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by Dr. M.G.R EDUCATIONAL AND RESEARCH INSTITUTE

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ACUTE GINGIVAL INFECTIONS

- ACUTE GINGIVAL INFECTIONS INCLUDE :

  - NECROTIZING ULCERATIVE GINGIVITIS
  - PRIMARY HERPETIC GINGIVO STOMATITIS
  - PERICORONITIS

NECROTIZING ULCERATIVE GINGIVITIS

- **Necrotizing ulcerative gingivitis is a microbial disease of the gingiva in the context of an impaired host response. It is characterised by the death and sloughing of gingival tissue and presents with characteristic signs and symptoms.**

HISTORY

- **NUG is characterised by sudden onset of symptoms, sometimes following an episode of debilitating disease or acute respiratory tract infection.**

- **A change in living habits ,poor nutrition, tobacco use, and psychologic stress are frequent features of the patient's history**

ETIOLOGY

ROLE OF BACTERIA :

- NUG is caused by specific bacteria fusiform bacillus and spirochetal organism.
- Fusospirochetal complex consisting of Treponema microdentium, intermediate spirochetes, vibrios, borrelia ,Prevotella intermedia

ROLE OF THE HOST RESPONSE :

- The role of an impaired host response in NUG has been associated with physical and emotional stress

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LOCAL PREDISPOSING FACTORS

- Preexisting gingivitis, injury to gingiva, smoking ,Deep periodontal pockets and pericoronal flaps are important pre-disposing factors

- Areas of the gingiva traumatized by opposing teeth in malocclusion, may also predispose to NUG.

- 98% of the patients with NUG were smokers and that the frequency of the disease increases with an increasing exposure to tobacco smoke.

SYSTEMIC PREDISPOSING FACTORS

- **Predisposing factors leading to immunodeficiency is important .It may be related to varying levels of nutritional deficiency; fatigue caused by chronic sleep deficiency and systemic disease.**

- **Nutritional deficiencies : eg; (vit B2, vit C) accentuate the severity of the pathologic changes**

- **Debilitating diseases and chronic diseases (syphilis, cancer), severe gastrointestinal disorders (ulcerative colitis), blood dyscrasias (leukemia, anemia) , AIDS.**

PSYCHOSOMATIC FACTORS

- **Psychiatric disturbance(anxiety, depression or psychopathic deviance) and the impact of negative life events (stress) may lead to activation of the hypothalamic-pituitary-adrenal–axis. This results in elevation of serum and urine cortisol levels, which is associated with a depression of lymphocyte and PMN function that predispose to NUG**

RELATION OF BACTERIA TO CHARACTERISTIC LESION

- The classic electron microscopic description of NUG lesions identified four zones
- ZONE 1 : The bacterial zone composed of bacteria with various morphocytes, including spirochetes.
- ZONE 2 : The neutrophil rich zone, consisting of many leukocytes and a predominance of neutrophils.
- ZONE 3 : The necrotic zone composed of dead cells, spirochetes and other bacteria.
- ZONE 4 : The spirochetal infiltration zone consisting of intact tissue elements infiltrated by spirochetes

CLINICAL FEATURES

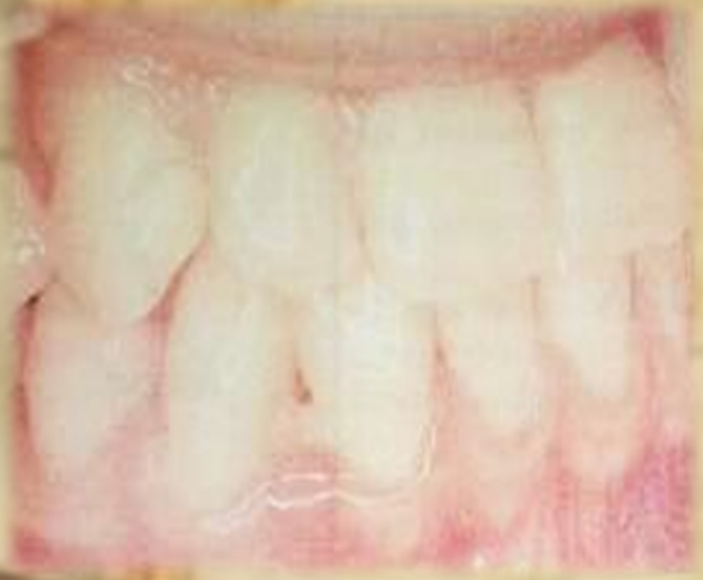
Oral signs

- Characteristic lesions are punched-out, craterlike depressions at the crest of the interdental papillae, extending to the marginal gingiva .
- The gingival craters is covered by a gray, pseudomembranous slough, demarcated from the gingival mucosa by a linear erythema.
- In some cases the lesions are denuded of the surface pseudomembrane, exposing the gingival margin, which is red, shiny, and hemorrhagic.
- The Characteristic lesions may progressively destroy the gingiva and underlying periodontal tissues.

