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ACUTE NECROTIZING ULCERATIVE GINGIVITIS-A REVIEW

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Abstract: Acute necrotizing ulcerative gingivitis (ANUG) is a non-communicable microbial gingivitis caused by a compromised host immune response. It is distinguished by the presence of "punched-out" crater-like lesions of the papillary gingiva, as well as the onset of sudden inflammation and pain.It is identified by necrosis of the gingival papillae crest, spontaneous bleeding, pain, and halitosis. If untreated, it can spread laterally and apically to involve the entire gingival complex, including the mucosa and alveolar bone, eventually leading to necrotizing ulcerative periodontitis, necrotizing ulcerative stomatitis, and Noma. Predisposing factors include poor oral hygiene, stress, smoking, hormonal imbalance, nutritional deficiencies, and so on.

Key Words: Punched out ulcer, Ulcerative gingivitis, Crater like lesions, Spontaneous bleeding

INTRODUCTION

Acute necrotizing ulcerative gingivitis (ANUG) is a painful type of gingivitis characterised by gingival pain, bleeding, and interproximal papillomacular necrosis. In 1896, Plaut (Barnes et al., 1973)[1] and Vincent described ANUG for the first time. Military historians have documented a condition characterised by painful, bleeding gingival tissues, necrosis, and fetor oris for centuries. This oral disease was not scientifically investigated until the work of Plaut and Vincent in the 1890s. During World War I, it was named as "trench mouth.It has several names, including Vincent's disease and trench fusospirochetal gingivitis .This type of gingivitis is uncommon. Proliferating oral anaerobic bacteria play a role in the development of the disease's clinical signs and symptoms, possibly as opportunistic pathogens. Secondary predisposing etiologic factors such as stress, impaired chemotaxis, poor oral hygiene, alcohol consumption, smoking, general debilitation, and malnutrition have all been studied.

ETIOLOGY

The exact cause of ANUG is unknown, but it is thought to be a polymicrobial infection caused by normal oral cavity commensals. However, when the local resistance of the human gingival area is reduced, the organisms become pathogenic.ANUG is most commonly caused by an opportunistic bacterial infection and is mostly caused by fusiform and spirochete bacteria. Spirochetes and the majority of Gram-negative bacteria, including Bacteroides intermedius and Fusobacterium spp., were identified as the most common causes in one study.[1,2]Another study identified Treponema spp., Selenomonas spp., Fusobacterium spp., and Prevotella intermedia among the microbiota associated with ANUG. [3] Eventually, ANUG is linked to spirochetes and gram-negative bacteria, which can be identified using the gram stain if performed. [4]

EPIDEMIOLOGY

Some centuries ago, ANUG was well known in Europe and North America. ANUG was reported in these Western countries, particularly amon personnel from the military As early as 401BC,Xenophon[5] described a clinical entity that resembledANUG in his soldiers' mouths. Bergeron[6] described a similar disease entity in 1859.Among the French troops with whom he served. a select fewcases reported in the European literature Prior to its association with AIDS, it was most commonly found among military personnel in North America.[7,8] However, because HIV infection is so common, ANUG has become widely recognised as a lesion that is strongly pathognomonic of the infection, especially when seen in otherwise healthy young adult.[9,10]The prevalence of ANUG among HIV-infected patients has been reported to range from 4.3 percent to 16.0 percent.[10-12] In marked contrast, the disease is still frequently seen in developing countries, especially in SubSaharan Africa where it occurs almost exclusively among poor children usually between the ages sof 3 years and 10 years from low socio-economic backgrounds.[13,14,15-19]Similar findings have been reported in India. 22 In Nigeria, hospital-based studies conducted over the last decade indicate that the incidence of ANUG is increasing among children, with a prevalence of up to 23% in children under the age of ten being reported. [13,19,20] In contrast, the disease is still prevalent in developing countries, particularly in Sub-Saharan Africa.

