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Research Paper

Aggressive Periodontitis-A Case Report

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Abstract; *Both generalized and localized aggressive periodontitis are severe in terms of development and extent. Early detection is critical for successful therapy and a positive prognosis. Antibiotic therapy combined with mechanical and surgical debridement has been shown to be effective. Periodontal screening, proper dental hygiene, and elimination of risk factors and causative microorganisms could help avoid it in families with a history of Aggressive Periodontitis.*

Key words; *aggressive periodontitis, clinical presentation, diagnosis, treatment*

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I. INTRODUCTION

Aggressive periodontitis can be defined as a group of rare, severe, rapidly progressing form of periodontitis characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families. Although its prevalence has been shown to be substantially lower than that of chronic periodontitis, it might result in early tooth loss in affected persons if not detected and treated early1. The amount of local etiologic factors is not proportional to the severity of periodontal damage. Gross deposits of dental calculus are unusual in most cases of aggressive periodontitis, and the gingiva has a normal clinical appearance with no clinical symptoms of gingival inflammation in the early stages of the disease. However, dental plaque is present on the affected teeth's root surfaces. Some patients have poor oral hygiene as well as visible plaque and calculus. Patients with advanced illness exhibit clinical indications of gingival inflammation.2The affected teeth may become progressively mobile as the disease progresses, with labial movement and incisor spacing. There is also bleeding on probing the periodontal pockets, indicating ulceration of the crevicular epithelium. Closed curettage and surgical curettage, along with scaling and root planing and concomitant antibiotic treatment, are effective treatments.

Aggressive periodontitis was defined by the 1999 International Workshop for the Classification of Periodontal Diseases according to three primary characteristics: absence of systemic conditions that might contribute to periodontal disease, rapid loss of clinical attachment and alveolar bone, and familial aggregation of diseased individuals.

II. CASE REPORT

A 31-year female patient reported to the department of periodontics with a chief complaint of spacing between the upper and lower front teeth. She also complaints slight mobility in relation to the area. She noticed the spacing about one year gradually increasing and associated with intermittent episodes of pus discharge from the lower front tooth and from the upper right back tooth. She gave a history of taking antibiotics as per advice from primary health center and gradually subsiding the complaint. There was no other cosmetic concern and only the mobility was the complaint of the patient.

There was no history of any previous dental treatment, family history of similar complaint or early tooth lose noted from her parents. patient is a known case of emotionally unstable personality disorder (EUPD)and also under medication for the same for 5 years.

There was no abnormality detected in extraoral examination. Full complement of teeth was present. The oral hygiene status of the patient was good as reveled by the oral hygiene index. The was minimal amount of calculus and plaque. There was a grade I mobility i.r.t 43,42,41,31,32,33, and 11. There is a papillary

penetrating type of frenal attachment i.r.t to the maxillary labial frenum. There was a labial migration and flaring of maxillary and mandibular anteriors.

Gingival examination revealed normal color except for the labial aspect of 22, where it was slightly reddish. The margins are knife edged except for the labial aspect of 11,21,41,31 where it was blunt and rounded. The gingiva was firm and resilient except in the region on 41,31,16 where it was soft and edematous. There was no loss of stippling in the anterior region. The position of the gingival margin was apical to CEJ i.r.t 41,42,31,32. There was generalized bleeding on probing and exudation from 16,21,11 region. All together there was minimal local factors.

A full mouth periodontal charting revealed generalized periodontal pockets and attachment loss. Grade I recession i.r.t 41,41,31, and grade II i.r.t 32 according to millers' classification. Pockets were especially deeper in the molars measuring up to (6-7mm) and incisor region it was about 6 mm., with slight lesser involvement in the premolar region clinical attachment loss ranged from maximum 9mm in the 11,16. Mesial aspect to a minimum of 2mm in the premolar region.