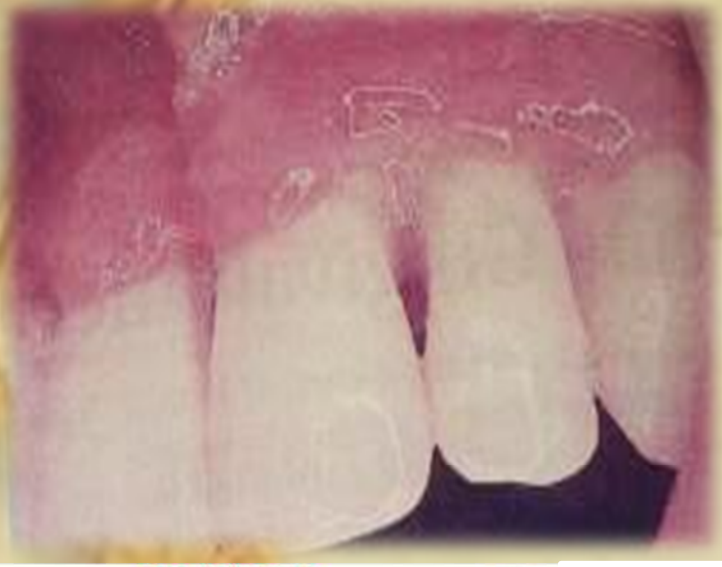
- **Spontaneous gingival hemorrhage or pronounced bleeding after the slight stimulation**

- **Other signs found are fetid odor and increased salivation.**

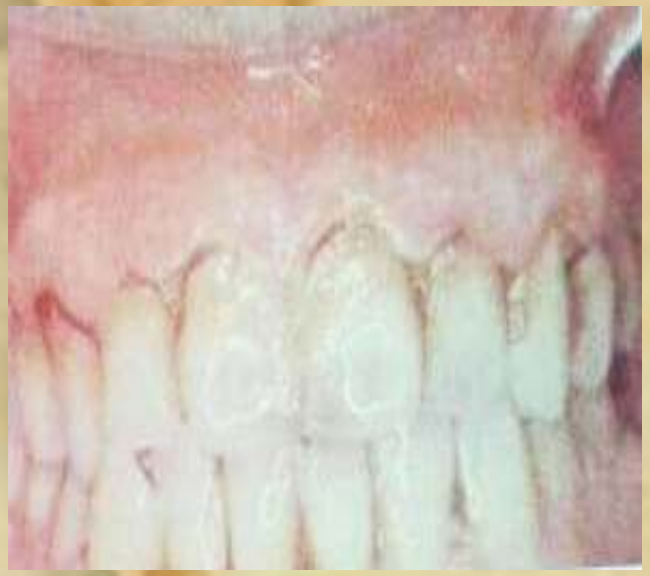
- **NUG can be superimposed on chronic gingivitis or periodontal pockets. NUG or NUP does not usually lead to periodontal pocket formation because the necrotic changes involve the marginal gingiva , causing recession rather than pocket formation.**



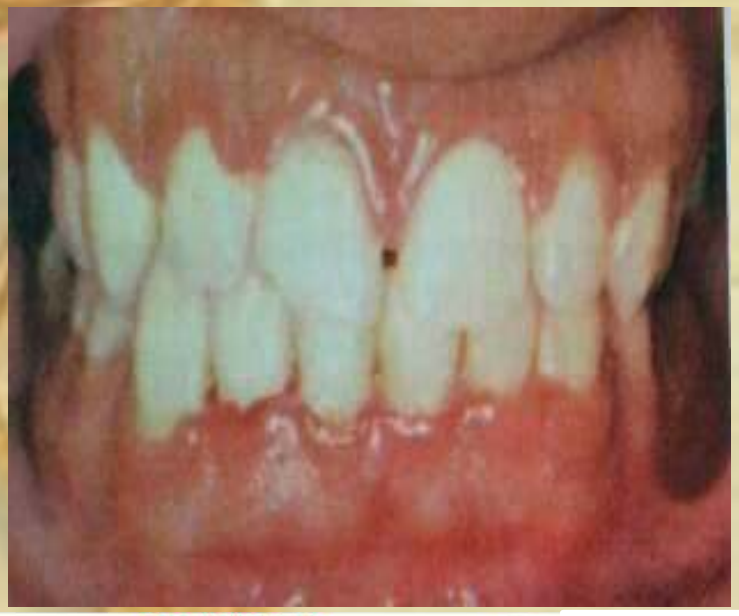
TYPICAL PUNCHED-OUT PAPILLA BETWEEN MANDIBULAR CANINE AND LATERAL INCISOR , COVERED BY GRAYISH PSEUDOMEMBRANE



