A multidisciplinary journal for publication of medical and biomedical research findings on issues pertinent to improving family health and related issues of public health
A multidisciplinary journal for publication of medical and biomedical research findings on issues pertinent to improving family health and related issues of public health

Editor – in – Chief
Dr. Phillip Kigodi

Associate Editors
Associate Professor Andrew Masta
Dr. Prem Rai
Professor Francis Hombhanje

Managing Editors
Professor Lohi Matainaho
Associate Professor Victor J. Temple

Speciality Editors and Editorial Board Members:
Dr. Adolf Saweri, Dr. Jacob Morewaya, Ms. Estelle Jojoga, Dr. Subhadda Perera, Dr. Jackson K. Lauwo, Dr. Wangi Linjim, Mr. Gairo Gerega, Dr. Paulus Ripa, Dr. K. Beaga, Mr. R. Kitau, Prof. Z. S. C. Okoye, Dr. David K. Obatomi, Prof. B. O. Ogunbanjo, Prof. C. E. Anyiwo, Dr. Reshma Suvarna, Dr Alphonsus N. Onyiriuka, Dr. Yama Oshiozokhai Eboetse, Dr. Florence Muga,

INFORMATION
SUBSCRIPTIONS: Correspondences concerning subscriptions, purchase of single copies and back issues, lost copies and related business should be addressed to the Secretary, Basic Medical Sciences, School of Medicine and Health Sciences, University of Papua New Guinea, P. O. Box 5623 Boroko, N.C.D., PNG. Official website is www.pacjmedsci.com; For Instructions to Authors please visit the official website.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Content page</th>
<th>Page #</th>
</tr>
</thead>
<tbody>
<tr>
<td>-----------------</td>
<td>-------</td>
</tr>
<tr>
<td>RESEARCH PAPERS</td>
<td></td>
</tr>
<tr>
<td>Pharmacological Management of Temporomandibular Joint Disorders – A Review:</td>
<td>3 – 14</td>
</tr>
<tr>
<td>Vagish L.S Kumar: Vol. 13, No. 2, December 2014:</td>
<td></td>
</tr>
<tr>
<td>CASE REPORTS</td>
<td></td>
</tr>
<tr>
<td>Instructions for Authors:</td>
<td>56 – 61</td>
</tr>
</tbody>
</table>
PHARMACOLOGICAL MANAGEMENT OF TEMPOROMANDIULAR JOINT DISORDERS – A REVIEW

Vagish Kumar L.S

Department of Oral Medicine and Radiology, Yenepoya Dental College and Hospital, Yenepoya Research Centre, Yenepoya University, Mangalore, India

Correspondence author: vagishkumar_12@rediffmail.com

ABSTRACT:
Patients frequently visit dentists with main intention of pain relief. Among various causes of pain, the temporomandibular disorder representing a group is one of them. Every dental practitioner should be aware of these disorders and the mode of treating them. Pharmacology usually represents the first stage of management or as an additional treatment for these disorders. Commonly used pharmacotherapeutic medicines include analgesics, corticosteroids, muscle relaxants, antidepressants and sedative-hypnotics. Analgesics such as naproxen, diclofenac, ibuprofen and corticosteroids such as methylprednisolone, triamcinolone acetonide are beneficial in relieving pain of acute and chronic temporomandibular disorders associated with inflammation. Muscle relaxants such as cyclobenzaprine and chlorzoxazone can be given for pain of temporomandibular disorders associated with muscular tensions and spasms. Sedative-hypnotics are helpful to patients who have muscular tension together with poor sleep patterns. Antidepressants like amitriptyline are effective in chronic myofascial pain syndrome and in patients with coexistent depression and tension headaches. Benzodiazepines such as clonazepam and diazepam are helpful in chronic myogenous jaw pain. The article attempts to review the pharmacological management of temporomandibular disorders in a concise manner so as to be helpful for medical and dental practitioners.

Key words: Pain, Pharmacotherapy, Temporomandibular disorders

Submitted: August 2014; Accepted: November 2014
INTRODUCTION:

Dental practitioners routinely encounter patients with pain arising from several sources like bone, joint, muscles, nerves, and somatic structures. It may be temporomandibular dysfunction as a result of myofascial, neurologic, bone, or joint derangements; Tooth pain as a result of dentin, enamel, pulpal, or periapical defects; atypical odontalgia; pain or burning sensations in the tongue; altered tongue sensations or cracked tooth syndrome. Pharmacology is a cornerstone in the treatment of pain and is aimed towards the source and the nature of pain [1]. The term “temporomandibular disorders” (TMD), is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ) and associated structures, or both [2, 3]. These group of disorders are characterized by facial pain in the region of the TMJ and/or the muscles of mastication; limitation or deviation in the mandibular range of motion, and TMJ sounds during jaw movement and function; headache; generalized tightness around face in the morning and Otalgia [4, 5]. Category 11 of International Headache society (IHS) classification includes temporomandibular joint disease and disorders of teeth, jaws, and related structures [5]. They are frequently associated with acute or persistent pain, and the patients often suffer from other painful disorders. The chronic forms of TMD pain often adversely affects the work or social interactions, resulting in depression, feeling of worthlessness and an overall reduction in the quality of life [6]. Pharmacologic therapy can be an effective method of managing symptoms associated with many TMDs. Medications in conjunction with appropriate physical therapy and definitive treatment can offer the most complete approach to many of these problems [6]. Anxiolytics are indicated for acute TMD pain; Non-Steroidal Anti-inflammatory drugs (NSAIDs), muscle relaxants, and local anesthetics may be used for both acute and chronic conditions; and the tricyclic antidepressants are primarily indicated for chronic orofacial pain management [7]. Treatment of TMD patients initially should be based on the use of conservative, reversible, and evidence based therapeutic modalities. Symptoms with TMDs have been observed to improve or resolve over time. Many conservative modalities of treatment provide symptomatic relief, have less risk of adverse effects and are nearly as effective as the invasive form of treatments [6]. Pharmacological intervention is usually considered an adjunctive therapy because more definitive treatments typically are used to correct the underlying pathophysiological process. Pharmacotherapy often is the primary approach in treating depression and the inflammatory processes that are frequently associated with temporomandibular disorder.
The pharmacologic management of TMDs rests on principles such as: demonstrated efficacy, an acceptable side effect liability, and safety when given for prolonged periods [9]. The commonly used pharmacologic modalities used for treatment of TMDs broadly include analgesics, corticosteroids, antidepressants, muscle relaxants and sedative-hypnotics.

**Non-steroidal anti-inflammatory drugs [10]:**
Non-steroidal anti-inflammatory drugs (NSAIDs) represent first-line drugs for treating TMD pain. Patients having painful disc displacement, capsulitis, synovitis and myositis [11], musculoskeletal pain, arthritis [10], masticatory myalgia and myofascial pain associated with the TMJ may benefit from these drugs [12]. They are particularly indicated for joint pains secondary to inflammation and painful articular disorders [2, 13]. NSAIDs are considered effective for acute postsurgical dental pain and chronic arthritic pain. A study by Ta and Dionne [14] showed that in patients with TMJ disc displacement, pain significantly reduced with naproxen 500mg taken twice daily for 6 weeks. Also maximal comfortable mouth opening improved in these patients: but there was approximately 40% increase in dyspepsia and pain with naproxen [14]. These groups of drugs should be discontinued after 7-10 days of use, if they fail to achieve the therapeutic goal or patient manifests serious side effects [12]. Topical diclofenac formulated with dimethyl sulfoxide applied four times a day is found to be equal to oral diclofenac sodium 50mg administered twice a day in subjects who have pain and tenderness due to joint osteoarthritis. [15] A study found that treatment of muscular TMD patients with sodium diclofenac 50mg twice a day promoted higher analgesia when associated to an occlusal splint [16]. NSAIDs are toxic when administered chronically at relatively high doses. Patients should be monitored very closely during first few weeks of treatment. Chronic use of highly selective cyclooxygenase-2 (COX-2) inhibitors may cause gastrointestinal (GI) events including ulcerations, perforations, and bleeds, than nonselective or semi-selective NSAIDs. Users of NSAIDs have a threefold greater risk of developing serious adverse GI events than nonusers, and the risk is greater in patients older than 60 years of age [17]. In addition, decreased renal function leading to water and sodium retention with concomitant hypertension has been observed in patients taking simultaneous antihypertensive drugs with these drugs. NSAIDs also carry increased cardiovascular risk, especially in elderly, in patients with hypertension, coronary artery and atherosclerotic disease, coronary artery bypass surgery and in patients with previous cardiovascular events.

Chronic pain patients taking antidepressant drugs concomitantly with NSAIDs have up to 16-fold increase in the risk for upper GI bleeds [10, 12]; they should not be used in asthma patients [18].
Corticosteroids:
Corticosteroids are powerful anti-inflammatory agents that can be administered orally or injected directly into the joint space. The primary clinical indication is synovitis that is not infectious, degenerative joint diseases and poorly responding to NSAIDs [9, 18]. They can also be used in acute, generalized muscle and joint inflammation associated with polyarthritides [7]. A 6-day methylprednisolone followed by 3 to 6 weeks of NSAID therapy in TMJ closed lock patients works equally compared to arthroscopy or open joint therapy in reducing jaw pain and dysfunction. Intra-articular steroids containing 0.7mL of methylprednisolone acetate 40mg/mL combined with local anesthetic in children or 1.0mL of triamcinolone acetonide or 1.0mL triamcinolone hexacetonide in adults significantly reduces pain and improves function in TMD arthritis [19, 20].

Schindler et al [21] discovered that intra-articular glucocorticoid injections used in a wrong way caused severe destruction of the joint. Oral methylprednisolone followed by NSAID’s for 3-6 weeks are effective with rehabilitation in TMJ closed lock patients [10, 22]. Corticosteroid and hyaluronic acid injected directly into TMJ decreases muscular pain and results in marked increase in the ability to open the mouth [22]. In addition, intra-articular corticosteroid injections and follow up for 8 years, evidenced improvement in clinical signs of TMD together with radiographic findings, suggesting remineralization of areas of condylar erosion [23]. Iontophoretic administration of steroids will result in high drug levels at the site of affected or painful TMJ, by applying an electric current to ionized drug solutions. Reid et al. found that iontophoresis with dexamethasone in a lidocaine vehicle in patients with TMD showed improvement and reported less pain with improved range of motion [24]. However this mode of drug delivery should be used only for severe cases and frequent injections must be avoided. Aggressive use of intra-articular steroids can cause articular cartilage destruction, infection and disease progression. Long term use may promote destruction of joint tissues [8]. Furthermore, oral corticosteroid use should be limited to no more than 2 weeks because of risks of decreased resistance to infection, elevations in blood glucose, osteoporosis, and suppression of hypothalamic-pituitary-adrenal axis. Patients having severe disc interference disorders and inflammatory conditions such as capsulitis, synovitis, and TMJ osteoarthritis/ rheumatoid arthritis, may benefit the most from this category of drugs.

Sodium hyaluronate intracapsular injection has been suggested for the treatment of TMJ articular disease. Its use following arthrocentesis may be helpful in reducing pain [7]. Hyaluronic acid injections are reliable in rheumatoid arthritis. It is also useful in painful disk displacement with reduction [25].
**Opioids** [11]

Opioid therapy should be considered only when: There is inadequate pain relief from prior nonopioid therapy; there is negative history of substance abuse; a confirmation that the pain being treated is of physiologic rather than psychologic origin; both patients and doctors are willing to adhere to an “opioid contract” between the doctor and patient which includes compliance with a scheduled administration of an oral opioid and close clinical follow-up [26]. Opioid should not be used as first-line drugs in patients with TMD. Also one should be aware of drug-seeking patients complaining of TMD pain. Opioids are indicated in palliative form when patient has severe unbearable TMD pain and is resistant to other modes of treatment [12]. However administration of opiates in patients with intractable TMD pain when other modes have failed is reasonable in specialist hands. Before prescribing an opioid, the patient’s level of pain and its interference with the quality of life should be determined. Assessment of previous drug use, past and current psychiatric status should be determined, often in consultation with a psychiatrist. Long-acting or sustained-release preparations of opiates, such as morphine sulfate and oxycodone limits cycles of breakthrough pain and opiate withdrawal symptoms. Doses should be increased to achieve efficacy or decreased to reduce side effects with continuous monitoring. Chronic use of opioid leads to constipation [26], and this can be dealt through intake of plenty of fluids and fiber, exercise, stool softeners and laxatives. Side effects like sedation and nausea dissipate with continued use. In TMD patients, morphine can be used as a 10mg intra-articular injection in arthrocentesis, arthroplasty procedure and intracapsular disorders. Long-term reductions in pain have been associated with this mode of arthrocentesis [27, 28]. The combination of acetaminophen 650 mg plus tramadol 75mg seems efficacious in postsurgical dental pain patients [29]. Osteoarthritis, fibromyalgia and diabetic neuropathy effectively respond to tramadol or tramadol with acetaminophen. When tramadol is combined with acetaminophen an opiate-sparing effect occurs compared with tramadol alone, resulting in better tolerability [10]. Any drug that is a Cytochrome P450 2D6 (CYP2D6) inhibitor, including the antiarrhythmic quinidine and antidepressants of the SSRI (selective serotonin reuptake inhibitors) class, such as paroxetine, reduces the analgesic activity of tramadol. Concurrent administration of tramadol with antidepressant class of drugs including tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), and SSRIs can produce tremors, convulsions, muscle rigidity, and hyperexia [30,31].

