IMPLANT FAILURES AND FACTORS AFFECTING IT -A REVIEW

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ABSTRACT

Dental implants are generally considered as a long-term successful treatment modality in a dental clinic. Though many cases have been reported in relation to the failure of implants. Successful implants are those which stay healthy in the oral cavity and also doesn't cause damage to its surrounding tissues. Recognizing any diseases or abnormalities in the oral cavity prior to the placement of the implant increases the chances of successful implant placement. Other factors such as age, sex, smoking, dietary habits, alcohol consumption and location of the abutment placement also play a major role in the success of implant placement. The follow up procedure after the implant placement is very important considering the longevity and stability of the implant.

INTRODUCTION

The dental implant is the gold standard in dental rehabilitation for missing teeth.[1,2] Different statistically analysed factors associated with implant failure are age and sex,[3] smoking,[4] systemic diseases,[5,6] maxillary location, bone type,[7] and implant surface treatments and characteristics.[8] Immunological[9] and genetic factors[10] have also been reported to be related to early implant failure. Implant failure is also associated with Periodontitis and cigarette smoking. Overall failure rates have been reported as 11% for smokers as compared to 5% for non-smokers. Mellado Valero et al. [11] found more failures in diabetic patients during the first year of functional loading. The failure of implant is seen in irradiated bone, excessive temperature elevation in bone during placement, resulting in necrosis of the supporting bone round the implant [12]

Age factor

Age is considered together of the important prognostic factors in implant success. Older patients are more prone to altered during the second and third decades of life when compared with the fourth and fifth.[16] The age and rate at which growth is complete is different from patient to patient.[17] The common growth spurt for males is 14 years and female is 12 years. However, this age can vary by the maximum amount as 6 years. Thus, when a dentist plan implant surgery for a child, the dentist should be aware of problematic age period that extends from 9 to 15 years for female and 11 to 17 years for male.[18]

Mesial drift of teeth within the maxilla and mandible

The spontaneous mesial drift of teeth within the step dentition phase is well understood. There is about 5 mm mesial movement/drifting of teeth within the lateral segment of jaw (canine to the primary molar) between 10 and 21 years aged. Thus, an implant can stop the mesial drift, leading to an asymmetric arch. An implant in the anterior part cannot follow the teeth and will become more lingual with time. Vertical growth can occur after puberty in slower phase. It may depend on many other factors like eruption of tooth pattern [19]

Oral hygiene and maintenance

The accumulation of bacterial plaque leads to gingivitis, periodontitis, and peri implantitis. Furthermore, the presence of any symptoms of infection, radiographic evidence of peri implant bone loss, and/or neuropathies reduced vascularity concomitant with parallel oriented collagen fibres may be indicative of

failing implant.[20] It is managed with the use of interproximal brushes penetrate 3 mm into the gingival sulcus, pocket. Evaluation of prosthetic component for plaque and calculus, stability of the implant abutment, peri implant tissue margin, implant body, and radiographs should be done after every 12–18 months in maintenance. oral lichen planus, parafunctional habits are some of the other clinical and conventional flap , flapless surgery, status of dentition, piezoelectric surgical technique and causes deleterious effects of smoking habit .

Bruxism

Glauser et al. evaluated 127 immediately loaded implants in 41 patients. Their results showed that implants failure is seen more in patients with bruxism than those placed in patients with no parafunction (41% vs. 12%). The higher failure rate among the bruxers is thanks to uncontrolled functional loading of the implant, which results in micro motions above the critical limit, leading to fibrous encapsulation of the implant instead of osseointegration. Early or immediate loading is not determine osseointegration if excessive micro motions occur at the bone implant interface during the healing phase leads to implant failure.[22]

The habit of cigarette smoking

Smoking also decreases calcium absorption. Smoking affects osseointegration process by lowering blood flow thanks to increased peripheral resistance and platelet aggregation. Smoking residues are carbon monoxide gas and cyanide, which delay wound healing capacity and alongside nicotine, inhibit cell proliferation rate. Tobacco directly inhibits osteoblast function. Strietzel et al. reported that smoking affects implant prognosis with/without augmentations. Studies show significant marginal bone absorption in smokers in comparison to non-smokers.[26] Loss of implant/graft material into the sinus The immediate implant insertion within the unstable residual bone can lead to the loss of implant or graft material into the maxillary sinus affecting the natural ciliary movement in the the maxillary sinus and mastication be managed surgically by different approaches, including intraoral, endoscopically, trans nasal route, and bone reconstruction of maxilla.[27,28]

Bisphosphonate related osteoradionecrosis

Bisphosphonates leads to active bone remodeling sites like jaws causing surgical trauma to the alveolar bone during implant surgery and further increases the postoperative accumulation of the drug to the implanted site. Bisphosphonates interfere with the bone formation and reduction and it reduce the peri implant bone resistance to oral flora, causing increased risk of peri implantitis.[29 31]

Injury to adjacent tooth

Improper placement of implant or an excessively large implant in which excess is space available can result in an injury to adjacent tooth, leading to non-vitality of the tooth. Dilacerated roots and excessive tilting in the mesiodistal direction corrupts the implant area and doesnt allow the perfect implant placement.[32] Alternatively, differences between the apical and crestal interdental spaces due to mesial or distal tipping of the roots are often orthodontically corrected.[33]

Peri implantitis

Peri implantitis may be a progressive inflammatory condition which affects the tissues surrounding an Osseo integrated implant, resulting in the loss of the supporting bone and implant failure. It is characterized by profuse bleeding, pus discharge, increased probing depth, implant mobility, and bone loss in radiograph. This inflammatory process is more severe, progresses faster and it goes down and affects alveolar bone too around the dental implant as compared to the inflammation around the adjacent natural tooth.[34] Peri implantitis can be caused by the most common pathogens like Cocci and nonmotile rods, sub gingival microflora leads to failure of dental implants. Soft laser irradiation is effective within the removal of the bacterial pathogens causing peri implantitis.[35] Systemic antibiotics for Gram negative anaerobes alters the microbial composition and clinically improves the

condition over a 1 year period.[36] To reduce bacterial count the local delivery devices such as Actisite which has fibres containing polymeric tetracycline hydrochloric acid can be used,.[37]

Hyperglycaemia

The osseointegration of dental implants is reduced in patients with increased blood glucose level. Parathyroid hormone gets altered in hyperglycaemic patients which helps in regulating the metabolism of phosphorus and calcium and inhibits osteoblastic differentiation. It effects bone matrix and its components, growth, adherence and accumulation of extracellular matrix. Hyperglycaemia may reduce bone recovery by 40% following circular osteotomies. Treatment with insulin normalizes this recovery index, thus indicating that the bone healing deterioration is strongly associated with poor diabetic control. Due to microangiopathy arising as a complication of diabetes the implant Failures that occur during the 1st year of functional loading or in second phase of surgery . This may compromise the vascularization of the flap, causing infection of sentimental tissue, and delayed wound healing. [38,39]

Irradiated bone

Malignant tumors in the craniofacial region is generally treated with irradiation alone with surgical excision. Hyperbaric oxygen therapy in irradiated patients prior to implant therapy increases the success rate of implants.[40] Osteoporotic patients Osseointegration failure in osteoporotic patients is due to the decrease in bone mass and density.[41]. In patients taking long-term bisphosphonates implant placement surgery should do with caution. [43,44]

Corticosteroid therapy

Reduced bone density, fragile epithelium, and decreases immune response is most common in patients under systemic corticosteroid therapy which in turn results in reduced osseointegration of the dental implant. Adrenal gland suppression rates and any medical intervention should be observed with caution.[45]

Immune deficiency

Frequent infection and constant tissue repair is seen in immune compromised patient. According to recent studies, dental implant placement has been performed successfully in patients with stable immune status, HIV-positive cases with a enough number of CD4+ cells and using antiviral drugs. [46,47]

Bleeding disorders

Uncontrolled haemorrhage can be caused by platelet disorders, coagulant factors deficiency, and using anticoagulant drugs such as aspirin and warfarin.[48] It is due to platelet deficiency It is due to platelet deficiency It is due to platelet deficiency <50,000/mm3.[49] The most life-threatening adverse effect of dental implant placement in these patients is upper airway obstruction.[50]

Cardiovascular disorders

Cardiovascular diseases interfere with healing and osseointegration process, resulting in reduced fibroblast activity, impaired macrophage function, and decreased collagen synthesis. [51] These pathologies include hypertension, atherosclerosis, and congestive heart failure. Cardiovascular disease does not have a significant influence on the long-term success rate of dental implant treatment.[52]

Organ (heart/liver/renal) transplantation

Patients having transplanted organs undergo long-term immunosuppressant medications to prevent graft rejection. Cyclosporine A is usually given in combination with steroids (anti-inflammatory action). Cyclosporine may have a negative impact on mechanical retention and healing of bone around the dental implant. [53,54]

CONCLUSION

There is a need to increase the knowledge and awareness regarding the potential risk factors that could impact on implant failures to those who are practicing dental implantology. This can be achieved through continuous dental educational programs and workshops. Regular assessment of the theoretical and practical knowledge of implant dentistry is mandatory to improve their implant experience.

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TONGUE LESIONS - A REVIEW

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ABSTRACT

Tongue is a vital organ within the oral cavity that has varied function,and it may act as an index for the underlying systemic diseases.The investigation of the tongue diseases may begin with mere clinical examination .This review is to highlight the signs and symptoms of the various lesions that affects the tongue and especially to talk in brief about the benign and malignant tumours that affect the tongue along with other inherited and congenital abnormalities.Tongue lesions are categorized as tumours,infections,reactionary,congenital,developmental,acquired,autoimmune and potentially malignant disorders for easy understanding and to arrive at appropriate diagnosis.Tongue playing an important role in maintaining the harmony in the oral environment,it should be treated from diseases.

Keywords: Tongue lesions, benign tumors, malignant tumors, diseases of tongue

CLASSIFICATION OF LESIONS AFFECTING THE TONGUE.

BENIGN TUMOURS OF THE TONGUE

- Capillary hemangioma
- Fibroma
- Cavernous hemangioma
- Giant cell granuloma
- Lipoma
- Lymphangioma
- Schwannoma

MALIGNANT TUMOURS OF TONGUE

- Squamous cell carcinoma
- Veruccous carcinoma
- Non-Hodgkin's lymphoma

TRAUMATIC/REACTIONARY LESIONS OF THE TONGUE

- Fibrous reactive hyperplasia
- Traumatic ulcer
- Pyogenic granuloma
- Frictional keratosis

INFECTIOUS LESIONS OF TONGUE

- Oral squamous papilloma
- Oral hairy leukoplakia
- Candidiasiis
- Median rhomboid glossitis
- Sublingual abcess

INHERITED, CONGENITAL, DEVELOPMENT AND ACQUIRED ABNORMALITIES OF TONGUE

- White sponge nevus
- Foliate papillitis
- Angina bullosa hemorrhagica
- Geographic tongue
- Fissured tongue
- Median rhomboid glossitis
- Bifurcated/tetrafurcated tongue
- Bald;depapillated tongue
- Papillomatous change

AUTO IMMUNE DISORDERS

- Lichen planus
- Vesiculobullous lesion

POTENTIALLY MALIGNANT DISORDERS

- Leukoplakia
- Oral submucous fibrosis

BENIGN TUMOURS OF TONGUE

Capillary hemangioma

Capillary hemangioma,a benign tongue lesion.Clinically,capillary hemangioma is a smooth or lobulated exophytic lesion manifesting as small, red erythematous papules on a pedunculated or sometimes sessile base, which is usually hemorrhagic

and compressible[1].Clinically the lesion is slow, asymptomatic and painless but it may also grow rapidly.Treatment includes observation for spontaneous remission and using topical, intralesional and systemic corticosteroids, cryosurgery, radiation, embolization and laser therapy.

Fibroma

Traumatic or Irritational fibroma is a common benign exophytic and reactive oral lesion that develops secondary to injury[2].Fibroma is a result of a chronic repair process that includes granulation tissue and scar formation resulting in a fibrous submucosal mass[3]. Recurrences are rare and may be caused by repetitive trauma at the same site. The lesion does not have a risk for malignancy.They may exhibit inflammation on the epithelial surface when traumatized .Surgical removal of the growth is needed, and the dental professional should evaluate any chronic habits that may be the causative factor.

Cavernous hemangioma

Cavernous hemangioma is formed by large, thin walled vessels, or sinusoids lined by epithelial cells separated by thin layer of connective tissue septa.It is almost never encapsulated. Treatment of the tongue vascular abnormalities is usually the preoperative endovascular embolization followed by the surgical resection of the lesion or partial resection of the tongue, depending on the size of the lesion.[4]

Giant cell granuloma

An exuberant reactive proliferation of multinucleated giant cells forming a mass on tongue.It is a reactive process with no malignant potential[5].Clinical features includes Reddish-blue rubbery nodules that range in size from a few millimeters to 3 cm.Treatment includes local surgical excision.

Lipoma

Lipomas are the most common soft tissue mesenchymal neoplasms and usually exhibit slow and asymptomatic growth.The lipoma is characterized by a significant fibrous component intermixed with the lobules of fat cells.Clinically, they usually present as well demarcated, slowly growing, painless, submucosal swellings which may appear yellowish in colour or similar in colour to the surrounding mucosa depending on the thickness of the overlying epithelium.Surgical excision is the treatment for these lesions. The recurrence rate for lipomas in tongue has been reported to be 3 – 62.5% .[6]

Lymphangioma

Lymphangioma is a rare, benign, congenital disease of unknown etiology that originates from lymph vessels [7].Tongue lymphangiomas typically demonstrate multiple blister like nodules or a pebbly surface that resembles a cluster of translucent vesicles on the enlarged dorsal surface of tongue.Lymphangiomas of tongue have been divided into four categories.This lesion is common and mostly occurs on the dorsal surface and lateral border of the tongue.The anterior two-thirds on the dorsal surface of the tongue is the most common site for intraoral lymphangiomas leading to macroglossia.Treatment is aimed at complete surgical excision. Partial surgical excision, injection of sclerosing solutions electrocoagulation, cryotherapy, embolization, steroid administration, radiation and laser surgery may be the other modalities of treatment of diffuse lymphangioma of the tongue

Schwannoma

Schwannoma is a benign, encapsulated, slow-growing and generally solitary tumour that arise from schwann cells of the peripheral nerve sheath[8].Schwannomas originate from schwann cells and are commonly encapsulated.Schwannomas of the tongue most commonly occur between the second and fourth decades of life and has no gender predilection, and often present as a painless

mass.Transoral resection is the standard approach for the treatment of the vast majority of these tumours. The recurrence rate is very low and malignant transformation is very rare.

MALIGNANT TUMOURS OF TONGUE

Squamous cell carcinoma

Squamous cell carcinoma of the tongue (SCCOT) is one of the most common tumors of the head and neck region.About 95% of oral cavity cancers are squamous cell carcinoma.Usually occurs between 50 - 70 years of age[9]. Usually occurs at the dorsal surface of tongue,base of the tongue and metastasize to submandibular and jugular nodes.The most commom sign of squamous cell carcinoma on tongue is a painless mass or ulcer that becomes more painful if secondarily infected.The tumour is usually superficially indurated with slightly raised borders and proceeds to develop a fungating exophytic mass,or to infiltrate the deep layers of the tongue producing fixation and induration without much change in the surface of the tongue.The posterior portion of the tongue is usually at a high risk of malignancy,metastasize earlier and offer a poorer prognosis due to inaccessibility to treatment. Metastasis occurs at a greater frequency in case of tongue cancer.Treatment procedure is usually surgical excision and radiation therapy.

Verrucous carcinoma

Verrucous carcinoma is a rare variant of well differentiated squamous cell carcinoma that has some unique characteristics.The neoplasm is usually exophytic and appears to be papillary in nature which has a pebbly surface which sometimes covered by white leukoplakic film.The lesions commonly have rugae-like folds with deep clefts between them[10].Regional lymph nodes are often tender and enlarged.Pain and difficulty in mastication are the common complaints. Treatment includes surgery or radiation.

Non hodgkin's lymphoma

Lymphomas are malignant neoplasms of the lymphocyte cell lines.Non-hodgkin's lymphoma (NHL) of the oral region is rare. It is usually identified by asymmetry of the tongue,on digital palpation a large sub-mucosal mass usually involves the lateral border of the tongue. The most common symptoms are swelling, pain and discomfort ,involvement of the intrinsic tongue musculature causing restriction of movement, dysarthria and dysphagia[11].Occasionally, the tumor may cause upper airway obstruction.The most common treatments for non-Hodgkin's lymphoma include chemotherapy,radiation,immunotherapy including monoclonal anti bodies tyrosine kinase inhibitors,stem cell transplant and surgery.

TRAUMATIC TUMOURS OF TONGUE

Reactive fibrous hyperplasia

Reactive hyperplastic lesions are tumor-like non neoplastic proliferations due to chronic irritation or trauma.The clinical appearance of reactive lesions is very similar to that of neoplastic proliferations.Clinically it is sessile or pedunculated mass.It is few millimeters in diameter, soft and painless, that gradually gets hard and sometimes pedunculated[12].Color may be similar to mucosa or vary depending on extent of inflammation.It is generally isolated.The lesions are asymptomatic.Recurrence rate is low and treatment is surgical excision.

Traumatic ulceration of tongue

If an ulcerative lesion lasts for two weeks or longer, it is considered chronic; otherwise, it is regarded as an acute ulcer.They are caused by mechanical damage, thermal, electrical, or chemical burns.Traumatic ulcers are most common on the tongue.These lesions may persist for a few days or even several weeks.The borders of traumatic ulcers are usually slightly raised and reddish, with a

yellowish-white necrotic pseudomembrane.Traumatic ulcers normally become painless within three days and after the injury has been eliminated , it mostly heals within 10 days.[13]

Pyogenic granuloma

Pyogenic granuloma or granuloma pyogenicum is a well-known oral lesion.Pyogenic granuloma of the oral cavity is known to involve the gingiva and tongue commonly[14].The etiology of the lesion is not known .It is said that pyogenic granuloma could possibly originate as a response of tissues to minor trauma and chronic irritation.Clinical features include a sessile lesion to an elevated mass. Pyogenic granulomas generally are soft, painless, and deep red to reddish-purple in color.Pyogenic granulomas have a relatively high rate of recurrence after simple excision.

Frictional keratosis

The clinical appearance can vary depending on the degree of trauma.The etiology of frictional keratosis includes mild abrasion of the mucosal membrane by sharp tooth,cheek and lip biting,irritation from masticatory function,due to constant rubbing of an external object eg.tobacco pipe or due to ill fitting or broken dentures[15].Linea alba is the term used to describe the white keratotic line on the buccal mucosa along the occlusal plane.The clinical findings can be of an ill-defined area of gray or white papules and plaques and may be associated with erosions and ulcers if the bite trauma is extensive.

INFECTIOUS LESIONS OF TONGUE

Oral squamous papilloma

Oral squamous papilloma is caused by HPV (Human Papilloma Virus).In oral cancers almost all lesions including the tongue are caused by HPV16, a subtype of the HPV virus.Researches indicates that approximately 70% of the oral cancers are caused by HPV16.Usually occurs as soft,painless pedunculated exophytic growth with numerous finger like projections that imparts a cauliflower like appearance.[12]Projections usually be pointed or blunted.Lesions are usually white,slightly red or normal in color of mucosa depending on the amount of surface keratinization.Treatment includes conservative surgical excision.

Oral hairy leukoplakia

Oral hairy leukoplakia is usually seen in HIV infected patients. OHL appears clinically as an asymptomatic white lesion on the lateral border of the tongue, unilateral or bilateral, with a flat, corrugated or hairy surface and non scrapable[16].The lesion is usually asymptomatic and microscopic examination shows hyperkeratosis, mild acanthosis, and a mild, chronic inflammatory infiltrate.Oral hairy leukoplakia appears to be caused by productive replication of EBV(Epstein Barr virus) in the oral mucosal epithelium, particularly of the lateral borders of the tongue.

Candidiasis

Candidiasis, is a fungal infection caused by Candida albicans.Oral candidiasis can be classified as pseudomembranous,erythematous,hyperplastic and angular chelitis.Most common are pseudomembranous and erythematous.Smoking has been recognized as the main risk factor associated with candidiasis[17].Smoking favors candidal colonization by causing localized epithelial alterations such as increased epithelial keratinization[18].Management of candidiasis involves the elimination of predisposing factors, chiefly smoking and antifungal therapy (both topical and systemic).

Median rhomboid glossitis

Median rhomboid glossitis (MRG) is defined as the central papillary atrophy of the tongue and it affects 0.01%–1.0% of the population.It occurs in the midline of the dorsum of the

tongue. It occurs as a well-demarcated, symmetric, depapillated area arising anterior to the circumvallate papillae[19].The surface of the lesion can be smooth or lobulated.It is usually asymptomatic.It gives persistent pain, irritation, or pruritus.A "kissing" lesion develops on the palate, directly opposite from the tongue lesion. This occurs due to the fungal organisms on the top of tongue being transferred to the palate during swallowing and similar movements.Predisposing factors associated with Median rhomboid glossitis are smoking, denture wearing, diabetes mellitus, as well as candidal infections[20].The most important diagnostic clue is the presence of candidal hyphae in the superficial epithelium. Treated using anti fungal drugs, including replacement of the toothbrush, and any other oral prosthesis that may cause reinfection by the organism.

INHERITED,CONGENITAL,DEVELOPMENT AND ACQUIRED ABNORMALITIES OF TONGUE

Foliate papillitis

Foliate papillae occasionally become inflamed or irritated, red in color, with associated enlargement and tenderness. These areas are enlarged, swollen and lobular with an intact overlying mucosa.It may occur as result of mechanical irritation of papillae, or as result of upper respiratory tract infection.Diagnosis is done by history of mechanical irritation to papillae site or history of upper respiratory tract infection,or by clinical features (inflammation of area, tenderness and color). The causative factors are treated to reduce the symptoms.[12]

Geographic tongue

Geographic tongue is a benign chronic relapsing recurring inflammatory condition of the oral cavity of unknown etiology.It can also be referred to as benign migratory glossitis, erythema migrans, annulus migrans, and a wandering rash of the tongue[21].It usually manifests as asymptomatic erythematous and migratory circinate patches that give its characteristic appearance of a map.The lesion persists for a period of several days to weeks and then disappears and reappears in a different location.Lesions usually occur on the lateral and dorsal aspects of the tongue.Psychosomatic and hereditary factors have been suggested to have a role in the etiology of geographic tongue.Geographic tongue has a slightly greater predilection for women than men.It is more commonly seen in patients with allergies to drugs, food, or others.Oral contraceptive pills that cause hormonal fluctuation have been associated with geographic tongue, further indicating a possible hormonal role in this disorder.Vitamin D, B6, B12, folic acid, iron, and zinc deficiency have also been proposed to play a role in the pathogenesis of geographic tongue.Geographic tongue usually does not require any treatment if it is asymptomatic.For symptomatic lesions, topical corticosteroids, antihistamines, cyclosporine, vitamin A, zinc, acetaminophen, topical tacrolimus have been shown to be effective. Avoidance of alcohol, hot, spicy and sour foods, acidic fruits and beverages, and maintaining good oral hygiene is recommended to avoid worsening symptoms.

Fissured tongue

Fissured tongue is a benign condition characterized by numerous shallow to deep grooves or furrows on the dorsal surface of the tongue.Aging, malnutrition and local factors such as infection may contribute to fissured tongue.Fissured tongue may have a familial occurrence and can be associated with certain underlying syndromes.The etiology is unknown but hereditary plays a significant role. The condition may be congenital, present at birth, or may become apparent during childhood or later in life.Aging and local environmental factors may also contribute to its development.Fissured tongue is diagnosed clinically on the basis of fissures. Based on the position of the fissures, fissured tongue can be classified as median and lateral

types.Fissured tongue is a benign condition and no specific treatment is indicated.Local measures to resolve the clinical manifestations can be done. The patient should be encouraged to maintain the oral hygiene and balanced diet.[22]

Glossitis

Glossitis is an inflammation of the tongue. The condition may present clinically as a painful tongue, as change in the surface appearance of the tongue changes in texture,color or both.The following could be the etiologies for glossitis. These include:
-Anemia
-Vitamin B deficiencies
-Infections(viral,bacterial,fungal,parasitic)
-Medications
-Psychological factors,Exposure to irritants
-Normal familial variants
-Mechanical irritation
-Poor hydration
-Down syndrome
-Psoriasis and other autoimmune conditions
-Burning mouth syndrome.
The history and physical examination are the most important evaluations in a patient with glossitis. Symptomatic treatment is possible with anti-inflammatory and analgesic mouth rinses.[23]

White sponge nevus

White sponge nevus is a rare benign autosomal dominant disorder with variable penetrance. It is characterized by asymptomatic white plaques affecting mainly the oral mucosa.Oral white sponge nevus appears as white or gray diffuse plaques thickened with multiple furrows and spongy texture located on buccal, labial, gingival mucosa and floor of the mouth.It is not most commonly seen in tongue and labial mucosa.[24]There is no treatment required in case of asymptomatic oral white sponge nevus.To reduce it clinically beta-carotene, antibiotics (penicillin, azithromycin, etc.), antihistamines, local applications of retinoic acid, tetracycline mouth rinses, surgical resection, and laser ablation, can be used as treatment modalities.

Lichen planus

Lichen planus (LP) is a chronic mucocutaneous disorder that affects oral mucousa. The etiology includes stress,systemic medications,dental materials,chronic liver disease and hepatitis C virus,tobacco chewing,graft versus host disease.
It is seen frequently in all regions of the oral mucosa, mostly noticed in buccal mucosa, gingiva and tongue. They are present bilaterally in most cases.Classically present as six types clinically: Reticular (fine white striae cross each other in the lesion), Atrophic (areas of erythematous lesion surrounded by reticular components), papular type, bullous type, plaque type, erosive or ulcerative type. The reticular type of oral lichen planus is often asymptomatic,only can be seen clinically. Ulcerative type in which erythematous areas are seen surrounded by reticular elements.Oral lichen planus is classically present as lesion with radiating whitish gray lines thread like papules, velvety appearance. They can be lacy or reticular, annular, patches or strings.Even though there is no specific treatment for oral lichen planus, symptomatic treatment is indicated. Corticosteroids provide relief and first choice of drug.[25]

POTENTIALLY MALIGNANT DISORDERS

Leukoplakia

Oral leukoplakia (OL) is the most frequent potentially malignant lesion.It was first defined by World Health Organization in 1978 as a white patch or plaque which cannot otherwise be characterized clinically or pathologically as any other disease.The etiology of oral Leukoplakia is considered multifactorial, but smoking is appreciated to be a frequently involved factor.Alcohol and conflicting results of studies related to the possible role of Human Papilloma Virus

infection.The clinical appearance of Oral Leukoplakia is classified in two main types,homogeneous type and non-homogeneous type which includes speckled and nodular.The homogeneous leukoplakia is a uniform, thin white area altering or not with normal mucosa. The speckled type is a white and red lesion, with a predominantly white surface.The clinical features of leukoplakia in tongue includes patches white or gray in color,thick or slightly raised,hardened and rough in texture.These patches may develop and change slowly over weeks to months. They are usually painless, but they may be sensitive to touch, heat, spicy foods, or other irritation.Patient should be advised to quit the habit.Treatment includes the surgical method that can use conventional surgery or laser ablation, electrocauterization, or cryosurgery.[26]

Oral submucous fibrosis

Oral submucous fibrosis (OSMF) is a chronic disease and potentially malignant condition that produces tissue fibrosis.The etiology of oral submucous fibrosis is mainly due to chewing areca nut.Oral submucous fibrosis occurs more often in women than men. The patient age range is 20–40 years(21).Clinical features include progressive inability to open the mouth (trismus) due to oral fibrosis[12].Oral pain and burning sensation on consuming spicy foodstuffs and increased salivation are present.The buccal mucosa is the most commonly involved site, but any part of the oral cavity can be involved.The features of the tongue involvement includes stiff and small tongue,blanched and leathery floor of the mouth.The treatment of patients with oral submucous fibrosis depends on the degree of clinical involvement. If the disease is detected at a very early stage, cessation of the habit is sufficient.Medical treatment is symptomatic and predominantly aimed at improving mouth movements. Treatment strategies include steroids weekly submucosal intralesional injections or topical application of steroids.Surgical treatment is indicated in patients with severe trismus and/or biopsy results revealing dysplastic or neoplastic changes.[26]

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EVALUATION OF ANTIULCER ACTIVITY OF SANGU PARPAM- AN EXPERIMENTAL STUDY

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ABSTRACT

*The Siddha System of Medicine is a traditional South Indian system of medicine. In the Siddha literature, Siddhar classified the diseases into 4448 and also mentioned the treatment for the diseases. Among the diseases that are classified, Gunmam is one of the diseases that is compared with Peptic Ulcer disease. As per the Siddha literature, Sangu Parpam is a unique medicine to treat the disease. Thus, the Sangu Parpam was prepared as per the literature and anti-ulcer activity was carried out by the methods of Pyloric ligation induced ulcer in rats and Ethanol/HCL induced ulcer in rats. The treatment with Sangu parpam shows a reduction in the gastric lesion area and promotes significant regeneration of the gastric mucosa in both methods. Thus, the traditional claim that Sangu Parpam is effective against peptic ulcers was proved. Further evaluation of the medicine is needed for its acceptance worldwide.**Keywords:** Peptic Ulcer Disease, Gunmam, Siddha, Sangu Parpam, Anti Ulcer Activity.*

INTRODUCTION

Traditional medicine has played an important role in meeting the demands of primary health care in many developing countries, and its use has expanded widely in many developed countries[1]. Siddha Medical System (SMS), also known as Indigenous Tamil Medicine, is a unique, significant, and scientific system that has been in use since time immemorial.The Siddha system of medicine deals not only with the external body but also with the internal soul.

The Alma Ata Declaration in 1978 at the WHO international conference on Primary Healthcare (PHC) advocated "the importance of integrating traditional practises as primary health care" and it also ascertained that "health is the state of complete physical, mental, and social well-being, not merely the absence of disease or infirmity".

Nowadays, modern lifestyle habits and fast-moving life have increased the rate of peptic ulcers[2.] Peptic ulcers are present in around 4% of the population. In 2013, nearly 53 million people developed peptic ulcers. 10% of people in the world develop peptic ulcers at some point in their life. In 1990, 327000 deaths were recorded, and in 2013, nearly 30000 deaths were recorded due to peptic ulcers.

Even with the advent of many advanced treatments for peptic ulcer disease, they all possess many side effects like cardiac arrhythmias, hypertension, and nephritis etc., [3]. Though the Siddha literature highly recommends **Sangu Parpam** for Peptic Ulcer Disease, the worldwide usage of this medicine will be on hand if the safety, efficacy, and mode of action of the medicine are established by standard scientific methods. In this work, **Sangu Parpam,** a herbo-mineral Siddha drug, is taken which is extensively used by traditional medicine practitioners.

Therefore, an attempt has been made to unveil the facts about the herbo-marine Siddha drug **Sangu Parpam[4],** a calcined product conch shell from the literature with anti-ulcer activity.

MATERIALS AND METHODS:

Preparation of Sangu Parpam (SP):

Purification of Sangu [5]:

Sangu was processed in the Thaalithal method (heating process**)** by covering it with Karchunnam (limestone).

Preparation process [4]:

100g of purified Sangu from each purification process was covered up by ground paste of Uthamani (*Pergularia damea*) and kept in the mud lid and closed by another mud lid. Cotton ribbon soaked in wet clay was winded over the rims of both mud lids and let to dry in sun light for 8 hours. Then this set up was subjected to Ganapudam. (100 cow cakes were used). After cooling, the set up was taken out and the calcinated Sangu was taken out, ground well, and stored in an airtight container.

Anti-ulcer studies:

Pylorus ligation method [6]:

Albino Wister rats of either sex weighing between 150 to 200gm were divided into six groups of 6 animals each.

Group I: Control (Ghee 5ml/kg)

Group II: Only pylorus ligation

Group III: pylorus ligation + Ranitidine 30 mg/kg body weight, oral.
Group IV: pylorus ligation + **SANGU PARPAM 9.36mg**/200gm
Group V: pylorus ligation + **SANGU PARPAM 46.8mg**/200gm
Group VI: pylorus ligation + **SANGU PARPAM 93.6mg**/200gm

"According to this method, the Albino Wister Rats were kept under fasting for 24 hours in metabolic cages and were taken care of in order to avoid coprophagy. In the control vehicle, three doses of SANGU PARPAM and the standard drug (Ranitidine 30 mg/kg) were given at different doses for five days orally. At the end of the fifth day, the animals were kept under fasting for 14 hours with water ad libitum. About 30 minutes before the ligation, SANGU PARPAM was administered to the animals. Under light ether anesthesia, the abdomen was opened and the pylorus ligated. Care was taken in order to avoid bleeding or to occlude blood vessels and the abdomen was sutured. The animals were then sacrificed after 6 hours of pyloric ligation under a surplus of ketamine hydrochloride, and the stomach was dissected out. Gastric juice was collected from the sacrificed animal and its volume, pH, free acidity, and total acidity were measured; the ulcer index was also determined. Evaluation of antioxidant enzymes, SOD, CAT, lipid peroxidation, myeloperoxidation, and histopathological evaluation were done on the excised stomach.

Ethanol/HCL induced ulcer method[7]:

Albino Wister rats were divided into 6 groups of 6 animals each. The animals were of either sex and were of nearly 150-200g in weight.
Group I: Control (Ghee 5 ml/kg)
Group II: Negative Control (HCL/Ethanol mixture containing 0.15 N HCL in 70% v/v Ethanol 1.5 ml) p.o
Group III: HCL/Ethanol+ Ranitidine 30 mg/kg body weight, oral.
Group IV: HCL/Ethanol+ **SANGU PARPAM 9.36mg**/200g
Group V: HCL/Ethanol+ **SANGU PARPAM 46.8mg**/200g
Group VI: HCL/Ethanol+ **SANGU PARPAM 93.6mg**/200g

"The animals were kept under fasting for 24 hours except for drinking water ad libitum until 2 hours before the start of the experiment. Gastric injury was induced with an acidified ethanol solution (150mMHCL/absolute ethanol) 40:60 v/v, (HCL/ethanol solution), as per a modification of the method. Ghee was administered orally to the normal control groups and normal saline was administered to the ulcer control groups. For the Reference group, 20mg/kg omeprazole was orally administered and for the experimental groups, oral administration of Sangu parpam 9.36 mg, 46.8 mg, 93.6 mg/200g was given. After one hour of this pretreatment, ghee and normal saline were

orally administered to the normal control group and the ulcer control group, respectively. Except for the normal control group, all the experimental groups were administered with HCL/ethanol solution (5ml/kg) orally for inducing gastric ulcers. With an excess of xylazine and ketamine anesthesia, the rats were euthanized 60 minutes after the treatment. Their stomach was immediately excised and the ulcer index determined. The anti-oxidant enzymes SOD, CAT, GPX, lipid peroxidation, and MPO were analyzed. [8].