DESTRUCTION OF PAPILLAE RESULTING IN IRREGULAR MARGINAL CONTOUR



GENERALIZED INVOLVEMENT OF PAPILLAE AND MARGINAL GINGIVA, WITH WHITISH NECROTIC LESIONS



TYPICAL LESIONS WITH SPONTANEOUS HAEMORRAGE

ORAL SYMPTOMS

- The lesions are extremely sensitive to touch
- Pain is constant radiating, gnawing
- "Metallic" foul taste, and there is excessive amount of "pasty" saliva.

EXTRAORAL AND SYSTEMIC SIGNS AND SYMPTOMS

- Local lymphadenopathy and a slight elevation in temperature.
- In severe cases, there is high fever, increased pulse rate, leukocytosis, loss of appetite, and general lassitude.
- Insomnia, constipation, gastro-intestinal disorders, headache, and mental depression sometimes

CLINICAL COURSE

- **If untreated, NUG may lead to NUP with a progressive destruction of the periodontium and gingival recession with systemic complications**

- **<u>Pindborg have described these stages in the progress of NUG</u>**

1) **Erosion of only the tip of the inter-dental papilla.**
2) **The lesion extending to marginal gingiva and causing a further erosion of the papilla and potentially a complete loss of the papilla.**
3) **The attached gingiva also being affected**
4) **Exposure of bone.**

STAGING OF ORAL NECROTIZING DISEASE

STAGE 1 : Necrosis of the tip of the interdental papilla.
STAGE 2 : Necrosis of the entire papilla.
STAGE 3 : Necrosis extending to the gingiva margin.
STAGE 4 : Necrosis extending also to the attached gingiva.
STAGE 5 : Necrosis extending into buccal or labial mucosa.
STAGE 6 : Necrosis exposing alveolar bone.
STAGE 7 : Necrosis perforating skin of cheek.

According to Horning and cohen stage 1 is NUG, stage 2 may be either NUG or NUP because attachment loss may have occurred, stages 3 and 4 would correspond to NUP, stages 5 and 6 would correspond to necrotizing stomatitis, and stage 7 would be Noma.

HISTOPATHOLOGY

- The NUG lesion is acute necrotizing inflammation of the gingival margin, involving both the stratified squamous epithelium and the connective tissue.

- The surface epithelium is destroyed and replaced by a meshwork of fibrin, necrotic epithelial cells, PMNs, neutrophils and various types of micro-organisms.

- At the border of the necrotic pseudomembrane ,the epithelium is edematous, and individual cells exhibit varying degrees of hydropic degeneration.

- There is infiltration of PMNs in the intercellular spaces.
- The connective tissue is extremely hyperemic, with engorged capillaries and a dense infiltration of PMNs.
- Plasma cells may appear in the periphery of the infiltrate ; this is interpreted as an area of established chronic gingivitis on which the acute lesion became superimposed

DIAGNOSIS

- **Diagnosis is based on clinical findings of gingival pain, ulceration, and bleeding.**

- **A bacterial smear is not necessary because the bacterial picture is not appreciably different from that in marginal gingivitis, periodontal pockets, or primary herpetic gingivostomatitis.**

- **Microscopic examination of a biopsy specimen is not sufficiently specific to be diagnostic. It can be used to differentiate NUG from specific infections such as tuberculosis or from neoplastic disease.**

- **It should be differentiated from other conditions that resemble it in some respects, such as**

  herpetic gingivostomatitis

  Chronic periodontitis

  Desquamative gingivitis

NECROTIZING ULCERATIVE GINGIVITIS	PRIMARY HERPETIC GINGIVOSTOMATIT
ETIOLOGY: interaction between host and bacteria, most probably fusospirochetes.	Specific viral etiology
Necrotizing condition	Diffuse erythema and vesicular eruption
Punched-out gingival margin, pseudomembranous slough that peels off, leaving raw areas	Vesicles rupture and leave slightly depressed oval or spherical ulcer.
Marginal gingiva affected; other oral tissues rarely affected	Diffuse involvement of gingiva ; may include buccal mucosa and lips.

