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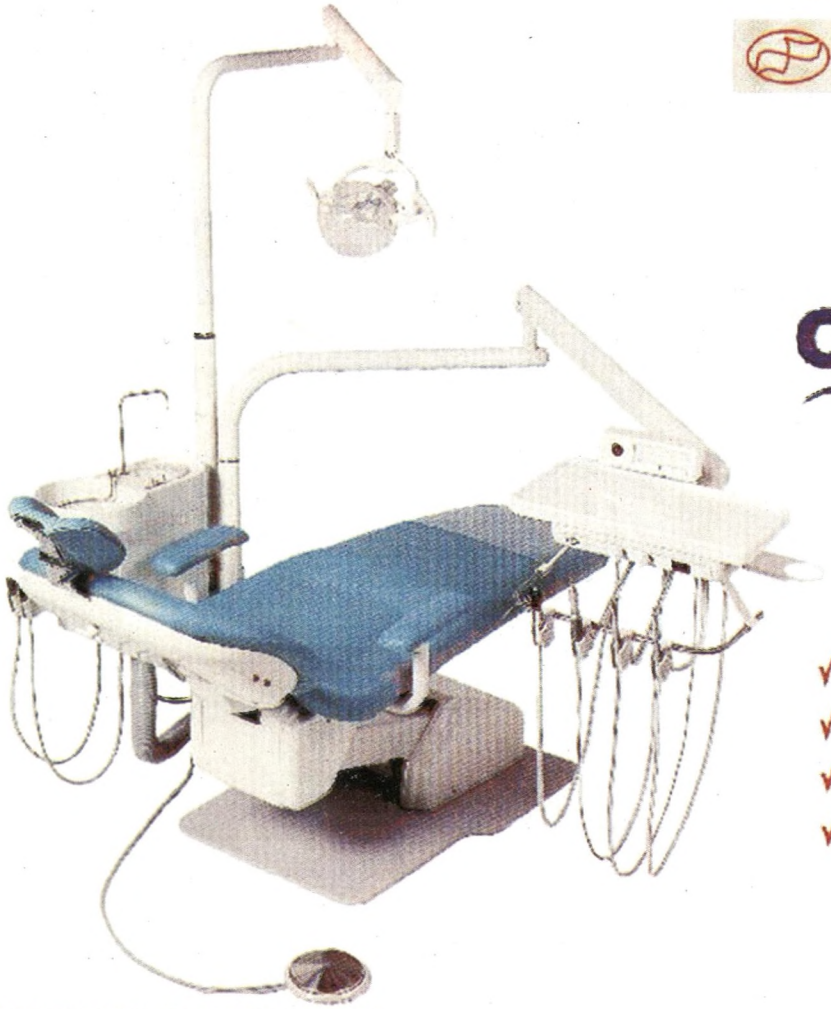

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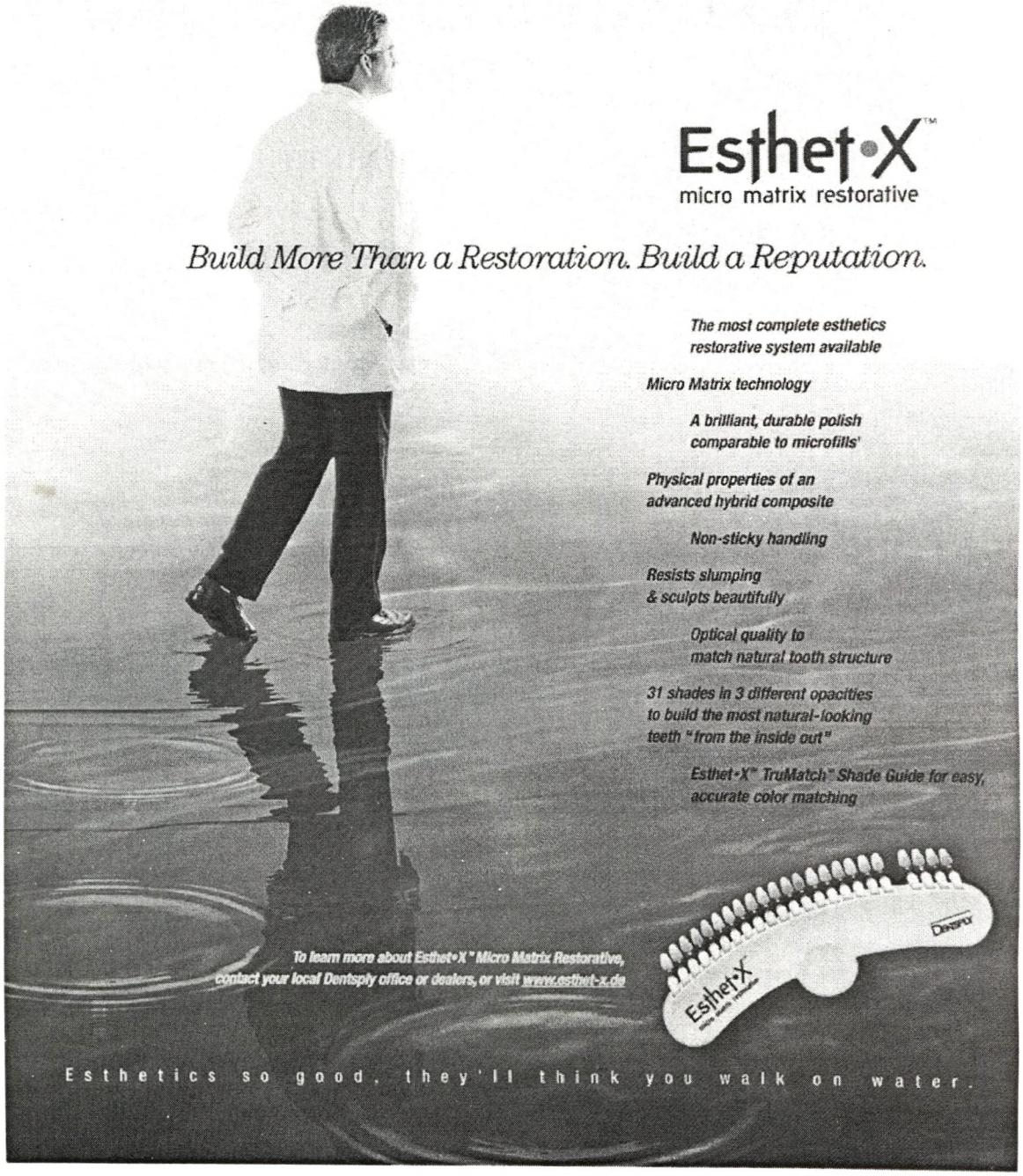

Sri Lanka Dental Journal

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EDITORIAL

The aspiring dental student: his future

Serious concern has been expressed over the problem of non-availability of employment for the newly qualified dental graduates in the Ministry of Health. While three batches of newly passed out dental graduates are facing a great crisis in their career, as the State is unable to provide them with employment in the Ministry of Health, a group of aspiring dental students are longing to commence their journey towards their chosen career in dentistry at the Faculty of Dental Sciences of the University of Peradeniya.

But how are our students chosen? The selection of students to the Faculty of Dental Sciences is based purely on their intellectual abilities as measured by their achievements at the GCE Advanced Level examination.

In the year 1999, approximately 25000 students sat for this examination in the bio- sciences stream. About 7000 of this number have been successful in securing the obligatory entry requirement to the universities. All 75 students who are enrolling with the study programme in Dentistry have been chosen on a district quota basis in the bio-sciences stream having obtained the minimum cut off point mark.

Clinical dentistry requires manipulative skills. Does this particular selection procedure help us to pick out a student who is particularly 'good with his hands'? The answer to this question is No. We on the other hand are picking out good examination students possibly highly intelligent, some of whom are deficient in manual dexterity. The latter student has little chances of making a successful career in dentistry except in a rare non-clinical area.

It is equally important to find out the attitudes of these undergraduates towards the dental profession and also the factors that prompted them to select a career in dentistry. In this respect, the results of a survey that has been carried out amongst the first year dental undergraduates by a group of academics will be most welcomed when it is published in the Sri Lanka Dental Journal in due course. Studies done in the West have shown that common reasons for choosing dentistry as a career include a positive public perception of the profession due to above average financial benefits and high public esteem.

Anyhow, to be successful in dentistry, one should possess an above average intelligence and manual dexterity to a moderate degree. They should also be kind and compassionate towards others. Those graduates opting to join general dental practice should have management skills, and a business sense if they are to be successful in the highly competitive market.

In order to attract the intelligent young ones of today the career prospects and employment avenues should be satisfactorily diverse and stimulating.

Deepthi Nanayakkara
Editor

Sri Lanka Dental Journal 29 (2) 2000 59-62

LEADING ARTICLE

Dental consideration in preventing infective endocarditis

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Infective endocarditis is a life threatening medical condition with an incidence ranging from 10 to 60 cases per million persons per year.[1,2] Though in the majority of instances it is an infection of the endocardial surface of the heart, structures outside the heart such as arteriovenous shunts, arterioarterial shunts may also be affected. Before the availability of antimicrobial agents, the mortality rate was 100%.[3] The mortality rate remains at a high level despite the availability of potent antimicrobial agents but has substantially improved with optimal treatment.[4] Early diagnosis is the single factor that is likely to improve the outcome. Because of its severity, it is agreed that infective endocarditis should be prevented whenever possible.

A number of prerequisites are necessary for the development of this condition. The main factors that play roles in the acquisition of endocarditis are a suitable cardiac lesion and a source of infection. One of these factors alone is insufficient to cause endocarditis. The endocardial surface should be altered to enable the attachment and colonization of microorganisms. Turbulence of blood and mechanical stress exerted on the endocardial surfaces play a key role in the deposition of platelets and fibrin on these surfaces. High velocity flow favours endothelial deposition of organisms as compared to low velocity flow. Thus left sided cardiac lesions such as mitral and aortic valve disease, ventricular septal defects and patent ductus arteriosus are particularly at risk. [5] Further deposition of fibrin and platelets causes creation of vegetation, resulting in a condition called nonbacterial thrombotic endocarditis. The vegetation serves as the nidus for bacterial invasion. Bacteria then reach this site and colonise prior to further multiplication beneath a protective covering of fibrin and platelets.