**Benzodiazepines:**

Benzodiazepines bind to specific receptors in the central nervous system (CNS) and are anxiolytic, sedative, and hypnotic. Diazepam
and clonazepam possess potent anticonvulsant activity. These drugs reduce muscle contraction, thus reducing the pain of TMD patients. Improvement of sleep patterns in subjects with chronic pain helps in breaking the pain cycle [32]. Diazepam and clonazepam, have long duration of action. Oxazepam, alprazolam, and triazolam are short acting drugs [10]. They are useful in patients with early disk displacement without reduction. Temazepam 10mg at night in the form of oral suspension is the best choice for these patients. Oral suspensions are used because patients can easily adjust the dose to avoid side effects and also to reach maximum effect when desired. It is contraindicated below 12 years of age [18]. Alprazolam (0.5-3mg/day) plus ibuprofen (2400mg/day) for 6 weeks is found to be effective in fibromyalgia patients [33]. Patients with chronic myogenous jaw pain given diazepam 5 mg four times a day for 4 weeks report significantly great decrease in pain than those taking placebo. Combination of ibuprofen and diazepam provides better pain relief from musculoskeletal origin than ibuprofen alone [34]. In patients with TMD who had failed appliance therapy and physical therapy, 1 month of clonazepam intake at bedtime was effective compared with placebo [35]. Longer-acting benzodiazepines with anticonvulsant activity, such as diazepam and clonazepam, may be more beneficial in relieving muscular pain of TMD [10, 36]. Oral benzodiazepines have side effects like drowsiness and psychomotor impairment. Peak blood levels of these drugs occur when the patient is asleep if taken immediately before sleeping. The dose should be halved in case of elderly people to prevent CNS depressant and memory impairment. Benzodiazepines like alprazolam, diazepam, midazolam, and triazolam, are Cytochrome P-450 3A4 (CYP3A4) substrates. Concomitant foods, such as grapefruit juice, and drugs including azole antifungals, erythromycin, clarithromycin, and calcium channel blockers that inhibit the CYP3A4 isoform, can significantly reduce the metabolism of these benzodiazepines leading to elevated blood levels and enhanced CNS depression. Therapy with these drugs should be limited to less than 4 weeks to prevent physical and psychologic dependence [10, 37]. The natural course of myofascial pain combined with conservative therapy will likely result in lowering of symptoms to acceptable levels [12]. Patients with depression should be referred to psychiatrist before prescribing benzodiazepines [8].

**Non benzodiazepine sedative hypnotics:**

Sleep disturbances are correlated with degree of pain severity and psychologic distress in patients with TMD. Eszopiclone, zolpidem, and zaleplon represent nonbenzodiazepine sedative hypnotics. In addition to inducing sleep, sedative doses of eszopiclone and zaleplon have muscle-relaxing activity.
However sleep walking has been reported in patients taking zolpidem [10].

**Centrally acting muscle relaxants:**
Muscle relaxants consist of two broad categories- centrally acting and peripherally acting agents. Peripheral muscle relaxants block muscle contraction and reduce skeletal muscle tone. Centrally acting muscle relaxants provide relaxation of muscle tissue by sedative effect on central nervous system (CNS). Muscle relaxants used in treating TMD are usually centrally acting, depress polysynaptic reflexes and are sedatives. These drugs help prevent or alleviate the increased muscle activity that might have resulted in TMD [8]. They relieve acute musculoskeletal pain without impairment in motor function and are often prescribed in conjunction with NSAIDs [9, 12]. Examples include carisoprodol, chlorzoxazone, cyclobenzaprine, metaxalone, methocarbamol, baclofen, and tizanidine. Since they have lower therapeutic indices they must be used with extreme caution in patients with concurrent depression. Cyclobenzaprine has been suggested to potentially benefit patients who have TMD with muscle contraction and spasm [8]. In a study, patients with TMD reported improvement in jaw pain when cyclobenzaprine 10 mg was taken at night. The effect was superior to either placebo or clonazepam 0.5 mg combined with self-care and education in the management. Cyclobenzaprine is effective muscle relaxant.

Low dosing 5-10mg taken 1-2 hours before bed time is usually effective [38, 39]. Carisoprodol has abuse potential and appears to be less effective in chronic pain conditions [39]. Sedation is a major side effect of skeletal muscle relaxant group of drugs. Cyclobenzaprine structure resembles tricyclic antidepressants, has anticholinergic activity, thereby causing side-effects like xerostomia and tachycardia. Thus cyclobenzaprine is contraindicated in narrow-angle glaucoma patients. Muscle relaxants are best used before sleep to reduce side effects. Skeletal muscle relaxants should be used for short duration in conjunction with physical therapy [8]. Metaxalone which has a few central effects is appropriate muscle relaxant for patient who must work while taking the medication [7].

**Topical medications:**
Topical NSAID’s are useful in reducing pain in acute and chronic musculoskeletal injuries. NSAIDs can be incorporated in transdermal creams for application on the skin over the painful joint or muscle. Ketoprofen, felbinac, ibuprofen, and Piroxicam have significant efficacy. These are also helpful in chronic conditions such as arthritis and almost are devoid of adverse effects [5]. Andrew [13] recommended their use regularly four times a day for 4 weeks. Food and Drug Administration (FDA)-approved topically applied agents that have potential usefulness in TMD pain include capsaicin 0.025% [40] to 0.075% and the 5%
lidocaine transdermal patch. Capsaicin [41] is a derivative of the chili pepper and is effective in osteoarthritis and neuropathic pain. So, topical capsaicin is likely to benefit TMD patients. Capsaicin is devoid of systemic toxicity. However patients may initially experience burning sensations which will terminate with continued application. Combining capsaicin with a topical anesthetic, such as benzocaine 20% in pluronic lecithin organogel may help reduce this burning sensation [42]. Capsaicin is best used as an adjunct to NSAIDs, benzodiazepines, or other systemic modalities. TMD patients may be benefited from 5% lidocaine transdermal patch. The patch has to be cut into smaller sizes with scissors before removal from the release liner. Various types of pain have been reported to be improved through the use of this patch [10].

Antidepressants:
Antidepressants are grouped into three main categories: TCAs, MAOIs, and SSRIs. Several studies have reported efficacy of the TCA drug amitriptyline in patients who have TMD. Fourteen days of treatment with low-dose amitriptyline (25 mg/d) was significantly more effective than placebo in reducing pain intensity in women who had chronic TMD pain [43]. Low-dose amitriptyline (10–30 mg/d) demonstrated significant improvement in pain in both depressed and non-depressed subjects between six weeks to one year [10]. Tricyclic antidepressants with both serotinergic and noradrenergic effects (e.g., Amitriptyline or doxepin) appear to be most effective. Lower dosages (25 to 75 mg) should be used initially for non-depressive patients with higher antidepressant doses reserved for patients who are depressed. Sedative antidepressants may be useful when patients have sleeping problems and may help to reduce the use of hypnotics [8, 12]. Amitriptyline 10mg just before sleep can have an analgesic effect on chronic pain but has little effect on acute pain. It is an important part of management of fibromyalgia [7]. Duloxetine 60mg/day is helpful in achieving relief of pain in diabetic neuropathy and fibromyalgia [10]. Dothiepin is found to be significantly more effective in a mixed group of TMD and Atypical Facial Pain patients [44]. Common side effects of TCAs and SSRIs include nausea, sedation, psychomotor impairment, xerostomia, and constipation. These drugs must be absolutely avoided in patients taking concomitant MAOIs because the combination can lead to a potentially lethal serotonin syndrome consisting of confusion, fever, shivering, diaphoresis, ataxia, myoclonus, and severe hypertension [10].

Anticonvulsants:
Gabapentin has relatively low side-effects and is efficient in various chronic pain syndromes [45]. Anticonvulsant pregabalin has demonstrated efficacy and favorable tolerability in neuropathic pain. Both drugs are used for the treatment of pain associated with
postherpetic neuralgia and pregabalin is also being used for the treatment of painful diabetic neuropathy [10]. Patients with TMD of myogenous origin who took gabapentin report significantly reduced spontaneous pain together with reduced number of tender sites in the temporalis and masseter muscles, compared with placebo. The initial dose of gabapentin is 300mg, with additional 300mg every 3 days until pain relief is achieved. The daily maximum dose is 4200 mg [45]. Dizziness, drowsiness, xerostomia, peripheral edema, weight gain and memory impairment can occur in patients using gabapentin and pregablin. Anticonvulsants should be used as adjuvant analgesics in TMD patients with history of failed TMJ surgeries or those with chronic unremitting pain.

**Botulinum toxin:**
Low concentrations and large volumes of injection of botulinum toxin at multiple muscular sites may be helpful for muscular disorders related to TMD and relieve muscular spasms [13, 46].

**Local anesthetics:**
They are used when a myofascial trigger point is present. As procaine has low toxicity to muscles, concentrations at 1% are used. Also 1% or 2% lidocaine is commonly used. Pain and muscle spasm may be relieved for long term by needling the area with local anesthesia. This may be due to long term release of endogenous endorphins in the area of needling [13, 47, 48].

**Miscellaneous medicines:**
Local massage with topical Chinese medicinal herb ointment like Ping-On Ointment may provide a cheap and effective relief of pain for TMD patients. The ointment contains peppermint oil, 18%; menthol, 20%; natural camphor 6%, wintergreen oil, 6%, sandal-wood Oil, 1%, eucalyptus Oil, 4%; bee wax, 8% and aromatic oil, 1%. The ointment is to be applied in a circular motion on the affected area for 5 minutes 2 times daily [49]. A gel provided rapid pain relief and patient comfort and speeded restoration of the jaw’s functional abilities, usually within 5 minutes after it is applied. It was composed of 18% potassium complex, 10% dimethylisosorbide, and 72% aqueous hydroxyethyl cellulose gel applied and gently rubbed onto the facial skin over the painful TMJs, muscles of mastication, and myofacial areas. This is because potassium and dimethylisosorbide inhibits inflammation and pain [50].

**CONCLUSION:**
In patients who have inflammatory pain, such as arthritis, capsulitis, or TMJ disc interference disorders, NSAIDs are first choice of drugs. Naproxen is most efficient in this group. In patients with GI problems etodolac is an alternative. Cyclobenzaprine is effective in TMD with muscular etiology.
Long acting benzodiazepines with anticonvulsant properties, such as diazepam and clonazepam, are used once a day in patients who don’t respond to other muscle relaxants. Topical agents capsaicin and transdermal lidocaine have high therapeutic index and their use is encouraged. TCAs or anticonvulsants may be considered in patients who do not respond to NSAIDs, benzodiazepines, or muscle relaxants. The injection of corticosteroids directly into the joint space should only be used in patients who have severe pain and limitations in function attributable to intracapsular inflammation. Narcotic therapy should be reserved for the patient who has truly intractable pain. Patients for whom therapy such as behavioral modification, appliance therapy, physical therapy, or TMJ surgery greatly improves the quality of life, should only use drugs on an as-needed basis. Because many TMDs present symptoms that are periodic or cyclic, there is a tendency to prescribe drugs on a “take as needed” basis. This type of management encourages drug abuse, which may lead to physical or psychologic dependency. Frequent use of drugs tends to lead to more frequent pain cycles and less drug effectiveness. When drugs are indicated for TMDs, they should be prescribed at regular intervals for a fixed specific period. Further, definitive treatment should be provided so that medication will no longer be necessary.