NECROTIZING ULCERATIVE GINGIVITIS	DESQUAMATIVE GINGIVITIS	CHRONIC DESTRUCTIVE PERIODONTAL DISEASE
Bacterial smears show fusospirochetal complex	Bacterial smears reveal numerous epithelial cells, few bacterial forms	Bacterial smears are variable
Marginal gingiva is affected	Diffuse involvement of marginal and attached gingivae and other areas of oral mucosa	Marginal gingiva affected
Pseudomembrane	Patchy desquamation of gingival epithelium	Generally no desquamation, but purulent material may appear from pockets.
Papillary and marginal necrotic lesions	Papillae do not undergo necrosis	Papillae do not undergo necrosis
Affects adults of both genders	Affects adults, most often women	Generally in adults

TREATMENT OF NUG

FIRST VISIT :

- **Evaluation of the patient, including medical history with special attention to recent illness, smoking and psychosocial parameters.**
- **The patient is questioned regarding the history of the acute disease and its onset and duration.**
- **The examination should include general appearance, presence of halitosis, presence of skin lesions.**
- **Vital signs including temperature, palpation for the presence of enlarged lymph nodes, especially submaxillary and submental nodes.**

- The oral cavity is examined for the characteristic lesion of NUG, presence of pericoronal flaps, periodontal pockets, and local factors.

- The goal of the initial therapy is to reduce the microbial load and remove necrotic tissue so that repair and regeneration of normal tissue are re-established

- A topical anesthetic is applied and after 2 or 3mins the areas are gently swabbed with a moistened cotton pellet to remove the pseudomembrane.

- After the area is cleansed with warm water, the superficial calculus is removed.

- Subgingival scaling and curettage are contraindicated at this time because these procedures may extend the infection into the deeper tissues and may also cause bacteremia.

SURGICAL PROCEDURES

- **Unless an emergency exists, procedures such as extractions or periodontal surgery are postponed until the patient has been symptom free for 4 weeks, to minimize the acute symptoms.**

- **Patients with moderate or severe NUG are placed on antibiotic regimen of amoxicillin (500mg orally every 6 hours).**

- **For amoxicillin sensitive patients erythromycin or metronidazole is prescribed.**

PATIENT INSTRUCTIONS

- Avoid tobacco and alcohol
- Rinse with a glassful of an equal mixture of 3% hydrogen peroxide and warm water every 2 hrs and/ or twice daily with 0.12% chlorhexidine solution.
- Confine tooth brushing to the removal of surface debris with a bland dentrifice and an ultra soft brush.

SECOND VISIT

- At the second visit, 1 or 2 days after the first visit, the patient is evaluated for amelioration of signs and symptoms.
- The patient's condition is improved the pain is diminished or no longer present .
- The gingival margins are erythematous, but without a superficial pseudomembrane.
- Scaling is performed if necessary and sensitivity permits.
- Shrinkage of the gingiva may expose previously covered calculus, which is gently removed

THIRD VISIT

- At the 3rd visit, approximately 5 days after the second visit, the patient is evaluated for resolution of symptoms.
- Plan for management of patient's periodontal conditions is formulated.
- The patient should be symptom free. Some erythema may still be present and the gingiva may be slightly painful on tactile stimulation.
- The hydrogen peroxide rinses are discontinued, but chlorhexidine rinses can be maintained for 2 or 3 weeks.
- Scaling and root planing are repeated if necessary.

ADDITIONAL TREATMENT CONSIDERATIONS

- **CONTURING OF GINGIVA AS ADJUNCTIVE PROCEDURE :**
- **Even in cases of severe gingival necrosis, healing often leads to restoration of the normal gingival contour occurs after several weeks**
- **If there has been loss of interdental bone, if the entire papilla is lost, healing results in the formation of a shelflike gingival margin.**
- **This may be an esthetic problem and favors retention of plaque and recurrence of gingival inflammation.**

- **This can be corrected by an attempt to restore lost tissue through periodontal plastic surgeries or by reshaping the gingiva surgically.**

- **Effective plaque control by the patient is particularly important to establish and maintain the normal gingival contour.**

PRIMARY HERPETIC GINGIVOSTOMATITIS

- It is an infection of the oral cavity caused by herpes simplex virus type1 (HSV-1).
- It occurs most often in infants and children younger than 6 yrs of age, but it is also seen in adolescents and adults.
- In most persons the primary infection is asymptomatic.
- As a part of primary infection, the virus ascends through sensory and autonomic nerves, where it persists as latent HSV in neuronal ganglia that innervate the site.

