Acute gingival diseases

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Acute Gingival Infections

1. Necrotizing Ulcerative Gingivitis.

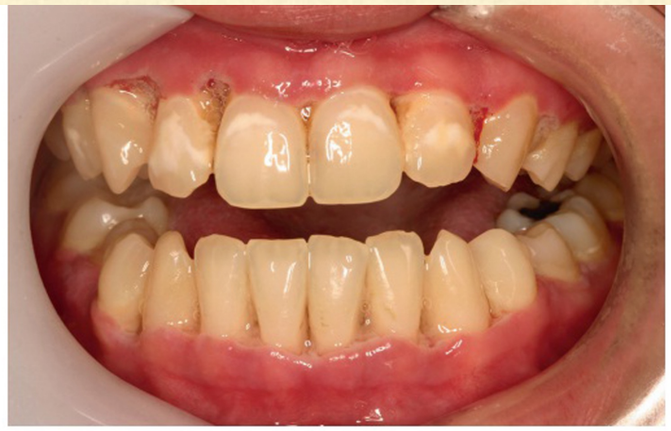


FIGURE 4-13 Necrotizing ulcerative gingivitis with typical punched out, necrotic and ulcerated interdental papillae. (Courtesy of Dr. Hani Mawardi, Boston, MA.)

2. Primary Herpetic Gingivostomatitis .

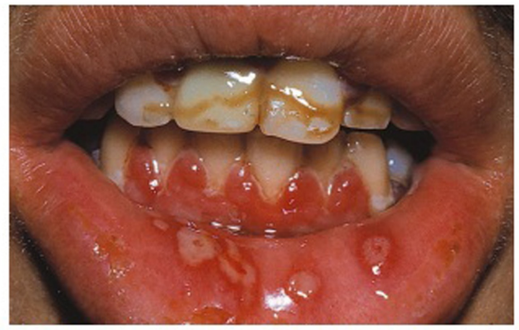
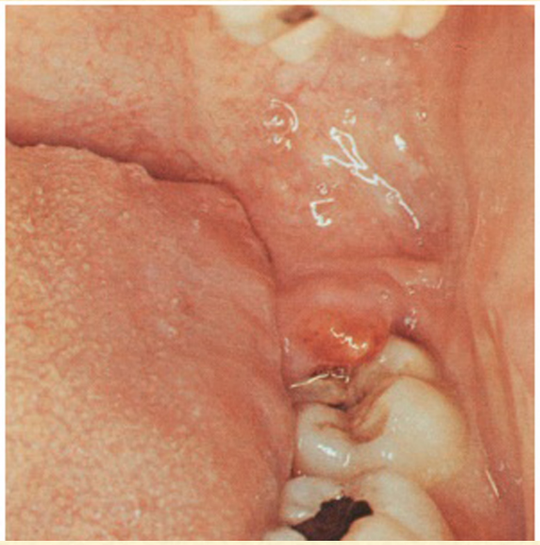


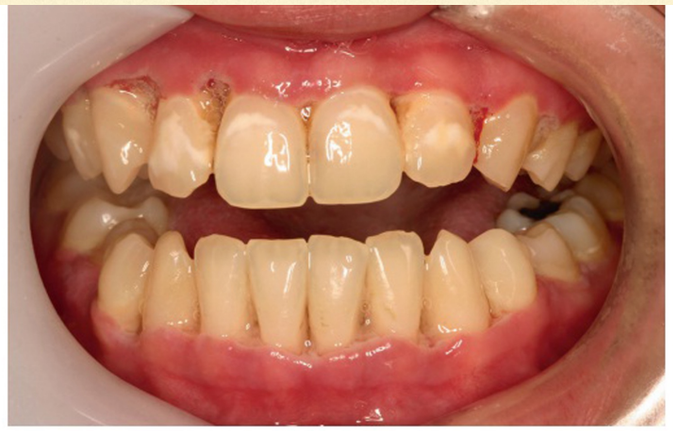
Figure 17-6 Primary herpetic gingivostomatitis in a 12-year-old boy, with diffuse erythematous involvement of the gingiva and a spherical gray vesicle in the lip. *(Courtesy Dr. Heddie Sedano, University of California, Los Angeles, and University of Minnesota.)*

3. Pericoronitis.



Acute gingival infections.

- **Prodrome Findings**…… **fever,** loss of appetite, **malaise,** and myalgia that may also be accompanied by **headache and nausea**. Oral **pain** leads to poor oral intake, and patients may require hospitalization for hydration.



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- Sudden onset.

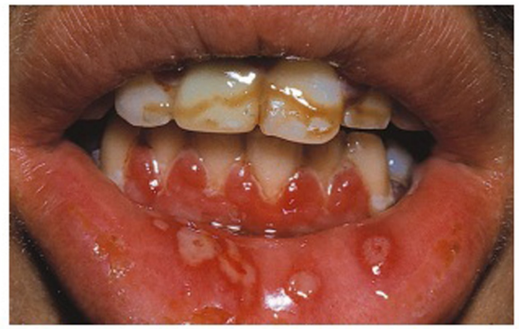
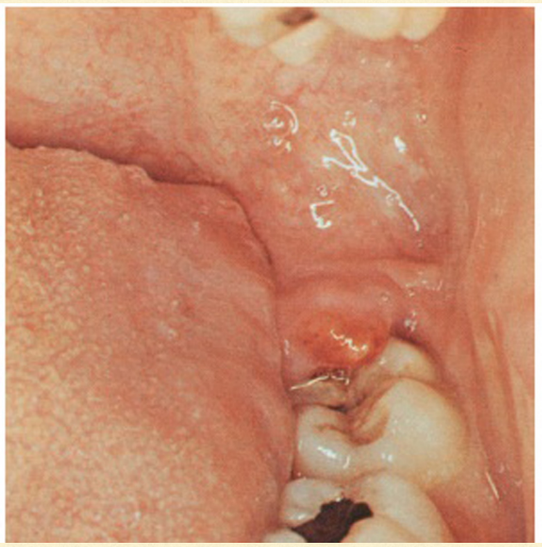


Figure 17-6 Primary herpetic gingivostomatitis in a 12-year-old boy, with diffuse erythematous involvement of the gingiva and a spherical gray vesicle in the lip. *(Courtesy Dr. Heddie Sedano, University of California, Los Angeles, and University of Minnesota.)*

- **Oral Findings** …..Within a few days of the prodrome,



1.Necrotizing Ulcerative Gingivitis:

- **Def:**

- Necrotizing ulcerative gingivitis (NUG) is a **microbial disease** of the gingiva in the context of an **impaired host response.**
- Necrotizing ulcerative gingivitis (NG) results from an impaired host response to a potentially pathogenic microbe.
- It is **characterized** by the **necrosis and sloughing** of gingival tissue, and it presents with characteristic signs and symptoms.