REFERENCES:


38. Herman CR, Schiffman EL, Look JO, Rindal DB. The effectiveness of adding pharmacologic treatment with clonazepam or cyclobenzapine to patient education and self-
EFFECT OF SMOKING ON LUNG FUNCTION OF STUDENTS IN THE NATIONAL CAPITAL DISTRICT, PAPUA NEW GUINEA

Majella P Norrie and *Shalon Taufa

Discipline of Physiology, Division of Basic Medical Sciences, School of Medicine and Health Sciences, University of Papua New Guinea

*Correspondence author: shalontaufa@gmail.com

ABSTRACT:
Smoking is a proven risk factor for a reduction in lung function. The amount of damage to the respiratory tract is associated with the amount of cigarettes a person takes and the duration of smoking. This prospective observational cross-sectional study assessed the lung function of students aged 19 to 25 years in higher learning institutions in the National Capital District, Papua New Guinea. Spirometry was used to assess the lung function of 77 students consisting of 34 (44.2%) males and 43 (55.8%) females that met the inclusion criteria. Among the 34 male students, 16 (47%) were smokers and 18 (53%) were non smokers; among the 43 female students 15 (35%) were smokers and 28 (65%) were non smokers. For the male students no statistically significant differences (p<0.05) were obtained in the FEV\textsubscript{1}, FVC, PEF and FEV\textsubscript{1}/FVC\% values of the smokers compared to non smokers. There were no statistically significant differences (p<0.05) in the spirometry parameters for the female smokers compared to non smokers. The mean FEV\textsubscript{1}, FVC and PEF values obtained for the male students were significantly higher (p=0.001) than the corresponding mean values for the female students. However, there was no statistically significant (p<0.05) difference between the mean FEV\textsubscript{1}/FVC\% for the male and female student smokers. The mean FEV\textsubscript{1}, FVC and PEF values for the male non smokers were significantly higher (p=0.001) than the corresponding mean values for the female non smokers. There was, however, no statistically significant (p<0.05) difference between the mean FEV\textsubscript{1}/FVC\% of the male and female students that do not smoke.

Key words: Smoking, lung function, students.
Submitted: August 2014; Accepted: October 2014

INTRODUCTION:
Smoking has been proven to be associated with lung cancer and chronic obstructive pulmonary disease (COPD) and thus has a detrimental effect on lung function [1-7]. Tobacco smoking was found to be associated with increased prevalence of respiratory symptoms and reduced lung function. Female smokers are reported to have a greatly reduced expiratory lung function compared to their male counterparts [2, 8].

Anderson [9] looked at lung function in an adult population in the highlands of PNG in 1979 and found that in the over 45 years age group, 20%
of men and 10% of women had an FEV₁/FVC less than 60% and that the most prominent inhaled pollutant was wood smoke from fires in the houses and not tobacco smoke [9].

Yanga and Datta [3] looked at the effects of chronic smoking and betel nut chewing on the respiratory and cardiovascular parameters in a Melanesian male population in Port Moresby. They reported that chronic smoking and betel nut chewing were harmful to respiratory function, but smoking cigarettes and chewing betel nut for more than two years and less than five years did not show any changes in cardiovascular function [3].

The major aim of this study was to assess the pulmonary function of male and female students in institutions of higher learning. The major objective was to compare the pulmonary functions of smokers to non-smokers among the students.

SUBJECTS AND METHODS:
This study was a prospective observational cross-sectional study done between April and June 2013 in the National Capital District (NCD) of Papua New Guinea. Students were tested from three institutions of higher learning. The institutions were the Taurama and Waigani campuses of the University of Papua New Guinea (UPNG), the Port Moresby Business College (PBC) and the Don Bosco Technological Institute (DBTI) [10]. The sample size calculated used a design effect of one, a relative precision of 10%, and a confidence level of 95%. A sample size of 150 was considered appropriate for this study. Students noted their demographic data and other information in a self-designed pre-tested questionnaire. A student with history of any respiratory or cardiac illness was excluded from this study.

The weight and height of each student was measured and the body mass index (BMI) was further calculated using the weight and height. Pulmonary function tests were then carried out on each student using a computerised spirometer, SpiroUSB model run with spida5 software. Calibration and testing was done using the American Thoracic Society (ATS) guidelines and criteria. Pulmonary function parameters tested were: FEV₁, Forced Vital Capacity (FVC), FEV₁/FVC, Peak Expiratory Flow (PEF) and Forced Expiratory Flow 25% and 75% (FEF₂₅₋₇₅).

Analysis of the data was done using Microsoft XP Excel Data Package and the Statistical Package for Social Sciences (SPSS) version 20. The Shapiro-Wilks test was used to assess normality of data. P-value of <0.05 is considered significant.

The Ethics and Research Grant Committee of the School of Medicine and Health Sciences (SMHS), University of Papua New Guinea gave ethical clearance for this study to be carried
out. All the institutional heads also consented for the tests to be carried out in their institutions. [10]

RESULTS:
One hundred and sixteen (116) randomly selected students out of the 156 who volunteered, were asked to complete a questionnaire before performing the spirometry. Of these 116 students, 39 (33.6%) were excluded during the analysis of the questionnaire and spirometry results as they did not fulfil the inclusion criteria. Norrie has already shown the reasons for exclusion. [10] 77 students, 34(44.2%) males and 43(55.8%) females were finally accepted for analysis. Mean age for the female students was 22 ± 1.6 years (Mean ± SD) and for the male students was 22 ± 1.5 years.

Out of the 34 male students, 16(47%) were smokers and 18(53%) were non smokers. Table 1 shows the descriptive statistics of the pulmonary function indices for the male students who smoke and those who do not smoke. There were no statistically significant differences (p<0.05) in the spirometry parameters for the male smokers compared to the non-smokers. The results indicate that smoking does not significantly affect the FEV₁, FVC, PEF and FEV₁/FVC% values for male students in the 19 to 25 years age group. Out of the 43 female students, 15(35%) were smokers and 28(65%) were non smokers.

Table 1: Descriptive statistics of the Pulmonary Function Indices for male smokers and non smokers

<table>
<thead>
<tr>
<th>Parameters</th>
<th>FEV₁ (Litres)</th>
<th>FVC (Litres)</th>
<th>PEF (Litres/min)</th>
<th>FEV₁/FVC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Smokers</td>
<td>Non smokers</td>
<td>Smokers</td>
<td>Non smokers</td>
</tr>
<tr>
<td>Mean</td>
<td>3.71</td>
<td>3.69</td>
<td>4.18</td>
<td>4.17</td>
</tr>
<tr>
<td>SD</td>
<td>0.44</td>
<td>0.43</td>
<td>0.59</td>
<td>0.51</td>
</tr>
<tr>
<td>Range</td>
<td>2.87-4.51</td>
<td>2.96-4.39</td>
<td>3.23-5.52</td>
<td>3.29-5.12</td>
</tr>
<tr>
<td>95% CI</td>
<td>3.48-3.95</td>
<td>3.48-3.90</td>
<td>3.86-4.49</td>
<td>3.92-4.43</td>
</tr>
</tbody>
</table>

95% CI: 95% Confidence Interval; IQR: Interquartile Range
Table 2: Descriptive statistics of the Pulmonary Function Indices for female smokers and non smokers

<table>
<thead>
<tr>
<th>Parameters</th>
<th>FEV₁ (Litres)</th>
<th>FVC (Litres)</th>
<th>PEF (Litres/min)</th>
<th>FEV₁/FVC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Smokers</td>
<td>Non smokers</td>
<td>Smokers</td>
<td>Non smokers</td>
</tr>
<tr>
<td>Mean</td>
<td>2.92</td>
<td>2.91</td>
<td>3.32</td>
<td>3.21</td>
</tr>
<tr>
<td>SD</td>
<td>0.44</td>
<td>0.36</td>
<td>0.67</td>
<td>0.44</td>
</tr>
<tr>
<td>Range</td>
<td>2.43-3.79</td>
<td>2.35-3.61</td>
<td>2.64-4.81</td>
<td>2.56-4.1</td>
</tr>
<tr>
<td>95% CI</td>
<td>2.68-3.16</td>
<td>2.77-3.05</td>
<td>2.95-3.69</td>
<td>3.04-3.38</td>
</tr>
</tbody>
</table>

Table 3: Duration of smoking habit among the male and female smokers

<table>
<thead>
<tr>
<th>Duration of smoking</th>
<th>Male Students (n =16)</th>
<th>Female students (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 6 months</td>
<td>4 (25%)</td>
<td>2 (13%)</td>
</tr>
<tr>
<td>1-2 years</td>
<td>5 (31%)</td>
<td>3 (20%)</td>
</tr>
<tr>
<td>3-6 years</td>
<td>4 (25%)</td>
<td>9 (60%)</td>
</tr>
<tr>
<td>&gt;6 years</td>
<td>3 (19%)</td>
<td>1 (7%)</td>
</tr>
</tbody>
</table>

Table 4: Frequency of smoking by the male and female smokers

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Male students (n=16)</th>
<th>Female students (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Everyday</td>
<td>11 (69%)</td>
<td>8 (53%)</td>
</tr>
<tr>
<td>Every other day</td>
<td>2 (13%)</td>
<td>2 (13%)</td>
</tr>
<tr>
<td>Once/week</td>
<td>2 (13%)</td>
<td>2 (13%)</td>
</tr>
<tr>
<td>Once/month</td>
<td>0</td>
<td>2 (13%)</td>
</tr>
<tr>
<td>Once or twice/year</td>
<td>1 (6%)</td>
<td>1 (7%)</td>
</tr>
</tbody>
</table>

Table 5: Amount of cigarettes smoked per day by the male and female smokers

<table>
<thead>
<tr>
<th>Amount smoked /day</th>
<th>Males students (n=16)</th>
<th>Female students (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>10 (63%)</td>
<td>7 (47%)</td>
</tr>
<tr>
<td>3-6</td>
<td>5 (31%)</td>
<td>7 (47%)</td>
</tr>
<tr>
<td>&gt;6</td>
<td>1 (6%)</td>
<td>1 (6%)</td>
</tr>
</tbody>
</table>
Table 2 shows the descriptive statistics for FEV₁, FVC, PEF and FEV₁/FVC% for female smokers and non smokers. There were no statistically significant differences (p<0.05) in the spirometry parameters for the female smokers compared to non smokers. The results indicate that smoking does not significantly affect the FEV₁, FVC, PEF and FEV₁/FVC% values for female students in the 19 to 25 years age group.

The data obtained for the male and female smokers, were analysed based on the duration of their smoking habit, their frequency of smoking and the amount of cigarette smoked per day. The results obtained are presented in tables 3, 4 and 5.

The result shows (Table 3) that 56% of the male students compared to 33% of the female students have been smoking for two years or less. More than half of the female smokers have been smoking for 3 to 6 years compared to 25% of the male smokers.

Among the male student smokers 69% have been smoking every day since they started compared to 53% among the female student smokers (Table 4).

Most of the male smokers (63%) have been smoking 1 to 2 cigarettes per day, compared to 47% of the female smokers (Table 5).

Weak non significant linear correlation was obtained between the duration of smoking and FEV₁ (rho = 0.053, p = 0.786) and also FVC (rho = 0.032, p = 0.856). A weak inverse non significant correlation (rho = -0.066, p = 0.712) between duration of smoking and FEV₁/FVC was also obtained.

There was a weak linear non-statistically significant relationship between frequency of smoking and FEV₁ (rho = 0.036, p = 0.081) and FEV₁/FVC (rho = 0.061, p = 0.732). However, an inverse non-statistically significant relationship (rho = -0.036, p = 0.839) was obtained between frequency of smoking and FVC.

Weak linear non-statistically significant correlations were also obtained when the amount of cigarettes smoked per day were compared with FEV₁ (rho = 0.078, p = 0.662), FVC (rho = 0.022, p = 0.90) and FEV₁/FVC (rho = 0.013, p = 0.941).

Inverse non-statistically significant relationships were obtained between the duration of smoking and FEV₁ (rho = -0.041, p = 0.796), FVC (rho = -0.007, p = 0.964) and FEV₁/FVC (rho = -0.134, p = 0.392) for the female smokers.

For the female smokers correlation coefficients showed linear non-significant relationship between the amount of cigarettes smoked per day and the FEV₁ (rho = -0.086, p = 0.584), FVC (rho = -0.056, p = 0.724) and FEV₁/FVC (rho = -0.108, p = 0.489).

Weak linear non-significant relationship was obtained between amount of cigarette smoked per day and FEV₁ (rho = 0.020, p = 0.901) and also FVC (rho = 0.056, p = 0.721). The relationship between amount of cigarette smoking per day and FEV₁/FVC (rho = -0.086, p = 0.584) showed a weak linear non-significant relationship.
smoked per day and FEV₁/FVC was weak inverse and non-statistically significant (rho = -0.180, p = 0.249) for the female smokers. When the male smokers were compared to the female smokers, FEV₁, FVC and PEF for the male students were significantly higher (p=0.001) than the corresponding mean values for the female students. There was no statistically significant (p<0.05) difference between the mean FEV₁/FVC% for the male and female student smokers. The mean FEV₁, FVC and PEF values for the male non smokers were significantly higher (p=0.001) than the corresponding mean values for the female non smokers. There was, however, no statistically significant (p<0.05) difference between the mean FEV₁/FVC% for the male and female students that do not smoke.