RESULTS:

TABLE 1-EFFECT OF SANGU PARPAM ON FREE ACIDITY AND TOTAL ACIDITY IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30mg/kg	Pylorus+ S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
FREE ACIDITY	36.12±1.1	54.67±1.43#	39.50±1. 3*	40.72±1.6	40.13±1.02	40.16±1.12*
TOTAL ACIDITY	58.14±1.43	84.32±1.47#	59.10±1.5*	59.20±1.5	58.38±1.09	58.18±1.31*

TABLE 2 -EFFECT OF SANGU PARPAM ON GASTRIC pH AND GASTRIC VOLUME IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30mg/kg	Pyloru s+ S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
GASTRIC PH	2.3±0.20	1.23±0.16 #	2.58±0.06**	2.35±0.12*	1.93±0.1*	2.1±0.2*
GASTRIC VOLUME	0.68±0.11	4.83±0.4#	2.27±0.12**	2.48±0.33*	2.86±0.14 *	2.39±0.32 *

Values are expressed as the mean ± S.D: Control vs. Negative Control # P<0.05, Negative Control vs. Treatment * P<0.05 Std ** P<0.01

TABLE 3 - EFFECT OF SANGU PARPAM ON ULCER SCORE AND ULCER INDEX IN PYLORIC LIGATION METHOD

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 Negative Control vs SP II 93.6 ** P<0.01 Std ** P<0.01						
Group	**Control**	**Only pylorus**	**Pylorus+ Ranitidine 30 mg/kg**	**Pylorus+ S.P(II) 9.36mg/200g**	**pylorus+ S.P(II) 46.8mg/200 g**	**pylorus+ S.P(II) 93.6mg/200g**
ULCER SCORE	**0±0**	**7.91±0.19##**	**3.95±0.22** **	**6.10±0.14***	**4.78±0.14***	**4.78±0.18****
ULCER INDEX	**0±0**	**8.02±0.39##**	**6.13±0.16****	**7.66±0.22***	**5.13±0.09***	**3.93±0.10****

TABLE 4 -EFFECT OF SANGU PARPAM ON TOTAL PROTEIN IN PYLORIC LIGATION METHOD

Group	**Control**	**Only pylorus**	**Pylorus+Ranitidine 30 mg/kg**	**Pylorus+ S.P(II) 9.36mg/200g**	**pylorus+ S.P(II) 46.8mg/200g**	**pylorus+ S.P(II) 93.6mg/200g**
TOTAL PROTEIN (g/dl)	**0.76±0.00**	**0.72±0.00***	**0.47±0.00****	**0.82±0.00**	**0.78±0.00***	**0.71±0.00**

Values are expressed as the mean ± S.D; Control vs Negative Control * P<0.05 Negative Control vs Std ** P< 0.01 Negative Control vs SP II * P<0.05

TABLE 5 -EFFECT OF SANGU PARPAM ON ANTIOXIDANT PARAMETERS IN PYLORIC LIGATION METHOD

Group	**Control**	**Only pylorus**	**Pylorus+ Ranitidine 30mg/kg**	**Pylorus+ S.P(II) 9.36mg/200g**	**pylorus+ S.P(II) 46.8mg/200g**	**pylorus+ S.P(II) 93.6mg/200g**
SOD (Unit/min/mg protein)	**0.65±0.01**	**0.33±0.00#**	**0.55±0.01***	**0.48±0.00**	**0.52±0.00**	**0.54±0.00***
CAT (µmol of H202 consumed /min/mgprotein)	**0.90±0.00**	**0.61±0.00#**	**0.81±0.00***	**0.76±0.00**	**0.80±0.00**	**0.82±0.00***
GPX (µmoles of glutathione oxidized/min/mg protein)	**0.69±0.00**	**0.47±0.00#**	**0.59±0.00***	**0.51±0.00**	**0.53±0.00**	**0.54±0.00***

Values are expressed as the mean ± S.D; Control vs Negative Control ## P <0.01 Negative Control vs Std -Non Significant SP II * P<0.05

TABLE 6 - EFFECT OF SANGU PARPAM ON LIPIDPEROXIDATION IN PYLORIC LIGATION METHOD

TABLE 7 -EFFECT OF SANGU PARPAM ON MYELOPEROXIDATION IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30 mg/kg	Pylorus+ S.P(II) 9.36mg/200 g	Pylorus+ S.P(II) 46.8mg/200g	Pylorus+ S.P(II) 93.6mg/200g
LPO (nmol of MDA/mg protein)	0.69±0.02	0.83±0.00 ##	0.67±0.00ns	0.53±0.00	0.52±0.00	0.51±0.00*

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative Control vs Std * P< 0.05 SP II * P< 0.05

Group	Control	Only pylorus	Pylorus+ Ranitidine 30 mg/kg	Pylorus+ SP 9.36mg/200g	Pylorus + SP 46.8mg/200 g	Pylorus + SP 93.6mg/200 g
MPO(µmol/ mi n/mg tissue)	0.87±0.00	1.06±0.08#	0.75±0.02*	0.75±0.02	0.75±0.03	0.77±0.02*

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 Negative control vs Standard ** P<0.01 SP II *

TABLE 8 - EFFECT OF SANGU PARPAM ON ULCER SCORE AND ULCER INDEX IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol + Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
ULCER SCORE	0±0	11±0.36##	2.33±0.21**	4.33±0.42	4.33±0.56**	3.33±0.42*
ULCER INDEX	0±0	15±0.36 ##	3±0.36 **	7.33± 0.42**	5.33±0.56 *	4.03±0.42 *

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative Control vs Standard * P<0.05 SP II * P<0.05

TABLE 9 -EFFECT OF SANGU PARPAM ON TOTAL PROTEIN LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
TOTAL PROTEIN (g/dl)	50.67±3.6	74±9.89$^{\#}$	67±1.67^{ns}	48.67±2.56^{ns}	44.67±2.56^{ns}	34.33±2.08^{ns}

Values are expressed as the mean ± S.D; Control vs Negative Control #P<0.05 No Significant changes in Negative Control vs Standard Negative control vs SP II

TABLE 10 -EFFECT OF SANGU PARPAM ON ANTI OXIDANTS ENZYMES IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
SOD (Unit/min/mg protein)	0.4±0.07	0.14±0.01$^{\#\#}$	0.49±0.08	0.39±0.07 ^{ns}	0.38±0.02 ^{ns}	0.44±0.01 ^{ns}
CAT (µmol of H2O2 consumed/min/ m g /protein)	5.31±0.34	2.59±0.19$^{\#\#}$	4.20±0.22*	3.39±0.15*	4.09±0.05	4.59±0.22
GPX (µmoles of glutathione oxidized /min/mg protein)	7.2±0.06	3.49±0.10$^{\#\#}$	6.15 ±0.11*	5.18±0.09^{ns}	5.38±0.90	5.66±0.27

Values are expressed as the mean ± S.D; SOD: Control vs Negative Control ## P<0.01 Negative Control vs SP II Non Significant CAT: Control vs Negative Control ## P<0.01 Negative Control vs Standard * P<0.05 SP II * P<0.05 GPX : Control vs Negative control## P<0.01Negative control vs Standard * P<0.01 SP II * P<0.05

TABLE 11 -EFFECT OF SANGU PARPAM II ON LIPID PEROXIDATION LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g

LPO (nmol of MDA/mg protein)	4.49±0.21	13.63±0.36$^{\#\#}$	5.03±0.48^{ns}	5.03±0.13^{ns}	4.67±0.63^{ns}	5.15±0.11^{ns}

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 No significant changes between Negative control vs Standard and SP II

Group	**Control**	**Only HCL/ Ethanol**	**HCL/Ethanol+ Ranitidine 30 mg/kg**	**HCL/Ethanol + SP 9.36mg/200g**	**HCL/Ethanol + SP 46.8mg/200g**	**HCL/Ethanol + SP 93.6mg/200g**
MPO (µmol/min/ mg protein)	0.37±0.061	0.47±0.05$^{\#}$	0.29±0.012*	0.41±0.04	0.35±0.02	0.26±0.05*

TABLE 12 -EFFECT OF SANGU PARPAM ON MPO LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	**Control**	**Only HCL/ Ethanol**	**HCL/Ethanol+ Ranitidine 30 mg/kg**	**HCL/Ethanol + SP 9.36mg/200g**	**HCL/Ethanol + SP 46.8mg/200g**	**HCL/Ethanol + SP 93.6mg/200g**
Mucus weight (g)	0.52±0.02	0.27±0.02$^{\#\#}$	0.39±0.08*	0.42±0.01	0.29±0.01	0.27±0.01*
PGE2 (Pg/ml)	132±1.46	46.67±1.84$^{\#\#}$	87±1.67*	47±2.03*	64.33±1.17	70.67±1.12

Table 13 -EFFECT OF SANGU PARPAM II ON MUCUS WEIGHTAND PGE2 IN HCL/ETHANOL INDUCED ULCER MODEL

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative control vs Standard * P<0.01 SP II * P<0.01

Values are expressed as the mean ± S.D; Control vs Negative control ## P<0.01 Negative control vs Standard *P<0.05 SP II * P<0.05

the free acidity level when compared to the normal control group. (Table 1).

Pyloric Ligation Model

The animals treated with all the dose levels did not produce any significant weight variations throughout the study period.

The animals treated with SP at the dose of 9.36, 46.8 and 93.6mg/kg showed a statistically significant decrease ($P < 0.05$) in

The pyloric ligation group alone showed a marked increase in the total acidity level when compared to the normal control group, which is statistically significant (P 0.05).

In animals treated with Sangu Parpam in different doses, there was a statistically significant variation in gastric pH (P 0.05) and total volume of gastric juice when compared to normal control animals (P 0.05) (Table 2). The ulcer score as well as the ulcer index of the Sangu Parpam also showed a significant variation (P 0.01) (Table 3) when compared with the control group.

There is no significant variation in the total protein (Table 4) level of the Sangu Parpam treated group compared with the control group. In the ulcer induced group, the anti-oxidant enzymes SOD, CAT, GPX, LPO, and MPO were decreased when compared with the control group. SP and the control group both have an increase in antioxidant enzyme levels, which protects against ulcer formation and has anti-ulcer activity.(Table 5,6,7)

HCL / Ethanol Induced Method

The ulcer score was found to significantly increase in the ethanol induced group of animals when compared with the control group (P 0.01). The ulcer index also showed a significant increase when compared with the control group. (Table 8).

In animals treated with SP in different doses, there was a statistically significant decrease in ulcer score and ulcer index when compared with the ethanol induced ulcer group (P 0.01) (Table 9). The animals treated with Sangu parpam did not produce any significant variation in total protein levels. (Table 9)

The antioxidant enzyme SOD level did not change significantly.But the animals treated with 46.8mg/200g showed a significant increase (P 0.01) in catalase and GPX levels, while the 93.6mg/200g group also showed a significant increase in values (P 0.01). But the LPO and MPO levels did not show any significant variation. (Table 10, 11,12)

The animals treated with Sangu parpam as well as standard drugs showed a significant increase in mucus weight. (Table 13).

DISCUSSION

The study concluded that Sangu parpam has anti-ulcer activity in rats using the Pyloric Ligation Model. The antiulcer property of Sangu parpam in the pylorus ligation model is evident from its significant reduction in free acidity, total acidity, number of ulcers, and ulcer index[9]. Moreover, this SP significantly suppressed the formation of the ulcers. The significant inhibition of gastric ulcer in rats pre-treated with SP was comparable to that of ranitidine, which is a standard drug used for curing gastric ulcers (Fig.1). Sangu parpam treated animals decreased both the concentration and the pH, and increased the gastric wall mucus and gastric mucosa, so it is suggested that Sangu parpam can suppress gastric damage induced by aggressive factors. As per the study, SP shows significant anti-ulcer activity.

HCL-Ethanol Induced Ulcer Model

Peptic ulcers are caused by an imbalance between the protective and the aggressive mechanisms of the mucosa, and are the result of the association of several endogenous factors and aggressive exogenous factors that are related to living conditions. Sangu Parpam could significantly protect the gastric mucosa against HCL-Ethanol induced injury. Compared to the control group, the test drug showed a significant increase in protection of the gastric wall mucosa and also in ulcer area by inhibiting oedema and leukocyte infiltration of the submucosal area (Fig.2). The PGE2, SOD, and CAT levels of tissue homogenate reveal increased levels of antioxidant enzymes in the treated group. This study provides complete evidence that the SP possesses an anti-ulcer activity.

Conclusion

Sangu Parpam was taken for anti-ulcer studies. The studies revealed that Sangu

Parpam had a significant anti-ulcer activity in both ulcer models. This study shows a reduction in the gastric lesion area and promotes significant regeneration of the gastric mucosa. Thus, the Sangu Parpam sample confirms its anti-ulcer activity inboth the Pylorus ligation method and the Ethanol/HCL induced ulcer method. This research work justifies and confirms the traditional claim that Sangu parpam is one of the important medications for peptic ulcer disease.

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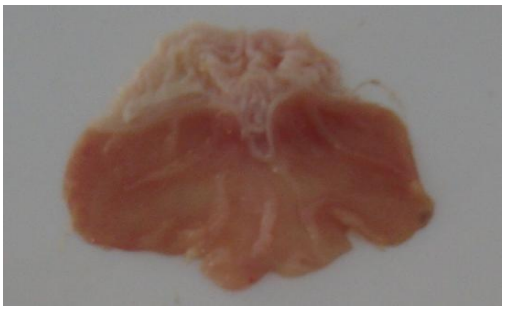
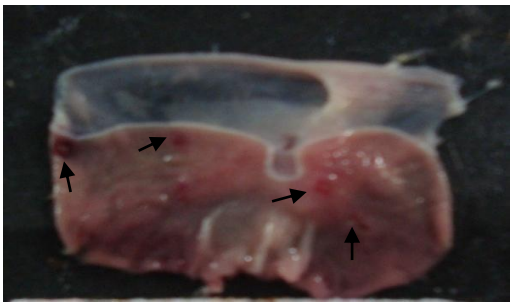
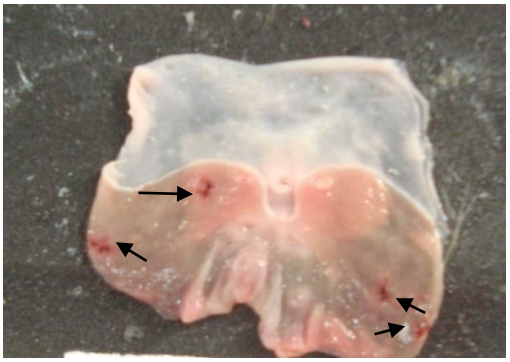
Fig 1: MACROSCOPICAL VIEW OF PYLORUS LIGATION (PL) INDUCED ULCER



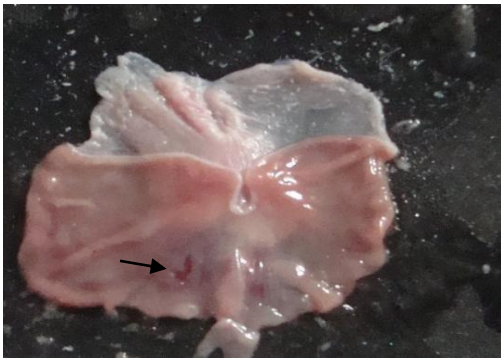
Control



Only PL shows severe damage of mucosal layer

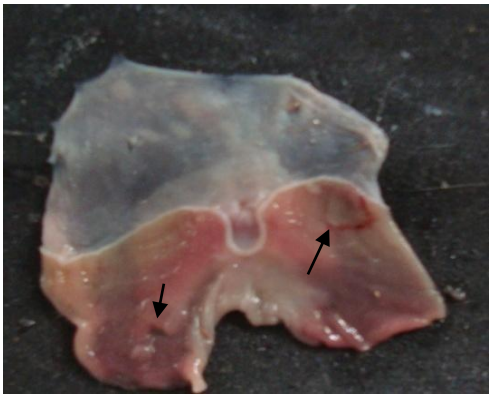


PL+ Ranitidine 30 mg/kg



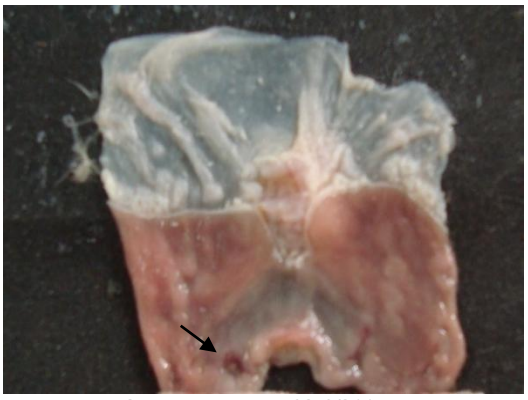
PL+ Sangu parpam -9.36mg/200g Shows

Moderately damage of mucosal layer



PL+ Sangu Parpam 46.8mg/200g Shows

Moderately damage of mucosal layer

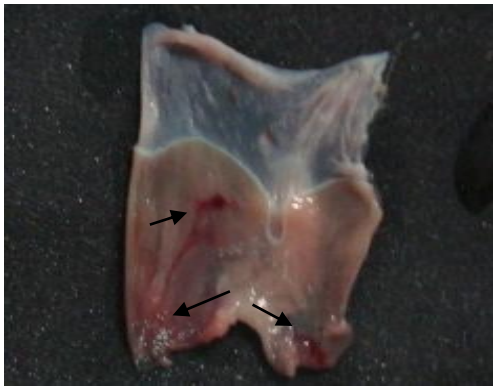


PL + Sangu parpam - 93.6/200gm

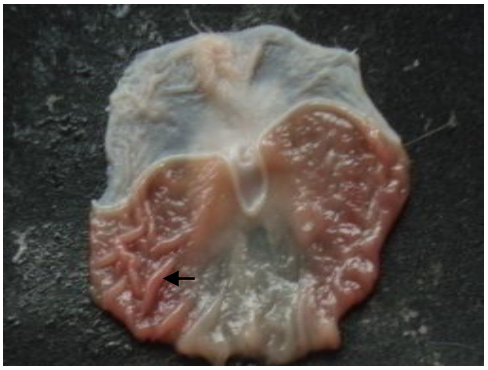
Shows Protected mucosal layer

Fig 2: MACROSCOPICAL VIEW OF THE GASTRIC MUCOSA IN HCL/ETHANOL INDUCED ULCER

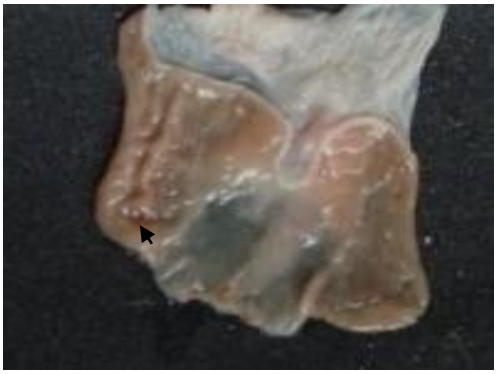
CONTROL



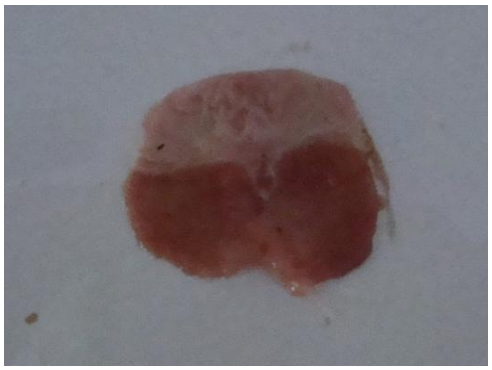
HCL/ ETHANOL



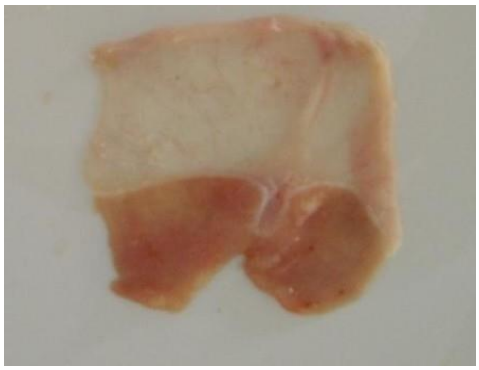
HCL/ ETHANOL+ RANITIDINE



HCL/ ETHANOL +SANGU PARPAM 9.36mg/200g



HCL/ ETHANOL + SANGU PARPAM 46.8mg/200gm



HCL/ETHANOL + SANGU PARPAM 93.6mg/200gm

THE EXIGENCY OF THIRD EYE FOR THIRD HAND SMOKE EXPOSURE IN CARDIOVASCULAR SYSTEM

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ABSTRACT

Environmental Smoke or Third hand smoke [THS] can affect heart and blood vessels that increase the risk of coronary heart diseases by 30% . Everyday dentists meet patients with tobacco smoking habit and some with precancerous lesions. Dentists are people who are most aware of the hazards caused by smoking. It is a need and responsibility of dental professionals to also be aware of various levels of smoke exposure and currently of Third Hand Smoke exposure. This article reviews on the nature and effects of THS, particularly on the cardiovascular system and ways to deal with THS exposure in our day to day lives.

Key Words: Second hand smoke, Third hand smoke, Hazards, Cardiovascular system

INTRODUCTION

More than a billion smokers exist around the world and about 12% of them are in India. Mainstream smoke [MSS] or First Hand Smoke is the primary level of smoke exposure, inhaled completely by the smoker. The exhaled smoke and smoke from the burning end of a cigarette (also called Side Stream Smoke or SSS) form the Second Hand Smoke. Third hand smoke [THS] is a relatively new term described by Jonathan Winickoff, a paediatrician at Dana-Farber/Harvard Cancer Centre, which refers to the tobacco smoke contamination that persists long after the cigarette has been extinguished. The authors emphasized on the potential hazards to children[1]. The article stated that children are particularly susceptible to THS exposure because they breathe and play near contaminated surfaces. However there are no conclusive studies linking THS with cancer, except for one study conducted on mice exposed to third hand smoke causing lung cancer[2].

TYPES OF SMOKE EXPOSURES

A cigarette is basically a cylindrical object made of tobacco scraps, fillers, and glue and sprayed with nicotine that is wrapped in a paper and lighted at one end to produce smoke. After lighting the cigarette, the smoke that is directly inhaled by the smoker through the opposite end is termed as 'First-Hand Smoke". The exhaled smoke and smoke released through the burning end is referred to as `Second-Hand Smoke`. Third hand smoke is essentially second hand smoke that is left on surfaces and progressively becomes toxic over time.

THIRD HAND SMOKE

Third Hand Smoke or THS is the residual second hand smoke that remains in surrounding area for months[1]. THS can cling to fabric from the clothes of the smoker, walls, carpets, furniture, hair, toys and vehicle surfaces. THS is a growing concern because unlike first and second hand smoke, THS affects unsuspecting common people, especially children.

Cigarette smoke contains a number of chemical components, particularly the notorious nicotine among many others. National Toxicology Program demonstrated the toxicity of low levels of cigarette constituents which included 250 poisonous gases, chemicals and metals, such as, hydrogen cyanide, carbon monoxide, ammonia, butane, arsenic, chromium, cadmium, polonium and toluene[3]. These compounds are well known carcinogens. A study conducted by the researchers of Lawrence Berkeley National Laboratory revealed Nicotine as a potential health hazard due to its ability to cling to surfaces and react with nitrous acid to produce harmful carcinogens[4]. It is exactly this ability of nicotine that forms the crux of THS. Nicotine reacts with ambient gases to form carcinogenic tobacco-specific nitrosamines or TSNAs, 4-(methylnitrosamino)-1-(3-pyridinyl)-4-butanal) or NNK, cotinine and formaldehyde. An experiment conducted on humans by exposing them to THS revealed changes in human nasal epithelium, THS induced cell survival responses, including up regulation of DNA repair genes, increased mitochondrial activity and inhibition of cell death[5]. Furthermore, THS components are suspended into air again and again over time, even after many days of the cessation of smoking activity[1].

The most dangerous part about THS exposure is that it affects non-smokers. Matt GE et al, in a study, concluded that dust and surfaces in homes of smokers are contaminated with environmental smoke or ETS. Not just smokers` homes but second hand homes of smokers, cars and even smokers` clothes retain harmful THS components that are inhaled, ingested or absorbed by innocent bystanders[6].

DIFFERENCE BETWEEN FIRST HAND SMOKE AND SECOND HAND SMOKE

First hand and second hand tobacco smoking are the two greatest causes of many illnesses and even death. First hand smoke refers to the smoke inhaled directly by the smoker and causes cancer, heart disease, stroke, lung diseases, diabetes and chronic obstructive pulmonary disease. On the other hand second hand smoke is the inhalation of exhaled smoke or smoke from burning end of a cigarette by non-smokers. SHS can cause heart disease, lung cancer and stroke in adult. Children and babies, because of their small bodies are more vulnerable to SHS. Babies who breathe SHS can die unexpectedly of sudden infant death syndrome (SIDS). [7] Second hand smoke can affect an individual much similar to MSS but on the other hand THS persists long after burning a cigarette, for months the toxins get accumulated, react with ambient gases and reemit in a toxic cycle.

IMPACT ON CARDIOVASCULAR SYSTEM

Cigarette smoking is a major risk for coronary heart disease. Both active and passive smoking seem to increase the risk of acute coronary thrombosis and myocardial infarctions. The chemicals in smoke alter the haemostatic mechanisms by alteration of endothelial cells, platelets, fibrinogen and clotting factors. In a study on mice exposed to THS it was found to cause increased haemostasis and a consequential increase in blood clots. The researches at Western University of Health Sciences in Pomona, California concluded that similar reactions in humans could lead to acute coronary thrombosis, which can obstruct blood flow to heart and lead to a heart attack[8].

EFFECT OF SMOKING: ATHEROTHROMBOTIC EVENTS

Atherosclerosis is the build-up of arterial wall plaques and it begins due to certain molecular changes in the innermost layer of arterial vessel wall called the tunica intima. The intima is lined with endothelial cells. Endothelial injury promote deposition of lipoproteins into the vessel wall. Monocytes infiltrate the site and endocytosis of lymphocytes occur leading to the formation of foam cells. Inflammatory mediators and smooth muscle cells migrate into the intima to promote plaque formation. Subsequently blood clot is formed which occludes the vessel leading to thrombosis[9].

This process of thrombosis is influenced by nicotine and other toxins present in cigarette smoke. Several studies[10] show that cigarette smoke increased vascular smooth muscle proliferation and migration due to the activation of the platelet-derived growth factor-protein kinase C signalling cascade. This causes hypercoagulability of blood and hemostasis[11].

Nitrous oxide production as a result of endothelial dysfunction decreases the flow mediated dilation [FMD]. This was shown in a study where the participants were exposed to THS[9].

HYPERTENSION

Nicotine can influence the release of catecholamine's by stimulation of the sympathetic nervous system via nicotine acetylcholine receptors present on peripheral postganglionic sympathetic nerve endings and the adrenal medulla[12].

Furthermore, nicotine acts on nicotinic cholinergic receptors present on endothelial cells to cause hypertension. The nicotinic acetylcholine receptors [nAChRs] are arranged in a barrel-like configuration to form a channel in the cell membrane. On activation of nAChRs the permeability to cations is increased leading to hypertension[13].

OTHER POTENTIAL HAZARDS MOLECULAR LEVEL

Genotoxicity is the ability of chemicals to produce damage to genetic information in a cell causing mutations and eventually cancer. THS and its residual components are such chemicals. Hang B et al conducted an experiment which used two different assays to evaluate the in vitro genotoxic nature of THS and its component NNA. Both the assays revealed that THS causes significant levels of DNA damage in human cells. It suggested that THS exposure is related to increased oxidative stress and could be a contributing factor in THS mediated toxicity[2].

DISEASES IN ADULTS

CANCER

Cigarette smoking is a common cause of cancer. It can cause cancer of mouth, throat, oesophagus, stomach, colon, rectum, liver, pancreas, larynx, trachea, bronchus, kidney, urinary bladder and acute myeloid leukaemia. Second hand smoke also causes lung cancer. Researchers have identified THS to increase lung cancer risk in mice. Antoine Snijders, Jian-Hua Mao and Bo Hang of Berkeley Lab reported in 2017 that brief exposure to THS is associated with low body weight and immune changes in juvenile mice[2].

These studies indicated that THS exposure induced molecular level damage, breakage of DNA double strands, increased cell proliferation and colony formation.

FATTY LIVER DISEASE

Non-alcoholic fatty liver disease [NAFLD] is one of the most common chronic liver diseases. Tobacco smoking is significantly associated with causation of NAFLD[14]. Animal studies have shown that THS stimulated the accumulation of fat in liver cells which caused non-alcoholic fatty liver disease[15]. NAFLD is known to worsen into cirrhosis and liver cancer.

POOR WOUND HEALING

Smoking in general is associated with interference in wound elasticity and poor wound healing which was also found to be true in the case of THS exposure. THS interferes with scar tissue development and wound contraction[14].

INSULIN RESISTANCE

Smoking and insulin resistance have a dose-dependent association[16]. Nicotine may be the potential cause; it indirectly causes insulin resistance mainly via hormone activation. Studies in mice have found that there is oxidative damage to insulin receptors caused by THS leading to reduction of insulin receptors on pancreatic cells and insulin resistance. As a result there is risk of diabetes in prediabetic or non-diabetic individuals[15].

PULMONARY FIBROSIS

Pulmonary fibrosis [PF] is a chronic illness affecting the respiratory system, is

characterized by thickening of lung tissue and scar tissue formation. Smoking is a main risk for PF. Animal studies suggest that THS affects collagen production in bronchioles leading to scar tissue formation[15]. This leads to complications such as asthma, chronic obstructive pulmonary disease or cystic fibrosis[17].

PREVENTION AND MANAGEMENT

Residual smoke is hard to manage as it is immune to traditional cleaning methods. Prevention is the best cure even in this situation.

- Banning cigarette smoking in public places is an effective method along with an additional waiting period of 10 minutes before entering indoor to prevent THS exposure[3].
- Opening windows do not offer sufficient protection against second and third hand smoke exposure, hence it is vital for smokers to move far away from children when they are smoking.
- Smokers must consistently get rid of their clothes that they used during smoking. People who have quit smoking must replace the rags and furniture at their houses, repaint the walls of their homes[18].
- Education of public is another effective method to prevent THS exposure.
- Last but not the least is 'quitting the habit'. Adults who smoke have to be counselled and given enough motivation to quit smoking. Nicotine replacement therapies can be used to aid the process[3].

CONCLUSION

It is very difficult to bring this review to a conclusion as THS is a phenomenon that is not completely understood and requires a lot of research. It is established that THS accumulates in households, cars and even on clothes. It can stay in surrounding for months and slowly remits into atmosphere producing harmful carcinogens. . It affects particularly non-smokers and children. The influence seen on the cardiovascular system is dangerous and potentially life threatening. Preventive modalities are very little and have scope for newer concepts and innovations. Many studies have been made showing molecular effects of THS compounds and potential for causing cancer however there is scope for further research in this topic.

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RESPONSE OF DIABETIC WOUND TO PACHAI ENNAI IN SIDDHA SYSTEM --A SINGLE CASE STUDY REPORT

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ABSTRACT

*People with diabetes mellitus can develop many different complications. Diabetic wounds are the most dreaded complication of diabetes mellitus due to peripheral neuropathy and peripheral arterial insufficiency. Diabetic foot disease is associated with major morbidity, mortality, and also a reduction in a person's quality of life. In Siddha literature, a diabetic foot ulcer (DFU) is referred to as a Madumega pun (a diabetic wound). A 55-year old female, post-menopausal woman, house wife was admitted to the Inpatient department of Sairam Siddha medical college & research center and hospital with complaints of an ulcer present in the big toe (plantar surface) of her right foot with pain. Pain, mild to moderate oozing, and mild swelling in the peri-wound area have changed gait for the past year, related to a known case of diabetes for 5 years. While examining the wound, which is unhealthy, no significant granulations seem to be covered with necrotic tissues that show a predominantly black color, which measures about 1.5*2.5*1cm in size. The severity of the ulcer was measured by the diabetic ulcer severity score (DUSS), classification of the ulcer by the 'Wagner Ulcer Classification system, and prognosis by the 'Leg Ulcer Measurement Tool. To draw special attention to DFU and to focus on siddha medication internally and externally with Madumega Chooranam-Anti diabetic siddha drug and Pachai Ennai (matthan thylam-wound healing external oil) respectively, The use of siddha medicine has the potential to heal nearly 90% of diabetic wounds (diabetic foot ulcers) and result in lower blood sugar levels.They may perhaps have a huge impact on the healing process, falling infection rates, amputations, plastic surgeries, and humanising the overall quality of life, reducing the monetary burden of treating DFU.*

INTRODUCTION

Nowadays, diabetes melitus is a common disease worldwide. Global diabetes prevalence in 2019 is estimated to be 9.3% (463 million people), rising to 10.2% (578 million) by 2030 and 10.9% (700 million) by 2045. The prevalence is higher in urban (10.8%) than in rural (7.2%) areas, and in high-income (10.4%) than in low-income countries (4.0%). About 422 million people worldwide have diabetes, the majority living in low-and middle-income countries, and 1.6 million deaths are directly attributed to diabetes each year. Both the number of cases and the prevalence of diabetes have been steadily increasing over the past few decades.

People with diabetes mellitus can develop many different problems, sometimes leading to severe complications like diabetic neuropathy, nephropathy, and retinopathy. Diabetic wounds are the most dreaded complication of diabetes mellitus due to peripheral neuropathy and peripheral arterial insufficiency. Diabetic neuropathy is nerve damage in diabetic patients, resulting in diabetic foot.It may lead to a secondary complication in the form of DIABETIC FOOT ULCER, which is an open wound commonly located on the bottom of the foot. In Siddha literature, diabetic foot ulcer (DFU) is referred to as Madhu mega pun (Diabetic Ulcer) or "Diabetic Ulcer" in Siddha literature. DFU is defined as a foot affected by an ulcer associated with neuropathy.

Diabetic foot disease is associated with major morbidity, mortality, and also a reduction in a person's quality of life. Around 15% of people with diabetes will develop foot ulceration during their lifetime, and 5–24% of them will eventually lead to limb amputation within a period of 6–18 months after the first assessment. MaththanTaylam (pachai ennai), a herbo-mineral classical Siddha formulation [1], is used as a remedy for healing suppurative wounds and is very useful in healing diabetic ulcers. PACHAI ENNAI is one of the best siddha medicines to cure a diabetic wound and prevent major complications like amputation.

This case study report outlines the response of siddha medicine to the successful management and healing of a diabetic wound. Here, we report the case of Madhu mega Pun, or (chronic non-healing diabetic foot ulcer) on the plantar surface of the right big toe, which was treated with internal and external Siddha medicine. The case patient was treated internally by Madhu mega chooranam (an Anti-Diabetic Siddha drug) and externally by Patchai ennai-Maththan Thailam (Wound Healing Siddha external oil).

