CASE REPORT

Conservative Management of Acute Necrotizing Ulcerative Gingivitis in Lactating Female

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Abstract
Acute necrotizing ulcerative gingivitis (ANUG) is described as “a rapidly destructive, non communicable, gingival infection of complex etiology”. It is characterized by necrosis of the crest of the gingival papillae, spontaneous bleeding, pain and halitosis. If it is left untreated, it may spread laterally and apically to involve the entire gingival complex, including the mucosa and alveolar bone, leading to necrotizing ulcerative periodontitis to necrotizing ulcerative stomatitis and finally noma. There are various predisposing factors like poor oral hygiene, stress, smoking, hormonal imbalance, nutritional deficiencies etc. This case report describes the conservative management of ANUG in lactating patient and probable mechanism of pathogenesis of predisposing factors involved.

KEY WORDS: Acute necrotizing ulcerative gingivitis, lactating female

Introduction
Acute necrotizing ulcerative gingivitis (ANUG) is a severe and painful form of gingivitis characterized by gingival pain, bleeding and necrosis of the interproximal papillae (Schluger, 1943). It has been called by many names like Vincent’s disease, trench mouth, and fusospirochetal gingivitis. This form of gingivitis is relatively rare. Proliferating oral anaerobic bacteria are involved in the development of the clinical signs and symptoms of the disease, possibly as opportunistic pathogens (Loesche et al., 1982). Primary risk factors for disease include smoking, psychological stress, poor nutrition. Defects in leukocytes and immune function may also be associated with disease occurrence (Cogén et al., 1983, Courtois et al., 1983)

Case Report
A 23 yr old female patient named Mubeena reported to the periodontology OPD of DR Z.A. Dental college and hospital with chief complaint of severe pain and bleeding gums with difficulty in eating food since one week. She also complained of bad breath. Patient was lactating mother and feeding her child for 7 months. She used to clean her teeth with finger.
On extra oral examination, there was no gross facial asymmetry detected, lips were competent, bilateral submandibular lymphnodes were tender on palpation and local rise in temperature was detected.
Patient gave the history of elevated temperature for 1 week. On intraoral examination poor oral hygiene was noticed with plaque and calculus deposition. There was swollen marginal gingiva and interdental papilla with rounded contour and also necrosis of the interdental papillae, causing it to separate into one facial and one lingual portion (Fig 1, 3, 4). Bleeding was present on slight stimulation of gums. There was traumatic bite in anterior teeth. Intraoral periapical radiograph revealed bone loss in lower anterior teeth (Fig 2).

In the first visit after thorough examination, only conservative treatment like removal of local factors and maintenance of oral hygiene was planned. Trauma relieved in anterior teeth by selective grinding. Supragingival scaling was attempted as thoroughly as the condition allowed. Patient was advised to take adequate rest, proper diet and maintain proper oral hygiene. She was prescribed amoxicillin 500 mg every 6 hours for 5 days and local application of gel containing metronidazole three-four times a day. She was also instructed to rinse with 3% H₂O₂ & sterile warm water (1:1) four times a day and also with 0.12% chlorhexidine rinses to maintain oral hygiene as she was unable to clean her teeth with brush. Patient recalled on second day and again supragingival scaling was done. After 3 days the patient was re-evaluated and scaling and curettege was performed. After 7 days patient was almost symptom free so thorough scaling & root planing was done. 3% H₂O₂ rinses were now discontinued but 0.12% chlorhexidine rinses continued. Patient was re-evaluated after one month & a good response was found in the form of healing of necrotic areas and reduction in the size of gingival craters (Fig 5, 6, 7). Patient was kept on maintenance with instructions of oral hygiene and proper nutrition.

Discussion
An eminent Bio statistician was contacted. Necrotizing ulcerative gingivitis is an inflammatory destructive disease of the gingiva, which presents characteristic signs and symptoms. This disease entity was present as early as 400BC in Greek soldiers, but it was first described by Plaut in 1894 and Vincent in 1896. It is caused mainly by Fusiform bacilli and Spirochetes. Characteristic lesions are punched out, crater like depressions of the interdental papillae. These lesions extend to marginal gingiva and rarely to attached gingiva and oral mucosa. The surface is covered by a pseudomembranous slough, demarcated from the remainder of the gingival mucosa by a pronounced linear erythema. In some instances, the lesions are denuded of the surface pseudomembrane, exposing the gingival margin, which is red, shiny, and hemorrhagic. Spontaneous gingival hemorrhage or pronounced bleeding may occur on the slightest stimulation. Other signs often found are fetid odor and increased salivation. NUG can occur in otherwise disease free mouths or can be superimposed on chronic gingivitis or periodontal pockets but does not lead to pocket formation because necrotic changes involve junctional epithelium and a pocket deepening requires viable junctional epithelium.