While virtually any bacterial organism can cause bacterial endocarditis, the vast majority of infections are caused by gram-positive cocci. The viridans group of streptococci is the most common cause of endocarditis involving native heart valves, in patients with congenital heart disease and in those who are not injection drug users.[6] Coagulase - positive staphylococci *(Staphylococcus aureus)* commonly cause bacterial endocarditis in patients with prosthetic valves and in injection drug users.[7] Presence of these organisms is also a common cause of acute bacterial endocarditis in persons whose heart valves were previously normal.

The ability of certain organisms to adhere avidly to the platelet – fibrin matrix explains why streptococci, staphylococci and enterococci are particularly common in infective endocarditis. Among the proposed pathogenetic mechanisms, the ability to elaborate the complex polysaccharide dextran appears to be critical factor at least in the streptococci. In others, platelet aggregability of the platelets may be an important factor. [8]

Bacteraemia plays a critical role in the development of endocarditis and the likelihood of it occurring forms an important basis of recommendations on prophylaxis. Bacteremia may occur spontaneously

such as through food chewing or tooth brushing, or it may develop as a complication of a focal infection, such as a periodontal or periapical infection, a urinary tract infection, pneumonia or cellulitis. Selected surgical and dental procedures and instrumentations, especially those involving mucosal surfaces or contaminated tissue, can cause a transient bacteremia that rarely persists for more than a few minutes.

The incidence of transient bacteremia after various dental procedures has been extensively studied. [9] Both the incidence and the magnitude of bacteremias of oral origin are proportional to the degree of oral inflammation and infection.[10] Extraction of one or more teeth, dental implant placement and reimplantation of avulsed teeth, periodontal surgery and prophylactic cleaning of teeth or implants, where bleeding is anticipated are examples of high risk procedures associated with significant transient bacteremia.[11] Placement of removable prosthodontic or orthodontic appliances, oral impressions, fluoride treatment, oral radiographs and orthodontic appliance adjustment are considered low risk procedures that require no prophylactic antibiotics. [11]

The issue is complicated by the fact that data on postprocedure bleeding and bacteremia show that there is no relationship between bleeding and bacteremia.[12] Significant bacteremia can occur in the absence of clinically discernible bleeding. The intensity of bacteremia in humans is significantly less than that used in experimental endocarditis models. It is unlikely that the intensity of bacteremia following dental procedures in children could readily lead to endocarditis.[12] A study among children randomised to either tooth brushing, scaling or professional cleaning showed equal chance of the likelihood to develop odontogenic bacteremia from tooth brushing at home as from professional scaling and polishing of the teeth at dental surgery .[13]

The cumulative exposure to bacteremia is significantly greater from everyday procedures when compared to dental operative procedures. It is far more likely that such everyday procedures are the cause of bacterial endocarditis because the cumulative exposure is often hundreds, thousands, or even millions of times greater than that occurring following surgical procedures such as extraction of teeth. The presence of certain bacterial species in blood may be more important than the overall frequency of bacteremia.

The degree to which the patient's underlying condition creates a risk of endocarditis should also be taken into consideration. Endocarditis is associated with some cardiac conditions more often than with others. These conditions are stratified into high, moderate and negligible-risk categories based primarily on the potential outcome if endocarditis occurs. The high-risk category includes individuals with prosthetic heart valves, a history of endocarditis, complex cyanotic congenital heart disease, or surgically constructed systemic pulmonary shunts or conduits.[5] The moderate risk category includes patients with congenital cardiac malformations (other than complex cyanotic disease), acquired valvular dysfunction (e.g. due to rheumatic fever), hypertrophic cardiomyopathy, mitral valve prolapse with valvular regurgitation and/or thickened leaflets, and certain types of heart murmurs. Individuals with isolated secundum atrial septal defects, surgical repair of atrial or ventricular septal defects, coronary bypass surgery, mitral valve prolapse without valvular regurgitation, innocent or physiological heart murmurs, previous Kawasaki disease or rheumatic fever without valvular dysfunction, and pacemakers do not necessarily need antibiotic prophylaxis.[14] The negligible-risk category suggests that endocarditis develops no more often than in the general population.

Antibiotic prophylaxis in at-risk patients has been accepted as reasonable practice for four decades, although data confirming its effectiveness are lacking.[15] While there are currently no carefully controlled studies to prove antibiotic prophylaxis helps protect against endocarditis, in vitro bacteria studies, experimental animal models, uncontrolled clinical observations and retrospective analysis of human antibiotic use provided the basis of guidelines recommended for prevention of bacterial endocarditis.[16] However, with little convincing evidence from human studies, expert groups continue to specify antibiotic prophylactic regimens.

Two important outcome studies that have been recently published have reopened the debate on endocarditis prophylaxis. These two outcome-based studies have similar findings and indicate that the current guidelines, which are not based on population-based outcome studies, require further review.

A Dutch study[1] assessed 427 patients with endocarditis and found that 64% of these patients would have been eligible for antibiotic prophylaxis based on previously known cardiac conditions. Twenty-three per cent had undergone a procedure that would have indicated prophylaxis within one-half year of onset of endocarditis, and 11% had undergone a procedure within 30 days of onset. Antibiotic prophylaxis had been administered to 26.6 % of the 64 patients and to 25.8 % of the 31 patients. It was thought that prophylaxis may have prevented 17% of cases within 180 days of onset, a period of time that extends beyond what many believe to be the appropriate incubation period, and 11% of cases within 30 days, representing only 5.3% of cases. Therefore, even if antibiotic prophylaxis was 100% effective and was provided for all at-risk patients receiving dental treatment, only a small fraction of cases of endocarditis (5.3%) would be potentially prevented.

The other study assessed patients in 54 hospitals in the Philadelphia area.[17] A total of 287 cases of endocarditis were identified. It was found that in the three months preceding the diagnosis of endocarditis, dental treatment was no more frequent in these patients than in non-infected age- and sex-matched control patients

However, the American Dental Association, like the American Heart Association, believes its current recommendations for endocarditis prophylaxis are valid and that modifications are not indicated at this time. They acknowledge that further research is warranted to determine whether some heart or valvular conditions would require coverage with pre-procedural antibiotics while others would be precluded.

Failure of protection may also occur due to antibiotics starting too early, using inadequate doses, disregarding the use in children and withholding in rheumatic fever patients in the belief that penicillin prophylaxis provides adequate protection when in fact it is insufficient. Prophylaxis fails even with antibiotic regimens recommended by the American Heart Association /American Dental Association.[11]

Experiences from dental practices has been shown to be varied and in conflict with guideline recommendation as to endocarditis prophylaxis prior to dental procedures.[18] When patients are diagnosed with a cardiac disorder, which predisposes to infective endocarditis they should have a dental examination as soon as possible. Much more importance should be given, however, to encouraging people to seek better routine dental care.

Infective endocarditis represents one of the few potentially fatal infections that may be caused in the patient by a dentist. It is desirable that guidelines for therapy be based on outcome

studies and on evidence of safety, efficacy and, increasingly, cost effectiveness as this disease still produces considerable morbidity and mortality. In spite of the uncertainty that elimination of all cases due to such procedures would be small, and the risk for adverse reactions, including anaphylaxis, and the possible occurrence of drug-resistant organisms the other reason to adhere to guidelines laid down by organizations is the threat of litigation for failure to provide prophylactic treatment.

The emerging data on infective endocarditis, suggesting limited risk associated with certain dental treatment, the time of incubation and the increasingly available outcome-based evidence, require continual review of the current historically and empirically based recommendations.

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Sri Lanka Dental Journal 29 (2) 2000 63-68

REVIEW ARTICLES

Risk factors for periodontal disease and periodontal disease as a risk factor for systemic conditions

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Introduction

It is now known that periodontal diseases are infections caused by bacteria and are multifactorial in origin. There are known risk factors that modify or aggravate the existing periodontal diseases. Although the medical researchers have worked steadily for decades to identify risk factors, it is only within the last 5 to 10 years have the dental researchers begun to look for risk factors apart from poor oral hygiene habits which may predispose a person to develop periodontal disease.[1]

The American Academy of Periodontology[2] indicates that the initial therapy for patients with advanced loss of periodontal support should include the elimination, alteration or control of risk factors that may contribute to periodontitis. Therefore the control of the risk factors involved in periodontal disease is considered to be of utmost importance in the optimal management of periodontal diseases. Diabetes mellitus, infection with human immuno-deficiency virus (HIV), physiological conditions such as pregnancy and menopause which lead to a hormonal imbalance, and the use of certain medications are known to be possible risk factors in periodontal disease. Therefore, the management of risk factors, along with antiinfective and regenerative therapy could be suggested as the most successful approach in the management of periodontitis in the future.

Periodontal diseases can also be a risk factor for other systemic diseases such as cardio-vascular disorders. A link between periodontal diseases with an increased risk for atherosclerosis and thromboembolism has been shown.[3]

Periodontal infections in pregnant women are now being recognized as a risk factor to have pre-term low birth weight (PLBW) babies. PLBW caused by infections is thought to be mediated by the translocation of bacterial products and by maternally produced inflammatory mediators. [4]

Risk factors for periodontal disease

These can either be systemic factors such as diabetes, HIV infection or local factors such as smoking.