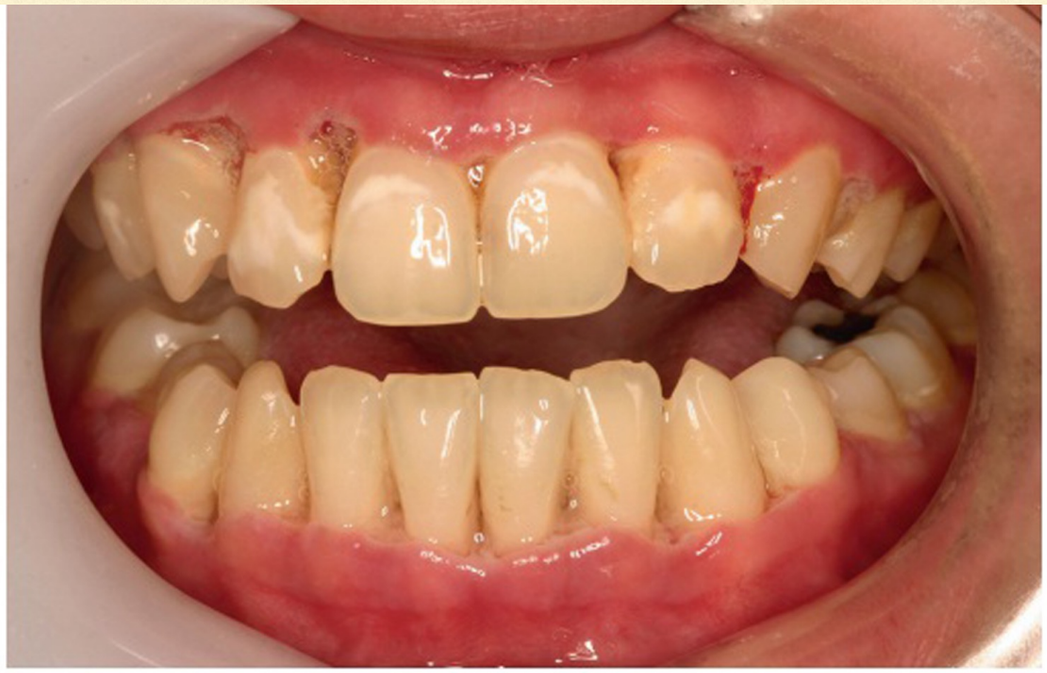


FIGURE 4-13 Necrotizing ulcerative gingivitis with typical punched out, necrotic and ulcerated interdental papillae. (Courtesy of Dr. Hani Mawardi, Boston, MA.)

1.Necrotizing Ulcerative Gingivitis:

- Necrotizing ulcerative gingivitis has been called many names:

1. - Vincent's disease
2. - Trench mouth
3. -Acute necrotizing ulcerative gingivitis
4. - Fusospirochetal gingivitis

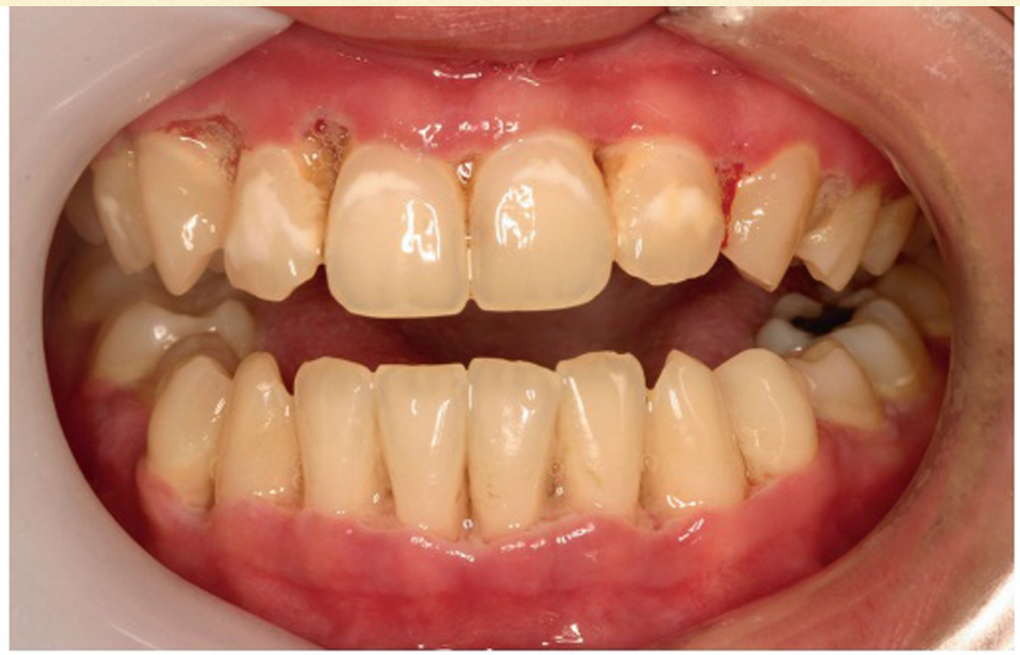
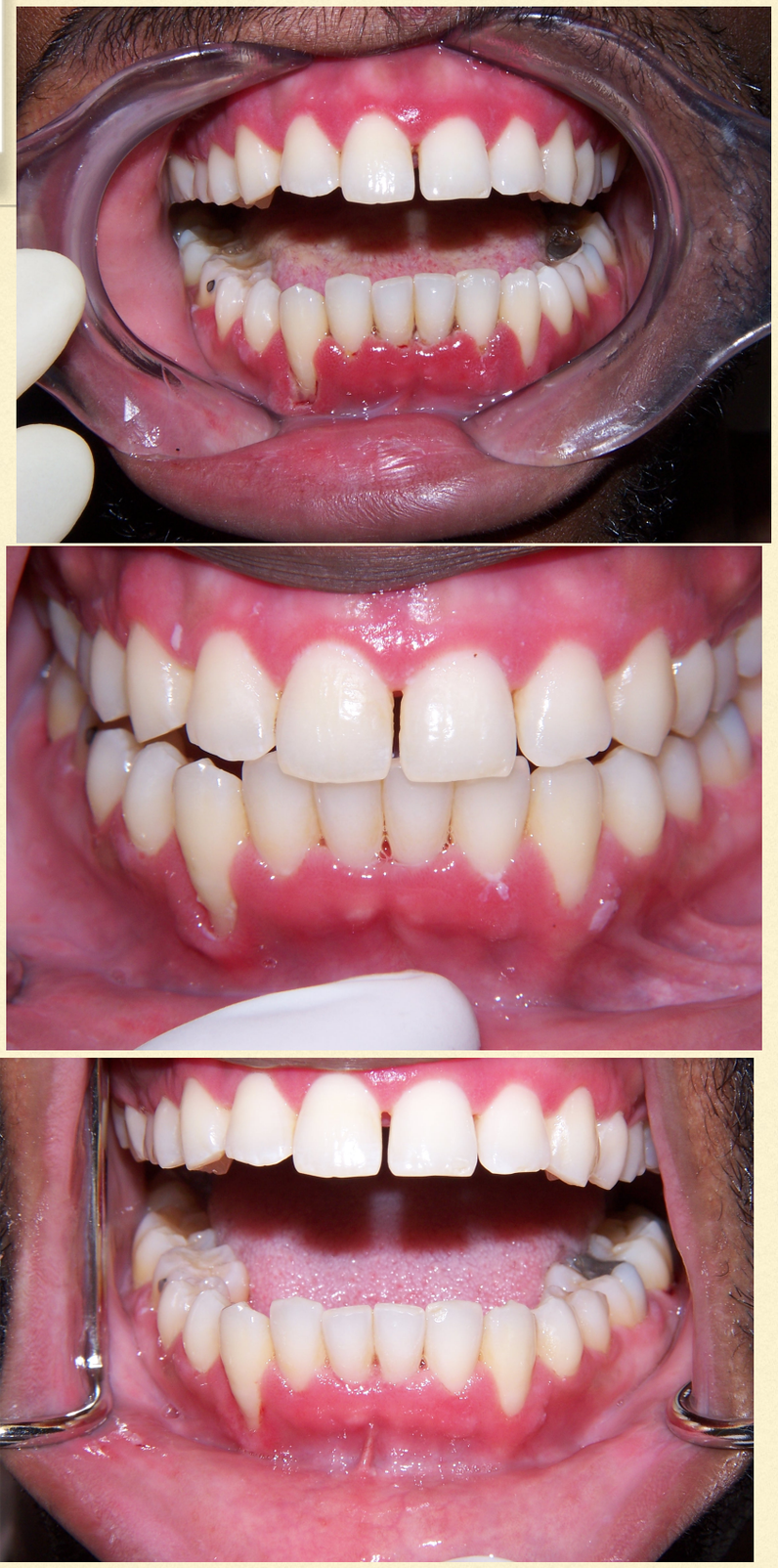


