International Journal of Applied Dental Sciences 2018; 4(2): 214-217

ISSN Print: 2394-7489
ISSN Online: 2394-7497
IJADS 2018; 4(2): 214-217
© 2018 IJADS
www.oraljournal.com
Received: 05-02-2018
Accepted: 06-03-2018

Dr. Pendyala Gowri
M.D.S Reader, Department of Periodontics, Rural dental College, Loni, Maharashtra, India

Dr. Mopagar Viddyasagar
M.D.S, Principal, Professor and Head Department of Pedodontics, Rural dental College, Loni, Maharashtra, India

Dr. Raurale Ashish
M.D.S, Reader, Department of Pedodontics, Yashwantrao Chavhan Dental College, Ahmednagar Maharashtra, India

Dr. Joshi Saurabh
M.D.S, Reader, Department of Pedodontics, Rural dental College, Loni, Maharashtra, India

Correspondence
Dr. Pendyala Gowri
M.D.S Reader, Department of Periodontics, Rural dental College, Loni, Maharashtra, India

An atypical form of a typical pathology: A report of a combined lesion

Dr. Pendyala Gowri, Dr. Mopagar Viddyasagar, Dr. Raurale Ashish and Dr. Joshi Saurabh

Abstract
Aggressive periodontitis, refers to a multifactorial, severe, and rapidly progressing form of periodontitis. Gingival fibromatosis is a heterogenous group of enlargement characterized by progressive increase in submucosal connective tissue elements. Here we present an atypical case report of a 13 year old male patient who presented with severe, bilateral gingival enlargement along with aggressive periodontitis around first molars bilaterally. Careful recording of the case history and results of clinical examination, laboratory blood analysis, radiological findings, and microbiological and histopathological investigations were noted and a critical review of similar conditions was taken into account to arrive at the said diagnosis. Treatment consisted of extraction of teeth with hopeless prognosis, modified widman flap procedure in maxillary arch, gingivectomy in mandibular arch as well as supportive use of antibiotics and an interdisciplinary approach to restore the teeth functionally. After a follow-up period of 2 years, the patient's periodontal status remains healthy with no recurrence of gingival enlargement, facilitating the eruption of permanent teeth.

Keywords: Localized aggressive periodontitis, fibrocellular proliferation, gingival enlargement, bone loss around first molars, interdisciplinary approach

Introduction
Gingivitis and Periodontitis, the two major forms of inflammatory diseases affecting the periodontium may be present concurrently [1]. Gingival enlargement due to its concomitant unesthetic appearance and the formation of new niches for the periopathogenic bacteria is considered as a serious adverse reaction [2].
Aggressive Periodontitis [AgP], a multifactorial disease results from complex interactions between the microbial attack and specific host responses. Exogenous factors and a genetic predisposition for the disease are of particular significance. Patients with AgP display functional defects of PMNL, monocytes or both, but without any systemic manifestations [3].
The localized form typically has a circumpubertal onset. A striking feature is the absence of clinical inflammation with minimal local factors despite the presence of a deep periodontal pockets and rapid rate of alveolar bone loss. Several species of bacteria are detected in the localized form; however, the role of *Actinobacillus actinomycetemcomitans* has been the predominant one [3].
We report a case of localized aggressive periodontitis associated with gingival enlargement at the affected site, while reviewing a plausible explanation for the concomitant gingival enlargement occurring at sites affected with localized aggressive periodontitis.

Case Report
A 13- year old boy reported to the Department of Pedodontics, Rural Dental College, Loni, with a complaint of swollen gums in the upper and lower right and left back regions of the mouth. He reported that the enlargement had begun 3 years ago, with a slow progression and had pain and difficulty during mastication. His medical history appeared non- contributory.
Extraoaral examination revealed a symmetric face with bimaxillary protruding profile and competent lips. The clinical intra-oral examination revealed a diffuse gingival enlargement around the first molars on the right and left side, extending significantly around the adjacent teeth, both in the maxillary and mandibular arches.

The enlargement involved the marginal, interdental and attached gingiva and extended upto the occlusal surface, from 36 to 34 and 46 to44 in the mandibular arch and 16 to 14 and 26 to 24 in the maxillary arch. [gingival overgrowth index =3, according to Angelopoulos and Goaz index]. Both buccal and palatal/ lingual gingiva were involved. [Fig 1&2]

The gingival overgrowth was firm, dense and fibrotic consistency, exhibited no color change/ altered surface characteristics. Minimal inflammation of the gingiva was noted, and there was no gingival bleeding.