1) Diabetes mellitus

Both insulin-dependent (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM), especially in the long-term are now viewed as significant risk factors for periodontal diseases. These findings were based on the epidemiological studies carried out in the recent past. In a longitudinal study it was found that the age and gender adjusted incidence of periodontal disease in patients with NIDDM was 78 cases per 1000 persons, about three times higher than in those without diabetes (29 cases per 1000 persons).[5] It is also known that patients with IDDM or NIDDM are at equal risk for developing periodontal diseases when other variables are controlled. Periodontitis progresses more rapidly and leads to more tooth loss in patients whose diabetes is poorly controlled.[6] These patients exhibit higher levels of the enzyme beta-glucuronidase in their gingival crevicular fluid than those in well-controlled

diabetes. It is still not very clear why diabetics increase the risk for severe periodontitis, although increased susceptibility to infection and impaired host response are likely factors. There are no significant differences in the micro-flora between people with either IDDM or NIDDM and those without.[7] In diabetics, especially in those whom the disease is poorly controlled or of long duration, the basement membrane of the small vessels thickens as a result of the nonenzymatic glycosylation of extracellular matrix components and intracellular proteins and there is a subsequent accumulation of deposits known as advanced glycosylation end products (AGEs), in vessel walls and on the luminal surface. These changes can narrow the lumen of the vessel interfering with transport across the vessel wall, imparting oxidant stress on the periodontal tissues and prolonging inflammation. This effect could be related to the increased incidence of periodontal disease in diabetics.[8] A build-up of AGEs may lead to the microvascular complications seen in diabetes which is a factor in other diabetic complications such as retinopathy, neuropathy and nephropathy.[9] There are other suggested mechanisms involved with diabetes, such as vascular changes, polymorphonuclear leukocyte dysfunction, abnormal collagen synthesis, abnormal bone matrix production and inflammatory mediator production, and an altered immune regulation which may have a role in complications of diabetes.[10]

It is reported that well-controlled diabetics who receive regular periodontal maintenance care, with good oral hygiene habits are less likely to develop severe periodontitis. Well-controlled diabetics have also been shown to respond to periodontal therapy as equally well as non-diabetics.[11]

2) HIV (Human Immuno-deficiency Virus) infection

This is a known risk factor for periodontitis. A well designed, 20-month study that included 114 men showed that the periodontal changes were related to HIV-1 serostatus, immunostatus, age and plaque deposits. The immunosuppression levels of helper-inducer T-cell (CD-4) counts of less than 200, especially in subjects of over 35 years of age. The risk for a greater than 3-mm clinical attachment loss was increased by more than 600%. Further, seropositivity, despite the immunostatus was a significant risk factor for gingivitis.[12] An estimated 5% of HIV patients develop a severe, rapidly progressing form of periodontitis or necrotizing ulcerative periodontitis (NUP). This lesion is associated with pronounced immunosuppression. Many of these cases do not respond to standard periodontal therapies, but when they are left untreated NUP can progress to necrotizing stomatitis.[2]

The regulation of polymorphonuclear leukocyte (PMN) recruitment into the gingival crevice is hindered in HIV-seropositive patients. This irregular PMN activity may also allow candida organisms to colonize subgingivally and thus increase the likelihood of developing periodontitis.[13]

Not only HIV infection, any condition that compromises host defense mechanisms, including irradiation and genetically transmitted diseases predispose patients to early-onset severe periodontitis.

3) Age

It has been found that the prevalence of periodontal disease increases with age. It is not clear whether this is the result of accumulating periodontal lesions over the years or to a greater susceptibility of the elderly to develop the disease. Some cross-sectional studies implicate age as a predisposing factor to periodontal disease. According to longitudinal studies, periodontal disease appears to be age-associated rather than to have a direct consequence with the body's

aging process. In a study using 14,690 Americans aged 15 to 74 years, 95% of them who maintained good oral hygiene did not have periodontitis regardless of age.[14] It has been documented that periodontitis frequently begins with loss of attachment during youth and early adulthood rather than in the older years.[15]

4) Osteoporosis

A relationship between osteoporosis and periodontitis has long been suspected. Oral bone loss associated with osteoporosis may be important in creating a susceptible host for dental disease.[1] There is a strong correlation between dental bone mass and the total bone mass among women, as well as total bone mass and number of teeth present in the mouth. Factors such as smoking, which predispose patients to osteoporosis also predispose them to alveolar bone loss.[16]

5) Menopause

The dwindling oestrogen levels that follow menopause are known to be linked to a greater risk of tooth loss. In the Nurses Health Study, carried out in the United States of America in 1996, it was found that decreased oestrogen levels in postmenopausal women who did not undergo hormone replacement therapy were associated with significantly greater tooth loss.[17] Similarly, in the Leisure World Study in 1995 which aimed to find out the benefits of oestrogen replacement therapy on oral health, it was found that women who received oestrogen replacement therapy had a lower risk for edentulism.[18]

It has been shown that bisphosphonate alendronate, an approved drug in the treatment of osteoporosis could lower the risk of bone height and density loss. [19]

6) Increased use of medications

More people are now using one or more prescriptive or over-the-counter medications than in the past. These may also affect the periodontal health. For example, some medications significantly decrease the salivary flow. These include anti-hypertensive drugs, narcotic analgesics, some sedatives, tranquilizers and antihistamines. The resulting xerostomia can help to accumulate more plaque. Other drugs, particularly those in liquid or chewable form that contain added sugar alter the pH and composition of plaque, making it more adherent to tooth surfaces.[1]

7) Smoking

Tobacco smoking is a highly predictive risk factor for periodontal disease, especially for severe periodontitis. It was found that light smokers (aged 25-74 years) had a relative risk of developing detectable periodontal disease that was 2 times higher than non-smokers, and heavy smokers had a relative risk that was 7 times higher.[20, 21]

There are a number of suggested mechanisms by which smoking may promote periodontal disease. Periodontal pathogens such as Bacteroides forsythus and Porphyromonas gingivalis could persist after mechanical debridement to a larger degree in smokers than nonsmokers.[21] Smoking has been shown to decrease both cell-mediated and humoral immune responses. Peripheral blood polymorphonuclear leukocyte phagocytosis is significantly impaired in smokers and they have lower levels of serum IgG_2, which normally provides the predominant serum antibody response to antigens of both Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis.[22, 23] One study has also shown that even passive smoking can over-stimulate oral PMN function for up to an hour after exposure to smoke, although it is not known how detrimental that effect might be cumulatively.[24] Smoking cessation seems to yield marked periodontal

benefits. It was found that, within a few months of quitting, the gingival inflammation of smokers worsens and bleeding is common during brushing for several months. After about a year, the gingival tissues may revert from a thickened fibrotic appearance customarily found in smokers to normal anatomy and contour.[25]

8) Other systemic factors
Some studies suggest that chronic stress, depression, financial problems, social isolation and other anxiety related psychosocial factors may be risk factors for periodontitis.[26, 27]

It is suggested that these stressors could depress the body's immune responsiveness to periodontal pathogens and thus increase the likelihood and severity of periodontal disease.

Alcohol abuse has also been suspected to have contributory effects on periodontal disease. However, further studies may be needed to confirm this. Other possible systemic factors that may increase the risk include a family history of periodontitis and certain genetic diseases.

Periodontal diseases as a risk factor for other systemic conditions

Periodontal disease may itself be a risk factor for other infections in the body, systemic diseases and pregnancy-related problems.

1) Cardio-vascular disease (CVD)
In recent years several researchers have found evidence linking periodontal disease with an increased risk for atherosclerosis and thromboembolism, independent of other risk factors for CVD. In a study adjusted for other risk factors of coronary heart disease (CHD), the researchers found that men with periodontitis were 25% more likely to develop CHD. The risk was particularly high for men under the age of 50, who had a relative risk for CHD.[3] In two different surveys, Umino and Nagao found heart disease to be the most common condition shared by patients with periodontitis.[28]

Given that periodontal disease is a chronic infectious disease that involves an abundance of gram negative species containing endotoxins and pro-inflammatory cytokines, it is not impossible that it may pose systemic challenges that predispose the periodontitis patient to coronary heart disease or exacerbate the condition.[1] Some authors suggest that microbial pathogens associated with chronic infections such as periodontal disease may disseminate through blood to infect the vascular endothelium.[1] Further, myocardial ischemia often is preceded by acute thromboembolic events, and several major species of dental plaque, such as Streptococcus sanguis have been shown to induce thrombosis.[29]

Periodontal disease may also interfere with blood lipid metabolism. Although the data show a consistent link between periodontal disease and CVD, there is still only limited evidence for a causal relationship.[30]

2) Pre-term low birth weight children
Although the infant mortality rate has decreased significantly since the mid 1960s, pre-term low birth weight (PLBW) babies are still common even in developed countries such as the United States, despite improved prenatal care. Even when traditionally believed risk factors for PLBW such as smoking, drug and alcohol abuse, nutrition are taken into account, about 25% of PLBW cases occur in women without these risk factors.[4] Since PLBW is a significant factor for infant mortality, there appears to be a great need to investigate for further causes of PLBW.

