



SRI LANKA DENTAL JOURNAL

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- * Alcohol and maxillofacial fractures
- * Effects of irradiation on salivary glands
- * A low cost filter to prevent dental fluorosis
- * Melanotic neuroectodermal tumor
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EDITORIAL

Accreditation

Products of diverse nature, quality and quantity of composition were found in the market at any given time both in the past and now at present. The difference in the amount of products in the foreseeable future is bound to increase. On-going research and advances in technology in the production of various items of produce contributes largely towards this boom. The increase in population is another factor which increases a demand for marketable products. Yet to the consumer the quality of products had only the often unsubstantiated claim of the manufacturer regarding its quality and efficacy in the not so distant past.

To illustrate, time was when, not very long ago, that the manufacturer of an infant milk food came out with a claim that their product was better than mother's milk, albeit unsubstantiated. Not surprisingly, to give of the best to their infants the general public of the day discontinued breast feeding of infants as they preferred this milk food because of the claim made regarding this infant milk. There were no Standards Bureau or similar Institution established at that time which could put the claim to test. However the medical Profession of the day, by sheer intuition felt that this claim could not be accurate. It was left to the conscience of the members of the then Medical Profession, whom the Manufacturer could ill-afford to displease, to protest at this abominable claim. The Manufacturer obliged to the extent of then claiming that his infant milk food was as good as mother's milk. The Medical Profession was not pleased. They argued that nothing surpasses or even equals a mother's milk to any infant in the entire animal world. They then grudgingly changed the advertisement to read "next to mother's milk". At that time there was no established body to verify even this claim. The Medical Profession was therefore compelled to refrain from further protestation for want of a better alternative. However the noteworthy feature was that the consumer was saved from the abominable claim and an ensuing disaster.

With the passage of time and the introduction on the market of products in abundance both in nature and quantity, government of various countries saw the wisdom of establishing organizations to standardize the quality of various products. Parliaments also appointed bodies to survey and monitor that products conformed to laid down criteria and penal measures

incorporated to punish erring manufacturers and producers. These functions were assigned to these bodies to protect the consumer from frivolous claims and sub-standard products. The latest move of the Sri Lanka Government towards this goal is the establishment of a Department of Measurement Units, standards and services as a new dimension to consumer protection in this age of market economy and a free market policy. This would be as an adjunct to the existing institutions in this field and established by the Government.

Though this be so, there are yet certain "grey areas" in this exercise. There will be professional bodies who would in most disciplines want to assess and certify that any product in addition to conforming to the criteria laid down by the above organizations would also meet the requirements of that profession. As an instance certain manufacturers have sought the accreditation of tooth-pastes and tooth-brushes as conforming to professional standards and criteria from the Sri Lanka Dental Association (SLDA). This would give suitable recognition of the product to the manufacturer and a reliable recommendation to the consumer - unlike in the earlier mentioned infant milk food fiasco.

Towards this end, the SLDA has with the unanimous approval of the general membership devolved upon the Council of this Association to accredit in the first instance tooth-brushes and tooth-pastes - much used items in the everyday life of a very high percentage of the Sri Lankan population. Accreditation or the popular synonym - endorsement, of such products by the National Body of the Dental Profession, in this case the SLDA, is not unique to Sri Lanka. It is much in vogue among the National Dental Associations of most developed countries including Great Britain, the United States of America and Australia, to mention a few countries.

It is worthy of mention that certain Members of the Medical Profession viewed this accreditation or endorsement, with raised eye brows. They were even concerned whether this action by the SLDA could be unethical. Yet it has to be understood that this meaningful step taken by this Association has been in the larger interest of the general public. Should they pause to

consider the valiant efforts of the Members of the Medical Profession of a by-gone era to safeguard the general public from the spurious claim of an infant milk food producer mentioned herein earlier, they would then acclaim that this action by the SLDA is commendable and a step in the right direction. This Association has filled a void, nay an astronomical black-hole to inform the consumer.

In granting accreditation or endorsement to these two products great pains have been taken to subject the products to stringent and rigid tests to ensure that they conform to the minimum standards laid down. The Association enlisted the support and services of the Ceylon Institute of Scientific and Industrial Research (CISIR) and the Sri Lanka Standards Institution (SLSI). These two bodies independently checked the conformity of laid down standards of the two products. The Manufacturer was then required by the SLDA to suitably alter the products to eliminate certain features of inadequacy to bring the products to the required Sri Lanka standards. The University of Colombo also helped to check the fluoride content of the toothpaste. These were finally evaluated by a sub-committee of the SLDA to determine that the requirements of the dental profession has been well met. The report of this sub-Committee was finally presented to the general membership of the SLDA who unanimously approved the accreditation or endorsement of the two products. It is fitting to mention herein that this process took three years to complete.

The end result is that the SLDA has been able to recommend a tooth-brush and a tooth-paste the consumer could use with confidence. This confidence would be an on-going feature as the SLDA is due to re-validate the accreditation or endorsement bi-annually to bring in any new research or technology into these products at re-validation. Furthermore the products are being monitored by the SLDA periodically and the accreditation or endorsement will be withdrawn if the products show any drop standards or quality.

Like in marriage, this exercise was undertaken not lightly or hastily but with much thought and attention to details to ensure conformity of standards and professional suitability.

Oral Manifestations of Systemic Diseases in Childhood.

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Most oral lesions in childhood occur as a result of poor oral hygiene causing carious teeth and gingivitis. However systemic diseases too could produce certain abnormalities in the oral cavity, some of which could be diagnostic (such as Koplik spots in measles).

The oral cavity starts from the lips and ends at the palatopharyngeal folds. The diseases are described below under their anatomical location.

* *The lips*

Unilateral or bilateral clefts of the upper lips are among the most common causes of congenital malformations. Cleft of the palate is a frequent association. In about one third of these babies there is a history of a cleft in a family member. Cleft of the lips and palate could be associated with defects in other systems such as gastrointestinal, cardiovascular, nervous system and musculoskeletal, or it could be a part of a syndrome such as Patau's (Trisomy 13) and Pierre Robin syndrome. The cleft could also be seen in babies following maternal Phenytoin therapy during pregnancy.

Dryness of lips with cracking may be due to mouth breathing which is frequently seen in children with enlarged adenoids obstructing the nasal airway. A reddened cracked lips are a feature of Kawasaki disease which is characterized by high fever, non-purulent conjunctivitis, oral changes including strawberry tongue, erythematous skin rash involving the face, trunk and extremities and lymphadenopathy. Some of these patients with Kawasaki disease develop coronary artery aneurysms.

Ulceration of lips could be due to burning as a result of ingestion of poison containing acid or alkali or as a result of infection around the mouth such as impetigo and herpes simplex (herpes labialis). Impetigo is a highly contagious skin infection caused by staphylococcus aureus, Group

A Streptococcus or a combination of these organism. The lesions begin with a transient vesicle or a bullous on an erythematous base and then the vesicles or bullae rupture and produce a thick yellow crust below which there is a moist superficial erosion. Herpes labialis is a recurrent infection which is caused by Herpes simplex type 1. As in adults, various factors such as fever and exposure to sun light may reactivate the virus. It appears as one or few lesions on and around the lips. These lesions initially appear as clear fluid filled vesicles, which later rupture and get crusted.

Several nutritional deficiencies may cause fissuring and inflammation at the angle of the mouth (Angular cheilitis) such as iron deficiency anaemia, Riboflavin and Niacin deficiencies. The angular cheilitis could also be due to chronic drooling of saliva with swallowing disorders such as in patients with cerebral palsy.

Cheilitis (inflammation of lips) may occur as a result of Stevens-Johnson syndrome. Generalized swelling of lips could result from acute anaphylactic reaction as in angioneurotic oedema.

Perioral depigmentation is a feature of vitiligo which is an acquired pigmentary defect. The depigmented well circumscribed lesions appear often on the face particularly around the eyes or the mouth. The cutaneous eruption of acrodermatitis enteropathica can affect the perioral skin. This is a rare inherited disorder of Zinc deficiency. The disease usually manifest in young children as vesiculo-bullous, eczematous and psoriasiform skin rash in the perioral, acral and perineal areas. L1

* *The oral mucosa*

Ulceration of oral mucosa most commonly occurs due to simple aphthous ulcers. These may be precipitated by local trauma. They heal spontaneously within 7-14 days. Recurrent and persistent aphthous ulceration occurs in

children with poor oral hygiene and in those with inflammatory bowel diseases such as Crohn's disease.

Extensive ulceration of oral mucosa occurs with ingestion of acid or alkali. It is also seen in Stevens-Johnson syndrome which is characterized by a vesiculo-bullous skin lesion, oral mucosal ulceration and genital ulceration. There is sometimes conjunctival ulceration and secondary infection. This could be due to infections such as herpes simplex and mycoplasma or drugs such as co-trimoxazole and sulfonamide.

Infections of the oral mucosa in children commonly occur with Herpes simplex type I virus (herpetic gingivostomatitis). It affects children often between 1 and 5 years. The stomatitis begins with fever, malaise, refusing to eat and drink, dribbling of saliva and foul smelling breath. Lesions appear as white plaques on the tongue, palate, gums, buccal mucosa, and tonsils. These plaques soon become shallow whitish yellow ulcers. Regional lymph nodes are enlarged and tender. The condition is self limiting over a period of 7-10 days. Chicken pox also affects the oral mucosa. Lesions which are similar to those on the skin appear in the oral mucosa causing pain during eating and drinking. Petechial haemorrhages are seen on the palatal mucosa in children with rubella infection and infectious mononucleosis. Koplik spots which are pathognomonic of measles are seen on the buccal mucosa near the posterior molar tooth as grayish white lesions usually as smaller as grains of sand. They appear and disappear rapidly, usually within 12 - 18 hours.

Oral candidiasis or thrush commonly occurs in young babies who become infected from their mothers or from infected babies. The disease manifests as white plaques which adhere to the tongue, buccal mucosa, gums and palate. Candidiasis could affect babies receiving long term systemic antibiotics and those who are immunosuppressed. The appearance of generalized pallor in the oral mucosa as well as in the tongue is a feature of anaemia of any kind. In addition, in iron deficiency anaemia, there is atrophy of the oral mucosa and atrophy of papillae in the tongue causing a smooth tongue (glossitis).

In pancytopenia (eg. aplastic anaemia) apart from pallor of the mucosa, there are petechiae, purpuric spots and gingival bleeding.

Central cyanosis manifests as purplish blue colour on the mucosa and tongue in diseases such as congenital heart

disease with a right to left shunt, acute severe asthma and acute severe bronchiolitis.

Abnormal pigmentation may be seen in Addison's disease and long standing thalassaemic patients who receive recurrent blood transfusion. In Addison's disease there are bluish brown macules on the buccal mucosa. Hyperpigmentation of the skin is also a feature of this disease. A bluish gray discoloration is seen in haemosiderosis due to repeated transfusion of blood in patients with Beta thalassaemia major. A bluish brown pigmentation along the gum margin is seen in heavy metal poisoning such as lead and mercury. Several genetically transmitted diseases can lead to abnormal pigmentation. They include Peutz-Jeghers syndrome and multiple neurofibromatosis. Peutz-Jeghers syndrome is characterized by melanotic macules on the lips and oral mucosa and by gastrointestinal polyposis. Multiple neurofibromatosis causes irregular brownish black macules in the oral mucosa.

* *The Gums*

Infection of the gums (gingivitis) in children is often a result of poor oral hygiene. Gingival hypertrophy is a feature of long term Phenytoin therapy. This is also seen in children with myelomonocytic and monoclastic leukaemias, and histiocytosis-X Haemorrhagic diseases such as haemophilia, von Willebrand's disease could present with gingival bleeding though local diseases such as gingivitis and trauma are the commonest causes of gum bleeding. Vitamin C deficiency too could manifest as gum bleeding.

* *The tongue*

Macroglossia (large tongue) is a feature in hypothyroidism, Beckwith Wiedemann syndrome and glycogen storage disease type 2. The tongue can be enlarged by a lymphangioma or a haemangioma. The latter could be associated with haemangiomas elsewhere. The normal sized tongue looks bigger in patients with Down's syndrome as they have a relatively small mouth.

The surface of the tongue becomes coated in patients with poor oral hygiene, dehydration and in conditions like typhoid fever and acute appendicitis.

In scarlet fever the tongue first becomes white and coated, then later becomes red, hence the name 'strawberry'.

Smooth tongue with no papillae is a feature of iron deficiency and megaloblastic anaemias, oral candidiasis and in a rare condition called familial dysautonomia. In megaloblastic anaemia, in addition the tongue becomes beefy red which is called Hunter's glossitis.

Fasciculation of the tongue is a feature of acute organophosphate poisoning and spinal muscular atrophy. Spinal muscular atrophy is a hereditary disease which manifests in very young babies as profound hypotonia (Werdnig-Hoffmann disease). It is progressive and the majority die within the first year of life.

* *The teeth*

The commonest problem in children is dental caries which is mainly related to poor oral hygiene. In addition, there are some diseases which could affect teeth. The primary dentition can be delayed in hypothyroidism, rickets, congenital syphilis and cleidocranial dysostosis. Premature shedding of primary teeth can occur in a rare condition known as hypophosphatasia and in mercury poisoning. Enamel hypoplasia is a feature of kernicterus, vitamin D deficient rickets, congenital rubella and congenital syphilis. Vitamin D resistant rickets causes dentine abnormality.

Shape of the teeth may be abnormal as a consequence of congenital syphilis (notched incisors) or in ectodermal dysplasia where the teeth may be peg shaped.

A brown discoloration of teeth is a feature in children when tetracycline is administered to the mother during late pregnancy or to children below 8 years.

Fluorosis is characterized by mottling of teeth with chalky white patches and there is a secondary infiltration of yellow or brown staining. Fluorosis occurs as a result of high concentrations of fluoride in drinking water in areas like Polonnaruwa, Kurunegala, Anuradhapura and Hambantota. Syrups containing iron could cause a blackish discoloration of teeth near their gingival margin, where as a green discoloration is a feature of kernicterus.

* *The salivary glands*

The commonest condition affecting salivary glands in children is mumps. It is an acute viral infection caused by mumps virus (Myxovirus parotidis) and in as many as 30% of cases the infection is inapparent. When the disease is manifested there may be a prodromal illness characterized by fever, malaise, headache and anorexia. Sometimes this prodrome is absent. The salivary gland involvement occurs 1-2 days after the prodrome and the parotids are the most frequently affected glands. Often only one parotid is involved but when both are swollen, they often reach their maximal size independently; in fact the first gland may return to its normal size before the other gland is involved.

Acute bacterial parotitis is uncommon in children but it can occur in neonates and in debilitated children.

Recurrent parotitis either unilateral or bilateral may occur where the symptoms are generally mild. The condition is self limiting and generally resolves by puberty.

Oral manifestations of systemic diseases in children often help in making a diagnosis. Thus awareness of these features would alert the clinicians to look for the underlying systemic condition.

REVIEW

Differential Diagnosis of Periapical Radiolucencies

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Summary

Periapical radiolucencies are commonly found in intraoral radiographs. Anatomic radiolucencies are caused by superimposition of the shadows of anatomical cavities, canals and foramina. The commonest cause of true periapical radiolucencies is pulpitis and its sequelae with 90% of pathologic periapical radiolucencies being either periapical granulomas or periodontal cysts. The uncommon conditions include periapical cementomas, periodontal disease, cysts, benign tumors and primary or secondary malignant tumors.

Introduction

Radiolucent areas in the region of the apices of teeth are commonly seen in intra-oral radiographs. Some of these are anatomic radiolucencies, whereas some others may be benign conditions which require treatment in order to preserve the tooth concerned. Very rarely, they represent oral manifestations of systemic diseases and malignancy. Therefore, it is most important for the dental clinicians to require a comprehensive working knowledge of these conditions.

All periapical radiolucencies may be readily divided into two groups - True or false. True periapical radiolucencies are produced by lesions that are located in contact with the apex of a root and it is not possible to shift their shadow from the region, by taking additional radiographs at different angles.

