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Abstract: Necrotizing lesions of the periodontium are considered to be unique in their clinical presentation and course and are found to have a low frequency of occurrence. A triad of pain, ulceration and bleeding are reported among individuals presenting with the same. This case report presents a necrotising lesion of the gingiva of lower anteriors in a systematically healthy adult. Of the many procedures advocated to manage necrotizing ulcerative gingivitis (NUG), the use of hydrogen peroxide mouth rinse, antibiotics and oral prophylaxis has provided a direct solution.

Key words: Antibiotics, Mechanical debridement, Necrotizing ulcerative gingivitis, Necrotising ulcerative periodontitis, Vincent’s disease

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I. Introduction
Necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP), classified as necrotizing periodontal diseases in AAP 1999 International Workshop for classification of periodontal diseases and conditions[1] are the most severe inflammatory periodontal disease caused by plaque bacteria. Vincent in 1896 had described an ulcerative lesion of the gingiva caused by fusiform bacilli and spirochetal organism, which came to be known as Vincent’s disease. A myriad of terms have been used to describe this condition of which fusospirochetal gingivitis, trench mouth, acute ulcerative gingivitis, acute ulcerative necrotizing gingivitis are a few to name.[1-3] The disease has an acute clinical presentation[1] with characteristic triad of gingival pain, interdental ulceration and bleeding. The onset of NUG is associated with increased emotional stress, increased physical demand and decreased nutrient intake.[2-4] The disease primarily affects the interdental and marginal soft tissue with little osseous involvement. At times, it might superimpose on preexisting periodontitis and complicate the diagnosis.[5]

Punched out or crateriform ulceration of interdental papilla covered with psuedomembrane and surrounded by erythematous borders are pathognomonic.[9] NUG and NUP represents different stage of the same infection and are believed to be associated with reduced systemic resistance to bacterial infection.[10-12]

Prompt resolution can be observed with removal of bacterial challenge[5,11,13] and evidence support that regeneration of involved site do occur with conservative treatment.[14] Periodic scaling, root planning and antimicrobial rinses helps in halting the disease progression and bring about regeneration of the necrotic area.

2.1CLINICAL PRESENTATION
36 year old male patient, reported to department of Periodontics, Government Dental College, Kottayam with complaint of pain and decaying lower gums. He also complained of pronounced bleeding while brushing, blood tinged saliva and bad taste in mouth since last three days. A comprehensive medical and dental history was taken and was found to be non contributory. He was referred for a routine blood examination and retroviral tests to eliminate possibility of any systemic illness and immunodeficiency disease. No unusual infection or abnormality in systemic function was detected from the evaluations made. Personal history revealed no deleterious oral habits. However sleep deficiency due to recent stressful life event was reported. There were no associated symptoms of fever, lassitude or malaise.

Clinical examination revealed ulcerated lesion with respect to labial and lingual aspect of lower anteriors, with marked severity seen lingually. Blunting of interdental tissues was noted. Punched out lesion involving the lingual papilla with respect to tooth 33 to 41 was seen with a central yellow grey area demarcated from remaining of gingival mucosa by pronounced erythema. Associated enlargement and tenderness of submental lymph node was obtained on palpation.

2.2 TREATMENT

2.2.1 First visit:
On first visit hydrogen peroxide irrigation of the site was performed. Gentle swabbing of the area with cotton pellet soaked with dilute hydrogen peroxide was done to remove the non attached surface debris (Fig 1). Supragingival scaling was delayed due to marked discomfort of the involved area. Patient was prescribed metronidazole 500mg for 7 days and was advised to rinse with glassful of hydrogen peroxide (3%) mixed with warm water, every two hours. Analgesics (paracetamol 500mg) was advised as and when necessary. Plaque control instructions were given.

2.2.2 Second visit:
Patient when reviewed on third day expressed relief of symptoms. His condition had improved with marked reduction in pain and tenderness. Bulk and redness of ulcerated margins had reduced. Previously sloughed areas showed signs of reepithelisation (Fig 2). Mechanical debridements with ultrasonic instruments were performed and irrigation with dilute hydrogen peroxide was repeated. From there on, patient was advised to discontinue hydrogen peroxide rinse and was prescribed chlorhexidine mouth wash (0.12% chlorhexidine mouth wash 10 ml bd × 2 weeks).

2.2.3 Third visit:
Scaling and root planning was repeated when patient reported on 7th post operative day. Tissue had restored to their normal tone (Fig 3). Plaque control instructions were reinforced.

2.2.4 Subsequent visit/ Follow up
Restoration of normal gingival contour and colour was noted on subsequent visits. There was also restoration of normal consistency and surface texture. Coverage of previously exposed root surfaces was observed at 6 week post operatively (Fig 4). The entire sequence of therapy is outlined in Fig.5.

III. Discussion

ANUG is a necrotizing painful condition that is seen in young adult males commonly affecting maxillary and mandibular anterior teeth. They are characterised by ulcerated and necrotic interdental papilla and gingival margins resulting in punched out appearance.[1]

A series of factors predispose the individual to develop this infection including psychological stress, immune suppression, malnutrition,[15-17] smoking,[18] trauma and preexisting gingivitis.[5, 19] There exists a positive correlation between stress and onset of ANUG as the former involves a down regulation of cellular immune response.[20, 21]

During periods of emotional stress, oral hygiene measures may decrease, nutrition becomes inadequate and tendency to smoke increases. Stressful life events thus activate hypothalamic pituitary adrenal axis that increase serum and urine cortisol.[22] Increased cortisol can depress functions of PMN and affect progression of NUG lesions.[23-25]

Microbial plaque control by mechanical debridement[3, 26], adjunctive antibiotics[13, 14] or both[2, 10] serves as the main stay in management of these lesions. Plaque control though considered simple, offers better results in ANUG patients. While calculus may act as a mechanical irritant to the gingival tissue, it is more likely that its presence on teeth decreases the patient’s ability to remove bacterial plaque.[27]

Antibiotics also have a role to play in the remission of disease. Treatment with metronidazole has been suggested in management of NUG as they effectively reduce Treponema species, Prevotella intermedia and fusobacterium.[13]

IV. Figures

Fig 1: Labial and Lingual gingival changes on initial presentation

Fig 2: Labial and Lingual gingival changes on second visit (day 3)

Fig 3: Labial and Lingual gingival changes on third visit (day 7)

Fig 4: Labial and Lingual gingival changes 6 week post operative.

Fig 5: Sequence of therapy

- **FIRST VISIT**
  - Blood examination
  - Swabbing and irrigation with hydrogen peroxide
  - Analgesics and antibiotics

- **SECOND VISIT**
  - Mechanical debridement
  - Hydrogen peroxide rinse changed to chlorhexidine mouth wash
  - Plaque control reinforced

- **THIRD VISIT**
  - Mechanical debridement repeated
  - Plaque control reinforced

- **FOLLOW UP**
  - Plaque control reinforced
V. Conclusion

As with other plaque associated periodontal disease, opportunistic bacteria are the primary etiologic agents in NUG. It is established that clinical signs and symptoms of NUG resolves in few days after adequate treatment and control of biofilm. The prompt diagnosis and treatment of the lesion is regarded crucial to prevent further progression of disease.

References