

Juvenile Periodontitis- A Case Report

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ABSTRACT

Aggressive periodontitis is a group of infrequent types of periodontal diseases with rapid attachment loss and bone destruction, which are initiated at a young age. Aggressive periodontitis has received considerable attention due to its peculiar clinical presentation, occurring around puberty, with an apparent lack of local factors such as heavy amounts of plaque and calculus,

in patients with reasonably good oral hygiene. Here, we present a case of a 21 years old female patient who complained of malaligned teeth and wanted the correction of these malaligned teeth.

Key Words: Aggressive periodontitis, Fibrocellular proliferation, Gingival fibromatosis, Puberty onset, Vertical bone loss around first molars

INTRODUCTION

Among all the forms of periodontitis, aggressive periodontitis has received considerable attention due to its peculiar clinical presentation, occurring around puberty, with an apparent lack of local factors such as heavy amounts of plaque and calculus, in patients with reasonably good oral hygiene [1].

Aggressive periodontitis is a group of infrequent types of periodontal diseases with rapid attachment loss and bone destruction, which are initiated at a young age. Though a variety of factors such as microbial, environmental, and behavioral factors and systemic diseases are suggested to influence the risk of aggressive periodontitis, an individual genetic profile is a crucial factor, influencing the systemic or host response-related risk [2],[3].

Aggressive periodontitis is characterized by a rapid and severe periodontal destruction in young systemically healthy subjects, and can be subdivided into localized and generalized forms according to the extension of the periodontal destruction [4].

Epidemiological surveys have shown that the prevalence of aggressive periodontitis varies among ethnic groups, regions and countries, and that it may range from 0.1% to 15% [5], [6]. This patient was later referred to the Department of Periodontics, where complete scaling and root planning, followed by the curettage of the required area was done. Then, she was put on analgesics and antibiotics to suppress the infection and was recalled after a week for follow up.

On extra oral examination, incompetent and everted lips were noted. On intraoral examination, proclined upper anteriors with spacing were noted [Table/Fig 1]. Anterior open and deep bites were also noted. On the right side, a class III molar relation and on the left side, a class II molar relation was noted. On probing, Grade I mobility of 11, 21, 32 and 42 was noted. [Table/Fig 2]



[Table/Fig-2]: Intraoral examination

The pockets were 5-6 mm deep, with an attachment loss of 2-3 mm; the mobility was grade I around the mandibular 1st molar. There was no significant pain and the probing revealed little subgingival plaque and calculus. Routine hematological investigations revealed normal readings. In the radiological examination, OPG showed moderate horizontal bone loss with the upper anteriors and mild bone loss with the upper and lower posteriors. A careful recording of the case history and the results of the clinical examination and the radiological findings confirmed the diagnosis of juvenile periodontitis [Table/Fig 3].



[Table/Fig-1]: Intraoral examination showing proclined upper anteriors



[Table/Fig-3]: OPG showed moderate horizontal bone loss

CASE REPORT

Here, we present a case of a 21 years old female patient who complained of malaligned teeth and she wanted them to be corrected.

DISCUSSION

A greater prevalence of aggressive periodontitis is reported in Africans and in African descent groups than in Caucasians and Hispanics [7], [8].

There are many reports in the literature which describe families with multiple aggressive periodontitis and affected individuals, thus suggesting familial aggregation [9-11].

Several research groups have used segregation analysis to determine the likely mode of inheritance for this trait. The patterns of disease in these families have led the investigators to postulate both the dominant and recessive modes of Mendelian inheritance for aggressive periodontitis [12-14].

Candidate gene approaches have been used to study aggressive periodontitis, but the results which have been obtained so far, are very diverse and conflicting [15], [16].

A case-control genome wide association study suggested a role of GLT6D1 in aggressive periodontitis in Germans [17].

One linkage study in African American families [18] showed that aggressive periodontitis is linked to the marker D1S492, which is located on chromosome 1q.

The disease appears to be the result of a defect in the immune response rather than it being plaque and calculus deposition [2].

It has been shown by many investigators that patients with aggressive periodontitis display functional defects of PMNL, monocytes or both, but without any systemic manifestations [19]. This results in a reduced defensive ability against some of the periodontal pathogens. Aggressive periodontitis has a familial tendency, which suggests a genetic predisposition [19].

The localized form of aggressive periodontitis predominantly affects the 1st molar and the incisors, with loss of attachment in at least two permanent teeth, one of which is the 1st molar. The rate of alveolar bone loss is considerably higher in aggressive periodontitis than in chronic periodontitis. A striking feature is the absence of clinical inflammation with minimal local factors, despite the presence of a deep periodontal pocket. Various periodontal pathogens have been implicated in sites of aggressive periodontitis, but the role of *Actinobacillus actinomycetem comitans* has been the predominant one. Several authors have referred to it as an arc-shaped bone loss which extends from the distal surface of the 2nd premolar to the mesial surface of the 2nd molar [19].

