

# Injury to the permanent tooth germ following trauma to the deciduous predecessor

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## Report of a Case

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*Key words: Injury, tooth germ, trauma*

## SUMMARY

*Trauma to the underlying permanent tooth germ following mechanical trauma to the deciduous predecessor may cause several enamel and dentine abnormalities which may present as local or diffuse defects on the tooth-crown after eruption of the tooth. A case of localized enamel and dentine hypoplasia is presented together with a very unusual pulpo-gingival soft tissue tag on the labial surface of the permanent tooth.*

## OPSOMMING

*Die tandkiem van 'n sekondêre tand mag sodanig deur meganiese trauma aan die oorliggende primêre voorganger beskadig word dat verskeie afwykings van glasuur en dentien later in die sekondêre tand voorkom. Hierdie afwykings mag gelokaliseerd of verspreid op die tandkroon sigbaar wees na erupsie. 'n Geval van gelokaliseerde glasuur- en dentien-hipoplasie word beskryf, tesame met 'n baie ongewone pulpo-gingivale sagte weefsel aanhangsel wat op die labiale oppervlakte van die kroon van die sekondêre tand voorgekom het.*

## INTRODUCTION

Trauma to the deciduous dentition occurs predominantly in children from 1½ to 2½ years of age. Without differentiating between upper and lower anterior teeth, van Gool (1973) reported that the deciduous central incisors were involved four times more frequently than the lateral incisors.

The germs of the permanent incisors are initially situated lingual to the apices of the deciduous teeth. During their further development the tooth germs are gradually positioned more closely to the resorbing roots of the deciduous teeth. A predominantly axially directed force may be transmitted via the apex of the displaced deciduous tooth to the uncalcified permanent tooth germ. If the ameloblasts are injured, enamel hypoplasia may arise and in the case of concurrent injury to the odontoblasts, would extend into the dentine. The developmental defects in the crown will be dependant on the severity and direction of the traumatic insult and the stage to which the process of amelogenesis has progressed. These developmental injuries may be simple or complex, and extensive or local (Torneck, 1982).

The most common clinical manifestation of injury to the crown of a developing permanent tooth is an area of whitish discoloration caused by an insufficient degree of calcification (Andreasen & Ravn, 1973). It may appear as a small dot or a large area in the enamel that does not change after scaling or prophylaxis. Other possible defects included partial crown or root duplication (Williamson, 1961), interruption or cessation of root completion (Pindborg, 1970), and occurrence of an odontoma-like structure (Rodda, 1960). All the above authors mentioned dilaceration and disturbance of enamel for-

mation as possible defects. No reference was made to the disturbance of dentine formation or soft tissue reaction.

The present report is concerned with mechanical trauma as the cause of the observed enamel and dentine defects.

## CASE REPORT

A white boy, aged 9, presented with an unusual pulpo-gingival soft tissue tag on the labial aspect of the left maxillary central incisor. A history of trauma to the primary maxillary incisor, occurring at 2 years of age, was given by the mother. The patient's main complaint was sensitivity and discomfort on brushing the tooth.

Clinically, a tag of soft tissue, arising from the labial gingiva extended as far as the middle third of the labial aspect of the tooth where it entered the tooth substance (Fig. 1). The colour of the soft tissue tag was slightly



*Fig. 1: Soft tissue tag extending from the gingiva to the middle third of the crown of the left maxillary incisor, entering the pulp chamber in that area.*



*Fig. 2: Radiolucency, representing the area of penetration of the soft tissue tag (arrow). An accessory canal is also visible in the apical third of the root (broad arrow).*

more red than the gingival soft tissue. Hypoplasia was evident in the enamel surrounding the entrance of the tag into the tooth substance. The tooth-mobility was normal and the tooth reacted positively to electrical sensitivity tests.

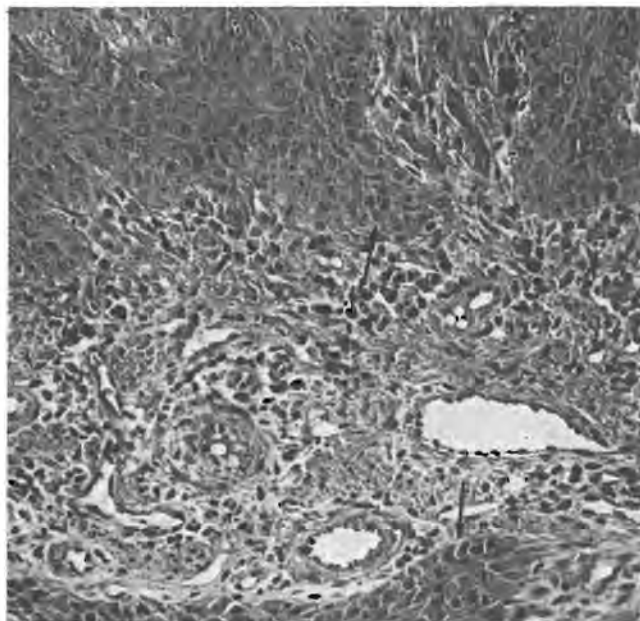
Radiographically the point of penetration was visible as a radiolucent area mesial to the pulpal chamber of the tooth. Root formation appeared to be complete. An accessory canal was also evident in the apical third of the root (Fig. 2).

Prior to root canal treatment an access cavity was prepared palatally in the tooth when it was established that the soft tissue tag was continuous with the pulp.

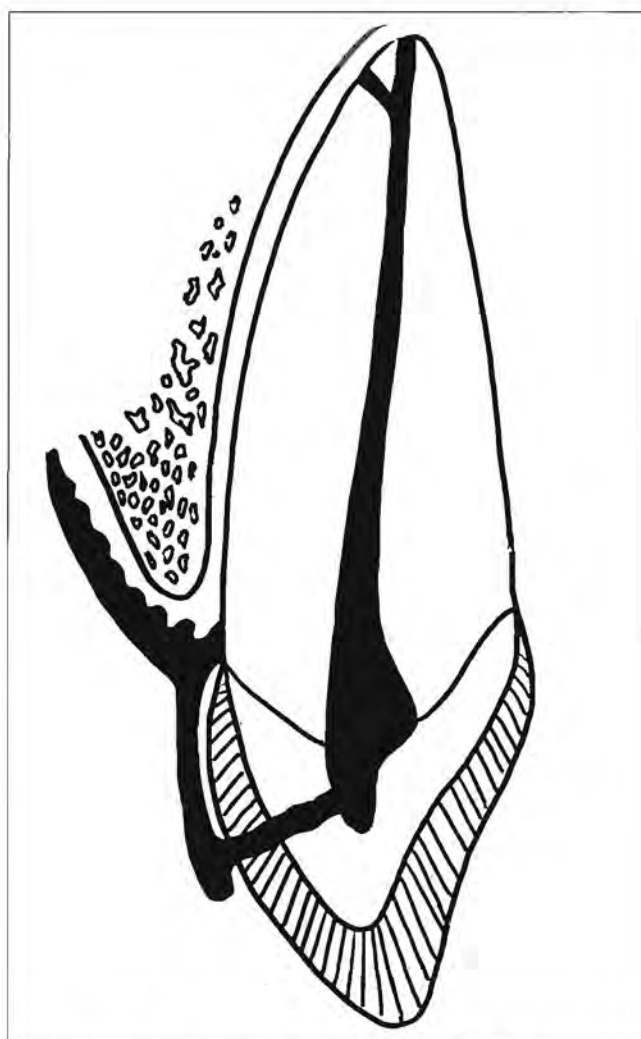
The soft tissue tag was then surgically removed from its pulpal connection and from the gingiva. On histological examination it was established that the soft tissue tag was completely covered by nonkeratinized epithelium and that the central core was made up of connective tissue which contained prominent blood vessels (Fig. 3). On completion of the root treatment, light cured restorative material was used to restore the hypoplastic defect on the labial surface of the tooth.

#### DISCUSSION

The reported case is unique in that both enamel and dentine defects appeared together with the very unusual soft tissue connection between the pulp and gingiva.



*Fig. 3: Histological appearance of the soft tissue tag. The covering epithelium (arrows) and prominent blood vessels within the central connective tissue of the tag are visible (H&E, x250).*



*Fig. 4: Schematic illustration of the soft tissue tag extending between the pulp and gingiva.*



Enamel matrix is laid down in a modified lamellar pattern and calcification normally takes place along the long axis of the tooth, proceeding from the incisal edge cervically. The position of the lesion on the tooth corresponded with the area of the tooth that had been forming and calcifying when the child was 2 years old (Wheeler, 1968). Severe insults either greatly disturb enamel production or produce death of the ameloblasts (Ten Cate, 1985). Odontoblasts are just as sensitive to insult (Linde, 1984). As a result of trauma to both the ameloblasts and odontoblasts that were actively forming enamel-and-dentine matrix at that time, subsequent necrosis and loss of the cells in that particular area of the forming tooth must have taken place. During the healing process, a fibrovascular connection was established between the dental papilla and the surrounding tooth follicle in the same location. In the final stage of the emergence of the tooth into the oral cavity the epithelium of the oral mucous membrane could have proliferated in an apical direction and merged with the epithelial cells of the reduced enamel epithelium in the affected area (Ten Cate, 1985) thus forming the attachment of the tag to the gingiva (Fig. 4).

#### CONCLUSION

This case clearly illustrates that trauma to the deciduous teeth may cause both enamel and dentine defects of the

permanent successors, while abnormalities in the soft tissue configuration may also occur.

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# Amelogenesis imperfecta: multiple impactions associated with odontogenic fibromas (WHO) type

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*Keywords: Amelogenesis imperfecta; fibroma; odontogenic fibroma*

## SUMMARY

Three types of amelogenesis imperfecta (AI) are recognised, namely hypoplastic, hypomature and hypocalcified varieties. We report on two cases of hypoplastic AI, the type which occurs most frequently. Both patients presented with multiple impacted permanent teeth. Odontogenic fibromas of the WHO type were found to be associated with the crowns of all the impacted teeth and are considered to have prevented normal eruption. Dentinal dysplasia found only in the furcation area of the multirrooted impacted teeth was evident. The macroscopic, microscopic and radiological appearance of the affected teeth, pericoronal lesions and interradicular dentinal dysplasia are described, and the most likely origins of the odontogenic fibromas and calcifications observed, are discussed.

## OPSOMMING

Drie tipes amelogenese imperfekta (AI) word aangetref namluk hipoplasties, hipovolwasse en hipogekalsifiseerde tipes. Hierdie artikel beskryf twee gevalle van hipoplastiese AI, diemees algemene tipe. Altwee pasiënte het veelvuldige geïmpakteerde tande gehad. Odontogene fibrome, WGO tipe, is aangrensend tot die geïmpakteerde tandkroon gevind en het moontlik erupsie vertraag. Dentinale displasie is slegs in die furkasiegebied van die geïmpakteerde molaartande gevind. Die makroskopiese, mikroskopiese en radiologiese beelde van die betrokke tande, perikoronale letsels en dentinale displasie word beskryf en die moontlike oorsprong van die odontogene fibrome en kalsifikasies wat waargeneem is, word bespreek.

## INTRODUCTION

Amelogenesis imperfecta (AI) is an inherited, congenital defect that primarily affects enamel formation and which is not accompanied by morphologic or metabolic defects in other body systems except abnormal tooth form or eruption (Witkop, 1989). The recent classification of Witkop (1989) describes different types of AI according to the predominant clinical and histological characteristics as well as the mode of Mendelian inheritance. The enamel abnormality can be either hypoplastic, hypomature, hypocalcified or a combination of these with autosomal dominant, autosomal recessive, sex-linked dominant or sex-linked recessive modes of inheritance (Table I). The hypoplastic type is characterised by thin, hard enamel of normal radiographic translucency. This type is the result of insufficient matrix formation with normal mineralisation. Hypomature enamel is a result of a defect in the formation of crystalline apatite in various parts of the enamel rods and sheaths. The enamel is of normal thickness with a mottled appearance, is slightly softer than normal and chips off the dentine. Radiographically it has approximately the same density as dentine. Hypocalcified enamel develops to a normal thickness but is lost soon after eruption. It is the result of defective mineralisation of the formed matrix and radiographically the enamel is less radiodense than dentine (Witkop and Sauk, 1976).

The combined prevalence of all types of AI has been reported to be 1:14 000 in the United States (Witkop and Sauk, 1976); 1:8 000 in Israel (Chosack *et al*, 1979) and 1:4 000 in Sweden (Sundell and Valentin, 1986). The most common

type of AI is the hypoplastic variety with a reported prevalence that varies from 1:8 000 (Chosack *et al*, 1979) to 1:6 700 (Sundell and Valentin, 1986). Impacted teeth are often associated with the smooth hypoplastic type and, less frequently, with the rough hypoplastic type (Witkop and Sauk, 1976).

The purpose of this paper is to report two cases of rough hypoplastic amelogenesis imperfecta associated with impacted teeth and pericoronal odontogenic fibromas of the WHO type.

## CASE 1

A 14-year-old girl presented for treatment with the main complaint of delayed eruption of her teeth. The child had no systemic abnormalities. Intraoral examination revealed thin, hard enamel on all the erupted teeth. The enamel surface varied from smooth to rough and had a yellow-white colour. The teeth failed to meet at the interproximal contact points. The patient had 5 sisters of whom 3 had AI with the same enamel appearance. The mother had normal teeth but the father was edentulous. His teeth had been extracted at a young age. This mode of inheritance was suggestive of an autosomal dominant inheritance pattern.

Radiographic examination revealed the normal number of teeth, of which 13 were unerupted, including the 4 developing third molars. Dilated follicles or cyst-like lesions were apparent as well demarcated radiolucencies with sclerotic margins associated with the crowns of the unerupted teeth (Fig 1). No well developed enamel could be seen. The roots of the molar teeth showed gross disfigurement with structures suggestive of pulpal calcifications.

**Table 1: Classification of amelogenesis imperfecta according to Witkop (1989)**

Type I	- Hypoplastic
IA	- hypoplastic, pitted autosomal dominant
IB	- hypoplastic, local autosomal dominant
IC	- hypoplastic, local autosomal recessive
ID	- hypoplastic, smooth autosomal dominant
IE	- hypoplastic, smooth X-linked dominant
IF	- hypoplastic, rough autosomal dominant
IG	- enamel agenesis, autosomal recessive
Type II	- Hypomaturation
IIA	- hypomaturation, pigmented autosomal recessive
IIB	- hypomaturation, X-linked recessive
IID	- snow capped teeth, autosomal dominant
Type III	- Hypocalcified
IIIA	- autosomal dominant
IIIB	- autosomal recessive
Type IV	- Hypomaturation-hypoplastic with taurodontism
IVA	- Hypomaturation-hypoplastic with taurodontism, autosomal dominant
IVB	- Hypoplastic-hypomaturation with taurodontism, autosomal dominant



**Fig. 1: Case 1. Pantomograph showing unerupted teeth with pericoronal radiolucencies (small arrows). No evidence of enamel is present and the unerupted molar teeth show gross root disfigurement (large arrows).**

All the unerupted teeth with the associated pericoronal tissue were removed surgically under general anaesthesia. The bone was found to be normal in texture and no excessive haemorrhage was encountered. Post-operative healing was uneventful.

Light microscopy of ground sections of the molar teeth showed irregular hypoplastic enamel with globular calcifications. The dentinoenamel junction lacked the normal scalloping (Fig 2). The dentine of the crowns and roots showed no abnormalities. An irregular mass of dentine was present in the interradicular area at the level of root bifurcation in all the molar teeth. Hypercementosis, consisting of cellular cementum extending into the interradicular space of the roots, was present.

Calcified globules with an onion-like appearance were present in the cementum in close association with the irregular dentine (Fig 3). False pulpal stones, not associated with the dentinal wall, were observed.

Scanning electron microscopy (SEM) of the outer enamel surface showed irregular globular and linear masses in association with depressions (Fig 4). SEM of the fractured surface confirmed the straight dentinoenamel junction and showed normal dentine. The enamel had voids and loss of structure with a resulting honeycomb appearance throughout its full thickness (Fig 5).



**Fig. 2: Ground section of an unerupted molar tooth with irregular enamel and globular calcifications (bold arrows). Note the straight dentinoenamel junction (fine arrows) x 100.**



**Fig. 3: Interradicular dentine dysplasia (bold arrows) associated with hypercementosis (c) and globular calcifications (fine arrows). Ground section, unstained x 10. Inset: Calcified globules with an onion-like appearance. Ground section, unstained x 200.**

The pericoronal tissue consisted of fibrous tissue that varied in cellularity. No evidence of a cystic lining was found. Odontogenic epithelial cell rests were scattered in the connective tissue. These epithelial cells appeared to be inactive with no peripheral pallisading of ameloblast like cells. Some of the epithelial cells had a vacuolated appearance. Two types of calcifications were present in the fibrous tissue. The most common type consisted of psammomatous lamellar bodies with an eosinophilic centre and a more basophilic peripheral zone. The second type consisted of eosinophilic material with a fibrillar matrix and peripheral tufts resembling Sharpey's fibres. Both types were closely associated with the odontogenic epithelial cell rests (Fig 6). The lesions were considered to be odontogenic fibromas, WHO type.