False periapical radiolucencies are produced by anatomic cavities, canals or lytic bony lesions, which are not in contact with the apex of the root, hence they can be shifted from the periapical region by taking radiographs at different angles.

Anatomic Radiolucencies

The shadow of a distant anatomic structure can get superimposed on the periapical region and the clinician should be aware of the normal location and appearance of the anatomical cavities, canals and foramina that produce radiolucencies with their normal range of sizes and variations. Also if the radiolucency is anatomic, a similar radiograph of the contralateral side will frequently reveal the same findings. Furthermore the past history, clinical findings and special investigations like vitality tests aid in the differential diagnosis.

Anatomic radiolucencies that are commonly seen in an intraoral radiograph may be,

1. Dental papillae
2. Maxillary sinus
3. Incisive foramen & canals
4. Naris
5. Nasolacrimal duct
6. Greater palatine foramen
7. Mental foramen
8. Mandibular canal (Inferior alveolar canal)
9. Marrow spaces
10. Submandibular fossa

True Periapical Radiolucencies

Pulpitis and its sequelae can be highlighted as the most important cause of periapical radiolucencies, as the commonest periapical lesions, initially share a common cause - irritating inflammatory products from a non vital pulp. These periapical lesions also share several clinical characteristics.

1. The periapical lesion is radiolucent
2. The associated roots are non vital
3. The crown may be discolored, with deep caries or have large restorations, close to the pulp.

4. The crown may be partially or completely missing, following trauma.
5. A past history of irreversible pulpitis and periapical infection may be present.

It is stated that the clinician is sometimes unable to differentiate between various periapical lesions purely on the basis of the history or by the clinical and radiological examination findings. However, in most instances, a working diagnosis can be arrived at, from a well conceived differential diagnosis and approximately 90% of the pathologic periapical lesions are noted to be either periapical granulomas or periodontal cysts.

Another factor to note is that 30% to 60% of the regional bone have to be destroyed for a radiolucency to be detected on a radiograph. Therefore an actively enlarging lesion might actually be slightly larger than it appears on radiographs.

Radiolucencies associated with periapical pathosis

1. Periapical granulomas
2. Periodontal or radicular cysts
3. Scars
4. Dentoalveolar abscesses
5. Osteomyelitis
6. Surgical defects

1. Periapical granulomas

The periapical granuloma represents the most common type of pathological radiolucency. The reported incidence of periapical granulomas range from 45% (Lalonde and Leubke 1968) to 94% (Block et al 1976) with an approximate incidence of 70% (Mean of 14 studies from 1954 to 1988).

Radiological features

The earliest periapical radiological change seen is thickening of the periodontal space and loss of lamina dura. As the lesion enlarges, it produces a well circumscribed radiolucency which is circular in shape, surrounding the apex of the tooth. Sometimes a thin radiopaque line or zone of sclerotic bone may be seen outlining the lesion and that indicates that the granuloma concerned is a slowly progressing lesion of long duration. It also indicates that the lesion has probably not undergone an active exacerbation of infection. The periphery in

other instances appears to blend with the surrounding bone. Occasionally some degree of root resorption is also found.

A periapical granuloma cannot be differentiated from a dental cyst by its radiological appearance alone.

A widely accepted earlier view was that periapical cysts could be differentiated from periapical granulomas on the basis of their larger size - more than 1 cm in diameter. However, it is very unlikely for a granuloma to be more than 1.6 cm in diameter (Lalonde 1970).

2. Periodontal or radicular cysts

The apical periodontal cyst is a common but not inevitable sequelae of a granuloma. The reported incidence ranges from 6% (Block 1976) to 54% (Priebe 1954) with a mean incidence of 26% of all periapical radiolucencies and periodontal cysts constitute 60-70% of all cysts.

Radiological features

The radiological appearance of a radicular cyst is almost identical to a granuloma in most instances. However, lesions with a radio opaque border and a diameter of more than 1.6 cm in diameter are much more likely to be cysts. Root resorption also can be present and a darker area in the radiolucency denotes the thinning and perforation of one or both cortical plates.

3. Scars

Scar tissue represents one of the possible end points of healing. The periapical scar is composed of dense fibrous tissue and is situated at the periapex of a successfully root filled tooth. 2% to 5% of periapical radiolucencies are estimated to be periapical scars, but little information is available in the literature regarding this entity.

When a tooth is successfully treated with non surgical endodontic therapy, periapical granulomas and cysts frequently resolve. Being replaced by bone, they are no longer apparent on radiographs. However in some instances, the granulation tissue slowly organizes with production of collagen fibres giving rise to a dense connective tissue scar. These are quite permanent and radiolucent.

The periapical scar produces a well circumscribed, circular radiolucency that resemble a granuloma or a cyst, but on serial radiographic examination, no change in the size (either an increase or decrease of the lesion) is evident.

4. Chronic and acute denoalveolar abscesses

Abscesses make up about 2% of all periapical radiolucencies. Primary abscesses or acute periapical abscesses/Periodontitis do not produce any radiolucency, as the associated teeth do not have existing apical lesions.

Secondary or recurrent abscesses develop in existing asymptomatic periapical lesions and therefore produce radiolucencies. Depending on the primary lesion the size and extent of the radiolucency may vary. In chronic cases the margin is blurred giving rise to an area of somewhat lessened density than the surrounding bone. The roots of the teeth concerned may also show apical resorption.

5. Surgical defects

A Surgical defect arises if the surgical area fails to fill in with osseous tissue following surgery. It is frequently seen following surgery for large lesions, when both labial and palatal plates have been destroyed. Approximately 45% of all periapical radiolucencies treated surgically, usually require 1 to 10 years for complete resolution and another 30% is said to take longer than 10 years. The remaining 25% of the surgical defects do not heal completely and it represents an area where the cortical plate is absent. In addition to the defect in the cortical plate, a periapical scar also may be present. Surgical defects account for about 3% of all periapical radiolucencies.

The periapical radiolucency produced by a surgical defect is round with smooth contours and a well defined border. It is usually small and does not exceed 1 cm in diameter. The radiolucent shadow may be directly over the projected apex or slightly away from the resected root of an endodontically treated tooth. The tooth and periapical area are completely asymptomatic. A careful clinical examination may reveal the mucosal scar from past surgery and if the defect is large enough it may be detected by palpation.

6. Osteomyelitis

Very rarely a periapical abscess can develop into an acute or a chronic osteomyelitis, specially in patients who have

an underlying systemic disorder that has depressed their resistance to infection or who have received radiation therapy to the jaw.

Acute osteomyelitis is similar to an acute primary dentoalveolar abscess and as the onset and course is rapid, bone resorption may not be present. Therefore a radiolucency may not be evident, initially. Chronic osteomyelitis on the other hand represents a low grade infection of bone, which if left untreated follows a protracted course of bone destruction and deposition. Therefore, it can give rise to lesions which may be completely radiolucent, mixed radiolucent and opaque or even completely radio opaque. Another important point to note is that osteomyelitis is extremely rare in the maxilla, where the other periapical radiolucencies described are commonly seen.

Uncommon lesions

1. Periapical cementomas

The periapical cementomas, cemental dysplasia and cementifying or ossifying fibromas in their initial or osteolytic stage, can present as radiolucent lesions. However, the associated tooth is vital with no coronal abnormality.

2. Periodontal disease

Advanced periodontal disease can give rise to periapical radiolucencies. The entire bony support of the involved tooth may be completely destroyed and the tooth will appear to be floating in the radiolucency. Sometimes a narrow vertical pocket extending to the apex can appear in certain projections, as a well defined radiolucency.

3. Other cysts

Although dentigerous cysts form adjacent to or surrounding the crowns of teeth, sometimes the position of the crown of the tooth is such that the pericoronal radiolucency is projected over the apex of a neighboring tooth. Very rarely the shadow of a circumferential or a lateral dentigerous cyst can be projected over the same tooth, giving rise to a diagnostic dilemma.

Solitary bone cyst, aneurysmal bone cyst, incisive canal cyst, mid palatal cyst and odontogenic keratocyst are some other cysts which can give rise to periapical radiolucencies.

4. Malignant tumors

malignant tumors either primary or secondary, can give rise to periapical radiolucencies. Tumors that produce this image are squamous cell carcinoma, malignant tumors of minor salivary glands, osteogenic sarcoma, chondrosarcoma, melanoma, fibrosarcoma, multiple myeloma and metastatic deposits from lung, gastrointestinal tract, breast, thyroid gland and kidneys.

Malignant tumors eg. squamous cell carcinoma, a usually erode much bone before they reach the apex. So they usually do not produce an isolated radiolucency. The apical area is included in a large radiolucency with ragged borders. On the other hand low grade, slow growing malignant tumors, eg. some salivary gland tumors, that destroy the cortical plate slowly, can appear as a well defined radiolucency. Mesenchymal malignant tumors and metastatic tumors originating within bone, can produce more localized periapical lesions.

The two basic but important principles of periodic clinical and radiological examination of endodontically treated teeth and histological examination of any surgical specimen will alert the clinician of this rare but extremely important entity in the event of an initial misdiagnosis.

5. Rarities

There are so many pathological entities, that at times, can present as periapical radiolucencies. Some of them may be Ameloblastoma, Giant cell granuloma, Giant cell lesions of hyperparathyroidism, Histiocytosis X and Leukaemia.

The differential diagnosis of these seemingly simple periapical radiolucencies is sometimes not quite easy and the clinician must always be alert to recognize the possibility of a rare lesion or an unusual presentation.

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Influence of alcohol in the causation of maxillofacial fractures in Sri Lanka.

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Summary

Alcohol is one of the main contributory factors in the causation of maxillofacial fractures. In order to investigate this aspect 818 fracture patients were presented with a questionnaire inquiring whether they had consumed alcohol prior to the incident that caused the injury. Two hundred and sixty seven patients returned the completed questionnaire and it was found that in road traffic accidents which caused facial fractures influence of alcohol could be implicated in 32.1%. With regard to assault 38.3% of the victims claimed that their assailants were intoxicated. Similarly 32.5% of patients whose fractures were caused by a fall stated they were under the influence of alcohol at the time of the accident. Though the results are not supported with more reliable information such as blood alcohol levels they could be considered as providing valuable information which reveals the need for enactment of legislation against alcohol abuse.

Key words: *Alcohol, Maxillofacial fractures, Road traffic accidents, Assault, Fall.*

Introduction

The common causes of Maxillofacial Fractures (MFF) in Sri Lanka are Road Traffic Accidents (RTA), Assault and Falls (Amaratunga, 1991). There may be other less common causes such as sports accidents, agricultural and industrial accidents etc. It is obvious that alcohol could influence all these causative factors to a greater or lesser degree. Surveys in Australia revealed that 25% of driver casualties had a blood-alcohol concentration over the legal limit (McDermott, 1976). In western societies one of the main causes of interpersonal altercation resulting in facial

injuries is alcohol (McDade et al, 1982). Similarly falls from a height as well as from the standing position could be commonly caused by intoxication (Hill et al 1984). There is evidence that even in sports intoxication may be a factor in the causation of a small proportion of accidents (Lamberg, 1978). Furthermore Lamberg (1978) reported that 4% of work accidents were due to alcohol related negligence.

In Sri Lanka MFF caused by RTA and Assault have been increasing over the last two decades (Amaratunga 1991). It may be necessary to find out the degree of influence of alcohol in the causation of MFF in Sri Lanka so that preventive and safety measures and appropriate legislation could be proposed for enactment.

Material and Method

In the present study only the common causes of MFF; RTA, Assault and Fall were investigated with regard to influence of alcohol. Three hundred and ninety patients who had received MFF due to RTA, 225 assault victims with MFF and 203 patients who sustained MFF due to falls formed the subjects of this retrospective study. Only males were selected for this study as alcohol consumption is rare among Sri Lankan Women.

These patients were treated by the author during the period 1970 - 1989 at the Oral Surgery Department, General Hospital Kandy. They were presented with questionnaires designed to obtain information regarding the consumption of alcohol before the incident that resulted in the facial injury. The questionnaire inquired about the type of liquor the quantity and whether it was consumed within three hours prior to the incident. It also questioned about the alcohol consumption habits of the patient. The patient

was given the liberty to remain anonymous and it was explained that the inquiry was made for the purpose of a scientific investigation and that there were no legal implications whatsoever. For purposes of comparison patients were grouped into; 1. those who took spirits (Arrack, Whisky etc.), 2. those who took beer and with regards to the quantity ; 1. those who took more than 04 fluid ounces (118ml) of spirits, 2. those who took less, 3.

he had consumed less than 04 fluid ounces of spirits prior to the incident. None apparently had taken more than 03 ounces. However 36(38.3%) of the assault victims claimed that their assailants were under the influence of alcohol at the time of the incident.

Eighty six (42.4%) of the subjects who had received MFF due to a fall returned the questionnaire. Of these

Table - Number (&%) of MFF patients who had consumed alcohol within three hours before the incident.

Cause of Fracture	Type & Quantity of alcohol		Spirit		Beer		Number who returned the questionnaire
	>4 oz	< 4 oz	>1 bottle	< 1 bottle			
RTA	5 (5.8%)	13 (14.8%)	0	5 (5.8%)	87 (23.3%)		
Assault	0	1(1.2%)	0	0	94 (41.8%)		
Fall	28 (32.5%)	11(12.8%)	0	0	86 (42.4%)		

Those who took more than one bottle of beer and 4. those who took less. RTA victims were classified as drivers, riders and pedestrians.

Results

Only 87 subjects (22.3%) out of the RTA victims returned the completed questionnaire. Of these 02 (2.3%) drivers, 01(1.2%) rider and 02(2.3%) pedestrians had stated they had consumed more than 04 ounces of spirits within three hours prior to the accident. All these people were habitual drinkers who consumed more than 10 fluid ounces of arrack almost everyday.

Four drivers (4.6%), 03(3.4%) riders and 06(6.8%) pedestrians had drunk less than 04 fluid ounces of spirits prior to the accident. These people too had the habit of drinking almost daily. A further 03 drivers and 02 riders had stated that they had consumed less than one bottle of beer. Six pedestrians and 4 drivers claimed that to the best of their knowledge drivers of the antagonist vehicle involved in the accident were intoxicated at the time of the accident.

Ninety four (41.8%) out of 225 assault victims responded to the questionnaire. Only one of these people had said

49(57.0%) had fallen from the standing position while 37(43.0%) had fallen from a height. Twenty one (42.9%) in the former group and 7 (18.9%) in the latter had taken more than 04 fluid ounces of spirit within three hours before the accident. A further eleven subjects belonging to the group that fell from the standing position had consumed less than 04 fluid ounces. None had stated they had taken beer.

Discussion

The study revealed that 5.8% of RTA victims had taken more than 04 fluid ounces of spirit which is the amount of liquor considered to be capable of causing measurable impairment of reflexes and coordination of movements for about three hours (Voss, 1982). This amount of liquor would result in a blood alcohol level of 0.05% which is the legal limit for driving in most Scandinavian countries. In the USA the legal limit of blood alcohol level for driving is 0.08%. Apart from this there was a further 14.8% of RTA victims who had consumed less than 04 fluid ounces of Arrack. All these people were habitual drinkers. Furthermore there were another 10(11.5%) RTA victims who claimed that to the best of their knowledge drivers

involved in the accidents in which they received the injuries were intoxicated at the time of the accident. If all this information is accepted as reliable there is evidence that in about 32.1% of RTA caused MFF influence of alcohol could be implicated. If reliability of the information is questioned it could be argued that people who are involved in RTA would not like to divulge the fact that they had consumed alcohol before the accident and therefore the proportion of people who drive or use the roads while being under the influence of alcohol could be more than the figure of 32.1%.