DISCUSSION:
According to Celli [11] FEV₁ in non smokers with no respiratory illness will start to decline by 25 to 30 mls per year starting at ages 25 to 30 years. People who smoke have a steeper decline than non smokers and heavy smokers have steeper decline compared to light smokers. The number cigarette smoked and the frequency of smoking is both important factors [11]. Thus age is correlated with the number of cigarettes smoked as well as number of cigarettes smoked per day [11]. In our study, it was shown that the FEV₁, FVC and PEF in male smokers were significantly greater than that of the female smokers (p=0.001). A study of young people in NCD in 1997 revealed that 90% of males and 63% of females smoked [12]. According to the WHO profile in 2014, Papua New Guinea has a current tobacco smoking prevalence of 41%, with more adult male smokers (55%) compared to adult females (27%) [13]. However, there is still no known operational policy, strategy or action plan to reduce the burden of tobacco use in the country.
In our study, comparison of smokers and non-smokers revealed no statistically significant differences in spirometry data for both male and female students. The age range in our study was narrower and younger than that of Cheng (25-55years), Yanga (18-40 years), Zhong (40 years and older), Zielinski (39 years and older) and Kim (18 years and older) [5,3,1,14,2]. According to Celli [11], the FEV₁ starts to decline at ages 25 to 30 years which is much older than our study population. This may be the explanation for the non-significant changes observed in our present study.
Gold et al found that adolescents (10-18years) who smoked developed mild airway obstruction and slowed growth of lung function and girls were more vulnerable than boys with regards to the effects of smoking on lung function growth [15]. Apostol et al [16] stated that starting to smoke at a very early age is associated with a
faster decrease in FEV₁. In our study most of the students started smoking between 1 to 6 years prior to the study which is still at a much later age than 10 – 18 years. Datta and Yanga noted that smoking for more than two years but less than five years did not show any changes in lung function [3]. Zhong et al did spirometry tests on selected urban and rural populations in China and reported a prevalence of COPD of 8.2% [1]. Smoking was a risk factor in two-thirds of those patients with COPD and the risk for COPD increased with the number of cigarettes smoked [1]. Kim et al did a nationwide spirometry study on selected population in Korea [2]. They found that 7.8% of adults over 18 years had lowered lung function indices suggesting airflow obstruction. However, after age 45 years, airflow obstruction prevalence increased with increasing age and was higher in men than women. They also found that airflow obstruction was higher in smokers compared to non smokers in subjects who were 45 years or older [2].

This suggests that the effects of smoking on lung function in our present study population may become more pronounced with time. A follow up study of these students in later years may identify changes in their lung function as a result of smoking.

CONCLUSION:
The results from this study showed no current statistically significant effects of smoking on the lung function of students aged 19 to 25 years in Port Moresby.

ACKNOWLEDGEMENTS:
We thank the School of Medicine & Health Sciences for the research grant used in this project. We also thank Associate Professor Victor Temple for the invaluable input into this project. We are grateful to Mr Sam Grant for his technical assistance and to all those students from Port Moresby Business College, Don Bosco Technological Institute and the University of Papua New Guinea for their participation in this study. Many thanks to Theresa Dunamb and Jennie Bautau-Grant in Basic Medical Sciences, SMHS, UPNG.

REFERENCES:
A CASE REPORT

PROSTHETIC REHABILITATION OF AN ORBITAL AND PERIORBITAL DEFECT:
A CASE REPORT

*Hegde Chethan, D Krishna Prasad and Shetty Ganaraj

Department of Prosthodontics and Crown and Bridge, A. B Shetty Memorial Institute of Dental Sciences, Nitte University, Deralakatte Mangalore Karnataka; India 575018

*Correspondence Author: drganaraj@gmail.com

ABSTRACT:
Losing an eye can be a fatal experience for a patient. The deformed appearance of the face resulting after an oncosurgery may result in psychological trauma as well as social embarrassment for the patient. It is a challenge to manage the defect on the face with surgery. Maxillofacial Prosthodontist can fabricate and rehabilitate defects with orbital and periorbital prosthesis. The defect can be restored with custom made orbital prosthesis consisting of orbital globe made up of heat cured acrylic resin and periorbital prosthesis made up of silicon elastomeric material, which can give real life like appearance and also improves the quality of life of the patient. The patient can feel more comfortable and accepted in the social circle. A multidisciplinary approach and team management are essential in providing more accurate and effective rehabilitation of such defects. This case report presents the fabrication of a custom made orbital and periorbital prosthesis for a patient.

Key words: Exenteration, Oncosurgery, Maxillofacial rehabilitation.

Submitted: March 2014, Accepted: June 2014

INTRODUCTION:
Orbital exenteration is the term given to a surgical procedure consisting of removal of entire orbital content including part or all of eyelids and periorbital tissue. The term orbital exenteration was first described by George Bartischcin in 1583 [1]. This surgical procedure is mainly done to treat unyielding progressive and life threatening malignancies, which is not responding to any other treatment modalities like medications and chemotherapy [1].
After the surgery, a large defect with a huge empty space is visible on the face [3]. Even though such defects can be prosthetically rehabilitated, the vision of the eye can never be restored, but the prosthesis which gives life like effect and looks more natural can be fabricated [1]. Fabrication of orbital and periorbital prosthesis is one of the challenging and most difficult procedures for prosthetist. The custom made prosthesis, should be more accurate and correctly match the contralateral eye; this ameliorates the patient’s self-acceptance [4].

This case report describes fabrication of a custom made orbital and periorbital prosthesis used to rehabilitate a facial defect of a patient who has undergone an orbital exenteration of the left eye. The most economical materials available, the heat cure acrylic resin was used to fabricate orbital prosthesis and medical graded silicon elastomeric material was used to fabricate the periorbital prosthesis [2].

CASE REPORT:
A 42-year-old male patient, reported to the department of prosthodontics department in our dental hospital with chief complaint of missing left eye and wanted it to be replaced. The case history reviled that the patient was diagnosed with inverted papilloma and squamous cell carcinoma of left eye and maxilla. The patient was treated surgically; excision of the left eye was performed followed by radiation therapy for 3 months Fig 1a.

After evaluating the case history informed consent was obtained from the patient, the anophthalmic socket and defect region was inspected, palpated and evaluated. The treatment was planned to fabricate a custom made orbital and periorbital prosthesis.

An impression compound was molded and adapted mediolaterally and superoinferiorly over the orbital and periorbital defect to make custom made impression tray Fig 1b. The area of the defect was lubricated with petroleum jelly and the impression was made using Irreversible hydrocolloid impression material (Neocolloid Alginate Impression Material). Subsequently, beading and boxing of an impression was made.

Cast was poured in two sections, where first half of sectional cast was poured with dental stone type 2 and orientation holes were made on the cast to maintain the exact orientation and placement of second poured cast Fig 1c. The second cast was poured with type 4 dental stone (Pearl Stone) Fig 1d.
Orientation of ocular prosthesis:
The two halves of cast were separated and the molten wax was poured into the orbital mold space, after the wax was set, the wax model similar to the contour and size of the orbital globe was retrieved from the cast and was carved. This wax pattern was transferred to the patient’s anophthalmic area; patient was instructed to look straight into observer’s eyes. The waxed up orbital globe was adjusted in patient’s orbital defect in accordance with the contralateral eye. When the desired size and position of waxed up orbital globe was obtained, the custom made iris disk with self cured acrylic resin was fabricated and placed onto the waxed up orbital globe model and the conformer was attached onto the iris disk [2]. The predictable iris positioning technique was used to confirm the position of iris so that the iris will exactly match the position iris of contralateral eye [6] Fig 1e.
After the desired position of iris was obtained, the wax model with the iris was then invested in dental plaster type 1, the conformer attached to the iris, was embedded into the plaster such that it prevents the displacement of iris after dewaxing procedure. The empty mold space obtained after the dewaxing procedure was packed with tooth colored heat cured poly methyl metha-acrylate of appropriate shade that matched exactly with sclera of contralateral eye. After polymerization, the heat cured acrylic orbital globe with iris and conformer attached, was retrieved from the flask, trimming of the prosthesis was done using acrylic trimming bur and finishing was done using sand paper and polishing buff was used to polish the prosthesis Fig 2a. The prosthesis was then tried in patient’s anophthalmic defect to check and adjust the prosthesis within the defect.

The next step was painting of the iris disk. The clinician should have an artistic skill and should possess thorough knowledge about color and pigments used to paint the iris disk. In the past, various methods and techniques to paint an artificial iris were described [2]. Though many types of paints and color pigments are
available to paint the iris disk, in the present case acrylic based color pigments and stains were used to paint the iris disk, once the desired color of an iris disk was obtained. The product obtained was then tried in anophalmic socket. The patient was asked to look straight to adjust the orientation and position of an orbital prosthesis to contralateral eye [2].

After the try-in procedure, the prosthesis was removed from the defect, red embroidery floss were then glued on the scleral part of the prosthesis using monopoly, which resembled blood vessels of natural eye [2]. The stone trimming bur was used to create the space for corneal prominence in the mold; the orbital globe was then packed with thin layer of heat cured clear acrylic resin. After polymerization, the prosthesis was retrieved from the flask; it was then properly trimmed and polished. After final finishing procedure of the orbital globe prosthesis, it was placed in the patient’s orbital defect and manipulated into the position corresponding to the contralateral eye Fig 2b.

The periorbital prosthesis was fabricated by using medical graded silicon elastomers, the wax pattern was prepared for fabrication of periorbital prosthesis, the wax sheets were softened, manipulated and placed over the periorbital section of the cast for evaluating the proper position and contour of wax pattern and adjusted to match the periorbital tissue of contralateral eye.

After carving the wax pattern to desired eyelid aperture, the skin texture was established by carving wrinkles and folds found around the contralateral natural eye [7] Fig 2c. Once the desired position, shape and contour of the wax pattern similar to contralateral eye were achieved, the wax pattern was invested in plaster. After dewaxing procedure; the mold space obtained was packed with silicon elastomeric material, adequate amount of medical graded silicone elastomeric material was dispensed (Room temperature vulcanized silicone, Cosmosil) on the glass slab.

The shade matching procedure was carried out in the presence of the patient to match the skin shade. The silicon elastomer material was mixed with intrinsic color pigments, stains and flocking of various shades (Cosmosil) to achieve the exact skin shade of the contralateral side of the face. The silicone material was packed in the empty mold space obtained after dewaxing procedure, the material was subjected to bench cure for 12 hours, after polymerization of silicon elastomeric material the molds were separated; the periorbital prosthesis was retrieved, finishing and polishing was done. When desired intrinsic shade of a skin was obtained, the periorbital prosthesis was tried in patient to check for accuracy of shade and color of the skin on contralateral side of the face, once the desired skin shade was obtained, the extrinsic
stains were applied to do final finishing of the prosthesis [7] Fig 2d.

**Extrinsic staining**

Extrinsic stains (Cosmosil) were painted on periorbital prosthesis to match the skin shade on contralateral side of the face. Dry air was blown over the prosthesis with the help of a dry air syringe to cure the extrinsic stain. Later prosthetic eyelashes were stitched onto the periorbital prosthesis using a natural hair. The prosthesis was tried in patient’s periorbital defect.

Few adjustments and minor modifications were done for better retention and marginal adaptation of the periorbital prosthesis. The patient was told about the limitation and retention aspect of the periorbital prosthesis. Satisfactory retention and stability was achieved by using skin adhesive (Beta Bond, Medical Graded Adhesives) and anatomical and soft tissue undercuts [2]. The patient was instructed to apply the skin adhesive over the defect area and leave it over for 2 minutes, so that the adhesive becomes more transparent and then to place the periorbital prosthesis over the defect. The frame of the eyeglass and anatomical undercuts engaged periorbital prosthesis to gain additional retention and stability Fig 2e. The patient was instructed about the use and follow up care of the orbital and periorbital prosthesis.

**DISCUSSION:**

After the cancer surgery of an eye, the orbital and periorbital defect on face has to be prosthetically replaced, either in the form of stock orbital prosthesis or custom made orbital prosthesis [2]. The difficulties faced during fabrication of custom made orbital prosthesis are; obtaining accurate impression of the defect without any compression or distortion of periorbital tissue, orientation of orbital globe in harmony with the contralateral eye, sculpturing the exact anatomy and position of the periorbital tissue, obtaining a satisfactory shade exactly matching to the skin complexion of contralateral side of the face [4].