Full- mouth periodontal charting, including assessment of probing depth and clinical attachment level, revealed pockets of 10-14mm deep with an attachment loss of 5-7mm around all the first molars, scanty plaque and calculus. There was grade I mobility with 16 and 26; grade II mobility with 36; and grade III mobility with 46 [Based on modified Millers index]. Malpositioning of 25, 35 in addition to all the first molars were seen due to bulbous enlargement of gingiva along with extrusion of 46 leading to slight open bite [Fig 3].

Haematological investigations

The patients complete blood count was within normal limits, including basal glucose and creatinine levels, coagulation factors, alkaline phosphatase levels, absolute T4 lymphocyte count, immunoglobulins G,A,M ang IgG subclasses. Absolute monocyte and neutrophil counts were slightly elevated.

Radiological examination

Radiographic examination [OPG and IOPA Radiographs] revealed considerable amount of horizontal bone loss around 1st molars in maxillary arch and an arc-shaped bone loss around 1st molars in the mandibular arch. External Root resorption with 36 and 46, grade III furcation involvement with 36, extrusion of 36 with a floating tooth appearance could also be appreciated in the radiograph [Fig 4].

Microbiological examination

For further evaluation, subgingival plaque was sampled from the deepest pockets using paper points. The results revealed aerobic and anaerobic flora.

Histopathological examination

Histopathological examination showed an increase in the amount of connective tissue that was relatively avascular and consisted of increased fibrocellular content. There was numerous immature collagen fibres with abundant fibroblasts. The surface epithelium was acanthotic and parakeratotic.

Diagnosis

Due to the age of the patient, the severity of bone loss in isolated areas, and lack of any detectable systemic disease, the diagnosis of localized aggressive periodontitis was made. Also co-existent was the clinical picture of gingival enalrgement corroborated by the histopathologically representing a combined lesion of gingival fibromatosis with localized aggressive periodontitis.

Treatment

The main focus of therapy is to overcome the local pocket infection and to establish a subgingival flora that is compatible with healthy oral conditions.

The treatment consisted of extraction of teeth with hopeless prognosis i.e.36 and 46. The extracted teeth had an irregular external resorptive pattern. The remaining teeth then underwent phase I periodontal therapy that comprised of scaling and root planing and oral hygiene instructions.

Phase II therapy consisted of modified widman flap surgical procedure performed for the maxillary arch and gingivectomy procedure in the mandibular arch at the involved sites. The surgical intervention was carried out under local anesthesia using internal and crevicular incisions to remove the fibrotic increments of hyperplastic tissues. Scanty dental plaque and hard deposits was seen upon elevation of the flap.

The patient received systemic antibiotic therapy of amoxicillin (50 mg/Kg/day, divided into 3 doses) in combination with metronidazole (30 mg/Kg/day) for 10 days along with an analgesic for 3 days postoperatively (Diclofenac sodium 50 mg, twice a day). Sutures (3-0, silk) and a periodontal dressing (coe-Pak; GC America Alslp, Illinpois, U.S.A) were placed and removed after one week. Post operative healing was uneventful.

Periodontal maintenance phase (performed at monthly intervals) comprised of hand and ultrasonic scaling and reinforcement of oral hygiene instructions. Six months postoperative follow –up revealed significant reduction in probing depth and a clinical gain in attachment with no recurrence of gingival enlargement [Fig 5&6]. There was also improvement seen in the attachment levels which was evident radiographically [Fig 7].

Once the patients periodontal condition was stabilized, and in view of his good compliance, he was rehabilitated with partial acrylic appliances in the mandibular arch to restore function and prevent the drifting of erupting 37 and 47 into the extracted space. [Fig 8] The patient was then referred to an orthodontist for the correction of malaligned teeth. The patient is currently undergoing orthodontic treatment.

