

Ulcerative Necrotic Gingivitis and Modern Treatment Methods

¹ Xudaykulov Kurbon; ² Ikromova Nozima; ³ Azimov Adxam; ⁴ Ergashev Ahmadjon;

⁵ Xaydarova Durdona Munisovna

^{1, 2, 3, 4} Students of the Faculty of Dentistry, Samarkand State Medical University

⁵ Scientific supervisor: Department of Therapeutic Dentistry, Samarkand State Medical University

Received: 2025, 15, Jan
Accepted: 2025, 21, Feb
Published: 2025, 08, Mar

Copyright © 2025 by author(s) and BioScience Academic Publishing. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).



Open Access

<http://creativecommons.org/licenses/by/4.0/>

Annotation: Acute necrotizing ulcerative gingivitis is a painful infection of the gums. Symptoms include severe pain, bleeding, and bad breath. Diagnosis is based on clinical presentation. Treatment consists of gentle brushing, improved oral hygiene, mouthwash, supportive care, and antibiotics if brushing is delayed.

Keywords: Clinical manifestations, Diagnostics, Treatment, Prevention.

Introduction: Acute necrotizing ulcerative gingivitis (ANUG) is most common in smokers and patients who are weakened by stress. Other risk factors include poor oral hygiene, malnutrition, immunodeficiency (e.g., HIV/AIDS, use of immunosuppressants), and sleep deprivation. Some patients also have oral candidiasis. The most common flora causing ANUG include the genera Treponema, Selenomonas, Melanogenic Bacteroides, and Fusobacteria (1).

Signs and symptoms of acute necrotizing ulcerative gingivitis (ANUG)

- Typically, the onset is sudden and may be accompanied by malaise or fever. The main manifestations are:
- Acutely painful, bleeding gums
- Excessive salivation
- Sometimes a very unpleasant odor from the mouth (halitosis)

Pathognomonic lesions are located on the dental papillae and the marginal margin of the gingiva (adjacent to the teeth). These lesions have the characteristic appearance of a puncture wound and are covered with a gray pseudomembrane. Similar lesions are rare on the buccal mucosa and tonsils. Swallowing and speaking may be painful. Regional lymphadenopathy is often present.

This rapidly progressing infection is characterized by acute, erythematous gingivitis, soft tissue necrosis with the formation of a necrotic superficial layer (called a pseudomembrane, best seen along the lower anterior gingival line), and cratering of the interdental papillae, known as "perforated suckers."

Image provided by Craig Fowler, DDS.

Often, ANH can manifest itself without a noticeable odor, and it can also manifest as a local pathology.

Rarely, the tonsils or pharyngeal tissue are affected; in this case, if the disease manifestations do not respond quickly to conventional therapy, infection due to diphtheria and agranulocytosis should be excluded using throat cultures and a complete blood count.

Treatment of ANUG

- a. Sanitizing the wound cavity
- b. Rinse (e.g., hydrogen peroxide, chlorhexidine)
- c. Improved oral hygiene
- d. Sometimes antibiotics are taken orally

Treatment of acute ulcerative necrotic gingivitis consists of careful necrotization using a hand scaler or ultrasound. Surgical treatment of the wound is carried out over several days. The patient should use a soft toothbrush or washcloth to clean the teeth.

In the first few days after the initial cleaning, rinsing with warm salt water every hour or 2 times a day with 1.5% hydrogen peroxide or 0.12% chlorhexidine may help.

Basic supportive measures include improving oral hygiene (primarily with caution), eating well, drinking plenty of fluids, resting, taking painkillers as needed, and avoiding irritants (such as smoking or hot or spicy foods). Noticeable improvement usually occurs within 24 to 48 hours, after which surgical debridement can be completed.

Research object and materials : If cleaning is delayed (e.g., dentist or cleaning equipment not available), oral antibiotics effective against normal oral flora (e.g., metronidazole 500 mg every 8 hours or amoxicillin 500 mg every 8 hours) may help provide symptomatic relief and may be continued for an additional 72 hours after symptoms resolve. If patients have a severe penicillin allergy, other antibiotics should be used (e.g., clindamycin 300 mg every 6 hours, erythromycin 250 mg every 6 hours, or tetracycline 250 mg every 6 hours).