People who are involved in interpersonal violence would be even more reluctant to divulge the fact that they were under the influence of alcohol at the time of the incident. Though 41.8% of the subjects complied by returning the questionnaire only one confessed that he had taken liquor before the incident. However 38.3% of the assault victims claimed that their assailants were intoxicated at the time of the altercation. Unfortunately no corroborative evidence was available to support these claims. Police records were found to be incomplete with regards to consumption of alcohol by alleged assailants. It may be concluded that influence of alcohol in assault seems to be of a much lesser degree in Sri Lanka when compared to Western Countries. Thorn et al (1986) reported that 85% of male assailants who caused MFF in Greenland were found to be intoxicated. In these countries a majority of interpersonal altercations occur in public houses where liquor is sold and there do not seem to be any underlying animosity between assailant and victim and altercations seem to occur without preplanning (Voss, 1982). In Sri Lanka interpersonal altercations seemed to be caused by underlying animosity and rivalry and alcohol is not a direct causative factor. An assailant who had planned an attack may consume liquor to work up "Courage" (Amaratunga, 1991).

With regard to Fall 32.5% of patients whose MFF were caused by a fall had consumed more than 04 fluid ounces of spirit. A majority of these people had fallen from the standing position. People who had fallen from a height had done so while working in an elevated position and they may have been reluctant to divulge the fact that they had taken liquor while at work. However the above mentioned figures are comparable with those reported in Western societies. Donaldson (1961) reported that 32% of people who received MFF due to falls in New Zealand were intoxicated.

Results of the present study shows that there is a significant degree of influence of alcohol in the causation of facial injuries in Sri Lanka. Though the accuracy of the methods employed are questionable the results provide a definite answer to the question whether the influence of alcohol in the causation MFF is significant in Sri Lanka. Information gathered from the questionnaire could have been supported if more detailed police reports were available. Drivers of vehicles involved in accidents, and alleged assailants are not routinely tested for intoxication by the police. The study has to be viewed in the context of such constraints and the lack of exactitude has to be disregarded in view of the importance of the information it supplies. Thus there is a case for enactment of laws to ensure that intoxication does not contribute to the causation of injuries. It has been reported that in Western Societies advice alone had proved futile but appropriate legislation and their strict enforcement have resulted in a significant reduction in alcohol related injuries (Voss, 1982).

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Effects of Irradiation on Male Mouse Submandibular Salivary Gland. An Ultrastructural Study.

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Summary

Single dose head and neck X-irradiation in male mice induced changes to the submandibular gland. Ultrastructural studies revealed that at a time of 72 hours after irradiation the serous ductal cells showed more damage than the mucous acinar cells. In the serous ductal cells mitochondrial membrane damage, dilatation of endoplasmic reticula and cytoplasmic membrane damage were clearly seen in. The presence of electronlucent cells scattered among the ductal cells, and change in the electron density of the serous granules are new observations reported in this study. Most of these anomalies however are comparable to those observed in other conditions of the salivary glands.

Key words: Submandibular Salivary gland, Male Mouse, Irradiation, Histology, Ultrastructure

Introduction

In Sri Lanka orofacial cancers constitute 35% of all malignancies (Randeniya 1987) and rank third of most frequent cancers (WHO 1995). The treatment procedures for these cancers include irradiation of the head and neck area. During these radiation schedules the major salivary glands are often found included in the field of irradiation. This results in damage to salivary gland tissue leading to xerostomia and other complications. It is therefore relevant to understand the mechanisms underlying irradiation damage to the salivary glands for appropriate patient care.

The effects of irradiation on the salivary glands have been studied earlier using several techniques. Light microscopy has allowed examination of the parenchyma: changes

such as decrease in number and staining ability of intracellular secretory granules, occurrence of karyolytic and pyknotic nuclei, occlusion of lumina (Abok et al 1984, Emestrom 1988), cloudy swelling and excessive liberation of secretory granules (Indran et al 1989) have been reported. Furthermore, salivary cells with serous secretory granules seemed to be extremely radiosensitive (Sholley et al 1974, El mofty and Kahn 1981). The radiosensitivity of the serous cells resulting in progressive destruction was attributed to the release of heavy metal particles and proteolytic enzymes that are contained within the granules (Stern et al.,1976). This theory led to studies involving the depletion of serous granules prior to irradiation schedules.

Norberg and Lundquist (1988), administered an alpha adrenergic agonist namely cycloctidine to deplete the serous granules from rat submandibular glands before being exposed to radiation. The animals pretreated with cycloctidine have shown less parenchymal and organelle damage at the ultrastructural level as opposed to the untreated animals which have shown extensive damage including mitochondrial membrane damage and the breaking up of cytoplasmic granular substance. More recent studies however have shown that depletion of serous granules prior to radiation treatment does not reduce the radiosensitivity of serous cells in the rat submandibular and parotid glands (Peter et al 1994, 1995).

Other findings of radiation damage in the rat submandibular gland include bleb formation in the serous duct cells (Messelt and Dahl 1983), changes in local immunoglobulin secretion (Emestrom et al 1988) and loss of epidermal growth factor in the ductal cells (Reade and Steidler 1985).

Thus studies have reported damage to salivary gland parenchyma for over a decade but the exact mechanism responsible for such damage is not fully understood. This study therefore aims to observe irradiation change in the mouse submandibular salivary gland using techniques of light and transmission electron microscopy with a view to elucidate probable mechanisms underlying damage to salivary gland tissue.

Materials And Methods

Three groups of mice, a control group, a sham control group and an experimental group were used in the study. Each group consisted of twelve male BALB C mice of 30gm of body weight. The experimental group was exposed to a single dose of 15 Gray head and neck X - irradiation from a 14 MeV cobalt 60 teletherapy unit. The mice were strapped to perspex sheets

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Materials And methods

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The sham controls were also strapped to perspex sheets and were taken to the radiotherapy unit along with the experimental group, but were not exposed to radiation. The three groups of mice were housed separately in wire cages. They received a standard pelleted diet and water ad libitum.

Specimen preparation

The mice were killed by cervical dislocation at a time point of seventy two hours after the radiation exposure. The submandibular salivary glands were perfused with 5% glutaraldehyde solution, dissected out and were then fixed in 5% glutaraldehyde solution in phosphate buffer for forty eight hours. The tissues were prepared in the following way : 4 x 4 mm square pieces of salivary gland tissue were washed in phosphate buffer and were then subjected to further fixation for thirty minutes in 1% aqueous osmium tetroxide (Sabatini et al 1963). This was followed by processing through a graded series of alcohol. They were then transferred to three changes of propylene oxide, before being transferred to a mixture of equal amounts of propylene oxide and TAAB embedding resin for two hours. The samples were then embedded in TAAB resin and were kept in an oven at 45oC for forty five minutes before being increased to 65oC for five days to allow the resin to harden. 1(m sections were cut in a Reichert ultramicrotome using glass knives and were stained with toluidine blue for light microscopic observations.

Transmission electron microscopy

70nm sections of selected areas were stained with uranyl acetate (Watson 1958) and lead citrate (Reynolds 1963). The stained sections were examined in a JEOL 100 CX (II) Transmission Electron Microscope. Images were recorded on kodak EM cut film no 4489, Ester thick base of size 6.5 x 9.0 cm.

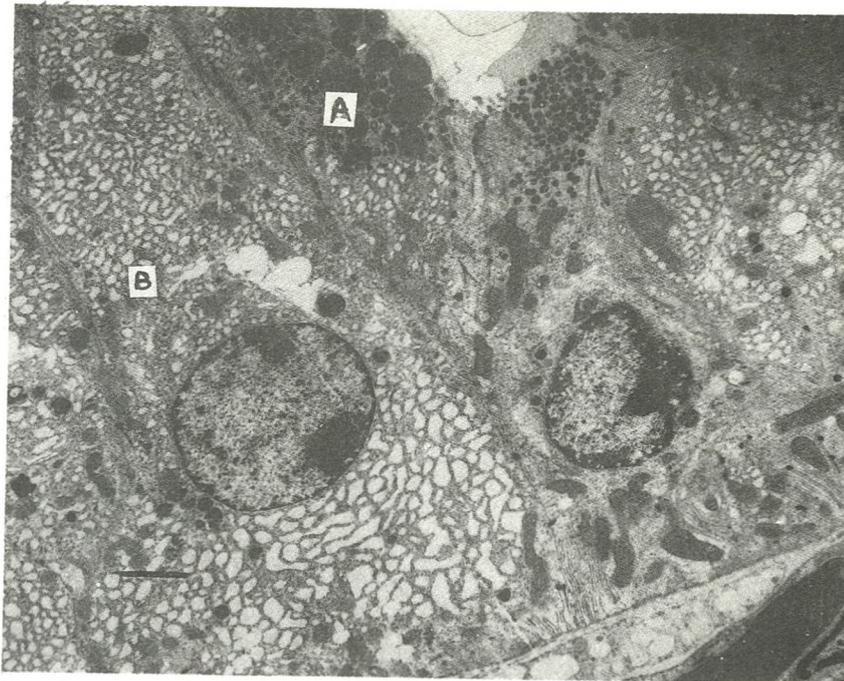


Figure 1: Ultrastructure of control granular duct. Granules (A), Steroid secreting cell(B). x 2000 Bar = 1(micron)

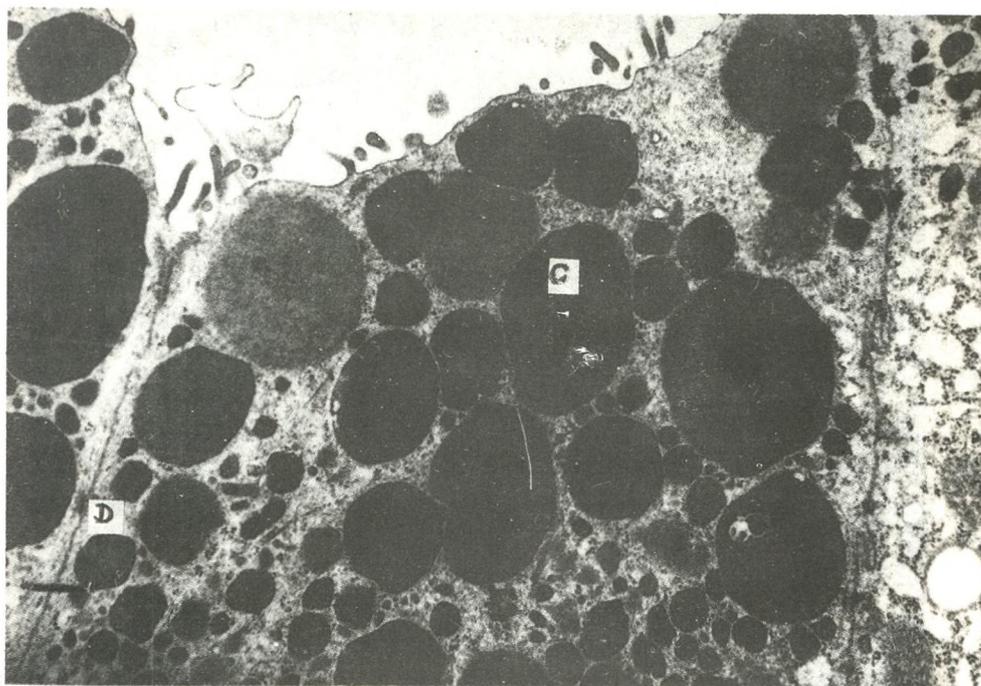


Figure 2: Ultrastructure of control granular duct cells. Granules (G), Desmosomal attachments (D). x 6700 Bar = 1(micron)

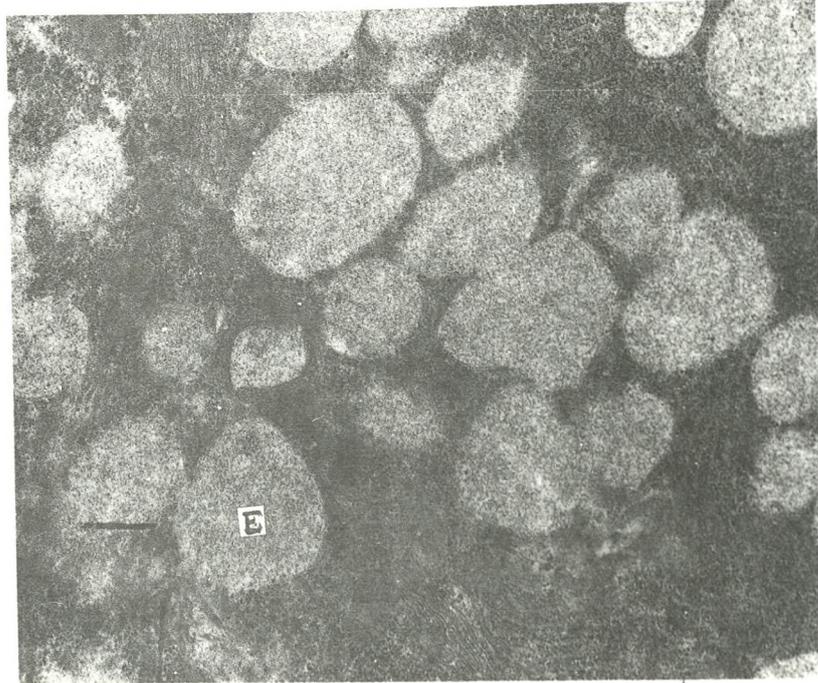


Figure 3: Ultrastructure of mucous acinar cell from control sample. Mucus globules (E) x 4000 Bar = 1(micron)

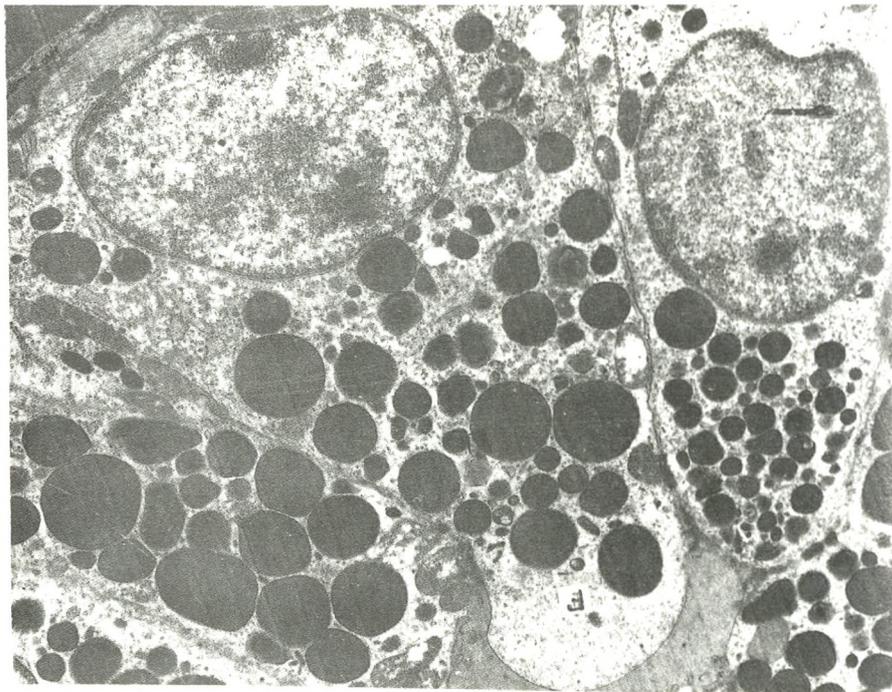


Figure 4: Ultrastructure of granular duct 72 hours after irradiation. Electron lucent cells among granular duct cells (F), x 2700 Bar = 1(micron)

