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Streptococcal Gingivitis: A Report of Case with a Description of a Unique Gingival Prothesis

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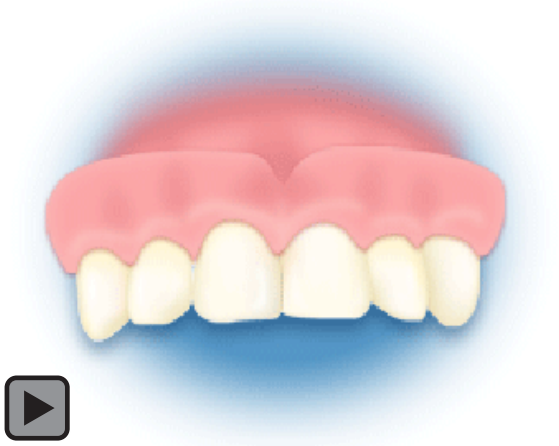

Abstract

Acute streptococcal gingivitis is an acute inflammation of the oral mucosa. Specific bacterial infections of the gingiva may be due to *neisseria gonorrhea, treponema pallidum, streptococci,* and other organisms. Streptococcal infections are seen rarely. This case report describes a patient who presented with severe gingival inflammation and pain that was diagnosed as an acute streptococcal infection. Bacterial cultures were obtained from the lesion, and biopsies were obtained from the gingiva of lower incisors for histopathologic evaluation. The patient was successfully treated using conventional periodontal therapy (scaling, root planning, curettage) and antibacterial agents. The reconstructive phase for this patient consisted of the fabrication of a heat-cured acrylic gingival facade to mask the gingival recession. The treatment of acute gingivostomatitis is of importance because of the possibility of systemic secondary infections. When esthetics is important, a gingival prostheses can be considered. The differential diagnosis, etiology, and treatment of acute streptococcal gingivitis are discussed and the literature is reviewed in this report.

Keywords: Periodontal disease, gingivitis, bacteria, infection, streptococcus bacteria, gingival prosthesis, recession, epithesis, gingival facade

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Introduction

Periodontitis is a multi-factorial disease with microbial dental plaque as the initiator of periodontal disease.[1] The manifestation and progression of periodontitis is influenced by a wide variety of determinants and factors. One of the important factors is the microbial composition of dental plaque. The composition of dental plaque in the oral cavity differs in healthy gingiva, gingivitis, and periodontitis.[2-4] Gingivitis represents a spectrum of diseases whose onset is commonly attributed to the presence of bacteria, but there are other forms of gingivitis that are not primarily plaque-related. Non-plaque induced gingival lesions can result from specific bacterial pathogens such as *neisseria gonorrhea, streptococci* from viral infections and from fungal infections.[5-8] The origin of gingival inflammation is occasionally different from that of routine plaque-associated gingivitis.[9] Acute streptococcal gingivostomatitis is an acute inflammation of the oral mucosa. Specific bacterial infections of the gingiva may be due to *neisseria gonorrhea, treponema pallidum, streptococci,* and other organisms.[9]

Streptococci are the earliest microorganisms found in the new formation of bacterial plaque on the teeth of humans.[10] Inflammatory periodontal disease and dental caries are caused by *streptococci*. The oral microflora is very complex. The microbial population that forms in gingival sulci and on the surfaces of teeth of a patient with healthy gingiva differs from the bacterial populations found in moderate gingivitis, specific bacterial infections of gingiva, and periodontitis.[2-4,11] While *actinomyces actinomycetemcomitans, porphyromonas gingivalis, prevotella intermedia,* and anaerobic gram-negative bacteria are increased in periodontitis, *fusobacterium nucleatum, treponema vincentii, treponema* and *veillonella* species are reduced in necrotizing ulcerative gingivitis (NUG). Streptococcus species (*streptococcus sangius, beta-hemolytic streptococcus A and B, streptococcus viridans, streptococcus pyogenes*) on the surfaces of teeth and gingiva are increased in streptococcal gingivostomatitis. The symptoms of acute streotococcal gingivastomatitis are usually described as diffusely swollen, bright red gingiva associated with malaise, fever, and submandibular lymphadenitis. Specific bacterial infections such as streptococcal gingivostomatitis, neisseria gonorrhea, and the others are very important, but little attention is given to these diseases because they are rarely seen. There have been two very well-documented cases of streptococcal gingivostomatitis reported in the medico-dental literature.[8,12]

Gingival recession or marginal tissue recession is defined as the location of the marginal tissueapical to the cement-enamel junction (CEJ) with exposure of the root surface.[13] Gingival recession defects may be treated by a number of procedures including rotational and advanced gingival flaps, free gingival or connective tissue grafts[14,15], and by applying principles for guided tissue regeneration (GTR).[16,17] A gingival facade is a treatment option in cases of severe recession.

A case report of periodontitis, gingival recession, and streptococcal gingivitis will be presented. The clinical features, differential diagnosis, etiology, and treatment of the disease are discussed and the literature is reviewed.