- Secondary manifestations result from various stimuli, such as sunlight, trauma, fever, and stress.

- These secondary manifestations include herpes labialis, herpetic stomatitis, herpes genitalis etc.

- Secondary herpetic stomatitis can occur as a result of dental treatment and may present as pain away from the site of treatment 2 to 4 days later.

- Inspection for characteristic vesicles may be diagnostic

CLINICAL FEATURES

ORAL SIGNS

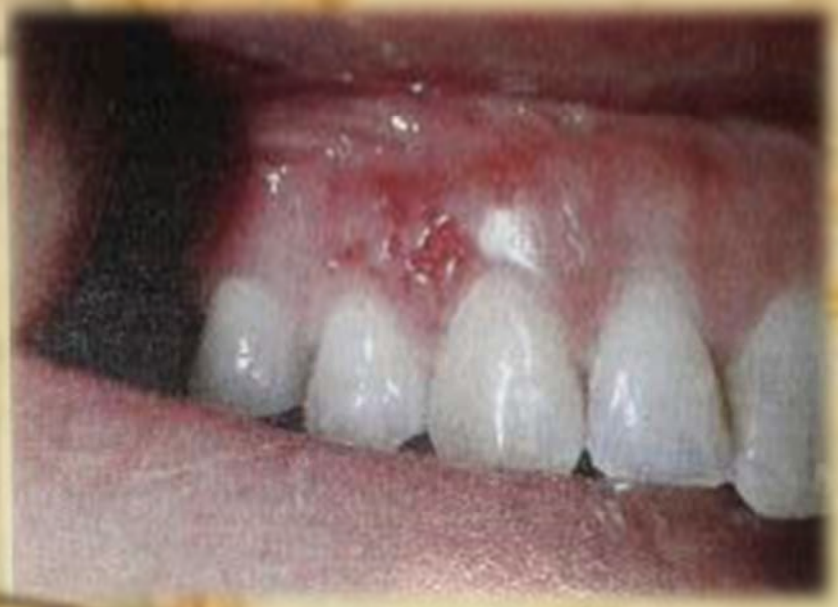
- Diffuse, erythematous, shiny involvement of the gingiva and the oral mucosa, edema and gingival bleeding .
- Initial stage, it is characterized by the presence of discrete, spherical gray vesicles, which occur on the gingiva, labial and buccal mucosae, soft palate, pharynx, and tongue.
- After 24 hrs, the vesicles rupture and form painful, small ulcers with a red, elevated, halo-like margin and a depressed, yellowish or grayish white central portion.
- The course of the disease is limited to 7 to 10 days.
- Scarring does not occur in the areas of healed ulcerations.

ORAL SYMPTOMS

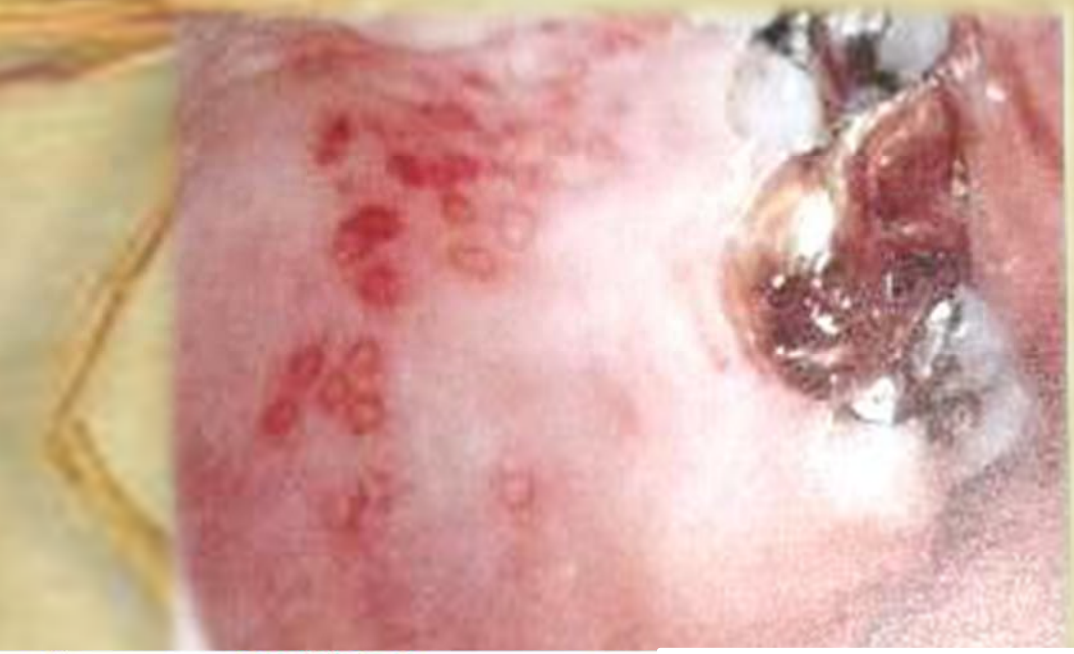
- The disease is accompanied by generalized soreness of the oral cavity, which interferes with eating and drinking.
- The ruptured vesicles are the focal sites of pain and are sensitive to touch and thermal changes,
- In infants the disease is marked by irritability and refusal to take food.