## CASE 2

A 26-year-old black female reported to the hospital, requesting that she be fitted with full upper and lower dentures. The patient was clinically edentulous and had marked vertical enlargement of the entire alveolar ridge in all four quadrants. No abnormalities were found on systemic examination.

Radiological examination confirmed the enlargement of all four quadrants with both maxillary tuberosities markedly overdeveloped. There was evidence of recent tooth extractions in the mandible in the form of healing sockets and 13

Amelogenesis imperfecta

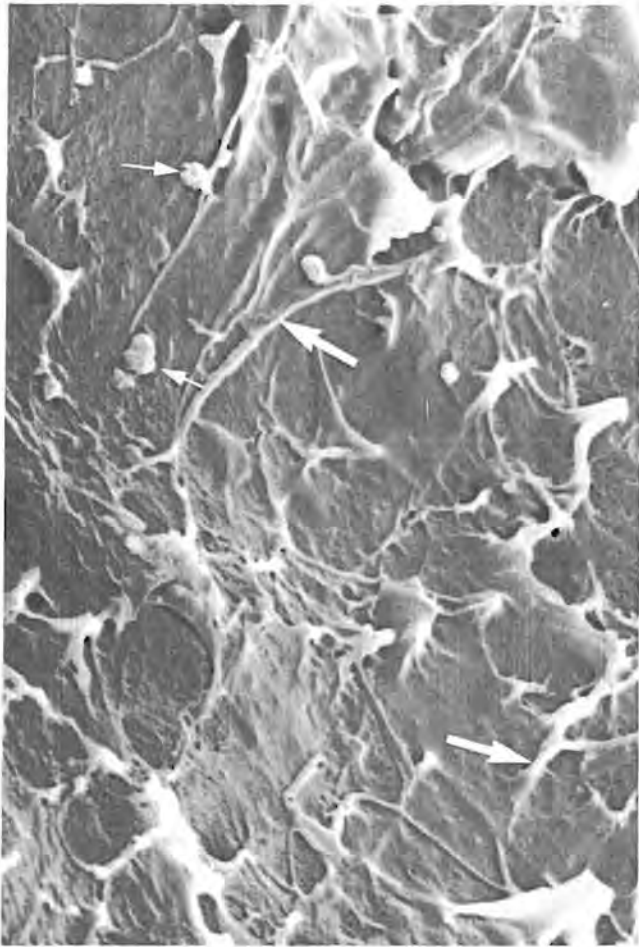


Fig. 4: The outer enamel surface showing globular (fine arrows) and linear (bold arrows) masses associated with depressions  $\times 2\ 000$ .

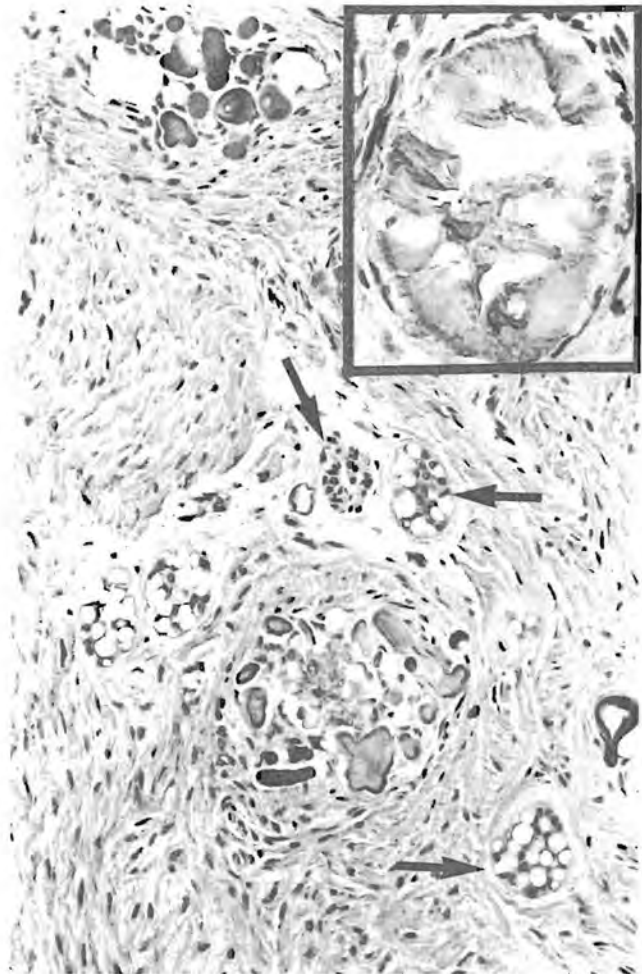


Fig. 6: Odontogenic epithelium (arrows) associated with psammomatous calcifications in a fibrous stroma. H and E  $\times 100$ . Inset: Fibrillar calcification with peripheral tufts. H and E  $\times 200$ .



Fig. 5: SEM of the fractured surface confirmed the straight dentinoenamel junction (bold arrows) and a honeycomb appearance in the enamel (fine arrows)  $\times 72$ .

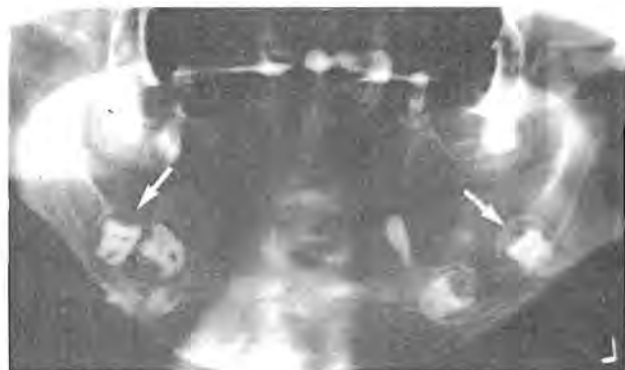


Fig. 7: Case 2. Pantomograph showing impacted teeth with pericoronal radiolucencies (arrows).

impacted teeth could be observed within the four quadrants. The enamel of the crowns of all teeth appeared markedly hypoplastic, with abnormally shaped pulp chambers which were smaller than normal. The roots of the teeth were malformed, shorter than normal, with occasional dilaceration. The crowns of the impacted teeth were surrounded by what looked like hyperplastic follicles. The follicular spaces were less radiolucent than normal (Fig 7). Radiological examination of the skeleton showed no abnormalities.

Macroscopic examination of an impacted molar tooth revealed thin, hard enamel with a granular appearance. The enamel could easily be chipped off. Microscopic examina-

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tion of a 50  $\mu\text{m}$  ground section showed normal dentine with an almost flat dentinoenamel junction. The enamel was thinner than normal and short curling enamel rods were seen. These were covered by irregular globular calcified masses (Fig 8). These features were consistent with rough hypoplastic amelogenesis imperfecta. The mode of inheritance could not be established. The pericoronal lesions had the same microscopic appearance as in case 1 (Fig 9).

## DISCUSSION

Calcifications associated with odontogenic epithelial remnants have been reported in odontodysplasia, impacted dens in dente, congenitally absent teeth in which there is an attempt at tooth formation and several types of AI (Witkop and Sauk, 1976). Odontogenic epithelium was present in 60 cases and calcifications in 54 cases of the 130 cases of opercula of impacted third molars (Cutright, 1976). Gardner and Sapp (1973) described two types of calcifications designated types A and B associated with the soft tissue and a periapical area of an involved tooth of a patient with regional odontodysplasia. The type A and B calcifications are similar in appearance to the two types that were found in our cases. Calcifications are also frequently found in the excised gingivae covering unerupted teeth in patients with AI (Nakata, Kimura and Bixler, 1985; Bab *et al* 1985, Ooya, Nalbandian and Noikura, 1988).

Our radiological differential diagnosis of pericoronal radiolucent lesions was dilated dental follicles, hyperplastic dental follicles, follicular cysts or odontogenic fibromas. Normally some teeth have dilated follicles in the pre-eruptive phase but according to Shear (1983) it does not signify a cyst unless the pericoronal width is at least 3-4 mm as measured on a radiograph. The hyperplastic follicle presents macroscopically as a solid rather than cystic lesion and no signs of a cyst can be seen microscopically. The histological appearance of hyperplastic follicles and odontogenic fibromas are similar. According to Gardner (1980) the distinction is based on the size and location of the lesion. The follicles are invariably associated with the crowns of unerupted teeth whereas it is not necessarily true for odontogenic fibromas. Sandler *et al* (1988) reported a case of a 16-year-old boy with 13 unerupted teeth, each one associated with hyperplastic pericoronal tissue that had histological features suggestive of the WHO type of odontogenic fibroma. The erupted as well as removed impacted teeth in their case were macroscopically normal. Gardner (1980) considers the WHO type of odontogenic fibroma to be a fibroblastic neoplasm. The pericoronal location of the tumours in our two patients suggested a follicular origin. The association of this fibroma-like tissue with impacted and unerupted teeth in AI suggested a hamartomatous lesion rather than a neoplasm. It is possible that the WHO type of odontogenic fibromas associated with impacted teeth, as in our cases, have a different histogenesis than the tumours described by Doyle, Lamster and Baden (1985), as none of their 6 cases was in a pericoronal location. Dunlap and Barker (1984) consider the central odontogenic fibroma of the WHO type to be the morphologic and histogenetic counterpart of the peripheral odontogenic fibroma. The authors postulated an ectomesenchymal-epithelial interaction in the histogenesis of this tumour. The close association of calcifications with odontogenic epithelium in both our cases supported their theory.



Fig. 8: Thin abnormal enamel covered by irregular globular calcifications (bold arrows). Unstained  $\times 100$ .

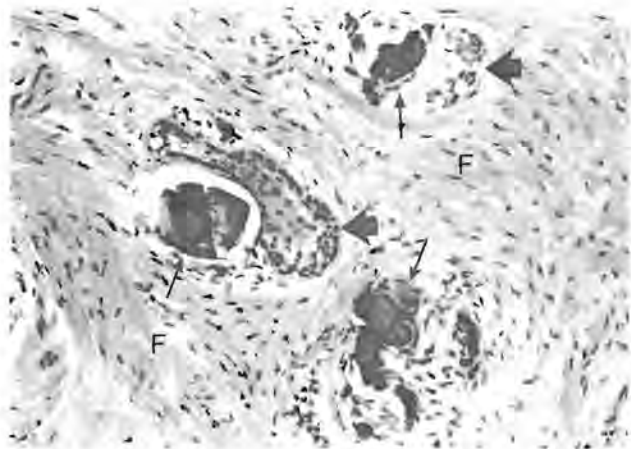


Fig. 9: Odontogenic epithelium (bold arrows) closely associated with psammomatous calcifications (fine arrows) in a cellular fibrous tissue (F). H and E  $\times 150$ .

AI associated with interradicular dentinal dysplasia has been reported by Nakata *et al* (1985). They suggested 3 possible mechanisms for the presence of dysplastic dentine: resorption of the interradicular area followed by secondary calcification; gene influence on matrix formation in this area; and secondary calcification for some unknown reason. No sign of resorption of roots or crowns of the impacted teeth in our cases was found. No abnormalities in the roots of single rooted teeth were seen on radiological and microscopic examination. This is an indication that the underlying cause is likely to be associated with the process of root branching. A genetic influence responsible for the abnormal interradicular dentine is unlikely since the abnormal

dentine present in our first patient did not occur in her 3 sisters who had AI. They had no other dental abnormalities or impacted teeth. The erupted molar teeth of the first patient showed no radiological evidence of root abnormalities. The association between the interradicular abnormalities and impactions was unclear. No abnormalities apart from AI could be seen on the impacted single rooted teeth. It is unlikely that a disturbance affecting the eruption occurred first and then caused a secondary abnormality of the interradicular area of the impacted teeth as suggested by Nakata *et al* (1985). It has been shown that eruption proceeds normally in the absence of root formation (Cahill and Marks, 1980). Both erupted and impacted molars in the AI patient reported by Nakata *et al* had interradicular dental dysplasia. The odontogenic fibromas WHO type associated with the pericoronal areas were probably the main reason for the impaction of teeth in both our cases. The suggested follicular origin of the odontogenic fibromas as a hamartomatous growth under the influence of the follicular epithelium supported this statement as Cahill and Marks (1980) have shown that a dental follicle is required for the eruption of a tooth.

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# REGIONAL ODONTODYSPLASIA ASSOCIATED WITH SOFT TISSUE SWELLING

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Odontodysplasia is an uncommon developmental anomaly with an unknown cause affecting both dentin and enamel of a group of adjacent teeth. The maxilla is involved twice as often as the mandible<sup>1</sup>. The condition is more common in the anterior than in the posterior regions and affects women more frequently than men (ratio 1.4:1)<sup>1</sup>.

This condition has been reported under a variety of names, such as odontogenic dysplasia<sup>2</sup>, localised arrested tooth development<sup>3</sup> and ghost teeth<sup>4</sup>. Regional odontodysplasia however, has become the accepted terminology, because the condition tends to affect several adjacent teeth within a particular segment or region of the jaw. Radiographically, the affected teeth demonstrate a ghost-like appearance with little demarcation between enamel and dentin, wide pulpal chambers and open apices. Clinically, the teeth appear discoloured, hypocalcified and hypoplastic. Delayed eruption of affected teeth is common.

**The purpose of this article is to report a case of regional odontodysplasia associated with a soft tissue tumour.**

## CASE REPORT

A healthy 8-year-old female presented at the clinic complaining of a painless tumour in the anterior mandible. She first noticed the lesion 4 months previously and it progressively increased in size over this period.

Examination revealed a round, bony hard tumour of 2 cm in size on the alveolus between the 41 and first 84 (Fig. 1). The tip of a tooth had erupted through the posterior aspect of the tumour. The mucosa was slightly erythematous in one area and pigmented in another, without any ulceration being present.

Radiographs revealed a mixed dentition, with the developing canines and premolar teeth, and 31 and 32 appearing normal. The 84, 85 and 46 were in their normal positions. The tooth of which the tip alone was visible was found to be the 83. The 41 and 42 were totally embedded in the tumour and of decreased radiodensity while their roots

were hypoplastic (Fig. 2). The tumour itself was well circumscribed and had a ground-glass appearance.

Under general anaesthesia the mucosa was reflected from the lingual and buccal aspects of the tumour. It shelled out easily from a bony cavity in the alveolus, with the malformed 41 and 42 completely enclosed. The wound was sutured primarily and healing was uneventful.

The surgical specimen consisted of 2 teeth embedded in a firm fibrous tumour. The teeth showed variable degrees of surface hypoplasia and were decalcified for histological examination.

Microscopic examination of the teeth and associated soft tissue showed hypoplastic dentin with a prominent predentine layer and numerous interglobular masses. The pulpal horns were high and pulp stones were present. The reduced enamel epithelium around the unerupted teeth showed numerous calcifications (Fig. 3). The adjacent tumour consisted of cellular fibrous tissue with numerous islands of small round amorphous calcifications distributed throughout the fibrous tissue. Odontogenic epithelium islands were noted in areas, the majority however, were associated with the calcifications. This fibrous tissue mass was well demarcated from the cortical and medullary bone. The diagnosis of regional odontodysplasia was confirmed.



Fig. 1



Fig. 2

## DISCUSSION

This case is unusual in that the patient complained about a soft tissue tumour and was unaware about the associated "dental problem". Regional odontodysplasia has been reported in association with ipsilateral hypoplasia of the face<sup>5</sup>, epidermal nevus syndrome<sup>6</sup> and hydrocephalus<sup>7</sup>. In a recent review of the world literature, 109 cases of regional odontodysplasia were described and it was found that none were associated with a soft tissue tumour<sup>1</sup>. Neupert & Wright however, later described a case of regional odontodysplasia in the maxilla associated with a soft tissue swelling with similar histologic features as the present case<sup>8</sup>.

The majority of cases reported in the literature were treated by extraction of the affected teeth. These teeth are

frequently painful and associated with abscess formation due to the inability of the aberrant enamel and dentine to resist bacterial invasion. Crawford and Aldred are of the opinion that noninfected affected teeth be saved wherever possible<sup>1</sup>. However, facial cellulitis appear to be a complication if these teeth are retained<sup>8</sup>.