PATHOPHYSIOLOGY

Psychological stress, poor diet, insufficient sleep, alcohol, tobacco, poor oral hygiene, pre-existing gingivitis, and HIV infection are all physiologic factors that contribute to ANUG. These factors have been shown to impair the host immune response, allowing bacteria to spread more easily. Psychological stress decreases gingival microcirculation and salivary flow while increasing adrenocortical secretions, both of which can alter the function of polymorphonuclear leukocytes and lymphocytes. This alters the patient's immune response as well as his or her behaviour and mood, resulting in poor oral hygiene, malnutrition, and increased tobacco consumption.[20] Similarly, a poor diet raises histamine levels and increases gingival capillary permeability, resulting in decreased PMN leukocyte chemotaxis. [20]

CLINICAL FEATURES

While some ANUG signs and symptoms appear to be pathognomonic for the disease, others appear infrequently. Perhaps the most widely used Interproximal necrosis is one of the agreed-upon signs8, with ulcération, pain, and bleeding in the affected area that the "classical symptoms" of Vincent's infection were [21] spontaneous interproximal haemorrhage without gingival redness and [22] inflamed papillae apices that bleeds easily without tenderness. They went on to say that the stereotypical CLINICAL description of interproximal destruction in Vincent's infection is not an unavoidable symptom [23].Schluger [24], on the other hand, provided the most widely recognised and accepted description of the pathognomonic signs of ANUG in 1943: This finding is supported by Barnes et al.8's large study (218 cases), in which gingival bleeding and interdental blunting or cratering were found to be the most commonly associated signs. Suzuki et al.[25]recently reported that all 35 patients they examined had interproximal cratering of the gingival papillae, 97 percent had foetid odour, 85 percent had pseudomembranous formation, and 76 percent complained of bleeding gums.

MANAGEMENT

Initially, treatment options for ANUG were nearly as numerous and diverse as its synonyms, but they all focused on reducing the bacterial flora. Vincent,In 1898, he described a treatment that included local iodine application and gargles with boric acid solution.consisting of potassium permanganate solution rinses, iodine tincture locally applied, and hydrogen peroxidePeroxide rinses and thorough mechanical debridement are both recommended.followed by the application of silver nitrate to the periodontal ligament sulci. Hirschfeld proposed treating gingival inflammation with frequent sodium peroborate rinses, thorough debridement, and no toothbrushing until gingival inflammation was reduced the following year. [23]Schluger[26]describes a streamlined treatment that consists of thorough, deep curettage followed by frequent rinses of diluted hydrogen peroxide or even plain water, primarily as a lavage.Fitch and colleagues report that immediate ultrasonic instrument debridement was highly effective in the treatment of ANUG, with rapid symptom relief and "remarkable tissue response." Goldhaber and Giddon12 agree with this approach to therapy, but they also advocate for the use of antibiotics, specifically penicillin, in the treatment of advanced cases. Gingivoplasty is thought to be important in preventing disease recurrence by removing the residual soft tissue craters.[27,26] Recent English dental literature supports the use of antibiotics in the treatment of ANUG, and researchers found metronidazole to be as effective as penicillin in causing remission of ANUG clinical symptoms in double-blind clinical studies. This is consistent with the findings of Loesche and colleagues, who reported that metronidazole treatment resulted in the immediate resolution of clinical symptoms. Clinical status improved in tandem with a decrease in the proportions of bacterial species associated with the disease.[28,29]

CONCLUSION

Over the years, there have been almost as many different ways to treat ANUG as there have been synonyms, but they all revolve around reducing the bacteria flora. The usage of the use of antibiotics in the treatment of ANUG has increased. It has been strongly urged. Metrondazole is a drug that is used to treat. It's also been discovered that it's just as effective as penicillin in treating infections.resulting in clinical symptom remission, and this occurred in tandem with a decline in overall of women in the workforce. The highly preventable ANUG, on the other hand, entails putting in place measures to combat malnutrition, improve oral hygiene, and improve overall health.minimising oral mucosa damage as well as keeping the oral environment free of contamination Bacteroidacease, particularly F. necrophorum, has a heavy load. Ulcerations of the oral mucosa and Traumatic lesions, such as traumatic tooth eruption, should be considered as having the potential to develop into ANUG. Furthermore, the prevention of water contamination due to faeces and weaning foods Another way to avoid this is to improve your nutritional status and practise good oral hygiene. This is a disease that is highly preventable.a list of bacteria species linked to the disease

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