Periodontal infections in pregnant women are receiving considerable attention as a potential

independent risk factor for PLBW infants. Studies have demonstrated a link between genitourinary infection and PLBW, and the widely held opinion is that PLBW caused by infections is probably mediated by the translocation of bacterial products, such as endotoxins associated with periodontal disease, and by maternally produced inflammatory mediators.[4]

Periodontal infections which serve as reservoirs for gram negative anaerobic organisms, lipopolysaccharides, and inflammatory mediators may also threaten the health of the feto-placental unit. This has been demonstrated in some animal studies. For instance, in one study the fetuses of hamsters with localized subgingival infections of a common periodontal pathogen weighed up to 25% less than the fetuses of healthy pregnant hamsters. These infections were also associated with increases in inflammatory mediators that appear to retard fetal growth.[31] A few human studies too show a similar link to this. In a study of 124 pregnant or postpartum mothers, controlled for traditional well known factors of low birth weight babies, Offenbacher et l. [32] found that those with periodontal disease were seven times more likely to deliver a PLBW baby. Hill[33], in 1997 reported that Fusobacterium nucleatum is the most frequent isolate cultured from the amniotic fluid of women with preterm labor, but that this organism is not prevalent in the vaginal microflora. It is commonly found in the mouth, however, and the authors theorize that it may spread to the amniotic fluid via a transient bacteremia in the presence of periodontal disease.[33]

3) Other systemic conditions

Periodontal disease, particularly in hospitalized patients or those in nursing homes may increase the risk for bacterial pneumonia, because dental plaque among these patients tends to harbor a high number of respiratory pathogens.[34] It was also noted that the elderly people who undergo prosthetic joint replacement are at a greater risk of developing infections in the prostheses as a consequence of untreated dental disease. This was thought to be due to the etiologic bacteria entering the bloodstream in the oral cavity.[35, 36]

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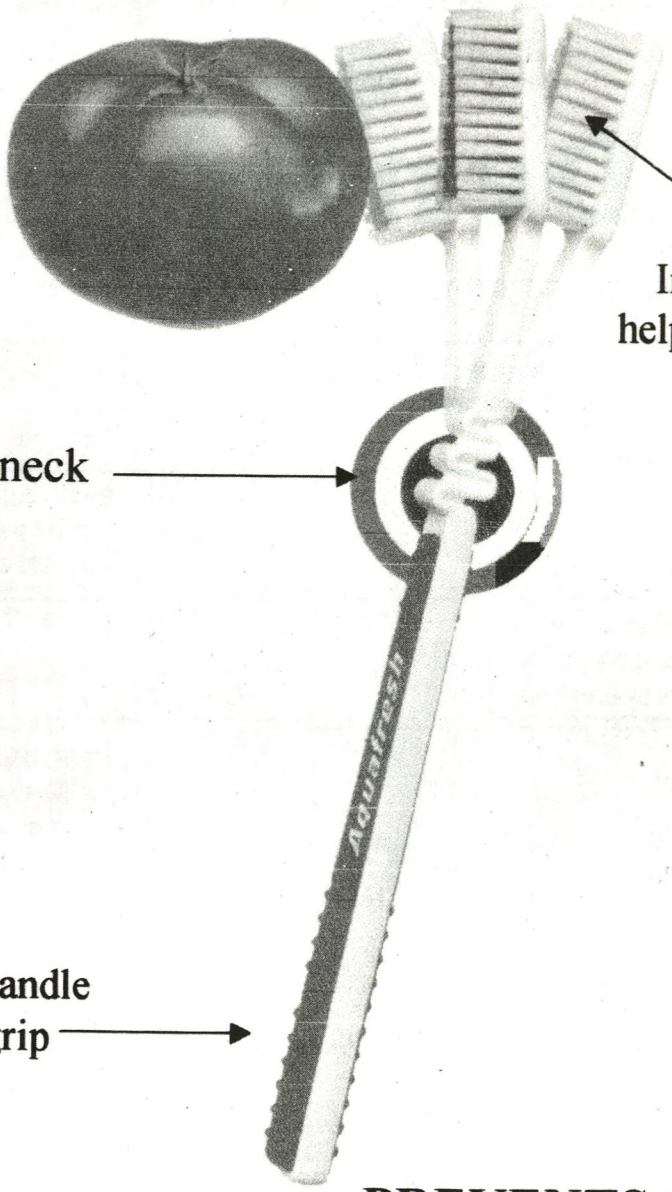

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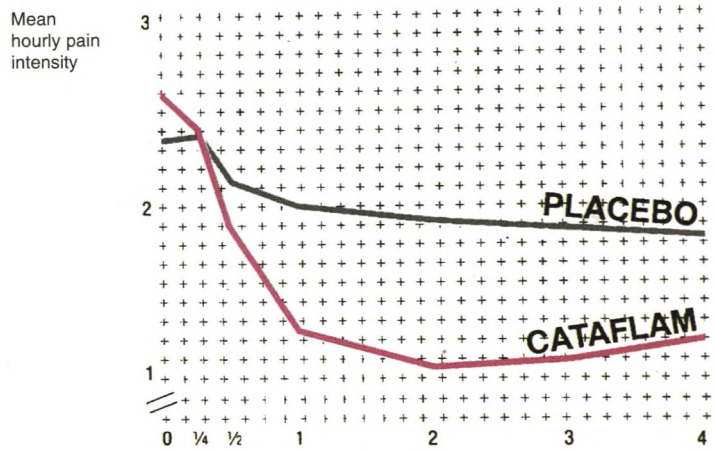

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Burning Mouth Syndrome

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Introduction

Burning Mouth Syndrome (BMS) has become a dilemma of diagnosis and treatment. Patients with burning mouth may consult not only dentists or a family doctor but also may seek help from physicians, oto-laryngologists, and dermatologists. Therefore, many patients receive varying types of treatment with unsuccessful results. BMS is a chronic oral pain disorder usually unaccompanied by mucosal lesions or other clinical signs.[1] This pain disorder can be regarded as a sensation perception abnormality affecting the oral mucosa and lips.[2]

Patients with BMS were labeled as "neurotic" because they do not show obvious physical causes. There is an increasing recognition of this disorder as a specific entity in the literature with emphasis on evaluation of the condition and the need for a long-term management strategy. The aim of this paper therefore, is to discuss the importance of medical and dental care which enable clinicians to manage the condition appropriately and with empathy.

Terminology

The syndrome has been identified by many names. Most of these names are derived depending on the involved site of the oral mucosa. Glossodynia or glossalgia have been used to describe pain symptoms associated with the tongue whereas burning sensation of the tongue was termed as glossopyrosis. Likewise when other areas of the mouth are affected the term stomatodynia or stomatopyrosis are used. However, all these can be described by the term Burning Mouth Syndrome regardless of the site affected.[3]

Burning sensation in the mouth can basically be divided into two main categories: a burning mouth with apparent clinical signs in the mouth or without any apparent clinical signs.[3,4] However, the term Burning Mouth Syndrome should be used to describe the burning sensation which is unaccompanied by clinical signs or idiopathic, which does not have identifiable aetiological factors.[3]

Epidemiological characteristics

It is evident that the epidemiological features of idiopathic burning mouth syndrome are imprecise due to the variable terminology and range of conditions included as BMS. Although, there are many studies on demographic and clinical characteristics, only few population based studies have been reported.[4,5,6,7] Basker et al.[8] in a clinical study using patients from general dental practice, menopause and diabetic clinics in Birmingham and West Midlands reported a prevalence of 2.6% in those over the age of 20 years. Locker and Grushka[9] in a population-based study reported a prevalence of 4.5% using a sample of 1000 residents of Toronto, Canada. Tammiala- Solonen et al.[6] in another population based study in Finland showed a prevalence of 15%. However, half of this 15% had some clinically observable oral mucosal lesion/s attributable for the burning sensation. Therefore, it can be argued that the prevalence of idiopathic BMS must be less

than this figure. Furthermore, Savage[10] reported that about 10% of new referrals to his clinic included patients with BMS.

Clinical features

BMS is more common in females with a female to male ratio of 7:1.[3,6,11,12] The majority of BMS patients are post-menopausal women [4,13] and middle aged women.[7] The occurrence of BMS under the age of 20 is very rare.[3] BMS generally encompasses the sensation of "burning" in one or more sites of the oral mucosa. However, it may be accompanied with other complaints such as dryness in the mouth, altered or disturbed taste sensation[7], dysphagia, sore throat, halitosis, cervico-facial muscular hypertonicity.[4,10] Symptoms are usually bilateral[11] affecting the tongue, lips, vermilion, anterior hard palate and occasionally circumoral skin. [2] Several authors[5,8,14,15] have reported on the prevalence of BMS by site (Table 1). Most patients complain of burning sensation as occurring at more than one site in the mouth. However, the comonest site of occurrence is the tip of the tongue. [2,11] Sites of complaints in order of frequency are shown in Table 2.

Table 1. Prevalence of BMS by site *

		Percentage by site						
Author	n	Tongue	Lip	Buccal mucosa	Palate	Throat	Upper denture bearing area	Lower denture bearing area
Basker et al., (1978)	22	50	36	14	_	13	50	18
Main and Basker (1983)	37	67	34	18	_	05	68	25
Lamey and Lamb (1988)	150	78	38	04	_	08	45	36
Gorsky et al., (1991)	130	48	24	_	25	_	_	_

* Percentages add up to more than 100 due to multiple site involvement.

Table 2. Sites of complaints in order of frequency

Order	Site
1	Tip of the tongue
2	Lateral borders of the tongue
3	Dorsum of the tongue
4	Lips
5	Buccal mucosa
6	Palate
7	Throat
8	Upper denture bearing area
9	Lower denture bearing area

Adapted from van der Ploeg et al.[16]

Burning mouth symptoms may take several months to evolve in a "waxing and waning" pattern with some patients showing complete remission and reappearance.[2] Symptoms are variable among patients and within an individual the severity varies from time to time.[10] Lamey and Lewis [11] have categorized the symptoms of BMS into three types. In type I, the patient wakes up with no symptoms, and burning sensation begins to appear gradually and the severity increases as the day goes on. In type II, burning is present on awakening and persists throughout the day whereas in type III, there may be symptom free days. This classification may be useful in assessing the progress in which type I shows the best prognosis.

Aetiology

The exact aetiology is yet to be determined. However, many local and systemic factors have been identified as causative factors for burning sensation. van der Waal[3] has divided the possible factors into 4 main categories namely, local, systemic, neurological and psychogenic.