EXTRAORAL AND SYSTEMIC SIGNS AND SYMPTOMS

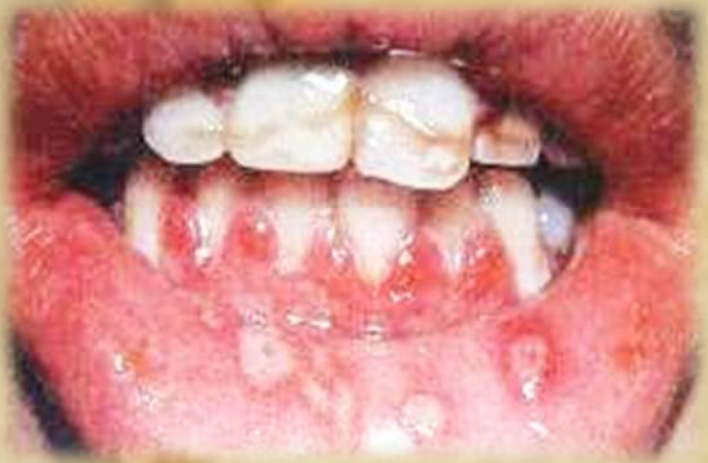
- Cervical adenitis, fever and generalised malaise are common.

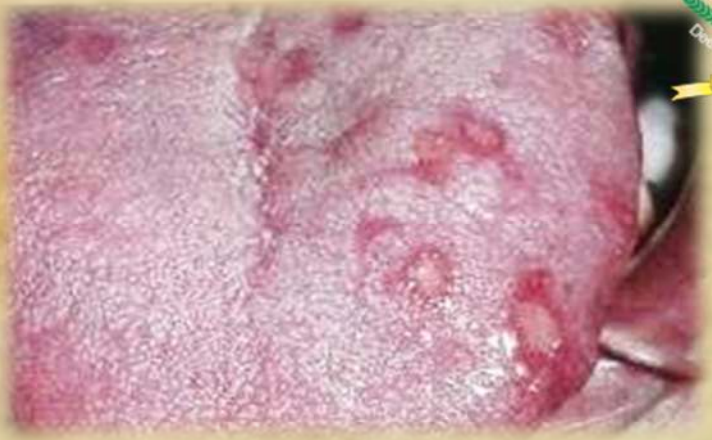


RECURRENT INTRA-ORAL VESICLES IN THE GINGIVA



RECURRENT INTRA-ORAL VESICLES IN THE PALATE



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HISTOPATHOLOGY

- The virus targets the epithelial cells, which shows "ballooning degeneration" consisting of acantholysis. These cells are called as Tzanck cells.
- Infected cells fuse, forming multinucleated cells
- Intercellular edema leads to formation of an intraepithelial vesicles that rupture and develop a secondary inflammatory response with a fibropurulent exudate.
- Discrete ulcerations have a central portion of acute inflammation, with purulent exudate, surrounded by a zone rich in engorged blood vessels

DIAGNOSIS

- **The diagnosis is usually established from the patients history and the clinical findings.**
- **Treatment with antiviral medications can alter the course of the disease, reducing symptoms and potentially reducing recurrences.**
- **Material may be obtained from the lesions and submitted to the laboratory for confirmatory tests.**
- **It includes virus culture and immunologic tests using monoclonal antibodies or DNA hybridization techniques.**

