Case Report

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ACTINOMYCOTIC GINGIVAL ENLARGEMENT: A RARE LESION

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ABSTRACT:

Actinomycosis is a chronic infectious granulomatous disease caused by saprophytic Actinomyces species. The purpose of this article is to report on a case of actinomycosis with clinical findings similar to any other chronic inflammatory gingival enlargement with pus discharge, cleft formation & heavy plaque accumulation. The diagnosis was based on histopathological findings. Due to the opportunistic characteristics of the actinomycotic infection, early diagnosis of the lesion, together with the adequate therapy and management are of great importance to prevent the spread of the disease. Proper knowledge of the different types of periodontitis is essential to distinguish any atypical presentation of tissue destruction.

Key words: Actinomycosis, Chronic inflammation, gingival enlargement, Granulomatous, Suppuration.

INTRODUCTION

Actinomycosis is a subacute to chronic bacterial infection characterized by contiguous spread, suppurative and granulomatous inflammation, with formation of multiple abscesses and sinus tracts that may discharge 'sulfur granules. Actinomycosis can affect people of all ages, but the majority of cases are reported in young to middle-aged adults (aged 20-50 yrs). Actinomycosis occurs worldwide, with likely higher prevalence rates in areas with low socioeconomic status and poor dental hygiene. As a disease process, the causative organism can primarily or secondarily invade gingiva, jaw, neck, pleura, lungs. The most common clinical forms of actinomycosis are cervicofacial (ie, lumpy jaw), thoracic, and abdominal.[1] Lesion in the oral cavity frequently involve the mandible, tongue, lips, and oral mucosa but gingival involvement is rare. [2] A predilection for male subjects is observed with a male : female ratio of 4:1 .The literature reports no racial predilection. The mandible is more commonly involved than maxilla (4:1). [3]

Actinomycosis first described in the early 19th century as disease entity found in cattle and later by Langenbeck in 1845 in humans as a rare disease that is caused by an inhabitant of the normal oral flora.[3] The causative organisms of this disease is bacteria of the genus

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Actinomyces belong to the Actinobacteria phylum and Actinomycetales order and are related to other genera such as Corynebacterium, Mycobacterium, Nocardia, and Propionibacterium. More than 30 species of Actinomyces have been described. Actinomyces israelii is the most prevalent species isolated in human infections and is found in most clinical forms of actinomycosis. Thus, A. israelii and A. gerencseriae are responsible for about 70% of orocervicofacial infections. [4]

Most bacterial infections are confined to one part of the body because the bacteria are unable to penetrate the body's tissue. However, actinomycosis is unusual in that the infection is able to move slowly but steadily and develop almost anywhere inside the tissue of the human body. In most cases, the bacteria live harmlessly on the lining of the mouth, throat, digestive system and the vagina (in women). The bacteria only pose a problem if the tissue lining becomes damaged by injury or disease, allowing the bacteria to penetrate deeper into the body. [5] Yellowish sulfur granules are constituted by conglomeration of bacteria trapped in biofilm. [6]

Gram staining of pus and pathology of infected tissue is of great interest for the diagnosis of actinomycosis, as it is usually more sensitive than culture, which remains sterile in more than 50% of cases. [7] Typical microscopic findings include necrosis with filamentous Gram-positive fungal-like pathogens.[6] Recently, a new species, Actinomyces radicidentis, has been isolated from apical periodontitis. Periapical actinomycosis is believed to be a non-resolving periapical lesion associated with actinomycotic infection and has been suggested as a contributing factor in the perpetuation of periapical radiolucencies after root canal treatment. [8]

The severity and rarity of this disease coupled with the fact that it is not an opportunistic infection present itself as an interesting area of study. This case report deal with the occurrence of actinomycosis infection related to gingival enlargement without involving the underlying bone, in less predelicted site with clinical findings similar to chronic inflammatory gingival enlargement. and provides a review of the literature related to actinomycosis affecting gingival tissue.