FIGURE 4-13 Necrotizing ulcerative gingivitis with typical punched out, necrotic and ulcerated interdental papillae. (Courtesy of Dr. Hani Mawardi, Boston, MA.)

Clinical Features:

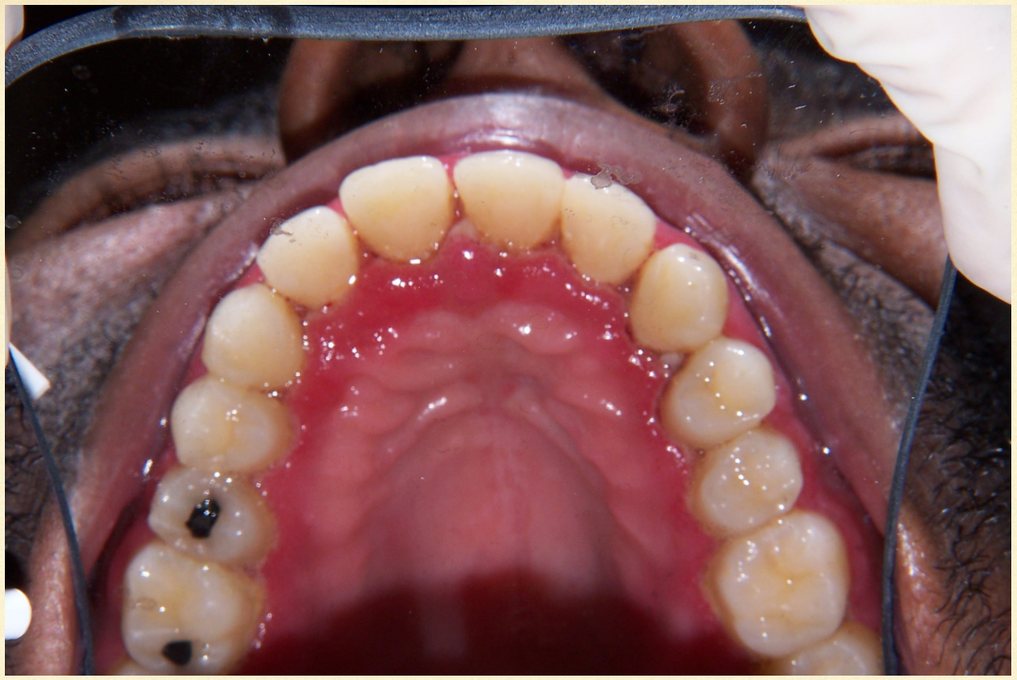
- NUG is usually identified as an **acute disease.** However, the term acute in this case is a clinical descriptor and should not be used as a diagnosis, **because there is no chronic form of the disease**.
- NUG often undergoes a diminution in severity without treatment, thereby leading to a **subacute stage** with milder clinical symptoms. Thus, patients may have a history of **repeated remissions and exacerbations**, and the condition can also recur in previously treated patients.