Declaration of Patient consent

The authors certify that they have obtained all appropriate written informed consent from the patients for the publication of this case report and accompanying images

CASE HISTORY

Name : XXXXXX
Age :55 Years
Sex : Female
Marital Status :Married
Menstrual History : Attained Menopause
Complaints : An Ulcer on the plantar aspect of big toe measuring about 1.5x2.5x1 cm size since 1 year.DM since 5 years.
O/E :Mild pain and swelling,Mild to moderate oozing, No significant granulationsseem to be covered with black colour, Necroticfibers predominantly
Place :Sri Sai Ram Siddha Medical College Hospital, Tambaram

CASE REPORT

A 55-year-old female housewife from the uptown area of Chennai, Tamil Nadu, was admitted to the in-patient department of Sri Sai Ram Siddha Medical College Hospital, west Tambaram, for 1 year with complaints of altered gait, mild pain and swelling in the peri-wound area, no significant granulations, and appearing to be covered with black colour necrotic granulations.

The patient was admitted to the IPD female ward, and the wound was cleaned and dressed with Maththan Thaylam after completing the appropriate examinations. The wound was analyzed in the following three ways:

The severity of the ulcer was measured by the DUSS (Diabetic Ulcer Severity Score) 14 In the DUSS assessment, the patient had pedal polices, probing to bone, and a toe ulcer numbered in singles. He scored about 1 out of 4.

1. Classification of Ulcer was measured by the ‘Wagner Ulcer Classification System (WUCS) 15, In the WUCS assessment, the patient has Grade 2 (ulcer extends to ligaments, tendons, deep fascia without abscess).
2. Characteristics of Ulcer and patient satisfaction were measured by the ‘Leg Ulcer Measurement Tool (LUMT) 16. Clinician Rated Domains it has 14 assessment questions rated by the clinician. Score about 20/56 and Patient Rated Domain (PRD) it has 3 assessment questions rated by the patients. Score about 4/12.

On the second day, blood and urine samples were collected for investigations, and the daily wound was cleaned with Thiripala wash and dressed with Maththan Thaylam. According to the Siddha structure, purgative is the early process to counter balance the Mukkutram (Three humours in Siddha). The patient was advised to take the oral route of Vellai Ennai – 15 ml for the mild purgative on the early morning of the third day. Following that, motion gut rest was advised for five times the loose stools. For oral administration on the fourth day, 2 g twice a day is recommended for Mathu mega Chooranam. Siddha medicines were ordered for 8 weeks. (Table 1).

Every day, the ulcer was cleaned and dressed. The oozing was stopped completely, and the pain and swelling in the peri-wound area were reduced.The depth of the ulcer was reduced, and granulation tissues developed around the

edges. Swelling and pain in the peri-wound area disappeared; gait returned to normal in the 4th week. The depth of the wound was closed. Necrotic tissues completely disappeared and granulation tissues covered the wound area in the 6th week of treatment (Figure 4). In the eighth week, blood and urine were collected for the investigation. There was a reduction in raised ESR and a raised level of haemoglobin (Table 2). In biochemical markers, blood sugar levels come to a normal limit (Table 3).

On the day of discharge (Figure 5), the ulcer was analysed by the measuring tools of DUSS, WUCS, and LUMT. DUSS scores nil, WUCS scores zero, and LUMT scores four out of fifty-six. The whole restoration of the wound and corresponding ongoing treatment was prescribed on the day of discharge. This exposed good results. The same medicines were continued for 2 months in the follow-up period. During the follow-up period, no recurrence was observed. No adverse drug reactions were observed during treatment and the follow-up period.

TREATMENT

- Mathumegachooranam – 2 gm. bd with Water
- Thiripala chooranam – External wash
- PACHAI ENNAI – Dressing the wound
- Treatment Period – 2 months

Table 1: Siddha Treatment & Observation

Day 1	Patient was admitted in IPD Female ward. Wound was cleaned with Thiripala wash and dressed with Maththan Thailam regularly
Day 2	Blood and Urine sample were collected for investigation
Day 3	Vellai Ennai 15 ml was given at the morning as a single dose for purgation therapy to regulate the Mukkutram (Three humors)
Day 4	Mathumega Chooranam -2gm twice daily with watergiven orally.
Week 1	Mild oozing in the wound was completely stopped.
Week 2	Pain and Swelling in the Peri-wound area was reduced
Week 3	Swelling and painin the peri-wound area were disappeared
Week 4	Depth of the wound was reduced, granulation tissues developed in the edges of the ulcer
Week 6	Blood and Urine samples were collected for the Investigations Necrotic tissues were completely disappeared,
Week 7	granulation tissues were covered the wound area
Week8	Wound was completely closed by the epithelial cells
Follow up	No recurrence was observed

Table 2.Comparative HematologicalParameters of the patient

Hematological Parameters	At the time of Admission	Before Discharge
Total count (cells/cumm)	8000	8200
Neutrophil %	65	70
Lymphocyte %	30	28
Eosinophil %	6	3
Basophil %	0	0
Monocyte %	0	0
ESR (mm/hr.)	40	20
Haemoglobin(gm%)	8.6	10.2

ESR*Erythrocyte Sedimentation Rate

Table 3.Comparative Bio Chemical Parametersof the patient

Biochemical Parameters	At the time of Admission	Before Discharge

FBS (mg/dl)	180	90
PPBS (mg/dl	318	176
HbA1c (%)	7.3	5.8
Urine Examination		
Albumin	-	-
Sugar	+ + + +	Nil
Deposit	Pus cells – occasionally Epithelial cell – occasionally Casts & crystals- Nil	Pus cells – Nil Epithelial cell – occasionally Casts & crystals- Nil

*FBS: Fasting blood sugar, PPBS: Postprandial blood sugar, HbA1c: glycated Hemoglobin

Fig.1Day I

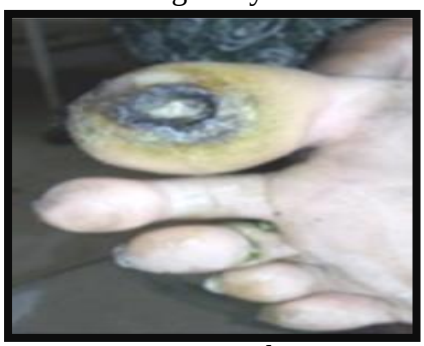

Fig.2 Week II

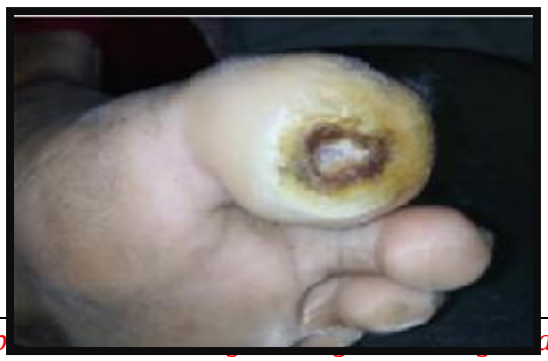

Fig. 3Week IV

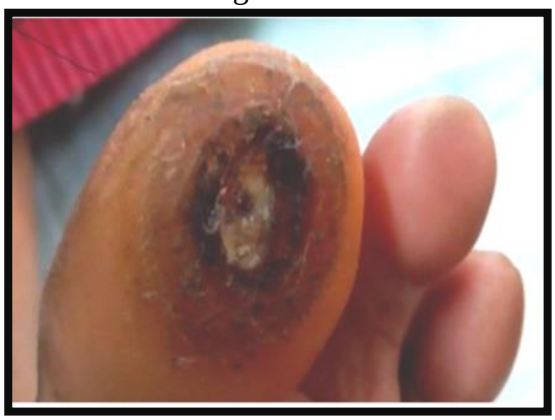

Fig:4 Week VI

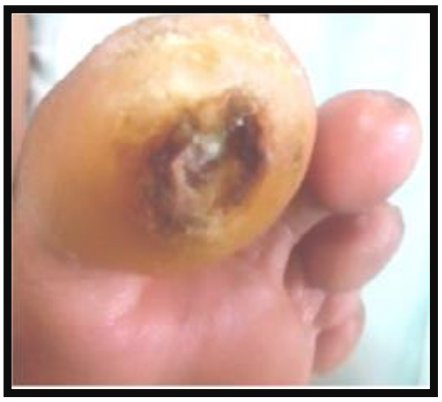

Fig.5 Week VIII

Diabetic wound is healed 95% at the end of treatment

DISCUSSION

In this intervention, Pachai Ennai is the commonly used Siddha topical medicine for wound healing; quoted as Maththan Thaylam in the Siddha literature, for external medication of chronic and non-curable ulcers.

The ingredients of Maththanthailam are coconut oil, copper sulphate, Datura metel, and Acalypha indica. A histopathological study found increased fibroblast proliferation and neovascularization in Coconut Oil-treated wounds compared to controls19. Datura metel extract was used in the wound and has significant antibacterial activity against Staphylococcus aureus and Pseudomonas aeruginosa 20.And copper is an essential mineral that plays an important role in angiogenesis, skin generation and representation, and extracellular skin protein maintenance 21. Acalypha indica plant

extract has adequate wound curing properties 22.

So, this case study confirms the response of the diabetic wound healing process of Maththan Thailam-Pachai Ennai. It has satisfactory improvement in the non-healing chronic diabetic foot ulcers based on the assessment tools of LUMT, DUSS, and WUCS, which compared the before and after treatment. The blood sugar level fasting was 90 mg/dl and postprandial was 176 mg/dl, extremely low compared to the initial levels. The patient's QOL was acceptably improved. 95 % of the wound was healed with normal gait, absence of pain, and the patient's QOL was acceptably improved.

CONCLUSION

Intervention by Siddha medicine can potentially cure or decrease the size of the foot ulcers associated with diabetes. As a result, PACHAI ENNAI is the best siddha medicine for treating diabetic wounds and preventing major complications such as amputation. They could have a significant impact on reducing infection rates, amputations, and plastic surgeries, as well as improving overall quality of life and lowering the economic burden of treating DFU.The greatest outcome from this case study is the control of diabetic complications and short-term improvement from DFU at a low cost. in comparison to other medical systems

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IMPLANT SURGERY: WHAT CAN GO WRONG?

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ABSTRACT

Implant surgery complications are frequent occurrences in dental practice and knowledge in the management of these cases is essential. The aim of this review was to highlight the challenges of treatment plan-related, anatomy related, and procedure-related surgical complications as well as to discuss the etiology, management and treatment options to achieve a satisfactory treatment outcome. (Implant Dent 2008;17:159– 168)

Key Words: *dental implants, implant complications, implant failures.*

Introduction

Surgical complications during implant placement are not uncommon. According to a retrospective study by McDermott et al, 1677 patients (2379 implants) were investigated, and an overall frequency of complications was 13.9%. Operative complications made up a mere 1% of the overall, whereas inflammatory and prosthetic complications were 10.2% and 2.7%, respectively.

Planning complications.

1. Lack of a proper history

Well organized, thorough treatment plans lead to successful implant treatment and patient satisfaction, which are the ultimate long-term goals. Patient selection is one of the most important determinants of success or of failure. Predictability of implant success can be jeopardized by absolute and relative risk factors. Therefore a complete medical record and patient analysis post treatment play a significant role.

2. Errors in angulation

Implant angulation is yet another determinant for implant success. Proper angulation should be determined according to the future prosthesis with the consideration of bucco-lingual, apicocoronal, and mesio-distal positions. Surgical guides can help control the implant placement angle if they are made and used correctly.

Mandibular teeth in the natural dentition are lingually inclined in relation to both the mandibular base, specifically as 109 degrees, as well as the maxillary opposing arch dentition (eg, lingual cusp buccal inclination)and therefore implants should be placed at a similar inclination. Failure to do so may result in perforation of the lingual concavity, constriction of the lingual space or damage of the lingual artery.

Teeth adjacent to implant sites and surgical guides with long drill channels, often require the use of drill extensions and maximum opening by the patient which may be strenuous. Short breaks to relieve muscle tension, using a bite block and having the patient shift their jaw to the opposite side can help ensure the correct angulation of the drill. Yet another issue is the finger placement. Due to the length of implant drills ("10#20 mm), using a finger rest while drilling, results in an inclination of the drill towards the hand that is steadied. Hence, using finger rests is an ergonomic principle that should not be used for implant placement.

3. Improper implant location

It should be ensured that the implant body is 1.5mm from the adjacent teeth nad 3-4mm

between adjacent implants. Preoperative measurements and planning are essential to achieve an ideal implant placement that facilitates future implant prosthesis. Hypothetically, a surgical complication could also occur, but not be realized by the surgeon at the actual time of surgery, especially when placing multiple implants, the one placed the away from an adjacent implant can have adequate stability and function but may later result in lateral bone loss.

4. Communication failures

An informed consent form is an excellent way of communicating potential surgical risks and complications to a patient. Common problems to address include but are not limited to postoperative infection, bleeding, swelling, facial discoloration, transient pain, paresthesia, neuralgia, fracture, joint pain, muscle spasm, tooth looseness and sensitivity, recession, speech change, trismus, and swallowing of foreign objects. Should a complication occur during the post operative healing time, it is recommended to give emergency contact information as well.

Anatomical complications

1. Nerve injury

When placing implants in the mandible, proper radiographs and pretreatment planning must be done to ensure complete aversion of the inferior alveolar, mental, incisive or lingual nerves. If the mandibular canal cannot be seen on a panoramic radiograph, a computer tomography (CT) scan should be taken to verify the location. Possible causes of nerve injury include poor flap design, traumatic flap reflection, accidental intraneural injection, traction on the mental nerve in an elevated flap, penetration of the osteotomy preparation and compression of the implant body into the canal. Radiographs should be taken if the surgeon has any doubt about where the drill is or if the drill or implant is in close proximity to or invading, neural anatomical structures. If the situation is the latter, the implant needs to be removed, or a shorter body implant should be placed instead. Within days or months, minor trauma injuries usually heal but permanent damage from neuritis can occur. Treatment options include neuronal anti-inflammatory drugs such as clonazepam, carbamazepine or vitamin B-complex.

2. Bleeding

Risk sites as described above in the posterior mandible include the sublingual fossa and lingual cortex. Life-threatening airway obstruction is a serious threat and early treatment is essential. Treatment involves having the patient stick out their tongue to compress the blood vessels against the body of the mandible. Placing pressure with gauze in the sublingual area does not work as one would intuitively think. Extraoral pressure to the submental or submandibular arteries for 20 minutes against the body of the mandible helps. The posterior superior alveolar and infraorbital arteries are located approximately 19 mm above the maxillary alveolar ridge, and the anastomoses of these arteries can pose a risk during sinus lift procedures by lateral window preparation. Bone wax, pressure, crushing, and electrocautery can alleviate hemorrhage. In summary, hemorrhage treatments at implant osteotomy sites include compression, finger pressure, vasoconstriction, cautery, bone graft, bone cement, and ligation of arteries.

3. Cortical Plate Perforation

The buccal cortical plate varies in thickness throughout the mouth and traumatic dental extractions can cause markedly thin plates or concavities, as well as overall ridge width deficiency. When preparing osteotomy sites or placing implant fixtures in areas with minimal labial plate thickness, or if the implant is placed too buccally, a fenestration or dehiscence implant defect is a common finding. Immediate correction with particulate bone grafting with or without a membrane during the time of implant placement, can be done as long as primary stability has been achieved. “Flapless” implant surgeries should be avoided in areas of potential perforation of the buccal or lingual bone.

4. Sinus Membrane Complications

Sinus complications often occurred when the membrane is perforated at time of surgery. Bone density after grafting should be assessed, regardless whether or not a perforation occurs, because poor bone quality often lead to a higher implant failure rate. Lastly, losing an implant into the maxillary sinus is a relatively uncommon surgical complication. However, in cases with less than 5 mm of bone, mastication can cause the implants to move during the graft maturation time frame. Transantral endoscopic surgery is a reliable, minimally invasive method for retrieving displaced objects from the maxillary antrum with minimal complications, but it does require having an endoscope or a referral to an ENT or oral surgeon.

Procedure Related.

1. Mechanical errors

Dense cortical bone (eg, type I bone quality), when compared with type III or IV soft cancellous bone, can be overheated when preparing osteotomies because more pressure is needed to advance the drill apically in comparison to soft bone. To reduce frictional heat, high speed handpieces, an up-down motion technique of the bone preparation, and copious irrigation can be used. According to Quirynen et al, 55 overpreparation or overheating osteotomies can result in inactive and active retrograde peri-implantitis lesions that can be detected on radiographs as periapical radiolucencies up to a month after insertion.

2. Lack of primary stability

It should be dealt with at the time of implant surgery. An unstable implant should be removed or an attempt to place a larger diameter should be completed. To leave an unstable implant without action can often lead to fibrous encapsulation that causes implant failure.

3. Mandibular fracture

The mandible is the most frequently fractured facial bone. Attempts to place implants in patients with severely atrophic mandibles increases the risk of fracture, especially when monocortical grafts and ridge-splitting surgeries are completed. A fracture of the mandible should be restored to maintain form and function. Management should include stabilization with an attempt to also simultaneously eliminate atrophy if indicated. The most relevant option of our field includes combined bone augmentation, fixation and simultaneous implant placement. Increasing mandibular height after augmentation may be unpredictable but using implants concurrently may reduce bone resorption.

4. Aspiration and ingestion

Most instruments have a special tip to help ensure screws and abutments transfer directly from the surgical tray into the patient's mouth, but nevertheless, accidents happen. For these reasons, preventative measures such as gauze throat screens and floss ligatures on implant pieces are encouraged. If a patient swallows or aspirates an implant component, they should be referred to the hospital because acute obstruction can be life threatening and prolonging the removal of foreign objects may make a bronchoscopy technically more difficult. If the foreign object is aspirated it should be removed within 24 hours.

Conclusion

Surgical implant complications are not uncommon and should be addressed immediately. Causality may be iatrogenic, due to poor treatment techniques, or lack of communication between dental disciplines. Time should be spent in the implant "planning" stages, such as tracing preoperative radiographs, measuring models, taking CT scans and making proper surgical guides. Basic anatomy must not be forgotten and should be reviewed by the surgeon in every case. As more surgically inexperienced dental professionals start placing implants an increase in surgical complications will likely occur. In summary, a competent surgeon should be able to treatment plan a predictable surgery, and recognize how to remedy a problematic dental-implant situation.

BISPHOSPHONATE RELATED OSTEONECROSIS OF MAXILLA FOLLOWING IMPLANT FAILURE – A CASE REPORT

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INTRODUCTION

Marx in the year 2003 , defined osteonecrosis of the jaw (ONJ) as exposed , necrotic bone in the maxillofacial region for atleast eight weeks in patients receiving anti – resorptive medication for primary or metastatic bone cancer , osteoporosis or Paget's disease , without history of radiation therapy to the jaws [1]. The AAOMS , modified the above definition to include exposed bone , or bone that can be probed through an intra oral or extra oral fistula in patients who are on antiresorptive or anti angiogenic medications [2].

Bisphosphonates treatment has been found to be a major factor contributing to the development of osteonecrosis of the jaws . Bisphosphonate related osteonecrosis of the jaws is the most common side effect of bisphosphonate treatment , without previous history of radiation therapy to the jaws [1]. According to Kurtzman , it is considered to be a long term and irreversible , even on discontinuation of drugs[3].

As mentioned by Ruggiero et al., oseteonecrosis of the jaw occurs more in th mandible and less in the maxilla [4]. The reason for this being good vascularisation and high bone turnover rate in the maxilla . Hence, maxillary ONJ lesions are extremely rare .

The possibility to perform dental extractions and dental implant placement in bisphophonate or anti resorptive agent users , continues to be a controversy . The American Association of oral and maxillfacial surgeons contraindicate implant surgery in patients who are under anti – resorptive therapy[2] .

This article reports the rare occurrence of osteonecrosis of the maxilla associated wht implant failure in a patient who underwent bisphosphonate therapy.

CASE REPORT

A 48 year old male patient , reported to Sree Balaji Dental College and hospital , Chennai . The patient complained of swelling , pain and burning sensation in the maxilla for 1 month .

The patient had a history of osteoporosis for the past 2 years and was under bisphosphonate therapy . All on four implants were placed in the maxilla 1 year back , the prosthetic rehabilatation was also completed , at a private dental clinic .

On examination , extra orally – no abnormality was detected . Intra orally , a firm swelling was observed in the maxillary region , upto the hard palate . Necrosed maxillary bone was exposed . The implants and the prothesis were mobile . It was also observed that the implants were exposed .

Local anaesthesia was administered and the prosthesis was removed , as the first step . The following (FIG. 1)is the intra oral picture after the removal of the prosthesis .

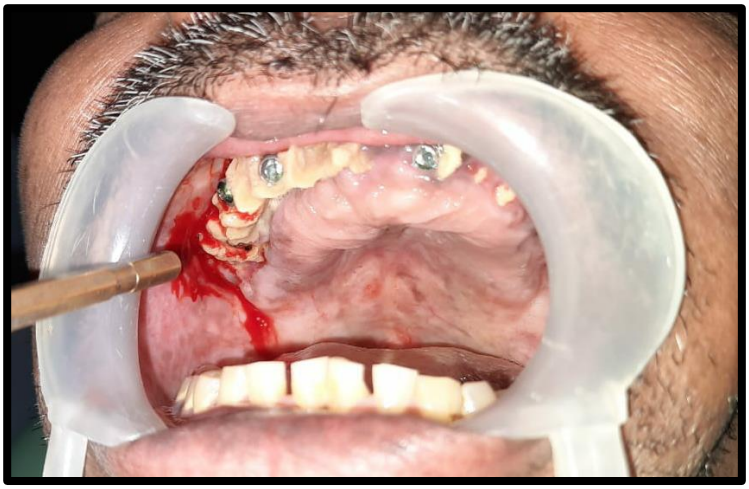

FIG 1: NECROSED MAXILLARY BONE AND EXPOSED DENTAL IMPLANTS

A crestal incicision was placed extending from 17 to 27 region . Mucoperiosteal flap was elevated (FIG 2 &3). The dental implants were removed . Sequestrum was removed (FIG. 3) . Debridement and curettage was done . Thorough metrogyl irrigation was carried out . Primary closure was done with silk suture (FIG. 4) .

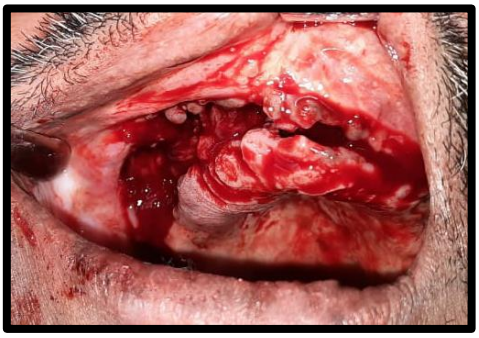

FIGURE 1

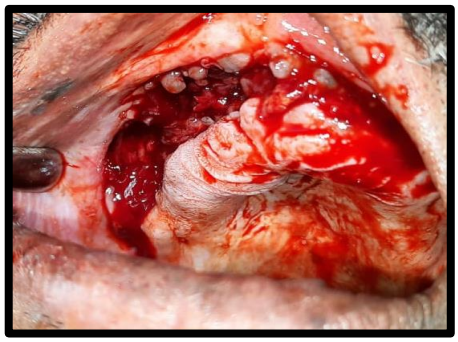

FIGURE 2

FIGURE 3

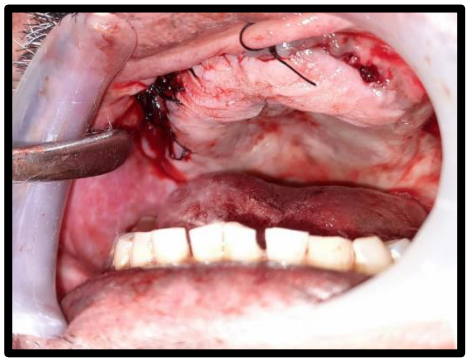

FIGURE 4

The patient was then administered with antibiotics and anti inflammatory medications . Review was done on the fifth and seventh post operative day .

DISCUSSION

The patient had a history of taking Alendronate for 2 years , when he had presented for implant placement in the maxilla at a private dental clinic . The presence of clinically visible necrosed bone in the maxilla ,mobile dental implants , chronic pain and burning sensation in the maxilla , as observed during examination of the patient , was suggestive of stage II Bisphosphonate related osteonecrosis of the jaw (BONJ) , as per the staging system given by Ruggiero et al[5]. The histopathological report confirmed the same.

As mentioned by Petropoulous et al. , several studies quote that there is a decreased incidence of osteonecrosis when the patient has been on oral bisphosphonate therapy for less than five years [6]. In a study conducted by Fuggazo et al. [7], it was conlcuded that patients who were on bisphosphonate therpay for an average duration of 3.3 years , did not experience any incidence of osteonecrosis following implant placement in intact alveolar ridges or following immediate implant placement after tooth extractions . Another study by Madrid and Sanz [8], stated that no BONJ was observed in patients who took

bisphosphonates for less than 5 years . Hence , the case reported in this article is a rare incident , as the patient ws on bisphosphonates for only 2 years .

A detailed treatment protocol was laid down by Rugeeiro et al. for evert stage of bisphosphonate related osteonecrosis of the jaw . It was stated that for stage III disease , as seen in this case , surgical debridement of necrotic bone ,followed by antimicrobial therapy , analgesics and anitmicrobial rinses is the best treatment protocol [7]. As per the prescribed treament protocol , surgical debridement was carried out for this patient , followed by medication , after the culture results were obtained .

CONCLUSION

In conclusion , although bisphosphonate related osteonecrosis of the jaw is not encountered very often , it is essential that the diagnosis and management of this condition is known by every oral and maxillofacial surgeon . A complete history must be taken by the oral surgeon , before placement of dental implants , so as to avoid the failure of implants .

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MIDFACE DEFORMITIES AND THEIR SURGICAL MANAGEMENT

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ABSTRACT

Background: *Midface deformities may be treated either by osteotomies that advance the maxilla or by osteotomies that retract the mandible. A transfacial technique is presented by which the cheekbones, the infraorbital rim, and the superior dental arch are advanced. The indications for this intervention are more widespread than those for Le Fort I osteotomies or mandibular osteotomies. Postoperative complications and risks of recidivism are reduced for this method because of the use of small screw-on plates for frontomalar osteosynthesis. Fifty-two cases of osteotomy by maxime champy et al is reviewed[1]. This review deals with his special consideration of problems caused by bone grafting and postoperative occlusion and with a discussion of the factors responsible for relapse.*

Keywords: *Osteotomies, le fort1, maxillary deformities.*

Introduction

This discussion of the surgical treatment of midface malformations is not intended to review extensively all surgical procedures used at present or to cover the entire spectrum of malformations that are known to occur in the midface. In any extensive discussion, one would have to include lesions as diverse as the Treacher Collins syndrome, craniofacial stenoses, hypertelorisms, tumoral malformations, losses of cutaneous substance, as well as facial clefts. In fact, this presentation will be limited to the study of skeletal dysmorphoses of the midface[2]. In an arbitrary way, with use of a topographic classification, the dysmorphoses could be categorized as follows: retrodisplaced maxilla and micromaxilla; extreme retrusion of the maxilla and combined anomalies, congenital malformations, deformities resulting from neglect or maltreatment of traumatic injuries, and deformities following surgical treatment of cleft lip or palate.

Considerable progress has been made in the treatment of these lesions during the last 20 years, and at present many bones can be cut, including the cranium, upper maxilla, and mandible, or a combination of these bones can be cut in a single operation[3]. As a result of the formation of well-trained maxillofacial surgery teams and improvements in surgical materials, it is possible today to displace almost any facial structure in any desired direction. Consequently, for a highly specialized surgeon, the difficulty in correcting malformations does not lie in the execution of the operation, but rather in choosing the best approach for the desired correction.

Discussion

The present consensus is that it is possible to move almost any facial structure in any desired direction, and the tendency is to correct the deformed bone structure by moving bones rather than by implanting bone transplants or by using prostheses[4]. The spaces between the bones may be filled with bone grafts, and postoperative fixation may be accomplished by wire osteosynthesis, by cranial fixation using frames, and almost always by intermaxillary wiring. We attempt to anticipate possible postoperative occlusion by studying models that give some idea of the degree of the necessary displacement and of the quality of the future intercuspation. Before the operation we also study the profile; the relative proportion of the cheeks, the nasal pyramid, and the chin; and the total facial appearance[6]. The preparation of the dental arches consists of simple interventions,

for example, extraction of poorly aligned teeth if a premolar is in a palatine direction. We do not expand the palate preoperatively, we only make sure that the teeth are well aligned. As a general rule, before the intervention, one should consider all possible postoperative developments in order to be prepared with prostheses, such as cap splints, that might be needed. This is particularly difficult if one wants to consider unexpected osseous adaptations under the influence of the postoperative functional conditions.

Incisions

Our approach is as discreet as possible, and if, simultaneously, a cranioplasty is necessary, we use a Cairns incision[5].lZA Our approach to the orbital margin is via an incision in the lower lid Our approach to the external orbital margin is via the same incision as for an aesthetic blepharoplasty and consists of two parts: first, the incision is made horizontally along the canthus without exceeding a distance of 1.5 cm from the outer margin, and second, the incision is continued 2-4 mm below the lower lid (fig. 5). A median vestibular incision allows access to the nasal fossa, and a posterior vestibular incision allows access to the pterygomaxillary sutures and is performed bilaterally[7]. Osteotomy. The osteotomy is performed by cutting the frontal process of the zygomatic bone at different levels according to the correction desired. Then one cuts the orbital margin, just outside the lacrimal sac, the frontal process of the maxilla between the orbital margin and pyriform aperture, the posterior wall of the antrum, the zygomatic arch as far as possible posteriorly, and the lower border of the nasal septum (fig. 6). Thereafter, we carefully break the orbital floor behind the orbital margin using a very fine osteotome (Stryker, Kalamazoo, MI), and we accomplish the interpterygomaxillary separation with another special Obwegeser osteotome (Medicon, Tuttlingen, West Germany). We loosen the last osseous attachment with a Rowe and Killey forceps and thus are able to move the entire facial skeleton with the exception of the nose. This maneuver can be adjusted according to the results desired and it can be a horizontal sliding movement alternating with a rocking movement either upwards or downwards (fig. 7). Aesthetically, it is more advantageous to rock the midface somewhat upwards. By this method, one achieves an exaggerated movement forward t.ogether with an overcorrection at the level of the dental arch. Bone Qraft. Spaces caused by moving the midface forward can be filled with bone transplants to avoid enophthalmos and the retraction which is necessarily exerted by scar formation on the advanced bone in the zygomatic bone cut and in the zone of the pterygomaxillary osteotomy, where a voluminous graft has to play the role of support. However, we try to avoid bone transplants as often as possible. In the orbits, for example, we have attempted to fill the diastema with lyophilized dura. The interpterygomaxillary space often does not need any special treatment[8]. Thus, we circumvent the painful bone sampling of the iliac crest and, furthermore, we are not convinced that one can avoid relapse by interposition of bone supports (fig. 8). Fixation. Only in exceptional cases do we use methods of orthopedic fixation such as extraoral pin fixation, intermaxillary wiring, or cranial frame. Sometimes we are forced to use intermaxillary wiring because of unusual circumstances and we even use the Delaire apparatus figure 9. frontozygomatic osteosynthesis by small screw-on plates: (A) frontal view, (B) profile. (Nichrominox, Lyon, France) when we are not sure how well the displaced fragment holds. Our favorite method of fixation consists of an osteosynthesis using two small plates (Gebruder Martin, Tuttlingen) on the cheekbone.'[10]' Its simplicity and its many advantages over all other methods of fixation bring us to this conclusion. We use small plates, 0.9 mm thick, with screws having a diameter of 2 mm. This material offers the advantages of allowing perfect insertion of the screws and easy adaptation of the plates to the shape of the bone because they are both pliable and strong[9]. These plates permit a perfect fixation in all three dimensions. They can be left for as long as 1 year, and they are

a good preventive measure against displacement. Furthermore, the biologic compatibility of the material is good (figs. 9-12).

Variation of the Technique. 'transfacial osteotomy allows the correction of many facial malformations. Nevertheless, in some cases one must make minor technical changes. When the desired surgical movements of different parts of the midface are not equal (for example, when the hypoplasia of the orbital margin is clearly delineated), the correction of this part of the deformities by a forward translation of all the midface creates an extreme projection of the upper dental arch, i.e., a promaxilla. We have observed many times that it is not necessary to be too preoccupied with the postoperative occlusion. A postoperative modeling which is either spontaneous or carried out by simple orthodontic means generally establishes satisfactory conditions for occlusion in the following weeks. Otherwise, it might be necessary to perform a Le Fort type I osteotomy10-12 or a sagittal osteotomy of the ramus according to the Obwegeser technique.[9] When there is a very narrow dental arch, the forward displacement of the midfiice does not allow a good intercuspation. The possibility of an intermaxillary bone cut can then be considered. In this case the osteotomy leads to two independent bone parts which are from the orbital margin, the maxilla, and the cheekbone on each side. In this case, the fixation is achieved by osteosynthesis using the plates on the cheekbone. To avoid the formation of spaces between the incisors, one uses a monomaxillary ligature. Postoperatively, one maintains the frontal extension of the palate by an expansion cap splint. The use of midface osteotomy is particularly interesting in patients with cleft lip and palate sequelae. In these cases, the bone cut is the same, but the lower central bone cut which leads from the orbital margin to the pyriform aperture will extend to the cleft. In this way, one obtains bilaterally two bone parts which are composed of the maxillae, the cheekbones, and the orbital margins (fig. 13). The special malformations in cleft lips and palates can then be corrected by movingthe individual fragments forward or outwards. Thus one obtains an opening of the cleft with separation of the mucous membranes, which necessitates a correction later. The fixation is achieved by osteosynthesis using two small plates on the cheekbones. To ensure the solidity of the fixation, it is best to use two plates bilaterally. A palatal cap splint is inserted as early as possible postoperatively. In fact, the postoperative risk of relapse is much greater in such a patient than in patients with other malformations. A long-term fixation is recommended. The normal scar retraction takes place in the usual front-to-back direction, but one also has to anticipate a concentric scar retraction in the former cleft region and an additional collapse of the fragments. Therefore, relapse can best be avoided with an osteosynthesis using plates.

Conclusion

In conclusion, the advantages of transfacial osteotomy in conjunction with fixation by plates on the cheekbone are such that this type of osteotomy should be chosen when there is any question about the best possible correction, except when there is a clear-cut indication or another method. The surgical treatment of skeletal deformity of the midface should be adapted to the nature of the anomalies, and the hest aesthetic result possible should be attempted. If the deformity is situated high on the midface, it is most logical to proceed with an intermediary transfacial osteotomy rather than with a Le Fort I osteotomy or a sagittal osteotomy of the ramus[10]. Provided that a careful surgical technique is respected and that inconveniences and factors of relapses in the particular method are well known, transfacial osteotomies give aesthetic results that are at least as satisfying for the surgeon as for the patients in their environment.

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MANAGEMENT OF PANFACIAL FRACTURE

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ABSTRACT

Traumatic panfacial fracture repair is one of the most complex and challenging reconstructive procedures to perform. Several principles permeate throughout literature regarding the repair of panfacial injuries in a stepwise fashion. The primary goal of management in most of these approaches is to restore the occlusal relationship at the beginning of sequential repair so that other structures can fall into alignment. Through proper positioning of the occlusion and the mandibular-maxillary unit with the skull base, the spatial relationships and stability of midface buttresses and pillars can then be re-established. Here, the authors outline the sequencing of panfacial fracture repair for the restoration of anatomical relationships and the optimization of functional and structural outcomes.