DIFFERENTIAL DIAGNOSIS

- Erythema multiforme
- Necrotizing ulcerative gingivitis
- Stevens-johnson syndrome
- Bullous lichen planus
- Desquamative gingivitis
- Recurrent aphthous stomatitis

TREATMENT

- Treatment consists of early diagnosis and immediate initiation of antiviral therapy.
- Therapy for primary herpetic gingivostomatitis consisted of palliative care.
- In a randomized double-blind placebo-controlled study, antiviral therapy with 15mg/kg of an acyclovir suspension given five times daily for 7 days substantially changes the course of the disease without significant side effects.
- Acyclovir reduced symptoms, including fever, decreases the new extraoral lesions and reduced difficulty with eating.

- If primary herpetic gingivostomatitis is diagnosed within 3 days of onset, acyclovir suspension should be prescribed.
- An NSAID(eg ibuprofen) can be given systemically to reduce fever and pain.
- Patients may use either nutritional supplements or topical anesthetics before eating to aid in proper nutrition.
- Periodontal therapy should be postponed until the acute symptoms subside.

- **Local or systemic application of antibiotics is sometimes advised to prevent opportunistic infection of ulcerations, especially in immunocompromised patients.**

- **If the condition does not resolve with 2 weeks, the patient should be referred to a physician for medical consultation.**

PERICORONITIS

- 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

CLINICAL FEATURES

- The partially erupted or impacted third molar is the most common site.
- The space between the crown of the tooth and the overlying gingival flap (operculum) is an ideal area for the accumulation of food debris and bacterial growth.
- In patients with no clinical signs or symptoms, the gingival flap is often chronically inflamed ,infected and ulcerations along its inner surface.
- Acute inflammatory involvement may be exacerbated by trauma, occlusion, or a foreign body

- The inflammatory fluid and cellular exudate increase the bulk of the flap, which then may interfere with complete closure of jaws .

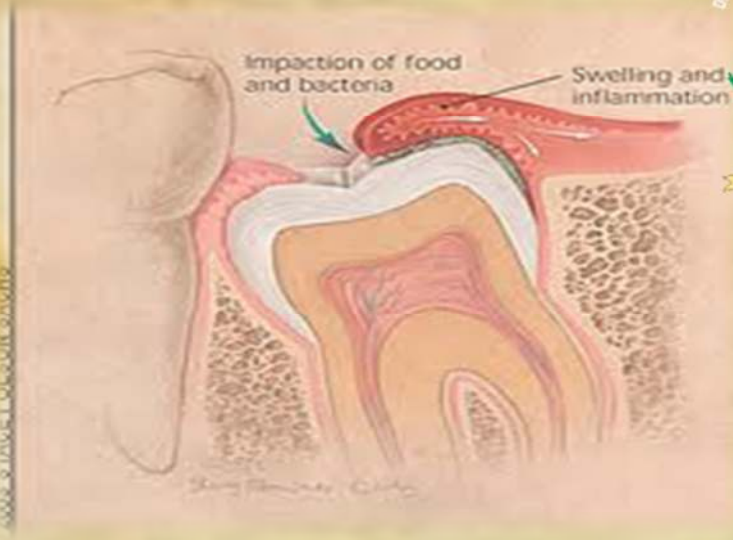
- The resultant clinical picture is that of a red, swollen, suppurating lesion that is tender, with radiating pain to the ear, throat, and floor of the mouth

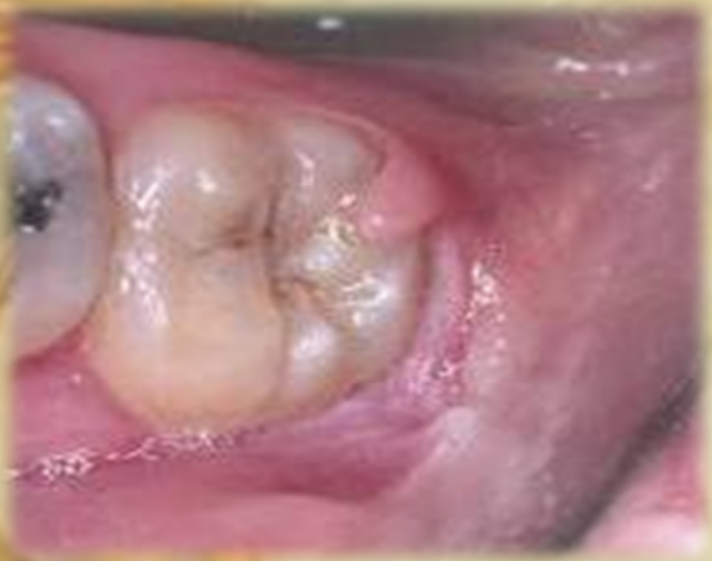
- The patient is uncomfortable because of a foul taste, inability to close the jaws in addition to pain.