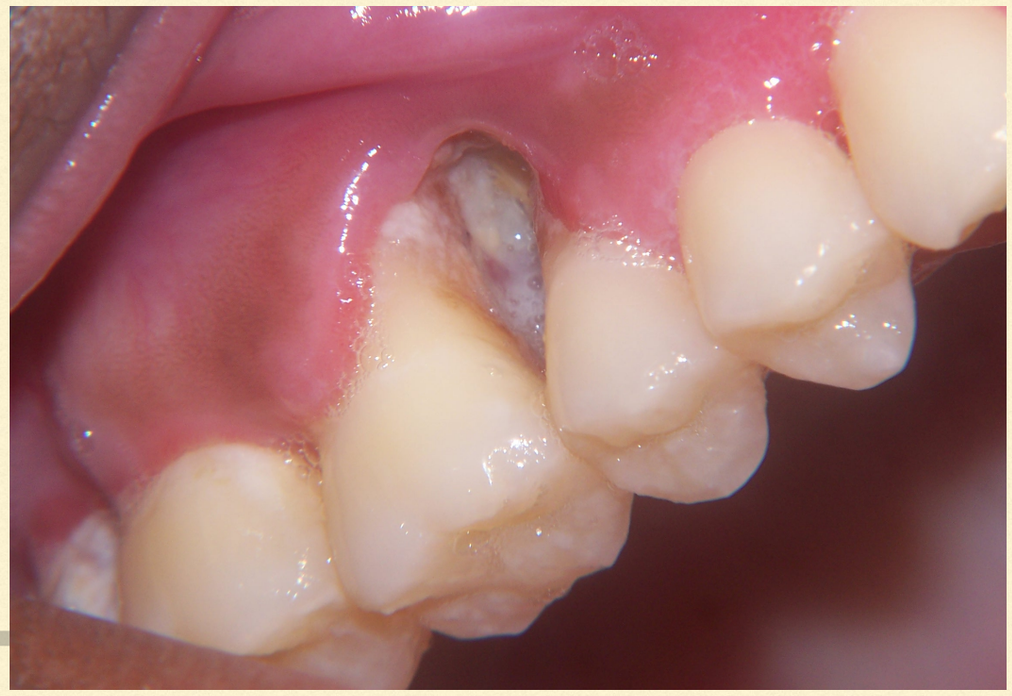


Clinical Features:

- Involvement may be **limited** to a single tooth or group of teeth, or it may be **widespread** throughout the mouth.

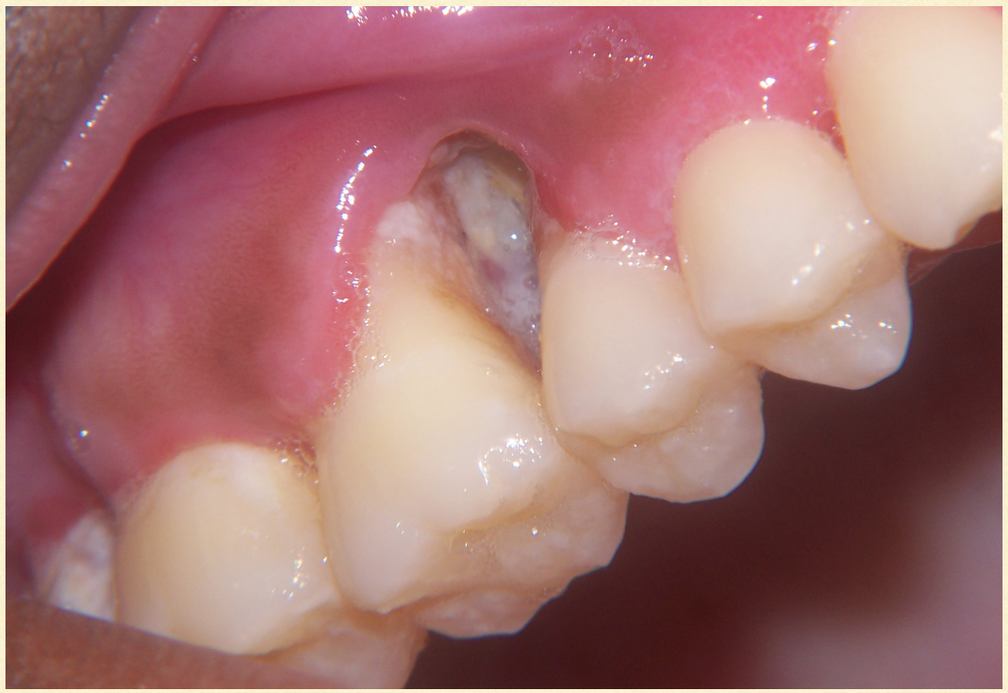
- UG can cause tissue destruction that involves the **periodontal attachment** apparatus, especially in patients with long-standing disease or severe immunosuppression. **When bone loss occurs, the condition is called necrotizing ulcerative periodontitis (NUP).**





Oral Signs.

- **However, NUG or NUP does not usually lead to periodontal pocket formation, because the necrotic changes involve the junctional epithelium; a <u>viable junctional epithelium is needed for pocket deepening.</u>**



Oral Signs.

- ***Punched out crater*** like depressions at the crest of interdental papillae, extending to the marginal gingiva and rarely to the attached gingiva and oral mucosa.

- **Grayish** ***pseudo-membrane slough*** covering the craters.

- ***Spontaenous bleeding*** on slightest stimulation.

- Fetid **odor**, metallic foul taste.