Keywords: *pan facial fracture, facial trauma, sequencing repair, occlusion restoration, spatial relationships of midrace and mandible.*

EPIDEMIOLOGY OF PANFACIAL BONE FRACTURE

Pan facial fractures are usually caused by high-energy injuries (e.g., motor vehicle or gunshot injuries).Panfacial fractures account for 4%–10% of all facial fractures. In Korea, the incidence was reported to account for 6.59% of all facial bone fractures. The most common site of mandibular fracture is the symphysis (33.5%), followed by the condyle (31.1%) and body (17.1%) .

HISTORY OF THE MANAGEMENT OF PANFACIAL FRACTURES

In the 1980s and early 1990s, craniofacial surgeons established the principles of wide exposure and direct visualization of fracture alignment for accurate craniofacial bone reduction. When these principles were applied, the sequence of alignment restoration was influenced[(1)]. The concept of facial buttresses was emphasized as the key to restoration of skeletal framework, and panfacial reconstructions began with the reduction of the frontal bone and proceeded with the midfacial bone alignment. Using the reconstructed maxillary framework as a template, the lower face was reconstructed last (top-to-bottom sequence) [(2)].

Following the advent of rigid internal fixation, surgeons started reduction of facial fractures with the mandibular condyle [(3)]. The condyles determine the facial posterior height, and restoration of this height allows the mandible, which is the strongest bone of the facial skeleton, to be used as a template for panfacial bone reduction. Because of this, the bottom-to-top sequence is widely used in craniofacial surgery today.

Principles of Approach to Panfacial Fracture

One of the primary concerns with regards to the repair of panfacial fractures is airway management. There are four established mechanisms for the airway: oral intubation, nasal intubation, submental intubation, and a tracheostomy. The latter three of these intubations allows for mandibular–maxillary fixation with full dentition. Oral intubation is possible when there is an absence of occlusion or absent teeth that allows the oral tube to be placed posteriorly in the mouth. Nasal intubation is often possible; however, with complex nasal and naso-orbito-ethmoid fractures, in addition to mandibular and palatal fractures, there is concern for postoperative management of the airway. There can be significant edema or packing within the nose in combination with mandibular–maxillary fixation that also leads to concern about maintaining airway patency. Submental intubation has been shown to be a safe approach with the tube out of the way, but the postoperative issues in regards to nasal packing and mandibular–maxillary fixation still exist. A tracheostomy allows the

tube to be away from the structures being repaired and also has postoperative control of the airway. Of course, there are concerns about postoperative tracheostomy-related complications; however, the risk of tracheostomy is relatively low when compared with the risk of airway management postoperatively.The inside-out thought process is reconstructing the maxillary–mandibular unit as the first major step and then focusing on the midface structures. This would allow the occlusal relationship to be restored and then "built out" from that process. The outside-in, or top-down, mentality would be reconstructing the outer facial frame and the bony pillars, such as the zygomatic arch and the frontal areas, and then addressing the interfacial frame.[4] These two thought processes have permeated the literature and teaching for decades. In actuality, the best course of action is to follow a combined process. The primary goal would be to restore the occlusal relationship and then the spatial relationship between the occlusal structures and the skull base.

Historically, the lines of weakness were first described by LeFort in 1901[5]. This was followed by descriptions of the buttresses in 1916 by Cryer, and by illustrations of the vertical pillars and horizontal buttresses. Epsteen and Dingman described the palatine and maxillary fractures as important for structural stability of the midface; finalized the importance of this relationship to the cranial base. This led to our understanding of the anatomy and physiology of the bony structural components of the midface in relation to the skull base and the mandible

Anatomical Relationships

The components of panfacial fractures are outlined in. The definition of panfacial fracture incorporates the lower-third, middle-third, and upper-third facial components usually in a combination of fractures. There are multiple buttresses within the midface that need to be approached to restore the midface height, midface projection, and midface width, in addition to restoring the occlusal relationship[6]. The medial buttresses are along the nasal frontal bone to the anterior maxillary alveolus. The lateral zygomatic maxillary buttresses extend along the zygoma and malar bone to the lateral maxillary alveolus.The pterygomaxillary buttress has a medial component that extends from the posterior alveolus and palate to the cranial base, and a lateral component that extends from the lateral pterygoid plate to the greater wing and lateral wall of the sphenoid. There is a central sphenovomerine buttress, which is along the central posterior palate to the floor of the sphenoid sinus.[7] With disruption of all of the buttresses and the occlusal relationship, there is a tendency for facial widening, flattening, and rotation of the maxilla. Consequently, there will be an appearance of an obtuse nasolabial angle with impaction of the midface, which will seem like an open-bite deformity. The loss of bony relationships along the sphenoid bone or the lateral walls of the orbit with fractures along the zygomatic arch will result in apparent facial widening.[8] With condylar fractures or ramus fractures, there is also a collapse of the mandibular relationship at the skull base. Therefore, the primary treatment goal is to approach these fractures in a stepwise fashion with proper sequencing of repairs by restoring the occlusal relationship and extending out to the repair of all of the buttresses.[9]

Sequencing

The key to sequencing in panfacial fracture management is to understand both the principles of buttress reconstruction and the need for restoring the spatial relationship of the occlusion in the skull base.[10] With panfacial fractures, there is a compromise of the mandibular–maxillary unit and the relationship between these two structures and the skull base. The midface is also violated with the loss of key components necessary for anatomical alignment. For example, the repair of mandibular–maxillary fractures often will rely on the stable structure of the upper face and vice-versa. With panfacial fractures, there

is a loss of the customary structures for anatomical alignment.

The sequencing that will be described assumes that all of the structures have been violated with the loss of anatomical stability. If there are any components that are minimally injured or not fractured, those can be assumed as "repaired" in the sequencing structure.

The first fracture to be repaired would be the palate. There is often a split within the midline or parasagittal component of the palate that needs to be realigned. Perfect structural alignment of the palate is somewhat difficult, so the goal would be to close the fracture and have this structure fixed in position. The options would be either to open up the mucosa and place a plate over the fracture site or to close the fracture and place transmucosal screws, utilizing a locking system.[11] Once the fracture has been closed, the fragments of the maxilla will be able to toggle, but not distract.

Two options for sequencing

There are two options for sequencing:

- Re-establish the maxillo-mandibular unit as the first major step of the sequencing (bottom-up).
- Starting with the reduction and fixation at the level of the calvarium and working in a caudal direction (top-down).

1) Re-establish the maxillo-mandibular unit as the first major step of the sequencing (bottom-up).

Once the maxillomandibular unit is established, most surgeons start from the calvarium and proceed in a caudal direction with reduction and fixation.

2) Starting with the reduction and fixation at the level of the calvarium and proceed in a caudal direction with reduction and fixation (top-down).

It should be noted that with this second option of sequencing, reestablishment of the proper maxillomandibular unit is still very important, but may be achieved later in the case.[12]

VARIOUS SEQUENCES OF PANFACIAL BONE REDUCTION

As no clear classification of panfacial bone fractures is available, various sequences of reduction (bottom-to-top, top-to-bottom, inside-out, and outside-in) are used in combination to restore facial contour. Numerous studies have compared combinations of these reduction sequences. However, the efficacy of inside-out or outside-in sequences have not been assessed independently of bottom-to-top or top-to-bottom sequences. The "bottom-to-top and outside-in" approach is the most widely used method in the panfacial bone reduction. Gruss and Phillips [12] advised starting panfacial reconstructions with reduction of the zygomatic arch and malar projection to establish the outer facial frame and to provide upper facial width and projection before NEO, maxillary, and mandibular reconstruction. Merville suggest the frontozygomatic suture line should be reduced first in panfacial bone fractures because this important structure determines facial width and projection. As NEO fracture fragments are fragile, it is difficult to find a stable fixation point in naso-ethmoid-orbital area. Therefore, experts often recommend the outside-in sequence for reconstruction of panfacial bone fractures.

Sequencing from the calvarium down

- The first priority is to address any significant calvarial, frontal sinus, and orbital roof fractures. Using the calvarium as the foundation for the remainder of the midface reconstruction, the surgeon progresses from this level down to the Le Fort I level. The fractures at the Le Fort I level are the second to last fractures to be plated.
- The zygoma is positioned into its proper three-dimensional position taking care to properly line up the

lateral wall of the orbit with the greater wing of the sphenoid.

- The proper alignment of the zygomatic arch and the infraorbital rim must be taken into consideration during the reduction of the various fractures[13].
- The completion of the reconstruction of the periorbital areas is performed by addressing the NOE and nasal fractures.
- Any condylar fractures may be treated open or closed depending on the wish of the surgeon.

Closure and Recovery

At this point, the face should be completely reconstructed. The mandibular–maxillary fixation will need to be released to assess the occlusion and make sure there is central occlusion with maximum intercuspation. The forced duction test will also need to be performed to verify freely moving orbital cone contents. This will all need to be done in anticipation of closure. The closure of the midface is a very important aspect of panfacial trauma management, so even part of the opening has to do with the anticipation of the closure. The intraoral incisions are closed in the usual fashion with reapproximating the muscle and then the mucosa. This could either be with running sutures or with multiple interrupted sutures, depending on the nature of the tissue that is left. The lateral midface will need to be closed by reapproximating the deep temporal fascia. The malar eminences will be completely degloved, thereby causing a risk of ptosis and the need for resuspension. Although there are techniques to suture the periosteum, there is really no periosteum available with extensive deglovings and panfacial fractures[14]. The deep tissue is grasped on either side of the infraorbital nerve with a suture placed through this tissue and then resuspended to the inferior rim; this is equivalent to the malar midface lift. With the resuspension of the malar tissue, the inferior rim hardware is covered, which may assist with preventing cicatricle retraction of the lower eyelids postoperatively. The scalp is enclosed with multiple interrupted buried sutures. Staples can be used; however, staples are often difficult to place because of their thickness. Thinner scalps allow staples to be placed with eversion. Staples placed in a thick scalp tend to have overlapping edges without eversion or coaptation of the skin edges. A safe closure would be suturing of the skin with the running locking suture or multiple simple sutures.

Conclusion

In summary, the sequencing of panfacial fracture repair should be in a stepwise fashion. The restoration of the occlusion is considered the primary goal in the beginning of the sequencing process. The LeFort I level of the maxilla will need to be restored in its width with mandibular–maxillary fixation. This mandibular–maxillary unit is then restored to its vertical height and position in relation to the skull base. The remainder of the midface is subsequently reconstructed by full exposure and reduction with the key elements of repair involving restoration of the lateral wall of the orbit at the zygomaticosphenoid junction and the projection of the zygomatic process of the temporal bone. The naso-orbito-ethmoid fractures are reduced at this point as well. After all of these fractures are addressed, then the LeFort I level can be plated because this is the area that is the most forgiving. Ultimately, panfacial fractures are managed through systematic sequencing steps focusing on the occlusion as the foundation for proper alignment.

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A REVIEW ON BASAL IMPLANTS

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ABSTRACT

The primary indication of implant prosthesis, essentially the endosseous implants, is to replace the missing tooth or teeth structure with a prosthesis that mimics the morphology of an original tooth and facilitates function and aesthetics. However, the main disadvantage of this prosthesis is that it shows very less success rate in the areas with less residual bone present. One of the designs to combat this problem is the use of basal implant in areas of very less bone height. This review aims to elaborate the features of the basal implant design

Keywords: *Basal Implant, BOI Implant, BCS Implant, Disk Implant, Basal Implantology*

INTRODUCTION

In present day dental practice restoring edentulous maxilla and mandible with dental implants based on crestal implantology become normal predictable treatment, where the implant placed in crestal alveoli. For successful dental implant placement minimum 10-13mm vertical bone height should be available. However, if the adequate bone height is not available in edentulous areas ridge argumentation procedures needs to be done to restore lost alveolar bone dimension for placing a successful dental implant. Such procedures involve autologous or allogenous bone graft placements, nerve repositioning, sinus lift procedures or even nasal lift. These above-mentioned procedures have their own indications and contraindications. In severely atrophic ridge patients to avoid these procedures implant design needs to be altered. Mini implants and basal implants can be used as an alternative.

Thus, basal implantology also known as cortical or bicortical implant system which the implant placed in the cortical portion of jaw gains a excellent retention from the basal cortical bone. In past several decades the basal implants undergone changes and modifications and designed specifically to for the purpose of gaining anchorage from the basal cortical bone. Now at present available basal implants are simple, less surgical protocol and can be loaded with immediately

REASON FOR USING BASAL IMPLANTS

As indicated by the idea of basal implantology the jaw bone contains two sections the tooth bearing alveolus or crestal part and the basal bone. The crestal bone is less thick in nature and presented to contaminations from tooth borne pathologies, wounds or iatrogenic factors and is consequently dependent upon higher rate of resorption while the basal bone is heavily corticated and less chances subject to contaminations and resorption.

It is this, i.e.; the basal bone that can offer a good supportto the implants on account of its thickly corticated nature, at a similar time the load bearing limit of the basal bone is ordinarily higher than that offered by the spongy crestal bone. Basal implants are additionally called as "Orthopedic Implants"[3, 4, 5, 6].

Basal Implant Types Based on Morphology

There are four fundamental kinds of basal inserts available

I Screw Form.

II Disk Form.

III Plate Form.

IV Other Forms.

Both of the kinds can be additionally arranged into.

1. Screw Form

a. Compression Screw Design (KOS Implant)

b. Bi-Cortical Screw Design (BCS Implant)

c. Compression Screw + Bi-Cortical Screw Design (KOS Plus Implant)

2. Disc Form

Basal Osseointegrated Implant (BOI)/Trans-Osseous embed (TOI)/Lateral Implant1)

According to Abutment connection.

i. Single Piece Implant.

ii. External Threaded Connection.

iii. Internal threaded Connection

a) External Hexagon.

b) External Octagon.

2) According to basal plate design.

i. Basal circles with angled edges.

ii. Basal circles with level edges called as S-Type Implant.

3) According to number of disks.

i. Single Disk.

ii. Twofold Disk.

iii. Triple Disk.

III. Plate Form

a. BOI-BAC Implant.

b. BOI-BAC2 Implant.

IV. Different Forms

a. TPG Implant (Tuberopterygoid).

b. ZSI Implant (Zygoma Screw).

Implant Morphology

The BOI and BCS implant being delivered today has a smooth and polished surface as it was discovered that polished surfaces are less inclined to irritation (mucositis, periimplantitis) than unpleasant rough surfaces[4, 5, 6]. The KOS and KOS plus Implants are surface treated (sand and coarseness with acid etching), in any case, the implant neck is keptprofoundly polished in KOS implant[4]. In the KOS Plus implant, its neck and the basal cortical screw part are kept vigorously polished4

A. BOI Implant Morphology

The BOI implant is produced either from unadulterated pure Titanium or then again from Titanium Molybdenum combination to upgrade strength of the implant[1, 4]. These can be either single piece or two pieces, following are the pieces of the BOI implant.

a) Abutment portion

In single piece BOI abutment the projection divide is tapered and stays uncovered in the oral cavity, though in two-piece BOI implant the abutment bit can be aexternallythreaded screw or an internallythreadedscrew with either an outer hexagonal or octagonal restorative platform[4].

b. Neck

It is the bit lying straightforwardly underneath the abutment portion. This part could be constricted in diameter; constriction gives better post-operative gingival healing and furthermore reduce inflexibility and permits for bending by 15°-25°1, 4.

c. Vertical Shaft

This is that portion that associates all the segments of the implant. The shaft is kept smooth and polished to debilitate plaque collection and irritation; moreover it tends to be either flexible or inflexible relying upon the kind of titanium utilized. The vertical shaft is simply a load bearing part and is typically 10 – 13.5 mm long[4].

d. Crestal Disk

It is the principal plate in the implant. It is called crestal disk as it lies in the crestal bone after positioning the implant. This disk serves a double purpose i.e.; following implant placement this disk gives and keeps up the

primary strength and stability and after osseointegration this disk changes over into a load bearing and distributing component[4,6].

e. Basal Disk

It is the second disk at the base of the implant and is the last part in the implant body. This part is additionally kept polished and is a load bearing and distributing segment. The piece of the shaft associated with the basal disk is versatile and can likewise be bent by 15°-25°[4,6]. Distance between the crestal and basal disk is typically 5 mm.

B. BCS Implant Morphology

These are single piece implantssimilar to the BOI implant with alterations in the abutment and the implant portion. BCS implantabutment can be Conical Straight, funnel shaped Angled and Multi-Unit abutments. In contrast to the BOI implant which contains disc in the implantportion, the BCS implant has wide diameter cutting screws which makes a difference in connecting with the buccal and palatal/lingual cortical plates also, at first give primary stability and load bearing ability to the implant and later on go about as a load bearing and distribution component[4,6]. These implantsare heavily polished and are flapless implants with a little mucosal infiltration diameter[3,6].

C. KOS and KOS Plus Implant Morphology

These implants are single piece implant and are produced from Titanium Molybdenum or Titanium aluminum Vanadium alloy. These implants are planned like compression screws, i.e.; these implants when screwed into the bone will pack the cancellous bone encompassing the implant to formmore compact and denser bone[1, 4, 8].

I. Abutment Portion[1, 4]

This is the restorative platform of these implants and stays uncovered in the oral cavity. These implant offer a wide assortment of abutment choices which are-

a. Conical shaped Straight abutment for cemented crowns, this abutment may likewise have a vertical microgroove that fills in as an anti rotational component.

b. Conelike Angled abutment.

c. locator abutment.

d. Ball abutment.

e. Multi-Unit abutment.

*(these abutments are essential for single piece implant)

ii. Neck[1, 4]

This piece of the implant is vigorously polished and is contracted to help in better gingival adaptation and to debilitate plaque aggregation. The neck of the implant is bendable by 15°-25°.

iii. implant Portion[1, 4]

This part of the implant has the thread which have wide construction and wide turns this empowers them to apply compressive forces on the cancellous bone and convert it to a denser cortical like bone. In KOS Plus the apical third of the implant contains the basal cortical screws these additionalscrews which help the implant engaging in the buccal and palatal/lingual cortical plates also, help in acquiring primary stability and later capacityas a load bearing and distributing part. It ought to be noticed that in KOS Plus implant the BCS part is consistently exceptionally polished.

SURGIAL TECHNIQUE

Not at all like ordinary implants, basal implants have an alternate surgical methodology. The method is straightforward and simple to execute and doesn't include extensive penetrating of bone drills prevents thermal injury[4, 9]. All through the surgical procedure the mode of water irrigation utilized is external and practically for any case of single pilot osteotomy with a "Pathfinder Drill" is adequate for KOS, KOS Plus and BCS implants, the kit additionally comprises of manual drills for a controlled osteotomy preparation[9,10].

Basal implantologists don't advocate raising a flap for these implants as it brings about a diminished blood supply and a sutured site is certifiably not a favorable place for the placement of immediate prosthesis[4, 9, 10]. For the BOI implant the methodology towards the bone is acquiredby raising a flap laterally and cutting into the bone with disc drills of required size a lateral direction to frame a "T" shaped osteotomy. The implant thus is placedlaterally and the flap is shut over it.[11, 12, 13, 14.]

Peri-Implant Healing (BOI and BCS Implant)

since these implants have an extraordinary design their peri-implanthealing is also unique. What regular implantologists call as "Osseointegration" is called as "Osseo adaptation" by basal implantologists, this stems from the way that the bone with nonstop functional loads remodels and adjusts over the outside of the implant, the redesigning of bone under utilitarian burdens is viewed as the fourth Dimension[4] . As indicated by reasoning of basal implantology the interaction of Osseoadaptation is done by a "Bone Multicellular Unit" (BMU), it is supposed to resemble a cutting cone with a tail, the cutting cone contains osteoclastic cells that destroy the peri-implant bone and the tail contains osteoblastic cells that lay down bone, as this unit moves in the bone the osteoclastic activity is along these lines followed by osteoblastic action. The formation of this BMU happens when the BOI and BCS implant are immediate loading and tends to remodeling of the bone under functional stress prompting improvement of this unit, and subsequently starts to healing phase and prompts formation of a thick peri-implant bone[4,15]. The course of cycles included is as per the following (4)-

Activation Phase

In this stage the precursor cells/human mesenchymalundifferentiated cellsform into osteoblasts and osteoclasts. This stage goes on for 3 days.

Resorption Phase

During this stage osteoclastic movement happens which uncoverssoft and porous bone. Osteoclastic activity happens at a pace of 40µm/day.

Reversal Phase

In this stage osteoblastic movement happens. Theosteoblasts set down neo bone in the haversian channels at a pace of 1-2µm/day.

Progressive Phase

This stage includes the osteoblasts shaping concentric lamella in haversian channels, which prompts decrease in diameter of the canal and expansion in bone thickness. At this stage the breadth of the haversian trench is 40- 50µm. The bone shaped is a Non-Mineralized Matrix Osteoid and this stage goes on for a very long time.

Mineralization Phase

After 10 days of osteoid arrangement mineralization stage starts. This stage includes two stages

a) Primary Mineralization Stage
   This stage confers primary hardness to the osteoid furthermore, represents 60% of all mineralization.
b) Secondary Mineralization Stage
   This stage confers final hardness and final morphology of bone. This stage goes on for 6-12 months.

Dormant Phase

In this stage osteoblasts form into osteocytes and line the haversian canals and take up mechanical, metabolic furthermore, homeostatic capacities.

It ought to be noticed that all through these stages the implants are under functional loads and due to which there is a continuous stimulations of the BMU for the duration of the existence of the implant, which causes the peri-implant bone dense (which increments all through the implant life) and to adjust over the surface of the implant, in the term "Osseo adaptation", and this is the manner by which rebuilding plays a key role and is called as the "fourth Dimension"[4] .

In basic terms it tends to be expressed that the peri-implant healing is a lifelong process using the idea of miniature movement furthermore, bone compression that is the reason these implants are too called as "Orthopedic Implants" as they utilize the equivalent standards of peri-implanthealing and bone densification[4]. To the extent the KOS and KOS plus implants are concerned, since these implants are surface treated, peri-embed healing happens as indicated by idea of osseointegration and remodeling is a lifelong process.

Basal Implants for Atrophied Ridges

Restoring atrophic edges is a challenge for theprosthodontist be it fixed or removable. Restoration of such cases includes broad planning including the choice of pre-prosthetic surgical procedure; basal implantology prevents any need for surgical procedures. Dissimilar to regular implantology where ridge augmentation shown to empower the placement of implant with suitable measurements, basal implants can be utilized in any size and in combination with any implant. In any case, there is a certain procedure to how atrophicridges should be restored. Following are the focuses that are considered before restoring atrophic maxilla and mandible

I. General Systemic Considerations[1,2]

As per basal implantologists it doesn't make any difference until the patient has had a recent myocardial infraction,cerebrovascular accident, immunosuppressant therapy, chemo or radiotherapy and bisphosphonate treatment. Diabetes is certifiably not an immense worry insofar as glucose levels are in control, likewise it doesn't make any difference if the patient is a smoker or not.

II. Biomechanical Considerations[4]

The evaluations of bone thickness given by Dr. Carl E. Misch are not relevant to basal implantology as the drilling succession and technique for placement is totally unique.Bone is a visco-versatile structure as is this implant, consequently, the stress shielding is dodged.

III. Where to load? [4, 15, 16]

As per theory of basal implantology the cranial bone is for all time in a condition of torsion, i.e.; there are consistent lateral stress being applied to thecranial bone consistently because of activity of the joined facial muscles, hence, there is nothing of the sort as an "unloaded" implant as lateral forces will consistently exist regardless of the implant gets a superstructure or not. Thinking about this, basal implants can be left without a superstructure till completion of the healing stage or they can get a superstructure quickly, following 3 days, 1 week, 6-8weeks, or temporary restoration can be possible for 3-6 months followed by finalrestoration.

IV. Which Jaw to Restore First???[4]

The stomatognathic framework comprises of stationary (maxillary bone) and a mobile (mandibular bone) part, the role of the mobile segment is to apply forces and the stationary part absorbs[3]a lot of the forces applied. Due to the previously mentioned purposeof the jaws, it becomes basic that the mandible ought to be restored first, likewise a conventional mandibular denture on aatrophied foundation is unstable, in this manner, chewing capacity becomes poor and continuously the related muscles lose their tonicity, on account of fixed restoration these afflictions are avoided, mandible ought to be restored first.

V. Treatment of Atrophied Ridges

a. Atrophic Mandible

Over the years two ways of thinking have created in regards to implant restoration in atrophic mandible, they are

i. Multi-Implant Concept of French School[4, 6]Engendered and established by Scortecci this school favors countless basal implants in the mandible generally around 7-12 implants. As indicated by this school basal and crestal Implants are joined to bring about a result that is rigid to the point that it doesn't allow any torsion across the mandible additionally this doesn't permit the jaw framework to reorient forces. Since, it is nearly difficult to stop

mandibular torsion, there is generation of extensive forces on the implant body which prompts over-load osteolysis and causes implant failure.

ii. Strategic Implant Positioning Concept of German School[4, 15] This school was established by Dr. Ihde. according to this school 4 implants are placed in the mandible ideally in the canine and second molar region this takes into account mandibular tortion and reorientation of forces which get compensated by flexibility of the prosthesis, in this manner, over-load osteolysis and implantfailure in prevented.

Infranerval Implantation Technique[4, 15, 17]

In the atrophic mandible with advancing resorption the IA nerve lies nearer to the crest, in such cases it gets troublesome toput crestal implants without bone augmentation or nerve repositioning. BOI implants don't need such procedures prior to the placementof the osteotomy preparation can be modified, i.e.; the osteotomy preparation for the basal disc is prepared approx. 2-3 mm below the nerve, this way the basal disc gets inserted beneath the nerve and need for extensive techniques is avoided. This strategy is likewise called as Infraneural Implantation.

b. Atrophic Maxilla[4, 6, 18]

The resorbed maxilla represents an extensive test for embed reclamations. The pneumatized sinus and the permeable bone make embed arrangement a difficult assignment. The permeable bone is dealt with by the pressurescrew inserts, though, for the sinus two procedures Have been portrayed, which depict substitute strategies of placement

i. Sinus Section Technique

In these two/three walls of the sinus are segmented to facilitate placement of the basal disc in the sinus. Basal implantologists leave the alternative of lifting the Sinus membrane and grafting on the operator. The sole motivation behind this procedure is to acquire bi-cortical support; additionally, just one implant can be put this route in each sinus.

ii. Tuberopterygoid (TPG) Screws

These implants are put in the pterygoid bone and help in offering support to the prosthesis. These are utilized in conjunct with Sinus Section procedure and are placed at 20º-45º in the bone also, the angulation between BOI implant and TPG screw ought not surpass 90º otherwise prosthesis placement gets troublesome.

iii. Zygomatic Screw Implant (ZSI)-

These are zygomatic implant that are placed in the zygomatic bone and like the BCS implant these likewise have sharp edged cortical screws that acquire bicorticalsupport

c. Cortically Fixed @ once [19, 20]

This is a new convention presented by Dr. Henri Diederich in 2013; this convention depends on basal cortical implantology and is explicitly pointed toward restoring atrophic jaws regardless of the measure of bone available with no requirement for augmentations. This isbasically a plate structure implant, which resembles smaller than usual plates (used for fracture reduction) with a abutment stage, this unique design permits them to be twisted and adapt to any surface and is anchored to bone utilizing bone expanding miniscrews . The quantity of holesrequired can be reduced; another advantage position is their isoelasticity enabling them to imitate bone. These implants are sub-periosteal implants thus far this convention has shown great outcomes however more clinical research is required.

Prosthetic Rehabilitation[4, 6, 15, 19, 20]

The point of prosthetic rehabilitation is to provide esthetics,enables hygiene practice and principally to stay away from over-load osteolysis. Esthestics are taken care with following the three FPs given by Dr. Carl E. Misch. Over-load osteolysis is prevented by providing suitable occlusal plans which can be reciprocal adjusted, group function, lingualized occlusion.

Conclusion

The innovative work these implants have gone through have made them a suitable choice for reestablishing atrophic jaws as they don't need extensive augmentation and aloe immediate loading and, they can be placed with a flapless method and can be joined with any implant. Regardless of the information accessible on their achievement in treating anvariety of cases these implants have acquired nearly nothing trust among conventional implantologists.However, it can't be rejected that basal implantology fits the guideline "Primum Nihil Nocere", i.e., "First Do No harm". At whatever surgeries are involved (conventional), basal implantcame as rescue. Additionally, with the proposed characterization we have attempted to extensively arrange basal implants primarily dependent on their morphology/structure, this grouping may help the in understanding the design that exist and will give a better comprehension of the applications and ramifications of each implant design.

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ACUTE AND SUB ACUTE TOXICITY OF METHANOLIC EXTRACT OF SPHAERANTHUS AMARANTHOIDES

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ABSTRACT

Sphaeranthus amaranthoides (SA) is one of the most popular medicinal plants used in Siddha system. It is used to treat blood disorders, helminthiasis, eczema, vomiting, abdominal discomfort, and also used to increase the semen consistency. It also prevents the destruction and aging of the viscera, muscles, nerves, bones, bone marrow, blood cells etc. To date there is no documented evidence corroborating its safety. This study thus aimed to evaluate the toxicity profile of the methanolic extract of Sphaeranthus amaranthoides. Acute and Subacute toxicity changes after oral administration of methanolic extract of sphaeranthus amaranthoides (SAME) were reported in Male adult Wistar albino rats. The results demonstrate that, a single dose and short term oral intake of methanolic *extract of Sphaeranthus amaranthoides caused no toxicity up to a dose of 2000 mg/kg b.w. Thus, prolonged uses of Sphaeranthus amaranthoides orally at lower doses and mid doses were proved to be very safe.*

KEYWORDS: *Acute, Toxicity, Lethal dose, Sphaeranthus amaranthoides, Herb, Methanolic extract*

INTRODUCTION

Plants are used traditionally for the treatment of various diseases. Therefore it is necessary to rule out the toxic effects of a particular plant. In addition toxic profile of a plant helps in enhancing the efficacy of that particular plant. Almost any substance can be harmful at some doses; similarly can be without harmful effect at lower doses. Between these two limits there is a range of possible effects, from subtle long-term chronic toxicity to immediate lethality [18]. The large array of toxic chemicals produced by plant based products, usually referred to as secondary plant compounds, is often said to have evolved as defense mechanisms against herbivorous animals, particularly insects and mammals. Many chemicals that have been shown to be toxic are constituents of plants that form part of the human diet [18].

Sphaeranthus amaranthoides Burm belongs to the family Asteraceae is a rejuvenator herb of Siddha system. Sphaeranthus amaranthoides has been used as an ingredient in certain Siddha polyherbal formulation possessing antioxidant property. The entire herb possesses therapeutic value, even though leaves has more value. The plants possess astringent and mild hot taste and hot potency. These tastes got biotransformation into hot taste after absorption. The plant has aromatic, astringent, stomachic, antispasmodic, emmenagogue and diuretic properties (12).

The phytochemical analysis of the plant showed that it contains steroids, triterpenoids, phenolic compound, flavonoids, tannins, and glycosides. The leaves of Sphaeranthus amaranthoides has have been reported for their antioxidant, antimutagenic, antimicrobial, and phytochemical activities [19]. Sphaeranthus indicus is a closely resembling plant of the same family on which many research works has been done. This article deals with the acute and subacute toxicity effects of methanolic extract of spahaeranthus amaranthoides on adult wistar albino rats.

EXPERIMENTAL SECTION

Plant Materials/Extract Preparation

The plant Sphaeranthus amaranthoides was sourced in September 2018, from the Agricultural lands of different parts of Tiruvarur and Salem district, Tamilnadu, India. The prime works like washing, drying were done. The plant materials were identified and authenticated by DR. P. Murugan M.Sc., PhD, Department of Medicinal Botany, Srisairam Siddha Medical College & Research Centre. The collected plant material was free from disease and contamination of other plants was strictly

avoided. About 2.5 kg of air-dried, powdered leaves of Sphaeranthus amaranthoides (SA) were defatted with petroleum ether (60–80 °C) to remove fat, latex and non-polar compounds of high molecular weights. The defatted plant residues were extracted by maceration in methanol for 24 h, with intermittent stirring at 45 °C, to obtain the methanol extract (SAME). The solvent was regularly changed until colour disappears. The collected extract was filtered through Whatman filter paper (No. 1). Finally the filtrate was concentrated in vacuum using rotary evaporator and the concentrated extract was dried using a freeze dryer followed by incubation in an oven (45 °C).

Oral Acute Toxicity Study: Experimental Design

The acute oral toxicity study was sanctioned to be conducted in compliance with OECD guideline 423, which stipulate the use of only three animals (OECD 423, [2]. Three of the test animals were fasted overnight (~12 h) and weighed. Test doses of Sphaeranthus amaranthoides methanolic extract (SAME) were calculated in relation to the body weight of every fasted animal; and administered via oral gavage at 2000 mg/kg (Fig 1)[1]. The animals were regularly and individually observed for behavioral changes and general toxicity signs after dosing for the first 24 h, with special attention being given during the first 4 h. Thereafter, observation was continued daily for a total of 14 days [3]. Finally, on the 15th day, weights of the animals were measured, and gross physical examinations were carried out. After sacrificing the rats, gross pathological observation was carried out on vital organs.

Oral Sub-Acute Toxicity Study (28 Days)

Procedure

The study was conducted in compliance with OECD guidelines No. 407. The experimental animals were divided into two groups of 5 rats each (160–240 g b.w.) in separate cages. The groups were treated daily with two doses of SAME (250 and 500 mg/kg b.w.) respectively for 28 days. All extracts were administered via oral gavage.

Observation

Clinical signs were observed at least twice a day during the 28-day treatment period. Body weights were measured once a week. On the 29th day, the animals were fasted overnight and blood samples collected from orbital sinus. Vital body organs were dissected, cleansed of adhering tissues and rinsed in normal saline before their weights were measured. The kidneys and livers were immediately stored in 10% paraffin for histology. Paraffin sections were made and stained with hematoxylin and eosin for a thorough histopathological study [5]. Hematological analysis of the blood samples was performed using an automatic hematology analyzer. The parameters which were evaluated included: red blood cells (RBC) count; hemoglobin (Hb); mean corpuscular volume (MCV); mean corpuscular hemoglobin (MCH); mean corpuscular hemoglobin concentration (MCHC); platelets (PLT); leukocytes (WBC) count; and neutrophils, eosinophils, basophils, lymphocytes and monocytes counts. For biochemical analysis purposes, the blood samples were centrifuged at 3000 rpm for 15 min. Diagnostic kits were used to evaluate these parameters, which included the serum levels of total proteins (TP), bilirubin, alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphate (ALP), creatinine, and albumin (ALB) . Histopathological examination was also conducted on the liver, Kidney and gonads of the treated control groups. In this study, the values obtained for the control group were considered as the reference values and statistical analysis was conducted against the control group.