CASE DETAIL:

A 20 year old female reported with a chief complaint of swollen & bleeding gums since 6 months. On clinical examination gingiva showed diffuse gingival enlargement in the mandibular anteriors irt 31- 42 covering three-fourth crown portion & adjacent teeth showed enlargement covering half of clinical crown length with salty taste in the mouth. Gingiva was erythematous in appearance & slightly indurated & cleft like formation was seen between 32 & 33 region with heavy plaque & calculus

(Fig 1). Localized pus discharge with mild loss of attachment, pocket formation & crestal bone loss were observed in the affected area. On general physical examination, the patient's gait was unaltered, and she was moderately built and nourished. Her medical history and personal history were not significant. Thorough scaling & root planing was done & patient was advised for strict oral hygiene regimen without any medication. Patient recalled after 15 days with very mild resolution of gingival enlargement & inflammation inspite of good oral hygiene (Fig 2). For proper diagnosis & management of case gingivectomy was performed in the enlarged areas & excised tissue was sent for histopathological examination.

HISTOPATHOLOGICAL EXAMINATION:

H & E stained sections of soft tissue revealed the presence of hyperplastic parakeratinized stratified squamous epithelium with underlying highly cellular connective tissue stroma with dense infiltration of mixed inflammatory cells along with blood capillaries that are proliferated, dilated and engorged with blood. The inflammatory component was predominated with neutrophils, lymphocytes and plasma cells. The deeper areas of this granulomatous lesion, revealed an actinomycotic colony containing small clumps of basophilic filamentous structures which represents colonies of bacteria. Therefore overall microscopic features were suggestive of gingival enlargement due to Actinomycotic infection (Fig 3 & 4).

MANAGEMENT:

Before gingivectomy procedure the oral cavity was irrigated with 0.2% chlorhexidine gluconate. Gingivectomy was performed in affected areas with complete excision of lesion, periodontal dressing was given and patient was asked not to disturb it. Based on histopathological findings systemic administration of 500 mg Amoxicillin three times a day for 6 weeks & 400 mg Metronidazole three times a day for 7 days was given to eliminate the other gram negative anaerobic microorganisms & patient was recalled after 7 days (Fig 5). The patient was instructed on and encouraged to engage in good oral hygiene program with periodontal debridement being initiated 7 days after systemic antibiotic usage. The patient was monitored weekly for the first month and then monthly. Healing was uneventful & the patient did not report any recurrence (Fig 6).

DISCUSSION:

Actinomycosis is considered as "the most misdiagnosed disease" even by experienced clinicians and is listed as a "rare disease" by the Office of Rare Disease (ORD) of the National Institute of Health (NIH). [9] Although there are few cases reported with the actinomycotic lesion involving the periodontium. [3,10,11] The present case was reported with diffuse gingival enlargement which was edematous, very erythematous and cleft formation seen between two papillas with mild pus discharge. The case was first clinically thought to be chronic

inflammatory gingival enlargement which did not respond to the non-surgical therapy & thus makes this particular case interesting. Hence there is a need to rule out the possibilities of other granulomatous lesions like Wegener's granulomatous lesion & chronic inflammatory gingival enlargement. The gingival tissue was excised for histopathological examination. When the histopathological section proved to be an actinomycotic lesion, literature and clinical situation was reviewed and a positive history of poor oral hygiene was considered as etiological agent.

The infection most commonly occurs from a few weeks to months after the entry of organism. The pathogenicity of the infection can be explained as ranging from an acute form of rapid onset with pus discharge from multiple sinus tracts to a slowly progressing chronic form which shows indurated fibrosis with little suppuration. [2] As these microorganisms are not virulent, they require a break in the integrity of the mucous membranes and the presence of devitalized tissue to invade deeper structures and cause gingival infection. [1] The present case had poor oral hygiene, inflammation & pocket formation this may be one of the cause of invasion of the microorganisms.