## ACKNOWLEDGEMENTS

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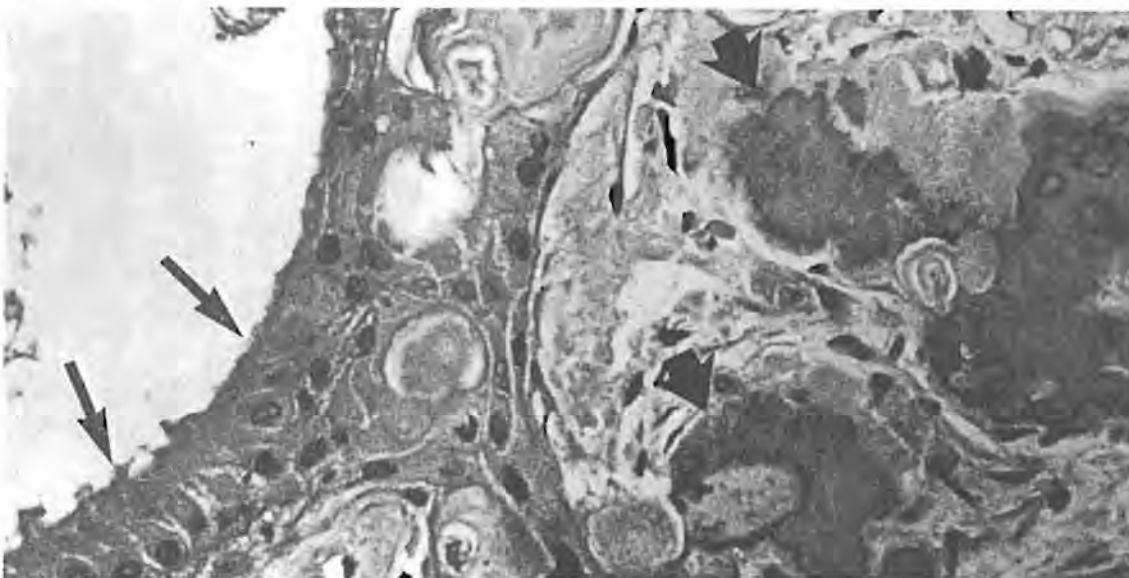


Fig. 3



# The use of tricure glass ionomer cement as an apical sealant after apicoectomy

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*Keywords: apicoectomy; glass ionomer cement; sealing ability.*

## SUMMARY

*The adaptation and sealing ability of a tricure glass ionomer material (Vitremmer), used in a retrograde cavity was assessed and compared with amalgam. Fifty single-rooted, extracted teeth were prepared and filled endodontically. All teeth underwent root resection and retrograde cavities were prepared. The teeth were divided into two groups. One group of 10 teeth received a layer of varnish and an amalgam filling in a retrograde cavity, while the other group of 40 teeth received a layer of primer and a glass ionomer filling. All the teeth were placed in an aqueous solution of Procion Brilliant Blue for 7 days whereafter ground sections were prepared. Micro leakage was determined according to the extent of dye penetration using an image analysis system. The results showed that significantly less dye penetration was observed in teeth filled with the glass ionomer cement than in those with the amalgam.*

## OPSOMMING

*Die adaptasie en seëlvermoë van 'n driedigekuuraksie glasionomeer materiaal (Vitremmer) wat in 'n retrograde kaweiteit gebruik is, is ondersoek en met amalgaam vergelyk. Vyftig enkelwortel verwyderde tande is endodonties gevul en in twee groepe verdeel. Een groep, bestaande uit 10 tande, het 'n laag vernis en amalgaamvulsel in 'n retrograde kaweiteit ontvang, terwyl die groep van 40 tande een laag "primer" en glasionomeer vulsel ontvang het. Al die tande is in 'n oplossing van Procion Brilliant Blue in water vir 7 dae geplaas waarna slypsels voorberei is. Mikrolekkasie is bepaal na gelang van die mate van kleurstofpenetrasie soos vasgestel deur 'n beeldanalise sisteem. Die resultate het getoon dat kleurstofpenetrasie betekenisvol minder was met die glasionomeer sement in vergelyking met amalgaam.*

## INTRODUCTION

An ideal material for retrograde apical seal has yet to be found. Studies have shown that amalgam does not provide an effective apical seal (Abdal, Retief and Jamison, 1982; Stabholtz *et al.*, 1985; Szeremeta-Browar, van Cura and Zaki, 1985). Further investigation into seeking an alternative retrograde root filling material would improve endodontic treatment. Vitremmer light-curing glass ionomer cement may prove to be an effective sealant that can be used in the moist environment found in root resection. This material exhibits almost no water sensitivity after 20 seconds of irradiation and is the only restorative material that forms a strong bond to dentine in a moist environment (Katsuyama, Tatsuya & Benji, 1993). The material has been shown to be biocompatible by both cell toxicity and biological testing (Zetterquist, Anneroth and Nordenram, 1987).

Previous studies (Chong, Pitt Ford and Watson, 1991; 1993) using a light-cured glass ionomer cement as a retrograde filling material showed that good adaptation was achieved to one cavity wall, but gaps were observed on the opposing wall.

We postulate that it is important to prepare a retrograde cavity when using glass ionomer material because at present it is still questionable whether the marginal seal will be maintained satisfactorily over a long period of time. This uncertainty is due to dimensional changes, such as from shrinkage at the time of setting, dissolution and exposure to various harmful factors such as external forces (Katsuyama *et al.*, 1993).

The purpose of this study was to assess the sealing ability of a tricure glass ionomer material (Vitremmer) when used as a retrograde root cavity filling material.

## MATERIALS AND METHODS

Fifty extracted single rooted human teeth consisting of incisors, canines and premolars were collected from the Department of Maxillofacial and

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WFP van Heerden, BChD (Pret), MChD (Oral Path) (Pret).

Oral Surgery at Medunsa and stored in de-ionized water at 37°C. An access cavity was prepared through the crown into the pulp space of each tooth. The root canals of all the teeth were prepared and filled with laterally condensed Gutta-Percha and Roth Root Canal Sealer (Roth International, Chicago, IL). The access cavity was sealed with IRM temporary filling material (L.D. Chaulk Co, Milford, DE). The external surfaces were coated with a layer of nail varnish. The teeth were then stored in de-ionized water at 37°C for 7 days. All teeth were subsequently resected with an ISO size 009 plain cut tungsten carbide bur (H23L) (Komet, Brassler, Lengo, Germany) at high speed (300 000rpm) with water coolant. Approximately 3mm of the root apex was removed and the root surface was bevelled labially at approximately 45° to the long axis of the root. All the resected root surfaces were of approximately similar dimension. The teeth were then divided into two groups: Group 1: 10 teeth for conventional amalgam fillings

Group 2: 40 teeth for tricure glass ionomer fillings

In both groups a single surface retrograde cavity was prepared in each tooth with an ISO size 016 round diamond bur (801)(Komet) at high speed with water coolant to a depth of between 2 and 3mm, measured from the labial margin of the cavity. The cavity was circular in cross-section, with a diameter of approximately 1,6mm. All the teeth were rinsed with water and dried with air from a three-in-one syringe before placement of the test materials.

#### Group 1:

The retrograde preparations received one application of cavity varnish (Copalite; H.J. Bosworth Co, Skokie, IL USA) applied with a paper point and blown dry to evaporate solvent, followed by a retro filling of high copper dispersed phase amalgam alloy (Dispersalloy; Johnson & Johnson, East Windsor, N.J. USA) mixed according to the manufacturer's instructions. The amalgam was carried to the retrograde cavities with an amalgam carrier and the condensation was done manually.

#### Group 2:

The primer was applied and air dried immediately. The Vitremer tricure glass ionomer material (3M Dental Products, St Paul, Minnesota, USA, Batch no: 19930116) was mixed and placed in bulk, according to the manufacturer's instructions. The glass ionomer cement was cured for 10 seconds using a light-curing unit (Chaulk Max; Caulk, Dentsply, Milford, DE, USA).

After material placement, all teeth from both groups were stored in an aqueous solution of Procion Brilliant Crestal Blue (Merck; Fedlife Park, Midrand, RSA) at 37°C for 7 days. The pH of the solution

was 7,59. Vitremer tricure glass ionomer material sets by means of exposure to visible light. It also has two self-curing mechanisms to provide a relatively rapid set where light does not penetrate and thus allows for bulk placement. It also shows much less contraction shrinkage than the previous light-cured glass ionomers (3M Dental Products, St. Paul, Minnesota, USA; Vitremer Technical Product Profile, 1992, 0.8)

After the storage period of 7 days the teeth from both groups were embedded in resin and sectioned longitudinally in a buccal-lingual plane with a low speed diamond saw (Low Speed Isomet Saw; Beuhler, Lake Bluff, IL, USA). A one millimetre section was cut off each tooth and then ground on carborundum paper to approximately 30µm thickness. The sections were subsequently mounted onto microscope glass slides with mounting media. The amount of leakage was scored according to the extent of dye penetration along the cavity walls. The extent of dye penetration was measured using an image analysis system (FIPS; Wirsam, Auckland Park, RSA) connected to a Nikon Ophthiphot microscope (IMP; Pretoria, RSA). For every tooth, two measurements were obtained from both sides of the retrograde cavity walls and expressed in micrometer penetration. Dye penetration beyond the prepared cavity was not measured. An average measurement was then calculated for each tooth.

The results from the dye leakage study were analysed using the Student's t-test for uncorrelated data.

## RESULTS

Three of the 40 teeth filled with Vitremer and one tooth filled with amalgam could not be used for dye penetration analysis, as they fractured during the cutting and grinding process. The dye penetration was present as a well defined blue line on the resected root surface and along the cavity walls. The mean distance of dye penetration in the group with amalgam fillings was 1774µm ± 947µm. These measurements ranged from 816µm to 3137µm (Fig. 1). The mean distance of dye penetration in the group with tricure glass ionomer fillings was 83,8µm ± 185µm. The measurements ranged from 14,9µm to 823,5µm (Figs. 2 and 3). The mean difference of dye penetration between these two groups was statistically highly significant (p < 0,001).

## DISCUSSION

Bacterial invasion of the pulp space through dentinal tubules have been described in previous studies (Hoshino *et al.*, 1992; Kiryu, Hoshino and Iwaku, 1994). The development and maintenance of a hermetic seal is considered to be a major prerequisite for success in root canal treatment. The

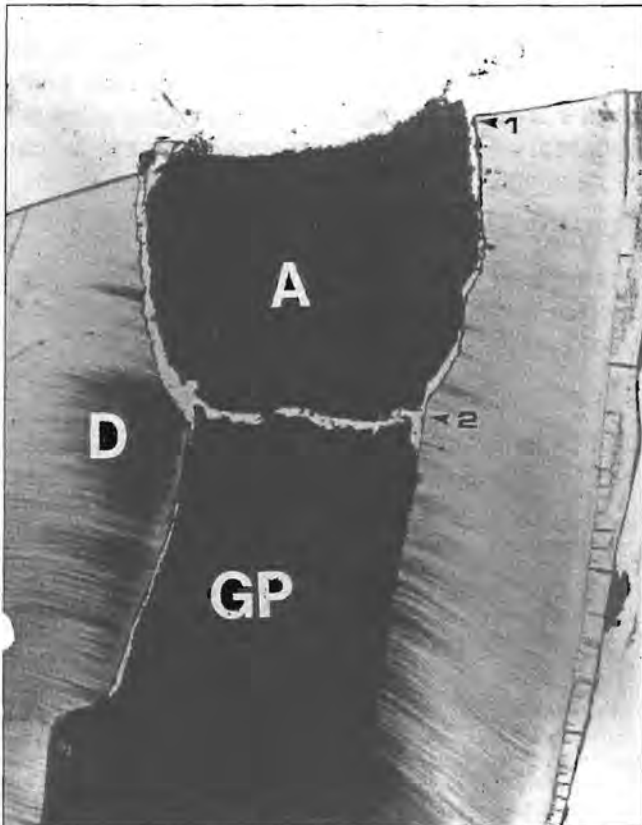


Fig 1: Photomicrograph of a ground section showing the amalgam retrograde filling (A) and Gutta Percha (GP). Dye leakage was observed extending the full length of the amalgam filling (1 to 2). Dentine = D. (Original magnification: x5).

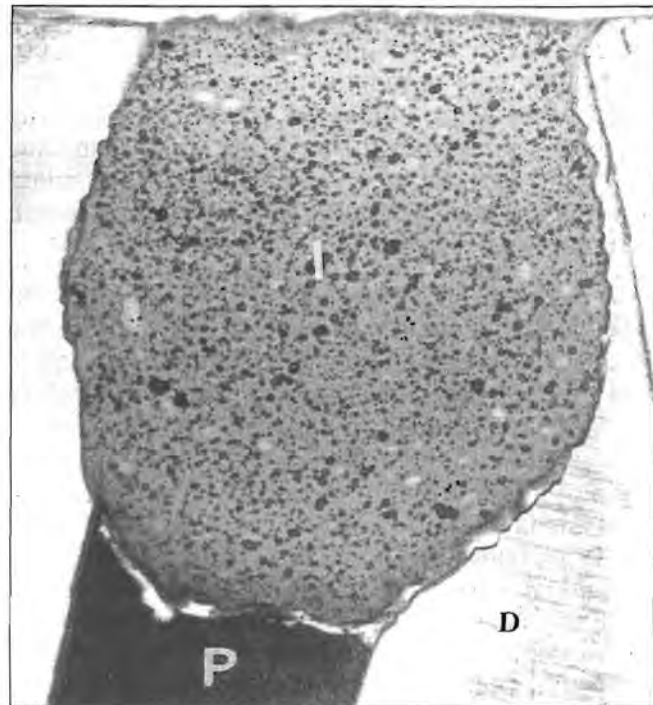


Fig 2: Photomicrograph of a ground section showing the glass ionomer retrograde filling (I) and Gutta Percha (P). Dentine = D. (Original magnification x10).

evaluation of the quality of the root canal filling using leakage tests is therefore still relevant (Wu and Wesselink, 1993). Glass ionomer materials, including cements, are technique sensitive (Bowen and Marjenhoff, 1992). The necessity to have a clean, dry dentinal surface on which to place these materials, remains the major problem to the clinician. Special instruments are available to facilitate debridement and obturation of the root canal space from a retrograde direction (Flath and Hicks, 1987), whilst root end isolation techniques for retrograde fillings in order to obtain and maintain a sterile, dry environment, have been described (Guerra, 1992).

Contrary to the findings of Chong *et al.*, (1993) using a light-cured glass ionomer material, the tricure glass ionomer material in this study was observed to be well adapted to both the cavity walls in all specimens. This finding is in line with the statement of the manufacturers that this material undergoes less polymerization contraction than previous glass ionomers and can be placed in bulk. The suggestion by Watson (1990) that a light-cured glass ionomer material should only be used in thin layers, is therefore not applicable to this specific material according to our results.

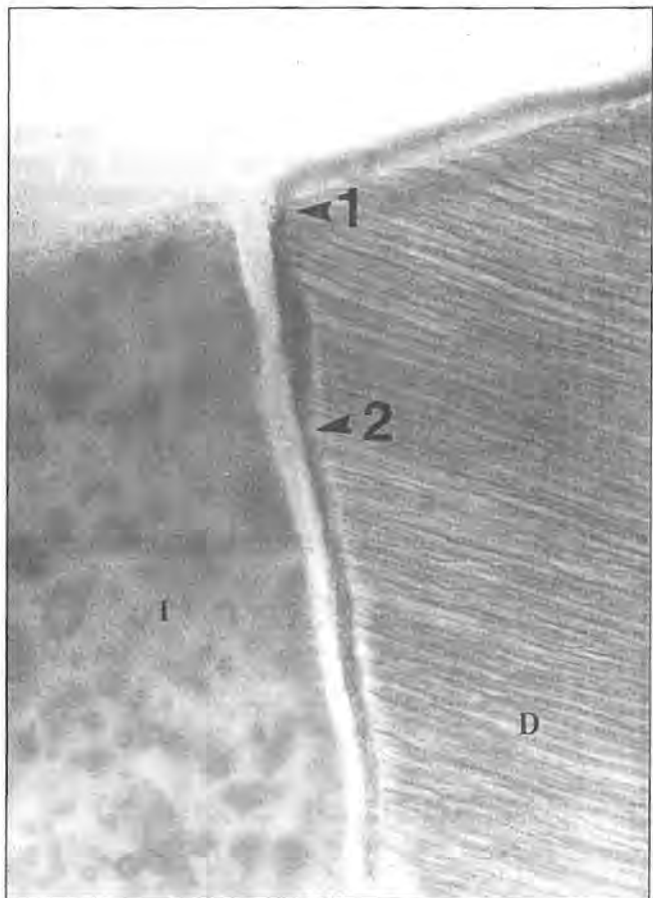


Fig 3: Photomicrograph of a ground section showing the extent of dye penetration (from 1 to 2) with a glass ionomer filling (I). Dentine = D. (Original magnification x100).

In this study the glass ionomer material was placed in bulk and cured for only 10 seconds (i.e., 50 per cent of the time recommended by the manufacturers) before being immersed in the dye. This was done in an attempt to make the study clinically more relevant since it is often difficult to isolate the apex of a tooth and keep it dry for any length of time.