Ethical Consideration

The study was conducted after having approval from the Institutional Ethical and Scientific committee, Sathyabama Institute of Science and Technology, Tamilnadu, India. Animals used in this study were not subjected to any unnecessary painful and terrifying

situations [15]. To keep the pain and suffering minimal during any surgical intervention all animals were given Xylazine anesthetic and the procedure was carried out by a well-trained person. The animals were protected from pathogens and placed in appropriate environment. The numbers of animals were reduced to the minimum possible that allows investigators achieving the scientific objectives of the study.

Histopathological Studies

The liver, brain, and kidney sections taken randomly for tissue processing were fixed in 10% neutral buffered formalin overnight at room temperature. After fixation, the tissue sections were washed with water to remove excess fixatives for about six hours and dehydrated with increased concentration of alcohol of 70% for two hours, 90% for two hours, absolute alcohol-I, II for one and half hours, and III overnight. The dehydrated tissues were cleared in two changes of xylene-for one and half hours and two and half hours. The tissues were then infiltrated with three changes of paraffin wax-for one and half hours, two and half hours, and overnight. Finally, the tissues were embedded in paraffin wax in square metal plates forming tissue blocks, by each tissue block was labeled and stored at room temperature till sectioned.

The tissue blocks were sectioned in ribbons at a thickness of 5 μm. The ribbons of the section were collected and put onto the surface of a warm water bath. The floating ribbons over the surface of warm water were mounted onto precleaned slides spread with egg albumin. The slides containing paraffin wax were arranged within the slide holder and placed in an oven with temperature of 40°c for about 20 minutes so as to fix the tissue to the slides and allowed to cool at room temperature for 30 minutes and stained regressively with routine Harris haematoxylin for 6 minutes and then eosin for 17-20 second (H and E).

The tissue sections were washed with tap water for five minutes and stained regressively with Harris haematoxylin for 6 minutes and then washed under running tape water for five minutes again. The slides were immersed in acidic alcohol for differentiation and controlling over stained haematoxylin for 1 second and then put in bluing solution (sodium bicarbonate) until they became blue. After bluing, the slides were counter stained with eosin for 17-20 seconds and then washed in tap water for two minutes. The sections were dehydrated with increasing alcohol concentration of 50%, 70%, 95%, absolute I and II for two minutes each. The dehydrated sections were cleared with xylene I and xylene II for three minutes each and permanently mounted on microscopic slides using DPX and cover slips and then observed under light microscope for the investigations of any histological change, thereby the histology of the treated groups was compared with histology of the control group. After examination, photomicrographs of selected samples of liver and kidney section from both the treated and control rats were taken under a magnification of x10 and x40 objective using automated built-in digital photo camera.

RESULTS AND DISCUSSION

Acute oral Toxicity Study of SAME (14 days)

Commonly Acute toxicity study assesses the adverse effects that occur within a short time following the administration of a single dose of a test drug. The present acute toxicity study did not show any toxicity signs and symptoms at 2000mg/kg. No morbidity or mortality was observed in the treated animals at this maximum dose during acute toxicity study. No sign of toxicity was observed in the wellness parameters during the 14-day observation period. As a result, the LD_{50} of the SAME extract could be greater than 2000mg/kg body weight. The methanolic extract of Sphaeranthus amaranthoides (SAME) may, therefore, be considered relatively safe on acute exposure.

Body weight change is an important index for assessment of toxicity [9]. In the present study, there was a gradual normal increase in

the mean body weight of the treated groups like control group. In the present study, there was a regular normal increase in the mean body weight of the treated groups like control group. At the end of Acute toxicity study the mean body weight for the control rats was 204 g. The mean body weight for rats treated with 2000 mg/kg 207 g, respectively. However, the weight gain difference between control and treatment groups was statistically insignificant

PARAMETERS	DOSES	MEAN ±SE
DAY 1	Control	180.83±2.19
	2000mg/kg	180.5±2.33
DAY7	Control	191± 3.61
	2000mg/kg	195±3.94
DAY14	Control	204±2.33
	2000mg/kg	207±3.41

Table 1: Comparison of the effect of SAME on body weight of treated and control rats during acute toxicity study.

Liver and kidneys of rats are used by many researchers to assess the safety or toxicity of herbal drugs or plant materials [10]. In the present acute toxicity study, gross pathological examination of the liver and kidneys of treatment groups did not show any major visual difference in size, shape, color, and texture compared with control group. In addition, there was no significant difference in the absolute weight of liver and kidneys of treated rats compared to control group. Our study agrees with the acute toxicity study done by Thanigavelan et al (12), which revealed that sphaeranthusamaranthoides was safe of acute exposure.

Sub-Acute (28 days) Oral Toxicity studyof SAME

Subacute toxicity study examines toxicity caused by repeated dosing over an extended period of 28 days of oral administration in rodents. This test provides information on target organs and on the potential of the test chemical to accumulate in the organism and then is used as the basis for the determination of the no observed effect level (11)

Effect of Oral Administration of Sphaeranthusamaranthoides Methanolic Extract (SAME) on General Behavior

In the sub-acute toxicity study, ratswere administered with 250 mg/kg b.w. and 500 mg/kg b.wof ofmethanolic extract of Sphaeranthusamaranthoides did not exhibit symptoms of toxicity. There was no morbidity or mortality during the study period. There was no behavioural variations in comparison with control rats.

Effect of Oral Administration of Sphaeranthusamaranthoides Methanolic Extract (SAME) on Body and Organs Weights

The rats showed increase in mean body weight in comparison with their initial body weights in a non –significant fashion. The initial mean body weight of control group was 189.681±4.9 g, and final mean body weight was 216.884±4.915 g. The initial mean body weight of rats treated with the dose of 200 mg/kg was 188.522±4.874 g, and final mean body weight was 217.5108±3.666 g. The initial mean body weight of rats treated with the dose of 400 mg/kg was 187.136±3.844 g, and the final mean body weight was 217.952±4.331 g.

The gross pathological examination of the liver and kidneys of the treated rats showed no change in color, shape, size, and texture compared to the control group. Gross observation of the liver and kidneys of the treated rats showed no significant changes compared with the control group and no significant difference was observed in the mean absolute organs weight between control and treated groups. The mean absolute weights of the liver were 7.07±0.1734g (at 200mg/kg) and 7.01±0.2277g (at 400mg/kg), compared with the control 6.61±0.7282g . Similarly, the mean absolute weights of the kidneys of rats were in the control and extract treated groups was not significantly different.

	DOSES	BODY WEIGHT (Mean ±SE)
I WEEK	CONTROL	189.681±4.9
	HD	187.136±3.844
	LD	188.522±4.874

II WEEK	CONTROL	198.585±5.703
	HD	198.538±4.874
	LD	197.146±3.844
IIIWEEK	CONTROL	208.136±5.469
	HD	208.719±4.871
	LD	206.36±3.354
IV WEEK	CONTROL	216.884±4.915
	HD	217.952±4.331
	LD	217.5108±3.666

Table 2: Comparison of the effect of SAME on body weight of treated and control rats during Subacute toxicity study.

	CONTROL	HIGH DOSE	LOW DOSE
Brain	1.87±0.0260	1.74±0.0313	1.89±0.0420
Kidney	1.41±0.0192	1.53±0.0598	1.19±0.0655
Liver	6.61±0.7282	7.01±0.2277	7.07±0.1734

Table 3: Comparison of the effect of SAME on organ weight of treated and control rats during Subacute toxicity study.

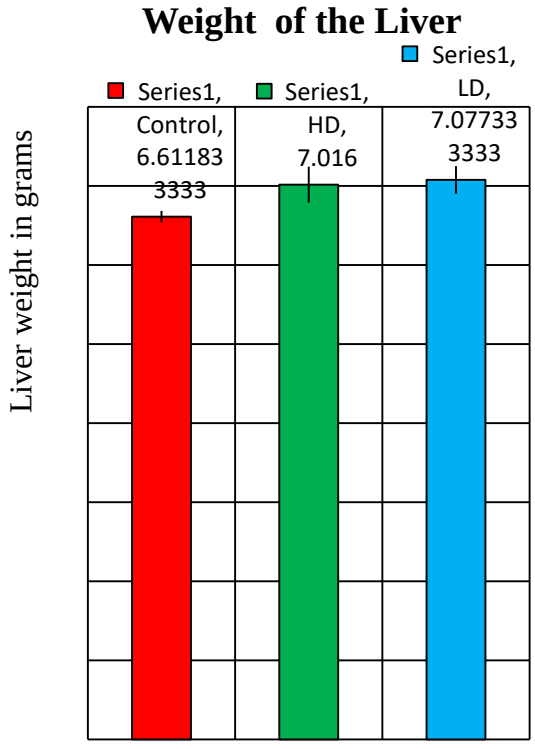


Figure 1: Bar graph of mean weight of liver in rats treated with (LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group during subacute toxicity study.

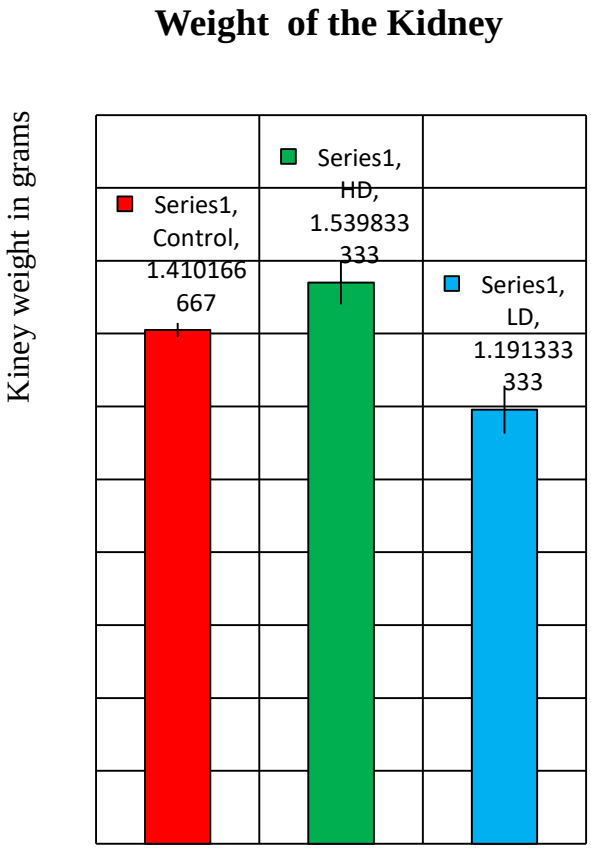


Figure 2: Bar graph of mean weight of kidney in rats treated with (LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group during subacute toxicity study.

Effect of Oral Administration of SphaeranthusamarnthoidesMethanolic Extract (SAME) on Plasma Hematological Parameters

Assessment of hematological parameters can be used to determine the extent of harmful effect of foreign compounds including plant materials on blood (14) Hematological parameters of the rats were examined as shown in Table 4. No significant changes were observed in plasma. Hematological parameters in animals treated with SAME compare with the control, with the statistical significance Hb(%) , RBC ($\times 10^6/mm^3$),

PCV(%), MCV(fl), MCH (pg),MCHC (g/L), WBC (mm^3), platelets($\times 10^3/mm^3$) ESR(mm) .

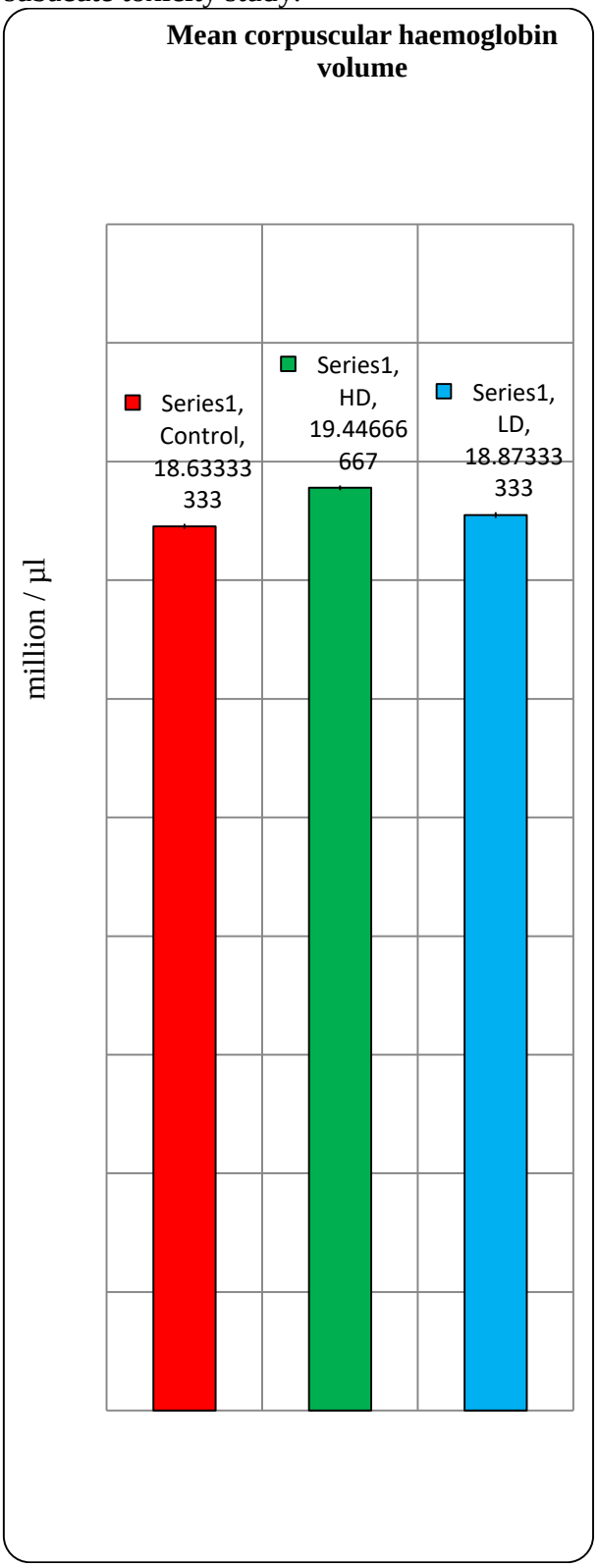
All the haematological parameters except a few are same for the animals administered with 400 mg /kg BW ,whereas all the haematological parameters are same as control for the animals administered with 250 mg /kg BW. The erythrocytes were slightly elevated when compared to control, but not in a significant in 500 mg /kg b.w. Haemoglobin values for control and low dose animals did not show any significant difference. This indicates that the 250mg of extract may not possess toxic substance that can cause anemia or other abnormalities. This may be well documented when given subchronically.

RBC-Red blood cell count, Hb-Hemoglobin ,WBC- White blood cell count,ESR-erythrocyte sedimentation rate, PCV- packed cell volume,MCV- Mean corpuscular volume, MCH- Mean corpuscular hemoglobin , MCHC - Mean corpuscular hemoglobin concentration Platelets

PARAMETERS	CONTROL	HD	LD
RBC	6.92±0.092	6.91±0.071	6.87±0.096
HB	0.44±0.07	0.37±0.062	0.46±0.076
WBC	11023.67±108	10938.67±149.97	11370.17±125.47
Platelets	1215.66±6.49	1286.66±14.34	1263.5±7.97
ESR	2	2	2
PCV	43.97±0.15	46.97±0.32	44.71±0.144
MCV	54.78±0.17	56.45±0.421	55.43±0.2678
MCH	18.36±0.05	13.44±0.048	18.87±0.06
MCHC	30.65±0.07	30.94±0.075	31.03±0.090

Table 4: Comparison of the effect of SAME on plasma haematological parameters of treated and control rats during subacute toxicity study.

Figure 3: Bar graph of mean Corpuscular haemoglobin volume in rats treated with (LD) 250mg/kg and (HD) 500mg/kg of SAME as compared to the control group during subacute toxicity study.



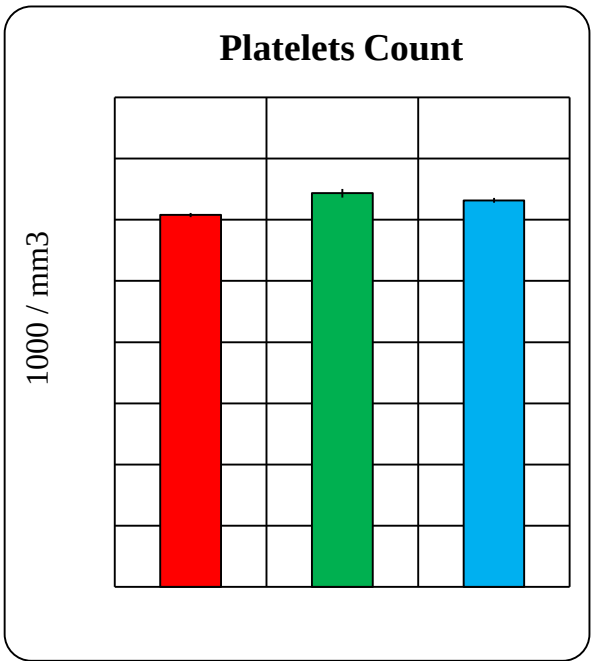


Figure 4: Bar graph of mean platelet count in rats treated with (LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group during subacute toxicity study

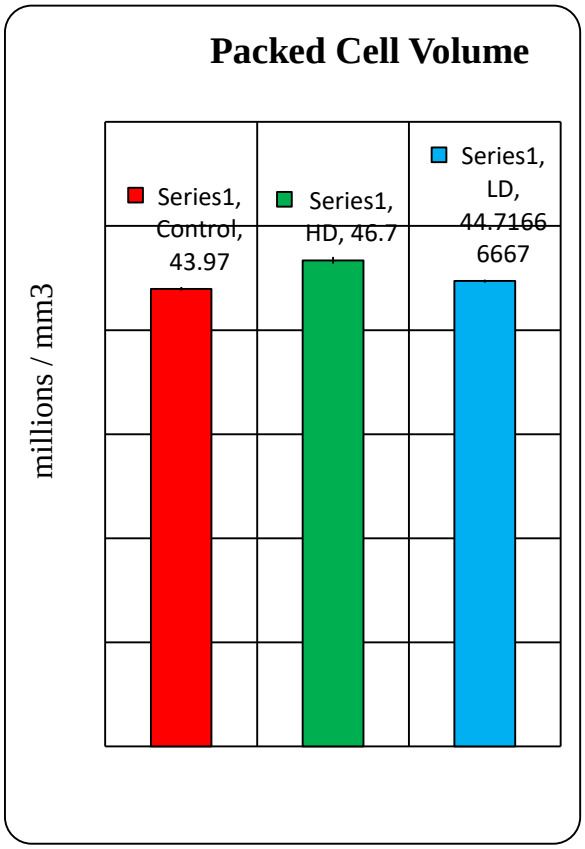


Figure 5: Bar graph of mean packed cell volume in rats treated with (LD)250 mg/kg and (HD)500 mg /kg of SAME as compared to the control group during subacute toxicity study.

Effect of Oral Administration of SphaeranthusamaranthoidesMethanolic Extract (SAME) on Serum Electrolytes Levels

	CONTROL	HD	LD
Sodium	136±0.346	135.76±0.348	135.5±0.341
Potassium	4.61±0.055	4.38±0.116	4.56±0.136
Calcium	8.4±0.08	8.38±0.071	8.25±0.072

Table 5: Comparison of the effect of SAME on mean serum electrolytes levels of treated and control rats during Subacute toxicity study.

Electrolytes (ions) play an important role in the body. They regulate the osmotic pressure in cells and help to maintain the function of muscle and nerves. If electrolyte levels vary, cell and organ functions will decline, which might lead to dangerous conditions. The electrolyte levels of the treated animals were insignificantly variable than control. The slight change in potassium levels may be physiological and should be correlated with other biochemical parameters.

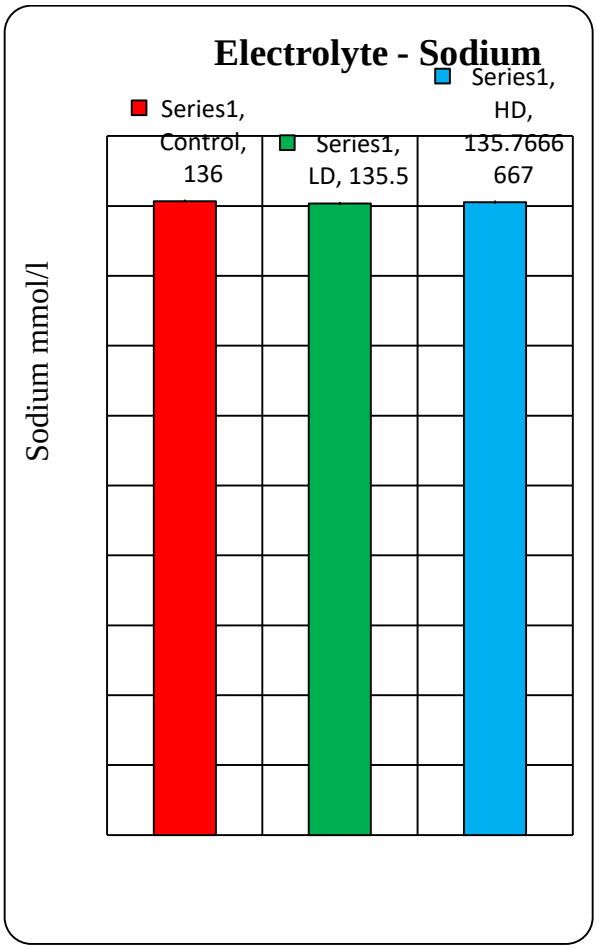


Figure 6: Bar graph of mean sodium level in rats treated with (LD)250 mg/kg and (HD) mg /kg of SAME as compared to the control group during subacute toxicity study during subacute toxicity study.

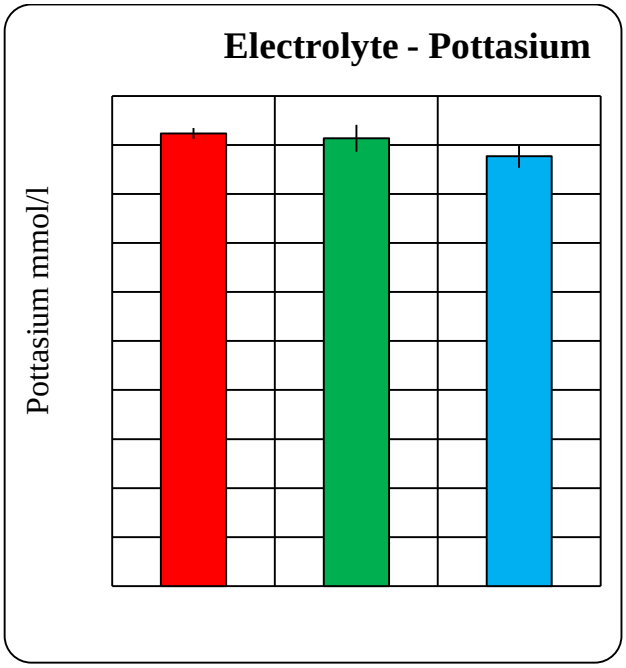


Figure 7: Bar graph of mean potassium level in rats treated with(LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group during subacute toxicity study. Sodium, potassium, chloride, calcium and Total protein concentrations in the treated groups were not significantly different from those in the control group.

Effect of Oral Administration of *Sphaeranthus amaranthoides (SAME)* Extracts on Serum Biochemical Parameters

In toxicological evaluation, biochemical parameters have significant roles as a marker because of their response to clinical signs and symptoms produced by toxicants. Evaluation of hepatic and renal function is of prime importance to assess the toxic properties of extracts and drugs .The albumin is the one of the most important plasma protein, maintain the osmotic pressure of blood and prevent the escape of fluids from blood to tissue. Almost all the Biochemical parameters of the treated animals did not show significant change when compared to the control group. GOT is more important than SGPT . The levels of markers were slightly elevated in those animals treated with 500mg / kg bw.but the changes were insignificant. These changes might be physiological.

Table 6: Comparison of the effect of SAME on mean serum Biochemical parameters of treated and control rats during Subacute toxicity study.

PARAMETERS	CONTROL	HD	LD
Bilirubin Total (mg/dl	0.26±0.008	0.009±0.009	0.008±0.008
Bilirubin Direct(mg/dl	0.1±2.53372581020338E-18	0.1±2.53372581020338E-18	0.1±2.53372581020338E-18
Bilirubin Indirect	0.16±0.16	0.15±0.009	0.16±0.008
SGOT	167.55±1.343	172.3±2.582	165.9±2.652
SGPT	58.23±3.148	58±1.715	60.06±1.972
Alkaline phosphatase	541.33±11.885	544.5±26.449	521.66±15.176
Total protein	8.3±0.014	8.41±0.024	8.35±0.031
Albumin	4.03±0.047	3.96±0.032	4.05±0.027
Globulin	4.3±0.014	4.43±0.020	4.36±0.008

Figure 8: Bar graph of mean SGOTlevel in rats treated with(LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group

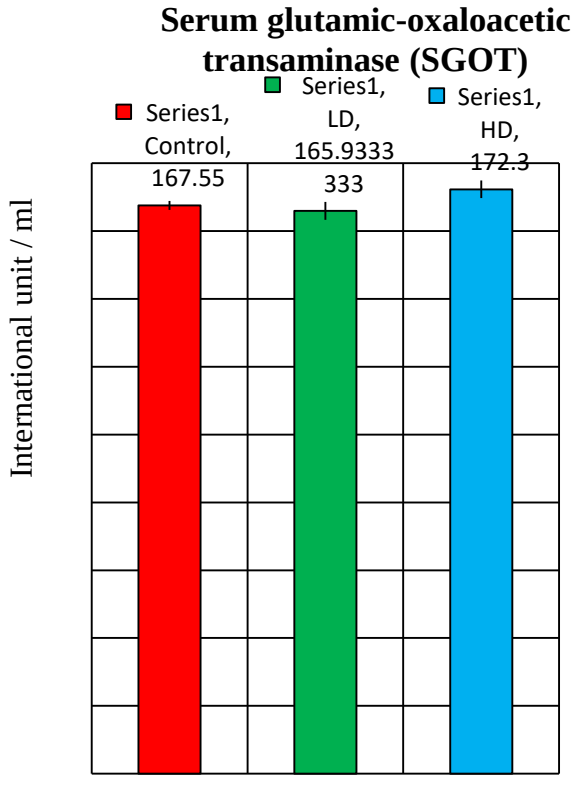


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Figure 9: Bar graph of mean SGPT level in rats treated with(LD)250 mg/kg and (HD) 500 mg /kg of SAME as compared to the control group during

3.2.7. Histopathological Examination

Histopathological studies of the liver sections in the control group (a) showed normal appearance of central vein (CV) and hepatic sinusoids (S) lined by endothelial cells with normal radiating hepatocytes. There was also normal appearance of the portal triad including hepatic portal vein, interlobular bile duct, and branches of hepatic artery. Rats treated with High dose 500 mg/ kg bw (b) of SAME and with Low dose 250mg / kg bw of SAME also showed normal appearance of the central veins (CV) and hepatic sinusoids lined with endothelial cells with normal radiating hepatocytes. Histological evaluation showed no specific change in the hepatic lobules in the treated rats as compared with the control. The result was also accompanied by the no adverse effects of the extract in any of the biochemical markers (such as SGOT and SGPT), which showed statistically insignificant changes compared with control group

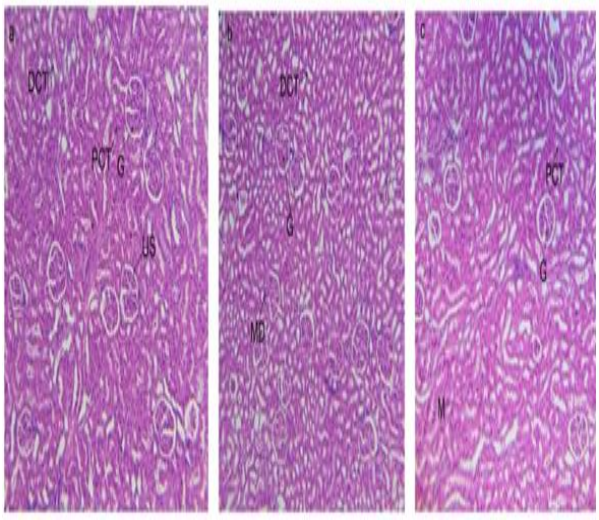

Figure : 10 Photomicrographs of the Liver sections. (H & E 10x). (a) Control rats , (b) High dose and (c) Low dose . CV= Central vein, PV= Portal vein, HC = Hepatocytes, HA= Hepatic artery, HD= Hepatic duct

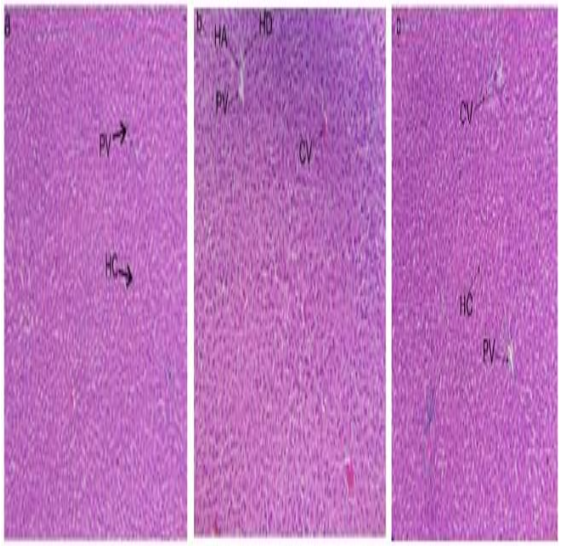

Figure 11 Photomicrographs of the kidney sections. (H & E 10x). (a) Control rats, (b) High dose (c) Low dose PCT= proximal convoluted tubule, DCT= distal convoluted tubule, MD= macula densa, G= glomerulus, US= urinary space, M = Medullary region

Conclusion

The acute toxicity study of the SAME did not produce adverse effects on the behavior and gross pathology of the rats at treated doses. Therefore, the oral LD_{50} of the methanolic extract of the Sphaeranthus amaranthoides was greater than 2000mg/kg. Meanwhile, subacute toxicity study of the SAME did not adversely affect the body weight and hematological and biochemical parameters of tested doses. There were no signs of toxicity observed in the kidney and liver sections of treated rats. However, well designed sub chronic and chronic toxicity studies should be carried out in order to set the clear picture of the safety of the plant part before develop in Sphaeranthus amaranthoides based health product.

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ADVANCEMENTS IN LOCAL ANAESTHESIA- A REVIEW

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ABSTRACT

Intra oral local anesthesia is perceived as a painful and an anxiety causing dental procedure. Most of the researches are focused on improvement in the area of anesthetic agents, delivery devices and technique involved. Newer technologies have been developed to reduce the pain of injection and adverse effects. These include buffering the local anesthetic, warming the local anesthetic, applying topical anesthesia before injection, reducing speed of injection using finer needles with electric delivery devices[1]. Sterile local anesthesia should be used and effort should be made to reduce the speed of injection[2]. This article will discuss in detail about the various advanced techniques of local anesthesia.

Key words: *Dental anesthesia, local anesthesia delivery device, computer controlled anesthesia, vibratory stimulation.*

INTRODUCTION

A significant number of patients still perceive local anesthesia as a painful and anxiety causing dental procedure. The achievement of good local anesthesia requires knowledge of agents being used, the neuroanatomy involved, and best techniques and devices available. Injection of local anesthetic is the greatest source of patient fear[3]. The most widely used devices are WAND, comfort control syringe and iCT.

Materials and methods:-

VIBROTACTILE DEVICES

Some of the newer local anesthetics delivery systems aimed at easing the fear of needle take the advantage of gate control theory of pain management[4], suggesting that pain can be reduced by the simultaneous activation of nerve fibres through the use of vibration.

Vibra Ject:-

It is a small battery operated attachment that snaps on to the standard dental syringe. It delivers a high frequency vibration to the needle that is strong enough for the patient to feel[5]. Research showed effectiveness of vibra jet showed mixed results. They also found no statistically significant decrease in pain scores at needle insertion or anesthetic injection.

Dental Vibe:-

This also utilizes vibratory mode of anesthesia delivery to the patients. It is a cordless, rechargeable, handheld device delivering pulsed, percussive micro-oscillations, to the site where it is administered. It also lights the injection area with an attachment to retract lip or cheek.

COMPUTER- CONTROLLED LOCAL ANESTHETIC DELIVERY SYSTEMS

It can reduce pain by controlling the speed of anesthesia delivery of a small amount of anesthetic at a slow speed, which reduces pain not only from the resistance felt in the tissues, but also from anesthesia taking effect simultaneously with injection, which in turn allows the anesthetic to be injected into tissue , that has already been anesthetized. The WAND is known for a longer time period in market , known for its ease in operation due to its light weight and circumference , which is half that of traditional syringe. However, it is difficult to consider it as speed controlling device , because it doesn't have a syringe needle and difficult to use in posterior teeth. It is suited for children's anterior teeth or as preliminary anesthesia method. Aspiration is not a mandatory criterion for selecting CCLAD device.

JET INJECTORS:-

It is based on the principle of using mechanical energy source to create a release of pressure sufficient to push a dose of liquid medication through a very small orifice, creating a thin column of fluid with enough force that it can penetrate soft tissue into subcutaneous tissue without a needle. Jet injectors are fast and easy to use, with little or no pain, less tissue damage and faster drug absorption at the site of injection.

Syrijet :-

It is been on market for past 4 decades and has been improved over the years. It accepts 1.8 ml cartridges of LA solution, actually ranging from 0.2 -2ml and is autoclavable.

SAFETY DENTAL SYRINGES

Use of safety syringe minimizes the risk of accidental needle stick injury. Surveys have reported with wide dissatisfaction on usage of the syringes.

UltraSafe Syringe:-

It is a disposable syringe and needle with a plastic syringe barrel, with a retractable needle sheath. The difference between the syringe is that an ultra safe plus XL syringe is that , the ultrasafe syringe is that , the entire assembly is disposable not autoclavable.

SafetyWand:-

Safety Wand has been used with CompuDent system. It has a pen like grasp that allows maximum tactile control and an auto retracting design that shield the needle, when not used. It is lighter and safe to use. It is the first patented device compliant with OSHA regulations under federal needlestick safety Act.

DEVICES FOR INTRA-OSSEOUS ANESTHESIA

Several systems have been devised for intraosseous anesthesia. Although, differences exist, their main aim is to inject into the cancellous bone adjacent to the tooth apex.

Stabident:-

It is safe and effective and its advantages being inexpensive and can be used with an equipment , existing in a dental office. Disadvantage being perforation is to be made in a reasonably accessible and visible location , distal to the tooth to be anesthetized.

Conclusion

An area of future interest is the development of newer improved devices and techniques for achieving profound anesthesia. Syringe micro vibrator , a new device introduced in dentistry , helps to alleviate pain and anxiety from intraoral injection.

Local anesthetics have grown leaps and bounds in the aspect of techniques , making the patients relieve from anxiety and fear from injections. Hence, more techniques are to be introduced and implemented in near future, to make patient comfortable and familiar with the devices available for the dental procedures in near future.

Conflict of interest:-

There are no conflict of interest declared.