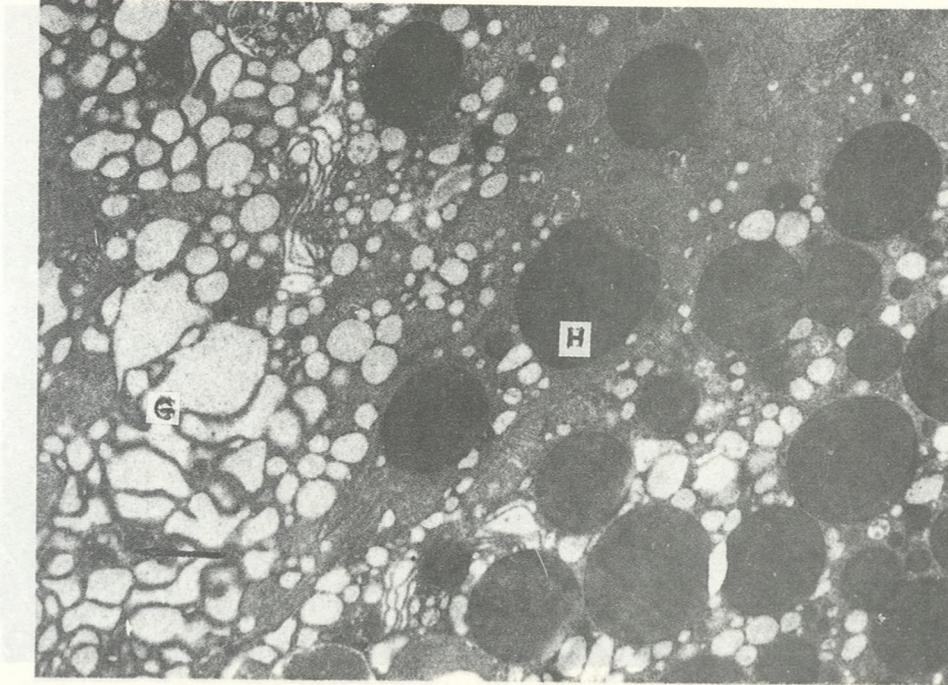


Figure 5: Ultrastructure of granular duct cells 72 hours after irradiation. Dilated endoplasmic reticula (G). The secretory granules are less electron dense (H). x 4000 Bar = 1(micron)

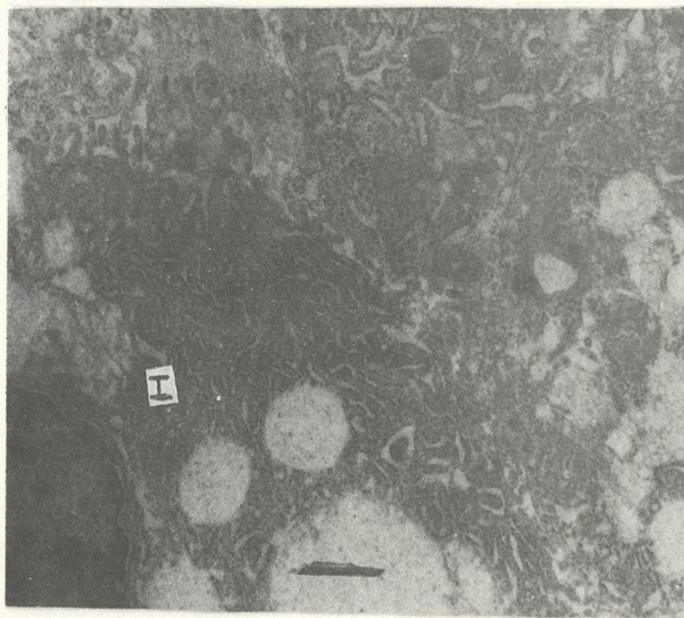


Figure 6: Ultrastructure of mucous acinar cells 72 hours after irradiation. Electron dense cell among mucous acinar cells (I). x 2700 Bar = 1(micron)

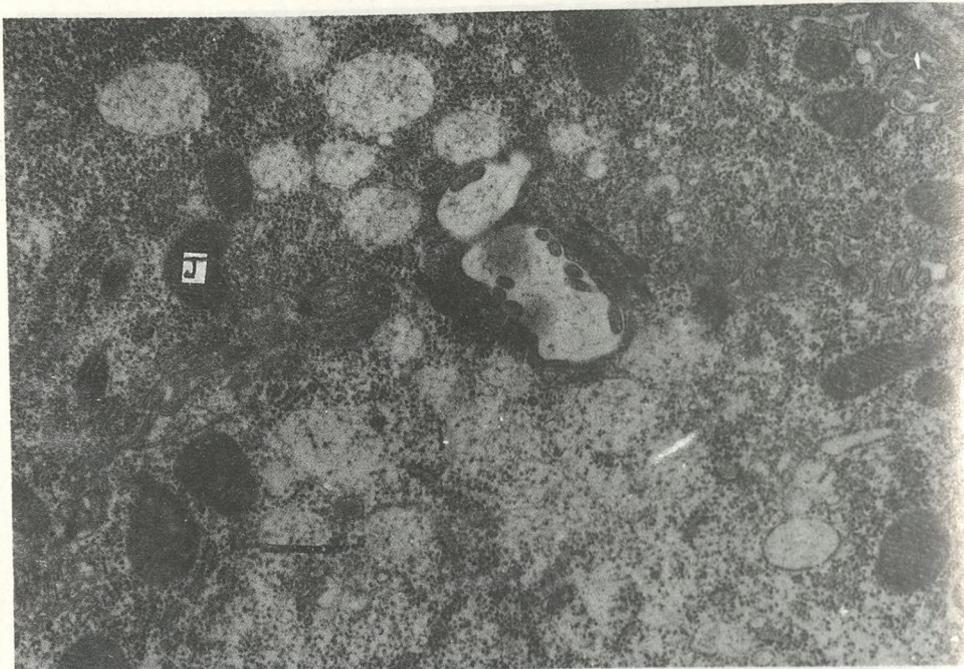


Figure 7: Ultrastructure of mucous acinar cell 72 hours after irradiation. Mitochondrial damage is not prominent (J). x 6700 Bar = 1(micron)

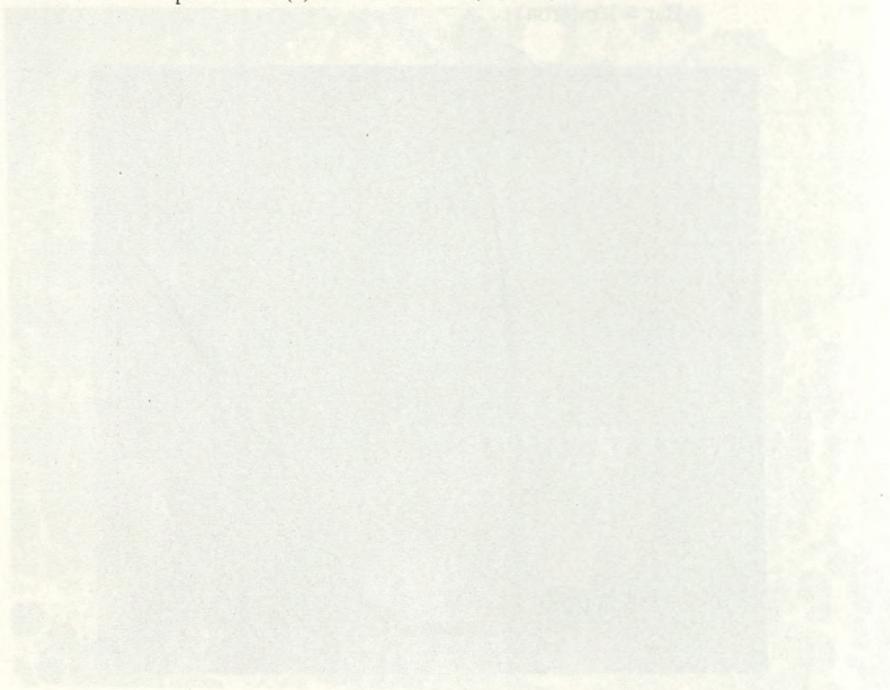


Figure 8: Ultrastructure of mucous acinar cell 72 hours after irradiation. Mitochondrial damage is not prominent (J). x 6700 Bar = 1(micron)

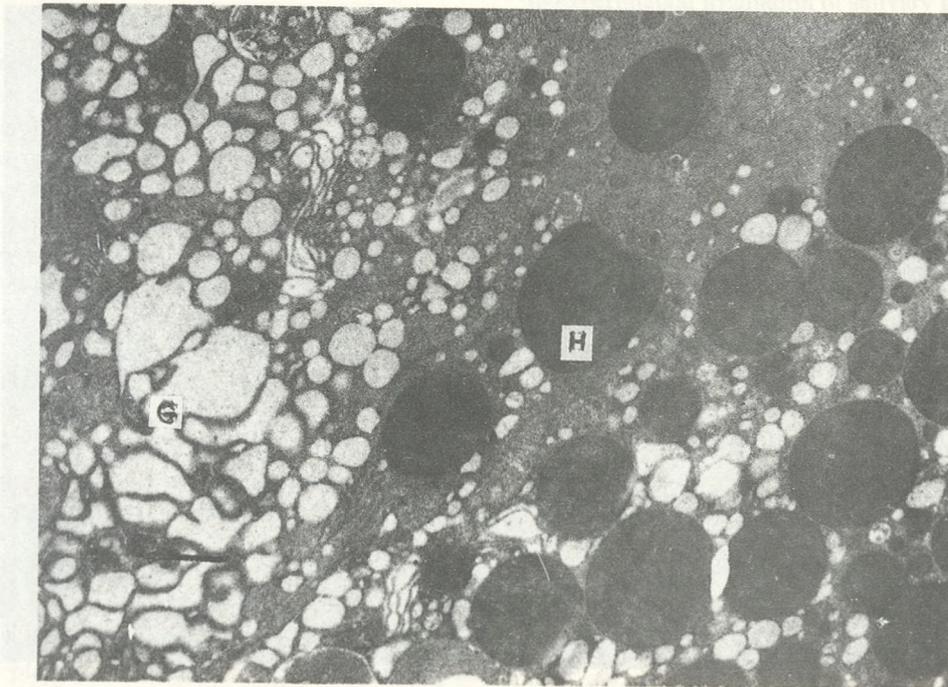


Figure 5: Ultrastructure of granular duct cells 72 hours after irradiation. Dilated endoplasmic reticula (G). The secretory granules are less electron dense (H). x 4000 Bar = 1(micron)

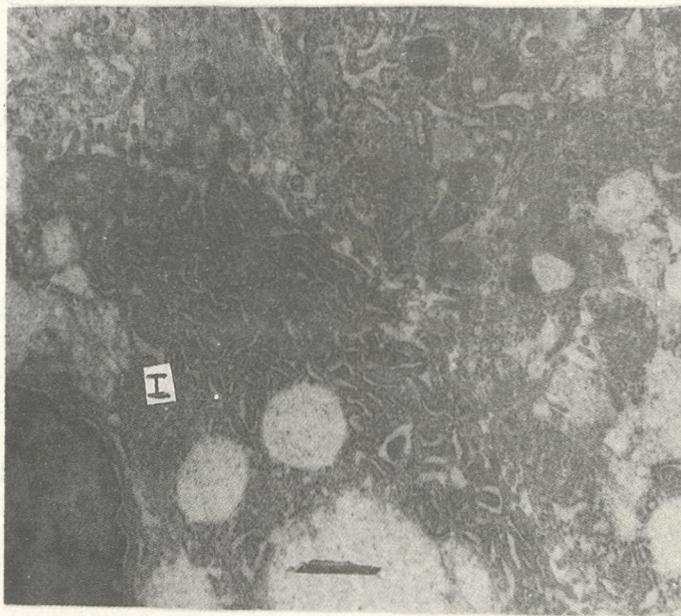


Figure 6: Ultrastructure of mucous acinar cells 72 hours after irradiation. Electron dense cell among mucous acinar cells (I). x 2700 Bar = 1(micron)

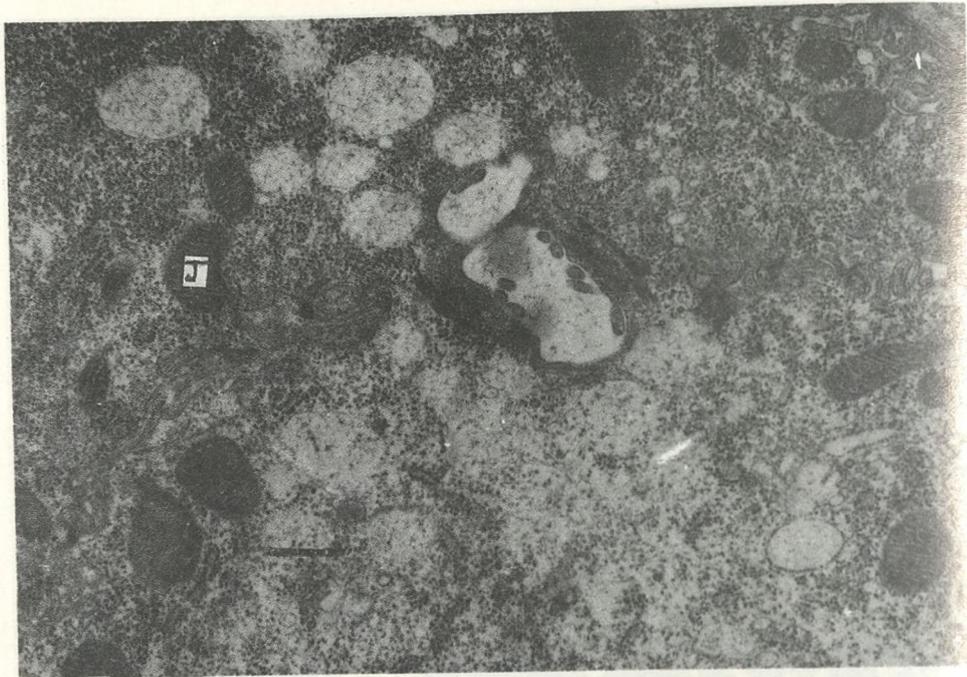


Figure 7: Ultrastructure of mucous acinar cell 72 hours after irradiation. Mitochondrial damage is not prominent (J). x 6700 Bar = 1(micron)

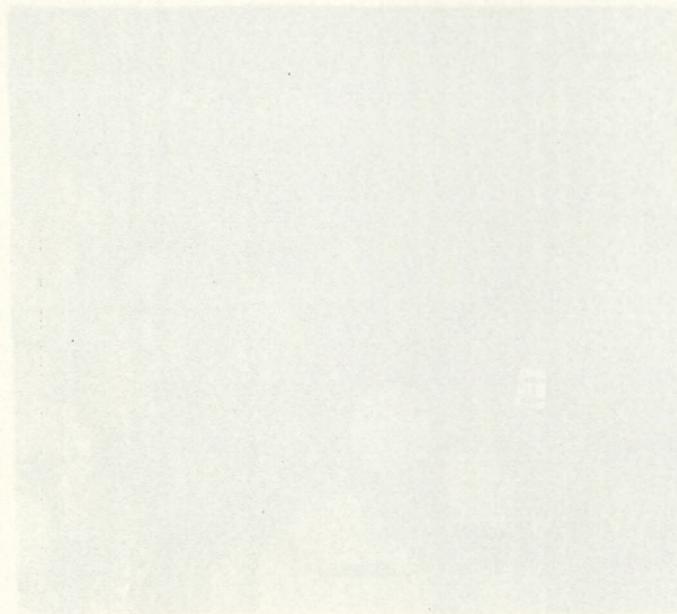


Figure 8: Ultrastructure of mucous acinar cells 72 hours after irradiation. Electron dense cell among mucous acinar cells (I). x 2700 Bar = 1(micron)

Results

The light microscopic observations of the control and irradiated salivary glands did not differ from previous descriptions. The control sections showed mucous acini, granular ducts and striated granular ducts containing serous granules and non granular intercalated ducts.

The irradiated tissues showed vacuolation in the acinar cells, discharged granules in the lumen of granular ducts and cloudy swelling of the cells (Indran et al 1989).

Transmission Electron Microscopy

In the control samples the granular duct cells showed a basally placed euchromatic nucleus and abundant membrane bound, electron dense secretory vesicles in the apical part towards the lumen. The granules were of different sizes and electron density (Fig.1). The mitochondria were scattered throughout the cytoplasm but were more concentrated along the basal and basolateral plasma membranes. Scattered among the granular duct cells were steroid secreting cells with extensive smooth endoplasmic reticula. Adjacent granular duct cells were attached by desmosomal attachments (Fig.2). The mucous acini showed electron lucent mucous globules, mitochondria and abundant rough endoplasmic reticula in the cytoplasm (Fig.3).

