Multiple dental anomalies and aggressive periodontitis: A coincidence or an association?

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ABSTRACT

Aggressive periodontitis (AgP) comprises a group of rare, often severe, rapidly progressive forms of periodontitis mostly characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families. Abnormal dental morphology and position have been associated with severe periodontal diseases. The purpose of this paper is to report a case of multiple dental anomalies associated with AgP. This paper reports a case of unusual association of multiple dental anomalies to AgP. Clinical findings and history led to the diagnosis of localized AgP, and radiologically. It was associated with multiple dental anomalies, especially supernumerary roots. Thus, the present case represents a very interesting demonstration of AgP association with supernumerary roots and the nature of this association merits further investigations.

Key words: Aggressive periodontitis, fusion, supernumerary roots

Aggressive periodontitis (AgP) comprises a group of rare, often severe, rapidly progressive forms of periodontitis mostly characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families.[1] AgP was characterized by: (1) noncontributory medical history; (2) Rapid attachment loss and bone destruction; (3) familial aggregation of cases; (4) lack of consistency between clinically visible bacterial deposits and severity of periodontal breakdown.[2] Based on specific clinical and laboratory features, special forms of AgP have been recognized: localized AgP (LAP) and generalized AgP (GAP). A diagnosis of LAP is done based on the evidence of circumpubertal onset and localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar and involving no more than two teeth other than first molars and incisors.[1]

Morphological dental anomalies of the permanent teeth are relatively common. These may be genetically determined; however, most arise sporadically and some may be affected by environmental factors acting during the morphodifferentiation stage of tooth formation.[3] Fusion is commonly identified as the union of two distinct dental organs. They are joined by the dentin; pulp chambers and canals may be limited and separated depending on the developmental stage when the union occurs.

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Supernumerary roots are one of the development anomalies pertaining to the tooth root morphology. These supernumerary roots may be due to the disturbances of the Hertwig's epithelial root sheath forming the root. They usually present no clinical problems other than difficulty in endodontic treatment and often go unnoticed.

Localized periodontitis has been associated with numerous dental anomalies such as fusion, talons cusp, root fusion, and developmental grooves.[4,5] Possible association of AgP with supernumerary teeth has been reported in the literature.[6,7] But there have been no published reports on supernumerary roots associated with AgP. The purpose of this paper is to report a case of multiple dental anomalies presenting with AgP and highlight the possibilities of a possible biological association.

CASE REPORT

A 23-year-old Indian female reported to the Department of Periodontics, Government Dental College and Hospital, Bangalore, with a complaint of pain and discharge in lower left permanent canine. She gave a history of similar pain in lower left first molar, three months back, for which she consulted a dentist and was prescribed medications which relieved her of pain and swelling. She was the second daughter of a consanguineous marriage and her 34-year-old elder sister was edentulous with a history of early spontaneous loss of teeth. Her medical history was unremarkable. She was a nonsmoker and had a normal blood glucose profile.

Hematological evaluation showed elevated ESR (68 mm/hr) and differential white blood cell counts showed slight elevations in the percentage of neutrophils (78%).

Clinical examination revealed deep pockets with exudation with teeth 16, 12, 26, 36, 42 and 46. Millers grade II mobility[8] was noted with teeth 42 and 36. Clinically visible bacterial deposits were minimal with plaque index of 1.23.[9] Rest of the teeth in the dentition were periodontally healthy with no increased probing depth or bleeding on probing.

Morphology of the permanent maxillary left first molar was irregular. The aspect of dental elements suggested the union of a supernumerary tooth crown with the mesial crown of this molar. In addition, increased mesiodistal crown width and distinct developmental occlusogingival grooves on labial and lingual surfaces were noticed. The permanent maxillary first molar of the right side showed single lingual cusp. The remaining maxillary and mandibular permanent teeth were normal in shape and all the third molars were absent. The fused supernumerary crown was carious but no tenderness on percussion could be elicited. Teeth # 22 and 19 were tender to vertical and horizontal percussion and teeth # 14, 19 and 22 responded negatively to electrical pulp testing.

Radiographic examination showed severe bone loss with teeth 16, 12, 26, 36, 42 and 46. The fusion of a supernumerary tooth with 26 was demonstrated with extensive periradicular radiolucency with the same. The lower first molars of either side revealed three roots and tooth 42 revealed two roots.