Often, such custom-made orbital and periorbital prosthesis provide satisfactory cosmetic and aesthetically improved facial appearance, especially for the patients who lost their orbital structures through disease, oncosurgery, trauma and accident [2]. The most specific and recommended treatment modality for large tumors and malignancies in the head and neck region is by surgical excision, with or without chemotherapy. After surgical removal of orbital content and the periorbital tissue, it has to be restored with orbital and periorbital prosthesis. Hence the prosthodontist plays major role in fabricating and rehabilitating such large defects on face by performing radical maxillofacial oncosurgery [7]. The success of prosthesis and esthetic outcome achieved after the rehabilitation of orbital defect depends mainly
on the total amount of tissue excised during surgery, the availability of the tissue around the defect and also by maintaining the good position, size and contour of the prosthesis with good retention, improved stability, marginal adaptation and fit of the prosthesis to the surrounding tissue [7]. The retention and stability of the prosthesis is an important factor for the prosthesis to look more natural and esthetically pleasant; hence the maxillofacial prosthesis can be retained by various methods of retention, either by using anatomical undercuts, frame of eyeglasses, magnetic devices, adhesives and implants [5]. Although implants can provide better retention and stability of the prosthesis, the reported drawback of implants was high number of failure rates due to the effect of radiation therapy on bone morphology, compromised blood circulation in and around the defect [9]. In addition, expense of implant surgery, cost factor of the implants, long waiting period for proper osseointegration to take place was not tolerated by patient. Another major disadvantage was that due to psychological trauma of undergoing oncosurgery, the patient hardly agrees to undergo another surgery for implant placement. Thus, due to these factors clinicians had no better option rather than using custom made prosthesis for such patients [7]. The custom made periorbital prosthesis can be retained with help of frame of eyeglasses, anatomical undercuts, magnetic devices and skin adhesives. The skin adhesive may degrade and results in reduced strength and bonding property over a long period of time; some skin adhesives have been reported to cause hypersensitive reactions [7]. Although the success rate of implant supported prosthesis is very high, the prosthesis retained with skin adhesives, anatomical and soft tissue undercuts are more successful due to their ease of application and are comparatively less expensive then implant supported prosthesis [3]. Other materials commonly used for fabrication of orbital prosthesis are epoxy resin, metal and light cured materials, ceramics and resilient vinyl copolymer acrylic resin [4]. Silicon elastomeric materials are more commonly used, because they provide better stability and good marginal adaptation, which satisfies patient’s cosmetic and esthetic needs; but the major disadvantage is that the manipulation of silicone requires more complex, advanced and multifaceted techniques which are rather more expensive [8]. The silicone elastomeric material posses’ excellent physical properties with good heat stability and are chemically inert materials, particularly when they are used in fabrication of prosthesis used to restore body parts [8]. Silicon elastomeric material posses soft tissue like consistency, provide additional advantage when they are used to restore the defects in movable soft tissues. Silicon materials are available in various shades provided by manufacturers to give exact shade and texture
of skin which closely simulate and resemble shade of patient's skin complexion. The drawback of the silicon prosthesis is that, in the long term the prosthesis material degrades easily and its additives undergo changes when exposed to moisture, high temperature, UV lights and sunlight, thus creating a need for replacement by a new prosthesis. To overcome these disadvantages newer polymeric materials have been introduced like polyphosphozenes, silicon block polymers, methacryloxypropyl terminated polydimethyl siloxane with enhanced mechanical, chemical and physical properties, such as increased elongation, high edge strength, improved heat stability, good tear strength, chemically inert, low hardness and viscosity for fabrication of maxillofacial prostheses [7]. Custom made prosthesis composed of orbital globe made up of heat cured acrylic resin and periorbital prosthesis made up of silicon elastomeric materials give patient a more lifelike appearance and esthetically improved looks.

CONCLUSION:
A case of orbital exenteration of left eye was managed with orbital and periorbital prosthesis. The prosthesis was made up of two separate parts. The orbital globe was made up of heat cure acrylic resin and the periorbital prosthesis was made up of silicon elastomeric material. The prosthesis was well retained with skin adhesives, additional retention was gained with help of frame of eyeglasses and anatomical undercuts, the patient was well convinced and satisfied with the prosthesis.

REFERENCES:
A CASE REPORT

DENTURE INDUCED INFLAMMATORY HYPERPLASIA – A CASE REPORT

Anjana Mohan Kumar, *Veena KM, Laxmikanth Chatra, Prashanth Shenai, Prasanna Kumar Rao, Rachana V Prabhu, Tashika Kushraj and Prathima Shetty

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Mangalore, Karnataka, India

*Correspondence author: veenaomr@rediffmail.com

Running Title: Epulis fissuratum

ABSTRACT:

Epulis fissuratum is a benign tumor like inflammatory hyperplastic growth which occurs on the mucosa along the borders of an ill fitting full or partial removable denture. If ulcerated, it can mimic oral squamous cell carcinoma. The treatment includes surgical removal of excess fibrous tissue and remodelling or reconstructing the denture suitably, ensuring better adaptability to the ridges. This case report describes a 55 year old male patient with characteristic clinical features of denture induced inflammatory hyperplasia.

Key words: Epulis fissuratum, ill fitting denture, inflammatory hyperplasia

Submitted May 2014; Accepted August 2014

INTRODUCTION:

One of the most common tissue reactions to a chronically ill fitting denture is the occurrence of hyperplasia of tissue along the denture borders [1]. This proliferation may be the result of resorption of alveolar ridge, leading to over extension of the denture borders causing chronic irritation to the oral mucosa in the sulcus area. Characterised by slow development of elongated rolls of tissue in the mucolabial or mucobuccal fold area into which the denture flange conveniently fits and is often asymptomatic unless ulcerations occur in the base of the fold. Epulis fissuratum (EF) can be treated conservatively or surgically based on the size of the lesion [2].

CASE REPORT:

A 55 year old, male patient reported to the outpatient department [OPD] with a complaint of missing tooth in the maxillary front region and desired replacement. The upper right
central incisor was mobile and fell off by itself two months back. He is a partial denture wearer on upper arch and complete denture on lower arch since 5 years. Intra oral examination showed completely edentulous lower arch and partially edentulous upper arch with only two teeth remaining, maxillary right lateral incisor and maxillary left central incisor, which were having grade 1 mobility. A sessile exophytic growth was also seen on the labial mucosa of the mandibular arch in the anterior region and extending symmetrically on either side of the midline [Figure 1a]. The tissue was split longitudinally all along its length forming two folds and the denture fitted comfortably in between the folds [Figure 1b].

Figure 1: A: Exophytic growth in the mandibular anterior region with two folds and a solitary ulcer in the centre. B: The lesion comfortably fits in between the two folds.

Figure 2:- A- Immediately After surgical excision. B- After 7 days
The superficial fold towards the labial mucosa was smaller measuring 1.5 x 0.5 cm and the fold towards the alveolar ridge was larger measuring 2.5 x 1.5 cm in size. The surface of the tissue was smooth and the colour and texture was same as that of the surrounding mucosa. In the centre of the two folds was a solitary ulcer about 1.0mm in diameter. The patient was not aware of the growth or the ulcer. On palpation the tissue was firm and non tender. On the basis of history and clinical examination a provisional diagnosis of denture induced inflammatory fibrous hyperplasia/ EF was made. The patient was instructed to discontinue the use of the denture. As the ulcer was a healing one no treatment was suggested. The patient underwent extraction of the two teeth and excision of the exophytic tissue [Figure 2a and 2b] and fabrication of new dentures. The excised tissue was sent for histopathologic examination.

**DISCUSSION:**

The term epulis, first described by Virchoff, has its origin in Greek language (epi on; oulon gum) describing something appearing on the gingival gumline [2]. EF is a common sequela of wearing ill fitting dentures, characterized by hyperplasia of the mucosa due to contact with denture border [3]. Other names used to describe the lesions are - Denture induced inflammatory fibrous hyperplasia, redundant tissue, denture injury tumor, denture epulis [4].

The lesion has a strong female predilection and is seen in age group of 30-60 years, with a peak incidence in the sixth decade [5]. Majority of lesions are seen in the maxilla than in mandible. Anterior portion of jaws is affected more often than posterior area. The strong female predilection is thought to be due to various factors like more women are denture wearers than men due to cosmetic reasons, they have a longer life span than men and hormonal deficiencies can enhance formation of epulis especially after menopause [6]. It is seen more in the maxilla than the mandible because the area of mucosa covered by a denture is greater in the maxilla than the mandible so the pressure being inserted to the underlying mucosa is higher in maxilla. There are some contradictory results, such as those in the De Baat et al. [5] study that shows that the lesions are more in the mandible than the maxilla.

In the present case the EF is seen in a male patient in the same age group as mentioned above and in the mandibular anterior region. The lesions may be single or numerous composed of flaps of hyperplastic tissue. Presence of inflammation is variable and if present is seen in the bottom of deep fissures. In some cases ulceration may occur. Diagnosis can be made based on the history and clinical examination of the patient. However after surgical excision histopathological examination is mandatory to yield a confirmatory diagnosis.
as there are many lesions that may appear in the area which can have a more serious outcome [7]. Histopathologic feature of epulis fissuratum include excessive bulk of fibrous connective tissue covered by a layer of stratified squamous epithelium [7]. Connective tissue is composed of bundles of collagen fibres, with few fibroblasts or blood vessels unless there an active inflammatory reaction is present. Lesions with almost similar clinical features are pyogenic granulomas, fibromas, peripheral giant cell granulomas, peripheral ossifying fibroma, neurofibroma, oral squamous cell carcinoma [4]. Pyogenic granulomas are purple-red nodular inflammatory hyperplastic lesion usually pedunculated, again seen more commonly in females, on the maxillary anterior region especially on the gingiva due to chronic irritation [8] It bleeds on slightest provocation, but is painless unless ulcerated and has a rapid growth pattern unlike epulis fissuratum and is not associated with denture wearing and also histologic picture shows granulation tissue. Fibromas are common benign soft tissue neoplasms more commonly seen in the buccal mucosa in the line of occlusion, though can be seen on other sites also including gingival [7]. They appear as elevated nodules of normal colour with a smooth surface and a sessile or occasionally pedunculated base. It is a slow growing lesion more common in females seen in the third, fourth and fifth decades. Histologic features include bundles of collagen fibers interspersed with fibroblasts and blood vessels. Here the distinction between hyperplasia and neoplasia may not be very clear cut in all the cases. Peripheral giant cell granuloma/epulis is a reactive lesion seen more commonly in females in the fourth to sixth decade of life occurring in the mandibular gingiva or alveolar process anterior to molars as a sessile or pedunculated mass [9]. Surface has a dark red or vascular appearance and ulcerations may be seen. Histologic appearance is characteristic here, with presence of multinucleated giant cells. In edentulous patients peripheral giant cell granuloma can cause superficial erosion of bone seen as peripheral cuffing in a radiograph. Peripheral ossifying fibroma are focal gingival over growths seen anterior to the molars, in young females [10]. The surface of the lesion smooth and is of the same colour as surrounding mucosa. Characteristic feature seen in the histopathologic examination is the presence of multiple calcifications, which is the differentiating feature of the lesion. Neurofibroma is a benign neoplasm of nerve tissue origin. Oral lesions are rare, but when present, are seen to occur on the buccal mucosa, palate, alveolar ridge, vestibule and tongue, as discrete non ulcerated nodules having same colour as the surrounding mucosa [11]. Histologic features of neurofibroma are considered to be virtually diagnostic with myxomatous peripheral nerve tissue within the
perineural sheath scattered within a collagen rich matrix. Oral squamous cell carcinoma is the most common malignant neoplasm of the oral cavity occurring at any intra oral site [12]. It can be seen as rapidly growing mass with ulcerations and indurations of the margins, affecting men more commonly than women. Based on histologic findings it can be well differentiated, moderately differentiated and poorly differentiated. Treatment includes conservative or surgical management depending on the duration and size of the lesion [13]. Conservative management includes repairing the denture, relining it or fabricating a new denture if it is ill fitting. And surgical managements include the use of surgical scalpel, the electro surgery or laser techniques (a carbon dioxide laser, Erbium:YAG laser, Neodymium-YAG laser, or diode laser) [2].Prognosis is usually good as long as the causative factor is removed successfully.

CONCLUSION:

Epulis fissuratum is a common lesion seen in elderly people associated with chronic trauma due to ill fitting dentures. Hence care should be taken while fabricating dentures and frequent review should be done to check for ridge resorption. Proper hygiene of the denture should be maintained by the patient. Surgical excision and biopsy of the tissue is recommended to rule out the other pathologies

REFERENCES:

CASE REPORT

DENTOALVOLAR ABSCESS WITH EXTRA ORAL SINUS IN A PEDIATRIC PATIENT:
A CASE REPORT

Nikhilraj, *Prashanth Shenai, Laxmikanth Chatra, Veena KM, Prasanna Kumar Rao,
Rachana V Prabhu, Tashika Kushraj, Pratima Shetty and Shaul Hameed

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University,
Mangalore, Karnataka, India

*Correspondence author: prashanthshenoy4@gmail.com

Running title: Dentoalvolar abscess with extra oral sinus

ABSTRACT:
Cutaneous sin tract of dental origin are often misdiagnosed and inappropriately treated because of their uncommon occurrence and absence of symptoms in about half of the patients. A case report describing the diagnosis and treatment of an extra oral cutaneous sinus tract of odontogenic origin in relation to mandibular left first molar with surgical treatment and proper antibiotic coverage is presented.