Measurement of dye penetration by using an image analysing system linked to a light microscope is the method of choice. It measures the actual dye penetration in scientific units and is therefore reproducible and allows for meaningful statistical analysis. The dye penetration results in this study indicated that a 3mm retrograde amalgam filling with one layer of varnish did not give a predictable seal, implying that communication between the periapical area and the pulp chamber was therefore still possible. This is demonstrated by the mean depth of dye penetration around amalgam (1774µm) which means that in most cases the dye leakage reached the base of the cavity (2000-3000µm deep). The results also demonstrated that it was possible to seal the canal orifice of a retrograde preparation to prevent the dye from reaching the pulp chamber by the bulk placement of a tricure glass ionomer material *in vitro*. This is demonstrated by the mean depth of dye penetration around Vitremer (185µm) which indicates that dye penetration never extended to the cavity floor. *In vivo* studies are necessary to determine the clinical relevance of these observations. The changing of formulae, as well as the addition of new chemicals to dental products, can serve as potential irritants and sensitizing agents. It is therefore important to do biological testing before clinical application of any new material.

#### CONCLUSION

A 3mm retrograde amalgam filling with one layer of varnish did not give predictable seal *in vitro*. Communication between the pulp chamber and

the periapical tissue may therefore still be possible immediately post operatively. Bulk placement of a 3mm tricure glass ionomer material filling gave a predictable seal *in vitro*. *In vivo* studies are necessary before this glass ionomer material can be recommended for routine clinical use.

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## SHORT COMMUNICATION

### AMINO ACID COMPOSITION OF DENTINE IN PERMANENT HUMAN TEETH

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(Accepted 20 August 1991)

**Summary**—Dentine of permanent mandibular incisors from nine individuals was hydrolysed and the amino acid composition determined by ion-exchange chromatography against a standard calibrant of 41 amino acids. Nineteen amino acids were detected, including small quantities of 1-methylhistidine and asparagine, two amino acids whose existence had apparently not been recorded before in human dentine. The total content of hydroxylysine plus lysine varied between 2.6 and 3.3 residues per 100 (SD, 0.74) in different teeth, which therefore did not support previous studies that had proposed a constant total value. This and other quantifiable differences between present and previous findings may be the result of the different methods and the influence of dietary and other regional factors on dentinogenesis.

**Key words:** human dentine, amino acids.

Dentine is the major component of teeth, responsible for most of the weight, volume and overall shape (Butler, Munksgaard and Richardson, 1979). Ninety per cent of the organic matrix of human dentine consists of collagenous proteins (Jones and Leaver, 1974) and the remaining 10% is made up of non-collagenous proteins, proteoglycans, glycoproteins and lipids (Avery, 1987). Various analytical methods, some of which are historic, have been used for determination of amino acids in dentine, namely: microbiological assay (Hess, Lee and Neidig, 1952), quantitative paper chromatography (Battistone and Burnett, 1956) and ion-exchange chromatography (Eastoe, 1963).

Our objective now was to determine the amino acid composition of human dentinal matrix with a modern technique and to compare it with previously published data.

Permanent mandibular incisors were extracted from nine bodies of known age and sex in the Forensic Medicine mortuary of Ga-Rankuwa Hospital, situated 32 km north of Pretoria. Before processing, the crowns and cementum were removed with a dental bur, and the pulp with an endodontic file. Pieces of radicular dentine of approx. 0.20 g were washed, dried and hydrolysed in 6 M hydrochloric acid (HCl) for 24 h at 110°C. The hydrolysates were neutralized with neutralizer (Spitz, 1973) and citrate buffers in the ratio 1:2:2, filtered (Millex-GS 0.22 µm) and then diluted further 1:1 with the citrate buffer. Calibrants containing 41 amino acids were prepared and diluted as above. The amino acids of dentine and the calibrants were separated in duplicate by ion exchange on a Beckman 6300 amino acid analyser which incorporates a 25-cm lithium column

and a four-buffer system. Chromatograms thus obtained were integrated and quantitated with a Hewlett-Packard 3390A integrator and the results expressed as a per cent residues detected. The results were tabled as the average of the total number of residues per 100 and the SD for each amino acid was calculated.

Nineteen amino acids were detected (Table 1). Asparagine and 1-methylhistidine, which have not previously been identified in human dentine, were present in small quantities. Asparagine was present in all our hydrolysates. In a serial study of hydrolysis, asparagine was detected only after 16 h and remained present in all acid hydrolysates for 24 h. In hydrolysates stored at 4°C, asparagine could be detected over as long as 12 months. No explanation for this phenomenon could be found. Acid hydrolysis of a pure mixture of aspartic acid, asparagine, glutamic acid and glutamine showed complete hydrolysis of asparagine and glutamine within 30 min. The concentration of aspartic acid and glutamic acid increased and high levels were detected under these conditions. It is suggested that asparagine in dentine may be 'protected' against complete hydrolysis. Furthermore, our monitoring system may have been more sensitive and with improved resolution as larger quantities of aspartic acid, glutamic acid, arginine, leucine, iso-leucine and valine were found than previously reported (Table 1).

Linde (1984) reported that the total content of hydroxylysine plus lysine in dentine is constant at 3.5 residues per 100. In our study, we found a variation between 2.6 and 3.3 residues per 100 (SD, 0.74), supporting Eastoe's (1963) finding of a variation in the total content of hydroxylysine and lysine over a



Table 1. Comparison of our findings (average of nine cases, with SD) with those of previous investigations (expressed as residues per 100)

Human dentine amino acids	Hess <i>et al.</i> (1952)	Battistone and Burnett (1956)	Eastoe (1963)	Linde (1984)	Present study (1992)	
					X	SD
Aspartic acid	4.4	5.4	5.5	4.5	5.9	0.06
Hydroxyproline	10.3	11.6	10.1	9.6	10.4	0.24
Threonine	2.7	2.0	1.9	1.8	2.1	0.11
Serine	3.4	3.0	3.8	4.1	4.0	0.28
Asparagine	—	—	—	—	0.3	0.08
Glutamic acid	7.4	7.6	7.3	7.2	8.8	0.15
Proline	14.5	9.7	11.5	11.9	11.8	0.28
Glycine	30.9	31.3	31.9	33.4	30.1	0.30
Alanine	9.8	11.2	11.2	10.2	8.6	0.31
Valine	2.6	2.5	2.5	2.3	3.0	0.15
Methionine	0.35	0.46	0.52	0.7	0.5	0.05
Iso-leucine	1.0	*	1.0	1.0	1.3	0.00
Leucine	2.8	*	2.6	2.4	3.0	0.04
Phenylalanine	1.2	*	1.4	1.4	1.5	0.05
Hydroxylysine	0.64	0.71	0.84	1.5	1.1	0.11
Lysine	2.4	2.2	2.3	2.0	2.1	0.17
1-Methylhistidine	—	—	—	—	0.3	0.07
Histidine	0.54	0.43	0.53	0.4	0.3	0.19
Arginine	4.4	5.0	4.7	5.2	5.6	0.17

\*Total of leucine, iso-leucine and phenylalanine is 6.4.

comparatively narrow range (2.91–3.35 residues per 100). The presence of two amino acids that have not hitherto been reported in dentine as well as other differences between our study and earlier ones may, amongst other factors, be the result of different methods. The more efficient buffer system and the modern lithium columns that we used facilitate the separation of isomers and increase the resolution of the various amino acids. The reported differences in the amino acid content of human dentine may also reflect dietary and other regional factors that may influence the formation of dental hard tissues.

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**9** The Effect of Modern Dentine Bonding Systems on Human Dentine. F. A. DE WET\* and M. R. FERREIRA, Faculty of Dentistry, University of Pretoria, Pretoria, South Africa.

Most modern dentine bonding systems contain primers or cleansers which are used to remove or alter the smear layer or dentine before resin application. The purpose of this study was to assess the effect of four modern dentine bonding systems (DBS), three with primers/cleansers and one without, on the appearance of human dentine.

Denthesive Bond (D, Kulzer), Pertac Universal Bond (P, E.S.P.E.), Prisma Universal Bond 3 (PUB3, Dentsply) and Syntac (S, Vivadent) were included in the study. The crowns of 80 sound, human, molar teeth were imbedded in rings, leaving the occlusal surfaces projecting. These surfaces were ground wet on 220 grit SiC paper to expose superficial dentine. Fifteen dentine surfaces were then treated with each of the four DBS. Cylinders of matching composites were thereafter bonded to these surfaces, using a rubber split mould. After 24 hours storage in water the specimens were stressed to failure using a shear load in an Instron. Fracture sites were evaluated in a SEM. Five abraded dentine surfaces were treated with the respective primer/cleanser of each of 3 DBS. Dentine treated with the D and S cleanser/primer, and debonded specimens of D and S, displayed open dentinal tubules. Subsequent application of an adhesive closed almost all the tubules. Pieces of fractured dentine were attached to some composite stubs, showing resin tags clearly penetrating deep into, and even through the dentine fragments. It can be concluded that only 2 of the 4 DBS tested opened the dentinal tubuli.

**11** Film Thickness Evaluation - Implementing the BENCOR MULTI-T System. C.H. DRIESSEN\*, F.A. DE WET and W.J.C. COETZEE. Faculty of Dentistry, University of Pretoria, South Africa.

Various techniques have been described for the determination of film thickness of dental cements. The purpose of this study was to assess the effectivity of the BENCOR MULTI-T system, and to explore the effect of load variation on the film thickness data. Four luting cements were included in this study i.e. X-R (IONOMER (X)), a glass ionomer, Mrege-FLC (M), a dual cure resin; UNITY (U), a self curing resin; and POLY-F Plus (P), a polycarboxylate. The BENCOR MULTI-T device (Driessen, 1990) was used for testing. The system is based upon direct force application derived from any calibratable source through its active rod onto the specimen material placed between two 22mmØ glass discs. The latter is covered with teflon discs for the purpose of: (1) simulating oral temperature, (2) transferring visible light (if needed) for the curing of a VLC material and (3) protecting the 150µm glass discs from direct metal impact. Measuring of the specimens was done by electronic digital calipers with control by reflex microscope data and SEM image observation. Ten samples of each product were tested using 10Kg and 15Kg forces in order to assess the effect of different loads on the film thickness. All data were statistically analysed. It was found that the technique enabled operators to measure film thickness of dental cements accurately, easily, fast and with only small variations. Statistical analyses showed a significant difference ( $p < 0.05$ ) between application of 10Kg and 15Kg load of (X) [25,76;19,60µm] and (U) [28,60;14,20µm] but not for (M) [31,14;30,80µm] and (P) [38,00;34,00µm]. It can be concluded that the BENCOR MULTI-T system is able to easily assess the film thickness of dental cements, and that a 15Kg load reduces film thickness compared to the 10Kg load.

**13** Effect of Respiratory Acidosis on Faecal Fluoride Excretion in Rats. S. D. JANSE VAN RENSBURG\* and C.A. VAN DER MERWE. University of Pretoria, and Medical Research Council, Pretoria, South Africa.

Respiratory acidosis is characterised by a primarily increased  $PCO_2$  with a compensated increased  $[HCO_3^-]$  which may affect the pH of blood. As the permeation of fluoride (F) through apatite is dependent on the pH, as well as F concentration, respiratory acidosis could have an effect on faecal F excretion, and therefore on the F balance of the body. Sixteen young adult female Sprague-Dawley rats were used in this study. They were divided into a control group (Group A); receiving a normal atmospheric gas mixture and an experimental group (Group B); subjected to an atmospheric  $CO_2$  content of 7%. All the rats were fed a low F diet (<1ppm) and received water with a fluoride content (FF) of 20ppm ad lib for 7 weeks. Water and food consumption were monitored daily. F intake/rat/day via water and food was calculated from the data. After sedation, blood was collected anaerobically in heparinised syringes from the descending aorta. Faeces were collected from the large intestine. The FF of the faeces was determined potentiometrically after HMDS diffusion. Blood gas analysis were done using the ABL blood gas analyser. The data were subjected to the Mann-Whitney procedure to detect differences between the groups, and to multiple regression analysis to explain the variation in the FF of the faeces. Although the F intake via the water differed significantly ( $p < 0.05$ ) between the groups, there was no significant difference between the F intake via food ( $p = 0.197$ ) or the total F intake ( $p = 0.071$ ). There was a significant difference ( $p < 0.05$ ) in the FF of the faeces (A:  $\bar{x} = 148,33$  ppm; S:  $\bar{x} = 213,20$  ppm) between the groups. Taking the FF of faeces as the dependent variable, 68,89% of the variation in the FF of faeces in the experimental group could be explained by the combination of the independent variables  $H_2O$ , or  $PCO_2$ , and total F intake. A 48,92% association between the variables was found in the control group. Respiratory acidosis enhanced faecal excretion of fluoride in rats. The higher FF of the faeces in the experimental group could possibly be due to net secretion of fluoride into the gut lumen. This project was partially supported by a grant from Afrrox Pty Ltd.

**15** Enamel Surface Roughness after  $CO_2$  Laser Radiation, in vitro Assessment. S.H. PAN\*, C. BAKER, J. DE VRIES, P.J. BECKER and S.S. MASHELE. Dept. of Operative Dentistry, Faculty of Dentistry, MEDUNSA.

Recently, a  $CO_2$  laser has been used successfully to enhance dental bonding. Knowledge of surface roughness is essential to reduce bond failures. The objectives were to: 1) determine the surface roughness after conventional drilling with/without laser radiation and with/without acid etching using the Bendix Profilometer, 2) examine under Scanning Electron Microscope (SEM) the ultrastructure of the enamel surface after  $CO_2$  laser radiation. Thirty-two human maxillary central incisor teeth were selected and stored in 10% buffered formalin. Conventional drilling and combined treatment with laser radiation were randomised on the labial enamel surface in the vertical dimension of 3 x 5mm. Laser radiation was set at a repeat pulse energy intensity of below 3W for a period of 10 seconds. Acid etching was performed on half of the specimens and the surface roughness was measured with the Bendix Profilometer. The SEM assessment was also noted. The experiment was designed as a randomised block but test results showed that roughness caused by laser produced the maximum measurable roughness of 10 micron for each sample point. The analysis then focused on conventional high/low speed drilling and no significant difference was found between acid etching techniques, ( $p = 0,4270$ ) or between drilling speeds ( $p = 0,7355$ ). Compared to laser the roughness after conventional drilling was clinically less.

An improved understanding of roughness caused by  $CO_2$  laser radiation on enamel surfaces may lead to possible clinical application in aesthetic restorative dentistry.

**10** Evaluation of the Effectiveness of an Oral Health Preventative Programme. S. Dhansay\*, R. Lalloo, A. Bawn, M.H. Moola. University of the Western Cape

This study was designed to measure the effectiveness of an oral health preventative programme based in a school population in the Cape Peninsula. The objective of the study was to measure the difference between schoolchildren who were exposed to the programme and children who were not.

Three experimental (programme) and two control schools were selected for the study. A total of 110 children in the experimental group and 102 in the control group were examined (Total = 212) in the age group 11-12 years. The examiners were calibrated for reproducibility using WHO (1986) criteria for dental caries. The results of the study showed a mean DMF(S) of 7.8 (+3.1) for the experimental schools and 24.5 (+8) for the control schools. 84% of the experimental group and 60% of control group were caries free. 98% of the experimental group and 55% of experimental group had an X component of 0.42% of the control group; 8% of experimental group had at least one 1st molar tooth decayed. These results clearly showed that the programme is successful in a community based school dental service. This project is supported by an MRC grant.

**12** Cross-infection Risks Associated with High-speed Dental Handpieces. C.H.J. HAUMAN\*, Department of Oral Pathology, Faculty of Dentistry, University of Stellenbosch, Tygerberg, South Africa

Dental handpieces are particularly prone to contamination with patient material, which can then be transmitted to the next patient. The common approach of disinfecting handpieces by external chemical wiping in combination with flushing may pose unacceptably high risks to those individuals treated soon after infected patients.

The aim of this study was to evaluate the efficacy of chemical disinfection of high-speed handpieces. Autoclaved high-speed handpieces were contaminated with an overnight culture of *Staphylococcus aureus* and dried in a hot air oven for 90 minutes. The outer surfaces of equal numbers of these handpieces were wiped with 70% alcohol, alcohol-in-hibitane and Asepsys (iodophor). The front, back and sides of the heads of these handpieces were pressed onto the surface of blood agar plates. In addition, contaminated handpieces were attached to the dental unit and water was flushed through the handpiece onto the surface of blood agar plates for 2 seconds. Handpieces used in the Tygerberg Dental Hospital were tested in a similar way after routine lubrication and alcohol swabbing. Residual contamination of handpieces after flushing for specific periods of time were also tested. After overnight incubation, growth was recorded. Although the numbers were reduced, *S. aureus* could still be cultured from the outer surfaces of artificially contaminated handpieces after wiping with all three disinfectants. Handpieces used in the clinic yielded virtually no growth from the external surfaces after routine cleansing. Confluent or 2+ growth was obtained with samples from the interior surfaces of both artificially contaminated handpieces and handpieces from the clinic. *S. aureus* was still present on the internal surfaces of artificially contaminated handpieces after flushing for 5 minutes. Sterilisation of both the internal and external surfaces of handpieces is necessary to exclude the risk of cross-infection in the dental surgery.