Treatment of oral candidiasis is described in another section.

If the gingival contour is inverted (i.e., the papillae tips are lost) during the acute phase, surgery may be necessary to prevent further periodontitis.

Hyperplasia is characterized by diffuse, relatively avascular smooth or nodular enlargement that almost completely covers a portion of the teeth. Often, the hypertrophied tissue can be removed. If possible, replace the medication causing the unwanted effect. Careful oral hygiene can reduce the likelihood of recurrence.

Squamous cell carcinoma of the oral mucosa can also occur in the gums, causing significant enlargement of the gum tissue.

Gingivitis is a type of periodontal disease that causes inflammation of the gums, swelling, redness, exudate, changes in normal contour, and sometimes bleeding with discomfort. Diagnosis is made based on a clinical examination. Treatment includes professional teeth cleaning and improved oral hygiene at home. In advanced cases, antibiotics or surgery may be required.

Normally, the gums are hard, firmly attached to the teeth and clearly define them, forming

interdental spaces. Keratinized gums near the crowns appear as pink and rough tissue. This tissue should fill the entire space between the crowns. The gingival areas located further from the crowns are called the alveolar mucosa and are non-keratinized, highly vascular, red, mobile, and continuous with the buccal mucosa. Normal gums should not bleed or pus when pressed with a spatula.

Inflammation or gingivitis, the most common pathology of the gums, can develop into periodontitis.

Caused by plaque buildup (usually due to poor oral hygiene)

Almost all gingivitis is caused by plaque. Mineralized plaque is a mixture of bacteria, food debris, saliva, and mucus with calcium salts and phosphate salts. Poor oral hygiene allows plaque to form between the gums and teeth; these. Gingivitis does not develop in areas where teeth are missing. Plaque irritation deepens the normal space (groove) between the tooth and the gum, forming gum pockets. These pockets contain bacteria that cause gingivitis and root caries. Other local factors, such as improper occlusion, tartar, food retention, poor tooth restoration, and dry mouth, play a secondary role.

Plaque-induced gingivitis can be triggered or worsened by hormonal changes, systemic diseases, medications, or nutritional deficiencies.

Hormonal changes that occur during puberty, menstruation and pregnancy, menopause, or taking oral (or injectable) contraceptives can increase inflammation.

Systemic diseases (eg, diabetes, HIV/AIDS, vitamin deficiencies, leukemia, leukopenia) can affect the response to infection. Some patients with Crohn's disease develop areas of granulomatous hypertrophy of the gums that have a rocky consistency as the intestinal stricture progresses.

Medications such as cyclosporine and nifedipine, as well as niacin (which causes pellagra) or severe vitamin C deficiency (rare in the United States), can cause gingivitis.

Exposure to heavy metals (e.g., lead, bismuth) can cause gingivitis and a black line on the gums.

Non-plaque gingivitis occurs in a small percentage of people. Causes include bacterial, viral, and fungal infections, allergic reactions, trauma, mucosal diseases (e.g., lichen planus, pemphigus), and hereditary diseases (e.g., hereditary gingival fibromatosis).

Simple gingivitis first causes a deepening of the groove between the tooth and the gum, followed by the formation of red, inflamed gums around one or more teeth, swelling of the interdental papillae, and easy bleeding. Pain is usually absent. The inflammation may subside, remain superficial for many years, or in some cases progress to periodontal disease.

Pericoronitis is an acute, painful inflammation of the gingival flap (operculum) over a partially erupted tooth, usually around the lower third molars (wisdom teeth). Infection is common and abscesses or cellulitis may develop. Pericoronitis often recurs when food gets stuck under the gingival flap and can damage the opposing upper third molar. The gingival flap disappears after the tooth is completely erupted. Most wisdom teeth do not erupt and are called impacted.

Desquamative gingivitis may occur during menopause. It is characterized by dark red, painful gum tissue that bleeds easily. Vesicles may precede desquamation. The gums are soft and lack keratinized cells that resist friction with food particles. Similar gingival involvement may be associated with pemphigus vulgaris, bullous pemphigoid, mucous pemphigoid, or atrophic lichen planus.