- Increased *pasty* **saliva.**

- Extreme *sensitive* to **touch.**

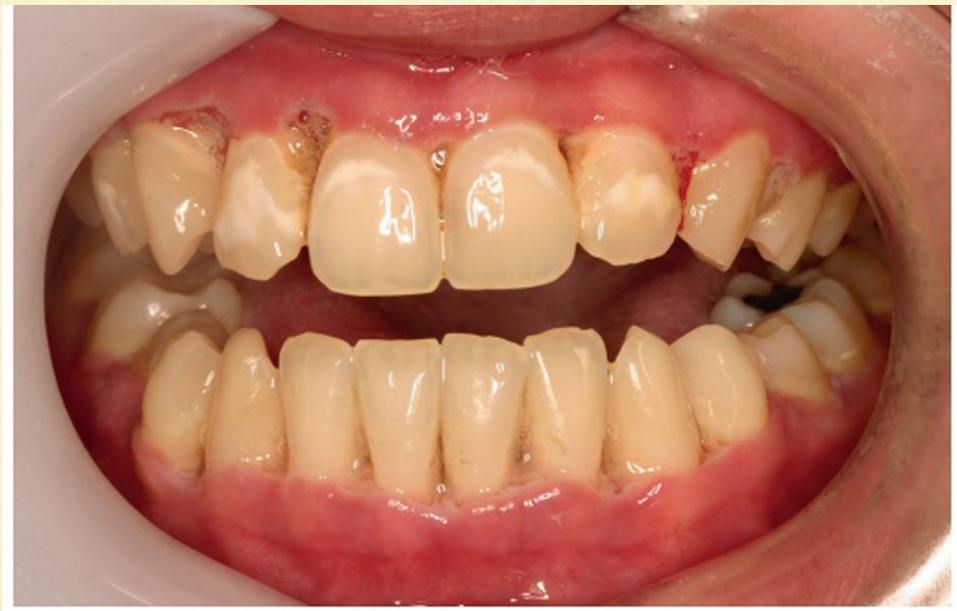


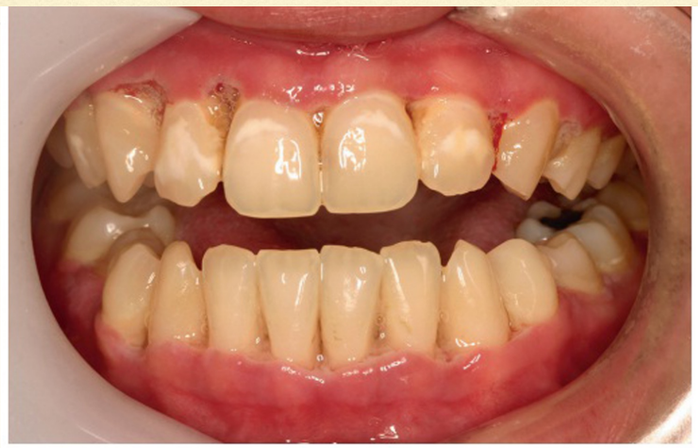
FIGURE 4-13 Necrotizing ulcerative gingivitis with typical punched out, necrotic and ulcerated interdental papillae. (Courtesy of Dr. Hani Mawardi, Boston, MA.)

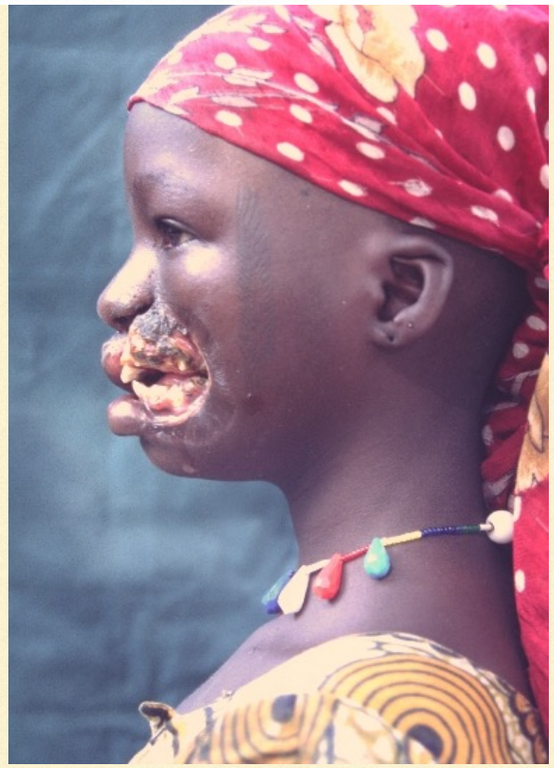
Extraoral and Systemic Signs and Symptoms.

- *Mild and moderate stages* – local lymphodenopathy, slight elevation in temperature.

- *Severe cases* – high fever, increased pulse rate, leucocytosis and loss of appetite.

- *Rarely* – sequel such as noma.





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Clinical Course.

STAGES.

1: Necrosis of tip of Inter dental papilla

2: Necrosis of entire papilla

3: Necrosis extending to the gingival margin

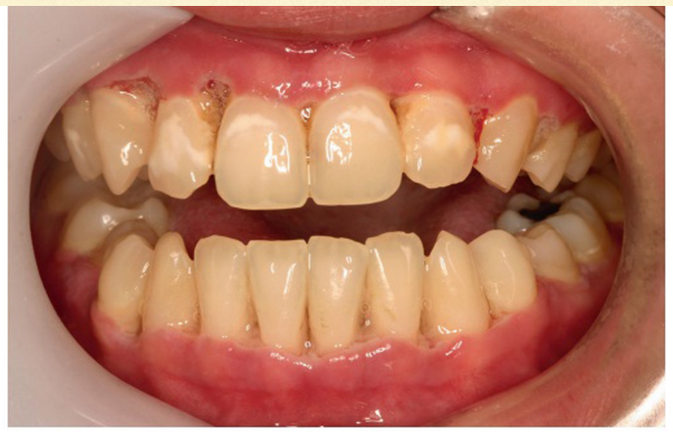
4: Necrosis extending to Attached gingiva

5: Necrosis into Buccal/Labial Mucosa

6: Necrosis exposing Alveolar Bone

7: Necrosis perforating skin of cheek

NOMA



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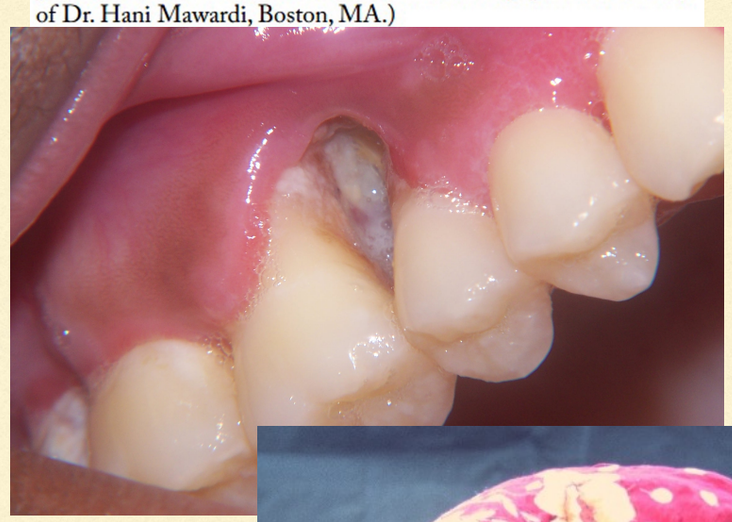
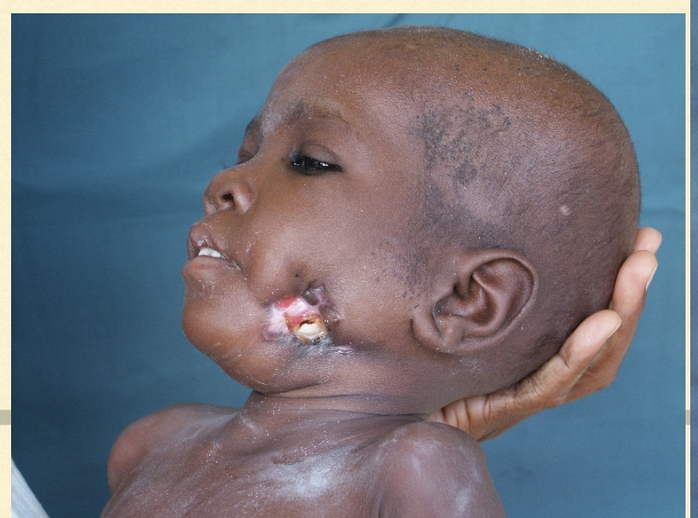
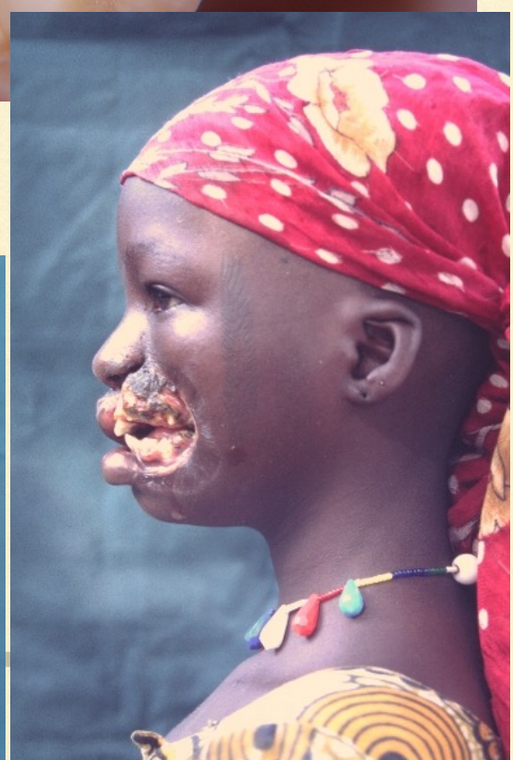
ANUG