**14** Inorganic Contents of Opaque and Translucent Radicular Dentine. F.S. NKHUMELANI\*, E.J. RAUBENHEIMER, W.F.P. VAN HEERDEN, M.L. TURNER and M.J. DREYER. Depts. Oral Pathology and Chemical Pathology, MEDUNSA, P.O. Medunsa.

This study was undertaken to compare the calcium, magnesium, phosphorus, zinc and fluoride contents of opaque and translucent radicular dentine. Twelve mandibular incisors were utilized. The crowns and cementum were removed using a dental bur. The specimens were then hydrolysed individually in 1M perchloric acid. Calcium, zinc and magnesium were determined utilising the ammonium phosphomolybdate calorimetric method and fluoride by ion selective electrode method. The mean values (mg/g) of opaque and translucent dentine respectively were:

Calcium (Ca)	244.65	(SD ± 3.60)	and	244.17	(SD ± 4.10);
Magnesium (Mg)	8.25	(SD ± 0.42)	and	6.97	(SD ± 0.69);
Fluoride (F)	0.22	(SD ± 0.01)	and	0.27	(SD ± 0.02);
Phosphorus (P)	127.70	(SD ± 2.43)	and	125.00	(SD ± 2.33);
Zinc (Zn)	0.14	(SD ± 0.01)	and	0.20	(SD ± 0.03)

The Mann Whitney test showed that there was a significant difference between the Mg, F and Zn contents of opaque and translucent dentine ( $p < 0.05$ ). Our findings do not support those of Moore and Leaver (1974) who found that only Calcium values were significantly lower in translucent dentine.

**16** Relation Between Blood, Molar, Cortical Bone and Trabecular Bone Lead Levels of Rats. R.J. ROSSOUW\* and S.R. GROBLER, Faculty of Dentistry, University of Stellenbosch, Tygerberg, South Africa

Bone is the major reservoir of body lead stores. Loosely and deeply bound lead compartments in bone provide short- and long-lived sources of this toxic element to blood and soft tissues.

Several groups of inbred BD-IX rats were continuously exposed to nebulized aerosols of lead for different time periods. Furthermore, the effect of different post-exposed periods on the lead concentrations were also investigated. The lead concentrations were determined in blood, molars, tail vertebra and iliac crest of the rat. The graphite furnace atomic absorption spectrophotometer was used.

The blood, molar, tail and iliac crest lead levels differed significantly ( $p < 0.05$ ) among the exposed groups. However, only the blood and iliac crest lead concentrations differed significantly ( $p < 0.05$ ) in the post-exposure periods.

It is concluded that lead is absorbed in the apatite crystal of different kinds of bone. Furthermore, cortical bone is identified as long term storage reservoirs of lead in the body.

This study was supported by the MRC.

# Giant ossifying fibroma: a clinicopathologic study of 8 tumors

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Clinical, radiographic and microscopic features of 8 ossifying fibromas diagnosed in 7 patients and measuring more than 8 cm in greatest diameter, were reviewed. The tumors occurred in both juvenile and middle aged patients and all lesions in women involved the maxilla. The abundance of fibrous connective tissue and resorption of mineralized deposits are indicative of altered cellular differentiation and proliferative activities in large ossifying fibromas. Focal areas of aneurysmal bone cyst formation were identified in the majority of lesions.

Key words: fibro-osseous lesion; giant ossifying fibroma; juvenile aggressive ossifying fibroma.

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Ossifying fibromas are generally regarded as slow growing and well circumscribed jaw tumors which contain foci of trabecular and spherical calcifications resembling bone and cementum respectively (1). They are reported to be more common in blacks, occur frequently in women and the majority of lesions involve the mandible (1, 2).

Although no convincing definition of giant ossifying fibromas are to be found in the literature, these neoplasms were reported by various authors as large tumorous fibro-cemento-osseous proliferations (3-7). Unfortunately, many of the reported cases are not documented satisfactorily. HAMNER *et al.* (1968) however, arbitrarily defined giant lesions as those exceeding 2 × 2 cm in size or involving the space occupied by two or more teeth.

There is no published series of giant ossifying fibromas in the literature. Therefore, this study was undertaken to determine the clinical and radiographic appearances and the microscopic features of the large ossifying fibromas diagnosed by the Department of Oral Pathology at the Medical University of Southern Africa (Medunsa) over a 6-yr period.

## Material and methods

All the cases diagnosed as ossifying fibroma over the last 6 yr were retrieved from the files of the Department of

Oral Pathology, Medunsa. Most patients seen at the hospitals served by the department are black and of rural origin. The pathology reports were reviewed and all lesions with a longitudinal diameter of 8 cm or more as measured on the excision specimen, were included in this study. Radiographs were available in all the selected cases. Histologic evaluation was done by means of light microscopy. Three specimens (Cases 1, 3, 4) were bivalved in the longitudinal diameter, and a 5 mm slice of the entire cut surface was obtained by means of a band-saw. The slice was then radiographed and blocked into multiple squares, each of which was numbered on a scheme corresponding to the radiograph and processed for routine light microscopy. Representative histologic sections were available in the remaining cases.

## Results

Seven patients from a total of 30 cases of ossifying fibroma were found to have tumors larger than 8 cm in greatest diameter (Fig. 1). The age, sex, site and size of the tumors are indicated in Table 1. Case 5 presented with a mandibular and a maxillary tumor, both exceeding 8 cm. Signs and symptoms varied, the most common of which was swelling. At the age of 2 yr, Case 1 presented with a mandibular tumor 4 cm in diameter. Biopsy showed a benign fibro-osseous proliferation and due to par-

enteral refusal the lesion was followed over a period of 7 yr during which it became less radiopaque and tripled in diameter. The duration of the other lesions could not be determined reliably. Although the post-operative follow-up is in some cases as short as one year, none of the lesions have recurred.

All tumors involved the tooth-bearing areas of the jaws and were well demarcated with scattered foci of radiopacities (Fig. 2). Root resorption were present in three cases.

Case 1 was treated with hemimandibulectomy. Enucleation of the tumor was done in the other cases. No cortical perforation was present, only expansion in all directions. On cut surface, the tumors had a gray-white color with a gritty consistency. Cystic spaces representing aneurysmal bone cyst changes and measuring up to 1 cm in diameter could be seen focally in six tumors.

The histologic features correlated with the radiographic appearance of the corresponding area. The inconspicuous radiodense areas consisted of woven trabecular bone, although a few lamellar bony trabeculae and psammomatous calcifications were also found. Active resorption of the trabeculae with accumulation of osteoclast type giant cells were evident in all tumors (Fig. 3). Vascularity was more prominent in the areas of resorption and the fibrous component adjacent to these areas were cellular.

The radiolucent zones consisted



Fig. 1. A, clinical appearance of Case 3. B, clinical appearance of Case 4.

mainly of fibrous tissue. The stroma varied from mature collagen to tissue with a cellular storiform pattern (Fig. 4). Small amounts of mineralized tissue, mainly of a psammomatous cementum-like nature were present in the fibrous tissue. The aneurysmal bone cyst changes were found in areas where the fibrous tissue had a loose, edematous structure.

### Discussion

The sizes of the eight tumors described surpass that of all giant ossifying fibro-

mas reported in the literature. HAMNER *et al.* (3), defining 'giant' lesions as those exceeding  $2 \times 2$  cm in diameter, found 17% of their cases of cemento-ossifying fibromas to have reached these dimensions. Seven (or 23%) of our collection of ossifying fibromas had a diameter of more than 8 cm. If the criteria of Hamner *et al.* (3) had to be applied to our collection of ossifying fibromas, almost all of the 30 cases diagnosed in our department over the last 6 yr will be regarded as 'giant'. The large dimensions of our tumors is related to the rural character of the populations served where proper diagnosis and treatment is often delayed through tribal customs.

The age range of our patients was 7–57 yr with an age peak in the first and fifth decades, a distribution corresponding to that generally reported for ossifying fibromas (1). The occurrence of large ossifying fibromas in young children is of particular interest as it is believed that these tumors require many years of growth to attain large dimensions (2). One of our cases, diagnosed at 2 yr of age, showed an in-

crease of 8 cm in diameter over a follow-up period of 7 yr. As far as we can ascertain, this represents the youngest age at which an ossifying fibroma had been diagnosed. Five of the eight tumors and all lesions in women involved the maxilla. This is in contrast to the generally held view that ossifying fibromas occur more frequently in the mandible (2).

Radiographically, the large ossifying fibromas in our study contain relatively less mineralized tissue than smaller lesions. This finding is substantiated by the microscopic appearance of the giant lesions where the balance of cellular activity favor fibrous tissue formation and bone resorption at the expense of new bone formation. Although the majority of our lesions showed foci of aneurysmal bone cyst formation, STRUTHERS & SHEAR (8) found this change to occur in only 4% of their ossifying fibromas and Eversole *et al.* (9) noted aneurysmal bone cyst features in three of their 64 cases. The high prevalence of aneurysmal bone cyst formation in our lesions is probably due to the prominent fibrous com-

Table 1. Clinical data of the seven patients.

Case No	Age	Gender	Site	Size
1	9	M	Mandible	12 cm
2	7	W	Maxilla	8 cm
3	13	W	Maxilla	13 cm
4	46	W	Maxilla	15 cm
5	40	M	Mandible	8 cm
			Maxilla	10 cm
6	49	M	Mandible	10 cm
7	57	W	Maxilla	10 cm

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ponent which contains more loose edematous areas than is found in smaller ossifying fibromas. This feature is not responsible for the giant dimensions, as the foci of aneurysmal bone cyst change are limited and the cystic spaces are of relative small size.

HAMNER *et al.* (3) stated that ossifying fibromas containing cementum are larger and more aggressive than pure ossifying or cementifying lesions. HALL *et al.* (10) consider mixed cementifying and ossifying fibromas as a potentially aggressive variant of ossifying fibroma. WALDRON & GIANANTI (11) however stated that a separation into cementifying and ossifying types is artificial as they could find no difference in the behaviour of tumors with these histologic designations. Cementum-like as well as osseous deposits were present in all tumors in our series and we believe that if representative tissues of ossifying fibromas are taken for microscopic examination, most tumors will be found to be of a mixed nature.

Our study furthermore suggests that ossifying fibromas with a gigantiform growth potential are characterized by the appearance of large fibrous areas which are represented radiographically by less radiodense areas. This is in contrast to the normal progression of these lesions where the islands of mineralizations are reported to increase in size and coalesce resulting in a more radiopaque lesion (2).

The microscopic appearance of the giant lesions does not resemble that of juvenile aggressive ossifying fibromas. The criteria defined by WALDRON (12) for the diagnosis of juvenile aggressive ossifying fibroma include a cellular vascular stroma with varying amounts of giant cells and little collagen production. Osteoid lined by osteoblasts are usually present. These lesions furthermore appear most often in young patients – predominantly younger than 20 yr and almost always below 40 yr of age (13). None of the lesions in the three young patients in our series can be classified as the aggressive variant because of the prominent fibrous tissue and collagen component and the scarcity of osteoid formation. This however does not exclude the possibility that at some earlier stage our lesions may have had the microscopic features of juvenile aggressive ossifying fibromas.

This study suggests that the shift in cellular activity from osteoblastic in small ossifying fibromas to fibroblastic in the giant lesions represents a phe-

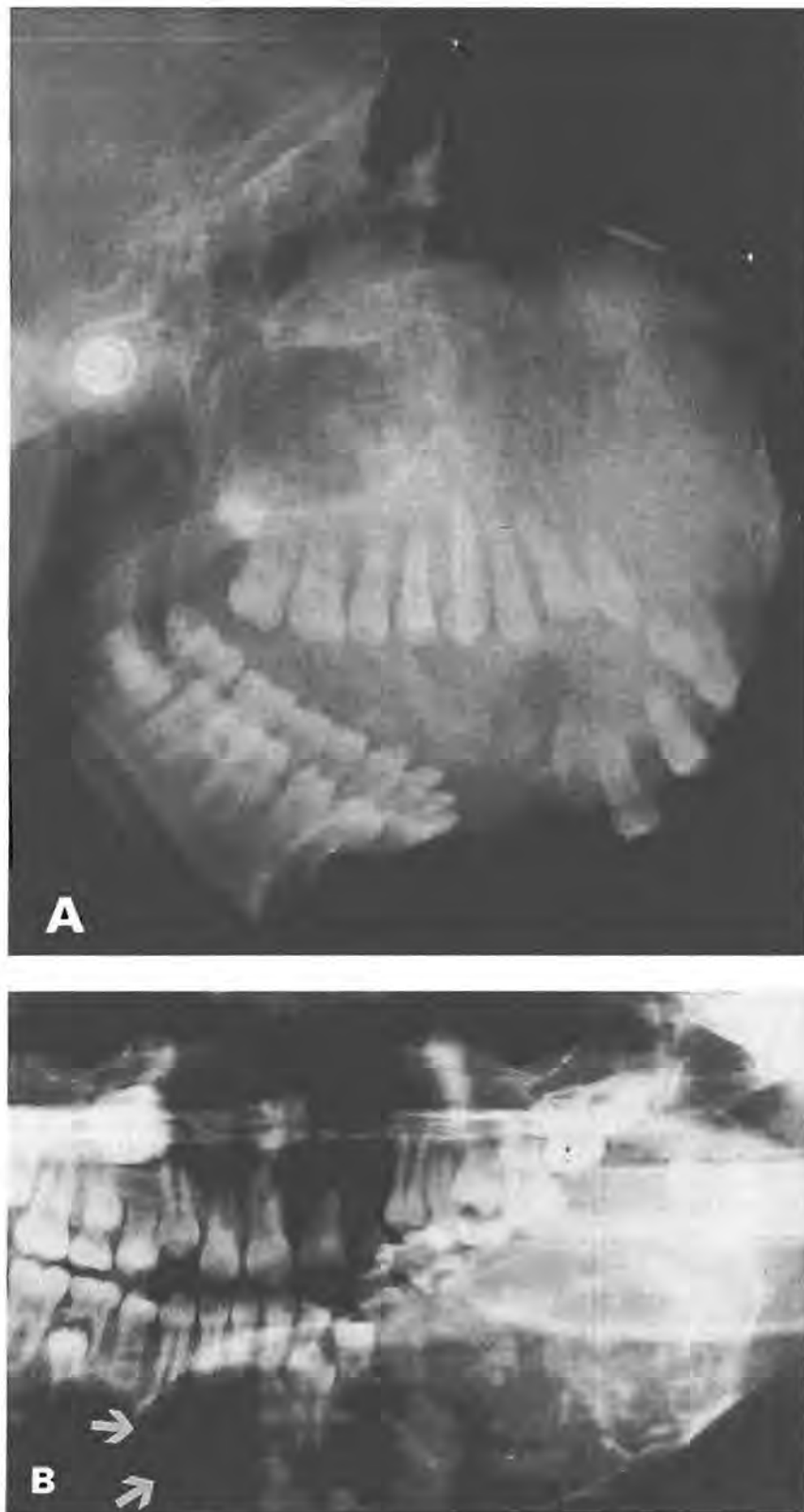


Fig. 2. A, radiograph of Case 3 shows a well demarcated lesion with smokescreen appearance and irregular radiolucent areas. B, Prominent lytic areas are present in Case 1 (arrows).

nomenon associated with gigantiform tumor enlargement.

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Fig. 3. Interface between dense fibrous zone and area of bone resorption. Note bony trabeculae (asterisks) and osteoclasts (arrows) in loose fibrous tissue. H&E,  $\times 40$ .