During pregnancy, swelling of the interdental papillae in particular may develop. Soft, reddish, pedunculated gingival growths often appear on the interdental papillae during the first trimester, which may persist throughout pregnancy and may resolve or persist after delivery. These growths are purulent granulomas, sometimes called pregnancy tumors. They develop rapidly and then become static. The triggering irritant is common, such as tartar or a rough-edged restoration. These

growths can also occur in nonpregnant women and men.

Uncontrolled diabetes can worsen the irritation of the gums, making secondary infections and acute gum abscesses common.

Results: In leukemia, the gums may appear filled with leukemic infiltrate, presenting with clinical signs of swelling, pain, and easy bleeding. Gingivitis is often an early manifestation of leukemia.

The gums become inflamed, hyperplastic, and bleed easily. Petechiae and bruising may appear throughout the mouth.

In pellagra, the gums become inflamed, bleed easily, and are susceptible to secondary infection. In addition, the lips become red and cracked, the mouth feels as if it has been burned, the tongue is smooth and bright red, and there may be ulcers on the tongue and mucous membrane.

The finding of erythematous, tender tissue at the gum line confirms the diagnosis of gingivitis. To detect gum disease early, some dentists often measure the depth of the pocket around each tooth. A depth of < 3 mm is considered normal; deeper pockets increase the risk of developing gingivitis and periodontitis.

Simple gingivitis is controlled by good oral hygiene with antibacterial mouthwashes. A thorough cleaning should be performed (manual or professional cleaning with ultrasonic instruments). If necessary, poorly fitted dental restorations should be repaired or replaced and local irritants should be removed. Excess gum tissue, if present, can be removed. If possible, medications that cause gingival hyperplasia should be discontinued; if this is not possible, improved home oral care and frequent professional cleanings (at least every 3 months) usually reduce hyperplasia. During pregnancy, swellings are removed.

If severe infection develops, antibiotics may be prescribed the day before removal and continued during healing. The general regimen is 500 mg of amoxicillin orally every 6 hours for 10 days (or until 3 days after all inflammation has resolved). Abscesses associated with pericoronitis require local incision and drainage, periodontal flap and root debridement, or extraction.

Discussion: When gingivitis is caused by systemic disease, treatment is directed at the underlying cause. Sequential administration of estrogens and progestins may be helpful for desquamative gingivitis in the menopausal period, but the adverse effects of this therapy (see "Hormone therapy") limit its recommendation. Otherwise, dentists may prescribe a glucocorticoid rinse or glucocorticoid paste applied directly to the gums. Pemphigus vulgaris gingivitis and related mucocutaneous diseases may require systemic treatment (e.g., corticosteroids).

Daily plaque removal with flossing and brushing and regular cleanings by a dentist or hygienist every 6 months to 1 year can help reduce the risk of developing gingivitis. Patients with systemic diseases that predispose to gingivitis require more frequent professional cleanings (every 2 weeks to 3 months).

Summary: Gingivitis is mainly caused by poor oral hygiene, but is sometimes caused by hormonal changes (e.g., pregnancy, menopause) or certain systemic diseases (e.g., diabetes, HIV/AIDS).

Professional cleaning with or without antibacterial rinse is usually sufficient treatment.

Peri-implant tissue diseases and pathological conditions are associated with soft and hard tissue deficiencies that help predict the long-term success of dental implants. Clinical manifestations of peri-implantitis include erythema, bleeding on probing, swelling, and suppuration.

Clinical signs of peri-implantitis mucosa include bleeding on probing, swelling, and plaque-related inflammation. The condition can be corrected with plaque control measures.

Peri-implantitis is a plaque-related pathological condition characterized by inflammation and subsequent progressive loss of supporting bone. It is associated with inadequate plaque prevention and occurs in patients with a history of severe periodontitis. Treatment is the same as for

periodontitis.

Normal healing after tooth loss can result in a lack of jawbone or dense, keratinized gum tissue. These abnormalities can be corrected by hard or soft tissue implants.