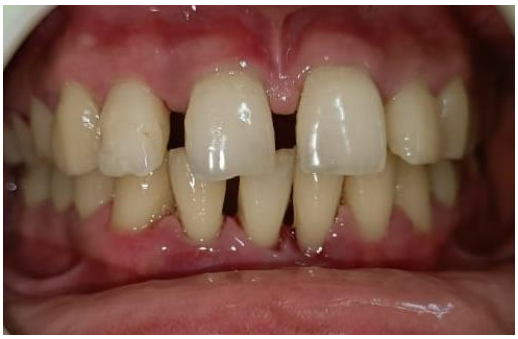


Figure1-afterphaseI therapy

An OPG was taken which revealed generalized distribution of bone loss which was combination of both horizontal and vertical bone loss. Routine blood investigation results were within the normal limit.

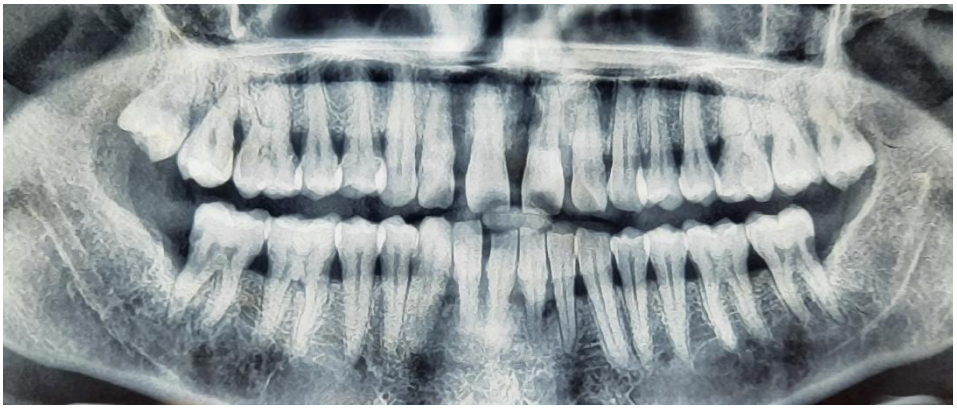


Figure2-OPG showing molar incisor pattern of bone lose

Based on the history, clinical examination, and radiographic findings a diagnosis of generalized aggressive periodontitis was made according to the criteria by AAP 1999 classification.

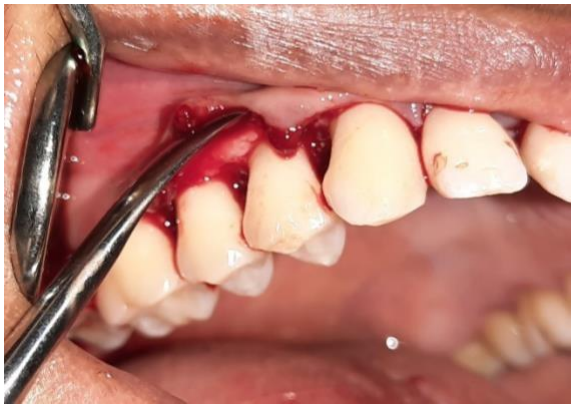
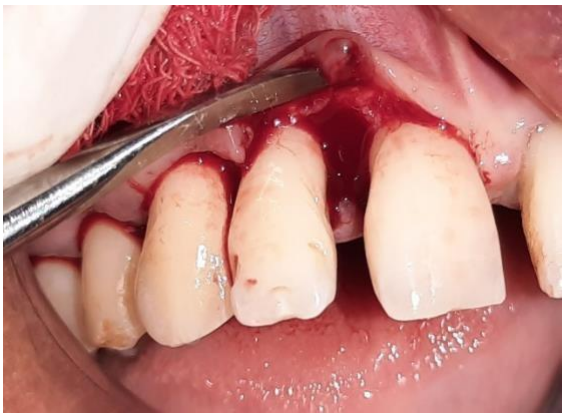
MANAGEMENT

A thorough supragingival scaling was performed following which the patient was motivated for better plaque control. Modified Bass technique was demonstrated, and the patient was educated on the use of interdental cleansing aids including dental floss and interdental brushes. Chlorhexidine mouth wash was prescribed for two weeks twice daily half an hour after brushing for better plaque control. Systemic antibiotics (Amoxycillin and Metronidazole, 250 mg of each thrice daily) were prescribed for 8 days, and the patient was recalled after 2 weeks for reevaluation. 2 weeks after subgingival scaling showed a reduction in probing depths and absence of bleeding on probing. Advice the patient for undergoing routine blood investigation.