Local factors

Local factors include mucosal lesions, prosthetic appliances, dental treatment, metal allergy, food allergy, smoking and use of alcohol. Many mucosal lesions and conditions may produce BMS like symptoms. Among them candidal infection, geographic tongue, oral lichen planus (mainly erosive type), oral ulceration and oral submucous fibrosis play a significant role. [3] Lamey and Lamb[15] have reported a prevalence of candida species in 40% of the patients with burning mouth symptoms. However, only one in seven of these patients having candida in their mouths had relief after anti candidal treatment. Geographic tongue is often asymptomatic and therefore most of the patients are not aware of its presence in the mouth. However, some may develop burning symptoms. Gorsky et al.[17] reported that 15% of their 98 patients had geographic tongues. Among many morphological types of oral lichen planus the erosive or atrophic forms are mainly responsible for burning mouth symptoms.[18] Dentures have been regarded as one of the aetiological factors in oral burning.[3,15] Burning sensation in these patients is considered to be attributable for faulty dentures (articulation and stability etc.) [14] and allergy to polymethyl methacrylate.[15] However, some authors questioned the possible correlation between burning mouth symptoms and faulty dentures.[17,19] Some patients may demand for many kind of dental treatments as a remedy for their burning symptoms.[20] In a retrospective study Mock et al.[21] had shown that symptoms of atypical facial pain can either persist or aggravate after dental treatment such as extraction, root canal treatment, in about 75% of patients. Oral galvanism, a phenomenon of currents resulting from an electrical reaction between metal dental restorations has also been identified as a causative factor for oral burning.[3] However, controlled studies on patients with BMS symptoms and those without, have shown similar currents in both groups.[22] Contact allergy to dental restorative materials has been suggested as an aetiological factor for burning mouth symptoms.[1] However, supportive evidence for this phenomenon is lacking.[15,23] A correlation between food allergy and burning symptoms has been mentioned in the literature. Sensitivity to some food-related products such as sorbic acid, propylene glycol, benzoic acid and cinnamon was discovered on patch testing. Dietary advice for patients to abstain from those materials led to the disappearance of symptoms.[15] Dysfunction or parafunction have been thought to have an aetiological value.[1,15,24] Individuals who grind teeth or clench can thrust their tongue against teeth leading to burning sensation especially in the lateral borders of the tongue. Dryness of the mouth or reduced saliva is a frequent complaint in patients with BMS.[2,5,25]

Systemic factors

Although, many systemic factors have been shown to be associated with BMS like symptoms, most of the investigators have not detected any consistent specific medical condition that is causally linked to BMS. Deficiency status of iron and vitamins including vitamin B1, B2, B6 and B 12 can lead to mucosal burning complaints.[26] Iron deficiency anaemia has been considered as an important aetiological factor in causing burning symptoms in the mouth.[7,15] However, recent studies have shown a non significant relationship of such deficiencies with BMS.[4,26] Diabetes mellitus has also been recognized as a causative factor for burning mouth symptoms.[15] Gibson et al.[27] have investigated 43 previously undiagnosed non-insulin dependent diabetes mellitus patients and showed that16 of them had burning mouth symptoms which disappeared after therapy for diabetes. Burning mouth symptoms may be attributed to metabolic alteration and oral candidal infection in diabetic patients.[1] Grushka [4] has suggested an immunologic basis for BMS. He has shown that 58% of BMS patients are associated with some immunologic abnormality. Reflux oesophagitis was also found as a factor in causing BMS.[15] However, no further studies have been conducted in this regard. Many drugs can cause side effects in the mouth. Some of them may directly or indirectly cause burning mouth symptoms.[28]

Neural mechanisms

It is likely that the BMS is a reflection of a neuropathic condition involving either central or peripheral mechanism or both.[20, 25] Nerve injury or dysfunction due to oral, facial and systemic trauma or certain medical conditions may lead to such changes in the central nervous system.[20,29] Lack of significant link with known diseases and no apparent abnormalities in the oral mucosa may support the above assumption. Burning pain is one of the features of several chronic pain conditions associated with nerve damage.[25] These changes in the central nervous system are considered to be due to neuroplasticity in the central nociceptive pathways. Damage or inflammation of the peripheral tissues or nerves can produce long lasting neuroplastic changes in the central nervous system. This central sensitization may lead to hyperalgesia, spontaneous pain and pain referral.[29] Therefore, it can be assumed that BMS is a central nervous system abnormality. The peripheral nerve alteration which leads to tonic input to the CNS can be responsible for burning sensation. This can occur in inflammatory conditions or nerve injuries. However, this mechanism in long term burning is doubtful.[25] Furthermore, local anaesthesia or topical analgesic solutions are ineffective in alleviating burning mouth symptoms.[25, 30]

Psychological factors

Psychiatric morbidity has frequently been cited as a causally related factor in BMS.[14,16,31-36] However, the cause and effect relationship is not very clear. Browning et al.[33] found that significantly a higher proportion of patients with a burning mouth (44%) had an associated psychiatric disorder compared to the control group (16%). Depression was reported more frequently in the burning mouth syndrome group. Rojo et al.[36] reported psychiatric involvement in 51% of patients with BMS. Depression was the commonest finding with BMS (31%) and anxiety was much less common (11%). On the contrary, Lamey and Lamb[34] claimed that anxiety to be more relevant than depression using Hospital Anxiety and Depression scale (HAD) to patients with BMS. Lamb et al.[37] had reported psychiatric morbidity in more than 50% of patients who suffer from BMS. A higher incidence was found in type II than in type I BMS. In a clinical study Humpf et al.[32] identified that 92% of patients with dysaesthesia were psychotic.

Twelve patients out of 34 who suffered from atypical facial pain were found to be psychotic and 7 out of 10 patients with BMS were suffering from pathological personality disorder. An underlying psychological problem, though the exact nature was uncertain has been found in many cases of chronic orofacial pain including orofacial pain and oral dysaesthesia.[21,32,38] Schoenberg[31] stated that the burning mouth is a symptom of depression resulting from psychological stress. The specific psychological stress could be a real or threatened loss of a loved person, a related object or a bodily function. Cancerphobia also has been found to be a significant factor. Lamey and Lewis[11] stated that approximately 20% of BMS patients were cancerphobic. In most of the studies carried out in the past, attempts have been made to ascertain the presence of psychiatric illness in association with BMS. However, they do not provide a causal relationship with BMS. Nevertheless, it is important to evaluate psychiatric involvement with regard to successful management of BMS. Indeed the psychiatric disorder could be a result of chronic pain or BMS rather than the opposite way.

Menopause and BMS

It is evident that BMS is more common in postmenopausal women.[3,8,13] Menopause (climacteric) is a natural process of cessation of menstruation which eventually occurs in all females. Climacteric period in which substantial hormonal changes occur is characterized by physical and emotional changes. Among them are vasomotor changes such as hot flushes, profuse perspiration, palpitations and psychological symptoms such as depression, irritability, tiredness, nervousness and other complaints such as headaches, insomnia and vaginal discomfort.[39] Oral discomfort with burning sensation is also a common complaint. Wardrop et al.[13] reported that the prevalence of depression, nervousness, apprehension and headaches were significantly higher in postmenopausal women who experienced oral discomfort than who did not. Basker et al.[8] reported oral discomfort in 26% of postmenopausal women. Out of this, 43% complained of only burning sensation, 27% had strange taste and 30% a combination of both. These figures are contrastingly higher than the prevalence in the general population[8] and premenopausal women.[13] Furthermore, Grushka[4] has shown that post-menopausal women experience burning mouth at about the time of menopause and symptoms are more severe than in the female control subjects. It has been claimed that hormonal changes that occur in menopause are responsible for the discomfort in the mouth.[3] Wordrop et al.[13] argued that the success of estrogen replacement therapy (ERT) in BMS is due to alleviation of psychological distress. Forabosco et al.[40] argued that the success of ERT in relieving BMS is attributed to the presence of estrogen receptors in the oral mucosa. However, some studies have not demonstrated a dramatic improvement of oral symptoms with ERT.[4,8,41] Although, it is difficult to explain the exact association with menopause, it appears that there may be a link, as yet unclear with the psychological changes that occur at menopause.

Assessment

It is important to assess the patient's condition properly prior to any treatment. Most of the patients probably would have consulted numerous specialists, all of whom would have proclaimed that the oral mucosa is healthy. Furthermore, these patients may claim that they have been treated with many medications with unsuccessful results. Some of the patients may believe that their problem is attributed to oral cancer or any other sinister disease. It is of utmost importance to be sympathetic towards the patient's problem. In addition to the pain history, medical and psychosocial evaluations are mandatory.[2] In all

circumstances, regardless of the extent and severity of the symptoms, a meticulous inspection of the oral cavity should be carried out. All mucosal changes should be thoroughly inspected. In addition to the examination of the mucosa, any signs of dry mouth, salivary consistency and flow should be determined.[3] If any positive finding is present, the appropriate tests including salivary analysis, allergytests, haematological and biochemical investigations.should be carried out to confirm the diagnosis. The need for specific laboratory tests should be assessed individually. Haematological investigations should include full blood count, differential count and blood picture. Other biochemical tests should include, serum iron, total iron-binding capacity, serum folate, serum B_{12}, B_1, B_2 and B_6 levels. In addition, the fasting blood sugar level also should be carried out to exclude diabetes in suspected cases. Furthermore, it is necessary to obtain a second opinion from a psychiatrist, haematologist, rheumatologist, allergist or physician wherever appropriate. When the organic cause for burning symptoms cannot be found the diagnosis of idiopathic BMS should be established. In other words the diagnosis of BMS is established by elimination of possible causes for the burning mouth symptoms.

Treatment

If an underlying cause for the burning mouth symptoms is found, the treatment should be directed towards the elimination of such a cause or causes.[5,24,42] If several causes are identified they should be addressed simultaneously.[42] However, treatment based on any of those suspected aetiologic factors is often ineffective in idiopathic BMS.[2,25] The treatment of idiopathic BMS is generally difficult. A long-term strategic approach should be used. Psychiatric referral may be necessary in some patients and a joint clinical management with a psychiatrist may be beneficial in successful management.[2] Self-regulation strategies such as diversion, distraction and thought stopping should always be discussed with the patient. After being properly informed about the various aspects of BMS, most patients do not insist on any type of drug treatment.[16] Elimination of cancerphobia is extremely valuable for those who suffer from it.[3] In any case the patient should be offered a follow up appointment in about 3-6 months time in order to evaluate the success of the management. However, in spite of proper explanation and reassurance some patients may demand further investigations and treatment.