Once Actinomyces spp. have invaded tissues, they develop a chronic granulomatous infection characterized by the formation of tiny clumps, called sulfur granules because of their yellow color. These formations of 0.1–1 mm in diameter, composed of an internal tangle of mycelial fragments and a rosette of peripheral clubs, are stabilized by a protein–polysaccharide complex, which is supposed to provide a resistance mechanism to host defenses by inhibiting phagocytosis. [12-14] The present case could not demonstrate sulfur granules probably the infection was intermittent between acute and chronic forms and no utilization of host calcium phosphate resulting from phosphate activity for tissue inflammation.

Actinomyces infections could be polymicrobial and associated with other bacteria, named "companion microbes", which contribute to initiation and development of infection by inhibiting host defenses or reducing oxygen tension.[4] Actinomyces are often isolated with other normal commensals, such as Aggregatibacter actinomycetemcomitans, Eikenella corrodens, Capnocytophaga, fusobacteria, Bacteroides, staphylococci, streptococci, or Enterobacteriaceae, depending on the site of infection. These companion bacteria appear to act as copathogens that enhance the relatively low invasiveness of actinomycetes. Specifically, they may be responsible for the early manifestations of the infection and for treatment failures. [15]

The most striking feature of the present case was that patient had no complains of pain & no relevant medical history. In a previous report, the majority of cases of actinomycosis were asymptomatic,

with only 18% presenting symptoms such as pain and sensory disturbances (80% and 20% of the symptomatic cases, respectively). Actinomycotic patients have often been afflicted by more than one medical condition. Won et al. have described actinomycosis as an opportunistic infection, suggesting cancer, immunodeficiency, steroids taken over a long period of time, and malnutrition as possible contributing factors. However, most of the actinomycotic patients from the Indian subcontinent have been systemically healthy. [11]

`Drug resistance is not considered a problem in actinomycosis. Indeed, Actinomyces spp. are usually extremely susceptible to beta-lactams, and do not produce beta-lactamase especially penicillin G or amoxicillin. As a consequence, penicillin G or amoxicillin are considered drugs of choice for the treatment of actinomycosis. Third-generation cephalosporins are less frequently used even if they are considered to be active on A. israelii; however, it is important to note that some species are resistant to ceftriaxone. Patients with actinomycosis require prolonged (6- to 12-month) high doses of penicillin G or amoxicillin, but the duration of antimicrobial therapy could likely be reduced (3 months) for patients in whom optimal surgical resection of infected tissues has been performed. Specific preventive measures (reduction of alcohol abuse, dental hygiene) may limit the occurrence of actinomycosis. [4] In this case report the duration of antibiotic therapy was reduced to 6 weeks as complete excision of inflamed gingival was done. Metronidazole was adviced for 7 days to eliminate the gram negative companion anaerobes.

CONCLUSION:

Infection by Actinomyces may initiate complications, which may not be diagnosed correctly unless the tissue is biopsied and the Actinomyces colonies are identified. In the clinical practice of periodontology, tissue is not routinely submitted for histopathologic analysis, and the authors would like to suggest more routine submissions of tissue removed from the oral cavity, especially during the treatment of periodontal disease. Moreover, due to the opportunistic characteristics of the actinomycotic infection, early and adequate differential diagnosis of actinomycosis, prior to attempts at therapy and management steps, is of great importance in the oral cavity because it can prevent the spread of the disease.

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FIGURES:

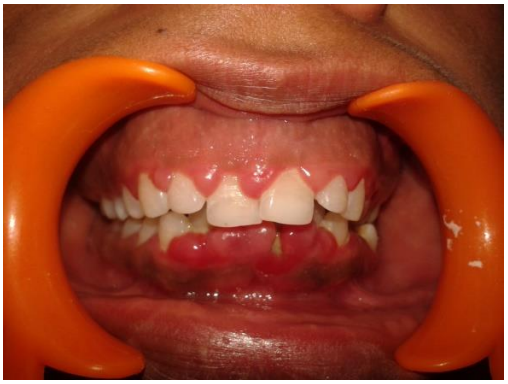

Fig 1: Patient with massive gingival enlargement irt mandibular anteriors

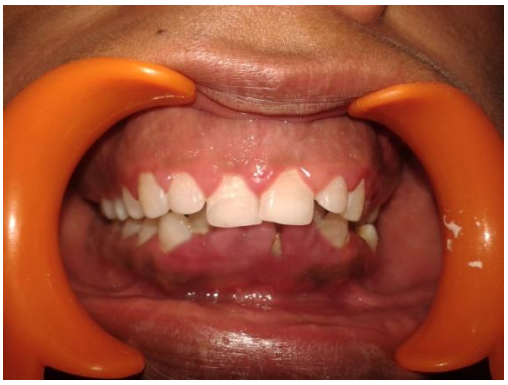

Fig 2: Mild resolution of inflammation after scaling and root planing.

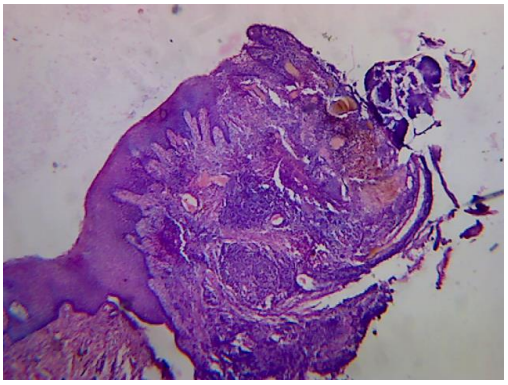

Fig 3: Photomicrograph under 10X magnification shows highly inflamed gingival tissue with deeper areas containing colonies of filamentous bacteria (thick arrow).

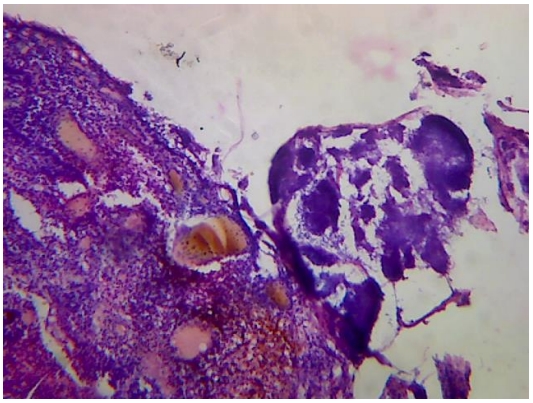

Fig 4: Photomicrograph under 40X magnifications shows colonies of filamentous bacteria (thick arrow

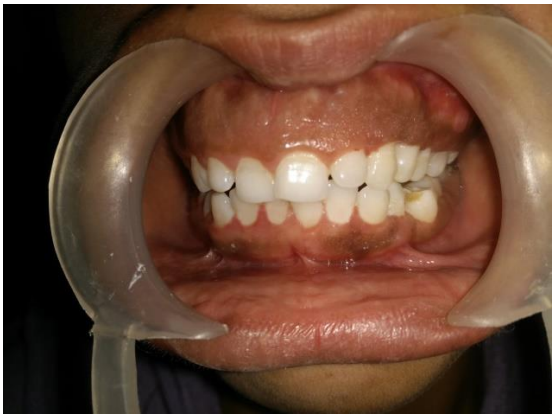

Fig 6: complete remission of the lesion after 6 weeks follow up.

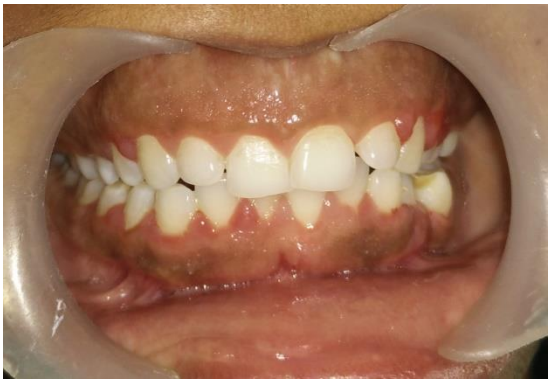

Fig 5: Healing observed after seven days of gingivectomy procedure along with antibiotic coverage. .