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6. Utility of vibratory stimulation for reducing intraoral injection pain
7. *Ozgur Erdogan, Anatachai Sinsawat, Sudeep Pawa, Duangtawan Rintanalert and Suchada Vuddhakanok.*

PHYTOCHEMICAL AND GC-MS ANALYSIS OF SPHAERANTHUS AMARANTHOIDES BURM

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ABSTRACT

***Objective**: To isolate and evaluate the phytochemical constituents of Sphaeranthus amaranthoides using GC-MS.*
***Method**: Preliminary phytochemical screening of the extract was carried out according to the standard method described by Brindha et al. GC-MS analysis was performed on the methanolic extract of S. amaranthoides to find out the chemical constituents.*
***Results**: Phytochemical screening revealed the presence of steroids, alkaloids, sugars, phenolics, flavonoids, saponins, tannins, and amino acids to a spotted degree. GC-MS results revealed the presence of 15 different phytocompounds, viz., 3,4-Xylyl, 3,5-di-tert-butylbenzoate, n-Hexadecanoic acid, , 17.beta. -Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid, 3-Cyclopenten-1-one, 3-hydroxy-2-(1-hydroxy-3-methylbutylidene)-5-(3-methyl-2-butenylidene)- 5 ,17.beta. -Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid methyl ester 6, Indan, 6-tert-butyl-4-ethyl-1,1-dimethyl -7, 9,12-Octadecadienoic acid (Z,Z)-, methyl ester 10(E),12(Z)-Conjugated linoleic acid , 9-Octadecenoic acid, (E)-Octadecanoic acid, 9.12-Octadecadienoic acid (Z, Z)-, 2,3-dihydroxypropyl ester,1,8 ,11-Heptadecatriene, (Z,Z)- , 11-Methyltricosane , Nonane, 5-butyl- ,1,3-Benzenedicarboxylic acid, bis (2-ethylhexyl) ester etc.*
***Conclusion:** The presence of various bioactive compounds confirms the application of Sphaeranthus amaranthoides for various diseases by means of a herbal system of treatments.*

Introduction

The significance of plants is known to us well. Many Medicinal plants have been used for centuries as remedies for a number of human diseases and from these plants many potential drugs have been isolated . Discovery of such herbal drugs increases the awareness on Siddha system . herbal drugs are easily available, less expensive, safe, and efficient . According to World Health Organization (WHO), medicinal plants would be the best source to obtain variety of drugs. About 80% of individuals from developed countries use traditional medicines, which has compounds derived from medicinal plants. However, such plants should be investigated to better understand their properties, safety, and efficiency [2]. Medicinal plants contain some organic compounds which provide definite physiological action on the human body. Some of the important bioactive substances include tannins, alkaloids, carbohydrates, terpenoids, steroids and flavonoids [6,7]. These bioactive substances are synthesized by primary or to a certain extent secondary metabolism of living organisms

Secondary metabolites are chemically and taxonomically diverse compounds with doubtful function. They are widely used in the veterinary, agriculture, scientific research and many other areas

Natural constitutions which is native of plant origin can be derived from any part of the plant like bark, leaves, flowers, roots, fruits, seed, etc.[4] The medicinal properties of the plants unique to particular plant species or groups are depends on the concept that the combination of secondary products in a particular plant is taxonomically distinct[5].The spectrometric and chromatographic screening method could provide the needed preliminary observations to select crude plant extracts with potentially useful properties for further chemical and pharmacological investigations[3].The determination of phytoconstituents is largely performed by the relatively laborious techniques such as gas (GC) and liquid chromatography (LC) combined with specific detection schemes. GC-MS has become firmly established as a key technological metabolic profiling in both plant and non-plant species.

Sphaeranthus amaranthoides. S. amaranthoides Burm.f. is a small procumbent herb, with appressed hairy

leaves palmately 3-foliolate. The species are low annuals with spreading branches, stem e erect, glabrous, sometimes as thick as the little finger, but short, branches e not winged and 7-13 inches, leaves 2-5 inches, linear, oblong narrowed at the base. This plant is well known for its medicinal value for the treatment of eczema, blood disorder, stomach worms, filarial, fever and as a remover of kapha, vata, and piles. It is also known to cure skin diseases.[8] In the present work, qualitative and quantitative phytochemical analysis were carried out in Sphaeranthus amaranthoides.

Material and methods

Collection of the plant material

The plant, S. amaranthoides, was collected from the Salem Dist., Tamil Nadu, India. The primary tasks, like washing, drying, etc., were done. The plant materials were identified and authenticated by DR.P.Murugan, M.Sc, Ph.D, Department of Medicinal Botany, Sri Siddha Medical College & Research Centre. The collected plant material was free from disease and also free from contamination of other plants.

Preparation of plant extracts

100 g of S. amaranthoides air-dried and coarsely powdered plant material was extracted with 500 mL of methanolic solvent by using a Soxhlet extractor. After extraction, the sample was kept in the dark for 72 h with intermittent shaking. Then the solvent was evaporated under reduced pressure using Rota-vapor to obtain viscous semi-solid masses.

Phytochemical analysis

The methanolic extract was tested for steroids, alkaloids, sugar, phenolic compounds, flavonoids, saponins, tannins, anthraquinone and amino acids. Phytochemical screening of the extract was carried out according to the standard method.15th

GC-MS examination

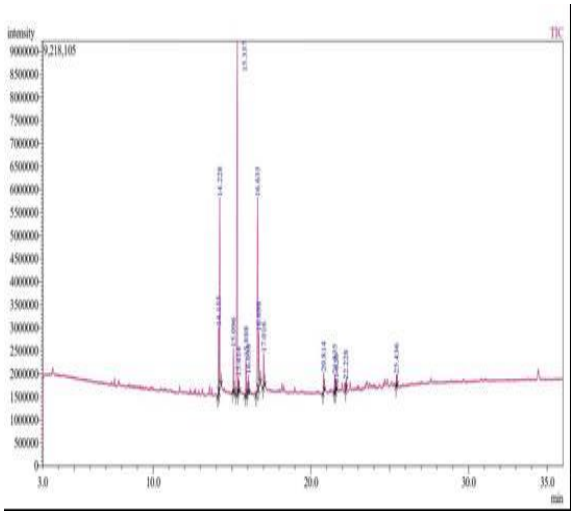
The shimadzu GCMS QP 2020 was used in the analysis. The method was employed with a fused silica column, packed with SH-Rxi-%Sil MS (30 m 0.25 mm ID 250m df) and the components were separated using Helium as the carrier gas at a constant flow of 1 ml/min. The injector temperature was set at 280°C during the chromatographic run. The 1L of extracted sample injected into the instrument at the oven temperature was as follows: followed by 280 °C at a rate of 10 °C min1 and 280 °C for 3 minutesThe mass detector conditions were: transfer line temperature of 280 °C; ion source temperature of 230 °C; and ionisation mode electron impact at 70 eV, a scan time of 0.2 sec, and a scan interval of 0.1 sec. The fragments range from 40 to 550 Da. The spectrums of the components were compared with the database of spectrums of known components stored in the GC-MS NIST (2017) library.

Results and discussion

The phytochemical screenings of S. amaranthoides extract revealed that the methanolic extract contains Steroids, Sugar, Flavanoids, Alkaloids, Phenolics, Saponins, Aminoacids, and Tannins except Anthraquinone (Table 1)

Table 1: Preliminary phytochemical screening of methanolic extract of *Sphaeranthus amaranthoides*.

S.NO	COMPOUNDS	METHANOLIC EXTRACT
1.	Steroids	+
2.	Sugar	+
3.	Flavanoids	+
4.	Alkaloids	+
5.	Phenolics	+
6.	Saponins	+
7.	Amino acids	+
8.	Tannins	+



The results pertaining to GC-MS analysis led to the identification of a number of compounds from GC fractions of the methanolic extracts of Sphaeranthus amaranthoides. They were recognised through mass spectrometry attached to GC. A GC-MS analysis of a methanolic extract of Sphaeranthus amaranthoides is shown in (Table 2). The result revealed the presence of 15 different phytocompounds viz.,(5.5%).3,4-Xylyl3,5-di-tert-butylbenzoate, (17.0%) Hexadecanoicacide, (3.5%) 17.beta.-Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid methyl ester, (34.19%) 3-Cyclopenten-1-one, 3-hydroxy-2-(1-hydroxy-3-methylbutylidene)-5-(3-methyl-2-butenylidene)-, (1.14%) 17.beta.-Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid methyl ester, (3.04%) Indan,6-tert-butyl-4-ethyl-1,1-dimethyl-,(1.38%) 9,12-Octadecadienoicacid (Z, Z)-, methylester, (21.08%) 10 (E), 12 (Z)-Conjugatedlinoleicacid, (5.65%) 9-Octadecenoicacid, (E)-(2.66% (1.51%) Octadecanoicacide9,12-Octadecadienoic acid (Z,Z)-, 2,3-dihydroxypropyl ester, (1.42%) 1,8,11-Heptadecatriene, (Z, Z)-, (0.77%) 11-Methyltricosane, [0.56%] Nonane, 5-butyl, (0.85%) 1,3-Benzenedicarboxylic acid, bis (2-ethylhexyl) ester respectively. In a study

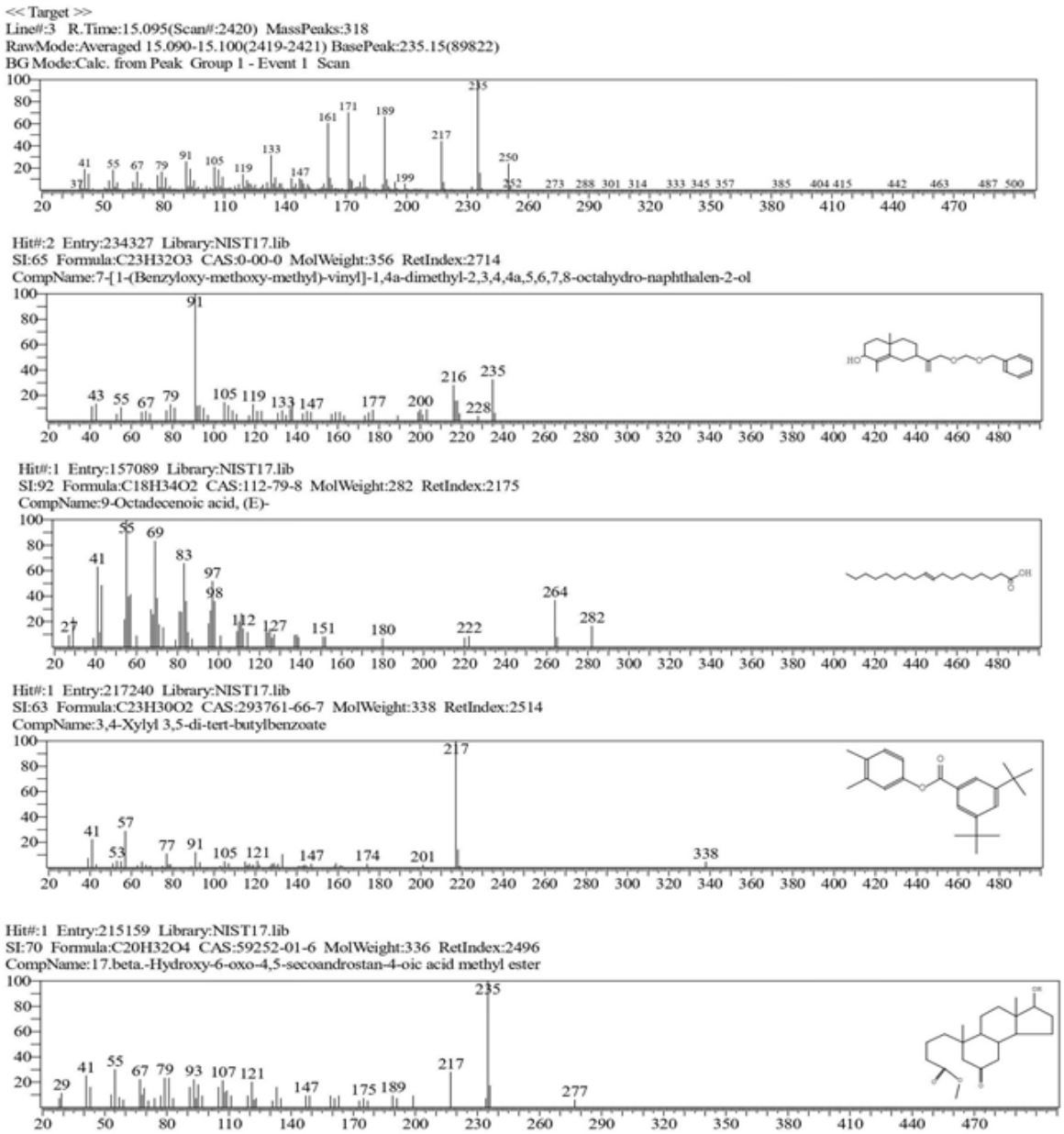
P #	R.Time	Area	Area%	Height	Height%	Name
1	14.155	3630368	5.50	1443174	6.22	3,4-Xylyl3,5-di-tert-butylbenzoate
2	14.228	11232709	17.02	4172076	17.98	n-Hexadecanoicacid
3	15.096	2146580	3.25	950028	4.09	17.beta.-Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid methyl ester
4	15.337	22564641	34.19	7622054	32.85	3-Cyclopenten-1-one, 3-hydroxy-2-(1-hydroxy-3-methylbutylidene)-5-(3-methyl-2-butenylidene)-
5	15.414	752352	1.14	330452	1.42	17.beta.-Hydroxy-6-oxo-4,5-secoandrostan-4-oic acid methyl ester
6	15.888	2003215	3.04	799939	3.45	Indan,6-tert-butyl-4-ethyl-1,1-dimethyl-
7	16.038	912144	1.38	417711	1.80	9,12-Octadecadienoicacid(Z,Z)-,methylester
8	16.633	13911518	21.08	4187155	18.05	10(E),12(Z)-Conjugatedlinoleicacid
9	16.698	3726515	5.65	1201783	5.18	9-Octadecenoicacid,(E)-
10	17.016	1754486	2.66	728746	3.14	Octadecanoicacid
11	20.814	993749	1.51	426771	1.84	9,12-Octadecadienoic acid (Z,Z)-, 2,3-dihydroxypropyl ester
12	21.535	936431	1.42	353713	1.52	1,8,11-Heptadecatriene,(Z,Z)-
13	21.620	509310	0.77	132960	0.57	11-Methyltricosane
14	22.228	366797	0.56	185721	0.80	Nonane,5-butyl-
15	25.436	558223	0.85	247539	1.07	1,3-Benzenedicarboxylicacid,bis(2-ethylhexyl)ester
		65999038	100.00	23199822	100.00	

done by Geethalakshmi et al., the methanolic extract of Spaeranthus amaranthoides showed the highest antioxidant activity of any other extract. 13th.Sphaeranthus amaranthoides methanolic extract is known to have antioxidant, antimutagenic, and antimicrobial properties.The rich antioxidant properties of Sphaeranthus amaranthoides might be due to the presence of carotenes, Neoxanthin, Chlorophyll a, Chlorophyll b, lactein, vialaxanthin, and pheophytin in it (15).

Individual diffraction of some dominating compounds is shown in fig.2.

Conclusion

The present study results confirmed the presence of phenolics, alkaloids, steroids, saponins, tannins and flavonoids with varied degree. In addition to this, GC-MS profile can be used asbiochemical markers in the pharmaceutical industries to identify the genuine mother plants and distinguish from its adulterants. Thus the presence of various bioactive compounds confirms the application of Sphaeranthus amaranthoides for various diseases by herbal system of treatments.



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RADIOLOGIST'S PERSPECTIVE ON COMPUTER GUIDED IMPLANT SURGERY: REVIEW

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ABSTRACT

Surgical guide gives stability in the surgical field and enhanced the accuracy in implant positioning based upon prosthetically driven restoration, thus ensuring predictable treatment outcomes. Successful rehabilitation lies in the correct sequence of surgical and prosthetic procedures. Whenever a staged approach of implant placement is planned, the clinician can effectively use the initially placed implants as anchors for the surgical template during the second phase of implant surgery. Keywords: Implantation, dental; dental implant; implant-supported dental prosthesis; dental restoration, surgical template.

Key Words: *Surgical Template, CBCT, Implant Planning*

INTRODUCTION

As dental implants increased in popularity as tooth replacement therapy, the accurate assessment of patient anatomy and the collaboration between restorative clinicians and surgeons have become critical determinants of successful outcomes. Advances in digital technology have enabled the development of systems that can assist the clinician in diagnosis, treatment planning, and the surgical treatment itself. Three-dimensional computer-assisted interactive implant planning software tools have sufficient accuracy and reliability required for predictable clinical use.[1,2]

Implant planning software allows one to virtually plan the implant surgery and to derive surgical templates from the information acquired. Surgical guide is the one that allows the practitioner to accurately place the implant with a predefined insertion path.

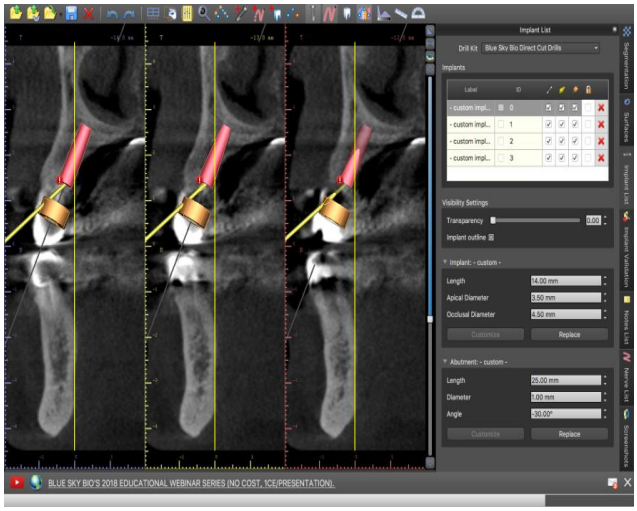
IMPLANT-GUIDED SURGERY PLANNING

Cone Beam Computed Radiograph plays vital role in Implant Assessments. The analysis of CBCT data includes the right qualitative and quantitative assessments of all relevant anatomy and boundary conditions. A condition is defined as any and every one anatomic constraints of an anatomic zone which will limit or influence implant placement and subsequent final restorations.[3]

Once measurements are wiped out the cross-sectional images, the clinician can virtually select and place implants within the region of interest. Most implant planning software packages do include implant libraries with most of the available implants within the market and every one the compatible abutments.4 (Fig1)

All systems of surgical guide use sleeve and drill combinations for the guidance. Different types of metal sleeves such as Pilot sleeve, Master Sleeve, Custom Sleeve and Drill Spoon during preparation of implant cavity, consistent with the virtual planning (Arisan et al. 2010). First, a fit of surgical guide with tooth or mucosa should be precisely the same thereupon at the time of preoperative CT acquisition, because a scan template used on the CT acquisition are often

customized to a surgical guide for implant surgery. Second, data on the diagnostic software as numerical form are often directly used for personalisation of surgical guide. Third, a milling cutter can reduce displacement possibility of implant cavity position from virtual planning, because the cutter is initially wont to shave bone surface as flat before drilling, and therefore, drills are difficult to slide. Moreover, sleeves are often selected due to inter-occlusal distance between jaws.[5,6] (Fig 2)



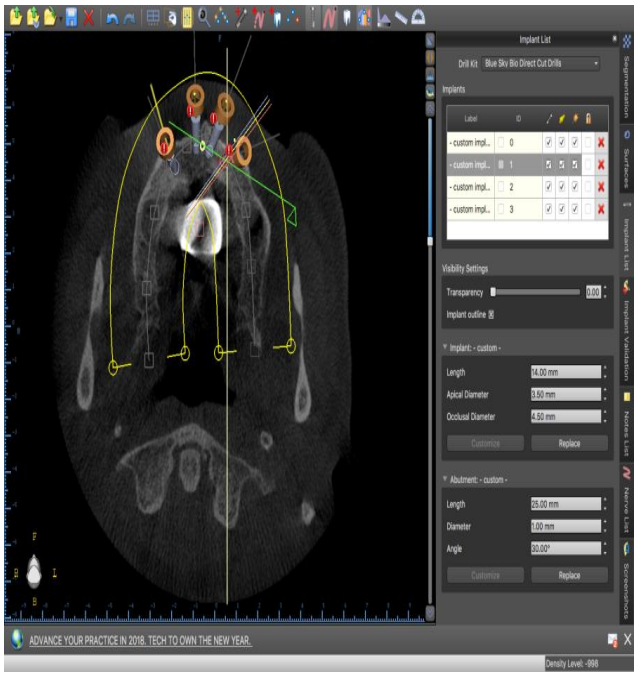


Fig 1: Virtual Implant placement using CBCT data (Bluskybio software)

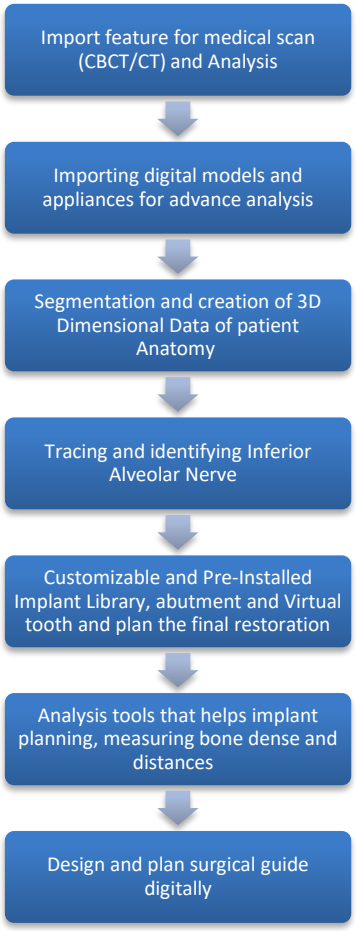


Fig 2: Schematic representation of Guided Surgery Planning

SURGICAL GUIDES

Once the implant is virtually planned, the project is often transferred to the clinical setting by employing a surgical guide. Surgical guides are appliances that are computer designed and are fabricated of an acrylic by a process called stereo lithography. The surgical guides contain steel sleeves with a predefined diameter to guide the drills during the osteotomy process. Parameters such as Inner Diameter, Outer Diameter, Height and Lip of the Sleeves should be considered before preparing the guide. There are differing types of surgical guides like a pilot guide, which allows the clinician to make the initial osteotomy. After the pilot osteotomy is made, the guide is removed and therefore the remainder of the method is completed free- handed. Surgical guides are often categorized consistent with the sort of stabilization they need like teeth, bone, or soft

tissue. Surgical guides for edentulous patients are stabilized through temporary fixation pins which will even be planned using advanced tools within the software packages. Two methods for a computer-based transfer are available: direct navigation and stereolithographic drill guides.[7]

At present, there are numerous third-party implant planning software programs like Implant (Materialise Dental Inc., Glen Burnie, MD, USA), Invivo5 (Anatomage, San Jose, CA, USA), Nobel Clinician (Nobel Biocare, Goteborg, Sweden), OnDemand3D (Cybermed Inc, Seoul, Korea), Virtual Implant Placement software (BioHorizons, Inc, Birmingham, AL, USA), coDiagnostiX (Dental Wings Inc, Montreal, CA, USA), and blue Plan (BlueSkyBio, LLC, Grayslake, IL, USA), 3SHAPE Etc., A CBCT reconstruction is obtained from all the pictures that are created and visualized from a special perspective than how the info were initially captured. Thus, a cross section, bird's eye view, multiplanar views, volume renderings, et al. are all considered CBCT reconstructions. For implant planning purposes, the cross section is that the primary diagnostic image used for the assessment of bone volume and quality. Completely edentulous patients should be scanned wearing radiographic stents with radiopaque markers (barium sulphate) to assist within the localization of specific cross sections and/or proposed implant site locations. These radiopaque markers are important to correlate tooth position with reference to the alveolar bone and CBCT cross sections. (Fig 3) Optical Scanning of models produces STL file which is additionally useful to correlate the teeth position. (Fig 4) If a surgical guide is to be produced, the precise protocols of the precise company must still be taken into consideration. In other situations, a replica of the denture is often used with fiduciary markers. a crucial factor when using radiographic guides both for dentate and edentulous patients is that the guides are positioned correctly, fully seated, and during a stable position during the scanning process.[8,9](Fig 5)

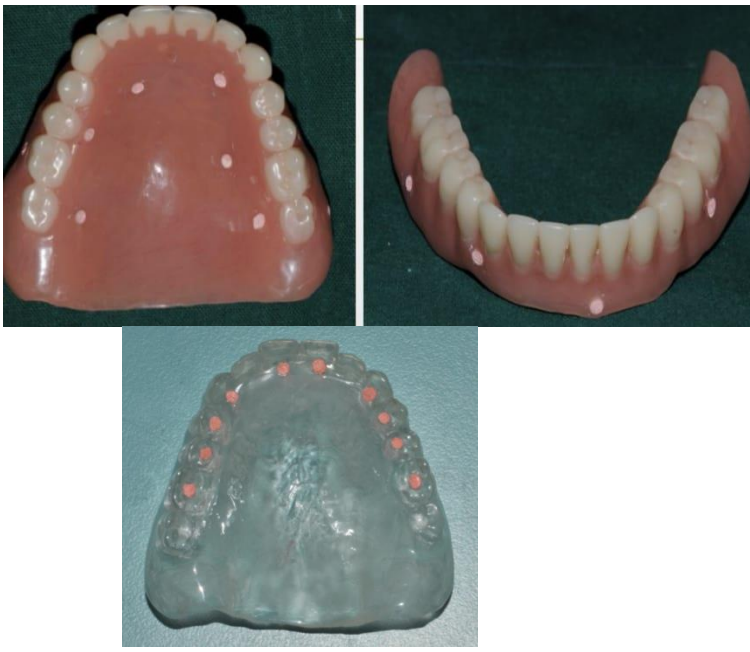


Fig 3: Radiographic Markers

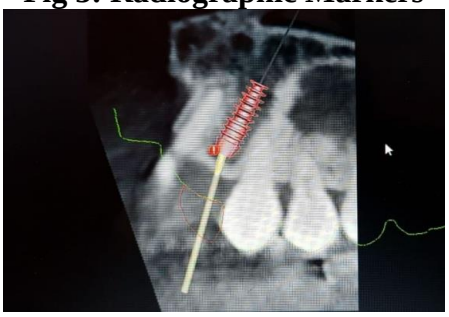


Fig 4: Stitching of optical scanning image to CBCT data

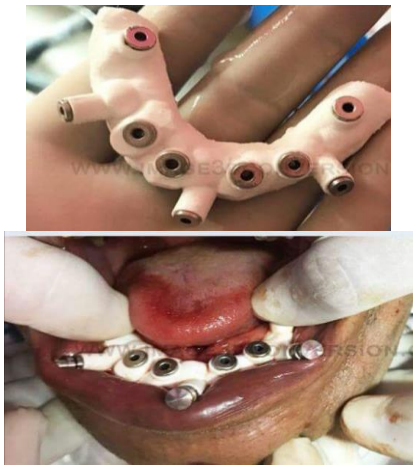


Fig 5: Fabrication of Surgical Guide (left) Placement of Guide inside mouth (right)

VOLUME RENDERING TOOLS

Volume renderings can greatly aid within the 3D visualization of implant locations and angulations and within the assessment of implants for restorative considerations. These renderings also can aid in assessing the

available space for any particular restorative goal. However, they're generally lacking within the ability to accurately depict the interior anatomy and thus should only be used after or with simultaneous cross sectional analysis. the precise surface morphology of volume renderings can also not accurately represent the patient's anatomy. due to this, it's important to recollect that the 3D models are to be used as a complement to the diagnostic and planning process. Three-dimensional models offer a broad picture of the general anatomy like root eminences, bone defects that originate from healing irregularities or concavities caused by bone atrophy, excess sharp bony edges, and tooth positioning. Finally, volume renderings can function an academic tool for the patients to know how the whole process works.

Some software programs allow manipulating the 3D volumes and creating high- resolution models by using advanced segmentation tools. Scatter originated from metallic restorations causes a detriment within the image and 3D rendering quality. Scatter are often manually erased or segmented with advanced tools from the 3D volume rendering but can't be eliminated within the 2D images.[10]

CONCLUSION

The invention of Cone beam Computed Radiography, guided surgery and digital scanning software's, provide clinicians more confident on both tissue supported and teeth supported implant surgeries. The stability of surgical guide's stable fixation ensures the accuracy in implant positioning based upon the future restoration. The outcome of treatment still relies on case selection, judgement of clinicians, prosthetic and surgical protocols. Complete treatment planning is important for selection between conventional and guided cases .

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The authors report no conflicts of interest related to this study.

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ALVEOLAR RIDGE AUGMENTATION – A REVIEW

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ABSTRACT

Bone Grafts and bone graft substitutes support regeneration of bone in bone defects and can be used for bone augmentation. Alveolar ridge augmentations are classified based on their morphology and severity. Bone graft augmentation techniques can be used for the application of socket defect grafting, horizontal ridge augmentation, vertical ridge augmentation and sinus augmentation[(1)]. To yield maximum results for each of these applications, a variety of different techniques is employed.This topic gives an overview on Alveolar ridge augmentation and various materials used and its application in the field of implant dentistry and oral and maxillofacial surgery.

Keywords: *ALVEOLAR RIDGE AUGMENTATION, BONE GRAFTS, BONE SUBSTITUTES*

INTRODUCTION

Bone Grafts and bone graft substitutes support regeneration of bone in bone defects and can be used for bone augmentation. Alveolar ridge augmentations are classified based on their morphology and severity. Bone graft augmentation techniques can be used for the application of socket defect grafting, horizontal ridge augmentation, vertical ridge augmentation and sinus augmentation.[(2)] To yield maximum results for each of these applications, a variety of different techniques is employed. The bone grafting methods include particulate grafting, membrane use, block grafting, and distraction osteogenesis, either alone or combination. With the availability of various grafting materials like autograft, allograft, xenograft and alloplast, though the autograft considered the "gold standard" by which other materials are osteoinductive, osteoconductive and osteogenic properties no risk of infections disadvantages of autograft are low availability of bone volume, required second operative site, morbidity associated with their harvesting, mainly from chin, particulate autografts resorption rate is high[(3)]. The disadvantages of allografts are possible infections and antigenicity risks. The artificial bone substitutes include combinations of calcium phosphates fabricated under different conditions, which yields different physical properties and resorption rates and it is readily available and easy to use. If an autogenous bone transplant is difficult to perform also it has low flexibility and ability to resorb and remodel to adapt to changing conditions fillings with artificial bone substitutes can be performed. some literature recommended the use of hydroxy apatite, beta tricalcium phosphate for alveolar ridge augmentation procedure. approximately 25% bone loss occur after the first year of the bone and 40–60% loss of alveolar volume occur during the first 3 years after a tooth is lost. Thus, the resulting ridge deficiency is primarily the result of the gradual loss of the horizontal dimension accompanied by a rapid loss of bone height (Carlsson et al. 1967). alveolar bone loss could be congenital, trauma, pathology, infection, or a consequence of periodontal disease and tooth extraction.

PRINCIPLES

To promote primary wound closure passive and tension-free wound closure. In orderto reduce the risk of membrane exposure, wound contraction, patient discomfort. Factors Assist in proper wound healing, enhancing cell proliferation and differentiation provides blood, oxygen, and nutrients to the tissues also acts as a source of angiogenic and osteogenic cells[(4)]. Protecting initial wound stability and integrity the placement of bone grafting materials to

favour and promote healing in osseous defects or to augment edentulous ridges to allow installation of dental implant become a gold standard treatment in implant dentistry. Cell Exclusion is used to prevent gingival fibroblasts and / or epithelial cells from gaining access to the wound site. Space is created beneath the barrier membrane, completely isolating the defect to be regenerated from the overlying soft tissue. Scaffolding: the space which is present initially becomes occupied by a fibrin clot, and it serves as a scaffold for the bone cells. protecting the clot is important for the formation of granulation tissue and subsequent bone formation (Schenk et al. 1994)[11,12].

TREATMENT OBJECTIVES

The objective behind any crestal bone augmentation procedure is to establish sufficient bone availability for safe and predictable dental implant therapy, as well as for getting adequate bone thickness around the installed implant.[10]

To achieve long-term stability of peri-implant health and good esthetics and avoid complications around functional implants by get at least 2 mm of bone on the buccal side

DIAGNOSIS AND TREATMENT PLANNING:

While diagnosing and treatment planning relative contraindications are need to be taken into consideration:

Medical conditions that may impair normal bone healing - diabetes mellitus (Colombo et al. 2011; Schlegel et al. 2013). when compared between controls diabetic to healthy patient in osseointegration was achieved in both groups (Retzepi et al. 2010)[5]. In previous studies, uncontrolled diabetes showed an increased rate of infection complications and a less predictable outcome

Smoking has also been found to affect the long-term prognosis of Osseointegration Clinical studies have reported that in smokers higher rates of implant failure.Larger number of complications around successfully integrated implants (Roos-Jansaker et al. 2006)[13,14], showed higher incidence of peri-implant mucositis and periimplantitis (Heitz-Mayfield 2008) (Bain &Moy 1993)[8]. non-smokers, the augmentation procedure was successful in 95% of the cases, whereas in smokers it was successful in only 63%

Cologne Classification of Alveolar Ridge Defects (2013)[7]

Cologne used Three-part codes to describe the effect of the alveolar ridge as comprehensively as possible with a view to existing therapeutic options:

Part 1: Orientation of the defect

h: horizontal

V: vertical

c: combined

S (or +S): sinus area

Part 2: Reconstruction needs associated with the defect

1. low: <4mm

2. medium: 4-8mm

3. high: >8mm

Part 3: Relation of augmentation and defect region

i: internal, inside the contour

e: external, outside the ridge contour

DEFECT CLASSIFICATION: According to Seibert (1983), alveolar crest defects

Class 1 defects: when the bone deficiency is predominantly present in the horizontal dimension

Class 2 defects: when the bone deficiency is predominantly present in the vertical dimension

Class 3 defects: when the bone deficiency is affecting both the vertical and horizontal dimensions.

HÄMMERLE AND JUNG CLASSIFICATION OF CREST DEFECTS IN FRESH EXTRACTION SOCKETS

Class I: extraction socket having intact bone walls after tooth extraction

Class II: extraction socket having marginal dehiscence fenestration of the buccal bone wall after tooth extraction

Class III: extraction socket having large dehiscence of the buccal bone wall after tooth extraction.

Bone Augmentation Therapies

Melcher (1976), He developed the concept of using barrier membranes to "guide" the biologic process of wound healing. Previous experimental studies demonstrated that the soft tissue invasion of the defect can be excluded by means of a barrier membrane[6], thereby allowing the cells with regenerative potential to migrate to the site (which was derived from the periodontal ligament or bone marrow) and promoted periodontal regeneration (Nyman et al. 1982).