Drug treatment

In the absence of identifiable causes pharmacological therapy has been suggested for the treatment of BMS. Among them antidepressants, benzodiazepines play a significant role.[2,24] Antidepressants such as amitriptyline or imipramine are useful for chronic pain disorders including BMS.[5] Their effect is mainly analgesic independent of antidepressant effects.[43] These drugs function by inhibiting the re-uptake of serotonin and norepinephrine.[1] As with chronic pain, improvement can be achieved in some patients with a low dose of 10-20 mg as a single dose at night. These medications should be taken regularly over some weeks before a change can be expected. Newer serotonin specific re-uptake inhibitors (SSRI) such as fluoxetine and paroxetine are also available. However, the efficacy of these agents in BMS or chronic orofacial pain is doubtful.[2] Nevertheless, they can be certainly beneficial for depressed patients with BMS.[1]

Anxiolytics also have been tried in some studies and the administration of chlordiazepoxide has some beneficial effects. Gorsky et al.[5] in a clinical study of 78 patients have shown marked improvement of symptoms in 49% of patients with administration of chlordiazepoxide and a 66% improvement in a small sample of 6 patients with

the administration of diazepam. However, clinically observable benefits were not found from benzodiazepines in the treatment of BMS.[2] A new modality of treatment was reported in the recent past using a neuropeptide " capsaicin".[44] Capsaicin, a neuropeptide extracted from hot pepper, has been shown to reduce and eliminate the accumulation of substance P. However, the author concludes that further controlled studies are needed to clarify the efficacy on neuropathic pain. The use of viscous solution of 2% lidocaine hydrochloride in carboxymethyl cellulose sodium also had been used as a symptomatic relief.[3] It has been shown that oral rinse of benzydamine hydrochloride have no significant efficacy in comparison with placebo solution.[30]

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RESEARCH ARTICLE

Recurrence of disease and survival of oral cancer patients treated at the Faculty of Dental Sciences, Peradeniya

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Abstract

Introduction: Pattern of local spread, metastasis and recurrence of oral cancer may be different in different societies where different aetiological factors could be in operation. Survival rate of patients would depend on these factors. ***Material and method*:** Hospital records of thirty three live patients and fifty dead patients were studied to determine personal details, site of the lesion, method of treatment, recurrence and period of survival after initial treatment. ***Results*:** Overall recurrence rate within three years was 50.6% and in 25.3% of these cases the recurrence had occurred during the first year after treatment. Three year survival rate was 69.8% and the five year survival rate was 39.7% and in both parameters the patients who had local resection only and those who had surgery and radiotherapy recorded higher survival rates than other groups. ***Discussion*:** Recurrence rate and three year survival rate compares favourably with the findings of previous studies. Five year survival rate appears to be poor in comparison. More comprehensive treatment modalities and predictive investigations may have to be employed for better results.

Key words: oral cancer, recurrence, survival rate, treatment protocol, neck nodes

Introduction

Natural behaviour with regard to local spread, metastasis and recurrence of oral cancer could be different when the aetiological factors are different. Aetiological factors in operation may differ from country to country, and society to society. Tobacco habits which is the main cause may be different in different countries. Some may smoke it in vaiours forms, some may chew it with different other substances, others may use it as a snuff. Behaviour pattern of oral cancer could be different in these different societies. Thus it may be necessary to determine the survival rate of oral cancer patients in Sri Lanka. This retrospective study is an attempt to do this using patients treated at the Oral Surgery Department, Faculty of Dental Sciences, Peradeniya.

Material and Method

One hundred and three patients who had been treated for oral cancer at the Department of Oral Surgery, Faculty of Dental Sciences, University of Peradeniya, prior to August 1994 were recalled in the month of August 1999. The letters sent to these patients were addressed to their next of kin who were requested to inform us if the patient was not alive giving us the date the patient died. They were also requested to let us know whether the patient had been treated elsewhere after the last date he/she attended our unit. Thirty Five patients attended our clinics while in the case of 54 the next of kin replied giving the date on which the patient had died. Thus, there was a total compliance rate of 89.3%. The hospital records of these eighty six patients were perused to

determine the personal details, site of the lesion, method of treatment and recurrence and its management, and period of survival after initial treatment. Four subjects were rejected as the information could not be accurately determined due to incomplete records. Thirty three of the patients who attended the clinic and whose records were satisfactory were included in the study and were further examined clinically to determine whether there was any recurrence and/or metastasis and also whether they had been treated elsewhere after the last date he/she had attended our unit. The final study group consisted of thirty three live patients and 50 patients who had died prior to August 1999. It was assumed that these 50 patients had died due to the oral cancer and its direct complications. In the statistical analysis the chi-square test was employed to determine the significance of difference between proportions.

Results

Age at time of initial diagnosis and sex distribution is shown in Table 1. A large majority (74.7%) of patients were in the age groups 50-59 and 60-69. Similarly there was a preponderance of males (84.3%).

Distribution of patients according to the site of the cancer is given in Table 2.The commonest site was the buccal mucosa and the next common site was the commisure and lip area. Cancer was found to be more common in the floor of the month than in the tongue. Palate was the least common site.

Table 1: Age (at initial diagnosis) and sex distribution of the study group

Age Group	Live		Dead		Total
	Male	Female	Male	Female	
40-49	04	03	06	01	14 (16.9%)
50-59	11	02	14	05	32 (38.6%)
60-69	10	0	19	01	30 (36.1%)
70-79	03	0	03	0	06 (7.2%)
80-89	0	0	0	01	01 (1.2%)
Total	28 (33.7%)	05 (6.1%)	42 (50.6%)	8 (9.6%)	83 (100%)

Table 2: Distribution according to site of primary lesion

Site	Male (%)	Female (%)	Total (%)
Buccal	23 (27.7%)	5 (6.0%)	28 (33.7%)
Commisure & Lip	20 (24.1%)	3 (3.6%)	23 (27.7%)
Tongue	10 (12.0%)	2 (2.5%)	12 (14.5%)
Floor	11 (13.3 %)	3 (3.6%)	14 (16.9%)
Palate	06 (7.2%)	0	06 (7.2%)
Total	70 (84.3%)	13 (15.7%)	83 (100%)

Methods employed in the treatment of these patients are given in Table 3. A combination of surgery and radio therapy was the most frequent (45.8%) method of treatment. Surgery involving neck dissection had been done in 18 (21.7%) patients, local resection only in 14 (16.9%) and radical resection without neck dissection in 13 (15.6%). In a total of 42 (50.6%) patients there had been recurrence of the disease which needed further treatment.

Table 4 shows the recurrence of disease after initial treatment. Twenty one (25.3%) of these patients had the recurrence during the first year after treatment, 9 (10.8%) during the second year and 12 (14.5%) during the third year. The commonest site of recurrence was the primary site of the lesion. A total of 24 (28.9%) patients which amounts to more than half (57%) had a recurrence at the primary site. Recurrence in the cervical lymph nodes was detected in 17 (20.5%). Distant metastasis was rare with only one patient being detected with it.

Crude three year survival rate in relation to the method of treatment is shown in Table 5. Patients numbering 14 who had a local resection only had a survival rate of 100%. Of the rest of the patients those who had radical surgery and radio therapy had recorded a three year survival rate of 68.4% while those who had surgery and neck dissection had recorded a survival rate of 61.1% and those who had radical resection without neck dissection recorded a survival rate of 53.8%. The difference between these proportions was statistically significant ($p<0.01$). The overall crude three year survival rate was 69.8%.

Table 3: Treatment method

Local Resection	Radical Resection	Resection & Neck Dissection	Surgery & Radiotherapy	Total
14 (16.9%)	13 (15.6%)	18 (21.7%)	38 (45.8%)	83 (100%)

Table 4 : Recurrence of disease after initial treatment

Site of Recurrence	Within First Year	Within Second Year	Within Third Year	Total
Primary	12 (14.4%)	5 (6.0%)	7 (8.5%)	24 (28.9%)
Neck nodes	09 (10.9%)	4 (4.8%)	4 (4.8%)	17 (20.5%)
Distant	0	0	01 (1.2%)	01 (1.2%)
Total	21 (25.3%)	9 (10.8%)	12 (14.5%)	42 (50.6%)

Table 5 : Three Year survival rate in relation to method of treatment

Method of Treatment	Three years after initial treatment			Survival Rate
	Alive	Dead	Total	
Local	14	0	14	100%
Radical	7	6	13	53.8%
Neck Diss.	11	7	18	61.1%
Surgery+RT	26	12	38	68.4%
Total	58	25	83	69.8%

RT – Radio Therapy

Table 6 presents the crude five year survival rate in relation to the method of treatment. The overall five year survival rate was 39.7%. The five year survival rate in patients who had local resection was 85.7%, that in patients who had radical resection was 30.7%, in patients who had neck dissections it was 16.6% and in those who had a combination of surgery and radiotherapy it was 36.8%. The difference between these proportions was statistically significant ($p<0.01$). Three year survival rate in relation to age and sex is presented in Table 7. The overall three year survival rate for males was 70% while it was 61.5% for females, the difference being statistically significant ($p<0.01$).There was no significant difference in the overall three year survival rate for the age groups 40-49, 50-59, and 60-69 ($p<0.01$). Within each gender however there were statistically significant differences in the survival rate between age groups.