The granular duct cells of the irradiated salivary glands showed the following changes: pale electron lucent cells scattered among other ductal cells (Fig.4), enhanced number of serous granules of low electron density (Fig.5), membrane damage, mitochondrial membrane damage and extensively dilated endoplasmic reticula (Fig.6). The desmosomal attachments were not damaged.

The mucous acini showed electron dense cells, numerous ribosomes scattered in the cytoplasm and marginally dilated rough endoplasmic reticula. Mitochondrial membrane damage or cytoplasmic membrane damage was not prominent (Fig.7). A qualitative comparison of damage between serous granular duct cells and mucous acinar cells indicated that serous cells were more damaged than the mucous cells.

Discussion

Numerous studies have previously been carried out on early light and electron microscopic changes subsequent

to experimental irradiation of salivary glands (Sholley et al 1974, Stern et al 1976), particularly the submandibular glands in rodents. Early light microscopic changes are not very striking: small foci of necrosis and a mild intensified inflammatory reaction. Electron microscopic examination reveals that as early as 16 hours after exposure, highly osmiophilic and granular karyolytic bodies can be seen. Forty eight hours post irradiation, autophagosomes and ductal mitochondrial changes are visible together with disappearance of basement membrane infoldings. All of these alterations reflect the direct effect of radiation on cells.

The current findings are largely consistent with these observations. The differences in the degree of cell damage and the observations of electron lucent cells may be attributed to the type of irradiation used (Cobalt 60) and the species and strain of the animal model. A new observation in this study, the alteration of electron density of the serous granules is concurrent with previous reports of altered enzyme activity (Abok et al 1984).

However, most of these changes are not specific for radiation or even for radiomimetic agents (Shiba et al 1972, Adkins 1974, Harrison and Garrett 1976) and are identical to changes seen after experimental ductal ligation (Cutler et al 1979). These alterations must therefore be viewed as a common mode of reaction of the salivary gland parenchyma to a wide variety of types of injury.

In a comparable situation the small intestinal villi undergo changes in villous height resulting in the formation of small conical villi to a flat mucosa without any villi after whole body irradiation procedures (Becciolini et al 196, Indran et al 1985). Change in villous height was attributed to damage to cells (Watanabe 1978). Indran et al (1985) however postulated that villous damage may not be attributed to cell death alone but was also due to changes in the stroma. This theory was subsequently confirmed by mimicking radiation damage using drugs that act on smooth muscle of the stroma (Indran 1986, Indran et al 1991). It is therefore necessary to study in depth the type and mode of stromal damage to irradiated salivary glands.

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Development of a Low Cost Domestic Filter to Prevent Dental Fluorosis

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Summary

Dental fluorosis had been identified as an endemic problem in dry zone areas in Sri Lanka. The unsightly brown discolouration of the teeth had led this young children effected in the villages to a severe psychological impact. This problem is not confined only to Sri Lanka but also exist in other parts of the world such as India, South Africa, America etc. Presently several methods are available to defluoridate the water using filter media such as serpentine, activated alumina, charred bone meal etc. In all these methods the main disadvantage is that the filter media used is not readily available for the affected community. In contrast the filter medium used in the suggested filter is freshly burnt bricks which is freely available in affected locality. In addition this has also the advantage of having increase contact time due to the upward flow technique used, and above all the low cost incurred for the development of the said filter.

A reasonably good fluoride removal can be obtained after four hours of retention time in the filter which is suitable for house-hold application. The fluoride removal efficiency of this defluoridator is 80 percent to begin with and then drops to around 25 percent after 75-80 days in operation, for a well with a fluoride content of 3 mg/l.

Key words: Dental fluorosis, Low cost domestic filter, Defluoridaton, Dry zone.

Introduction

Dental fluorosis occurs when water containing excessive amounts of fluoride is consumed during the developmental stage of the permanent teeth. Although it is endorsed by

the WHO that the recommended amount of fluoride in the drinking water is 1.0 mg/l, it is suggested that for a tropical country like Sri Lanka, this figure should be 0.7 mg/l.

In Sri Lanka several endemic areas where dental fluorosis is seen had been identified (Seneviratne et al 1973 & 1974, Warnakulasuriya et al 1990, Abayaratne 1989/1990). It has been shown that the prevalence of this condition in the North Central Province to be quite high, and equally to contain high levels of fluorides in drinking water as well.(Dissanayake and Seneviratne 1982, Raghava Rao 1987).

Dental Fluorosis is seen as an unaesthetic stain which sometimes is intensified to brown or a blackish stain, specially relegated to the permanent anterior incisors. Keeping in line with the saying "prevention is better than cure" it is imperative that we concentrate on prevention once and for all, rather than treatment, when once it occurs. Therefore the best mode of preventing the occurrence of this unaesthetic condition is to prevent the ingestion of "high fluoride" water. Although defluoridation using complicated defluoridation plants, is one of the methods used in developing countries, it may not be practicable for Sri Lanka, because of the enormous cost involved. This probably may not be realised, at least for a long time to come. Therefore in this paper an effort taken to develop a low cost house hold fluoride filter to eliminate the ill effects of the malaise of unaesthetic dental fluorosis is described.

Materials and Methods

The newly designed household filter is 100 cm in height, 20 cm in diameter and is fabricated using PVC pipes (Figure 1). The inner pipe is 2 cm in diameter with a

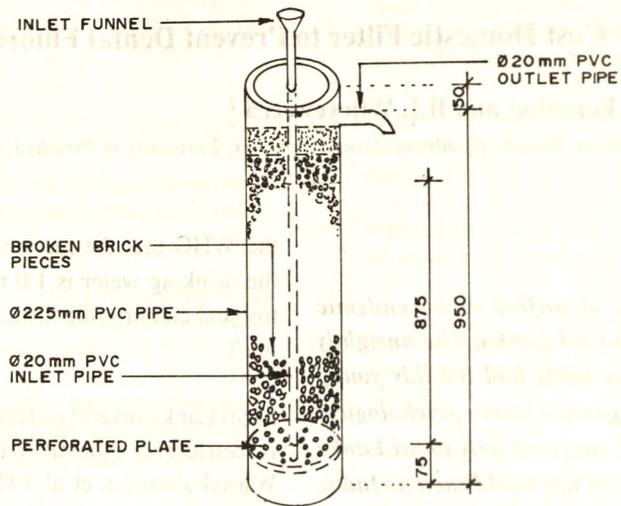


Figure 1 Shows the newly designed upward flow domestic defluoridator, after the completion of its fabrication.

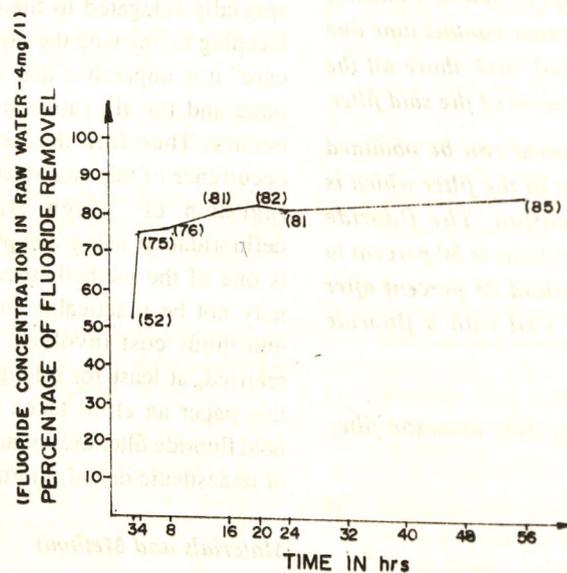
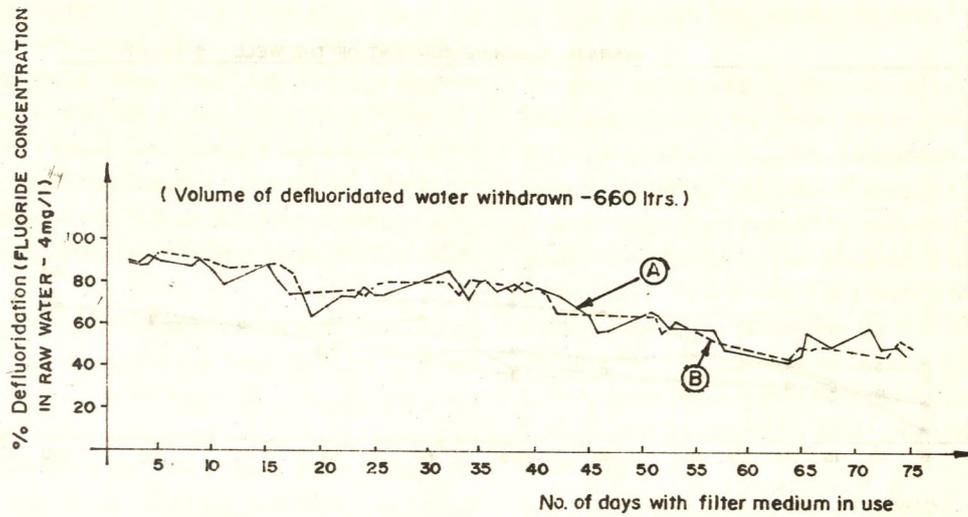


Figure 2 Indicates the percentage removal of fluoride at different time intervals.



Retention time 8 hrs and 16 hrs

Figure 3 Shows the fluoride removal efficiency of the defluoridator.

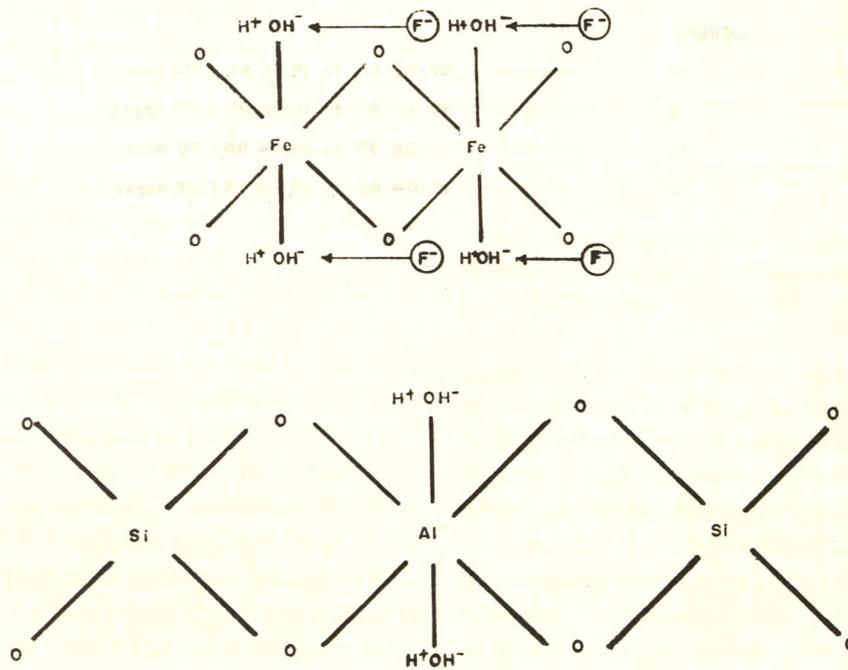


Figure 4 Shows the mechanisms of the fluoride removal.

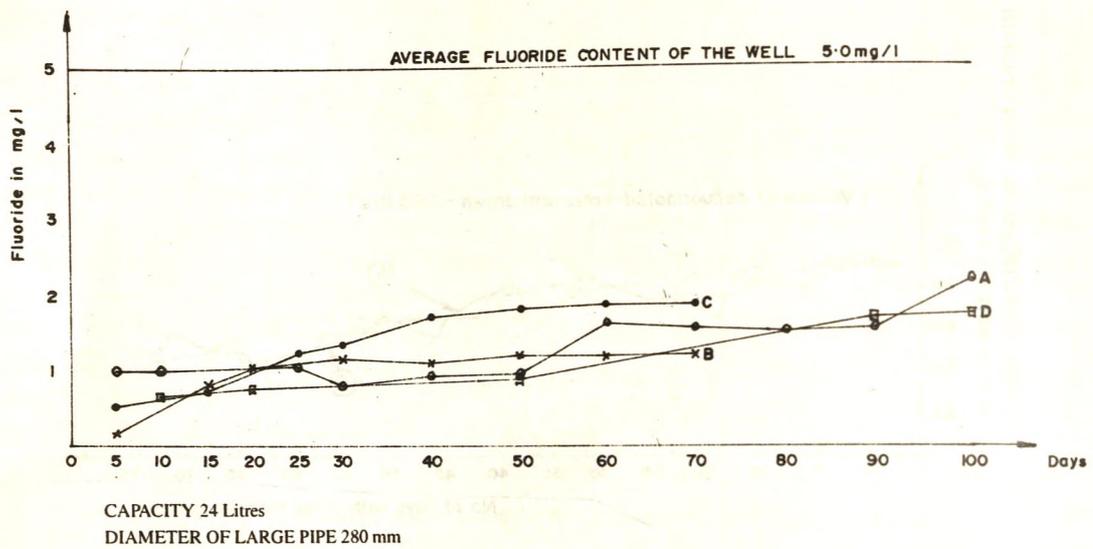


Figure 5 Shows the efficiency of one of the defluoridators no.42 operated in a house hold in Kekirawa.

LEGEND

- | | | |
|---|-----|---------------------------------|
| A | —○— | 07-08-94 to 15-11-94 (100 days) |
| B | —×— | 20-11-94 to 10-02-95 (70 days) |
| C | —●— | 11-02-95 to 20-04-95 (70 days) |
| D | —□— | 25-04-95 to 05-08-95 (100 days) |

circular perforated plate fixed at 5 cm from the base of the filter. The outlet is fixed 5 cm below the top of the filter. The filter unit is packed with broken pieces of bricks of sizes 8-16 mm to a height of 75 cm. The high fluoride water (4 mg/l) is fed through the inlet pipe. The fluoride analysis was carried out using a HACH DR 2000 programmable spectrophotometer using the 4-5 dihydroxy - 3 - (p-sulfophenylazo)-2-7-naphthalene disulfonic acid, tri sodium salt (SPANDS) reagent method (HACH 1980).

At the beginning the filter unit is filled with fluoride rich water and kept over night for at least 12 hours to obtain an equilibrium. Thereafter when the fluoride rich water is fed through the inlet pipe and equal volume of defluoridated water comes out automatically through the outlet pipe. The efficiency of the said filter was further tested by analyzing the fluoride rich water that was fed in and the defluoridated water that was collected from the outlet pipe at various time intervals (Padmasiri 1994).

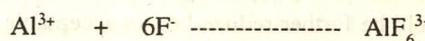
Discussion

The figure 2 indicates the percentage removal of fluoride at different time intervals. After keeping the high fluoride water in filter for 12 hours, the water was fed and withdrawn at these time intervals. 50 percent removal has been achieved after 03 hours. Furthermore 75 to 80 percent of fluoride removal could be obtained if the withdrawal of water is carried out after a longer time interval. Therefore it seems appropriate to infer that the minimum lag phase for usage of the filter to obtain subsequent batch of water should be four hours. The volume of water fed and withdrawn at a particular time was 8 liters. The four hour duration period is therefore more practical for a household application. This time interval suits well, the cultural patterns of our rural population where the usage of the water is mainly for cooking and drinking purposes. In another similar trial a 16 liter capacity filter unit was run for 60 days in the laboratory. The high fluoride water was fed and 10 liters was withdrawn in the morning giving a retention time of 16 hours (Figure 3A) while 5 liters was withdrawn in the afternoon giving a retention time of 08 hours(Figure 3B) in the filter unit. The fluoride removal efficiency of the filter could maintain at the same level provided small

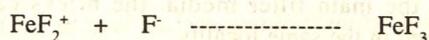
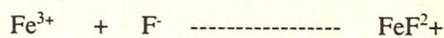
volumes of water are withdrawn with less retention time or large volumes withdrawn with longer retention time.