Key words: dentoalvolar abscess, sinus tract, periapical region
Submitted June 2014; Accepted October 2014

INTRODUCTION:
In the paediatric patient, dental abscesses are not uncommon. Even though the decline in dental caries in past decades, many young children are still at risk for dental decay, and pulpal infections from caries in primary teeth have been reported [1]. In addition, dental abscesses resulting from trauma are also encountered in young children. In most children, these infections usually present as chronic inflammation, which are localized to the offending tooth. In such cases, management of localized pulpal infections in the primary dentition includes root canal treatment or extraction and space maintenance [1]. On the other hand, the treatment of a spreading, acute dental abscess centres on pain control, antibiotics, surgical drainage and removal of
the source of infection, which may include endodontic treatment or extraction of the tooth. Sinus track is defined as the channel leading from the enclosed area of the inflammation on the epithelial surface. Opening of sinus tract may be located either intraorally or extra orally [2]. Cutaneous sinus tract of dental origin is uncommon. Even though they have been well documented in medical and dental literature the lesions continue to be misdiagnosed challenging and stance a diagnostic dilemma [2, 3]. Studies indicate that extra oral sinus tract is most commonly found in cheek, chin and angle of the mandible [4]. Most commonly the ethology of odontogenic sinus tract involves chronic periradicular abscess that arises from bacterial invasion and chemical irritation or trauma [5]. Here we report a case of dentoalvolar abscess with extra oral sinus of an 11 year old female patient.

CASE REPORT:
An 11 year old female patient reported to the dental hospital with a complaint of ulcerative area on the left region of the lower jaw since one week. The ulcer was painful and associated with purulent discharge since 2 days, fever since 3 days with sleep disturbance. She was given medication for the pain and fever. On presentation, extra oral inspection showed diffused ulceration below the border of the mandible measuring 2 × 2 cm in diameter, extended anteriorly 3 cm short of the chin and posteriorly 4 cm short form angle of the mandible. The colour of the lesion was brown and borders appear to be rough with erythematous surrounding skin pus discharge seen from the ulcer; no other ulcerated area was seen elsewhere on the face (Fig. 1). On palpation the lesion was tender and not fixed to the underline structures, paraesthesia was also not present. Intra oral inspections showed grossly decayed 36 with obliteration of buccal vestibule (Fig. 2). Based on the history and clinical examination a provisional diagnosis of dentoalvolar abscess with extra oral sinus was made. Intra oral radiograph (Fig. 3) showed coronal radiolucency involving enamel and dentin and approaching pulp periapically diffused radiolucency with ill-defined borders. Based on the radiographic findings a final diagnosis of dentoalvolar abscess with extra oral sinus was made.

The treatment plan was incisional and drainage of extra-oral sinus with extraction of 36. The treatment done was prescribed. Initial management included, antibiotic coverage, Amoxicillin paediatric 250 mg thrice daily (TID) Flagyl 200mg TID and Ibugesic paediatric TID on the first visit. After the infecting subsided, the extraction of 36 (Fig4) was done with debridement of extra oral sinus with suturing. The patient was recalled after a week for suture removal with application of betadine ointment and Neosporin powder. On the fourth visit the lesion site showed complete healing.
DISCUSSION:
Chronic dentoalvolar abscess is long standing of low grade infection of periradicular tissue result from acute pulpitis or acute non-suppurative periodontitis or acute exacerbation of periapical granuloma, cyst or abscess [5]. A dentoalvolar abscess may be initiated by caries, periodontal disease, trauma, or thermal and chemical injuries. An intra–oral or extra–oral sinus can develop, depending on the path of the inflammation, which is dictated by surrounding muscular attachment and facial
planes [6, 7]. The site of dental sinuses is usually anatomically close to the causative tooth. Occasionally, the opening of the sinus tract may be found at a far distance from the dental infection, which makes the diagnosis challenging, especially with respect to intact teeth. It is usually a non–vital tooth, but in edentulous patients, it could be a retained tooth fragment, an impacted tooth, or an odontogenic cyst. On the basis of clinical appearance the differential diagnosis includes pustules, actinomycosis, osteomyelitis, pyogenic granulomas, furuncles neoplasms; squamous cell carcinomas, epidermal cyst [8, 9].

In this present case the apparent cause of sinus formation was the pulp necrosis of 36, which was grossly decayed with complete loss of crown structure except the lingual wall. The major management guidelines for the treatment of a sinus include draining the pus and removing the source of infection. Antibiotics may be used as an adjunct to conventional treatment; when a drainage cannot be established immediately, if the pus has spread to the superficial soft tissues or when the patient is in the setting of diabetes, immunosuppression, or systemic signs of infections such as fever. Antibiotic therapy alone may not be effective in these cases, because of the absence of adequate circulation in a necrotic pulp system and abscess. If antibiotics are to be used, penicillin V potassium is the first choice. Clindamycin or amoxicillin-clavulanate may be used if the infection is unresponsive [10]. In penicillin hypertensive patient’s erythromycin and metronidazole can be given, as most of the infections are caused by obligate aerobes. Recognition of the true nature of the lesion facilitate as the quick treatment, it minimize the patient discomfort and aesthetic problems and reduce the possibilities of developing further complications prominently.

CONCLUSION:
The eradication of the dental source of infection invariably terminates suppuration, establishment of healing and resolution of the cutaneous lesion. Communication between the dentist and the physician is suggested to provide timely acknowledgement and treatment of rare cases.

REFERENCE:


8. Wood G. Pits, Fistula, and draining lesions (H. M. cherrick, chaptered.):


CASE REPORT

ORAL MUCOCELE TREATED USING DIODE LASER: A CASE REPORT


Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Deralakatte, Mangalore, Karnataka, India

*Correspondence author: drdeepsts@gmail.com

Running title: Oral Mucocele

ABSTRACT:
The mucocele is a salivary gland pathology that results from rupture of salivary gland duct and spillage of mucin into the surrounding tissues. The term mucous extravacation is also used to describe this lesion. The rupture of the gland or duct may be due to local trauma. The most common site of mucocele is lower lip. The treatment of mucocele includes cryosurgery, intra-lesional corticosteroid injection, micro-marsupialization, marsupialization of the mucocele, conventional surgical removal of the lesion, and laser ablation. The advantages of laser ablation over other methods include less treatment time, avoidance of suturing, minimal complications and relapse. Here we report a case of mucocele on lower lip treated using diode laser.

Key words: Mucocele, Diode laser, Nodule

Submitted: July 2014, Accepted: October 2014

INTRODUCTION:
The term “Mucocele” (from Latin terms mucus, or mucus, and coele, or cavity) is used to define the accumulation of mucus secreted from salivary glands and their ducts in the oral cavity’s sub-epithelial tissue [1]. Clinically a mucocele is characterized by increase in volume, with a dome shaped swelling, bluish in color or sometimes of the same color as the surrounding mucosa. Conventional treatment of the mucocele is excision with the associated overlying mucosa and the glandular tissue down to the muscle layer. If the mucocele is merely incised, the contents will drain, but the
lesion will reform as soon as the incision heals. Another treatment option available is excision using lasers. With the advent of high-intensity lasers, this type of lesion may be treated efficiently due to its prompt hemostasis and no need to suture, which reduces surgical time and reduces wound infection [1]. Here we report a case of mucocele treated using diode laser.

CASE REPORT:
A 21 year old female patient reported to the department of oral medicine and radiology with a chief complaint of swelling seen on the lower labial mucosa (figure 1). The swelling was asymptomatic and was not associated with any symptoms. The patient was more concerned about the esthetics. She noticed the swelling before 4 months, which gradually increased in size. General and extra-oral examinations were non-contributory. On intraoral examination a sessile dome shaped nodular swelling measuring approximately 5mm × 5mm was noticed on the lower labial mucosa 2mm below the vermillion border of the lower lip. Color of the swelling was same as that of the adjacent mucosa, with no erythema, pus discharge and ulceration. The incisal edge of the right central and lateral incisors was impinging on the nodule. On palpation the swelling was fluctuant and soft in consistency. Based on the history and clinical appearance the differential diagnosis includes giant cell fibroma, mucocele and papilloma. The lesion was advised for excisional biopsy. The excision was done using 810nm diode laser in a continuous wave mode at a power setting of 4.5W under local anesthesia in minor operation theatre. The patient was advised to wear a safety goggles throughout the surgical procedure to avoid ocular damage. The excision site was bleeding free (figure 2A and 2B) and the procedure was less time consuming with avoidance of suturing. Patient compliance was excellent. Histopathological features include mucin pooled areas with numerous mucinophages surrounded by a fibrous connective tissue wall infiltrated by chronic inflammatory infiltrate predominantly lymphocytes and plasma cells. Overlying epithelium was parakeratinized stratified squamous in nature. Mucous and muscle tissues were also evident. The impression was given as fibroed mucocele. Patient was recalled on seventh and twenty eighth post-operative days and was examined for healing and pain. Seventh post-operative day the Visual analogue scale (VAS) pain score was 1 and showed good healing with no connective tissue exposed and no bleeding on palpation (figure 3A). On the twenty eighth day of recall the VAS pain score was 0 and healing was excellent with the mucosa appearing similar in color to that of normal surrounding mucosa, with no granulation tissue and no connective tissue exposed (figure 3B).
Figure 1: Nodular swelling seen on the lower labial mucosa

Figures 2A and 2B: Bleeding free excision site during the procedure

Figures 3A and 3B: Seventh post-operative day and 28th post-operative day
DISCUSSION:
The incidence of mucocele in the general population is 0.4-0.8%, with scant differences between males and females [2]. Two types of mucocele can appear - extravasation and retention. Extravasation mucocele results from a broken salivary glands duct and the consequent spillage into the soft tissues around this gland. Retention mucocele appears due to a decrease or absence of glandular secretion produced by blockage of the salivary gland ducts [3]. When this mucocele is located in floor of the mouth it appears as the underbelly of a frog, so it is called as ranula. These lesions are devoid of epithelial lining and are also termed as: Superficial mucocele, Classical mucocele. Superficial mucoceles are located under the mucous membrane and classical mucoceles are seen in the upper submucosa [4]. The literature describes different treatment options for mucocele, including cryosurgery, intrallesional corticosteroid injection, micromarsupialization, marsupialization of the mucocele, conventional surgical removal of the lesion, and laser ablation [2]. In comparison with conventional scalpel, laser has many benefits, such as ease of soft tissue ablation, hemostasis, instant sterilization, reduced bacteremia, little wound contraction, reduced edema, minimal scar, reduced mechanical trauma, less operative and post-operative pain, increased patient acceptance, no or few sutures, no need for topical anesthesia [5]. Lasers possess all these excellent properties which help in considering it as a better option in treatment of mucocele.

The word laser is an acronym for light amplification by stimulated emission of radiation [6]. The application of lasers in dentistry includes incisional and excisional biopsy, management of tongue lesions, white lesions, vesiculobullous lesions, malignant lesions, treatment of salivary gland pathologies mainly mucocele and ranula, herpetic lesions, aphthous ulcers, frenectomy, gingivoplasty, crown lengthening, pre prosthetic surgery, implant exposure, hypersensitivity, bony surgeries [7]. Diode laser is an excellent soft tissue surgical laser indicated for cutting and coagulating gingiva and mucosa and for soft tissue curettage or sulcular debridement. Care must be taken when using the continuous emission mode because of the rapid thermal increase in the target tissue. The chief advantage of the diode lasers is the use of a smaller size instrument. The units are portable and compact and are easily moved with minimum setup time and are the lowest priced lasers currently available [6]. The diode lasers have been effective in treatment of mucocele since minimum use of anesthesia, less bleeding, no scarring, no postoperative discomfort, more patient acceptance, and most importantly it is precise and provides a
sterilized field. Thus diode lasers can be considered in treatment of mucocele [8]. Appropriate protective eyewear for the patient and the entire surgical team must be worn when the laser is operating so that any reflected energy does no damage. The surgical environment must have a warning sign and limited access. High volume suction must be used to evacuate the plume formed by tissue ablation. The laser itself must be in good working order so that the manufacturer’s safeguards prevent accidental laser exposure. Masks and gloves must be worn by the operator [6].

In our case the diode laser provided with an excellent patient compliance, less operating time, less bleeding, no suture, less post operative pain, excellent healing and no scar formation.

CONCLUSION:

Mucocele is a salivary gland pathology which requires proper care and management as the recurrence rate is high. The properties of diode lasers make it more effective in the treatment of mucocele. Instructions given by the manufacturer should be strictly followed to avoid complications during and after surgical procedures.

REFERENCES:

CASE REPORT

RADICULAR CYST: A CASE REPORT

Anjali S, *Prashanth Shenai, Laxmikanth Chatra, Veena KM, Prasanna Kumar Rao, Rachana V Prabhu, Tashika Kushraj, Prathima Shetty and Shaul Hameed

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Mangalore, Karnataka, India

*Correspondence author: prashanthshenai4@gmail.com

Running title: Radicular Cyst

ABSTRACT:
Radicular cysts are the most common inflammatory odontogenic cystic lesions. It usually originates as a sequel to a periapical inflammatory process, following chemical, physical or bacterial injury. Due to its chronic etiology, the cyst usually appears towards the later stage of life. It has a male predilection. The maxillary anterior region is the most common site of involvement. This case report presents the clinical features, radiographic features and management of radicular cyst.