ANUP

ANUS





Etiology.
1. Role of Bacteria.

- Plaut in 1894 and Vincent in 1896 postulated that NUG was caused by **specific bacteria**: **fusiform bacillus and a spirochetal organism**.

- **Treatment with metronidazole** results in a significant reduction of Treponema species, Prevotella intermedia, and Fusobacterium,with resolution of the clinical symptoms. The antibacterial spectrum of this drug provides evidence for the **anaerobic** members of the flora as etiologic agents.

Etiology.
2. Role of the Host Response.

- **In the etiology of NUG, the presence of these organisms insufficient to cause the disease, this because.**

1. The fusiform–spirochete flora is frequently **found** in patients who do not have NUG.

2. Exudates from NUG lesions produce fusospirochetal abscess rather than typical NUG when such exudates are inoculated subcutaneously into experimental animals.

3. NUG was associated with physical and emotional **stress .**

4. NUG is **not found in well-nourished individuals** with a fully functional immune system. All of the predisposing factors for NUG are associated with immunosuppression particularly in PMN chemotaxis and phagocytosis.

Etiology.
2. Role of the Host Response.

- **Immunodeficiency may be related to**

1. Nutritional deficiency,
2. Fatigue caused by chronic sleep deprivation,
3. Health habits (e.g., alcohol or drug abuse),
4. Psychosocial factors, or systemic disease.
5. NUG may be the presenting symptom for patients with immunosuppression related to HIV infection.

Etiology.
3. Local Predisposing Factors.

- Preexisting **gingivitis, injury to the gingiva, and smoking** are important predisposing factors.
-
- **Deep periodontal pockets and pericoronal flaps** are particularly vulnerable areas, because they offer a favorable **environment** for the proliferation of anaerobic fusiform bacilli and spirochetes.

- Areas of the gingiva that are **traumatized by opposing teeth** in malocclusion (e.g., the palatal surface behind the maxillary incisors, the labial gingival surface of the mandibular incisors) may predispose an individual to the development of NUG.

Etiology.
4. Nutritional Deficiency.

- A poor diet has been cited as a predisposing factor for NUG and its sequelae in developing African countries, although the effects appear primarily to **diminish the effectiveness of the immune response.**

- Nutritional deficiencies (e.g., vitamin C, vitamin B2) accentuate the severity of the pathologic changes induced when the fusospirochetal bacterial complex is injected into animals.

Etiology.
5. Debilitating Disease.

- Debilitating systemic disease may predispose patients to the development of NUG.

- Such systemic disturbances include,

1. Chronic diseases (e.g., **syphilis, cancer**),
2. Gastrointestinal disorders (e.g., **ulcerative colitis)**,
3. Blood dyscrasias (e.g., **leukemia, anemia**),
4. Acquired immunodeficiency syndrome.

Etiology.
6. Psychosomatic Factors.

- The disease often occurs in association with **stressful situations** (e.g., **induction into the armed forces, school examinations**).

- Psychologic disturbances as well as **increased adrenocortical secretion** are also common in patients with the disease.

- The mechanisms whereby psychologic factors create or predispose an individual to gingival damage have not been established, but alterations in digital and gingival capillary responses that suggest increased autonomic nervous activity have been demonstrated in patients with NUG.

Diagnosis

- **Diagnosis is based on.**

1. **Clinical findings of gingival pain, ulceration, and bleeding.**

2. **Past medical history.**

3. **A bacterial smear is not necessary or definitive But are useful for the differential diagnosis of NUG and specific infections of the oral cavity (e.g., diphtheria, thrush, actinomycosis, streptococcal stomatitis).**

Differential Diagnosis.

- **NUG should be differentiated from other conditions, such as**

1- Herpetic gingivostomatitis.
2- Chronic periodontitis;
3- Desquamative gingivitis, streptococcal gingivostomatitis; aphthous stomatitis; gonococcal gingivostomatitis; diphtheritic and syphilitic lesions. tuberculous gingival lesions; candidiasis, agranulocytosis, and dermatoses (pemphigus, erythema multiforme, and lichen planus); and stomatitis venenata.

- **Treatment options for these diseases vary dramatically, and improper treatment may exacerbate the condition.**

- **In the case of primary herpetic gingivostomatitis, early diagnosis may result in treatment with antiviral drugs that would be ineffective for NUG, whereas the treatment of a case of herpes with the debridement required for NUG could exacerbate the herpes.**

TREATMENT

- Alleviation of the *acute inflammation and* alleviation of generalized *toxic symptoms* such as fever or malaise.

- Treatment of gingival *disease* underlying the acute involvement or else where in the oral cavity.

- Correction of *systemic conditions* that contribute to the initiation or progress of the gingival changes.

- Correction of gingival defect…… Plastic surgery.

TREATMENT

- Treatment should follow an orderly sequence and is divided into treatment for

- 1. **Non-ambulatory patient (bedridden)**: With symptoms of generalized systemic complications.

- 2. **Ambulatory patient**: With no serious systemic complications

TREATMENT

Non-Ambulatory Patients

- **Day 1:**

- *Local treatment* limited to gently removing the necrotic pseudomembrane with a pellet of cotton saturated with hydrogen peroxide (H_2O_2).