Table 6 : Five year survival rate in relation to method of treatment

Method of Treatment	Five years after initial treatment			Survival Rate %
	Alive	Dead	Total	
Local	12	02	14	85.7
Radical	04	09	13	30.7
Neck Diss.	03	15	18	16.6
Surgery+RT	14	24	38	36.8
Total	33	50	83	39.7

RT – Radio Therapy

Table 7 : Three year survival rate in relation to age and sex

Age Group	Male			Female			Total		
	Alive	Dead	SR %	Alive	Dead	SR %	Alive	Dead	SR %
40-49	7	3	70	3	1	75	10	4	71.4
50-59	19	6	76	3	4	42.8	24	10	70.5
60-69	21	8	72.4	1	0	100	20	8	71.4
70-79	3	3	50	0	0		3	3	50
80-89	0	0		1	0	100	1	0	100
Total	50	20	70	8	5	61.5	58	25	69.8

SR – Survival Rate

Discussion

This series of patients was homogeneous in the sense that two surgeons were responsible for the surgery and two oncologists were responsible for radiotherapy. However this was a retrospective study that relied heavily on hospital records which could be a source of error. One major draw back was the fact that the oral cancers had not been satisfactorily staged according to an acceptable

method. Further details of radiotherapy was not available and there was no way of controlling this variable factor. Yet as only two surgeons and two oncologists were involved in the treatment it was not difficult to ascertain that treatment regimens were based on acceptable protocols. Due to the above constraints the investigation into recurrence and survival could not be related to the stage of malignancy but was related to less important criteria such as method of treatment, age and sex. However the results of the study are useful as they give a good idea of recurrence rates and survival rates of oral cancer patients in Sri Lanka and when these are related to treatment protocols useful information could be gleaned from the data so analyzed.

The overall recurrence rate of oral cancer was found to be 50.6% which compares favourably with figures reported in other studies.[1,2,3] Commonest site of recurrence was the primary site and in half of the patients the recurrence was observed during the first year. This could mean inadequate marginal resection. Invaded resection margins were found to be a factor in recurrence in previous studies[4]. The finding that most of the recurrence occurred during the first two years postoperatively is in agreement with that of other studies[3]. Next common site for recurrence was the cervical lymph nodes. Whether this finding supports the view that neck nodes should be treated irrespective of whether or not they are clinically affected could not be clearly ascertained due to lack of proper records. However a nodal recurrence rate of 20.5% could be considered as satisfactory when compared with the 32% reported by Pradier et al.[3] who recommend nodal clearance for early stage tumours. In this series neck nodes had been treated only when they were clinically affected.

The overall three year survival rate of 69.8% compares favourably with figures reported in other studies. [5,4,1] Local resection without neck dissection and/or radiotherapy had given the best survival rate of 100%. This finding cannot be taken at its face value. It is probable that these patients had Stage I or II tumours which required only a local resection. The five year survival rate (85.7%) too was satisfactory for these patients. Thus the question arises whether the early stage tumours in Sri Lankan patients need to be treated with neck dissection and also radiotherapy. If they are subjected to such intensive treatment perhaps their survival would improve further. This point of view is supported by other authors.[6,5] Raybaud et al.[7] say that it has been observed that tumours of the same grade or stage do not necessarily behave similarly; small tumours may show a high recurrence and mortality rate whereas larger tumours may fail to recur. Such behaviour is not commonly observed in Sri Lankan patients. However it may be necessary to identify more reliable prognostic indicators and plan treatment accordingly. For instance radio-resistance could be predicted using p53 protein accumulation[8] and tumour DNA content.[7] Such expensive investigations may not always be possible in Sri Lanka but what could be appropriate is the use of multifactorial histologic grading system for malignancy [9,10] in combination with clinical TNM staging system in order to acquire a more accurate basis for prognosis prediction and choice of treatment.[11]

Apart from the group who had local resection only, the highest three year and also five year survival rates were recorded by the group who had a combination of radical surgery and radiotherapy. Similar findings have been reported by other authors.[5,1] Patients who had neck dissection without radiotherapy had a better three year survival rate (61.1%) compared to those who had only radical resection (53.8%) ($p<0.01$). However this order was reversed in the five year survival rate, indicating that in the patients who did need a

neck dissection (lymph nodes affected) the disease process had been more extensive than in those who did not. In this context the possibility of extra nodal spread must be taken into consideration. Shingaki et al.[12] found extra nodal spread to be present in 46% of neck dissection specimens and also they reported that five year survival rate of patients with extra nodal spread was 40% compared to 72% in those without. A feature of extra nodal spread is the increased frequency of distant metastasis. In the present series distant metastasis was detected in only one patient. However this finding is not reliable as the screening for distant metastasis has not been very satisfactory especially in the patients who had died. The possibility that they died due to distant metastasis cannot be ruled out. Therefore, the policy regarding the management of cervical lymph nodes have to be reviewed. Clinical palpability alone may not be a satisfactory criterion.

The frequency of false negative neck nodes could be significantly high when clinical examination was relied upon to detect lymph node metastasis.[13] Ishii et al.[14] found that ultra sound and CT examination were superior to conventional clinical examination in detecting metastatic nodes. On the other hand Woolgar et al.[15] have concluded that the most stringent clinical protocols supplemented by CT scanning cannot be expected to achieve 100% accuracy. They stated that detailed histopathological assessment provides the most reliable currently available method of diagnosing cervical metastatic disease. Thus the Sri Lankan cancer surgeon is in a quandary as to the best approach in the management of cervical lymph nodes. Costly diagnostic methods may not be always available whereas clinical methods may be inadequate.

Another weakness of the study is that the survival rates have not been corrected for age and sex. There was significant difference in the survival rate between age groups in each gender and also between male and female. However when males and females were taken together there was no significant difference in the three year survival rate between age groups; 40-49, 50-59 and 60-69 to which the large majority (91.6%) of patients belonged.

This study raises more questions than it provides answers. Thus there is a need to study the natural behaviour of oral cancer in Sri Lanka more thoroughly and develop protocols for its accurate diagnosis and adequate treatment.

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CASE REPORT

Cervical mucocele – an unusual presentation of a ranula

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Introduction

The term "ranula" is used to denote a "blue–domed" cystic lesion of the floor of the mouth that has some resemblance to a frog's belly. Ranulae usually present as unilateral swellings of the floor of the mouth. They have been classified as superficial and deep (plunging). [1] A superficial ranula is submucosal and lies entirely above the mylohyoid muscle and the deep ranula lies partly superficial and partly deep to the mylohyoid muscle.[2] Intraoral presentation of ranula can be easily recognized as a bluish fluctuant swelling in the floor of the mouth and usually does not pose a diagnostic difficulty.

Plunging ranulae though rare, are well documented as neck swellings. [3-8] On some occasions plunging ranulae can occur as extraoral swellings without an associated intraoral swelling. [5,7] Such a lesion must be differentiated clinically from a thyroglossal cyst, dermoid cyst, branchial cyst, lymphangioma, laryngocele, lipoma, haemangioma, cyst of the parathyroid and thyroid glands, abscesses or tumours. [4] However as specific tests are not available to diagnose ranulae, it may be difficult for a clinician to differentiate it from other extra oral swellings that occur in this region.[9]

Histologically, a plunging ranula does not have an epithelial lining. Hence, it is called a pseudocyst. The cyst wall of a ranula is made by compressed fibrous connective tissue containing fibroblasts, vascular channels and inflammatory cells. [6]

The treatment of choice for such a cyst is extirpation of the secreting salivary gland via intra-oral route. It is not necessary to excise the cervical extension of the cyst since it does not contain a true cyst lining. [4,8]

This case report describes an unusual presentation of a plunging ranula in that the patient presented only with cervical swelling. The probable pathogenesis is discussed.

Case Report

A 16-year-old girl was brought by her parents to the Oral Medicine Clinic, University Dental Hospital, Peradeniya. She had complained of a swelling under the lower jaw on the left side. The swelling was observed over a period of three weeks and had gradually enlarged. In addition, she had noted the enlargement of the swelling during mealtimes. Her past medical history was insignificant. Physical examination revealed that she was a healthy looking well grown girl. There was a swelling of 5 cm in diameter over the submandibular region (Figure 1)

There was no tenderness on palpation and the lesion was fluctuant. The lower border of the mandible was intact. The lesion became more evident when the patient blew her nose which indicated that raised intraoral pressure pushed the lesion into the neck. There was no evidence of intraoral swelling. Fine needle aspiration was carried out and a viscous straw coloured fluid was found. (Figure 2)

Ultrasound scan of the swelling revealed the cystic nature of the swelling (Figure 3)

The diagnosis of a plunging ranula was made and the patient was referred to the Department of Oral Surgery for the extirpation of the sublingual salivary gland of the left side. However, the patient was reluctant to undergo surgery and had got herself discharged.

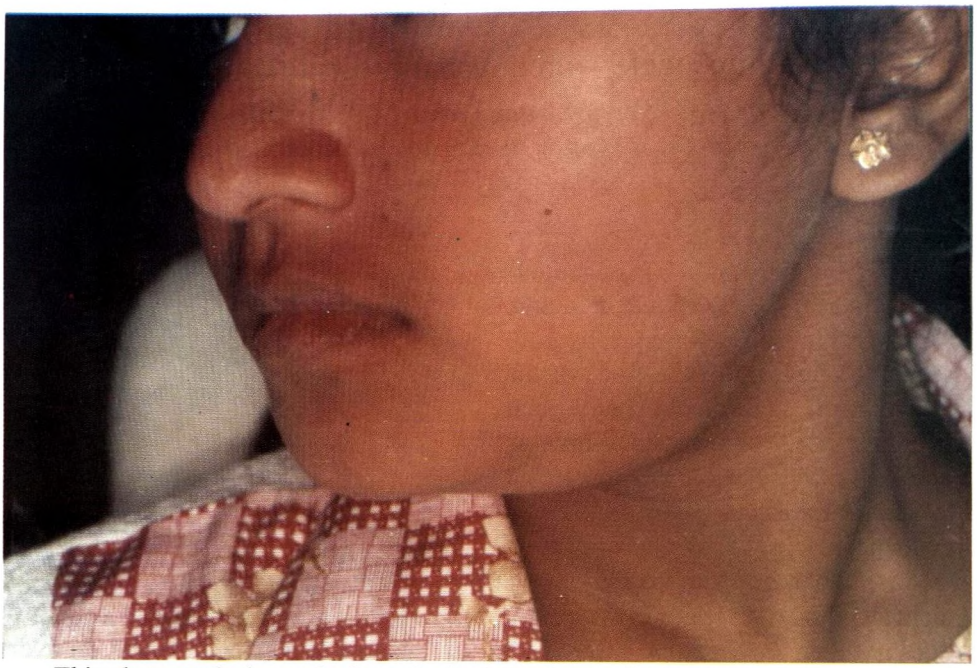


Figure 1 This photograph shows the lump in the left submandibular region.