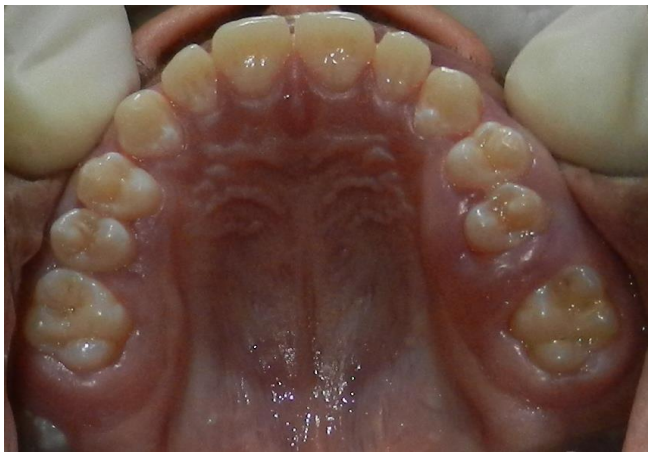

Fig 1: Preoperative picture depicting the enlargement seen in maxillary arch

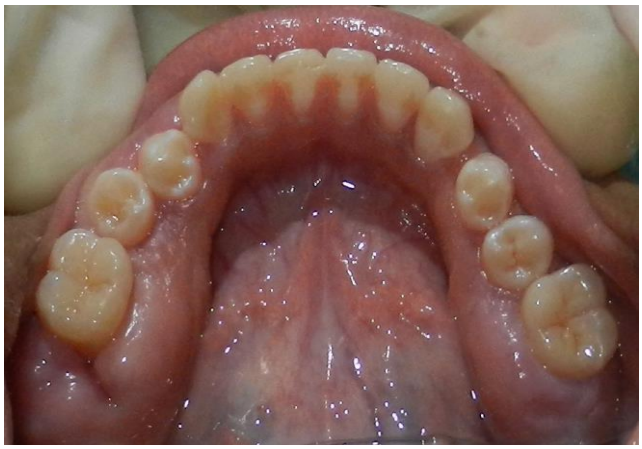

Fig 2: Preoperative picture depicting the enlargement seen in mandibular arch

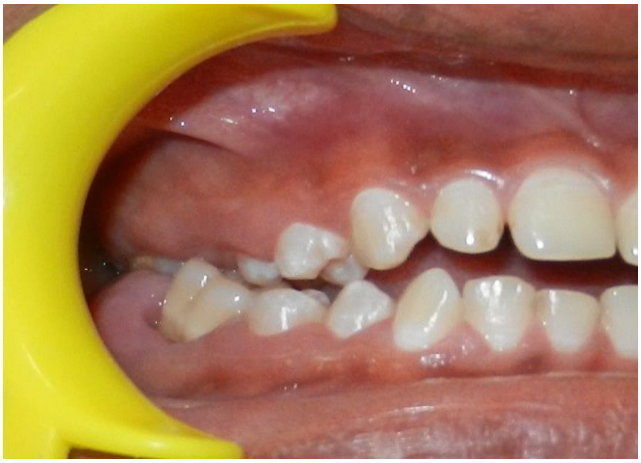

Fig 3: Extrusion of first molar leading to open bite

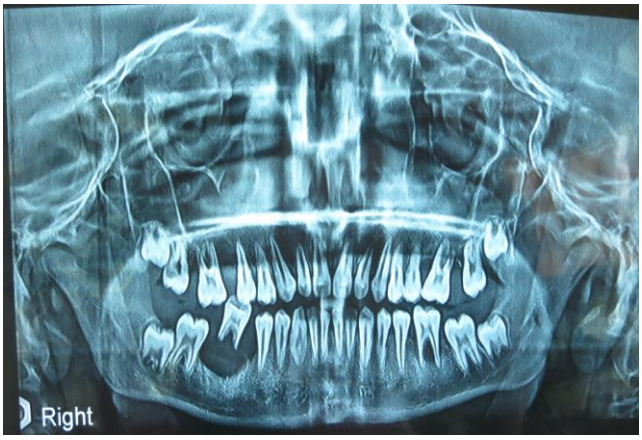

Fig 4: Radiographic picture depicting bone loss around 1st molars, extrusion of 46 and external resorption around 36 and 46