Key words: radicular cyst, odontogenic cyst, maxillary anterior
Submitted: July 2014; Accepted: October 2014

INTRODUCTION:
Radicular cysts are the most common inflammatory odontogenic cystic lesions [1]. It originates from the epithelial cell rests of the Malassez, periodontal ligament or of the surrounding bone, secondary to inflammation [2]. Around 60% of all jaw cysts are radicular cysts. The cyst is most common in third and fifth decade of life [3]. These cysts are considered to be rare in primary dentition [4]. The radicular cyst commonly shows a male predilection with maxillary anterior region as its prevalent site of involvement. Radicular cysts have been regularly associated with carious, non-vital teeth or teeth with a history of trauma. Radicular cysts can heal spontaneously after root canal treatment or extraction. However, some authors propose that suspected radicular cysts must be totally enucleated surgically to remove all epithelial remnants [5].
Here we report a case of large radicular cyst in the maxillary anterior region in a 35 year old female patient. Ethical clearance was obtained from the University Ethical Committee.

**CASE REPORT:**
A 35 year old medically fit female patient reported to the department of Oral Medicine and Radiology with a chief complaint of pain on the upper left front teeth region since fifteen days. Patient gave history of swelling on the hard palate since fifteen years. Swelling was initially small in size which increased to the current size a month ago (Figure 1). She had difficulty in speech for the past one month. Patient had a fall and blunt trauma to the upper lip about twenty years ago. No obvious swelling or facial asymmetry was noted on extra oral examination. No sinus or fistula was evident extra-orally. Regional lymph nodes were non-enlarged and non-palpable. A diffuse, soft, swelling was noted on the hard palate extending from maxillary right first premolar to maxillary left first premolar which was approximately 4 x 4cm in diameter. Anteriorly it extends from rugae and posteriorly up to anterior two-third of hard palate. Mucosa over the swelling appears stretched. Dull pain was elicited on palpation. Swelling was fluctuant and not fixed to underlying structure. No local rise in temperature, pus discharge and paraesthesia was noted. Maxillary left central incisor was tender on percussion. Gingiva bleeds on probing in relation to maxillary left central incisor. Maxillary left central incisor was discoloured. All teeth were vital on electric pulp vitality testing except maxillary left central incisor.

A maxillary anterior occlusal radiograph was taken which showed well circumscribed unilocular radiolucency involving the apex of maxillary right incisors and maxillary left incisors and canine, with well defined, radio opaque sclerotic borders (Figure 2). Aspirational biopsy was done which revealed straw coloured fluid. On histopathological examinations presence of shiny cholesterol crystals were identified suggestive of radicular cyst. Based on clinical, radiologic and histopathologic examination, a diagnosis of radicular cyst was given. The patient was advised to go for endodontic treatment followed by surgical enucleation and referred to concerned department.

**DISCUSSION:**
Odontogenic cysts constitute frequent benign lesions of the jaw bones, due to the ubiquitous presence of epithelial rests after odontogenesis. Radicular cysts appear as the most common of all odontogenic cysts, with an incidence around 50% [6, 7, 8]. It is also known as periapical cyst, apical periodontal cyst, root end cyst or dental cyst. Radicular cyst commonly occurs in the maxillary anterior region in the third to fifth decade of life, more commonly in men. In the present case, the radicular cyst was in a female patient.
The pathogenesis of radicular cyst is commonly considered as occurring in three phases: initiation, cyst formation and cyst enlargement [9]. A radicular cyst is one which arises from the epithelial residues in the periodontal ligament as a result of inflammation. The inflammation usually follows the death of dental pulp and cysts arising in this way are found most commonly at the apices of the involved tooth. Most of the radicular cyst are symptomless and are usually discovered during routine radiographic investigations [3]. Pulpal necrosis leading to inflammation appears as the most frequent etiology of the radicular cyst. A lesser known but likely cause of pulpal necrosis is traumatic injury to teeth. In the present case, none of the associated teeth were found to be carious, while only one left maxillary central incisor was found to be non-vital but non-carious. The patient however did report blunt trauma to the upper lip about twenty years ago. No injury or bleeding was reported and no treatment was taken at that time. Thus, significant trauma twenty years ago appears to have initiated the pathology.

Radicular cyst most commonly occurs in maxilla. It may be due to the spongy nature of the maxillary bone and reluctance to extract anterior teeth, the over retention of which leads to cyst formation. The initial swellings of these radicular cysts are usually bony hard, but as they increase in size, the covering bone may become very thin despite initial sub-periosteal bone deposition. Finally, with progressive bone resorption, the swellings exhibit ‘springiness’ or ‘egg shell crackling’. Differential diagnosis of adenomatoid odontogenic tumor (AOT) and traumatic bone cyst can be given.

AOT shows maxillary swelling. The AOT is a benign, non-neoplastic (hamartomatous) lesion.
with a slow progressing growth. The tumor has three clinicopathologic variants, namely, intra-osseous follicular, intra-osseous extra follicular and peripheral [10]. The extra follicular type (24%) has no relation with an impacted tooth, whereas follicular type (73% of all AOT cases) is associated with an unerupted tooth [10]. The peripheral variant (3%) is attached to the gingival structures. Follicular and extra follicular types are more common in the maxilla than in the mandible, and most of the tumors involve anterior aspect of anterior maxilla. There is a slight female over male predilection, almost 2:1. Radiographically, they usually appear unilocular and may contain fine calcifications, and irregular root resorption is rare [10].

The traumatic bone cyst (TBC) is an uncommon non-epithelial lined cavity of the jaws. TBC occurs most commonly during childhood and adolescence, usually in the second decade of life. Some reports [10] suggest that males are affected more often than females (3:2). In the maxillofacial region, most TBCs occur in the mandible, rarely the maxilla have been reported. Expansion of the cortical plate of the jaw bone is often noted, usually buccally, resulting in intraoral and extraoral swelling and seldom causing deformity of the face.

On radiological examination, a traumatic bone cyst usually appears as a unilocular radiolucent area with an irregular but well defined (or partly well defined) outline, with or without sclerotic lining around the periphery of the lesion [11].

Several treatment options are available for a radicular cyst which includes surgical and nonsurgical method. Surgical methods include Enucleation and Marsupilization. Enucleation procedure is usually indicated for a small cyst, which can be done when the vital structures are not involved [12]. Combined approach reduces morbidity and hastens complete healing of the defect [12]. In this technique marsupialization is done first and the enucleation is done at a later date.

Non-surgical methods include conservative endodontic treatment, decompression technique, active nonsurgical decompression technique, aspiration and irrigation technique, method using calcium hydroxide, lesion sterilization and repair therapy, and apexum procedure [13]. Other methods under research are the use of Simvastatin and Epigallocatechin [13]. In our case, root canal treatment followed by surgical enucleation was done.

CONCLUSION:
Radicular cyst is a common condition found in the oral cavity. However, it usually goes unnoticed and rarely exceeds the palpable dimension. In the present case the clinical features were examined, investigations carried out and successful management of a radicular cyst was achieved.
REFERENCES:


CASE REPORT

CAPDEPONT'S TEETH: A CASE REPORT

Kota Sravani, *Prasanna Kumar Rao, Laxmikanth Chatra, Prashanth Shenai, Veena KM, Rachana V Prabhu, Tashika Kushraj, Pratima Shetty and Shaul Hameed

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Mangalore, Karnataka, India

*Correspondence author: drjpkrao@gmail.com

Running title: Dentinogenesis imperfect

ABSTRACT:

Dentinogenesis imperfecta is an autosomal dominant disorder of tooth development characterized by the presence of opalescent dentine, resulting in a dusky blue to brownish discoloration of the teeth. It is the most common dental genetic disease. This condition is genetically and clinically heterogeneous, it may affect only the teeth or it may be associated with the osteogenesis imperfecta. Diagnosis is based on history, clinical examination and radiographic features. This report describes an 18 year old male patient who showed the characteristic dental features of dentinogenesis imperfecta.

Key words: Dentinogenesis imperfecta, Amelogenesis Imperfecta, Dentine Dysplasia.

Submitted: August 2014; Accepted: November 2014

INTRODUCTION:

The teeth which are regarded as the hardest structures of our body are made up of enamel the outermost covering, dentine the middle layer and the pulp which consists of nerves and blood vessels. Tooth development like the development of all epithelial appendages is regulated by inductive tissue interactions between epithelium and mesenchyme. Numerous genes interact, either act in conjunction or antagonize each other in odontogenesis [1, 2]. Certain genes involved in enamel and dentine structures are highly specific for tooth. Mutations in these genes have been identified as causes of Amelogenesis Imperfecta (AI), Dentinogenesis Imperfecta (DGI), Dentine Dysplasia (DD) and anomalies in tooth number [1]. Dentinogenesis
imperfecta is also known as Capdepont’s teeth, hereditary opalescent dentin, Brown teeth. It is a genetic disease transmitted as an autosomal dominant trait, and characterized by disturbance in dentin formation [3, 4]. The condition was first described by Barret in 1882 [4]. The term Dentinogenesis imperfecta was coined by Robert and Schour in 1939 [1]. The affected teeth have opalescent, amber color and darken with age and exhibits attrition of incisal and occlusal surfaces [2]. Our present case report is about an 18 year old patient who reported to the department with the chief complaint of discoloration in the anterior teeth.

CASE REPORT:
An 18 year old male patient reported to the department of Oral medicine and radiology, with the chief complaint of discoloration of his anterior teeth (Figure 1). There was a history of similar discoloration of the deciduous teeth which were exfoliated uneventfully. The permanent teeth which erupted were brown in color at the time of eruption. The same type of discoloration was seen in patient’s siblings. On intra oral examination the maxillary and the mandibular anterior teeth were brownish in color with mild upper anterior crowding. The incisors were a darker shade of brown when compared to the other teeth. The incisal and occlusal surfaces of all the teeth were attrited (Figures 2 & 3). Both the upper first maxillary molars were decayed with caries present on the occlusal surface. The mandibular first molars and the premolar on the lower right side were found to be missing. The patient was further subjected to radiographic investigations. Orthopantomograph showed normal anatomical landmarks with full complement of upper teeth and partially edentulous mandible. Generalized cervical constriction of all the teeth was noticed with obliteration of pulp chambers in some suggestive of Dentinogenesis imperfecta. Radiolucency involving enamel and dentin and approximating the pulp was seen in respect to upper first maxillary molars suggestive of dental caries (Figure 4).

DISCUSSION:
The classification of hereditary dentine disorders is currently complicated. The most familiar classification system is that formulated by Shields in 1973 [3]. This categorization discriminates three types of dentinogenesis imperfecta and two types of dentine dysplasia [3].The Shields' system is increasingly out of date as it does not account for the molecular etiologies of the hereditary dentine defect elucidated so far [4]. The genetic defects that have been discovered to date are insufficient to allow for the construction of a comprehensive classification based on the knowledge of the underlying mutations. Shield classified dentinogenesis imperfecta into three types based on clinical and radiographic features [3].
Dentinogenesis imperfecta type I: Individuals with DGI-I also have osteogenesis imperfecta. The teeth of both dentitions are typically amber and translucent and show significant attrition. Radiographically, the teeth have short, constricted roots and dentine hypertrophy leading to pulpal obliteration either before or just after eruption. Expressivity is variable even within an individual, with some teeth showing total pulpal obliteration while in others the dentine appears normal [5].

Dentinogenesis imperfecta type II: The dental features of DGI-II are similar to those of DGI-I but penetrance is virtually complete and osteogenesis imperfecta is not a feature. Bulbous crowns are a typical feature with marked cervical constriction. Normal teeth are never found in DGI-II. Short stature and blue sclera are extra oral features which may be seen in individuals affected. Sensorineural hearing loss has also been reported as a rare feature of the condition [6].
Dentinogenesis imperfecta type III: This is a form of DGI found in a tri-racial population from Maryland and Washington DC known as the Brandywine isolate. The clinical features are variable and resemble those seen in DGI-I and -II but the primary teeth show multiple pulp exposures and radiographically, they often manifest “shell” teeth i.e. teeth which appear hollow due to hypotrophy of the dentin.

The present case comes under type II in the Shield’s classification and there was absence of blue sclera. The conditions that have similar clinical or radiographic features to DGI need to be considered to give a correct diagnosis. Some of the conditions may mimic the appearance of DGI either clinically or radiographically. Hypocalcified forms of Amelogenesis imperfecta initially develop normal enamel thickness but the poorly calcified enamel is soft and friable and is rapidly lost by attrition leaving dentine cores. But unlike DGI the teeth are usually sensitive and on radiographs enamel is less radio-dense than dentine [7]. Pulp chamber and root canals are usually not sclerosed.