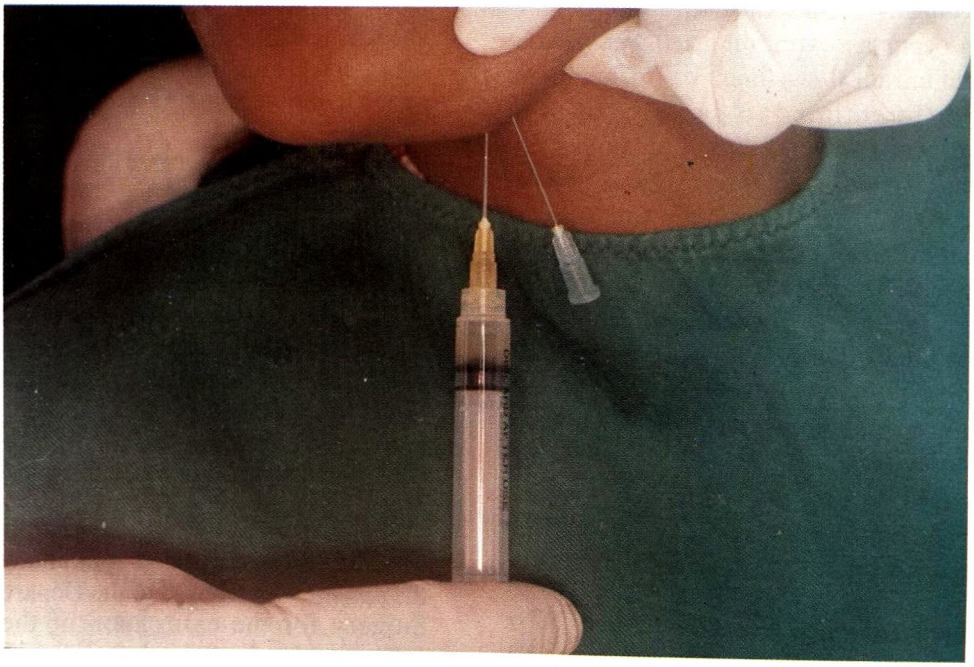
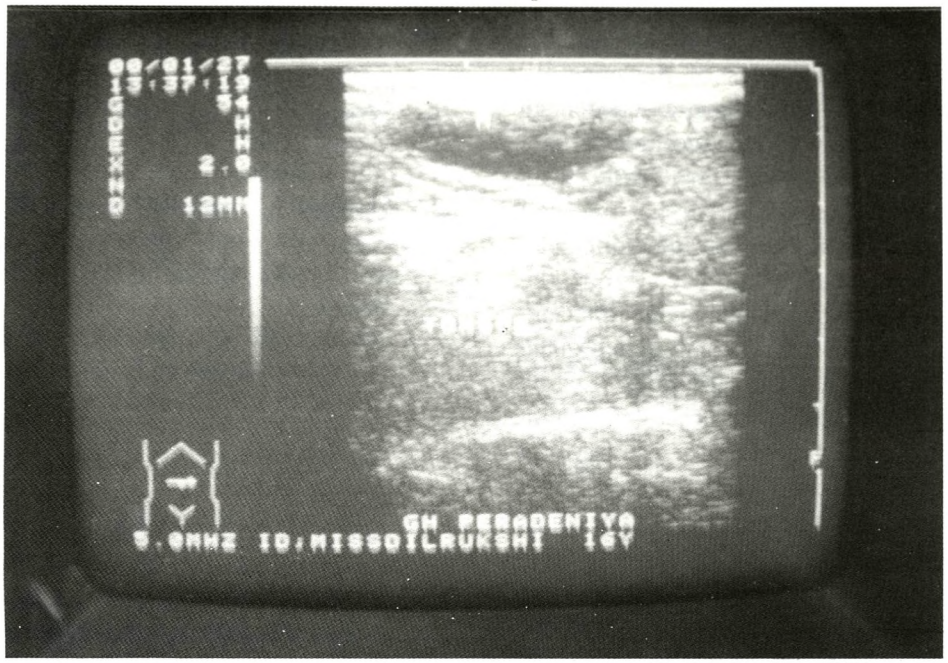


Figure 2 The aspiration procedure showing withdrawal of straw-coloured viscous fluid.



3. This photograph of the ultrasound scan shows the cystic nature of the lesion in the submandibular region.

Discussion

When extra-oral swelling is the only sign, the diagnosis of a plunging ranula poses an obvious problem. There would have been no doubt that the lesion was a plunging ranula if a ranula was evident on the floor of the mouth. However, aspiration of straw coloured viscous saliva from the submandibular swelling led us to think that the swelling was of salivary gland origin. Furthermore, a possibility of haemangioma was eliminated easily due to the colour of the aspirate. Epidermoid or dermoid cysts invariably present as a symmetrical midline swelling, whereas branchial cysts are confined to the lateral neck according to the developmental origin. Therefore, they were excluded from the differential diagnosis. Possibility of an abscess was excluded due to the absence of inflammatory features.

There is little value of sialography and ultrasound scanning in identifying the origin of a plunging ranula.[8] Takimoto[10] described a radiological technique in which herniation of a ranula to the neck region could be detected. In this technique radiographs of the submandibular region were taken after injecting 0.5ml of radiopaque medium into the sublingual space. If the contrast medium extends into the neck region on the radiograph, it indicates a plunging ranula. However, this was not possible since the patient was not co-operative for invasive procedures. High protein and amylase levels in the salivary aspirate may pave the way to a diagnosis of a ranula. [3] However, in this patient the salivary amylase and protein analyses were not done due to the non-availability of facilities. Therefore, a process of elimination established a probable diagnosis of a plunging ranula herniating through the mylohyoid muscle. However, histopathological diagnosis was not possible since the patient refused surgery.

A plunging ranula develops as a result of

herniation of a ranula through the mylohyoid muscle and along the fascial planes of the neck. [11] However, pathogenesis of plunging ranulae remain controversial. There was considerable debate regarding as to which salivary gland was responsible for the lesion and it is now well established that it originates from the sublingual gland. Roediger et al.[3] reported that the development of a plunging ranula is due to the extravasation of mucous from the sublingual gland which ramifies diffusely into the neck. Lyall[7] reported that a cervical extension of a ranula appears only when the patient blows his nose or at times while eating. Barnard[8] has reported a case of bilateral ranulae that arose asynchronously as cervical swellings.

Gaughran[12] described a deficiency or hiatus in the mylohyoid muscle in one third of the population through which a portion of sublingual salivary gland may herniate. Lyall[7] pointed out that certain. activities which increase intraoral pressure such as nose blowing, eating and swallowing can encourage herniation of tissues through weak points in the mylohyoid muscle. Therefore, this anomalous herniation of the gland certainly encourages the accumulation of mucous in the submandibular region rather than in the mouth itself.

Furthermore, ectopic sublingual glands superficial to the mylohyoid muscle may also be a source of a cervical ranulae[9], though much attention has not been paid in the literature to this theory. The presence of ectopic sublingual glands or herniation of a part of the sublingual gland was not revealed by the ultrasound report. Furthermore, a surgical verification could not be obtained as the patient refused surgery.

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Journals

Standard journal article

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Corporate (collective) author

WHO COLLABORATING CENTRE FOR ORAL PRECANCEROUS LESIONS. Definition of leukoplakia and related lesions: an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol 1978; 46: 518-539.

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Boyde A. Amelogenesis and the structure of enamel. In: Cohen B, Kramer KH (eds). Scientific Foundations of Dentistry. William Heinemann Medical Books Ltd. London, 1976; 335-352.

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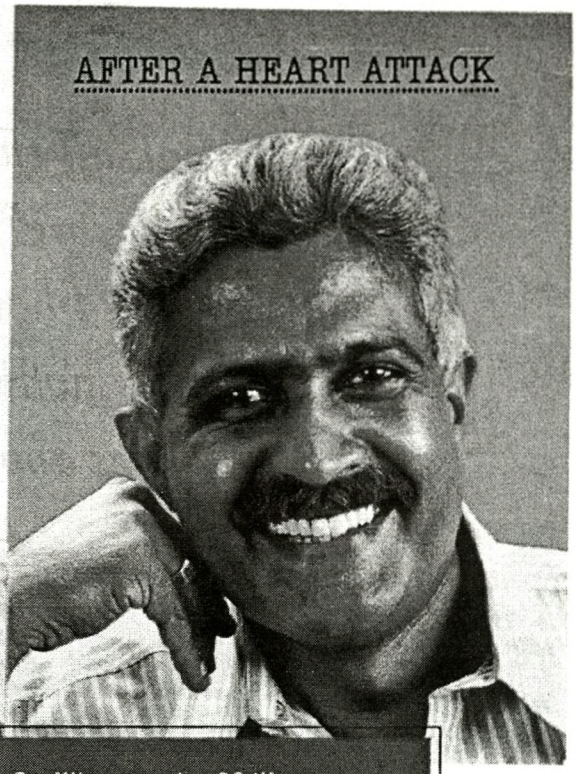

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