- Advised *bed rest* and *rinse* the mouth every 2 hours with a diluted 3 percent hydrogen peroxide.

- *Systemic antibiotics* like penicillin or metronidazole can be prescribed.

TREATMENT

- **Day 2:**

- If condition is improved, proceed to the treatment described for ambulatory patients.
- If there is no improvement at the end of the 24 hours, a bedside visit should be made. The treatment again includes gently swab the area with hydrogen peroxide, instructions of the previous day are repeated.
- **Day 3:**

- Most cases, the condition will be improved, start the treatment for ambulatory patients.

TREATMENT

Ambulatory Patients

- **First visit:**

- Clinician should obtain a *general* information regarding **recent illness, living conditions, dietary background, type of employment, hours of rest and mental stress.**

- Patient's ***general appearance*** should be observed, apparent **nutritional status** and temperature noted.

- Sub Maxillary and Sub Mental ***lymph nodes*** palpated.

- Oral cavity examined for ***characteristic lesion of NUG*****,** it's distribution and possible involvement of the orpharyngeal region.

- *Oral hygiene* evaluated, presence of pericoronal flaps, periodontal pockets and **local irritants determined.**

TREATMENT

- Topical anesthesia applied and area gently swabbed with a cotton pellet to *remove the pseudo membrane* and non-attached surface debris.

- **Superficial** calculus is removed.

- *Subgingival scaling and curettage contraindicated* to prevent extension of infection to deeper tissues and also bacteremia.

- Unless an emergency exists, extractions or periodontal surgery are postponed until the patient has been symptom free for a period of **4 weeks** to minimize likelihood of exacerbation.

TREATMENT

- Patients with *moderate or severe* NUG and local lymphadenopathy or other systemic symptoms are **placed on an antibiotic regimen** of Penicillin (500mg 6th hourly);
- Metronidazole 500mg bid (500mg twice per day) for 7 days is also effective.
- **Antibiotics continued until systemic complications subside.**

- .

INSTRUCTIONS AFTER FIRST VISIT.

- *Avoid tobacco*, alcohol and condiments.

- Rinse with a glassful of an equal *mixture of 3% H_2O_2* and warm water every 2 hours and/or twice daily with *0.12% chlorhexidene* solution.

- Avoid excessive *physical exertion* or prolonged exposure to the sun.

- Confine tooth brushing to the removal of surface debris with a bland dentifrice; over zealous brushing and the use of dental floss or interdental cleaners will be painful.

TREATMENT

- **<u>Second visit:</u>**

- 1 to 2 days later, the patient's condition is usually improved; pain diminished or no longer present.
- Gingival margins of the involved areas are erythematous, but **without a superficial pseudo membrane.**
- Scaling performed if sensitivity permits.
- Instructions as same as above.

TREATMENT

- **Third visit:**

- 1 to 2 days after the second visit, patient should be essentially **symptom free.**

- Scaling and root planing repeated.

- Instructed in plaque control procedures.

- H_2O_2 rinses **discontinued;** chlorhexidene rinses maintained for 2-3 weeks.

TREATMENT

- **Fourth visit:**

- Oral hygiene instructions are reinforced and thorough scaling and root planing are performed.

- **Fifth visit:**

- Appointments are fixed for treatment of chronic gingivitis, periodontal pockets and pericoronal flaps, and for the elimination of all local irritants. Patient is placed on maintenance programme.

2.Primary Herpetic Gingivostomatitis

- **Def.**

- Primary herpetic gingivostomatitis is an infection of the oral cavity **caused by** herpes simplex virus (HS) type 1.

- It occurs most often among infants and children who are **less than 6 years old**, but it is also seen in adolescents and adults.

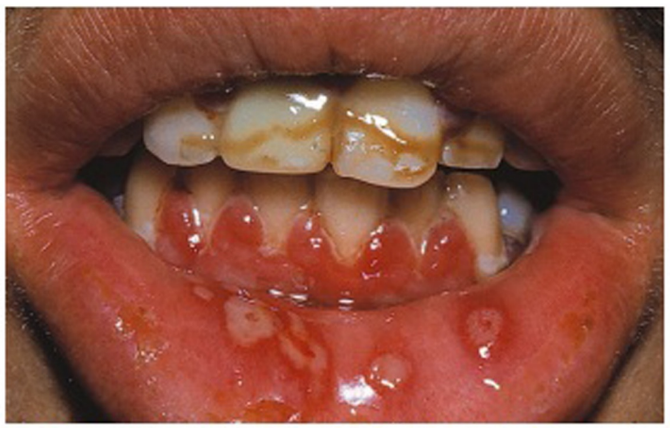


Figure 17-6 Primary herpetic gingivostomatitis in a 12-year-old boy, with diffuse erythematous involvement of the gingiva and a spherical gray vesicle in the lip. *(Courtesy Dr. Heddie Sedano, University of California, Los Angeles, and University of Minnesota.)*

2. Primary Herpetic Gingivostomatitis

- As part of the primary infection, the **virus ascends through the sensory and autonomic nerves, where it persists as latent HS in neuronal ganglia that innervate the site.**
- In approximately one third of the world's population, **secondary manifestations result from various stimuli,** such as sunlight, trauma, fever, and stress.
- These secondary manifestations include herpes labialis, herpetic stomatitis, herpes genitalis, ocular herpes, and herpetic encephalitis.
- Secondary herpetic stomatitis can occur on the palate, on the gingiva, or on the mucosa as a result of dental treatment that traumatizes or stimulates the latent virus in the ganglia that innervate the area it may present as pain away from the site of treatment 2 to 4 days later. Careful inspection for characteristic vesicles may be diagnostic.

- **Clinical Features**
- ***Oral Signs.***

- Primary herpetic gingivostomatitis appears as a **diffuse, erythematous involvement of the gingiva and the adjacent oral mucosa, with varying degrees of edema and gingival bleeding.**
- **During its initial stage,** it is characterized by the presence of discrete, spherical gray **vesicles,** which may **occur on the gingiva, the labial and buccal mucosae, the soft palate, the pharynx, the sublingual mucosa, and the tongue.** After approximately 24 hours, the vesicles rupture and form **painful small ulcers** with red, elevated, halo-like margins and depressed yellowish or grayish-white central portions.
- The course of the disease is limited to 7 to 10 days. The diffuse gingival erythema and edema that appear early during the course of the disease persist for several days after the ulcerative lesions have healed.
- Scarring does **not** occur in the areas of healed ulcerations.