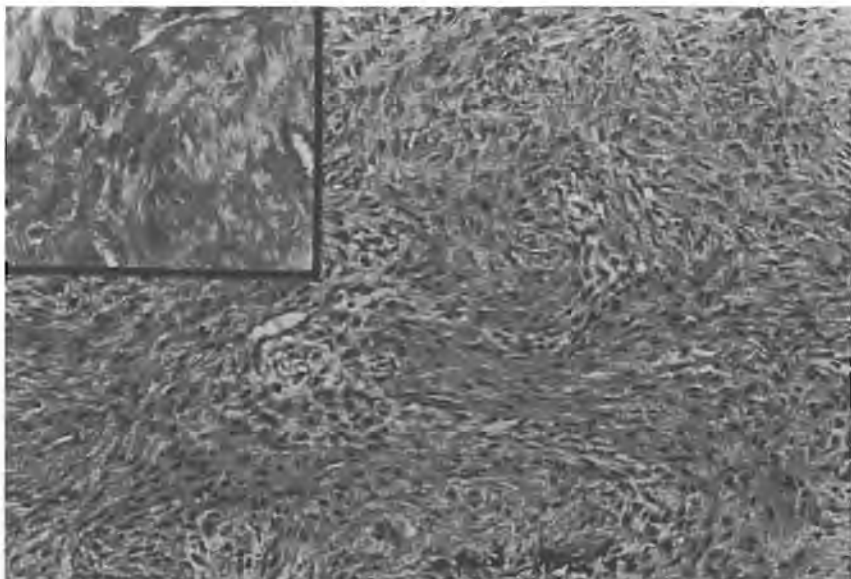


Fig. 4. Cellular storiform growth pattern. H&E,  $\times 100$ . Inset: mature collagen. H&E,  $\times 150$ .

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# Adenomatoid odontogenic tumour: a report of two large lesions

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Adenomatoid odontogenic tumours with a diameter of more than 4 cm are uncommon. Two cases, both measuring in excess of 7 cm, are described and the differential diagnosis discussed. The progressive growth and cortical perforation in these two cases support the view that it is a benign neoplasm rather than a hamartoma.

*Keywords: Odontogenic tumours; maxilla*

The adenomatoid odontogenic tumour (AOT) is a rare, benign odontogenic tumour of which approximately 170 verifiable cases have been reported in the English literature. It occurs most often in young females and commonly involves the anterior maxilla. Although many AOTs are detected during routine radiographic examination, patients may present with a gradually enlarging, painless swelling which can lead to facial asymmetry. Radiographs generally show a clearly demarcated, radiolucent lesion surrounding an unerupted tooth, usually a maxillary canine<sup>1,2</sup>. Radiopaque foci frequently occur in the tumour. The size of an AOT varies between 1.5 and 3.0 cm but some as large as 9.0 cm have been reported<sup>3</sup>. It is usually diagnosed radiographically as a follicular, lateral periodontal<sup>1</sup>, residual<sup>4</sup> or 'globulo-maxillary' cyst<sup>2,5</sup>. If calcification is present, the differential diagnosis should also include calcifying odontogenic cyst, central ossifying fibroma, calcifying epithelial odontogenic tumour and ameloblastic fibro-odontome. Microscopically, AOTs are characterized by a well-defined fibrous capsule surrounding sheets, strands and nodular masses of epithelial cells which form tube-like structures and rosettes<sup>1</sup>. The purpose of this paper is to report two large AOTs with diameters of more than 7 cm.

## Case reports

### Case 1

A 12-year-old black female presented complaining of a maxillary swelling obstructing her nose; the duration of the swelling was unknown. On examination, a maxillary tumour, extending from the lower border of the right eye and crossing the midline of the face, was present. The size of the lesion interfered with lip closure. The nose was deviated, the nasal passage obstructed and on palpation the bony cortex was perforated, resulting in fluctuation. The skin overlying the lesion had three parallel scars (each 4 cm long). Intra-oral examination showed mobile and displaced maxillary permanent incisors and a primary canine, and bulging of the right palatal shelf and buccal plate

(Figure 1). Radiographs revealed a well-circumscribed unilocular radiolucency containing the crown of the unerupted permanent canine (Figures 2, 3). A clinical diagnosis of follicular cyst was made and the lesion was enucleated through an intra-oral approach. The specimen submitted for pathological examination consisted of a cystic lesion measuring 12 × 10 × 10 cm which contained a normal maxillary canine. The lining of the cyst contained multiple nodules measuring up to 5 mm in diameter. Microscopic examination showed an epithelial lining containing nodular masses of odontogenic epithelial cells forming rosettes and pseudoglandular spaces. A diagnosis of AOT was made.

### Case 2

A 9-year-old black female presented with a complaint of swelling in the right maxilla obstructing her nose. The lesion was painless and had been present for 3 years. On examination, there was a 9 × 8 cm swelling in the right maxilla, which had elevated the right ala (Figure 4). The maxillary permanent central incisors,

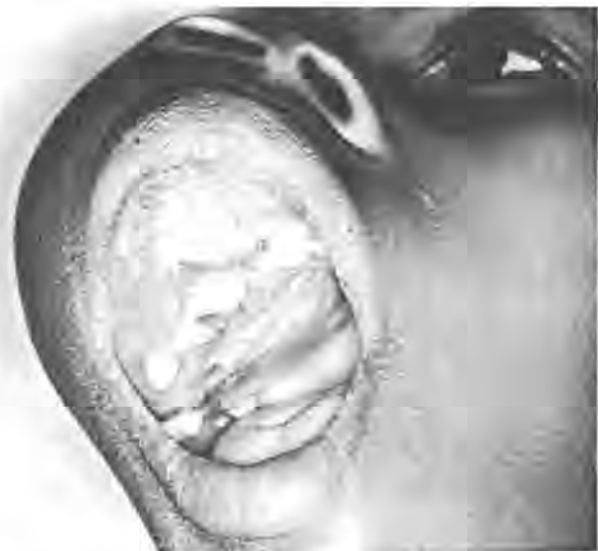


Figure 1 Case 1. Intraoral view of the tumour



Figure 2 Case 1. Lateral cephalometric radiograph. Note the displacement of the permanent maxillary canine (arrow)

primary canines and molars were erupted and the palatal shelf and buccal plate were expanded by a firm swelling. Radiographically, there was a well-circumscribed radiolucent lesion, surrounding the crown and neck of an unerupted maxillary canine; the developing maxillary premolars were dilacerated and the second incisor displaced and impacted (Figure 5). In the absence of calcification in the wall of the lesion, a clinical diagnosis of a follicular cyst was made. The lesion was enucleated and an opened cyst, 8cm in diameter, surrounding the crown and neck of an unerupted maxillary canine and containing mural granules, was submitted for pathological examination. Microscopically, the cyst wall consisted of abundant connective tissue lined by thin and inactive odontogenic epithelium which surrounded nodular masses of epithelial cells exhibiting rosettes and pseudoglandular structures. A diagnosis of AOT was made.

**Discussion**

As far as can be ascertained, Case 1 represents the largest AOT reported in the English literature.



Figure 3 Case 1. Panoramic radiograph. Note the well circumscribed, expansile lesion in the right maxilla with displacement of the roots of involved teeth as well as the canine



Figure 4 Case 2. Frontal photograph

Another large tumour had a diameter of 9cm and formed part of a series of 13 cases occurring in Nigerian patients<sup>7</sup>. The large size of our two cases could be related to their more rapid growth in younger patients, certainly, the average age is higher in previous reports. However, the size may also result from a delay in seeking proper dental treatment. This view is supported by the presence of linear scars on the skin overlying the tumour in Case 1, an indication of regular visits to tribal medicine men before seeking hospital treatment.

The histogenesis of the AOT is unknown but the possibilities range from the dental lamina to reduced enamel epithelium<sup>6-8</sup>. One investigator<sup>9</sup> suggested that the epithelial rests of Malassez at the apex of deciduous teeth is the progenitor tissue. His argument is based, in



Figure 5 Case 2. Panoramic radiograph. Note the dilacerated first and second permanent maxillary premolars (arrows).



part, on the fact that AOTs have never occurred in association with impacted deciduous teeth nor in areas not preceded by deciduous teeth. The existence of those lesions not associated with an unerupted tooth and therefore not arising from the reduced enamel epithelium, may be explained on this basis.

Courtney and Kerr<sup>10</sup> from a study of 20 AOTs, as well as others<sup>11-13</sup>, believe the lesion to be an hamartoma rather than a benign neoplasm. However, hamartomas have a limited growth potential and progressively differentiate into more mature tissue with ageing<sup>14</sup>. Our cases do not support a limited growth potential as postulated by Saito *et al.*<sup>4</sup> nor did they exhibit maturation into more differentiated dental tissues. We therefore believe the lesion to be a benign neoplasm. The growth potential of AOTs is supported by Ajagbe *et al.* and others<sup>15,16</sup> and earlier detection is likely to be the reason for the small size of most cases reported in the literature.

Radiographically both of our cases resembled a follicular cyst, the most common lesion to consider in the differential diagnosis of AOT. The well demarcated radiolucency associated with an AOT is reported to extend more apically on the root of the associated unerupted tooth than in the case of a follicular cyst<sup>17</sup>. Another feature that could be helpful in distinguishing between these two lesions is the virtual absence of root resorption in AOTs<sup>18</sup>. The dilaceration of the permanent premolars in Case 2 is most likely the result of pressure exerted by the enlarging tumour on the roots of the developing teeth.

Nasal obstruction is a common complaint in patients with maxillary AOTs measuring 5cm or more in diameter<sup>3,19</sup>. Furthermore, erosion of bone has been reported in a large AOT and actual perforation has led to it being described as a 'fluctuant mass'<sup>19</sup>. Our Case 1 also exhibited this feature but we do not agree with Poulson and Greer<sup>20</sup> that its presence warrants the exclusion of an AOT and the consideration of a more aggressive tumour in the differential diagnosis.

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# Diffuse peripheral odontogenic fibroma: report of 3 cases

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Weber A, van Heerden WFP, Ligthelm AJ, Raubenheimer EJ: Diffuse peripheral odontogenic fibroma: report of 3 cases. *J Oral Pathol Med* 1992; 21: 82-4.

Since peripheral odontogenic fibroma (POF) is characteristically described as a solitary lesion and no diffuse POF had been reported in the literature, our cases should be considered as extremely unusual. Three diffuse cases of POF are described of which one case was seen in association with ocular and skin lesions. The question arises whether POF should be considered as a true odontogenic tumor rather than a diffuse hamartomatous lesion caused by uncontrolled induction of the gingiva. It is also possible that such lesions could be part of a yet undescribed syndrome.

Key words: hamartoma; mouth, neoplasms; odontogenic fibroma; peripheral odontogenic lesions.

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The odontogenic fibroma is defined by the World Health Organization as a benign odontogenic neoplasm of fibroblastic origin characterized by relatively mature collagenous fibrous tissue and varying amounts of odontogenic epithelium, with the potential to occur in either a central or extraosseous location. The extraosseous counterpart is designated peripheral odontogenic fibroma (POF) (1).

All the POF's described in the literature presented as single, exophytic tumors which frequently prompted a clinical diagnosis of localized gingival hyperplasia. Diffuse involvement of the gingiva has not yet been reported. Three cases with the histologic appearance of POF with diffuse involvement of the gingiva, of which one case was associated with dermatological and ocular abnormalities, are presented.

## Material and methods

### Case 1

An 8-yr-old Black girl presented with diffusely enlarged gingiva in both jaws, causing delayed eruption of the perma-



Fig. 1. Clinical photograph of Case 1 showing diffusely enlarged gingivae in both jaws.

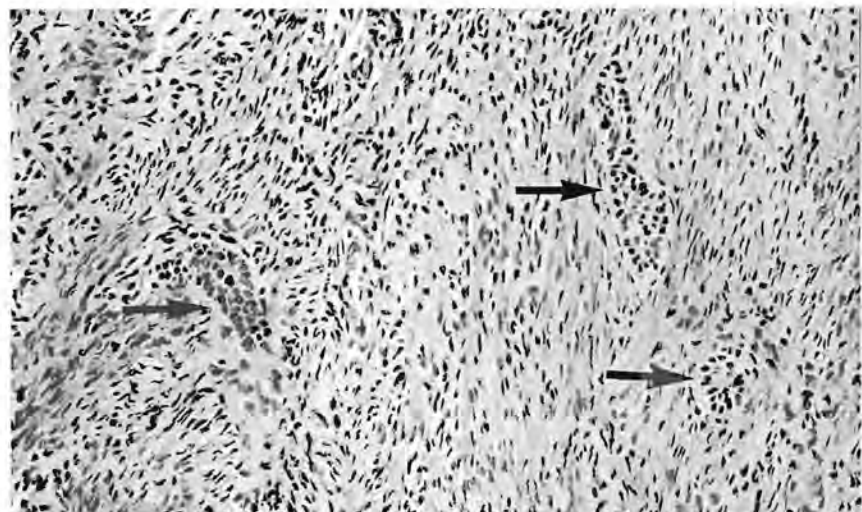


Fig. 2. Histologic appearance of lesion in Case 1 showing cellular fibrous tissue with inactive odontogenic epithelium arranged in nests and strands (arrows).100.

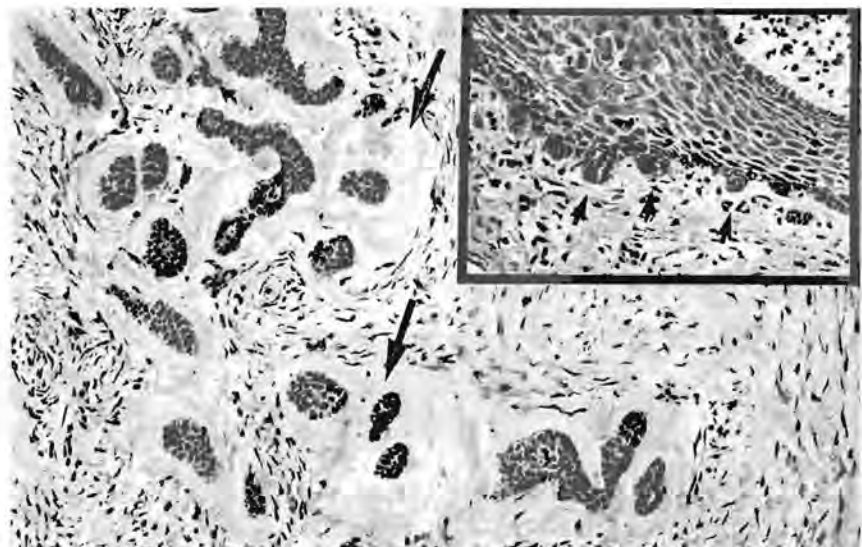


Fig. 3. Microscopic examination of lesion in Case 2 showing strands of odontogenic epithelium with prominent hyalinization (arrows). Inset: budding of overlying epithelium (arrows).100.



Fig. 4. Diffuse nodular gingival hyperplasia (arrows) in Case 3.

dent incisors. Nodules were present in the gingival masses which were firm in consistency (Fig. 1). The duration of the lesions could not be determined and no evidence of a family history was found. Radiographic examination showed no bone involvement or disturbance in tooth development. Gingivectomy of the hyperplastic tissue was done and the tissue sent for histologic examination.

Microscopically, the lesion consisted of cellular fibrous tissue with myxomatous areas. The odontogenic epithelium appeared inactive and was arranged in cell nests and strands (Fig. 2). No hard tissue formation was seen. The overlying epithelium was hyperplastic without evidence of downward proliferation of the rete ridges.

#### Case 2

A 56-yr-old black woman presented with diffuse gingival hyperplasia of both jaws resulting in enlarged alveolar ridges. The duration of the lesions was not known. The mandibular canines were displaced. All teeth were severely afflicted by plaque and calculus deposits. A biopsy of the lesion was performed and oral hygiene procedures implemented.

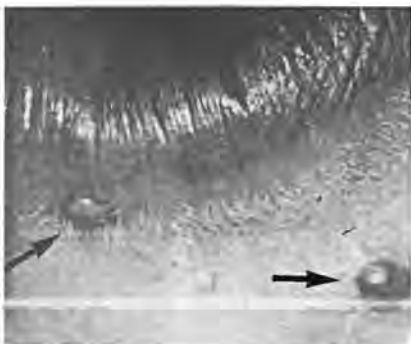


Fig. 5. Clinical photograph of Case 3 showing xanthogranuloma on skin (arrows).

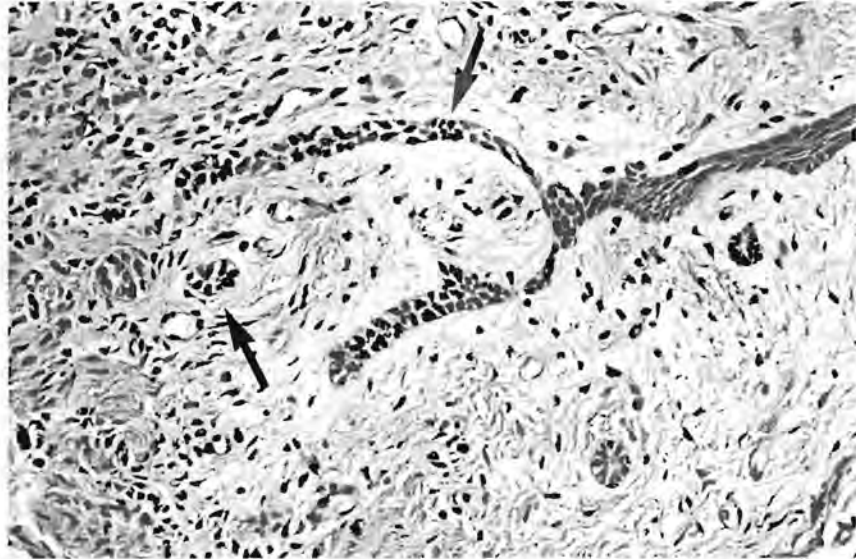


Fig. 6. Histologic appearance of tumor in Case 3 showing islands and strands of odontogenic epithelium (arrows) scattered in connective tissue.100.