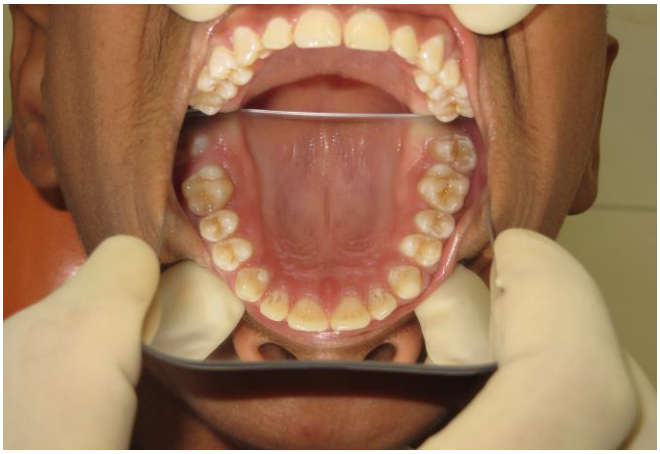

Fig 5

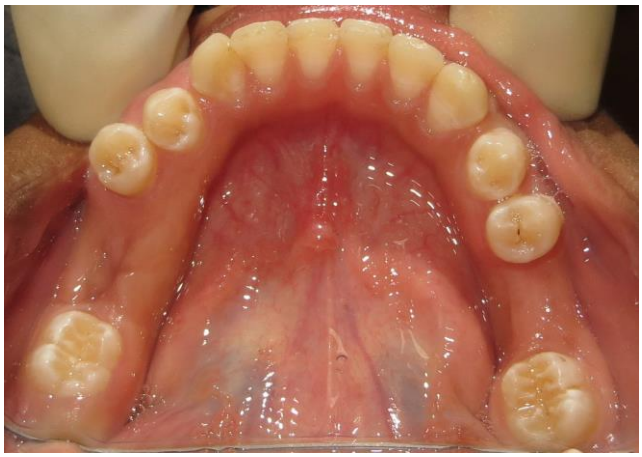

Fig 6

Fig 5, 6: Post operative view

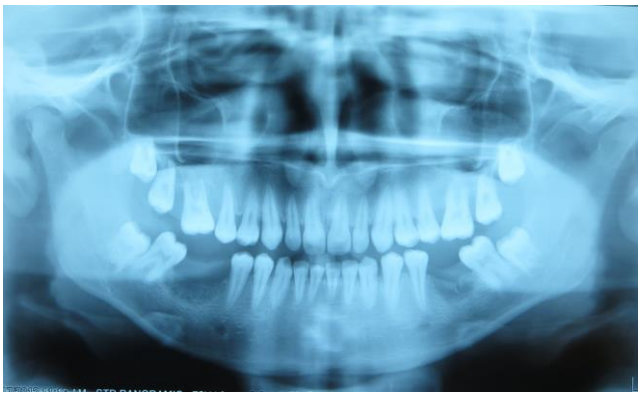

Fig 7: Radiographic picture depicting gain in bone around 16 and 26

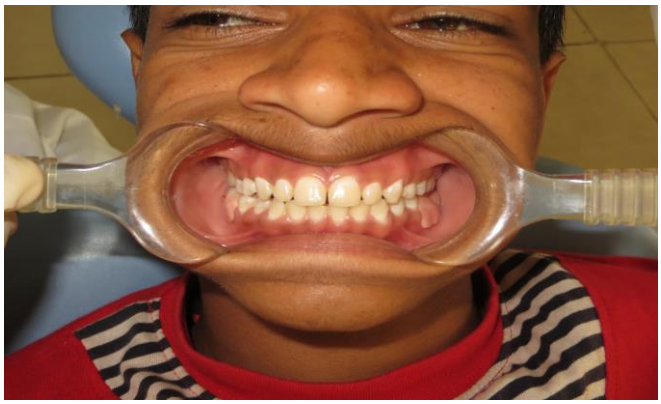

Fig 8: Picture depicting prosthetic rehabilation with partial dentures in mandibular arch.

Discussion

Treatment of Aggressive periodontitis must be pursued with a logical and regimented approach. One of the most important aspects of treatment success is to educate the patient about the disease, and to stress the importance of the patient's role in the success of treatment [4].

Teeth with moderate to advanced periodontal attachment loss and bone loss often have a poor prognosis. In our case, the teeth 36 and 46 with mobility equal to or greater than grade II were extracted. The extracted teeth had an irregular external resorptive pattern.