The remaining teeth were normal morphologically and no bone loss was observed with them. The severe bone loss associated with tooth 26 could be attributed to abnormal crown morphology favoring plaque accumulation but the attachment loss in the other periodontally involved teeth could not be justified with such a reason because the anomalies were localized to root portion and could not possibly have played role in the initiation or progress of periodontal breakdown until late in the disease process. The severity of bone loss relative to the age of the patient suggested a rapid rate of progression. Based on the clinical findings and a positive family history, a diagnosis of localized AgP was given.

Patient was educated about her oral condition and explained the treatment plan, which included thorough scaling and root planning (SRP) supplemented with antimicrobial agents. Systemic antibiotics (Doxycycline 100 mg once daily for 14 days after initial loading dose of 200 mg on first day), were prescribed along with thorough SRP. Teeth 42 and 36 were extracted because of extensive bone loss and hopeless prognosis. Endodontic treatment of 26 was suggested followed by flap for access and regenerative procedures.

Following extraction patient was relieved of pain and wished to continue her further treatment at her home town. Thus, the patient was referred with the complete case file to the competent person at her home town for further management.

DISCUSSION

The international classification workshop consensus indicated that not all listed primary and secondary features need to be present in order to assign an AgP diagnosis and that diagnosis may be based on clinical, radiological, and historical data alone.[1] Diagnosis of AgP in the present case was based on clinical findings of rapid bone loss in absence of significant clinically visible bacterial deposits, angular bone loss on radiographic examination, unremarkable medical history, and a positive family history.

Morphological dental anomalies of permanent teeth are relatively common. These may be genetically determined; however, most arise sporadically and some, including shape and size may be affected by environmental factors acting during the morphodifferentiation stage of tooth formation.[3] Abnormal dental morphology and position have been associated with severe periodontal diseases due to increased plaque accumulation and interference with plaque control. There have been numerous reports of carious and periodontal involvement of fused teeth.[10,11] Fusion can occur between teeth of the same dentition or mixed dentitions and between normal and supernumerary teeth. A diagnostic consideration, but not a set rule, is that supernumerary teeth are often slightly aberrant, and fusion between supernumerary and normal tooth will generally show difference in the two halves of the joined crown. Similar was the clinical picture of the present case. Teeth with this abnormality, if in anterior region, are unaesthetic due to their irregular morphology. They also present a high predisposition to caries and periodontal disease, and spacing problems. The main periodontal complication in fusion cases occurs due to the presence of fissures or grooves in the union between teeth involved. If these defects are very deep and extend subgingivally, the possibility of bacterial plaque accumulation in this area is quite high. The carious and periodontal involvement of the fused maxillary molar with a supernumerary tooth in the present case can be explained on the similar basis. On the contrary, severe attachment loss in the other periodontally involved teeth could not be explained on the similar grounds because the morphological anomalies were localized to the root portion, and could not have possibly played a role in initiation or progress of the periodontal breakdown until late in the disease process.

Earlier studies have documented that anatomic variations in molar root morphology may favor the development of localized periodontal problem by providing an environment favorable to plaque retention. Hou *et al.*, in 1997,[12] investigated the relationship between molar root fusion and localized periodontitis and found a statistically significant association. They found a rapid progression of attachment

loss in molars with fused roots and attributed it to gradually decreasing attachment dimension, unfavorable crown to root ratio, short root length, and a tapered shaped root. These negative characteristics of the molars with root fusion, probably offered less resistance to heavy occlusal loads and torque forces. Molar cervical enamel projection, one of the most common developmental abnormalities affecting furcation regions, are probably related to more rapid progression of pocket formation because of their anatomy and location.[13,14] But no such association has been demonstrated between supernumerary roots and localized periodontitis. A possible relationship of AgP to supernumerary teeth was described by Eley in 1974[15] Odell and Hughes in 1995[6] hypothesized on the possible association between supernumerary teeth and AgP. They reported that both AgP and supernumerary teeth are uncommon but have familial tendency and at the same time both are consistent with multifactorial and multigenetic etiology. But in a recent retrospective study by Gokhan *et al.*,[7] the association was suggested to be a random occurrence, rather than a biological one.

To the best of our acquaintance, no such association of supernumerary roots with AgP has been reported earlier in the literature, this being the first reported case of such an occurrence. The association of AgP to the supernumerary roots in this case may be only incidental; but absence of periodontal destruction in other normal teeth, the familial tendency and multifactorial etiology of both points towards a possible biological relation. Thus, the present case represents a very interesting demonstration of association of AgP with supernumerary roots and the nature of this association merits further investigations.

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