A quadrant-wise full-mouth conventional flap surgery was planned including bone grafting in relation to the molar and incisor regions where predominantly vertical or intrabony defects were detected.


Figure 3-pre operative clinical evaluation of probing pocket depth

Conventional flap surgery and curettage for all the four quadrant under local anesthesia with 1:80,000 adrenaline was done.i.r.t 11,16,15 there was a an angular bone defect which was filled with demineralized bovine bone matrix (DMBM ,Osseo graft).Flap was approximated and simple interrupted suture place followed by periodontal pack was placed. After one-week suture removal was done, healing was satisfactory. All the quadrants with open flap debridement were completed in 1 month. Recall visit at one month showed stable and good results with patient's full satisfaction.

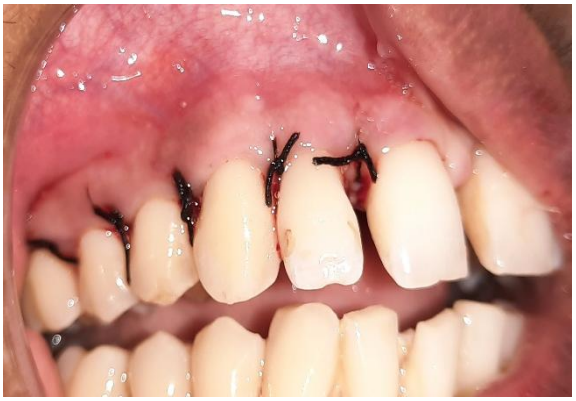


Figure 4 intra operative photograph showing bone defect, placement of graft and suture

III. DISCUSSION

The presence of aggressive periodontitis is distinguished by its early onset, rapid disease progression, involvement of several teeth with a specific pattern of periodontal tissue loss, and absence of systemic illnesses. In some people, tissue loss begins before puberty, however in the majority of cases, it begins at or shortly after puberty. Genetic susceptibility is important in the development of aggressive periodontitis and contributes to the disease's early onset of tissue destruction. The extent and shape of bone loss are largely determined by whether the lesion is detected at an early or advanced stage. Antibiotic therapy, scaling and root planing, and conventional flap surgery were all done, and the therapy was successful in revealing good results with considerable attachment gain during the 6-month follow-up period, which is still ongoing.

In aggressive periodontitis, attachment loss occurs at a much faster rate than in chronic periodontitis, the more common variant. Because the course of destruction occurs rapidly, evaluation of historical, radiographic and clinical data is necessary to estimate the start of disease. Some clinicians insist that massive attachment loss in a relatively young patient automatically indicates a rapid disease progression.

Immunological mediators play a role in the pathogenesis of several oral diseases, periodontal disease among them.

IV. CONCLUSION

Even though the prevalence of aggressive periodontitis is substantially smaller than that of chronic periodontitis, aggressive periodontitis management is more difficult than that of chronic periodontitis due to its strong genetic predisposition as an unmodifiable risk factor. Early detection is critical to successful treatment, aids in the prevention of disease progression, reducing the likelihood of extensive tissue destruction and alveolar bone loss. Management of aggressive periodontitis patients include nonsurgical phase, surgical therapy an interdisciplinary therapy and a lifelong supportive periodontal therapy. For better management protocols in the treatment of aggressive periodontitis, further understanding of the etiology, risk factors, pathogenesis, and host immune response is required, as well as improvements in regenerative ideas, tissue engineering, and gene therapy.

Vieira et al5 analyzed the clinical parameters of periodontal diseases to assess familial aggregation of such traits. The study found there was familial aggregation of dental plaque measurements, even after adjusting for confounding factors, such as age, sex, race and oral hygiene habits.

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