Clinically, the patient had characterized "first molar" presentation with interproximal attachment loss on the two permanent teeth, the left upper and the left lower molars. The pattern of alveolar bone loss was "arcuate", extending from the distal surface of the second premolar to the mesial surface of the second molar, both in the upper as well as the lower jaws on the left side. There was a lack of clinical inflammation despite the presence of deep periodontal pockets and advanced bone loss. The amount of plaque on the affected teeth was minimal, which seemed to be inconsistent with the amount of periodontal destruction which was present. The facts that the patient was a female and that the onset had been circum pubertal also supported the clinical picture of localized aggressive periodontitis [20].

The involvement of the first molars and the typical arcuate bone loss pattern at the given age of the patient suggested localized aggressive periodontitis.

CONCLUSION

The early diagnosis and the management of these cases can help oral clinicians to maintain the health and function of the permanent teeth and their surrounding structures.

REFERENCES:

- [1] Sharn Pal Sandhu, Vipin Kakar, Guneet Gogia, and S. C. Narula. Unilateral gingival fibromatosis with localized aggressive periodontitis (involving first molars): An unusual case report. *J Indian Soc Periodontol* . 2009;13 (2); 109-113.
- [2] Kinane DF, Shiba H, Hart TC. The genetic basis of periodontitis. *Periodontol* 2000;2005; 39: 91-117.
- [3] Meng H, Xu L, Li Q, Han J, Zhao Y. Determinants of host susceptibility in aggressive periodontitis. *Periodontol* 2000. 2007; 43: 133-159.
- [4] Armitage GC Development of a classification system for periodontal diseases and conditions. *Ann Periodontol*. 1999;4: 1-6.
- [5] Albandar JM, Brown LJ, Genco RJ, Loe H Clinical classification of periodontitis in adolescents and young adults. *J Clin Periodontol*. 1997;68: 545-555.
- [6] Tinoco EM, Beldi MI, Loureiro CA, Lana M, Campedelli F, et al. Localized juvenile periodontitis and *Actinobacillus actinomycetem comitans* in a Brazilian population. *Eur J Oral Sci*. 1997;105: 9-14.
- [7] Albandar JM, Tinoco EM Global epidemiology of periodontal diseases in children and young persons. *Periodontol* 2000;2002;29: 153-176.
- [8] Van der Velden U, Abbas F, Armand S, de Graaff J, Timmerman MF, et al. The effect of sibling relationship on the periodontal condition. *J Clin Periodontol*. 1993;20: 683-690.
- [9] Novak MJ, Novak KF. Early-onset periodontitis. *Curr Opin Periodontol*. 1996; 3: 45-58.
- [10] Tinoco EM, Sivakumar M, Preus HR The distribution and transmission of *Actinobacillus actinomycetem comitans* in families with localized juvenile periodontitis. *J Clin Periodontol*. 1998;25: 99-105.
- [11] Boughman JA, Halloran SL, Roulston D, Schwartz S, Suzuki JB, et al. An autosomal-dominant form of juvenile periodontitis: its localization to chromosome 4 and linkage to dentinogenesis imperfecta and Gc. *J Craniofac Genet Dev Biol*. 1986; 6: 341-350.
- [12] Hart TC, Marazita ML, McCanna KM, Schenkein HA, Diehl SR Re-evaluation of the chromosome 4q candidate region for early onset periodontitis. *Hum Genet*. 1993;91: 416-422.
- [13] Marazita ML, Burmeister JA, Gunsolley JC, Koertge TE, Lake K, et al. Evidence for autosomal dominant inheritance and race-specific heterogeneity in early-onset periodontitis. *J Periodontol*. 1994; 65: 623-630.
- [14] Carvalho FM, Tinoco EM, Govil M, Marazita ML, Vieira AR Aggressive periodontitis is likely influenced by a few small effect genes. *J Clin Periodontol*. 2009;36: 468-473.
- [15] Brett PM, Zygogianni P, Griffiths GS, Tomaz M, Parkar M, et al. Functional gene polymorphisms in aggressive and chronic periodontitis. *J Dent Res*. 2005;84: 1149-1153.
- [16] Covani U, Marconcini S, Giacomelli L, Sivozhelevov V, Barone A, et al. Bioinformatic prediction of leader genes in human periodontitis. *J Periodontol*. 2008;79: 1974-1983.
- [17] Schaefer AS, Richter GM, Nothnagel M, Manke T, Dommisch H, et al. GLT6D1 as a susceptibility locus for periodontitis. *Hum Mol Genet*. 2010;19: 553-562.
- [18] Li Y, Xu L, Hasturk H, Kantarci A, DePalma SR, et al. Localized aggressive periodontitis is linked to human chromosome 1q25. *Hum Genet*. 2004; 114: 291-297.
- [19] Takei N, Carranza K. *Gingival enlargement: Carranza's Clinical Periodontology*. 10th Ed. Elsevier: 2007. pp. 379-80.
- [20] Shafer, Hine, Levy Shafer's Text Book of Oral Pathology. 5th edition. Elsevier: 2004.

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