List of used literature:

1. Munisovna X. D. COMPLEX METHODS OF TREATMENT OF CHRONIC PERIODONTITIS //Conferences. – 2023. – С. 36-40.
2. Munisovna K. D. et al. GINGIVITIS IN PEOPLE: FEATURES OF DIAGNOSIS, CLINICAL MANIFESTATIONS AND TREATMENT //ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ. – 2023. – Т. 20. – №. 3. – С. 58-62.
3. Haydarova D., Tilavov X. TREATMENT OF PULP PATHOLOGY IN PATIENTS WITH CHRONIC PERIODONTITIS //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 79-82.
4. Хайдарова Д. ПРИМЕНЕНИЕ СОВРЕМЕННЫХ АНТИСЕПТИКОВ ДЛЯ ПРОФИЛАКТИКЕ В РАЗВИТИЕ ПЕРЕИМПЛАНТИТАХ //Евразийский журнал медицинских и естественных наук. – 2022. – Т. 2. – №. 6. – С. 62-68.
5. ВАЛИЕВА, С. Ш., НАБИЕВ, О. Р., ХАЙДАРОВА, Д. М., ГАППАРОВ, Ж. З. У., & НАСРЕТДИНОВА, М. Т. ВЕСТНИК НАУКИ И ОБРАЗОВАНИЯ. ВЕСТНИК НАУКИ И ОБРАЗОВАНИЯ Учредители: Олимп, 76-81.
6. Asrorovna X. N. et al. Anatomy And Topography of The Tooth //Texas Journal of Medical Science. – 2022. – Т. 4. – С. 1-3.
7. Xolboeva N., Haydarova D. BIOLOGICAL METHODS OF TREATMENT OF PULPITIS //Science and innovation. – 2022. – Т. 1. – №. D8. – С. 73-78.
8. Asrorovna X. N., Munisovna X. D. COMPLEX METHODS OF TREATMENT OF CHRONIC PERIODONTITIS //Journal of Integrated Education and Research. – 2023. – Т. 2. – №. 1. – С. 53-56.
9. Kholboeva N. A., Khaydarova D. M. MECHANICAL TREATMENT AND EXPANSION OF ROOT CANALS WITH CHEMICAL PREPARATIONS (ENDOLUBRICANTS) //Bulletin of Science and Education. – С. 4-1.
10. Munisovna I. R. H. D. et al. TREATMENT OF TEETH DAMAGED BY SURFACE CARIES IN REM-THERAPY MODE //Galaxy International Interdisciplinary Research Journal. – 2023. – Т. 11. – №. 11. – С. 513-515.
11. Холбоева Н. А., Хайдарова Д. М. МЕХАНИЧЕСКАЯ ОБРАБОТКА И РАСШИРЕНИЕ КОРНЕВЫХ КАНАЛОВ ХИМИЧЕСКИМИ ПРЕПАРАТАМИ (ЭНДОЛУБРИКАНТЫ) //Вестник науки и образования. – 2022. – №. 4-1 (124). – С. 88-92.
12. Xolboeva N., Haydarova D. PROVISION OF THERAPEUTIC DENTAL CARE AND PREVENTIVE MEASURES DURING PREGNANCY //Science and innovation. – 2022. – Т. 1. – №. D6. – С. 179-181.
13. Raxmonova B., Haydarova D., Sadikova S. TREATMENT OF FRACTURES OF THE UPPER AND LOWER HEAD IN ELDERLY PATIENTS USING THE IMMOBILIZATION METHOD IMPACT ON PERIODONTAL TISSUE //Science and innovation. – 2023. – Т. 2. – №. D10. – С. 194-198.
14. Farrukh S. ORGANIZATION OF DIGITALIZED MEDICINE AND HEALTH ACADEMY AND ITS SIGNIFICANCE IN MEDICINE //Science and innovation. – 2023. – Т. 2. – №. Special Issue 8. – С. 493-499.

15. Валиева С. Ш. и др. Наша тактика лечения больных с болезнью Меньера //Вестник науки и образования. – 2021. – №. 7-3 (110). – С. 76-81.
16. Xaydarova D., Karimov I. RESULTS OF THE ASSESSMENT OF CHANGES IN MASTICATORY MUSCLE TONE IN RELATION TO THE PATIENT'S BODY POSITION //Science and innovation. – 2023. – Т. 2. – №. D10. – С. 155-157.