Microscopically the gingival enlargement resulted from a proliferation of loose cellular connective tissue with scattered islands of inactive odontogenic epithelium. Hyalinization and calcifications were present in relation to the odontogenic epithelial rests. Budding of the overlying oral epithelium (Fig. 3) and focal areas of chronic inflammation were seen.

#### Case 3

A 3-yr-old white boy presented with diffuse nodular maxillary and mandibular gingival hyperplasia which became evi-

dent soon after birth (Fig. 4). The normal eruption pattern was disturbed but no other abnormalities were found radiographically. The patient also had small nodular skin lesions diagnosed as xanthogranulomas (Fig. 5), as well as corneal opacities. Corneal transplants were done in the eye lesions which could not be diagnosed as any specific pathological entity as yet. The oral lesions were clinically diagnosed as gingival hyperplasia and a biopsy performed.

Microscopically the lesion was composed of cellular fibrous tissue with islands and strands of odontogenic epithelium scattered in the connective

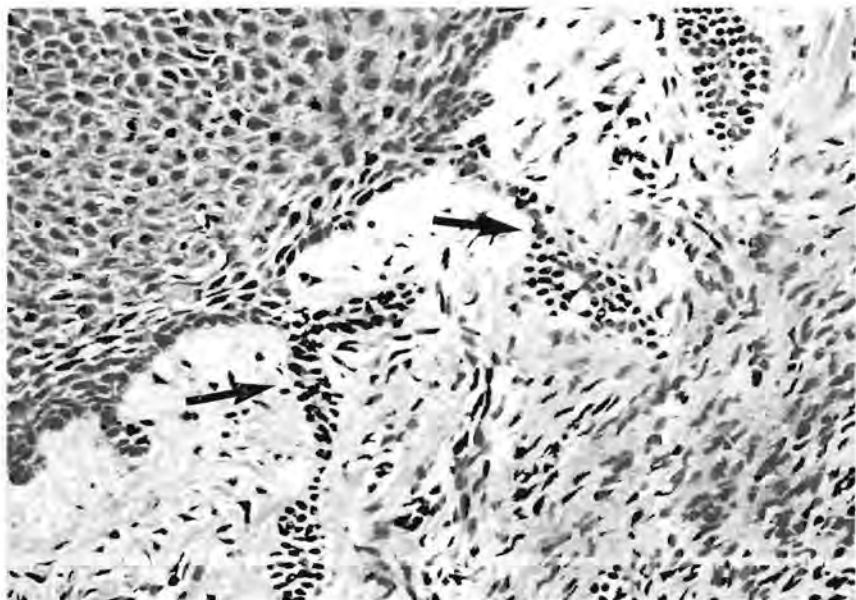


Fig. 7. Overlying epithelium in Case 3 showing downward proliferation into the connective tissue (arrows).200.



tissue (Fig. 6). No mineralized matrix formation was evident and the surface epithelium exhibited mild hyperplasia with downward proliferation (Fig. 7). A diagnosis of POF was suggested and gingivectomy of the hyperplastic tissue was performed. All the tissue submitted exhibited similar microscopic features.

#### Discussion

Not one of the accepted cases of POF in the literature were described as a diffuse gingival lesion. Furthermore, POF was never before described in relation to any other lesions as was seen in Case 3.

The question arises as to whether

POF is a true neoplasm or whether it should be regarded as an hamartomatous developmental anomaly. The diffuse involvement of the gingiva in our three cases supports the possibility that POF does have an hamartomatous origin rather than being a true benign neoplastic lesion. We agree however, that the distinction between an hamartoma and a benign neoplasm is at best difficult and is differently interpreted.

The authors are of the opinion that POF should be considered as solitary or diffuse hamartomatous lesions which are caused by uncontrolled induction in the gingiva in a local or diffuse manner. Furthermore, the possibility that POF is

an hamartomatous growth, which could be part of a yet undescribed syndrome, cannot be excluded, and should be investigated.

*Acknowledgments* – The authors thank C. S. BEGEMANN for secretarial services.

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# Peripheral dentinogenic ghost cell tumor

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Raubenheimer EJ, van Heerden WFP, Sitzman F, Heymer B: Peripheral dentinogenic ghost cell tumor. *J Oral Pathol Med* 1992; 21: 93-5.

A case of dentinogenic ghost cell tumor, that has originated peripherally in the jaw, is presented and the literature reviewed with particular reference to the origin of the tumor. The total number of central and peripheral cases reported in the English literature is 10 and although mucosal infiltration is common, peripheral origin of the neoplasm could be verified in only 3 cases.

Key words: dentinogenic ghost cell tumors; jaws, neoplasms; odontogenic tumors

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The calcifying odontogenic cyst is a unique jaw lesion described as a distinct entity in 1962 by GORLIN *et al.* (1). In a study of 16 cases by PRAETORIUS *et al.* (2), it became evident that this group of lesions contains two entities, a cyst (Type I) and a neoplasm (Type II), and for the latter the term 'dentinogenic ghost cell tumour' was proposed. The neoplasm occurs predominantly in later life and consists microscopically of ameloblastoma-like odontogenic epithelial proliferations infiltrating the bone and connective tissue. Ghost-cells are present as well as varying amounts of dentinoid the latter being closely associated with odontogenic epithelium.

The purpose of this paper is to present an unique case of a dentinogenic ghost cell tumour originating in an extraosseous location.

The surgical specimen measured 6 × 6 × 4 mm and had a firm consistency with foci of calcifications. Microscopic examination revealed hyperplasia of overlying epithelium and a solid tumor, composed of odontogenic epithelium associated with calcifications in the sub-epithelial connective tissue (Fig. 2). The neoplastic epithelium showed a well defined cuboidal to cylindrical basal cell layer closely associated eosinophilic cells with abrupt keratinization, resembling ghost cells (Fig. 3). Although the epithelial cells displayed nuclear pleomorphism, no mitotic figures were present. In focal areas, stellate reticulum-like differentiation as well as the formation of dental lamina-like structures were observed. Masses of acellular calcified material, resembling dentinoid,

were evident in close association with the epithelium (Fig. 3). The surrounding connective tissue contained strands of inactive epithelium associated with small globular dentinoid deposits. Slight inflammation with vasodilatation and edema was present and a diagnosis of peripheral dentinogenic ghost cell tumor was made. Six year follow-up after removal failed to reveal a recurrence.

## Discussion

Peripheral occurrence of the cystic types of calcifying odontogenic cysts (Type I) is well documented in the English literature (1, 3). This may result from cortical bone perforation by a central lesion or more rarely, true peripheral origin from gingival epithelial remnants (4).

## Case report

An 82-yr-old man presented in the Department of Dental Surgery and Radiology, University of Ulm, complaining of a slow growing nodule on the mandibular right alveolar ridge. The lesion started 6 yr ago after extraction of the mandibular right canine. On examination the patient was found to be edentulous. A 6 mm broad based polypoid lesion was located on the mandibular right alveolar ridge. Radiographic examination revealed no underlying bone involvement (Fig. 1) and a clinical diagnosis of a peripheral giant cell granuloma was made. During surgical removal, the lesion was found to be located within the alveolar mucosa and the alveolar bone was not involved.



Fig. 1. Panoramic radiographic view showing lack of bony involvement of the mandibular right alveolar ridge.



Fig. 2. Well encapsulated tumor with ghost cell formation (arrows) and dentinoid deposits (asterisks). H&E stain,  $\times 30$ .

Conflicting data on the origin of the solid type calcifying odontogenic cyst (Type II) or dentinogenic ghost cell tumor are present in the literature. A recent review article summarizes the clinico-pathological features of 10 cases published in the English literature. In this article gingival swelling is considered to be the most frequent clinical feature. Radiographically all cases presented as lucencies with poorly defined margins (5). An earlier paper, reviewing 5 cases, proposed that dentinogenic ghost cell tumors usually occur peripherally and on the gingiva (6). Analysis of the original publications, mostly case reports, proves the discrepancy to lie in the interpretation of the clinical descriptions. Both cases reported by PRAETORI-

US (2) are described as being 'extraosseous', despite radiographic signs of bony and dental involvement. Although peripheral involvement is probably implied by the authors, true peripheral origin of these lesions need to be questioned. The first of two cases reported by FEJERSKOV & KROGH (7) is described as an exophytic palatal mass. Unfortunately roentgenograms were not available and central origin of this case can therefore not be excluded. Central dentinogenic ghost cell tumors were also reported by GÜNHAN & SENGÜN (8) - 1 case, - TAJIMA (9) - 1 case - and COLMENERO *et al.* (5) - 1 case -, bringing the total number of central dentinogenic ghost cell tumors in the literature to 7. The 'peripheral odontogenic tumor with

ghost-cell keratinization' reported by VULETIN *et al.* (10) contained no dentinoid deposits and exhibited odontogenic epithelium surrounded by cellular fibroblastic tissue, resembling and primitive dental pulp. On microscopical grounds, this lesion can not be classified as a dentinogenic ghost cell tumor since ghost cells are found in many other odontogenic neoplasms (2).

Peripheral presentation of dentinogenic ghost cell tumors is related to their infiltrative behaviour and although this feature appears to be common, true peripheral origin is not as frequently reported. ABRAHAMS & HOWELL (11) describe a dentinogenic ghost cell tumor located entirely extraosseous and palatal to a maxillary cuspid. Peripheral dentinogenic ghost cell tumors involving only the gingiva or alveolar mucosa, with radiographic support, were also reported in the lingual mandibular left premolar region (6) and the anterior part of the maxilla (12). This brings the number of true peripheral tumors to four, including our case.

The average age reported in the literature is 50, the oldest being 72 and the youngest 17 (5). Our patient, with an age of 82 yr, represents the most advanced age at which a dentinogenic ghost cell tumour has been diagnosed. Although the central tumors have a high rate of recurrence after removal (5), long term follow-up of our case and lack of proof of recurrence of any of the other peripheral dentinogenic ghost cell tumors suggests a favourable course for the peripheral type.

*Acknowledgments* - We are indebted to Ms C. S. BEGEMANN for secretarial assistance.

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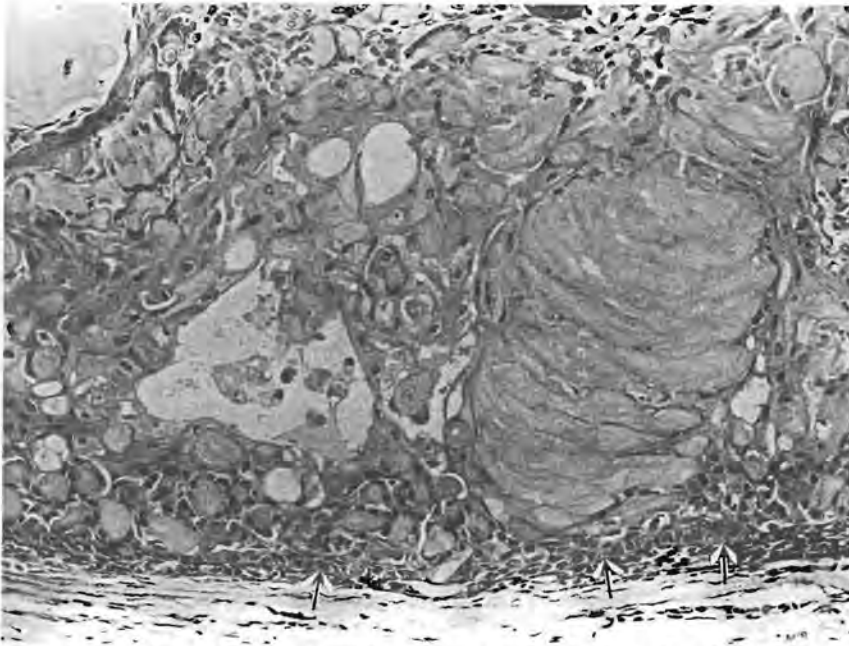


Fig. 3. Cuboidal peripheral basal cell layer (arrows) and adjacent cells exhibiting abrupt keratinization with ghost-cell formation. H&E stain,  $\times 150$ .

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# Infrequent clinicopathological findings in 108 ameloblastomas

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One hundred and eight ameloblastomas diagnosed in a rural black Africa population were analysed for clinicopathologic findings other than those classically described. One patient had a polycystic ameloblastoma adjacent to an ameloblastic fibroma. Two other polycystic ameloblastomas showed aneurysmal bone cyst formation and one mandibular tumour was diagnosed as a keratoameloblastoma. Microscopic changes resembling an adenomatoid odontogenic tumour were present in association with two unicystic ameloblastomas and a HPV18-positive verrucous lesion occurred in the lining of a cystic space of a polycystic ameloblastoma. Two ameloblastomas contained eosinophilic granules in all tumor cells and melanocytes were diffusely present in another. One case exhibited a focus of mucous cell metaplasia. Two polycystic ameloblastomas showed diffuse interstitial ossification. One mandibular tumor was diagnosed as a desmoplastic ameloblastoma and another as an odontoameloblastoma. This study demonstrated that although ameloblastomas are regarded as a fairly homogeneous group of neoplasms, detailed investigations prove clinicopathologic diversity in a significant number of lesions.

Key words: ameloblastoma; infrequent findings; jaw tumors; odontogenic tumors.

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Ameloblastoma is the most common neoplasm affecting the jaws. It is derived from odontogenic epithelium and although located primarily intraosseously, peripherally occurring ameloblastomas involving soft tissue only have occasionally been reported. Two clinicopathologic variants of intraosseous ameloblastomas are commonly recognised. Polycystic ameloblastomas occur mainly in the body and ascending ramus of the mandible and most patients with this type present in the 4th decade of life (1). Unicystic ameloblastomas present on average a decade earlier (2) and are generally associated with a lower post-operative recurrence rate than the polycystic types. They can be divided microscopically into Groups 1, 2 or 3 depending on either the presence of a non-proliferating lining, intraluminal proliferations or mural invasion respectively (2). Ameloblastomas may occur more frequently in black Africans than in other racial groups (3).

Several recently published large series on ameloblastomas make no mention of coincidental and infrequent clinicopathologic findings (2-5) and most information about these is obtained through individual case reports. The secretion of interleukin-1 and a parathyroid hormone-like substance by an ameloblastoma was alleged to be the cause of hypercalcemia in one patient (6). A multilocular lytic mandibular lesion in a patient with hyperparathyroidism was proven microscopically to represent an ameloblastoma associated with a brown tumor of hyperparathyroidism (7). The occurrence of an ameloblastoma in a patient with the basal cell nevus syndrome (8) appears to be a sporadic rather than a regular feature. Other tumors that have been reported to be associated with ameloblastomas include the calcifying odontogenic cyst (9), acinic cell carcinoma and adenolymphoma of salivary gland origin (10), osteogenic sarcoma (11), traumatic neuroma (12) and aneu-

rysmal bone cyst (13). HPV capsid antigen was proven positive with the immunoperoxidase staining technique in 3 out of 10 ameloblastomas in children (14) and mucormycosis infection was reported to have been superimposed on an ameloblastoma in an elderly diabetic woman (15). Stromal desmoplasia in a significant number of ameloblastomas has led to the use of the term 'desmoplastic ameloblastoma' (16) and extensive interstitial bone formation in ameloblastomas has recently been reported in two Japanese patients (17, 18). A case of papilliferous keratoameloblastoma was reported by ALTINI *et al.* (19) and other microscopic rarities include melanocytes between (20), and granular cell change in all tumor cells (21).

The purpose of this study was to determine the spectrum of uncommon clinical and pathological findings in a large sample of ameloblastomas diagnosed in a rural black African population.

### Material and methods

Clinical records, radiographs and hematoxylin and eosin-stained microscopic slides of biopsies and surgical resections of 108 primary intraosseous ameloblastomas were scrutinized for extraordinary and coincidental pathologic features. At least four wax blocks were available in most cases. The following special staining techniques were employed on selected cases: Mucicarmine for epithelial mucins, Masson-Fontana for melanin, Perl's Prussian blue for hemosiderin pigment and the *in situ* hybridization technique for the presence of HPV antigen. All cases were diagnosed and treated in the Medunsa Dental Hospital which serves a black and mainly rural African community.