Case Report

A systemically healthy 23-year old Turkish man presented with a complaint of severe pain. The patient had not used any topical or systemic medications. However, he smokes one pack of cigarettes a day. The gingiva was red, swollen, and acutely inflamed. There was a substantial amount of supragingival and subgingival calculus present (Figure 1).

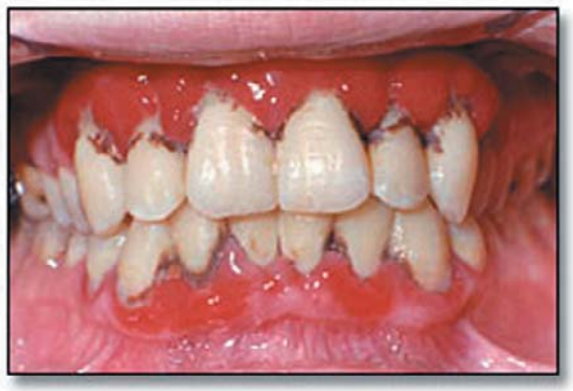

Figure 1. Diffuse erythematous areas in maxillary-mandibular gingiva.

Involvement was mainly limited to the marginal and attached gingiva areas of the maxillary and mandibular anterior regions. A panoramic radiograph was taken of the patient (Figure 2).

The patient suffered a high temperature (38.5°C), pain, and a sore throat. The pharynx was red. The total white blood cell count was 15,000. Bacterial samples were obtained by scraping the surface of the gingival lesion on the anterior lower jaw with a sterile chip. The anaerobic-culture technique is used to identify the bacteria. Cultures of the gingival samples grew mostly *streptococcus pyogenes* and a few other microorganisms.

Biopsies were obtained during surgical periodontal treatment (open curretage using the modified Widman flap) from the gingiva of lower incisors for histopathologic evaluation. The histopathologic evaluation revealed the specimen was covered with stratified squamous epithelium, with a mononuclear inflammatory cell infiltrate within the lamina propria (Figure 3). However, a histological analysis alone does not provide a definitive diagnosis to confirm acute streptococcal infection of the gingiva. After calculus was removed by scaling and root planning, open curettage was carried out using a modified Widman flap. Amoxicillin, (1000 mg. daily for 10 days), benzidamin HCL (150 mg. daily), and Chlorhexidine gluconate (0.2%) mouthwash were prescribed. The treatment outcome is shown in Figure 4.

After two months of healing, the patient presented for re-examination in preparation for removable prosthodontic treatment. After the periodontal condition resolved completely, a removable gingival prosthesis was fabricated to mask the gingival recession (Figures 5, 6). Heat-cured acrylic (QC-20, Dentsply Detrey Prothesen Material, UK) was used to fabricate the gingival prosthesis which is only worn for esthetic purposes.

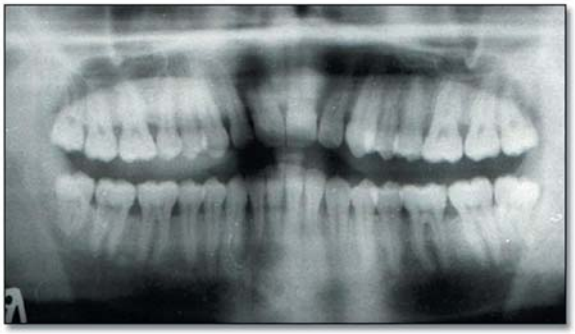

Figure 2. Mild bone loss in jaws.

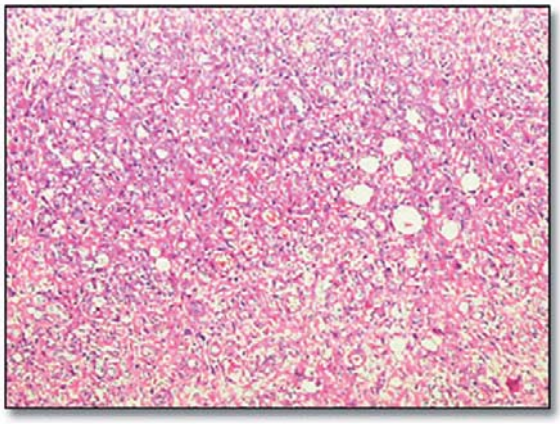

Figure 3. Biopsy of the gingival specimen.

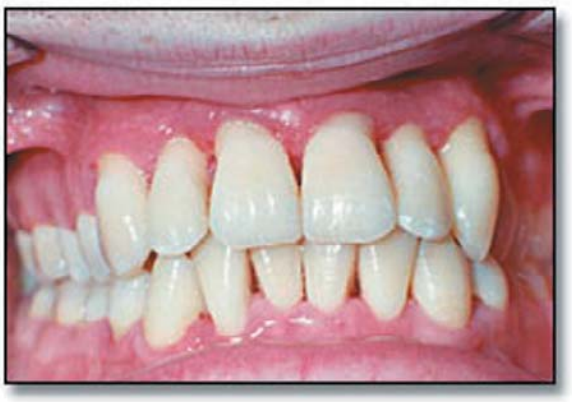

Figure 4. After treatment.