Resoprtion of root surfaces, requires some form of stimulus to begin, most commonly, trauma by mechanical, bacterial, or chemical event causes the various forms of root resoprtion to continue developing [5]. The probable explanation in our case could be that the microflora and the acidic environment combined in the periodontal pocket area could have caused the root resorption [6].

Also co-existent in our case was the clinical picture of gingival fibromatosis. In vitro studies have suggested that despite the degradation of the fibrillar collagen, fibroblasts are able to synthesize a new type of collagen, type I trimer which accumulates in the gingiva or that inflamed human gingiva contains fibroblasts with different phenotype than those from the normal tissue, the myofibroblasts, which are able to synthesize large amount of collagen leading to enlargement [7].

The periodontal pathogens, specifically *Actinobacillus actinomycetemcomitans*, remain in the tissues after therapy to reinfect and has been implicated as the reason that aggressive periodontitis does not respond to conventional therapy alone.

[8]. Hence, adjunctive systemic antibiotic use along with mechanical debridement is an acceptable treatment option for Aggressice Periodontitis. Given the possible emergence of tetracycline-resistant *A. actinomycetemcomitans* [9] a combination of metronidazole and amoxicillin was prescribed because when taken concurrently amoxicillin has been shown to increase the bacterial cell uptake of metronidazole [10].

Successful management of patients with AgP must include interdisciplinary approach as part of the treatment plan. In our 13 year old severely compromised teeth were extracted and a removable prosthesis was designed to accommodate future eruption of succedaneous teeth [37 and 47] without drifting into the extraction space. He is currently undergoing orthodontic treatment for the correction of malaligned teeth.

Frequent maintenance visits is one of the most important factors in the the success of treatment in patients with AgP. The duration between these recall visits was short during the first period after the patient's completion of therapy, no longer than 3-month intervals. Over time the recall maintenance interval was adjusted (less often) based on the patient's ability to maintain optimal oral hygiene and control of disease, as determined by each examination. No recurrence was observed during 2 years follow up period and patient showed remarkable functional improvement.

Conclusion

The present case was unique as it depicts an unusual condition of two independent disease entities affecting only specific areas of dentition due to submergence of inter-related factors. Aggressive forms of periodontitis are a challenge for the clinician as they are infrequently encountered and also the predictability of treatment success varies from one patient to another. The best treatment for these patients is a combination of conventional treatment with antimicrobial therapy and close follow – up care. Further understanding of the etiology, risk factors, pathogenesis and host immune response along with advances in regenerative concepts, tissue engineering and gene therapy is needed for formulating better management protocols of aggressive periodontitis cases.

References

1. Armitage GC. Development of a Classification System for Periodontal Diseases and Conditions. Ann Periodontol. 1999; 4:1-6.
2. Takada K, Sugiyama H, Umezawa K, Mega J, Hirasawa M. The subgingival microflora in phenytoin- induced gingival hyperplasia. J Periodontol Res. 2003; 38:477-81.
3. Shafer, Hine Levy. Shafer's Text Book of Oral Pathology, 5 th edition, Elsevier, 2004, 543-8.
4. American Academy of Periodontology: Parameter on Aggressive Periodontitis, J Periodontol. 2000; 71:867-869.
5. Bergmans L, Van Cleynenbreugel J, Vergeken E, Wevers M, Van Meerbeck B, Lambrechts P. Cervical external root resorption in vital teeth. J Clin Periodontol. 2002; 29:580-585.
6. Toto PD, Restarski JS. The histogenesis of pulpal odontoclasts. Oral Surg Oral Med Oral Pathol. 1967; 16:172-178.
7. Bartold M, Narayanan AS. Molecular and cell biology of healthy and diseased periodontal tissues. Periodontol 2000, 2006; 40:29-49.
8. Saglie FR, Carranza JR, Newman MG. Identification of tissue invading bacteria in human periodontal disease. J Periodontal Res. 1982; 17:452-455.
9. Walker C, Karpinia K. Rationale of use of antibiotics in periodontics. J Periodontol. 2002; 73:1188-1196.
10. Pavicic MJ, Van Winkelhoff AJ, Pavicic – Temming YA, DeGraaff J. Amoxicillin causes an enhanced uptake of metronidazole in Actinobacillus actinomycetemcomitans: A mechanism of synergy. J Antimicrob Chemother. 1994; 34:1047-1050.