The filter media used for the removal of fluoride is low temperature burnt clay pieces (broken bricks). The burnt clay consists of silicate, aluminate and hematite complexes. When this is soaked in water for several hours these complexes get converted to oxyhydroxides of iron, aluminium and silica. The Si-O, Al-O bonds are much stronger than Fe-O bonds. The iron rich bricks which are reddish colour are preferable for fluoride removal. In addition the geochemistry of fluoride ion (ionic radius 1.36 A) is similar to that of the hydroxyl ion (ionic radius 1.40 A) and these can be easily interchanged between them (Figure 4).

The other possible formation is



The ferric ions form complexes with halides and its affinity for fluoride is high (Cotton F.A. and Willkington G 1988).



Perhaps this could be one of the contributory factors that is responsible for the unsightly brownish stain that occurs in dental fluorosis.

Figure 5 shows data of a filter with a larger diameter of 280 mm, thereby increasing the capacity of the filter. This facilitates the filtering of water containing higher content fluoride (5 mg/l). The beneficiary was provided with this filter and instructions were given on its usage. The consumption rate of this house hold was 8 liters of defluoridated water per day. Accordingly after every 70 to 100 days of operation the filter media had to be changed as shown in figure 5 graphs A,B,C and D to obtain the best efficiency. The table 1 gives the analysis of the fluoride levels of the defluoridated water at the beginning and at the end of the cycles.

Table 1 : Defluoridator No. 42

Inlet fluoride level	Period	Graphs	Defluoridated water outlet	
			mg/l	
mg/l			Start	End
5.0	07-08-94 to 15-11-94 (100 days)	A	1.00	2.00
5.0	20-11-94 to 10-02-94 (70 days)	B	0.24	1.40
5.0	11-02-95 to 20-04-95 (100 days)	C	0.50	1.85
5.0	25-04-95 to 05-08-95 (70 days)	C	0.66	1.75

The above data shows that even high fluoride level of 5 mg/l could be brought down to less than 2 mg/l by using this defluoridator. Perhaps by using a second defluoridator this also could be further reduced to an acceptable level where fluorosis can be completely avoided.

Conclusion

The suggested filter unit is simple in design.

This is a low cost filter.

The maintenance and operation cost is negligible since the main filter media, the bricks can be obtained in the same locality.

The rural community could change the filter media by themselves after a specified duration depending on the fluoride level in the well water.

This defluoridator has the capacity to reduce the fluoride content from 4 mg/l to less than 1.0 mg/l which conforms to WHO standards for fluoride in the drinking water.

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Enamel Surface Defects of Permanent Teeth. A Scanning Electron Microscopic Study.

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Summary

Tooth enamel surface was investigated using a scanning electron microscope. The normal surface exhibited regional variations of perikymata patterns and enamel rod end pits. Different types of developmental abnormalities in the form of pits and outcrops were encountered on the surface. Formation of calculus were more marked on such surfaces. Surface irregularities favors retention of plaque and makes restorative procedures difficult. Further, periodontal instruments by damaging the surface may make the tooth more vulnerable to caries and periodontal diseases.

Key words : Enamel Surface, Perikymata, Rod End Pits, Surface Overlapping Projections, Isolated Deep Pits, Brochs, Calculus, Caries.

Introduction

The structure of the tooth enamel surface has a direct relationship with the two major classes of dental diseases; the dental caries and periodontal disease. All forms of surface roughness predispose towards the retention of bacterial plaque on the tooth surface and may therefore have some effect on the occurrence of these conditions.

It was Pickerill (1923), who reported that the tooth surface was never smooth but rather consisted of horizontal ridges and furrows called "imbrication lines". The principal microscopic details which have been observed on tooth surface are perikymata, enamel rod end pits, cracks, scratches, micropits and different types of outcrops.

Earlier workers employed many different techniques to study the tooth enamel surface.

Wolf (1940) using a plastic film replica technique (zelluloid) reported that perikymata differ from one part

of the tooth surface to another. Perikymata are transverse, wave like grooves: they are the external manifestation of the growth lines (striae of Retzius). Scott and Wycoff (1946) using a different plastic film replica technique (collodion) found the perikymata to vary from 3 to 10 microns in depth and to be from 15 to 100 microns apart.

Different types of light microscopes and transmission electron microscopes have also been used in the past to study the enamel surface. However, the scanning electron microscope has been established as the best instrument to investigate the enamel surface due to its large depth of focus and wide range of magnifications (Boyde, 1971, 1975).

The perikymata are not found on the tips of cusps, since growth lines here are parallel with the cervix. They increase in frequency towards the cervix. In the midlateral region of the enamel surface, perikymata become more closely spaced and usually present as a regular wave like variation in the enamel thickness. The enamel rod end pits are found in the troughs of these waves. In these troughs there is usually a line at which there is an abrupt transition from region in which the pits are prominent, to a region in which they are shallow, and this line corresponds to the continuation of the formation of a new layer of enamel during formation (Boyde, 1971).

The enamel rod end pits are concave, about 6 microns in diameter, and vary in depth and shape. They are shallowest in the cervical region of surfaces, and deepest near the incisal or occlusal edges. The outline has been classified as round, scalelike or hexagonal. It has been reported that such variations in shape are most likely due to external influences, and are not typical of the prism ends as formed (Scott, 1951). These pits are synonymous

with the hexagonally packed pattern of 0.1 to 0.2 micron deep depressions, 6 to 7 micron in diameter, indicating the last location of individual ameloblasts which can usually be seen at the deciduous enamel surface (Boyde, 1975).

The above features can be considered as normal for the enamel surface. Many other features in the form of developmental abnormalities have been reported by past workers. These include various micropits and outcrops (Pedersen and Scott 1951, Newman and Poole 1974), surface overlapping projections (SOP) and isolated deep pits (IDP) (Boyde, 1975), Brochs in permanent (Boyde, 1971) and deciduous (Jayasinghe, 1987) teeth.

During this study the scanning electron microscope was used to examine enamel surface defects of extracted teeth and to observe their relationship to retention of bacterial plaque and formation of calculus.

Materials and methods

185 permanent teeth (105 male and 80 female) extracted at a private dental clinic for various reasons were used for the study. The teeth were stored in 70% Alcohol immediately after extraction. They were immersed in a solution of 2% Hydrogen Peroxide for 24 hours to remove organic matter and after washing with distilled water were kept at 37°C for 3 hours in a solution of 50:50 Chloroform and Methanol. The teeth were then air dried and examined using a Binocular Microscope. 20 teeth were selected for Electron Microscopy. They were given a conductive coating of Platinum using a sputter coater and examined in a Jeol Scanning Electron Microscope at 10kV.

Results

The teeth were extracted mainly due to caries and periodontal disease. Therefore, carious lesions and calculus were present in most of teeth. It was possible to identify gross abnormalities and developmental defects under the Binocular Microscope. Six samples showed discoloration due to Fluorosis and 5 samples showed mottling possibly due to Tetracycline.

The Electron Microscopy revealed detailed features of tooth surface. Fig. 1 is a low power micrograph of the cervical region of a premolar tooth extracted due to periodontal disease. Closely spaced perikymata present above the cervical margin shows the adherence of calculus. Towards the cervical margin the enamel has a smooth appearance and is thinner. In high power of the same specimen (Fig.2) more details of this surface is apparent. Enamel rod end pits can be seen in the trough of a perikymata and on the smooth cervical region brochs are evident. Some deep pits and outcrops can also be seen on the upper part of the specimen. The upper of the figure shows a part of enamel that has been chipped away during preparation.

Fig. 3 shows buccal surface of a lower left molar tooth. In addition to normal rod end pits this surface shows outcrops which appear to be over production of enamel by ameloblasts before they ceased to function. The sizes of these outcrops vary from production of one to several ameloblasts. Fig. 4 shows a surface similar to Fig. 3 from a lower right molar tooth. Similar outcrops to Fig. 3 and a deep pit show associated growing bacteria on the surface. Fig. 5 shows a deep pit in high power. Calculus is growing around the edge of this pit. Fig. 6 is a micrograph of palatal surface of a upper molar tooth showing two isolated outcrops. One of these projections has been broken away revealing a deep pit underneath. Fig. 7 is a high power micrograph of calculus from a premolar tooth. Calcified bacteria on this surface mainly exhibits morphology of rods and cocci. Fig. 8 shows a lingual surface of a lower premolar tooth. Outcrops in the form of surface overlapping projections can be seen on this surface. Several chains of calcified cocci shaped bacteria are also present on the surface.

Fig. 9 shows a lingual surface of a upper incisor tooth. A hypoplastic region can be seen on the centre of the micrograph. In high power (Fig. 10) this area shows deep pits indicating that they may have resulted from premature death of ameloblasts before completion of matrix secretion. Fig. 11 shows a typical broch found in a upper premolar tooth. Fig. 12 is the labial surface of a fluorosed canine tooth. The normal morphology of a tooth surface is not evident on this surface indicating interference of



Fig.1 Cervical region of a premolar tooth showing perikymata and calculus. Field width 2.3mm.



Fig.2 Same field as Fig. 1 showing enamel rod end pits in the trough of a perikymata. Brocks can be seen towards the cervical margin (left). Field width 0.58mm.

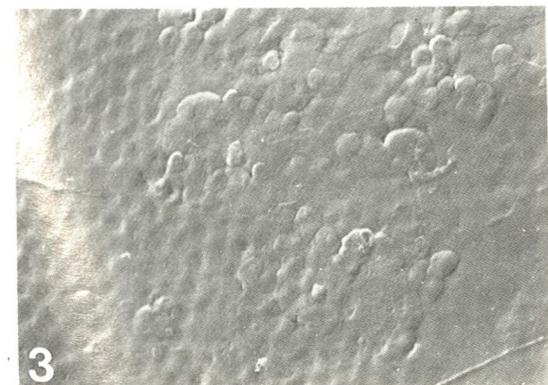


Fig.3 Buccal surface of a lower left molar showing outcrops of different sizes. Field width 156 microns.



Fig.4 Buccal surface of a lower right molar tooth. Outcrops and pits are associated with bacterial growth. Field width 156 microns.

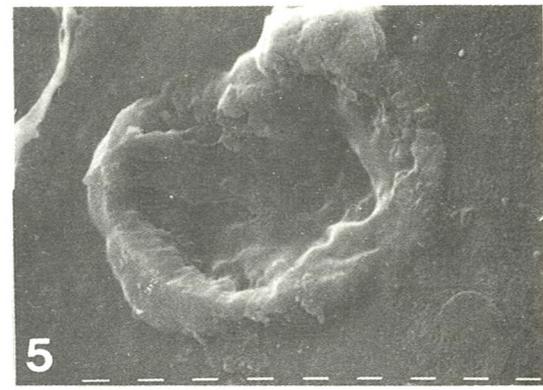


Fig.5 A deep pit from a upper right molar tooth. Calculus is attached to the margin of this pit. Field width 17.5 microns.

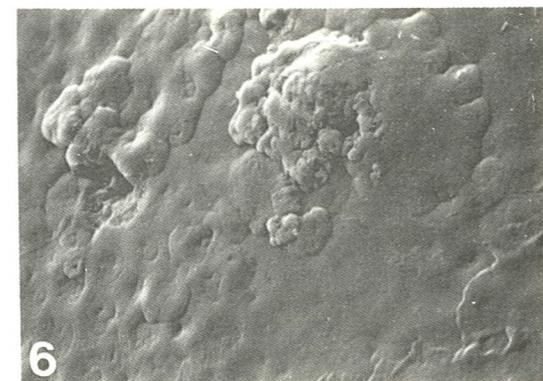


Fig.6 Palatal surface of a upper left molar showing two isolated outcrops, one broken away revealing a pit underneath. Field width 82 microns.

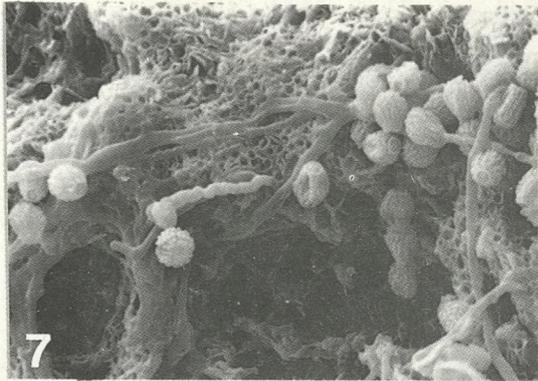


Fig.7 High power of calculus from a premolar tooth. Calcified bacteria show the morphology of rods and cocci. Field width 39 microns.

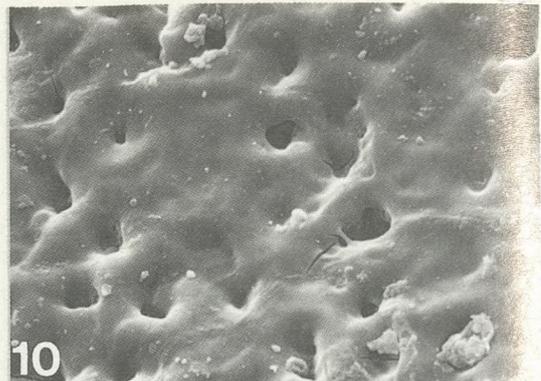


Fig.10 High power of Fig. 9 showing deep pits in the hypoplastic region. Field Width 41 microns.

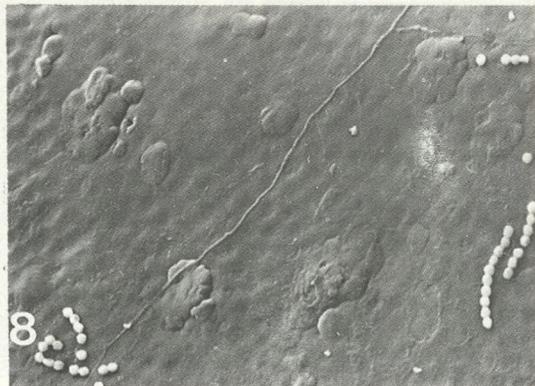


Fig.8 Lingual surface of a lower premolar tooth showing several SOPs and chains of cocci shaped bacteria. Field width 175 microns.



Fig.11 A typical brooch from buccal surface of a upper left premolar tooth. Surface of the brooch is smooth and no rod end pits can be seen. Field width 41 microns.



Fig.9 Lingual surface of a upper incisor tooth showing a hypoplastic region at the centre of the micrograph. Field width 140 microns.

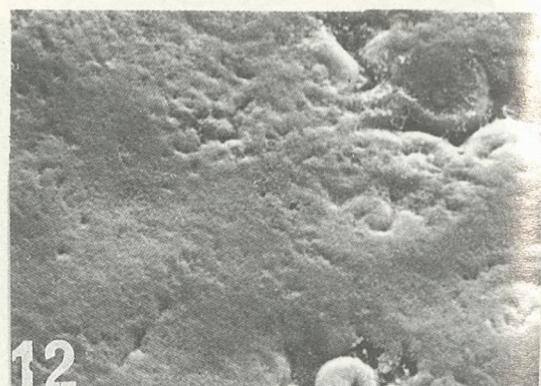


Fig.12 Labial surface of a fluorosed canine tooth. Normal enamel surface features cannot be seen on this surface. Field width 17.5 microns.

fluoride with formation and mineralisation of normal enamel.

Discussion

It has been shown that rough enamel surface not only aid in accumulation of plaque and formation of calculus, but interfere with various restorative procedures (Boyde, 1972). Further, use of different periodontal instruments may actually damage rough areas of enamel thereby exposing such areas to caries (Jones et al, 1972).

The normal enamel surface itself is not smooth but presents perikymata that are more prominent towards cervical margins (Fig.1). It has also been shown that 30% of all teeth (Boyde, 1971) and 50% of premolars (Jayasinghe, 1986) have surface projections called brochs around cervical margin. It is therefore apparent that the cervical margin may readily available for formation of calculus compared with other regions. Any cavity margin cut into these areas will produce an irregular surface that will lead to a defective surface finish. Further, scaling carried out to remove calculus can easily damage surface irregularities making the surface more vulnerable to caries and retention of plaque.