Description of the Gingival Prosthesis

An irreversible hydrocolloid impression material (Alginoplast; Heraeus Kulzer, Holland) was mixed according to the manufacturer's directions and an impression of the upper jaw was taken. A cast was then made with type IV dental stone (Bego Bremer Goldschlagerei Wilh, Herbst GmbH & co. Emil sommer, Bremen). A wax pattern for the prosthesis was prepared, invested in denture flasks, and processed with heat-cured acrylic resin (QC-20, Dentsply Detrey Prothesen Material, UK) using the short processing method. Heat-cured

Figure 5. Epithesis model.

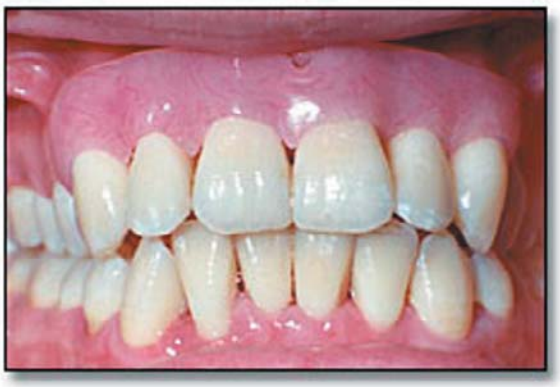

Figure 6. Epithesis and adaptation on teeth.

acrylic resin was prepared in accordance with the manufacturer's recommendations. After curing and deflasking, the prosthesis model was polished, disinfected, and adapted to the upper teeth of the patient during an insertion visit.

Discussion

Streptococcal gingivitis or gingivostomatitis is a rare condition that may present as an acute condition with fever, malaise, and pain associated with acutely inflamed, diffuse, red, and swollen gingival with increased bleeding and occasional gingival abscess formation.[18] Similar descriptions of the clinical features of this disease can be found in the literature.[8,9,12] The diagnosis of streptococcal infection generally depends on clinical symptoms. A histopathologic examination does not provide a definitive diagnosis to confirm acute streptococcal infection of the gingiva. The differential diagnosis includes primary herpetic gingivostomatitis, necrotizing ulcerative gingivitis, and edematous type of acute and chronic marginal gingivitis. Necrosis of the gingival margin is not a feature of this disease, however, NUG causes the necrosis of the gingival papilla.

Herpetic gingivostomatits occurs frequently in infants and children. In its initial stage, it is characterized by the presence of discrete, sypherical gray vesicles. Laboratory diagnosis requires cultural isolation and identification of the causative *streptococci*. Techniques that have been applied to detect periodontal pathogens are immuno-assays (ELISA), enzyme tests, anaerobic culture, and polymerase chain reactions (PCR). Each of these techniques have advantages and disadvantages.

The anaerobic-culture technique is the most extensive technique to study the composition of supra and subgingival plaque. The anaerobic-culture technique is an open test system. *Streptococci* are a large group of gram-positive *spherical cocci* that are classified into several groups. Serologic group A *streptococci* are the predominant group in the production of human infections.

In past articles, cultures were reported to grow mainly *beta-hemolytic streptococcus* from group A.[8,12] In addition *Streptococcus viridans* has been cultured by previous authors.[19] *Streptococcus pyogenes* was predominantly cultured from the lesions in this case. Anti-infective therapy includes both mechanical and chemotherapeutic approaches to minimize or eliminate microbial biofilm (bacterial plaque), the primary etiology of gingivitis and periodontitis. Mechanical therapy consists of debridement of the roots by the meticulous use of hand or power-driven scalers to remove plaque, endotoxins, calculus, and other plaque-retentive local factors. The term mechanical therapy refers to both supragingival and subgingival scaling as well as root planing.

The recommended antibiotics are penicillin-metronidazole, ampicillin-sulbactam, or clindamycin.[20] However, the treatment choice for *streptococci* infections is penicillin. Penicillin and derivatives were administered to the patient in addition to scaling, root planning, and curettage.

Gingival defects may be treated with surgical or prosthetic approaches. Prosthetic replacement is especially useful when a larger amount of tissue needs replacement. These appliances have the advantage of being able to hide extensive amounts of tissue loss very conservatively. They are easy to fabricate and repair. In addition, gingival facades do not require surgical intervention. Tissue replacement prostheses may be used to replace tissue lost through surgical gingival procedures, trauma, ridge resorption, or traumatic tooth extraction.[21] With this method, large tissue volumes are easily replaced by artificial materials.

Conclusion

Streptococcal gingivitis has been given little attention in the dental literature in general and in the periodontal literature in particular. The recognition, identification, and treatment of acute gingivostomatitis is of great importance because of the possibility of systemic secondary infections. Systemic antibacterial therapy is recommended to prevent unfavorable sequellae of streptococcal gingivitis such as meningitis, pericarditis, rheumatic fever, pneumonia, otitis media, mastoiditis, sinusitis, and septicemia. If this disease is not treated, these complications can endanger the systemic health of the patient.

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