### Results

The sample consisted of 108 ameloblastomas of which 75 were polycystic and 33 unicystic. All cases originated intraosseously. The sex and age distributions are shown in Table 1. All polycystic and 29 of the unicystic ameloblastomas occurred in the mandible and four unicystic ameloblastomas presented as maxillary swellings. The left mandible was involved in 65 cases, right mandible in 26 and symphysis area in 13 cases. Twelve polycystic and 4 unicystic ameloblastomas perforated the bony cortex and caused soft tissue ulceration. A mandibular ameloblastic fibroma in a 19-year-old woman was adjacent to and continuous with a polycystic ameloblastoma (Fig. 1). Aneurysmal bone cyst changes were identified in the latter patient as well as in another polycystic ameloblastoma in a 20-year-old woman. A polycystic ameloblastoma which involved the anterior mandible of a 13-year-old girl was associated with a compound odontome (Fig. 2).

Microscopically, 47 polycystic ameloblastomas were follicular, 23 plexiform and 5 were of mixed follicular and

Table 1. Sex and age distribution

	N	Sex		Average age (range in years)
		M	F	
Total sample	108	50	58	29.3 (8-84)
Polycystic	75	33	42	35.4 (13-84)
Unicystic	33	17	16	16.5 (8-37)



Fig. 1. A resected specimen showing a polycystic ameloblastoma associated with an ameloblastic fibroma (arrows).



Fig. 2. Panoramic radiograph of the odontoameloblastoma.

plexiform patterns. The stellate reticulum showed no differentiation in 35 cases, 22 cases presented with acanthomatous differentiation, 9 cases with granular cell differentiation, 6 cases with both granular cell and acanthomatous differentiation, and basal cell differentiation was seen in one case. A follicular ameloblastoma showed acanthomatous differentiation and foci of mucous cell metaplasia (Fig. 3). One tumor, which occurred in the mandible of a 57-year-old woman, showed extensive keratinisation and was diagnosed as a keratoameloblastoma. The unicystic ameloblastomas showed mural invasion in 15 cases and intraluminal proliferation in one case. The remaining 17 unicystic ameloblastomas were lined by

non-infiltrative epithelium. In 9 of the latter group, less than 3 blocks were available for microscopic examination. Microscopic changes resembling an adenomatoid odontogenic tumor were presentation the walls of two unicystic ameloblastomas (Fig. 4). HPV type 18 was identified in a verrucous lesion which occurred in a polycystic ameloblastoma and in one case melanocytes were uniformly present between the neoplastic epithelial cells. In two patients aged 15 and 26 years respectively, ameloblastic epithelium contained eosinophilic granules in all tumor cells (Fig. 5). Hemosiderin pigment was identified in the cytoplasm of neoplastic odontogenic epithelial cells next to an area of hemorrhage. A desmoplastic re-



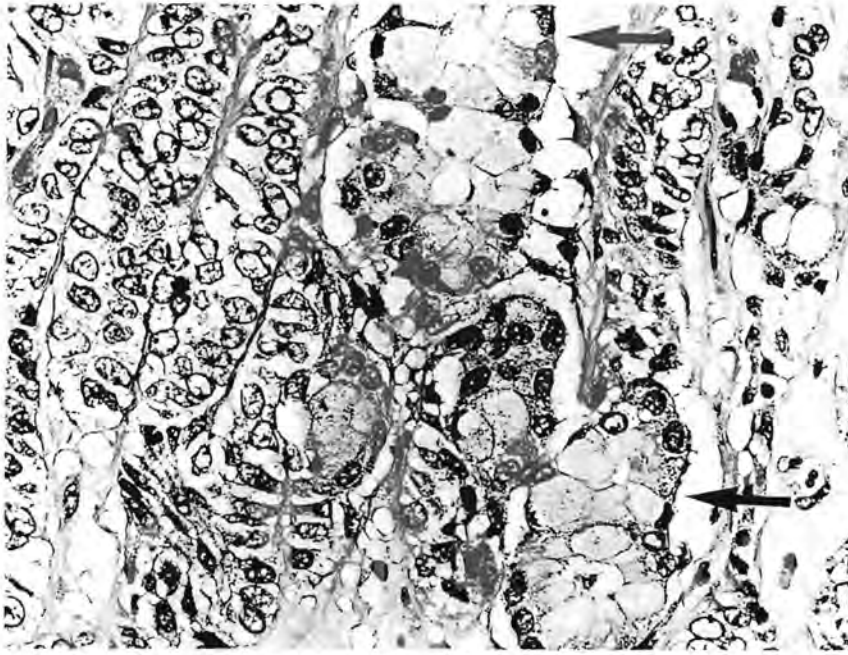


Fig. 3. Mucous-cell metaplasia (arrows) in an ameloblastoma (H&E, X240).

action was a common feature in the extraosseous component of ameloblastomas which perforated the bony cortex. Desmoplasia of the intrabony part of ameloblastomas was variable both between tumors and within the same tumor and depended upon the degree of inflammation. Only one case was diagnosed as a desmoplastic ameloblastoma on the basis of a uniform and mature connective tissue proliferation in the absence of inflammation and which impinged upon the neoplastic epithelial component (Fig. 6). Two polycystic ameloblastomas were associated with diffuse interstitial bony deposits which led to radiographic diagnoses of fibroosseous lesions.

#### Discussion

Large series published on ameloblastomas often make no mention of features other than those classically described and most infrequent findings are re-

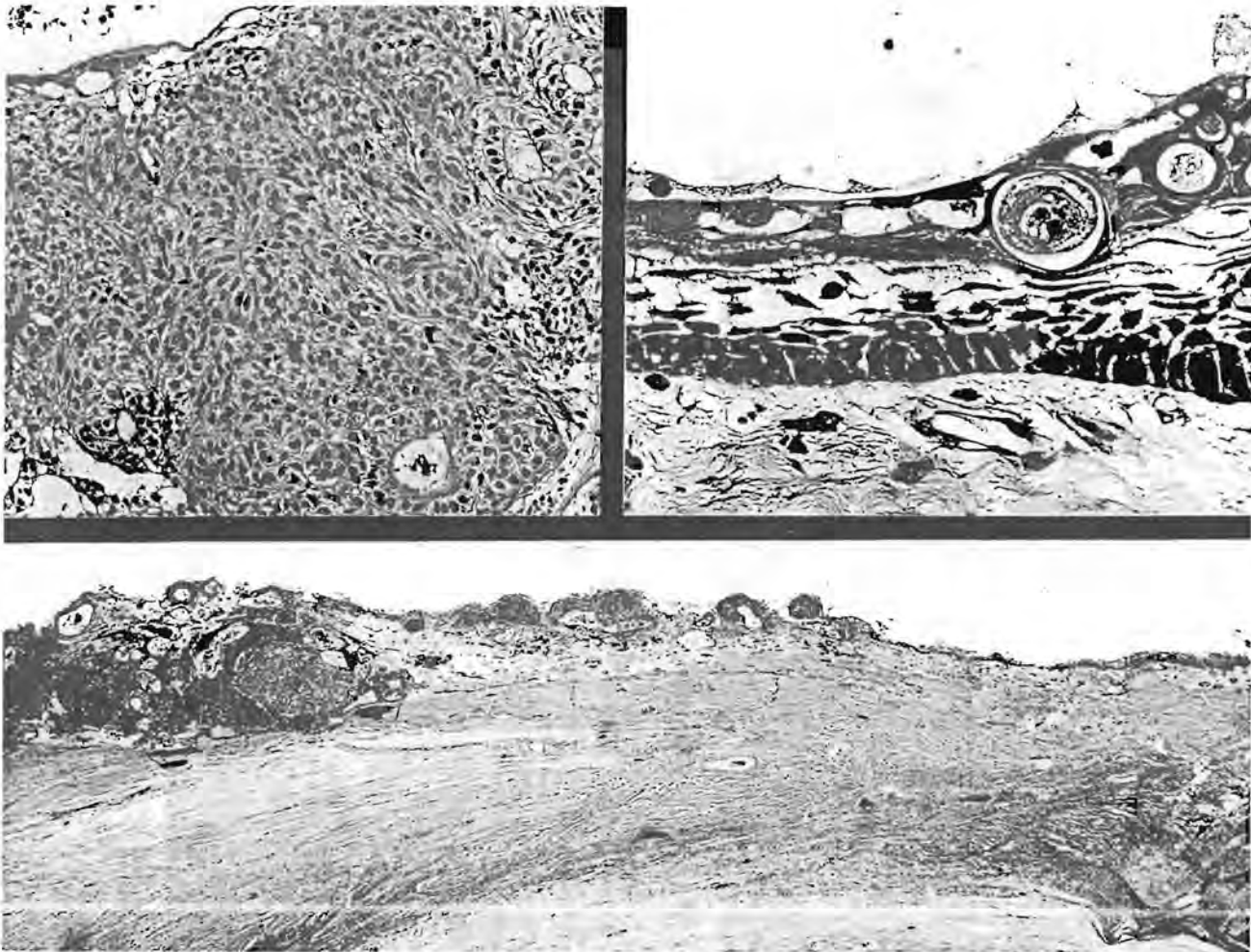


Fig. 4. A unicyclic ameloblastoma (right side of low power view and top right insert) which exhibited adenomatoid odontogenic tumour differentiation (left side low power view and top left insert). (H&E,  $\times 40$ ,  $\times 120$ ,  $\times 200$ ).

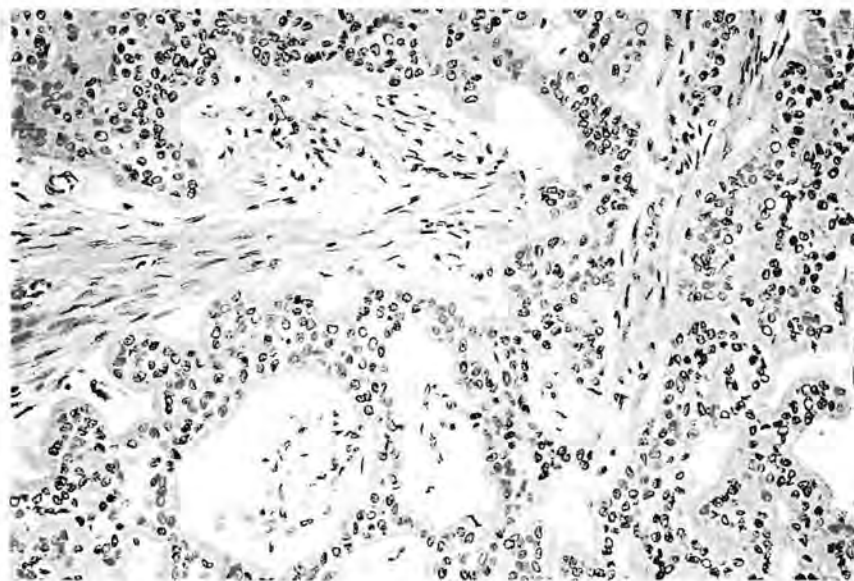


Fig. 5. A plexiform proliferation of ameloblastic epithelium exhibiting granules in all tumour cells. Typical features of ameloblastoma were not present (H&E,  $\times 180$ ).



Fig. 6. Desmoplastic ameloblastoma characterized by compressed epithelial strands in a mature connective tissue stroma. Typical features of ameloblastoma were not present (H&E,  $\times 180$ ).

ported as case studies. This has led to a generally accepted view that ameloblastomas are fairly homogeneous in their clinical and pathologic presentation. This review was undertaken to establish the spectrum of extraordinary and coincidental clinical and pathologic findings in a large collection of ameloblastomas diagnosed in a rural African population.

In the total sample, the left mandible was affected 2.5 times more commonly

than the right. Although there appears to be no apparent explanation for left mandibular predominance in our study, this finding is supported by a large Japanese series in which only 40% occurred on the right hand side (4).

The subclassification of unicystic ameloblastomas according to the microscopic appearance of the lining (2) was found to be impractical. Although areas of intraluminal proliferation or mural invasion positively confirmed un-

icyclic ameloblastomas in Groups 2 and 3 respectively, the subdivision into Group 1 lesions was found to be of limited value unless the whole tumor was processed for microscopic examination. In the case of large unicystic ameloblastomas, this was either impractical or even impossible, making the microscopic identification of foci of mural invasion in larger lesions unlikely. This dilemma is clearly illustrated in our study where fewer than three wax blocks were available for microscopic examination in 9 cases ultimately subclassified as Group 1 unicystic ameloblastomas.

Tumors which occur within bone generally predispose to pathologic fracture and aneurysmal bone cyst formation (22). Both these findings are, however, infrequently reported in association with ameloblastomas. The presenting symptom in only three of our patients was directly associated with pathologic fracture of the mandible. Aneurysmal bone cysts are reported to be rare in the jaws, occur mainly in young patients, and approximately one-third are found in association with other pathologies (23). A microscopic study of 42 ameloblastomas found hallmarks of aneurysmal bone cysts in 7 (13). Although the frequency of aneurysmal bone cyst change in our study was not as high, both our cases occurred in young patients. The coexistence of aneurysmal bone cyst with ameloblastoma is significant because of the excessive bleeding which may be encountered during surgery.

The association of an ameloblastic fibroma with ameloblastoma has not previously been reported. The example described here could be regarded as coincidental, as the patient was at an age when ameloblastic fibroma occurs most frequently. Simultaneous occurrence of ameloblastoma and odontoma is rare (24). These tumors, which have been designated as ontoameloblastomas, consists of epithelial proliferations typical of ameloblastomas associated with highly differentiated dental tissues either scattered throughout the tumor or, as in our case, as a single radiopaque mass (1). Squamous metaplasia is a well described and variable feature in ameloblastoma. Extensive squamous change, where follicles consist entirely of squamous epithelium with only traits of the original ameloblastomatous structure, is less frequent (23). One tumor in our series, which occurred in an elderly woman patient, exhibited this change and was diagnosed as a kera-



toameloblastoma. Although adenomatoid odontogenic tumor-like differentiation has been reported in a dentigerous cyst (25), this change in a unicystic ameloblastoma has not yet been described. Both our examples occurred in the anterior mandible and this, together with the hitherto undescribed phenomenon of mucous cell metaplasia, illustrates the differentiation potential of ameloblastic epithelium.

The presence of an HPV-induced verruca in the epithelial lining of a polycystic ameloblastoma adds an interesting parameter to the study of papillomavirus-induced epithelial proliferations. These lesions occur commonly on the skin and lining mucosa, to our knowledge this case, which was published recently (26), represents the first description of a verruca in a cystic epithelial tumor, the lining of which was within bone and not in contact with the oral mucosa. The origin of the melanocytes in odontogenic tumors and cysts is speculative. The cells of the neural crest interact in the development of teeth and it is not surprising that melanocytes can occur in odontogenic tissues. Although it is believed that melanotic odontogenic tumors occur more frequently in black patients (25), the number of cases reported is too low to draw conclusions. Our one melanotic ameloblastoma in the 108 cases studied places the frequency of this phenomenon below 1% in a black African sample and thus can hardly be regarded as common. The presence of hemosiderin in the neoplastic epithelium adjacent to an area of hemorrhage emphasizes the phagocytic capacity of neoplastic odontogenic epithelium. Varying amounts of granular cells are seen in the stellate reticulum of granular cell ameloblastomas. However, the presence of granules in all cells, including the peripheral layer, is rare. We believe this feature, which was present in two of our cases, represents full expression of granular cell differentiation. The plexiform granular cell odontogenic tumor reported by ALTINI *et al.* (1986) (27) is probably an example of this variant.

Quantification of stromal desmoplasia was found to be difficult as it varied between tumors and within the same tumor. Generally, ameloblastomas which infiltrate soft tissue and those which are inflamed exhibit more desmoplasia than others. Terminology such as "desmoplastic ameloblastoma" (16) should be used with great circumspection as

the intensity of the desmoplasia is variable and forms part of a continuous spectrum of stromal fibroplastic reaction in ameloblastomas. We suggest that certain criteria must be applied before the diagnosis of a desmoplastic ameloblastoma is made. These should include the absence of inflammatory changes and cortical bone perforation as well as the uniform presence of a mature, diffuse collagenous stromal tissue compressing the neoplastic epithelial component into strands. The two tumors which contained extensive interstitial bony deposits resemble the Japanese cases published recently (17, 18) and their clinical and radiographic differentiation from benign fibro-osseous proliferations is a pitfall to be avoided. The prominent bone formation appeared to be reactive in nature, probably linked to a process of interstitial connective tissue metaplasia rather than a result of induction, as the bony deposits were generally separated from the neoplastic epithelium by a broad band of inactive connective tissue. Unlike a recently published paper (28), we believe the desmoplastic and osteoplastic ameloblastomas are not distinct clinicopathologic entities but rather variants of the microscopic spectrum of stromal reactions in ameloblastomas.

This study demonstrates that although ameloblastomas are generally regarded as a homogeneous group of neoplasms, detailed investigations prove clinicopathologic diversity in a significant number of tumors. Many of these changes emphasize the differentiation potential of neoplastic odontogenic epithelium and add interesting parameters to the study of tissue reactions associated with this common odontogenic tumor.

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