It was also shown by this study that bacterial growth occurs mainly around various defects found on the surface. Boyde et al (1968) found that in a group of unerupted teeth the greatest number of pits were between 1 and 3 microns in depth. The deepest pits, which would form the "best" micro-stagnation areas, were found in the mid lateral region, which corresponded approximately with the region of highest caries incidence on approximal surfaces just below the contact area. A tooth brush bristle is too big to remove plaque that accumulate in such pits.

Isolated deep pits (IDPs) (Fig.5) usually several times larger than the developmental type pits were commonly encountered on all surfaces. As Boyde (1975) reported, these isolated deep pits may result where surface overlapping projections (SOPs) (Fig.8) break away (Fig.6). SOPs may be found anywhere on the tooth surface. The smallest of these probably represents the last product of single ameloblast, and the largest, the last product of considerable group of cells.

Different types of hypoplasias (Fig.10) may also retain plaque and predispose caries. Fluorosed teeth (Fig.12) showed hypoplasia that interrupted the normal perikymata

pattern. Similarly other conditions such as amelogenesis imperfecta that produce defects on the enamel surface will aid occurrence of caries and periodontal disease.

The brochs (Figs.11) observed during this study were the first to report in Sri Lankan teeth.

It is clear that the accumulation of plaque, formation of calculus and occurrence of caries are related to the quality of enamel surface. It is possible for a clinician to identify gross surface defects during restorative procedures, so that adequate precautionary measures could be taken. However, most defects are microscopical and may lead to imperfect filling margins and resultant accumulation of inaccessible plaque may lead to recurrent caries or to gingivitis. Similarly damage caused by periodontal instruments to the enamel surface may be an important factor in future plaque and calculus retention and caries.

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CASE REPORT

Melanotic neuroectodermal tumor of infancy

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Introduction

Melanotic Neuroectodermal Tumour of Infancy is a rare neoplasm arising from cells derived from the neural crest cells, with approximately 164 cases been reported in the literature from 1983 to 1994 (Shah R.V.,etal, 1994). Krompecher (1918) is reported to have been the first to recognize this tumour which he termed as a 'congenital melanocarcinoma'. Since then several other authors have coined deferent terminology for this condition, being - 'Melanotic epithelial odontome' (Mummery and Pitts, 1925), 'Retinal analage tumour' (Halpert and patzer, 1947), 'Pigmented congenital epulis' (MacDonald and white, 1954), and 'Melanotic neuroectodermal progonoma' (Stowens, 1957).

This tumour of rather obscure nature occurs exclusively in infants and in most cases the lesion is situated in the jaws, although similar tumours have been reported in extra oral sites.

This lesion occurs in infants within 12 to 18 month of life with average age being between 1 and 3 months. The maxilla is affected very often than the mandible. This tumour forms a mass that expands the bone, starting in the alveolar ridge, apparently without pain or tenderness with a variable rate of growth.

In some cases the lesion grows to quite a large size fairly rapidly, while in others it has been described as of slow growth. Radiologically there may be a well defined area of translucency suggesting a cyst but often the margin of the area of bone destruction are irregular, giving the impression of an invasive tumour. The destruction area may be containing or displacing a developing tooth or teeth. Both sexes are equally affected.

Case Report

A four month old female infant was brought to the oral and Maxillo facial surgery clinic at Base Hospital, Nuwara

Eliya by her parents, with a gradually increasing lump in the anterior Maxilla. They gave a history of 10 days since noticing the lump. Apparently the lump had not bothered her and she had been breast fed with out any problem. She was the forth child of these parents with all other three children having died before reaching the age for 1 year., due to reasons other than similar lesion.

The past medical history was not relevant and generally appeared healthy.

Extra oral examination revealed a swelling over the upper jaw in the mid line. The lips couldn't be brought to gether with the lesion showing just under the upper lip. The both canine fossae and alae of the nose were raised. There were no cervico facial lymphadenopathy. She was 1.6Kg of weight.

Intra oral examination revealed a soft, with few areas being firm , non fluctuant, non tender Lump of 2.5cm in dimension over the anterior alveolar region, obliterating the labial sulcus and involving the premaxillary region palataly. The surface was smooth. It was dark bluish in colour (Fg1). Rest of the oral cavity appeared normal.

Taking into consideration of the age, site and clinical appearance of the lesion a differential diagnosis of melanotic neuroectodermal tumour of infancy and malignant melanoma were considered.

At this stage following investigations were carried out.

1. Radiography
 - Upper standard occlusal view
 - Occipito Mental view
 - True lateral view of the skull

The radiographs revealed a radiolucent region in the premaxilla with irregular margin. Crowns of the upper incisors were also seen on the radiographs.

2. Incisional biopsy.

Incisional biopsy was carried out under general anaesthesia. The histopathological features were reported as supportive of Melanotic neuroectodermal tumour of infancy.

3. The routine haematological, biochemical and urinalysis were carried out and were reported as within normal limits.

During the period of taking biopsy and receiving the biopsy report, left deciduous central incisor erupted through the mass.

With the histopathological diagnosis definitive surgery was planned.

Under oro tracheal intubation incision were made on the labial, alveolar and palatal mucosa at the demarcation between the mucosa covering the tumour and healthy mucosa. Mucoperiosteal flaps were raised and tumour enucleate. There were areas of erosions into the maxillary bone, with tumour tissue and these tumour tissue were also enucleated, with pathological bone conservatively. The enucleated mass was soft with firm areas in between and appeared bluish to black in colour (fig.2). Sharp bony edges were filed down on the surrounding bone and haemostasis achieved (fig.3). The mucoperiosteal flaps were mobilised and the surgical defect was closed down primarily with 4/0 braided black silk (fig.4).

Post operatively she was on intra venous antibiotics for five days and analgesics. She made an uneventful post operative recovery and was discharged after suture removal in ten days time. After one month post operatively the wound was healed and the raised canine fossae were getting reduced gradually. The infant was very active and back to normal. At three months post operative the face appeared normal with slight deficiency in the premaxilla. She is being kept on long term six monthly review to check on possible recurrence and subsequently for restoration of the dentition and the appearance.

The excision biopsy specimen macroscopically appeared 3.5x3.0x3.0cm of size including brownish black soft tissue and hard tissue.

Microscopic findings were that the section was showing an alveolar pattern of tumour cell nest with a small amount

of interning connective tissue. The cells located centrally in the nests are dense. The peripheral cells are larger and clear cell type in nature. The deeper areas showing darkly staining neuroblast like cells. Most of the area showing melanin pigmentation. The histopathological features are consistent with that of a Melanotic neuroectodermal tumour of infancy.

Discussion

Melanotic neuroectodermal tumour of infancy is a rare tumour, arising from neural crest cells, though earlier it was thought of arising from odontogenic epithelium (Kerr and Pullon, 1964). Borello and Gorlin (1966) presented a case in which some 6-8 times the normal amount of Vanilmandelic acid was being excreted in the urine. When the tumour was removed the urinary level of this substance fell to normal. Since other tumours with which Vanilmandelic acid in excessive amounts is excreted in the urine include neuroblastomas, ganglioneuroblastoma and phaeochromocytoma, these authors suggested an origin from neural crest cells. They also accepted that certain tumours found in sites outside the jaws were histologically the same. Such tumours have been found in the anterior fontanelle, long bones, the shoulder, the epididymis and soft tissue of the limbs (Pettinato, G,etal,1991, Kapadia,S.B.,etal,1993). Koudstaal, etal (1968) compare the enzyme pattern of the cells with that of malignant melanoblastoma, paraganglioma and phaeochromocytomas and that of the small tumour cells with neuroblastoma cells. They concluded that both types of tumour cells in the melanotic neuroectodermal tumour of infancy were of neural crest origin.

The current view is that these tumours are of neural crest origin and the preferred name is Melanotic neuroectodermal tumour of infancy. The odontogenic epithelium found in the jaw tumours, but not in those from other sites, is considered to be from the normal dental lamina and included secondarily in the tumour mass as it grows.

Conservative enucleation of the tumour is the accepted form of treatment. Any marked damage to the jaw at this stage will produce a disfiguring deformity as the child grows. Any how, care should be taken to remove heavily involved bone and any pockets of tumour. Any tooth germ present within the lesion will be included in the specimen.



Fig. 1 Pre operative view of the tumour

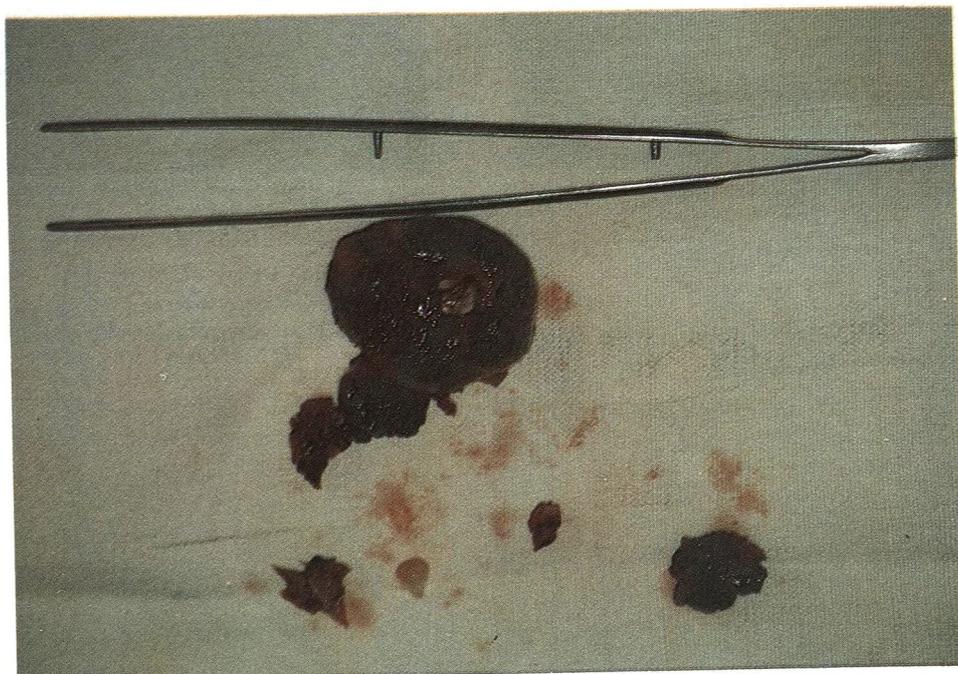


Fig. 2 View of excised specimen

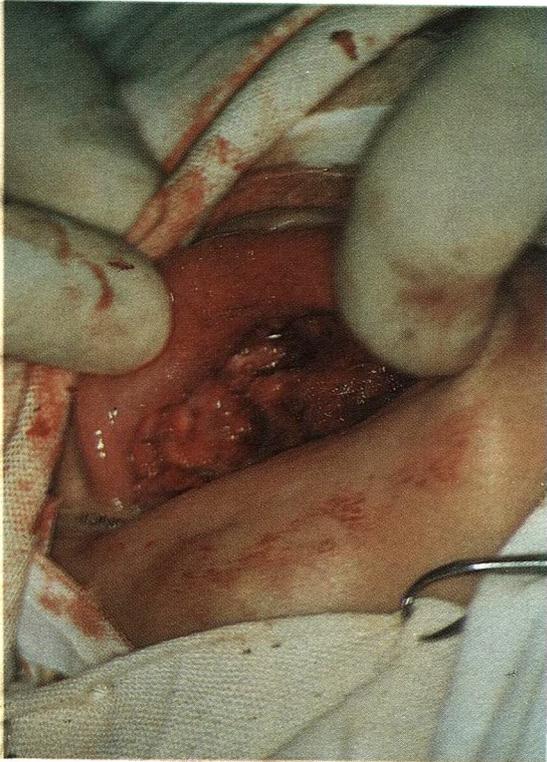


Fig. 3 View of the residual surgical site after enucleation of the tumour



Fig. 4 View after closing the operative site.

In spite of the poor prognosis normally associated with invasive and pigmented neoplasms, with this particular variety the prognosis is good. In some instances there may be a single local recurrence, which results from a failure to remove a pocket tumour, but further enucleation and curettage is all that is required to effect control. In a case described by Hovell and Spencer(1952) a recurrence of melanotic neuroectodermal tumour of infancy in the mandible after six months of initial enucleation had grown into massive size, but after six years it had spontaneously regressed into a localized mass. It seems, therefore, that this condition may regress spontaneously, but it would be taking unnecessary risk to treat all cases expectantly as by it's enlargement the tumour damages the adjacent structures(Killey and Kay,1975). The tumour is usually benign, but some cases have behaved in a locally aggressive fashion, while few others have resulted in distant metastasis (NavasPalacios, J.J.,1980, Block, J.C., etal, 1980).

In the case reported a conservative surgical approach was taken by removal of all visible tumour tissue. The urinary vanilmandelic acid levels could not be ascertained due to inadequate laboratory facilities.

Acknowledgments

The author is grateful to Prof. B.R.R.N Mendis and Dr. Prasad Amaratunge for providing the histopathological reports and to Drs. N.S. Manoratne and S. Paraneetharan for their assistance in managing the patient.

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British Dentists in Sri Lanka on a Study Tour - A report.

Dr. Hilary W.M. Cooray

President - General Dental Practitioners ' Association

A delegation of 82 British Dentists and their spouses arrived in Sri Lanka on Sunday 11th February 1996. The Dentists who are Members of the British Society for General Dental Surgery were here at the Invitation of their counterpart organization in Sri Lanka namely the General Dental Practitioners Association. A Scientific Programme was organized in Collaboration with the Sri Lanka Dental Association.

On the 14th of February they visited the Faculty of Dental Sciences at Peradeniya had dialogue, discussions and seminars with the staff and students of the Faculty. The program at the Faculty was as follows.
Wednesday 14 February.

Wednesday 14 February.

Tour round Dental School at Peradeniya.
Dean's Address - Prof. A.N.I. Ekanayake
Welcome address by Chairman Organising Committee - Prof. B.R.R.N. Mendis.
Forensic Odontology - Dr. Ian McIntyre
Address by President Sri Lanka Dental Association Kandy Branch - Dr. J.U. Weerasinghe
Presentation by Scientific Programme Organiser - Dr. Mike Mulcahy.

On *Sunday the 18th of February* a Joint Scientific Sessions between the British Society for General Dental Surgery, Sri Lanka Dental Association and the General Dental Practitioners Association was inaugurated by the Minister of Health Social Services and Highways, Hon. A.H.M. Fowzie. This was held at Triton Hotel Ahungalle.

The Programme details were as follows :-

Sunday 18th February

Addresses -

- President Sri Lanka Dental Association - Dr. R. Weerasinghe

- President Elect British Society for General Dental Surgery - Dr. Ian McIntyre.
- Minister of Health, Highways and Social Services - Hon. A.H.M. Fowzie.
- President General Dental Practitioners' Association - Dr. Hilary Cooray

The development of General Dental Practice in the United Kingdom - Dr. Mike Mulcahy.

Community Trial to remove traces of Fluoride from drinking water - Dr. K.D.G. Saparamadu.

The formation of a Professional Group dealing with Aesthetic Dentistry - Dr. Howard Stean of ADAPT.

Implementation of an outreach programme using Dental Auxiliaries - Dr. Adly Mohamed.

Restorative Dentistry and pulpal pathosis - Dr. Gary Unterbrink

Dentistry in the Ancient Ayurveda System - Dr. Hilary Cooray.

Bolts in faces - Dr. David Madan.

Table Clinics

- Full and Partial Denture problems and Solutions - Dr. Richard Bulter.
- Tooth surface loss - Dr. Dan Earp.
- Motivating staff and team leadership skills in the Dental Team - Dr. Charles Scol
- Crown preparation - Dr. Rasesh Patel.
- Orthodontics : current concepts of removable appliance therapy - Drs. Helen Jones and Mike Coleman.
- The Hanson Speed Bracket : the ligature free Orthodontic bracket - Dr. Ken Alexander.
- TMJ problems and treatment using appliance therapy - Dr. Philip Lang.
- Technique tricks - Dr. Mike Jones and Mike Hodgson.
- The DGDP (UK) Examination - Dr. Paul Howard.