Regenerative Materials are barrier membrane, Bone grafts and Bone substitutes.[15]

Barrier membranes Purpose is to prohibit the penetration of cells, primarily epithelial, through its structure. There are five criteria that has been considered to be important in the design of barrier membranes used for GBR 1. biocompatibility, 2. cell occlusion properties, 3. integration by the host tissue, 4. Space making capacity. 5. clinical manageability

Types of barrier membranes: Barrier membranes have been derived based on two principal varieties:

1- Non resorbable as titanium,e-PTFE(expanded PTFE) non-degradable barrier membranes require a second surgical intervention to remove them.The material of choice usually depends on the amount of bone regeneration needed, mainly in the vertical dimension. e-PTFE barrier membranes have demonstrated more favorable results when compared with resorbable devices, mainly due to their betterspace-making capacity, longer barrier function, lack of a resorbption process that may affect bone formation (Hämmerle& Jung 2003).

dPTFE(High-density polytetrafluoroethylene), Textured dPTFE(Cytoplast)[9]

2- Resorbable membrane:

A.Synthetic: 1.polylactide,2.polyglycolic acid,3.vicryl mesh,Cargile membrane

Autogenous

Autogenous intraoral (obtained from chin, mandibular ramus, maxillary tuberosity)

Autogenous Extraoral (obtained from tibia, anterior ilium, posterior ilium, cranial bone)

Block and particulate

Xenograft

Bovine

Porcine

Block and particulate

Allograft

Demineralised freeze-Dried bone

Freeze dried bone allograft

Block and particulate

Alloplast

Bioactive glass

Calcium phosphate

Calcium sulphate

Calcium carbonate

Synthetic polymers

Particulate HA

Biological agents

PRF, PRP

Growth factors

BMP

Comparison of different grafting materials:

Xenografts and allografts resulted in least loss of socket dimensions

Alloplast has the larger amount of vital bone and the less amount of remnant graft material and remnant connective tissue.

Ridge augmentation procedures:

1) Ridge preservation
2) Bone regeneration in fresh extraction sockets
3) Horizontal bone augmentation
4) Ridge splitting/expansion
5) Vertical ridge augmentation

CONCLUSION

ridge augmentation procedures and using bone grafts for the procedures have become increasingly predictable. The proper selection and application of the available techniques and biomaterials are key to determinants of implant survival/success rates.

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PRAYER BEADS-*ELEOCARPUS GANITRUS*(RUTHRACHAM)AND ITS MEDICINAL IMPORTANCE-A REVIEW

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ABSTRACT

Eleocarpus ganitrus is one of the most dominant medicinal plants known as Ruthracham. This highly spiritual bead is a treasure trove of many health benefits.It is frequently cited as "Prayer beads" and it is contemplated as the most sacred bead.It is habitually accessible and cultivated in the Himalayan regions.It is renowned for its salubrious usage. In the Siddha system of medicine, which is the oldest system of medicine, "Ruthracham" is extensively used for numerous ailments together with stress, depression, anxiety, epilepsy, migraine, bronchial asthma, hypertension, arthritis, nerve pain, palpitations, ADHD (attention deficit hyperactive disorder) and liver diseases. It is generally acknowledged for its anti-inflammatory, immune stimulatory, anti-hypertensive, anti-asthmatic, anti-microbial, anxiolytic, anti-diabetic, nephroprotective, and anti-oxidant activities. This article allocates all the imperative information concerning its long established literature and research upgrades on ethnopharmacology, phytoconstituents, traditional properties, and pharmacological importance.

KEYWORDS: *Eleocarpus ganitrus (Ruthracham), Ethnopharmacology, Traditional Properties, Phytoconstituents, pharmacological importance.*

INTRODUCTION

Although medicinal plants are unequivocally the preeminent source of drugs in human history, the inadequacy of authentic literature to brace the pharmacological potential of the plants has outstandingly reduced their acceptability among people. *E. ganitrus* (Synonyms: E. sphaericus) is a large, eternal tree, universally cited as the Rudraksha tree in India. It is a sacred tree and is relished with exceptional acclaim and devotion in the Hindu community. In Hindi it is known as "Rudraki" and in Sanskrit it is known as "Rudraksha". The word "Rudraksha" literally means "Tears of Shiva". In our ancient Siddha system of medicine, it is extensively used in treating stress, anxiety, depression, palpitation, nerve pain, epilepsy, migraines, lack of concentration, asthma, hypertension, arthritis, and liver diseases. In consonance with our Siddha medicinal system, wearing Rudraksha beads generates positivity and soothes numerous ailments, but most vitally, it has age-defying effects. Besides the fact that Rudraksha beads are traditionally utilized in India, they are used widely in other Asian countries too. This species' name is synonymous with E. sphaericus, whose fruits or seeds are used in Siddha for psycho-somatic diseases [5] [6]. The main focus of this article is to encapsulate the morphology, distribution, phytochemistry, and medicinal properties of *E. ganitrus* for nurturing the scientific investigation into the evolution of effective therapeutic compounds [1].

Taxonomy

Kingdom: Plantae
Division:Magnoliophyta
Class: Magnoliopsida
Order: Oxalidales
Family: Elaeocarpaceae,
Genus: Elaeocarpus
Species: *E. ganitrus* Roxb.

MORPHOLOGY

*E. ganitrusis*a huge eternal tree with large leaves. Its growth ranges from 50-200 feet in height. On the sun-facing side, the leaves are large and shining and are dull and leathery on the dorsal side. Flowers will start appearing in the month of April to May and are yellow or white in colour with fringed petals. Fruits will start appearing in the month of June and will ripen in the month of October. The ripe fruit is so fleshy and has a seed with a blue shell. The inner part of the bead lying in the seed is known as "Rudraksha".

Geographical distribution

E. ganitrus is contemporarily grown on plantations and hillock's for its mercantile and pious values in Nepal, India, Bangladesh, Bhutan, Maldives, Pakistan, Indonesia, New Guinea, Australia, Guam, Hawaii, and Sri Lanka. Capitalistically, there are three varieties of "Rudraksha" that are up for grabs: Nepalese, Indonesian, and Indian. In India, *E. ganitrus* is dispersed in the Eastern Himalayas in Arunachal Pradesh, Bihar, Madhya Pradesh, and the Konkan Ghats. In Asia, the genus Eleocarpus consists of 120 species, out of which 25 have been delineated from India.

Ethno pharmacology

E. ganitrus is a crucial medicinal plant with several medicinal uses in the traditional medicine system. It is used to cure many health problems in several parts of the world. Leaves and seeds are known for their various medicinal properties and are customarily used to heal stress, anxiety, depression, palpitation, nerve pain, epilepsy, migraines, lack of concentration, asthma, hypertension, arthritis, and liver diseases [3] [4].

ETYMOLOGICAL & MACROSCOPICALDESCRIPTION

The leaves are shorn, ovate, mucronate, lobed, and sporadically serrated, cuspated, or aciculated. The flowers are in opaque racemes, yellow or white in colour and mainly from stipules of leaves which have fallen. They come into sight from April to May. The tiny fruits are blue or violet in colour, circular or ovoid in shape, and they taste acidulous. The stone pit is solid, sphere-shaped, and it is reddish brown in colour.

COMPOSITION OF "RUDRAKSHA"

The rudraksha beads encompass oxygen, hydrogen, carbon, nitrogen and other micro elements in integrated form. The gaseous elements in Rudraksha beads and their configuration in percentage are specified underneath.

Oxygen - 30.53%
Hydrogen - 17.897%
Nitrogen - 0.95%
Carbon - 50.031%

LEAF EXAMINATION – MACROSCOPICAL

APEX	**BASE**	**COLOUR**	**MARGIN**	**SHAPE**	**SIZE**
Stiff	**uniform**	**Gleaming green**	**ripple**	**ovoid**	**2 inch breath 5-6 inch length**

Chemical Constitutions of *Elaeocarpus ganitrus*

Dynamic constituents in Rudraksha are elaeocarpidine, elaeocarpine, rudrakine, flavonoids, and quercetin (Johns SR., et al., 1971; Ray AB., et al., 1979; Chand L., et al., 1977). Excerpts exhibit the presence of phytosterols, fat, alkaloids, flavonoids, carbohydrates, ethanol, proteins and tannins, acid and ellagic acid. It holds 50.03% C, 0.95% N, 17.89% H, and 30.53% O2. Phytonutrient inspection with different extractions displays distinct chemicals. Extraction with petroleum ether shows the existence of fatty oil fats and phytosterols. Extraction with ethanol ether appearance displays the existence of alkaloids,

flavonoids, carbohydrates, proteins, tannins. Extraction with water displays the existence of carbohydrates, proteins, and tannins. Elaeocarpus sphaericus capitulates mainly indolizidine alkaloids, including isoelaeocarpine, epiisoelaeocarpiline, epielaeocarpiline, alloelaeocarpiline, and pseudoepiisoelaeocarpilline.

Traditional Properties

The conventional use of the unripe and ripped fruit of *Elaeocarpus sphaericus* manifests as follows:

- *Elaeocarpus sphaericus* is revered as the sacred bead that bestows constructive sequels on trepidation, stress, inadequacy of concentration, epilepsy, and other ailments.Those who are enduring stress are cited as saying that holding a larger size of five muki
- *Elaeocarpus sphaericus* in their right palm tightly for ten minutes would bring tranquilly to their mind. It also assists in regaining self-possession and solidity in individuals.
- *Elaeocarpus sphaericus* is an excellent pregnancy bead. Wearing Garbh Gauri Elaeocarpus sphaericus helps women who are vulnerable to miscarriages and aids in natural conception.
- *Elaeocarpus sphaericus* is beneficial for both delirium and somnolence in women.
- *Elaeocarpus sphaericus* is additionally potent for children who experience frequent fevers. Those children should put up three-faced Elaeocarpus sphaericus.
- To treat small pox, pulverise an equal amount of black pepper and *Elaeocarpus sphaericus* and take it with water.
- *Elaeocarpus sphaericus* is even more effective in psychiatric disorders. Milk boiled with four-faced Rudraksha seeds is a sweet medicine for cognitive illness. This also helps in intensifying memory.
- *Elaeocarpus sphaericus* also has anti-aging properties [5].

Pharmacological Activities

Antioxidant properties

Ethanolic extract of leaves of *Elaeocarpus ganitrus* was scrutinized for their total antioxidant capacity, reducing power, hydroxyl radical scavenging activities. The extract at 500 µg/ml manifested utmost Iron chelating activity (76.70%). Nevertheless, the extract exhibited only modest hydroxyl radical scavenging activity (13.43%). Total antioxidant capacity was 24.18 mg ascorbic acid equivalents at 500?g/ml extract concentration. There was a positive correlation between the total phenolic content and antioxidant capacity, R^2 = 0.8547, whereas the correlation between the total flavonoids and antioxidant capacity was determined to be R^2=0.8413. The results suggest that phenolics and flavonoids in the leaves provide substantial antioxidant activity. [15]

Antihypertensive activity

It is experimentally proven that extracts of *E. ganitrus* Roxb can be utilized in stress-induced hypertension. Whereas the ethanolic extract of E.garcinia failed to bring down nicotine, it persuaded hypertension. An aqueous extract of *E. ganitrus* seeds evinced antihypertensive activity in renal artery-clogged hypertensive rats. Treatment of animals with aqueous extract of *E. ganitrus* for 6 weeks outstandingly depleted the upraised blood pressure of the animals [9].

Antimicrobial activity

It is cited that certain solvents should be used to extract active phytochemicals from the epicarp and endocarp of *E. ganitrus*, which could be a prospective source of dynamic phyto-constituents such as alkaloids, phenols, flavonoids, tannin, glycosides, and coumarins. It is long established that *E. ganitrus* seeds have antimicrobic activities and would play a vital part in the pharmaceutical industry. Numerous extracts of E. sphaericus were assessed for their antimicrobic activity in opposition to 28 gram-positive and gram-negative bacteria. Petroleum ether, benzene, chloroform, acetone and ethanol extract formulated from dried fruits of E. sphaericus showed broad spectrum antimicrobial activity with regard to the spread of microorganisms. Amidst all the extracts, the aqueous extract revealed

astounding antimicrobial activity greater than that of other extracts [10]. Petroleum ether, chloroform, ethanol and aqueous extracts formulated from E. ganitrus seeds demonstrated potential broad spectrum antifungal activity against Asperagillus niger, Candidum geotrichum, Candida albicans, C. glabrata and C. tropicalis [11].

Immunostimulatory activity

E.*Ganitrus*seeds appear to evince remarkable immunostimulatory activity and it impacts both non-specific and specific arms of the immune system. Alkaloidal fragments of *E. ganitrus* seeds have been shown in vitro and in vivo to brace immune mediators from peritoneal exudates cells and intensify immune cells in in vitro and in vivo models [12].

Anti-inflammatory activity

Copious solvent excerpts (Petroleum ether, benzene, chloroform, acetone and ethanol extracts) from E. sphaericus fruits evinced noteworthy anti-inflammatory action in opposition to both acute and sub-acute models in mice. Over and above, all excerpts secured guinea-pigs against bronchospasm, persuaded by histamine and acetylcholine aerosols [13]. Petroleum ether, chloroform, methanol, and aqueous excerpts of E. sphaericus leaves displayed outstanding analgesic and anti-inflammatory potential in carrageenan-induced paw oedema (inflammation) in rats and tail flick tests in mice. Methanol and aqueous extracts displayed remarkable dose-dependent anti-inflammatory activity in the course of the experiment [14].

Antioxidant activity

E.ganitrus leaf excerpts were delineated to evince noteworthy antioxidant activity in numerous in vitro methods. Ethanolic excerpt of *E. ganitrus* leaves against the spread of free radicals exhibited remarkable total antioxidant activity, reducing power potential, metal chelating activity, and ABTS scavenging activity. Furthermore, a positive correlation between total phenolic content and antioxidant capacity, as well as total flavonoid content and antioxidant activity, was declared.[15] Formalized paraphrase

Antiasthmatic activity

E.sphaericusfruits were found to manifest antiasthmatic activity *in vivo.* Numeroussolvent extracts (petroleum ether, benzene, chloroform, acetone and ethanol extracts) of *E.*
E. sphaericus fruits evidenced mast-cell sustaining activity, validating the potency of *E. sphaericus* against bronchial asthma [16].

Anxiolytic activity

E. sphaericus fruits were found to have anxiolytic activity in a Swiss albino mouse model. Methanolic extract of E. sphaericus fruits (200 mg/kg bw) expressed exceptional anxiolytic effects in a mouse model. It has been affirmed that the anxiolytic activity of the extract may be due to its high flavonoid content [17].

Antidiabetic activity

Aqueous excerpts of *E. ganitrus* seeds exhibited an appreciable hypoglycemic sequel after 2 hours of treatment in streptozotocin-persuaded diabetic rats. Extract treatment remarkably decreased the blood glucose level in a dose-dependent manner during the 30 days of treatment [18]. Chitosan-based extract and aqueous extract prepared from *E. ganitrus* leaves were assessed for their antidiabetic potential in Arrayed Albino rats. Amidst both extracts, the chitosan-based extract of *E. ganitrus* leaves exemplified more consequential antidiabetic activity than that of the aqueous extract [19].

Nephroprotective activity

Ethanolic excerpts of *E. ganitrus* seeds were established to exhibit a striking nephroprotective effect. In male Wistar rats, GM induces nephrotoxicity. The extract treatment of diseased mice exceptionally minimised the upraised levels of serum creatinine, blood urea nitrogen, uric acid, and albuminuria with a substantial increase in serum albumin and urine creatinine [20].

Toxicology

The majority of the plants are predominantly contemplated as nontoxic, but it's vital to substantiate their level of toxicity before therapeutic use. Aqueous extract of *E. ganitrus* seed was established to be nontoxic up to a dose of 5.0 g/kg weight (highest dose checked) in the Swiss albino mouse model [21]. Literature corroboration is not available to report the future exposure of the extract. Another study, aqueous extract of *E. ganitrus* leaves did not display any hemolytic activity against human erythrocytes up to 1000 µg/ml concentration; henceforth it might potentially be scrutinized secure with regard to human erythrocytes [22].

AN ANTIDEPRESSANT ACTIVITY

Ethanol and petroleum ether excerpts of Elaeocarpus spharicus's fruit lessened the swim stress stolidity in mice, designating some scale of antidepressant activity. Pharmacological investigations with the 90% ethanolic extract of the fruits of *E. ganitrus* revealed the phenomenon of a serious central nervous system depressant effect, signaled by quintessential behavioral actions, morphine analgesia, anticonvulsant, potentiating of hexobarbitone hypnosis, and anti-amphetamine effects. In this study, the extract emphasised a cardio stimulant and a depressor, part of these being arbitrated by way of beta adreno receptor stimulation and in part through direct musculotropic effects. The present unearthings prop up the implementation of *Elaeocarpus ganitrus* as an antidepressant endorsed in the conventional system of medicine and open an avenue to develop a proxy antidepressant agent from distinguished herbal remedies [23].

ANTI-ANXIETY ACTIVITY

Ethanolic fruit excerpts of Euganitrus procure antidepressant effects. *E. ganitrus* was appraised for antianxiety activity in mice utilizing an elevated plus maze model. The chloroform and ethanol extracts were found to be effective against anxiety in low doses, but a dose of 200 mg/kg of ethanol extractive was equal to diazepam, as evidenced by statistical equivalence between the results of this dose and those of diazepam.Chloroform extractives are also effective in the lowest doses, but best at a dose of 400 mg/kg. Anxiolytic effect of *Elaeocarpus sphaericus* fruit extract (methanolic) in Swiss albino mice

Conclusion

In the wake of its remarkable medicinal perquisites, rudraksha has made a nook in medicine and religion. Good luck. It ushers in added exuberance to our system, contributing to health and harmony by eliminating negativity. Ethno medical and scientific reports about the medicinal properties of *E. ganitrus* describe it as a treasured plant and initiate it as a candidate for enduring drug development.

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TMJ ANKYLOSIS- A REVIEW

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ABSTRACT

The TMJ is a diarthrodial, ginglymus ,synovial joint that is capable of both rotational and translator movement.it is formed by the articulation of the glenoid fossa of the temporal bone and the head of the condyle. TMJ ankylosis refers to a partial or complete inability to open the mouth which result in functional or growth deformities if the mandible. being responsible for the various movements of the jaw ,any pain or restriction of function can cause immense distress to the patient

Key words: *-temporomandibular joint, tmj disorder, ankylosis, trauma*

INTRODUCTION

TMJ Ankylosis is the pathological fusion of parts of joints resulting in restricted movement across the joint[1]. It is an arthrogenic disorder of the tmj, refers to restricted mandibular movement with deviation to affected side on opening of the mouth. Ankylosis may range from simple fibrous restriction of jaw movements to a bone formation within the joint restricting movement completely

ETIOLOGY

TRAUMA: -Delivery, Intracapsular fractures, Congenital

INFECTION AND INFLAMMATION: -

- Otitis media
- Parotitis
- Mastoiditis

SYSTEMIC CAUSE: -

- Scarlet fever
- Meningitis
- Small pox

PATHOPHYSIOLOGY: -

Intracapsular fracture of bone

⬇

Bleeding within joint cavity

⬇

Bone fragments with very high osteogenic potential

⬇

Organisation of haematoma within joint

⬇

Conversion to fibrous tissue

⬇

Subsequently to bone

CLASSIFICATION OF TMJ: -

Based on the type of tissue causing the ankylosis; -

- Fibrous ankylosis
- Bony ankylosis

Based on the side involved: -

- Unilateral
- Bilateral

Based on the severity of the ankylosis: -

- Partial
- Complete

Based on the type of etiology for trismus: -

- Pseudo ankylosis
- True ankylosis

KAZANJIAN CLASSIFICATION

- Intra articular
- Extra articular

SAWHNEYS GRADING OF ANKYLOSIS

Type 1: -flattening or deformity of the condyle with little joint space seen on the radiograph. Extensive fibrous adhesion seen during operation

Type 2: -bony fusion of the outer edges of the articular surface with no fusion in the deeper areas of the joints

Type 3: - A bridge of bone is seen between ramus of the mandible and zygomatic arch

Type 4: - Entire joint is replaced by a mass of bone

DIAGNOSIS OF TMJ ANKYLOSIS

History: -

Accurate history is important to differentiate the condition of pseudo ankylosis and true ankylosis. History of trauma either directly to the joint or indirectly to the chin. Duration of trismus should be asked[2].Extracapsular causes such as an untreated zygomatic arch fracture should be ruled out. History of ear infection in childhood. History of forceps delivery of the child

Clinical examination: -

- Restricted mouth opening patients will complain of difficulty in mastication
- Protrusive movement are absent on involved side
- Partial mobility or complete immobility of the condyle is noticed on palpation

UNILATERAL ANKYLOSIS

1. Facial asymmetry
2. Affected side appears normal
3. Opposite side appears flat
4. Chin deviated to ankylosed side
5. Deep ante gonial notch on ankylosed side
6. Reduced condylar movement on affected side
7. Class 2 malocclusion on the affected side
8. Posterior cross bite
9. Poor oral hygiene

BILATERAL ANKYLOSIS

Bird face

1. Trismus
2. Class 2 malocclusion
3. Deep ante gonial notch
4. Poor oral hygiene
5. Crowding of teeth
6. Protrusion of upper anterior teeth
7. Anterior open bite
8. No condylar movement palpable

INVESTIGATION

Radiographic findings

Orthopantomography: - will show both the joints picture which can be compared in unilateral area

Lateral oblique view: -will give anteroposterior dimension of the condylar mass.Elongation of coronoid process can be seen[3]

Cephalometric radiograph: -is taken to evaluate the associated skeletal deformities

Posteroanterior radiograph: -will reveal the mediolateral extend of the bony mass.it will also highlight that asymmetry in unilateral cases

CT scan: - very helpful guide for surgery. Relation to the medial cranial fossa, the anteroposterior width, mediolateral depth can be assessed. Any presence of fractured condylar head on the medial aspect of ramus can be located

SEQUELAE OF AN UNTREATED ANKYLOSIS

- Facial deformity
- Speech difficulty due to decreased mouth opening, maloccluded teeth and tongue position
- Nutritional deficiency
- Respiratory distress
- Malnutrition
- Malocclusion
- Poor oral hygiene

MANAGEMENT OF T.M.J Ankylosis

Basically 3 types: -

- Condylotomy
- Gap arthroplasty
- Interpositional arthroplasty

APPROACHES TO TMJ: -

1. Preauricular incision
2. Postauricular incision
3. Hemi coronal
4. Submandibular incision
5. Post ramal
6. Endaual incision

CONDYLECTOMY: -

Advocated in case of fibrous ankylosis, where joint space is obliterated with deposition of fibrous bands but there is not much deformity of condylar head .Preauricular approach used commonly ,other include AI kayat Bramley, inverted hockey stick[4]

GAP ARTHROPLASTY: -

Section consists of two horizontal osteotomy cuts and removal of bony wedge for creation of a gap .NO substance is interposted between the two cut bony surface. Minimum gap of 1cm to prevent reankylosis

INTERPOSIIONAL ARTHROPLASTY: -

Involves creation of gap, but in addition a barrier is inserted between the cut bony surface to minimize risk of recurrence and to maintain vertical height of ramus

COMPLICATION DURING SURGERY: -

DURING ANESTHETIC: -

As the patient cannot open mouth, awake blind intubation has to be done where co-operation is required which is difficult to achieve sometime. Because of small mandible and altered position of larynx, intubation poses a problem. Aspiration of blood clot, tooth or foreign body during extubation. Danger of falling back of tongue and obstructing airway is always there after extubation[5]

DURING SURGERY: -

- Haemorrhage
- Damage to external auditory meatus
- Damage to zygomatic and temporal branch of facial nerve
- Damage to auriculotemporal nerve
- Damage to parotid gland
- Damage to glenoid fossa

DURING POST OPERATIVE FOLLOW-UP: -

- Infection
- Open bite
- Recurrence of ankylosis

RECURRENCE OF ANKYLOSIS

Several factors said to be responsible

Inadequate gap created between fragments

Fracture of costochondral graft

Loosening of costochondral graft due to inadequate fixation to ramus

Inadequate postoperative physiotherapy

Inadequate coverage of glenoid fossa surface

Higher osteogenic potential and periosteal osteogenic power maybe responsible for high rate of recurrence in children

DISCUSSION

Prior to 1951 TMJ ankylosis was considered to be incurable. Brisment force was tried without success. Esmarch suggested wedge resection of the body of mandible. condylectomy in early cases. Gap arthroplasty led to recurrence. Osteoarthrotomy improved the prognosis. Excision of callus & joint reconstruction become the treatment of choice.

Ethical clearance – Not needed as it is a review article

Source of funding- Nil

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ALVEOLOPLASTY AS A SECONDARY TREATMENT FOR CLEFT PALATE: A REVIEW ARTICLE

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ABSTRACT

Cleft palate is the most prevalent congenital defect. It is more prevalent in 3rd world countries. The internationally accepted steps of treatment are: 3 to 6 months – surgery to repair a cleft lip if present, 6 to 12 months – surgery to repair a cleft palate, 18 months – speech assessment, 3 years – speech assessment, 5 years – speech assessment, 7 to 12 years – bone graft to repair a cleft in the alveolar bone,12 to 15 years – orthodontic treatment and monitoring jaw growth. Secondary alveoloplasty serves to enable the patient to have a better quality of life through the augmentation of the alveolar ridge and hence open up the doorway to various modes of dental rehabilitation for proper nutrition and development. The most commonly used method is Boyne's technique. The use of growth factors has increased the success rate of the contemporary method. With the advent of virtual planning and surgery, the quality of care has recently skyrocketed, yet is still out of reach of most patients due to its high cost. In this article, we shall delve deeper into the topic of secondary alveoloplasty.

KEYWORDS: *ALVEOLOPLASTY IN CLEFT PALATE, SECONDARY BONE AUGMENTATION IN CLEFT CASES*

INTRODUCTION

Among all of the congenital deformities, cleft palate is the most common. Average prevalence rate is 1:1000, and has several grades of severity. It is generally characterized by a discontinuity in the plate which may or may not extend to the premaxilla and alveolar ridge. Over the years various theories have been proposed to explain the phenomenon, namely (1) alteration in intrinsic palatal shelf force, (2) Failure of tongue to drop down, (3) Non fusion of shelves, (4) Rupture of cyst formed at site of fusion. The condition has been deemed to be multifactorial in etiology, which are, (1) Genetic, (2) Nutritional disturbances during development, (3) Stress during development, (4) Ischemia, (5) macroglossia, (6) Environmental factors like infection, use of teratogenic drugs, antibiotics, radiation, hormonal disturbances, (7) consumption of alcohol and tobacco by the mother during development. Classifications:

1. VEAU' CLASSIFICATION:

Group I- Involving only the soft palate
Group II- Cleft on soft and hard palate extending only till incisive foramen
Group III- Complete unilateral cleft involving the soft palate, hard palate, alveolar ridge and lip on one side
Group IV- Complete cleft on soft palate, hard palate, alveolar bone and lips bilaterally

2. KERNAHAN AND STARK'S CLASSIFICATION:

GROUP I (primary palate only)- (a) unilateral (b) bilateral (c) total (d) subtotal
GROUP II (secondary palate only)- (a) total (b) subtotal (c) submucous
GROUP III (both primary and secondary palate)- (a) unilateral (total and subtotal) (b) median (total and subtotal)

Steps in treatment plan for cleft palate cases:

Birth to 6 weeks – feeding assistance (use of obturators), support for parents, hearing tests andpediatric assessment

- 3 to 6 months – surgery to repair a cleft lip if present
- 6 to 12 months – surgery to repair a cleft palate
- 18 months – speech assessment
- 3 years – speech assessment
- 5 years – speech assessment
- 7 to 12 years – bone graft to repair a cleft in the alveolar bone
- 12 to 15 years – orthodontic treatment and monitoring jaw growth

The word ALVEOLOPLASTY means to surgically mould the size or/and shape of the alveolar process. Historically alveoloplasty has been known and used for more than 170 years. A. T. Wilard of Chelsea, Massachusetts, in 1853 was the 1st known surgeon to reduce alveolar process in order to accomplish complete approximation ofsoft tissues over the ridge. Bone is a living tissue where osteoclastic as well as osteoblastic activities takes place, so the surgeon must start with the maxim that bone is precious and must not be wasted, therefore, its conservation is desired. FollowingWolff's law of bone adaptation, alveolar bone remodels itself in response toeach new situation of pressure. It will heal after dental extractions, and itwill usually attempt to adapt itself to the general configuration of the rest ofthe alveolar arch, Alveolar cortical bone will re-form in approximately 3months, more or less[1]. Thus, it is an irreplaceable tool in the arsenal of surgeon in cleft palate cases involving alveolar bone.

DISCUSSION

The protocol for treatment of cleft palate cases which is widely accepted is that, 7-11 years of age is ideal for correction of maxillary bone defect. Boyne et al were the 1st to consider alveoloplasty as a viable secondary corrective surgery in cleft cases. Alveoloplasy performed before 2 years of age is called as primary alveoloplasty. After that it is known as secondary alveoloplasty. It has been observed that alveoloplasty in cleft palate case has had more success and lesser adverse outcomes when performed befor the eruption of the permanent canine. If done after eruption of the permanent canine, the incidence of adverse outcomes has been much higher. The perfect timing for an alveolar bone graft in a cleft palate case has been determined to be in the mixed dentition stage when the root of the canine is between ¼th to ½ of being complete, which is generally around 7-9 years of age. Reconstruction of the bone is done with autologous bone graft and the objective ofthe procedure is to close the oro-nasal communication, allow canine eruption, give adequate bone support to adjacent teeth,facilitate orthodontic treatment, contribute to stabilityand continuity to maxillary arch (avoiding collapse ofstructures previously expanded by orthodontics), givesupport to nasal ala and allow dental rehabilitation withosteointegrated dental implants when necessary.

BOYNE'S METHOD

This is the most common procedure followed. Alveoloplasty along with autologous bone grafting is done. Bone is usually harvested from the iliac crest or if a very large defect is to be closed, then from the ribs. Two separate surgical teams work simultaneously on the operating table, one performing the alveoloplasty and the other harvesting the graft and preparing it. Preoperatively the surgery is planned with the help of panoramic X-rays and CT scans. General anaesthesia is induced, intubation is done and the two teams simultaneously prepare the two surgical sites. Incisions are placed, mucoperiosteal flaps are reflected, alveoloplasty is done to remove irregular borders and to give a definite shape to the discontinuity in order to help ease the grafting procedure. The discontinuity is measured. Simultaneously the other team harvests the bone graft in he desired size and shape. The graft is then transferred to the host site and placed so as to considerably approximate the defect. The flaps on both sites are reapproximated and sutures are placed in a layered fashion. Generally, no drain may or may not be placed. This technique is a tried and tested approach and shows excellent results even after the initial graft resorption.

USING PRP

Boyne's method can be further augmented by the use of Platelet Rich Plasma (PRP), which is a highconcentration of autologous platelets in a smallvolume of autologous plasma[4]. About 10ml ofPRP is harvested on the day of surgery in the operating room, it is mixed with calcium chlorate and incubated for 3 mins at 37 degrees Celsius and the mixed with the autologous bone graft, triturated thoroughly to form a malleable mixture which is then placed in the defect and the flaps are closed. Here placing a drain is contraindicated in the host site to enable the growth factors to work. This technique shows far better results than Boyle's method in terms of healing and lesser bone resorption and faster remodelling.

USING OF SCAFFOLD TO TRANSFER STEM CELLS

The use of stem cells obtained by the autologous bonemarrow and appropriately treated in order to obtainosteoblasts is a possible alternative[3]. Adult stem cells havethe capacity to form many different tissue types.Technical advances have helped to identify multipotentialstem cells and their ability to regenerate tissues isbeing studied in transplantation models[3]. The idea is to place bone marrow stem cells at close proximity to the cleft through a scaffold place in the bone defect. This technique can be used as an alternative to autologous bone grafting, to prevent the morbidity and added complexity due to the procedure. A suitable scaffold of pre formed shape is used which needs to have both osseoinductive as well as osseoconductive properties in order to enable the autologous stem cells to form complete bone and approximate the bone defect. The scaffolds commercially available maybe be natural or synthetic in origin and sometimes are devoid of osseoinductive property. Hence, they are mixed with PRP or Bone Morphogenic Protein (BMP) contained in a collagen matrix. Generally, about 20-30 mL of bone marrow is harvested from the iliac crest under local Anesthesia or conscious sedation. In this method hyalinization can be observed as early as 8 weeks and then proceeds onto mineralization gradually. This technique shows comparable degree of results to autologous bone grafting and hence is a viable alternative.

3D VIRTUAL PLANNING AND 3D PRINTING

With the advent of virtual surgery planning software, surgeons have added another very important tool in their arsenal. This enables the surgeon perform precise surgery virtually before the actual surgery and thus plan accordingly so as to reduce operating time, inaccuracies and complications. Cone beam CT scans are used to construct an exact replica of the maxillofacial skeleton, on which the surgeon can apply any approach or technique to see the outcome in real time. Another boon of technology has come in the form of 3D printing, where the 3D models already generated are used to create customized patient specific scaffolds. And in future it will be possible to 3D print actual live tissues as well which can then be directly placed into the bone defect. Yet the one disadvantage of this procedure is the lack of cost effectiveness and the fact that Cleft Palate cases are more prevalent in 3rd world countries where the average citizen has poor financial status.

CONCLUSION

The contemporary method shows excellent results but can be further augmented and improved with the use of growth factors. When the harvesting of autologous bone is not a viable option, then, the use of stem cells and scaffolds to naturally grow bone in the defect is a viable option, but is not very cost effective. 3D planning and 3D printing although an excellent mode of treatment, is not a viable option in the context of poorer countries due to its high cost. At the end enabling the patient to have a chance at a normal life and proper rehabilitation is the goal of alveoloplasty in cleft palate patients and the financial angle is a very important factor as well.

Ethical clearance – Not required since it is a review article

Conflict of interest – nil

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TOXICITY PROFILE OF HERBO MARINE SIDDHA DRUG SANGU PARPAM

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ABSTRACT

The present study was carried out to investigate the toxicity profile of Herbo marine Siddha formulation Sangu Parpam. Three types of purification was done on three samples of raw sangu and sangu Parpam was prepared from each purified sample and named as SP I,II,III. Acute and Sub-acute toxicity study was conducted as per the OECD guidelines 423 and 407 respectively. Acute toxicity study revealed Sangu Parpam I, II, III did not produce drug-related toxicity. The maximum tolerable dose obtained from the acute toxicity study is about 2000mg/kg b.wt. Sub acute toxicity study revealed Sangu Parpam I, II, III can be considered as safe and did not produce any toxic effects over a period of 28 days. Histopathological study shows normal architecture of oragans. Thus the Sangu Parpam I, II, III are safe in prescribed dose level as per the literature.

Key Words: *Siddha, Sangu Parpam, OECD, Purification, Herbo marine drug*

INTRODUCTION

The Siddha Medical System is based upon the teachings of the Siddhas. A great deal of the Siddha Medical System comes to us from the selfless work of untiring souls who preferred obscurity and austerity. Those who attained or achieved the above-mentioned powers were known as Siddhas1. It is a one-of-a-kind system that has existed among the Tamil people of South India for more than five thousand years, serving humanity in combating diseases and maintaining physical, mental, social, and spiritual health2.Unlike contemporary medicine, the Siddha system uses more than one ingredient for preparing medicines because of their synergistic activity and lower toxicity. These drugs possess increased bioavailability as the mineral drugs are treated with herbal juices, which leads to a reduction in particle size up to the nano level. As a result, even the smallest doses provide increased potency.