- ***Oral Symptoms.***

- The disease is accompanied by **generalized soreness** of the oral cavity, which interferes with eating, drinking, and oral hygiene.

- The ruptured vesicles are the focal sites of **pain** they are particularly sensitive to touch, thermal changes, foods such as condiments and fruit juices, and the action of coarse foods. In infants, the disease is marked by irritability and refusal to take food.

- ***Extraoral and Systemic Signs and Symptoms.***

- Cervical adenitis, fever as high as 101F to 105F (38C to 40.6C), and generalized malaise are common.

- ***History.***

- Primary herpetic gingivostomatitis is the result of an acute infection by HS, and it has an acute onset

• Diagnosis.

- It is critical to arrive at a diagnosis as early as possible in a patient with a primary herpetic infection. Treatment with antiviral medications can dramatically **alter the course** of the disease **by reducing symptoms and potentially reducing recurrences.**

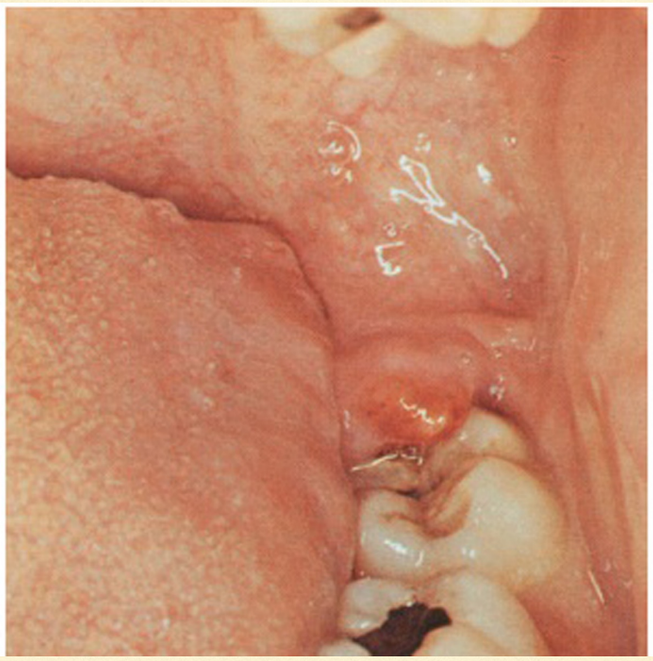
- **<u>The diagnosis is usually established from.</u>**

1. **The patient's history.**
2. **The clinical findings.**
3. Material may be obtained from the lesions and submitted to the laboratory for confirmatory tests, including **virus culture and immunologic tests .**

3. Pericoronitis

- **Def.**

- The term *pericoronitis* refers to inflammation of the gingiva in relation to the crown of an incompletely erupted tooth. It occurs most often in the mandibular third molar area.
- Pericoronitis may be acute, subacute, or chronic.



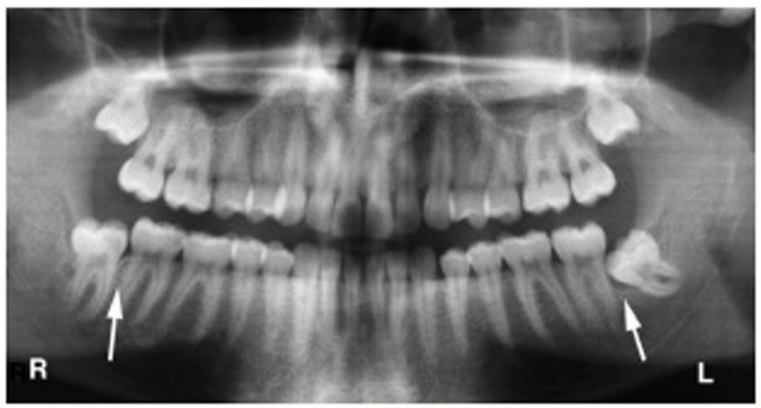


Figure 7-28 Panoramic radiograph illustrating a mesial impacted lower left third molar with a widened follicle and no apparent bone on the distal interproximal surface of the second molar. Alternatively, the lower right third molar is vertically impacted and exhibits interproximal bone distal to the second molar and mesial to the third molar.

Clinical Features.

- The partially erupted or impacted mandibular third molar is the most common site of pericoronitis. The space between the crown of the tooth and the overlying gingiva (operculum) is an ideal area for the accumulation of food debris and bacterial growth.
- Acute inflammatory involvement is a constant possibility it may be exacerbated by trauma, occlusion, or a foreign body trapped underneath the tissue .
- The inflammation and cellular exudate increase the bulk of the gingiva, which then may interfere with complete closure of the jaws and which can be **traumatized by contact with the opposing jaw,** thereby aggravating the inammatory involvement.

Clinical Features.

- The resultant clinical picture is a red, swollen, suppurating lesion that is tender, with **radiating pains to the ear, the throat, and the floor of the mouth**.
- The patient is extremely uncomfortable as a result of pain, a foul taste, and an inability to close the jaws. **Swelling of the cheek** in the region of the angle of the jaw and lymphadenitis are common findings.
- **Trismus** may also be a presenting complaint.
- In addition, the patient may have **systemic complications** such as fever, leukocytosis, and malaise.

Complications.

- Involvement may be localized in the form of a **pericoronal abscess.** It may spread posteriorly into the oropharyngeal area and medially to the base of the tongue, thereby making it difficult for the patient to swallow.

- Depending on the severity and extent of the infection, there may be involvement of the submaxillary, posterior cervical, deep cervical, and retropharyngeal **lymph nodes.**

- Peritonsillar abscess formation, cellulitis, and Ludwig's angina are infreuent but potential sequelae of acute pericoronitis.

TREATMENT

- Procedure for relieving the pain is surgical removal of the operculum, inject local anesthetic directly into the overlying tissue and then cut it away using the outline of the tooth as a guide for the incision. Sutures are not required .
- Irrigate with a weak (2%) hydrogen peroxide solution.
- Prescribe oral analgesics for comfort as well as penicillin over the next 10 days (penicillin VK 500mg).
- Instruct the patient on the importance of cleansing away any food particles that collect beneath the gingival flap. This can be accomplished by simply using a soft toothbrush or by using water jet irrigation.
- Follow-up should be provided to observe the resolution of the acute infection and to evaluate the need for removal of the gingival flap or molar.
- Do not undertake any major blunt dissection while draining pus. This could spread a superficial infection into the deep spaces of the head and neck or follow a deep abscess posteriorly into the carotid sheath.

TREATMENT