Congenital erythropoietic porphyria is a condition resulting from an inborn error of porphyrin metabolism. This deficiency leads to hemolytic anemia, photosensitivity, blistering of the skin, and deposition of red-brown pigments in the bones and teeth [8]. In case of Rhesus incompatibility, the discoloration ranges from yellow through to green, brown and grey to black is usually found at the necks of teeth and the enamel hypoplasia’s are usually located in the coronal third of the teeth [9].

Tetracycline’s have the ability to chelate calcium ions and to be incorporated into developing teeth, cartilage and bone, resulting in discoloration of both the primary and permanent dentitions. This permanent discoloration varies from yellow or grey to brown depending on the dose or the type of the drug received in relation to body weight [10].

In the present case discoloration of the anterior teeth was more pronounced than the rest and generalized attrition of teeth was noticed. Other causes of early loss of teeth as in DGI include hypophosphatemia, immunological deficiencies e.g. severe congenital neutropenia (Kostmann’s disease), cyclic neutropenia, Chediak-Hegashi syndrome, neutropenia’s, histiocytosis X, Papillon- Lefevre syndrome and leucocyte adhesion deficiency syndrome [11]. With the exception of hypophosphatasia, all of these conditions have an underlying immunological defect which makes those with these conditions susceptible to periodontal breakdown. Mobility of teeth in those with hypophosphatemia however is due to aplasia or marked hypoplasia of cementum.

Vitamin D-dependent rickets and vitamin D-resistant rickets have clinical and radiographic features of DGI. Vitamin D-dependent rickets is characterized by yellowish to brown enamel, chronic periodontal disease, large quadrangular pulp chambers and short roots. Features of vitamin D-resistant rickets include
attrition and exposure of abnormally formed dentine of primary teeth and abscessed non-carious primary or permanent teeth [11].

The aim of treatment provided would be to restore function, aesthetics and protect posterior teeth from wear and maintain the occlusal vertical dimension.

Treatment varies according to the age of the patient, severity of the problem and the presenting complaint. Modern dental technology and materials have promoted new treatment strategies, including the use of an extended provisional phase to better determine the functional and esthetic aspects of a specific case. In the present case the patient was referred to the department of conservative dentistry for the endodontic treatment of the tooth with caries.

CONCLUSION:

Dentinogenesis imperfecta is the most common autosomal dominant disorder causing discoloration of the teeth which in turn affects the quality of life of an individual. Correct diagnosis and carefully planned management would help to restore not only the function but also the aesthetics there by improving the quality of life of the individual.

REFERENCES:

INSTRUCTIONS FOR AUTHORS:

AIMS AND SCOPE:
Pacific Journal of Medical Sciences is a peer-reviewed, multidisciplinary journal published by the School of Medicine and Health Sciences (SMHS), University of Papua New Guinea (UPNG).

The aim of The Pacific Journal of Medical Sciences is to provide the forum for researchers, medical practitioners and other professionals to record, publish, and share ideas and research findings that serve to enhance the understanding of the aetiology, symptoms, diagnosis, prevention, control and management of human disease conditions world wide.

The Pacific Journal of Medical Sciences publishes original scientific research reports, case reports, short communications, letters to the editor and reviews, representing new and significant findings in all areas of medical, biomedical and health sciences (including epidemiology, public and environmental health). Book reviews, scientific news and conference proceedings are published on special request.

EDITORIAL POLICIES:
The Pacific Journal of Medical Sciences (Pac. J. Med. Sci.) editorial policies require that: All manuscripts accepted for publication must reflect novelty and originality of quality research and be of interest to a multidisciplinary audience. All papers submitted for publication are peer-reviewed by two anonymous reviewers and the editor-in-chief or a designated member of the editorial board.

The editorial board may request for review articles, commentaries or short reviews on contemporary medical or biomedical issues that the board considered important to the advancement of the aims of the journal.

Original research papers should be both complete and concise; they should essentially offer conclusive results, but they should not exceed 7,500 words, including abstract, tables, figures and references.

Short communications and reports should not exceed 3500 words, including abstract, tables, figures and references. Review articles should not exceed 6,000 words, including tables, figures and references. Letter to the Editor should be brief and to the point.

On preliminary editing, all manuscripts that fail to meet the basic requirements indicated above and those that contain significant and obvious typographical errors are returned without further processing.

Manuscripts submitted will be reviewed and considered for publication only if they have not been published, simultaneously submitted or
already accepted for publication in another journal. The author responsible for correspondence must show evidence of approval of all co-authors when submitting a paper for publication.

All relevant ethical approval for research involving human and animal subjects must conform to the provisions of the Declaration of Helsinki in 1995 (as revised in Edinburgh 2000). Only research methods that comply with internationally accepted principles of humane animal experimentation are accepted for publication in The Pacific Journal of Medical Sciences. The decision to accept, revise or reject any manuscript for publication in The Pacific Journal of Medical Sciences is the responsibility of the editor-in-chief; this is done after reviewing the reports and comments from the reviewers, in consultation with members of the editorial board.

Disclaimer:
All statements and opinions expressed in any of the manuscripts published in The Pacific Journal of Medical Sciences are of the authors and co-authors, and not necessarily of the editors or members of the editorial board. The editor-in-chief and members of the editorial board of The Pacific Journal of Medical Sciences disclaim any responsibility or liability for such material and do not guarantee or endorse any products or services mentioned in the articles, nor guarantee any claims made by the authors of the articles.

SUBMISSION OF MANUSCRIPT:
Manuscript should be written in clear and concise English and be intelligible to those that are not specialists in the particular scientific area. Manuscript that does not satisfy these requirements but is acceptable for publication in the Pacific Journal of Medical Sciences because of its essential scientific content will be returned to the authors for extensive and appropriate revision, as recommended by the reviewers and editors.

A covering letter to clarify the following should accompany any manuscript submitted for publication in the Pacific Journal of Medical Sciences: (a) That the scientific data contained in the manuscript has not been published or submitted for publication in any other journal; (b) That ethical clearance and permission for the research had been obtained from the appropriate committee(s) in the institution(s) where the work was carried out; (c) That all the authors have read and approved the content of the manuscript; (d) The name, address and email contact of the author responsible for correspondence and for communicating with others about revisions and final approval of proof of manuscript; (e) Statements quantifying the contribution of each author to the manuscript (this is a requirement in the latest
guidelines of the International Committee of Medical Journal Editors);

Only electronic copy of the manuscript sent as e-mail attachment should be submitted using the approved format indicated in the appropriate sections of this document. Manuscript should be sent by email to any of the following: pacjmedsci@gmail.com; templevictor@gmail.com;

PREPARATION OF MANUSCRIPT:
Manuscripts should be prepared on one side of A4 paper, using double-spacing. Pages are to be numbered consecutively in the bottom right-hand corner. Manuscript should include the following sections: Title page, abstract and keywords, text, acknowledgements, references, tables and figures.

Style: The Pacific Journal of Medical Sciences uses both UK and US spelling. Only one or the other should be used throughout a manuscript. SI units should be used for all measurements. Use abbreviations to avoid repetition of long technical terms: Indicate the abbreviation in parentheses when the word is used in full for the first time. Use the approved generic names of chemical substances and drugs. Do not use trade names or brand names of chemicals and drugs.

Title page: The following should be on the title page: (a) Title of the manuscript – it should be concise and informative; (b) Short running title of not more than 40 characters (optional); (c) Name of each author (first name, middle initial and last name), including highest academic degree; (d) Name and address of institution(s) in which the work was carried out; (e) Name, postal address and email contact of the author responsible for correspondence; Source(s) of research or other types of support for the research project, if any,

Abstract and key words:
The abstract should not be more than 300 words. The following should be clearly stated in the abstract: the purpose of the study, basic procedures, main findings (specific results and statistical significance, if any), and principal conclusions. Abbreviations and references should not be included in the abstract. Not more than 8 key words should be put below the abstract. Key words are used to assist indexers in cross-indexing published articles and may be published with the abstract. Medical Subject Headings (MeSH) list of the Index Medicus should be used for selecting key words (www.nlm.nih.gov/mesh/meshhome.html)

Text:
Text of an original manuscript should be separated into the standard IMRAD format as follows: Introduction, Materials and Methods, Results, Discussion. Sections on Acknowledgements and References should be included.
Introduction: This section should: (a) summarize relevant previous work, using appropriate references, without any extensive review of the subject; (b) clearly state the purpose of the study and summarize the rationale for the study or observation; (c) avoid giving any data on the work being reported.

Materials and Methods: This section should: (a) clearly indicate either the sampling procedure or observational subjects; (b) give appropriate references for established techniques and procedures; (c) new techniques and procedures and extensive modifications of existing ones should be presented in sufficient details so that other researchers can easily reproduce and evaluate them; (d) indicate appropriate quality control procedures used for laboratory methods and techniques; (e) indicate ethical procedures if either human subjects were involved [if informed consent was obtained from each subject] or if appropriate guideline for using laboratory animals were followed [see editorial policies above]; (f) indicate statistical methods used, if any.

Results: Data obtained should be presented in logical sequence in the text, tables and figures should be adequately explained to facilitate their interpretation. Avoid duplicating the results by repeating in the text all the data presented in the tables and figures. The text in the results section should only emphasize or summarize the important data.

Discussion: Major findings should be highlighted before the minor findings. All findings should be related to other relevant studies, if any, using appropriate references. Indicate the implications of the findings and their significance or limitations, including implications for future research. When warranted, propose new hypotheses with appropriate data, but be sure to clearly label them as such. The conclusions should be linked with the goals of the study and be clearly supported by evidence/data. Include recommendations, if applicable.

Acknowledgements:
The following should be acknowledged: Research or other financial grants; Material support, Contributions of Institutions, Colleagues, and other relevant participants.

References:
The Pacific Journal of Medical Sciences uses the Vancouver system of referencing. The references should be numbered, using Arabic numerals in square brackets, in the order in which they are first used in the text, tables, figures, and legends. In the reference section, list the references in the order of appearance in the text, tables, figures and legends. Abstracts, unpublished data, oral communications, and personal communications should not be included in the reference section. All references should
be verified against the original documents. In the reference section, the names of all authors should be included. Avoid using “et al.” in the reference section. Names of journals should be abbreviated, using the approved style indicated in Index Medicus/PubMed. References should be listed according to the examples given below:

**Journal articles:**

**Book:**

**Chapter in a Book:**

**Published proceedings paper:**

**Tables:**
Tables should be numbered sequentially in Arabic numerals, typed double-space on separate A4 paper for each table; vertical lines should not be used to separate columns. Each table should be self-contained with a comprehensive but concise legend/heading; column headings should be brief, with units in parenthesis. All non-standard abbreviations used in tables should be explained in footnotes, using the following symbols in this sequence: *, §, ¶, #, $.

**Illustrations:**
Graphs, line drawings, bar charts, maps, etc., should be labelled as ‘figures’ and numbered consecutively, using Arabic numerals. All figures should be drawn using computer graphics. Legends should be brief but understandable without referring to the text. Photographs should be unmounted sharp, glossy black and white prints. Photographs should contain scale bars, not magnifications. Colour photographs are not acceptable. Figures, reproduced from another source, should be clearly indicated and appropriate references and written permission from the copyright holder must be submitted with the manuscript.

**Electronic copy of manuscripts (e-mail):**
When a manuscript is accepted for publication, the corresponding author will be required to send an electronic copy of the corrected or
modified manuscript by email. All email attachments should be scanned with anti-virus software before sending. Automatic software for referencing, footnotes, headers, footers, etc., should not be used during formatting.

All manuscripts should be formatted using MS WORD.

GALLEY PROOFS:
Galley proof will be sent by email to the correspondent author. Only minor corrections should be made, no major alterations to the manuscript will be accepted. Galley proof should be returned within maximum 5 working days from the date of receipt. If any major modification is made, the manuscript will be rejected at this stage. Correspondent author should correct all printing errors and ensure that the typesetting is accurate in the galley proof. Note that the correspondent author, not the publisher will be responsible for any such errors, should they occur in the published paper.

REPRINTS:
Reprints will not be sent to authors, because the PJMS is an on-line journal. The Web-link will be sent by email to the corresponding author. One copy of a paper may be sent to the correspondent author on request only, subject to availability of funds to cover postage.

Authors may download and print the journal at their own expense.

COPYRIGHT:
Manuscripts accepted for publication in The Pacific Journal of Medical Sciences become the property of the journal. Therefore, all authors may be requested to sign a transfer of copyright form and send scan copy by email attachment to the editorial office. The copyright gives the journal the exclusive rights to reproduce, translate and distribute the article for academic purposes.

CHECKLIST FOR AUTHORS:
- Running title included (Can be omitted if not applicable);
- Abstract prepared according to instructions and include key words;
- Manuscript typed double-space on one side of A4 paper;
- References cited in square brackets in text and listed according to approved style for this journal; Uniform spelling throughout the text;
- Tables and Figs on separate A4 pages;
- Covering letter written as required by this journal;
- E-mail address of corresponding author indicated.