In this study, acute and sub-acute toxicity studies were done on *Sangu Parpam*, a herbo-mineral Siddha drug that is extensively used by traditional medicine practitioners. Three various types of purifications were done on raw sangu and from each purified sangu, Sangu parpam was prepared. The same preparation was followed to prepare the Sangu parpam. It has high therapeutic value in treating diseases like Peptic Ulcer, Gastrointestinal Disorders, Cough, Piles, etc.

MATERIALS AND METHODS

Preparation of Sangu Parpam

Purification of Sangu [5]

Process I (Spu I) [5]

35g of Sangu (1 palam) was soaked in 175g of Juice of Ilaikkalli (Common Milk Hedge – *Euphorbia ligularia*) and let to dry in sunlight from morning to evening. This process was repeated for another 3 times with fresh juice.

Process II (Spu II)[5]

Sangu was processed in thaalithal method (Heating process**)** by covering it with Karchunnam (limestone).

Process III (Spu III)[5]

Equal parts of Karchunnam (limestone) and Uvarmann (Alkaline earth) was mixed up with 8 parts of water and the clarified water was collected. Sangu was processed by heating with this clarified water. After heating, Sangu was washed with water and dried.

Preparation process [6]

100g of purified Sangu from each purification process (Spu I,II,III) was covered up by ground paste of Uthamani (*Pergularia*

damea) and kept in the mud lid and closed by another mud lid. Cotton ribbon soaked in wet clay was winded over the rims of both mud lids and let to dry in sun light for 8 hours. Then this set up was subjected to Ganapudam. (100 cow dung cakes were used). After cooling the set up was taken out and the calcinated Sangu was taken out ground well and stored in an airtight container. Sangu Parpam prepared from above mentioned purified process (Spu I, II, III) were named as SP I, II, III respectively.

Acute toxicity study of SP I, II, III

The complete protocol of the animal experiments has been approved by the Institutional Animal Ethics committee, National Institute of Siddha, Chennai. The IAEC approval number is **1248/AC/09/CPCSEA -9/Dec 2013/8.** Acute toxicity study of Sangu Parpam was evaluated in rats as per the Method - OECD 423 - Acute toxic class method[7].The animals were fasted overnight with water *ad libitum.* The Sangu Parpam I, II, III was administered in four different dose levels i.e. 50mg, 300mg, 1000mg & 2000mg/kg b.wt. as OECD guidelines insist the stepwise administration first 50mg/kg b.w of Sangu parpam I, II, III was given as a single oral dose. As no death was noticed, 300mg was administered. Likewise, 1000mg and 2000mg/kg b.wt was administered from group II to Group 13. Group I was served as control which received vehicle i.e. ghee (2ml/200gm). After drug administration, all animals were observed for 14 days. Observations were made and recorded systematically and continuously observed as per the guidelines after substance administration. On day 15, the overnight fasted animals (water allowed *ad libitum*) were sacrificed and examined for gross pathological changes in the major internal organs. All the animals were observed at least two times a day to record abnormal behavior. In clinical signs of toxicity, they should be observed daily for 14 days.

Sub-Acute Toxicity Study Of SP I, II, III 28 days Repeated Dose Oral Toxicity

Study were conducted as per the Method - OECD 407- Sub-Acute Toxicity study (Repeated Dose 28-Day Oral Toxicity Study in Rodents)[8]The IAEC approval number is **1248/AC/09/CPCSEA -9/Dec 2013/8.** Animals were divided into 4 groups and each group contains 5 animals per sex. The control animals were administered ghee 2ml/200gm. Group II received SP I, Group III received SP II, Group IV received SP III with low dose of 100mg/kg b.w, middle dose of 200mg/kg b.w and high dose of 300mg/kg b.w respectively. Administration was by oral for 28 consecutive days. Experimental animals were kept under observations throughout the study for the following, clinical signs and mortality, body weight, food and water consumption, Haematological parameters and Biochemical parameters. All the animals were sacrificed on day 29. Necropsy of all animals were carried out and routine histopathological examination was done. The various organs collected were fixed in 10% neutral buffer formaline. They were dehydrated through a series of graded alcohol, fixed in paraffin, and routinely processed for histopathological assessment. The tissues cut into 4-5µm thick sections and stained with Haematoxylin-eosin. Tissue slides were examined and photographs were taken by using N-400ME photomicroscope (CELL-TECH diagnostics, Hamburg, Germany) in X40, X100 and X400 objectives.

The mean changes in body weight, daily food and water intake, organ weight relative to body weight, and biochemical and haematological parameters were statistically analyzed and significant differences within groups were calculated using the one-way ANOVA test followed by Dunnett's test to compare mean differences of control and test drug treated groups. The results were expressed as the mean ± SD. Statistical analysis was performed using the SPSS version 18.

RESULTS

TABLE I - PHYSICAL AND BEHAVIOURAL EXAMINATIONS FOR SP I, II, III

Group no.	Drug	Dose (mg/kg)	Observation signs	No. of animal affected.
I	Ghee	Control	Normal	0 of 3
II	SP I	50mg/kg	Normal	0 of 3
III	SP I	300mg/kg	Normal	0 of 3
IV	SP I	1000mg/kg	Normal	0 of 3
V	SP I	2000mg/kg	Normal	0 of 3
VI	SP II	50 mg/kg	Normal	0 of 3
VII	SP II	300 mg/kg	Normal	0 of 3
VIII	SP II	1000mg/kg	Normal	0 of 3
IX	SP II	2000mg/kg	Normal	0 of 3
X	SP III	50 mg/kg	Normal	0 of 3
XI	SP III	300mg/kg	Normal	0 of 3
XII	SP III	1000mg/kg	Normal	0 of 3
XIII	SP III	2000 mg/kg	Normal	0 of 3

TABLE II- EFFECT OF SP I, II, III ON BODY WEIGHT

GROUP	O day	7th day	14th day	21st day	28th day
CONTROL	125±2.76	133±3.09	143±2.47	152±2.86	161±2.044
S.P I-LOW DOSE	128±2.49	136±2.5	143±1.69	157±1.93	161±1.86
S.P I-MID DOSE	128±3.20	136±3.22	147±3.23	157±3.58	165±2.52
S.P I-HIGH DOSE	139±2.17	149±2.53	159±2.43	168±3.26	174±2.49
S.P II-LOW DOSE	146±3.79	153±5.05	145±5.69	147±3.93	158±2.61
S.P II-MID DOSE	145±2.02	148±2.5	149±2.82	155±5.09	164±3.24
S.P II-HIGH DOSE	147±4.23	142±3.14	151±2.80	146±2.79	159±4.35
S.P III-LOW DOSE	149±4.42	146±1.37	151±3.30	152±2.85	163±2.64
S.P III-MID DOSE	141±2.47	141± 3.41	153±5.43	153±4.55	163±3.08
S.P III-HIGH DOSE	149±2.66	136±2.90	158±4.0	161±1.71	158±3.45

Values are expressed as the mean ± S.D; Statistical significance (p)calculated by one way ANOVA followed by dunnett's $^{c}P<0.001$, $^{b}P<0.01$, $^{a}P<0.05$ calculated by comparing treated group with CONTROL group.

Table III –EFFECT OF SP I, II, III ON HAEMATOLOGICAL PARAMETERS

GROUP	WBC cells/mm^3	POLY MORPHS	RBC cells/lt	HB g/dl	PCV g/dl	LYMPHOCYTES cells /µlt	MONOCYTES cells /µlt	EOSINOPHILS cells /µlt	MCH Picogm/cell
CONTROL	10±0.79	7±1.53	4±1.14	15±0.58	48±2.41	84±1.52	3±0.63	6±1.15	26±0.59
S.P I-LOW DOSE	12±0.83	9±1.85	6±0.27	15±0.80	45±2.47	81±1.67	3±0.76	6±0.58	24±0.79
S.P I-MIDL DOSE	13±0.63	4±1.85	5±0.07	13±0.15	41±1.70	87±1.87	3±0.76	4±0.88	24±0.50
S.P I-HIGE DOSE	12±0.55*	5±0.33	5±0.22	15±0.56	45±1.06	84±0.73	4±0.56	5±0.88	28±0.32
S.P II-LOW DOSE	11±0.34	5±1.20	5±0.25	14±0.67	40±2.17	77±3.83	3±0.36	3±0.66	22±0.67
S.P II-MIDL DOSE	10±0.35	6±1.33	5±0.24	15±0.78	49±6.07	73±3.15	4±0.21	4±1.15	26±1.19
S.P II-HIGE DOSE	11±1.02	6±1.53	5±0.15	14±0.54	42±4.69	77±4.20	4±0.56	4±1.16	17±1.69
S.P III-LOW DOSE	10±0.35	7±0.67	6±0.20*	14±0.75	46±7.55	81±2.01	5±0.36	5±0.88	20±2.03
S.P III-MIDL DOSE	12±0.79	6±0.58	6±0.21*	14±0.52*	41±1.88	88±1.26	4±0.36	5±1.76	21±2.03
S.P III-HIGE DOSE	12±0.59	7±0.67	5±0.27	15±0.73	40±1.51	77±3.48	3±0.76	4±0.88	23±1.48

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

TABLE IV - EFFECT OF SP I, II, III ON ORGAN WEIGHT IN GMS

GROUP	BRAIN	HEART	LUNGS	LIVER	TESTIS
CONTROL	1.12±0.06	0.89±0.02	2.31±0.21	6.86±0.43	2.95±0.29
S.P I-LOW DOSE	1.20±0.12	0.89±0.03	1.01±0.10	5.04±0.45	2.39±0.23
S.P I-MIDL DOSE	1.65±0.09	0.82±0.03	1.32±0.16	5.53±0.59	2.00±0.19
S.P I-HIGE DOSE	1.49±0.23	1.0±0.05*	2.06±0.14	6.05±0.12	2.57±0.21
S.P II-LOW DOSE	1.52±0.23	1.08±0.08*	1.71±0.19	6.42±0.39	2.44±0.16
S.P II-MIDL DOSE	1.08±0.07	0.96±0.03	1.69±0.23	5.78±0.55	1.37±0.15
S.P II-HIGE DOSE	1.02±0.03	0.97±0.02	1.41±0.15	6.78±0.59	3.04±0.20
S.P III-LOW DOSE	1.29±0.10	0.92±0.04	1.46±0.20	6.43±0.69	3.27±0.19
S.P III-MIDL DOSE	1.31±0.04	0.87±0.02	1.548±0.2	6.42±0.19	2.79±0.47
S.P III-HIGE DOSE	1.38±0.09	0.94±0.05	2.06±0.10	6.43±0.93	3.25±0.15

Values are expressed as the mean ± S.D; control vs SP I High dose * P< 0.05 control vs SP II Low dose * P<0.05

TABLE V - EFFECT OF SP I, II, III ON ORGAN WEIGHT IN GMS

GROUP	KIDNEY (L)	KIDNEY (R) WEIGHT	UTERUS WEIGHT
CONTROL	1.05±0.09	1.04±0.04	0.49±0.02
S.P I-LOW DOSE	0.69±0.014	0.75±0.03	0.56±0.06
S.P I-MID DOSE	0.69±0.03	0.68±0.01	0.52±0.05
S.P I-HIGH DOSE	0.95±0.09	0.89±0.03	0.72±0.02
S.P II-LOW DOSE	0.82±0.08	0.81±0.08	0.51±0.04
S.P II-MID DOSE	0.98±0.06	0.94±0.03	0.48±0.06
S.P II-HIGH DOSE	0.81±0.08	0.79±0.08	0.54±0.12
S.P III-LOWDOSE	0.91±0.08	0.79±0.07	0.43±0.07
S.P III-MID DOSE	0.79±0.07	0.78±0.07	0.41±0.02
S.P III-HIGH DOSE	0.95±0.16	0.87±0.11	0.43±0.08

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

Table VI - EFFECT OF SP I, II, III ON BLOOD GLUCOSE LEVEL

GROUP	BLOOD GLUCOSE LEVEL mg/dl	
	Male	**Female**
CONTROL	78±3.12	79±4.02
S.P I-LOW DOSE	83±1.64	95±3.45
S.P I-MID DOSE	75±6.63	83±1.87
S.P I-HIGH DOSE	92±7.62	92±1.46
S.P II-LOW DOSE	92±5.93	79±4.10
S.P II-MID DOSE	91±8.10	93±2.95
S.P II-HIGH DOSE	90±4.10	78±4.93
S.P III-LOW DOSE	87±3.31	89±4.02
S.P III-MID DOSE	94±9.02	90±6.28
S.P III-HIGH DOSE	89±4.39	92±2.35

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

TABLE VII – EFFECT OF SP I, II, III ON LIPID PROFILE

GROUP	TOTAL CHOLESTEROL (mg/dl)	TRIGLYCERIDES (mg/dl)	HDL-CHOLESTEROL (mg/dl)
CONTROL	37±2.89	101±1.59	8±0.99
S.P I-LOW DOSE	38±1.99	98±2.09	8±1.48
S.P I-MID DOSE	37±3.97	96±10.89	8±0.93
S.P I-HIGH DOSE	39±2.54	96±9.59	8±2.60
S.P II-LOW DOSE	36±2.69	100±3.83	7±0.42
S.P II-MID DOSE	37±3.61	99±2.22	8±0.13
S.P II-HIGH DOSE	37±1.65	98±1.83	7±0.092
S.P III-LOW DOSE	36±6.38	97±2.56	7±0.24
S.P III-MID DOSE	37±3.29	101±3.65	8±0.40
S.P III-HIGH DOSE	38±1.52	99±3.31	8±0.15

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

TABLE VIII - EFFECT OF SP I, II, III ON SGOT, SGPT, ALP LEVELS

GROUP	SGOT (U/L)	SGPT(U/L)	ALP(U/L)	TOTAL BILIRUBIN (g/dl)
CONTROL	79±5.56	62±6.88	297±7.87	0.53±0.08
S.P I-LOW DOSE	81±17.53	61±5.99	301±13.18	0.75±0.13
S.P I-MID DOSE	77±4.79	56±9.44	267±17.32	0.64±0.03
S.P I-HIGH DOSE	80±0.83	59±1.56	254±2.30	0.69±0.06
S.P II-LOW DOSE	83±15.30	52±1.33	322±20.45	0.55±0.09
S.P II-MID DOSE	82±4.74	68±1.42	210±4.40	0.66±0.09
S.P II-HIGH DOSE	71±0.86	61±1.79	260±6.07	0.72±0.06
S.P III-LOW DOSE	85±30.4	64±3.27	314±24.85	0.64±0.03
S.P III-MID DOSE	69±1.08	59±5.36	288±1.47	0.75±0.02
S.P III-HIGH DOSE	63±4.56	6 2±3.08	268±10.08	0.73±0.08

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

TABLE IX - EFFECT OF SP I, II, III ON UREA, URIC ACID, CREATININE LEVELS

GROUP	UREA(mg/dl)	URIC ACID (mg/dl)	CREATININE (mg/dl)
CONTROL	33±4.42	1±0.08	0.33±0.01
S.P I-LOW DOSE	32±2.49	0.94±0.16	0.28±0.04
S.P I-MID DOSE	38±0.61	1±0.03	0.19±0.02
S.P I-HIGH DOSE	29±1.91	1±0.08	0.31±0.03
S.P II-LOW DOSE	45±4.75	8±4.39	0.36±0.04
S.P II-MID DOSE	37±1.52	0.89±0.10	0.42±0.05
S.P II-HIGH DOSE	38±5.79	0.61±0.11	0.34±0.04
S.P III-LOW DOSE	40±2.5	1±0.17	0.40±0.06
S.P III-MID DOSE	33±1.31	1±0.16	0.37±0.02
S.P III-HIGH DOSE	45±0.96	0.68±0.04	0.26±0.01

Values are expressed as the mean ± S.D; No significant difference between SP I, II, III when compared with control group

Acute toxicity study

The trial drugs sangu parpam I, II, III were tested for their acute toxicity study as a step wise procedure. The different doses did not produce any mortality and morbidity throughout the study period. The animals were healthy and they had gained weight throughout the study period. There was no body weight changes noted. No animals in the groups showed significant variation in food and water intake. After the completion period, there was no grass pathological change noted in all the group of animals. The animals did not show any mortality and morbidity up to 2000 mg/kg body weight. So, the maximum tolerable can be obtained as 2000 mg/kg body weight was shown in (Table I)

28 days repeated oral toxicity study

The repeated oral administration of Sangu parpam II in three different dose levels did not produce any significant changes in the body weight (Table II) when compared to normal control animals.

There was no significant difference was noticed in the food intake as well as the water intake.

The animals treated with sangu parpam of different doses did not produce any significant variations in the haematological parameters like WBC, polymorphs, RBC,HB, PCV& MCH (Table III)

The organs which were collected for histopathological studies were weighed and the mean organ weights were tabulated. There was no significant variation noted in the major organs like brain, heart, lungs, liver, testis kidney and uterus (Table IV, V)

The animals treated with sangu parpam of different doses did not produce any significant variations in the Blood glucose level. (Table VI)

The animals treated with Low Middle and High dose of SP I, II, III did not produce any significant changes in HDL cholesterol level, Total cholesterol, Triglyceride (Table VII).

The animals treated with Sangu parpam of different doses did not produce any variation in Total Bilirubin level, SGOT, SGPT and

ALP (Table VIII) when compared with the control group animals.

Various dose treatment of Sangu parpam after 28 days did not produce any significant deviation in and urea, Uric Acid and Creatinine (Table IX) levels when compared to the normal control levels. There were no significant observation in histopathology examination and revealed normal architecture in comparison with control and treated animal (Fig I).

FIG I: HISTOPATHOGY SLIDES OF CONTROL & SP I, II, III HIGHER DOSE LEVEL

DISCUSSION

The oldest form of healthcare in the world is the use of herbs as a medicine[9].·During the past decade, developed and developing countries were highly accepting the natural therapy. Because of poverty and limited access to allopathy medicine, about 80% of the world's population, uses traditional system of medicines as their source of healthcare[10].Most of the herbal preparations are safe for consumption, some herbs like most biologically active substances could be toxic[11] Mostly, without any proper scientific evaluation herbal products are launched into the market. And also there is no effective tool to regulate manufacturing practices and standards of drug.

The standardization of herbal drug is very essential for the global acceptance. In this study Sangu Parpam which is highly recommended for the disease Peptic Ulcer in Siddha Literature is taken up for standardization methods. Proper standardization techniques, toxicological and pharmacological evaluation on these medicines to meet the criteria will support its use worldwide. Therefore, in this study an attempt has been made to evaluate the toxicological analysis of a Herbo-marine Siddha drug **Sangu Parpam.** Sangu (Conch) is a Marine origin drug. Loads of research has been conducted in drugs of plant origin but very little amount research work done in the marine origin drugs. This may help to reveal the quality and safety of the drug and will lead to universal acceptance of the drug Sangu parpam for the disease Peptic Ulcer.

Acute toxicity

SANGU PARPAM I, II, III was administered single time at the dose of 50 mg/kg, 300mg/kg, 1000mg/kg and 2000mg/kg and observed for consecutive 14 days after administration. All animals were observed daily once for any abnormal clinical signs. Weekly body weight and food consumption were recorded. No mortality was observed during the entire period of the study. Data obtained in this study indicated no physical and behavioral signs of any toxicity due to administration of SANGU PARPAM I, II, III at the doses of 50mg/kg, 300mg/kg, 1000mg/kg and 2000mg/kg.

14 days, all animals were observed for functional and behavioral examination. In functional and behavioral examination, home cage activity, hand held activity were observed. Home cage activities like body position, respiration, clonic involuntary movement, tonic involuntary movement, palpebral closure, approach response, touch response, pinna reflex, sound responses, and tail pinch response were observed. Handheld activities like reactivity, handling, palpebral closure, lacrimation, salivation, piloerection, papillary reflex, abdominal tone, limb tone were observed. Both functional and behavioral examination was normal in all treated groups.

Sub-acute toxicity

All the animals from control and all the treated dose groups up to 300mg/kg survived throughout the dosing period of 28 days. No signs of toxicity were observed in animals from different dose groups during the dosing period of 28 days. Animals from all the treated dose groups exhibited comparable body weight gain with that of controls

throughout the dosing period of 28 days. Thus food consumption and water intake of control and treated animals was found to be comparable throughout the dosing period of 28 days. Haematological analysis conducted at the end of the dosing period on day 29, revealed no abnormalities attributable to the treatment. Biochemical analysis conducted at the end of the dosing period on day 29, no abnormalities attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Mild changes observed in the SP I high dose and SP II low dose. No significant changes was observed in organ weight during the treatment. But the range is with in the laboratory limit. Histopathological examination revealed normal architecture in comparison with control and treated animal.

Acute toxicity study revealed Sangu Parpam I, II, III at the doses of 50mg/kg, 300mg/kg, 1000mg/kg and 2000mg/kg to the rats did not produce drug- related toxicity. No mortality was observed during the entire period of the study. Data obtained in this study indicated no significance physical and behavioral signs of any toxicity. The maximum tolerable dose obtained from the acute toxicity study is about 2000mg/kg b.wt. Sub-acute toxicity study revealed **Sangu Parpam I, II, III** can be considered safe, as it did not cause either any lethality or adverse changes with general behaviour of rats and also there were no observable detrimental effects (100 to 300mg/kg body weight) over a period of 28 days. Both acute and Sub-acute toxicity studies of various preparation of Sangu parpam revealed safe in animals tested. Thus the Sangu Parpam I, II, III are safe in prescribed dose level as per the literature.

CONCLUSION

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EFFECT OF MUSCLE ENERGY TECHNIQUE VS EXERCISE FOR SUBJECTS WITH TEMPOROMANDIBULAR JOINT DYSFUNCTIONS.

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ABSTRACT

Objectives: *To determine the effectiveness of muscle energy technique on range of mouth opening and pain in subjects with temporomandibular joint dysfunctions.*

Materials and methods: *40 subjects aged 20 to 30 years both male and females were recruited into the study. Double blinded randomized controlled trial design was carried out for 4 weeks duration.Subjects with temporomandibular joint dysfunctions were assessed and examined bydental surgeon and referred to physiotherapy OP department. Subjects were randomly assigned to Group A and Group B. Group A (N - 20) received Muscle energy techniqueand Group B (N - 20) received Active ROM exercises alone. Pre and post treatmentmeasures were recorded by dental surgeon and treatment provided by physiotherapist tomake the study double blinded randomized trial. Outcome measures were recorded usingVAS scale and Inter incisal distance in mm.*

Results: *Statistical significance were obtained withmuscle energy technique group A, while no significance achieved in general ROM exercise group in this study.*

Key words: *Muscle energy technique, ROM exercise, VAS scale*

Introduction

Temporomandibular joint dysfunction (TMD), is an umbrella term for a group of pathologies affecting the masticatory muscles, the temporomandibular joint (TMJ) and its related structures[1]. Although, traditionally viewed as a syndrome, the recent studies supports that TMD is a cluster of related disorders in the masticatory system, which has many common features[2,3]. The term has been used synonymously with a host of other terms including TMJ disorders, TMJ dysfunction syndrome, craniomandibular disorders and myofacial pain dysfunction syndrome[25]. TMD is considered a musculoskeletal disorder of the masticatory system that affects more than 25% of the general population[2]. Studies reported thatone or more symptoms of TMD will be exhibited by 85 to 90 percentage of population in their lifetime and 5 to 6% will have clinically significant TMD related jaw pain[10].

Epidemiological studies in specific population showed that about 75% of population have at least one sign of joint dysfunction (tenderness, joint sounds etc.) and about 33% have at least one symptom (face pain, joint pain, muscle pain etc.)[21,23]. Clinical studies have revealed that a small percentage of people have problems severe enough to seek care for TMD . Mostlywomen are affected than men in the ratio of about 8:1. TMD accounts for large percent of non-dental pain in the oro-facial region, with pain being one of the most frequently presenting symptoms[24]. Usually involving the muscles of mastication (temporalis, masseter and themedial and lateral pterygoid), the pre-auricular area and/or the temporomandibular joint. TMD usually have a wide range of symptoms including restricted mouth opening, locking, clicking and commonly joint and muscle pain. It is also commonly associated with other factors affecting the head and neck regions such as headache, ear-related symptoms and cervical spine disorders [9]. Patients with chronic TMD frequently report of depression, poor sleep quality and low energy[7]. Chronic TMD interferes with normal social activity, interpersonal relationships and ability to maintain employment[17].

Management of TMD often involves a multidisciplinary approach; dentists, orthodontists, physical therapists, physicians and psychologists work together[6].

Current treatment of TMD

includes behaviour modification,pharmacological intervention, nutritional counselling, occlusal therapy, physical exercise therapy and, orthodontics, prosthetics, orthotics and surgery. Considering the complexity of TMD, certain patients may benefit from more than one treatment modality at any one time[9]. Physical therapy interventions including electrophysical modalities, exercise and manual therapy techniques are potentially effective in managing TMD.Electrophysical modalities include interventions such as ultrasound, microwave, laser and TENS[16]. Physical therapy interventions often include therapeutic exercises for the masticatory or cervical spine muscles toimprove strength and mobility in the region[19]. Manual therapy helps toreduce pain and restore mobility[16]. The Maitland technique and Muscle energy technique are most widely used technique for musculoskeletal disorders.

Muscle energy technique(MET)

Muscle Energy Techniques (MET) are used to treat muscles with excessive tension, which limitmotion and cause pain[4]. MET can be used to strengthen a physiologically weakened muscle, to lengthen a shortened or spastic muscle and also to reduce localized oedema and to relieve passive congestion. MET is useful in, improving joint motion by reducing tension in the jaw muscle and subsequently reducing pain, be it localized or referred to the face and head. MET is of a valuable therapeutic use in resolution of themusculoskeletal component of TMJ pain and dysfunction. As MET works both on the TMJ andthe muscles of mastication (mainly the temporalis and masseter) it is considered to be more appropriate in treating both the limitation of mouth opening as well as the muscular component (strain and/or hypertonicity) of TMD[4].

Materials and methods

The participants of this study were adults aged 20-30 years of both sexes, were diagnosed by the dentists as having unilateral TMD based on their clinical findings and imaging techniques.

The sample size included in the study were 50 participants using simple random sampling method, each receiving a self-reportingquestionairre by mail. Out of 50 participants in the study, 40 participants returned the survey, a response rate of 80% Participants completed demographic questions such as age, gender and marital status andspecific informationabout the level of impairment of their report of TMJ pain. The participants were enquired about the frequency of their TMJ pain. The examination included observation, palpation, pain assessment and range of mouth opening (inter-incisal distance) assessment. The participants fulfilled the following inclusion and exclusion criteria.

Inclusion criteria

- Pain in temporomandibular joint
- Unilateral pain
- Reduced mouth opening as measured interincisal distance < 40mm
- Symptoms < 3 months

Exclusion criteria

- Degenerative Temporomandibular joint
- Infective temporomandibular joint
- Inflammatory temporomandibular joint
- Malignancy
- History of dislocation and surgeries of temporomandibular joint
- Ankylosis of temporomandibular joint
- Hypermobile temporomandibular joint

Patients were explained about the purpose of the study, types of evaluation and intervention procedures to undergo as part of the study. They were assured that their identity will be concealed, and informed that the results of the study will be shared among other professionals and even may be published in scientific journals. The subjects were asked for their queries regarding the purpose of the study and their anxieties and

doubts were cleared. A written informed consent was obtained. Participants were treated with MET which includes post isometric relaxation and reciprocal inhibition, thrice a week for 4 weeks. The time period of each contraction was 10 seconds and was repeated for about 5 times.

Evaluation was done for the level of self-reported pain and TMJ range of mouth opening, in case of each subject, before the commencement of interventions and at the end of each week of interventions. Pain was measured using Visual Analogue Scale (VAS scale), which is a 10 cm horizontal line. The left end in the line was marked 0 which represents no pain at all and the right end of the line was marked 10, which signifies the worst pain experienced by the subject. The subjects were asked to mark the intensity of the pain perceived on the scale. The TMJ range of motion was measured as the Maximum Mouth Opening (MMO) and measurement was taken with a flexible intra-oral ruler as the participant actively opened his/her mouth to the maximum possible distance. The distance between the upper central incisors and the lower central incisors was determined as the inter-incisor range of opening[14] (Figure 1 & Figure 2).

Statistical analysis: The collected data were analyzed using parametric tests as it is interval in nature. The intra group pre and post-test data for MMO were analyzed using paired t-test, while the post test intergroup data were analyzed with unrelated t-test. The visual analog scale data were analyzed using non-parametric tests as it is ordinal in nature. Wilcoxon signed rank test is used to analyse the intra group pre and post-test VAS scores and Mann Whitney U test is used to analyse the post-test inter group VAS scores . The statistical significance of the p-valuefor data was fixed at 0.05.

Result:

The study consisted of 40 participants (n = 40), with a gender distribution of 21 females (55%)and 19 males (45%). The age of the participants ranged between 20 and 30 and the mean was25.5+2.96. The mean duration of symptoms for the participants was 47.7+23.85 days. Withinsame group, the comparison of pain as measured by VAS at the end of each week of intervention(Table 1). The gradual and definitive reduction in the self reportedpain on a week-on-week basis is reflected in the median values.

The median score for the pre-test by VAS was 7, which reduced to 6 at the end of week 1, 4 at the end of week 2, 3 at the end of week 3, 1 at the end of week 4 of the intervention period (Table 2). The results showed that there is a reduction in the level of pain at the end of each week and at the end of the 4 week intervention period is statistically significant ($p<0.05$).

Similarly, the MMO scores show that there is considerable increase between the pre test value
and post test value. Moreover the MMO scores recorded at the end of each week of intervention also shows gradual improvement. The pre test mean MMO score was 21+1.26 (Table 3). At the end of week 1, the mean MMO scores showed a remarkable improvement with the value of (25.95+1.28). This improvement was maintained in subsequent weeks as can be seen from the values measured at the end of each week.

It shows that the Mean Difference (MD) between the pre test score and the score at the end of
week 1 is 4.95. The statistical analysis (t-value = 56.18) reveals that the improvement in MMO
value is statistically significant (p-value = 0.00). The improvement in MMO value at the end of
week 2 when compared to the pre test value showed a MD of 8.85, which was statisticallysignificant (t-value - 67.41, p-value = 0.00). At the end of week 3 the MMO scores showed aMD of 13.25 when compared to the pre test value, which on statistical analysis (t-value =85.08)showed a significant improvement (p-value = 0.005). The MD in MMO at the end of week 4when compared to the baseline was 17.85 and on analysis (t-value = 107.13) it showed asignificant improvement (p-value = 0.005).

The post test MMO score at the end of the 4 week intervention period when compared to the pretest score showed a MD of 22.45, which on statistical analysis (t-value = 132.25) revealed a significant improvement (p-value =0.005) (Table 4).

Discussion

MET reduces tension in the jaw muscles and subsequently reduces pain, be it localized or referred to the face and head[20]. In thispresent study the maximum mouth opening has increased, it is similar to the findings by Anderson. The muscle energy technique thus stimulates the muscle spindles and Golgi tendon organs reducing excessive activity. Stretching of the muscle fibers stimulates the Golgi tendon receptors, which have an inhibitory influence on muscle tension, leading to muscle relaxation[19]. When a particular muscle isactively contracted, its antagonists are reflexively relaxed. Therefore, opening the mouth against resistance is inclined to relax contracted elevator muscle and vice versa for opening muscles[20], which can increase the ROM. However findings of present study did not support the work of Gosling and Frois[7] and Freshwater and Gosling[9] The reduction in pain by MET is similar to the findings by Lewit and Simons[5] and Brodin[3]. Although, Brodin's work whichis involved in the treatment of lumbar spine rather than TMJ, it is reasonable to assume that the mechanisms involved in both are similar. The possible reason for this discrepancy is that both the studies involved only a single session of MET which may not have been adequate in bringing about more positive results for the study clinically.

Limitations of the study:

- The study duration was short, only 4 weeks and the results apply to short term only, which might differ in the longer run
- No long term follow up was done to ascertain the differences in long term gains in the protocols
- Sample size taken for this present study is small and bigger sample might have led to some differences in the results
- Limited parameter of outcome measure was used which might bias the results Conclusion;

This study showed that, muscle energy technique demonstrated a significant reduction in TMJ pain and increase in TMJ range of motion as measured by Maximal mouth opening after four weeks of intervention in TMD. However, due to the lack of control group, small sample size and a dearth in the literature supporting the findings of this study, the results should be interpreted with caution.

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TABLE 1: PRETEST AND WEEK WISE VAS

Test	VAS score
Pre test	7
Week 1	6
Week 2	4
Week 3	3
Week 4	1

Table 2: Pre - Post test comparison of VASscore

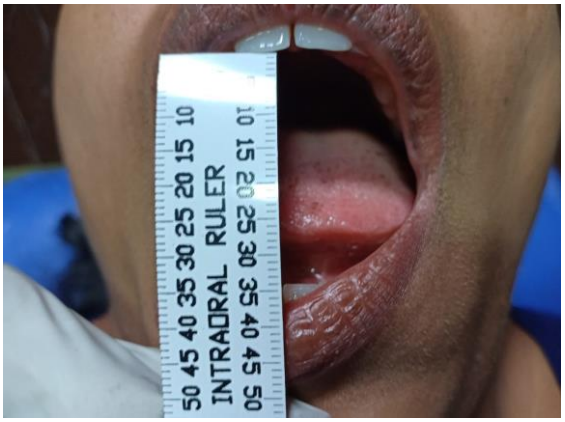
Pre test and Week wise comparison	Mean difference	Z- value	P - value
Pre test week 1	1	-4.13	0.00*
Pre test week 2	2	-3.98	0.00*
Pre test week 3	3	-3.97	0.00*
Pre test week 4	4	-3.96	0.00*

TABLE 3: PRETEST AND WEEK WISE MEAN AND STANDARD DEVIATION OF MAXIMUM MOUTH OPENING

Test	Mean	SD
Pre test	21.00	1.26
Week 1	25.95	1.28
Week 2	29.85	1.42
Week 3	34.25	1.48
Week 4	38.85	1.22

Table 4: Pre Post test comparison of Maximal mouth opening

Pre test and Week wise comparison	Mean difference	Z- value	P - value
Pre test week 1	4.95	56.18	0.00*
Pre test week 2	8.85	67.41	0.00*
Pre test week 3	13.25	85.08	0.00*
Pre test week 4	17.85	107.13	0.00*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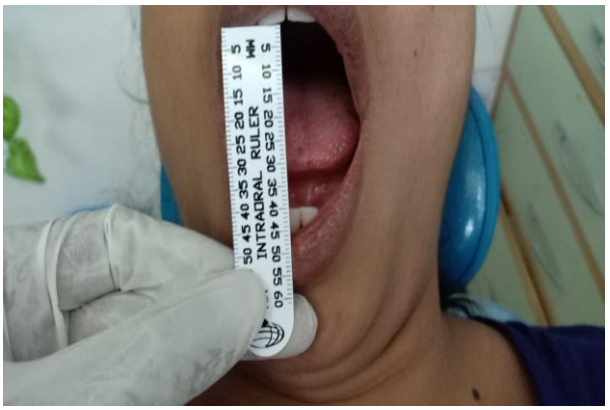
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