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AGGRESSIVE PERIODONTITIS WITH STREPTOCOCCAL GINGIVITIS: CASE REPORT

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ABSTRACT

Acute streptococcal gingivitis is a condition of acute inflammation of the oral mucosa. The pathogens implicated in gingival inflammation are rarely different from that of the routine plaque-associated gingivitis. Streptococcal infections of gingiva are seen rarely and the origin of such gingival inflammation is different from that of plaque associated gingivitis. This case report article states a patient who presented with severe gingival inflammation with attachment loss that was diagnosed as an acute streptococcal infection associated with aggressive periodontitis. A case of aggressive periodontitis with streptococcal gingivitis was reported which was diagnosed and treated with no postoperative complications.

Key words: Aggressive Periodontitis, Acute Streptococcal Gingivitis, Gingival Inflammation, Attachment Loss etc.

INTRODUCTION

Acute streptococcal gingivitis is a condition which causes acute inflammation of the oral mucosa. The pathogens implicated in gingival inflammation are rarely different from that of routine plaque-associated gingivitis such as Treponema pallidum, Neisseria gonorrhea and recently streptococci infections [1, 2]. Streptococci seen in the composition of microbial dental plaque causes inflammatory periodontal disease and dental caries [3]. The streptococci related disease of gingiva has very specific microbiological and clinic features but there have been only a few cases reported in different articles. An important feature of streptococcal gingivitis includes that it can be seen on gingiva with the other inflammatory diseases like throat inflammations [4, 5].

Case History

A 43 year old female came with complain of bleeding gums, gingival hyperplasia and halitosis. The patient had a history of pain and bleeding while brushing. Past medical history was not significant. On examination, generalized edematous, hyperplastic gingiva, bleeding on probing and calculus formation was seen. Gingival index, plaque index, periodontal probing depths (PPD) and clinical attachment levels (CAL) of the teeth were recorded before and after the treatment by using a manual probe. Periodontal examination revealed gingival swelling, 78.2% bleeding at probing sites, 52.7% plaque control record, and 72.9% of the sites had a periodontal pocket depth of 3 mm or more. Upon initial examination, radiographs demonstrated extensive generalized vertical intra-bony defects in the maxillary and mandibular arches. Based on the clinical and radiographic findings, a diagnosis of generalize aggressive Periodontitis was assigned to the patient. Treatment was started with systematic scaling and planning of all accessible root surfaces and the introduction of healthy oral hygiene. During oral hygiene instruction and subsequent initial preparation as a chemotherapeutic support tetracycline had been given to the patient with a mouth rinse including Chlorhexidine Digluconate and Benzidamin HCL. After optimal oral hygiene was provided, based on persistence of periodontal lesions, a second phase of therapy was planned and the advanced

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periodontal therapy, flap and gingivectomy operations were done and remaining roots were removed. After resolution of the periodontal infection, the patient was placed on personal maintenance care program including continuous evaluation of the occurrence and the risk of disease progression. After a week, bleeding, edema and dark red discoloration on the marginal areas of gingiva developed again. Further medical tests were carried out to look for searching an underlying systemic disease and its impact on the etiology of the disease. Acute streptococcal infection was suspected because of the results were in normal limits. Cultures of the gingival samples were done and it was seen that it grew streptococcus pyogenes and a few other microorganisms. The treatment was given accordingly and patient was treated. No post operative complications were seen.

DISCUSSION

In a revised classification system for periodontal diseases, Aggressive Periodontitis (AP) was redefined to comprise a complex entity of microbial alterations and cellular dysfunctions that differentiate the underlying molecular mechanisms from chronic periodontal disease [6]. Once AP has been diagnosed, a comprehensive periodontal treatment plan must be developed and followed. The treatment of periodontal diseases is divided into four phases: systemic, hygienic, corrective and maintenance/supportive therapy [7]. As treatment progresses through the four phases, the dentist uses both surgical and non-surgical therapy to remove biofilm created by the bacterial pathogens. The adjunctive systemic use of antimicrobials along with mechanical debridement of the root surfaces to disrupt the biofilm is effective in patients with AP [8]. Tetracycline was chosen for chemotherapy because of its affectivity for periodontal diseases and high secretion capacity from gingival tissue. Although a successful and appropriate treatment was applied to the patient according to the recommendations for AP, streptococcal inflammation increased the worsening of the oral findings present in AP.

Penicillin is the treatment of choice for streptococci infections. Sulfonamides and broad spectrum antibiotics can also be used to treat streptococcal infections. Identification of etiology, diagnosis of disease and treatment of acute gingivo-stomatitis are critically important of untreated group A. beta-hemolytic streptococcal infection, which has many serious complications [9]. In addition to this primary etiologic factor, other factors such as differentiation of saliva composition can help in progression or control of the disease by acting as a disease marker or helping in defense mechanism.

Saliva has some antimicrobial activity against many different microorganisms due to the presence of immunoglobulin and non-immunoglobulin agents in its content. At physiologic concentrations and neutral pH, it prevents the bacterial glycolysis by inhibiting the pH and potentiates the antibacterial defense mechanisms as a bacteriostatic agent. Even though saliva has all those beneficiary antimicrobial effects that were mentioned above, sometimes it may not be sufficient enough to kill some specific bacteria which can be available in oral pH values of 6–8 and for streptococcus species which can survive at a low pH and to continue producing acid [10].

CONCLUSION

There are various advanced diagnostic and treatment modalities available in the management of aggressive periodontitis though the conventional techniques retain their popularity to date. Penicillin is the drug of choice for such cases but other drugs like broad spectrum antibiotics can also be used. A case of aggressive periodontitis with streptococcal gingivitis was reported which was diagnosed treated with no postoperative complications.

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