The Minister in his address stated that the "Dental Team" is an important modality for rendering effective relevant Oral Health Services as we enter the 21st century and that Sri Lanka will be introducing the "team concept" in order to provide effective Oral Health Care specially to the rural population. He wished the sessions success and invited the delegation to come again to Sri Lanka for the Asian Pacific Dental Congress in April 1997.

Dr. Hilary Cooray President of the General Dental Practitioners Association stated that the concept of this trip was planned during his meeting with Dr. Raj Rayan the President of B.S.G.D.S during his visit to London in October 1994. He traced the Historical links of British Dentistry from its inception in Ceylon in 1912 to present time and stated that further cooperation was possible in many aspects of Dentistry and particularly the development of General Dental Practice in Sri Lanka.

The President Sri Lanka Dental Association Dr. Ranjith Weerasinghe in his address thanked the B.S.G.D.S. for this gift of dental materials equipments and books and

also said that this trip has given us a great opening to step up our promotion for the 19th Asian Pacific Dental Conference to be held in April 1997.

The British Society of Dentistry presented books, manuals and guides on behalf of itself and the Faculty of General Dental Practitioners for the Library of the Dental Faculty at Peradeniya and the Library of S.L.D.A. and the G.D.P.A. . The society members also made a generous donation of dental materials and equipment. The B.S.G.D.S. President Dr. Raj Rayan expressed their and on behalf of the Faculty of General Dental Practitioners the wish to further collaborate with the Sri Lankan counterparts to develop programmes in Sri Lanka. A Television Interview with Vice Dean of the Faculty of General Dental Practitioners of the Royal College of Surgeons, Dr. Mike Mulcahy and President Elect B.S.G.D.S. Dr. Ian McIntyre was also recorded and telecast during this time.

This visitation has indeed provided us with an opportunity of renewing old ties and making new friendships with the members of the Dental Profession in the United Kingdom.

New Dental Faculty / Hospital Complex funded by Japanese Grant Aid Project

The 18th of October, 1996 was a memorable day for the Dental Profession, Dental students and the entire staff of the Faculty of Dental Sciences and for the University of Peradeniya as a whole. The foundation stone for the proposed building complex for the new Dental Faculty/ Teaching Hospital complex was laid on this day by the Hon. Minister of Education and Higher Education, Mr. Richard Pathirana, with the participation of the Hon. Acting Minister of Health, Mrs. Pavithra Wanniarachchi, the Hon. Deputy Minister of Higher Education, Prof. Vishwa Waranapala and His Excellency the Ambassador of Japan in Sri Lanka, Mr. Yasuo Noguchi.

This new Dental Faculty and Hospital Complex is an outright grant given by the Government and people of Japan to the Government and people of Sri Lanka under Japanese Grant Aid after recognising the absolute need to improve the facilities at the faculty of Dental Sciences, University of Peradeniya. This will include one 4-storeyed building complex for clinical studies, one 4-storeyed complex for pre-clinical studies and administration and one 2-storeyed building complex for student activities. All these physical facilities will be provided with appropriated and modern equipment. This will improve the quality of service given to patients, quality of teaching and research, and finally the quality of students produced from this Institution.

This Grant is the highest that the University of Peradeniya has ever received, amounting to 22 Million American dollars, being the equivalent of One thousand two hundred million rupees. This is also the first time that the Government of Japan has gifted a fully fledged Dental Faculty to a country anywhere in the world. We therefore become the first country to receive a Dental Faculty and a Hospital Complex from Japanese Grant Aid. Therefore, it is envisaged that this Dental Faculty/ Hospital Complex will be the future Model for Japanese Grant Aid projects, while it is expected to be a centre of excellence in dental man power training in the South Asian region.

By: Dr. R.L. Wijeyeweera, Chairman, The Japanese Aid Project, of the Dental Faculty University of Peradeniya.



The Foundation Stone being Layed by the Hon.Minister of Education and Higher Education Mr.Richerd Pathirana. The Hon. Deputy Minister of Health Mrs. Pavithra Wanniarachchi, His excellency the ambassador of Japan in Sri Lanka Mr. Yasuo Noguchi. The Dean, faculty of Dental Sciences Dr. Ajith.W. Ranasinghe and Chairman, Japanese aid project, of the Dental faculty Dr. Wijeyeweera are also seen in the picture



Unveiling of the plaque by the Hon.Minister of Education and Higher Education after the laying of the Foundation stone of the New Dental Faculty Hospital complex with Japanese Government Grant Aid. The others in the picture are Dr.R.L. Wijeyeweera, Chairman, Japanese Aid Project of the Dental Faculty, Dr. Ajith W.Ranasinghe, Dean, Faculty of Dental Sciences, Prof. C.M. Madduma Bandara, Vice Chancellor University of Peradeniya and His Excellency Yasuo Noguchi, Ambassador of Japan in Sri Lanka.

Examination Results

Following candidates have been successful at examinations conducted by the PGIM

M.S. Part I

Dr. Thushari Nishanthi Hewapathirana
Dr. Jayasinghe Arachchige Vijira Pushpa Jayasinghe
Dr. Sampath Kumara Jayasinghe
Dr. Kalahe Acharige Kalyanaratne
Dr. Perippanayagam Kirupakaran
Dr. Thiraviam Sabesan
Dr. Poojitha Wajirapani Nikapitiya Wickramaratne
Dr. W.M. Parakrama Sarath Kumara Wijekoon

M.S. Part II

Dr. Sudath Wickramarachchi

DGDP

Dr. M. Sriyani Olivia Dias
Dr. K.H. Marie Pushpa Fernando
Dr. W.P. Ramani Gunasekara
Dr. Mohomed Farhim Jameel
Dr. Thusitha Janaka Liyanaarachchi
Dr. Shanthinie Logeswaran
Dr. Uma Iswarie Narenthiran
Dr. Adonchiyalage Ranjani premalatha
Dr. R.A. Sisira Kumara Ranasinghe
Dr. Kamalavathy Senthivasan
Dr. Janagee Sivaanantarasa
Dr. Suntherekantha Sivapathasundaram
Dr. A.L. Delicia Wickramasekere.

APPRECIATION

Dr. Neil Gunawardene -An Appreciation

The sudden demise of Neil after a very short illness had removed from our midst a loving and caring friend and a true colleague. The void created is well nigh impossible to bridge.

My association with Neil dates back to about forty years, first at Ananda College Colombo then at the Dental School Peradeniya. He was my batch-mate and also my room mate at Jayatileke Hall during our student days. Having qualified in 1960 he joined government service and was a lecturer at the Dental Nurses Training School at Maharagama.

Subsequently he was invited to take up the Principalship at the School for Dental Auxiliaries in Jamaica and re-organize the teaching programme there. He always loved teaching and was a very popular teacher here and abroad.

Having returned to Sri Lanka he set up in Private Practice and enjoyed a very lucrative practice in Colombo. His kind and gentle manner won the hearts of many patients, and they came to him from all parts of the island, to seek his healing touch. The organizing capabilities he had was evident even in his student days and he was chiefly instrumental in organising many social functions as Secretary of the Dental Students Union.

Integrity and devotion to duty characterized his professional life. He was actively involved in professional work. He served the Sri Lanka Dental Association in Various capacities for many years and as its President in the year 89 - 90. As President of the College of General Dental Practitioners he contributed substantially for the steady growth of the discipline of General Dental Practice in Sri Lanka.

He loved community work and gave leadership to the Lions movement at Nugegoda. He never hesitated to give his professional expertise to these Organization. His professional Colleagues always sought his assistance and advice, which he readily gave.

Till very recently he was the Secretary to the Organizing Committee of the 19th Asia Pacific Dental Congress to be hosted in Sri Lanka in April 1997. He made critical comment and valuable contributions at the Committee Meetings and his love and desire to see the hosting of a successful Congress was such that when I visited him at the Intensive Care Unit of the Hospital he made inquiries about the progress of the APDC arrangements. He was a devoted and loyal husband to Anula and a father who was always a councilor and a path finder to his two sons Ravi and Channa.

He had a wide circle of friends from all social strata who were deeply touched by his gentle personality.

He always had a friendly and eternal smile for every one, and his unique sense of humor and his ready wit won the hearts of many.

Till very recently he was in good physical health and always enjoyed the camaraderie that followed after every APDC Organizing Committee Meeting at the Organization of Professional Associations. His sudden death seems that an unseen had had snatched him from our midst for greater services.

For me personally it was the emotional end of a long and very close friendship filled with many happy moments of joy fun and laughter. Going back the memory lane it brings back fond memories of the fun filled care free undergraduate days at the Dental School Peradeniya.

The large and distinguished crowd that gathered at his cremation to pay their last respects bear ample testimony to his simplicity and to the noble qualities he possessed.

May he attain the eternal bliss of Nirvana.

Dr. K.D.G. Saparamadu.

INSTRUCTIONS TO AUTHORS

The SLDJ publishes following categories of articles which have relevance to Dentistry and allied sciences.

1. Leading Article- One article per issue. It may be solicited by the Editor. Authors are welcome to submit leading articles on current topics of interest, one's expertise or commentaries on general practice etc. They should be approximately 1500 words in length. References should be 20 or be less.

2. Reviews - Reviews are detailed surveys of published research pertinent to dentistry and associated sciences. They should be critical in nature and should not normally exceed 3000 words or 30 references.

3. Papers - Articles resulted from research work belong to this group. Results from routine clinical examinations or laboratory investigations will not be considered under this category. Subjects may vary from clinical trials to basic science research, historical analysis to dental economics. They should not exceed 3000 words and 25 references. A reasonable number of tables and illustrations will be accepted.

4. Short-Reports - These include reports on current topics, modified techniques new materials, practice management etc. Interesting results from routine clinical work or laboratory investigations may also be accepted.

5. Case Reports- Reports such as of rare diseases or conditions, modifications to accepted treatment procedures, new management methods etc. may be included in this category.

6. Letters to Editors - Subjects unlimited, but may include short critique of published papers in SLDJ.

7. Miscellaneous Topics- Subjects unlimited and the format is free. These may also include details of scientific meetings, conferences, annual sessions, examinations, news and views, visits, and obituaries.

8. Proceedings of Annual Sessions- Abstracts from annual sessions of SLDA and other colleges will be published under this category.

The following instructions are mainly applicable to research papers. However, other articles should also conform as far as possible to these instructions.

Submission of Manuscripts

1. General- Manuscripts must be submitted in triplicates. Text must be typed double - spaced with wide margins throughout in A14 (212 X 297 mm) size papers. They should be carefully scrutinized for errors before they are submitted. Correctness of spelling, grammar, and typing is the responsibility of the author. Three sets of figures and tables must be submitted. The number and the size of the illustrations must be consistent with the minimum requirement for clarification of the text. Previously published figures cannot be accepted. Manuscripts should be accompanied by a letter stating that the contents have not been published or submitted elsewhere for publication. Where applicable a copy of the ethical clearance certificate should be attached.

2. Title page- Following information should be furnished in the title page.

Title of paper, names of authors in the order in which they are to appear in the published article, departmental and institutional affiliation and an address for correspondence. Five English key words must be supplied for subject indexing. These key words should be taken from index Medicus or composed on the same principle

3. Summary - The brief summary is limited to 250 words. It should convey the main points of the paper and outline the results and conclusions.

4. Introduction - The introduction should carry sufficient background information on the subjects of study.

5. Materials and Methods - These should be described and referenced in sufficient detail.

6. Results- This section should present the findings of the research supported by statistical or illustrative validation of assertions. It should be free from discussion.

7. Discussion- The discussion should be focussed on experimental findings and their interpretation. Unsubstantiated speculations and plans for future study are unacceptable.

8. Legends for figures- Must be concise and should provide a brief, self sufficient explanation of the illustration. Sentences such as "see text for details" are unacceptable. Magnification should be indicated at the end of the legend if a calibration bar is not included in the figure.

9. References- References should be cited in the text as follows:

One author- (Jones 1992)

Two authors - (Jones and Amett 1986)

Three or more - (Jones et al, 1972)

Number references in the order in which they are first cited in the text.

Some common examples for the style of references are given below.

Bartlett J.G. O'Keefe P (1979) The bacteriology of perimandibular space infections. *J.Oral Surg* 37: 407-409.

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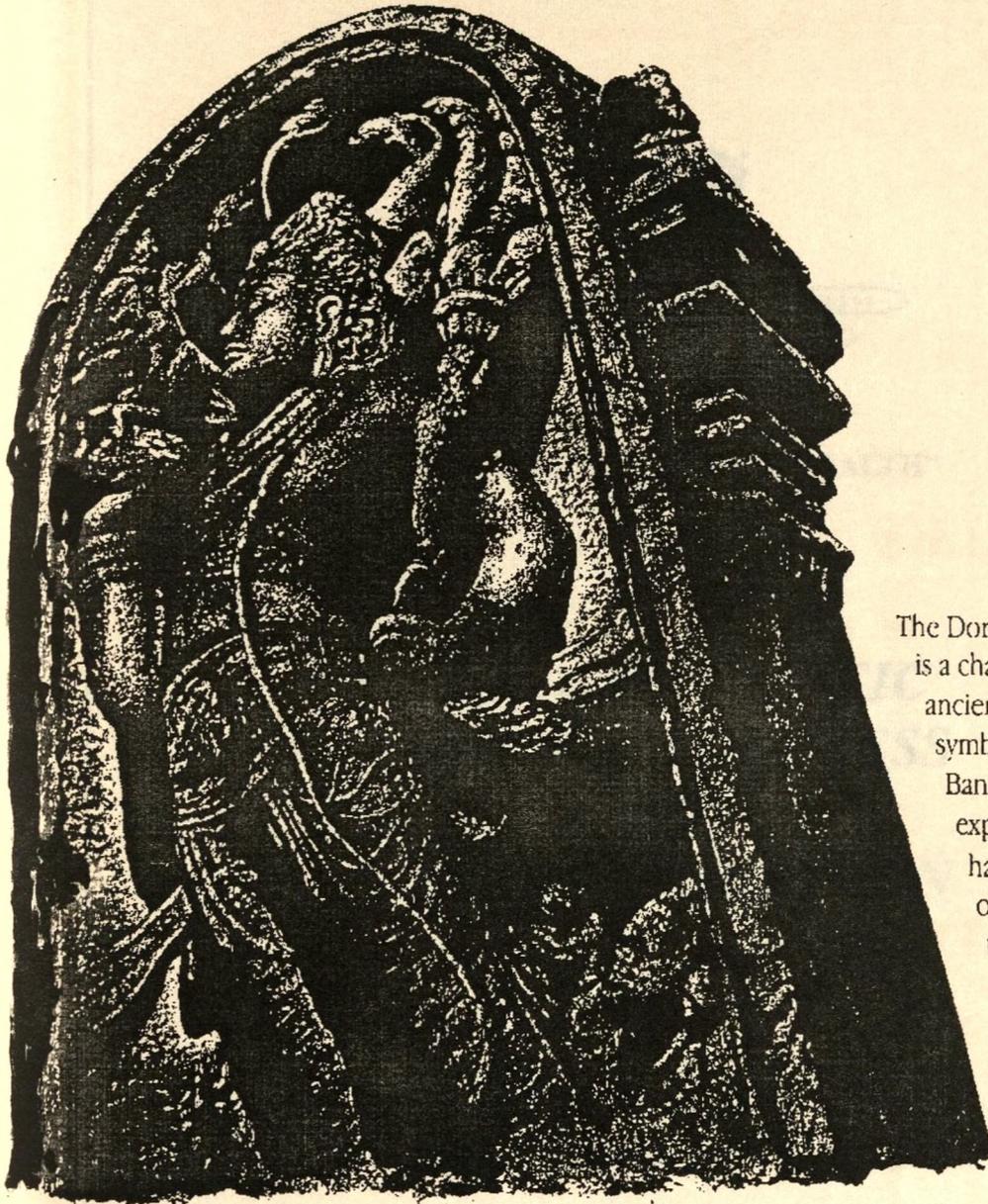
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