NUG can cause tissue destruction involving the supporting structures. It usually runs an acute course and therefore the term acute is often included in the diagnosis. When bone loss occurs the condition is called necrotizing ulcerative periodontitis. If left untreated infection reaches into systemic circulation, depicted as in following flow chart:
In the present case report we have discussed the pre-treatment and post treatment clinical picture of ANUG and its management in lactating patient. The local debridement and proper oral hygiene practice with antibiotic coverage has healed the lesion considerably. Patient was relieved from pain, swollen gingiva and fetid odor completely with this initial approach. This conservative mode of treatment is reliable method for treating ANUG. The main predisposing factors in our case were stress probably because she was nursing her child. Maestripieri D et al found in their study on female rhesus macaques that lactating females had significantly higher plasma cortisol levels than nonlactating females.1 Wendy Saltzman et al reported that in several mammalian species, hypothalamic-pituitary-adrenal (HPA) and behavioral responses to stressors were down-regulated in lactating females, possibly preventing stress-induced disruptions of maternal care.2 Early reports have shown a positive correlation between acute necrotizing ulcerative gingivitis and psychological stress suggesting psychosomatic effects on the periodontium, including endocrine dysfunction, lowered resistance to infection and changes in diet, personal oral hygiene and parafunctional habits.

Increased adrenocortical activity, which occurs in response to emotional stress lead to altered cytokine profiles3 that affect the recruitment of cells, such as macrophages and fibroblasts ultimately causing the reduction in host immune response to periodontal pathogens4 alters host immune responses5 and results in an inability to control the indigenous bacteria. Stress also causes reduction of tissue matrix metalloproteinase levels, which leads to impaired tissue turnover.6 Moreover, altered Th1/Th2 (Helper T cell-1/Helper T cell-2) ratio, may lead to an increased susceptibility to periodontal disease.7

In our case plaque and calculus initiated the gingival and periodontal disease and then transiently suppressed immune system aggravated the condition. Traumatic bite and previous periodontal pocket are also contributing factors for ANUG and both were present in anterior teeth of patient. Though patient was feeding her child, metronidazole was not advised to take orally as it should be avoided in lactating patient.

**Prescribing Drugs to Lactating Patients**

The rate of passage of a drug from plasma to milk is an important determinant of the concentration of the drug in milk. Mechanisms of excretion of drugs in breast milk include both passive diffusion and carrier-mediated transport.

The amount of a drug excreted in breast milk depends on the characteristics of the drug, such as the **drug's molecular weight, lipid solubility, pKa, and plasma protein binding**.

The pKa of weak electrolytes is an important determinant of drug concentration in milk, because the pH of milk is generally lower (more acidic) than that of plasma, and milk can act as an "ion trap" for weak bases. At equilibrium, basic drugs may be more concentrated in milk relative to plasma. Conversely, acidic drugs are limited in their ability to enter milk, because the concentration of nonionized free form in milk is generally higher than in plasma, and a net transfer of the drug from milk to plasma occurs.

In **Table1** simple classification of drugs given which is adapted from “Breastfeeding counselling: A training course”, WHO/CDR/93.3-6.

**References**


Table 1: **Breastfeeding and Mother’s Medication**

<table>
<thead>
<tr>
<th>Breastfeeding contraindicated:</th>
<th>Anticancer drugs ( antimetabolites): Radioactive substances ( stop breastfeeding temporarily)</th>
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<tr>
<td><strong>Continue breastfeeding:</strong></td>
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<tr>
<td>Side-effects possible</td>
<td>Selected psychiatric drugs and anticonvulsants</td>
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<tr>
<td>Monitor baby for drowsiness</td>
<td>Chloramphenicol, tetracyclines, <strong>metronidazole</strong>, quinolone antibiotics (e.g. ciprofloxacin)</td>
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<tr>
<td>Use alternative drug if possible</td>
<td>Sulfonamides, dapsone, sulfamethoxazole+trimethoprim (cotrimoxazole) sulfadoxine+pyrimethamine (fansidar)</td>
</tr>
<tr>
<td>Monitor baby for jaundice.</td>
<td>Estrogens, including estrogen-containing contraceptives, thiazide diuretics, ergometrine</td>
</tr>
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<td>Use alternative drug (may inhibit lactation)</td>
<td>Most commonly used drugs: analgesics and antipyretics: short courses of paracetamol, acetylsalicylic acid, ibuprofen; occasional doses of morphine and pethidine, antibiotics: ampicillin, <strong>amoxicillin</strong>, cloxacillin and other penicillins, erythromycin, antituberculosis drugs, anti-leprosy drugs (see dapsone above), antimalarials (except mefloquine, Fansidar), antihelmintics, antifungals, bronchodilators (e.g. salbutamol), corticosteroids, antihistamines, antacids, drugs for diabetes, most antihypertensives, digoxin nutritional supplements of iodine, iron, vitamins.</td>
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<th>Safe in usual dosage</th>
<th>Monitor baby</th>
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