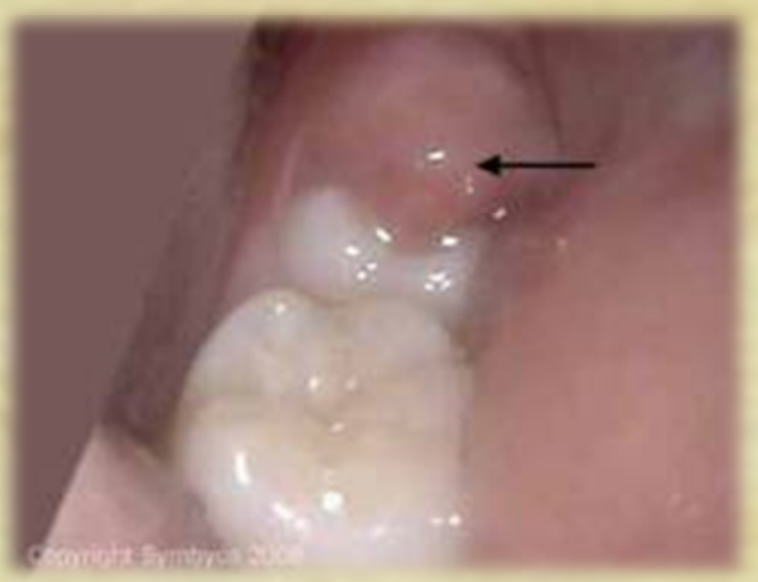
- Trismus may also be a presenting complaint.

- Swelling of cheek in the region of the angle of the jaw and lymphadenitis are common findings


Inflamed gums around the wisdom tooth (pericoronitis)
Lower Wisdom Tooth


Impaction of food and bacteria
Swelling and inflammation





TREATMENT

- The treatment of pericoronitis depends on the severity of the inflammation, the systemic complications, advisability of retaining the involved tooth.

- Persistent symptom-free pericoronal flaps should be removed as a preventive measure against subsequent acute involvement.

- The treatment of acute pericoronitis consists of

1) Gently flushing the area with warm water to remove debris and exudate.
2) Swabbing with antiseptic after elevating the flap gently from the tooth with a scaler.

- The underlying debris is removed, and the area is flushed with warm water.
- The occlusion is evaluated to determine if an opposing tooth is occluding with the pericoronal flap.
- Removal of soft tissue or occlusal adjustment may be necessary.
- Antibiotics can be prescribed in severe cases. If the gingival flap is swollen and fluctuant, the clinician uses a no 15 blade to make an anteroposterior incision to establish drainage.

- After the acute symptoms have subsided, a determination is made about whether to retain or to extract the tooth

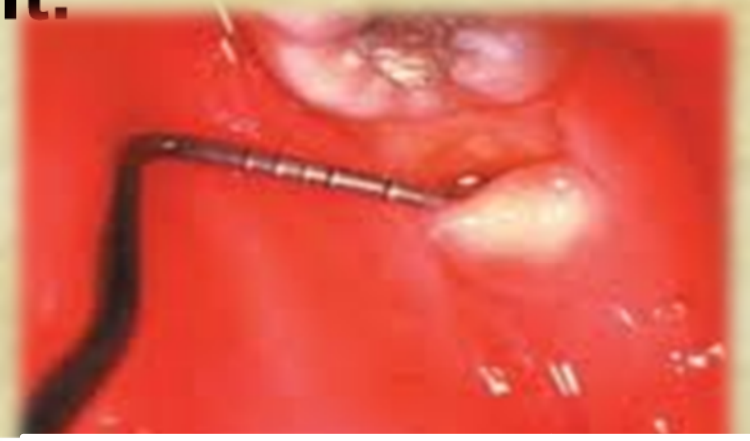
- Bone loss on the distal surface of the second molars is a hazard after the extraction of partially or completely impacted 3rd molars.

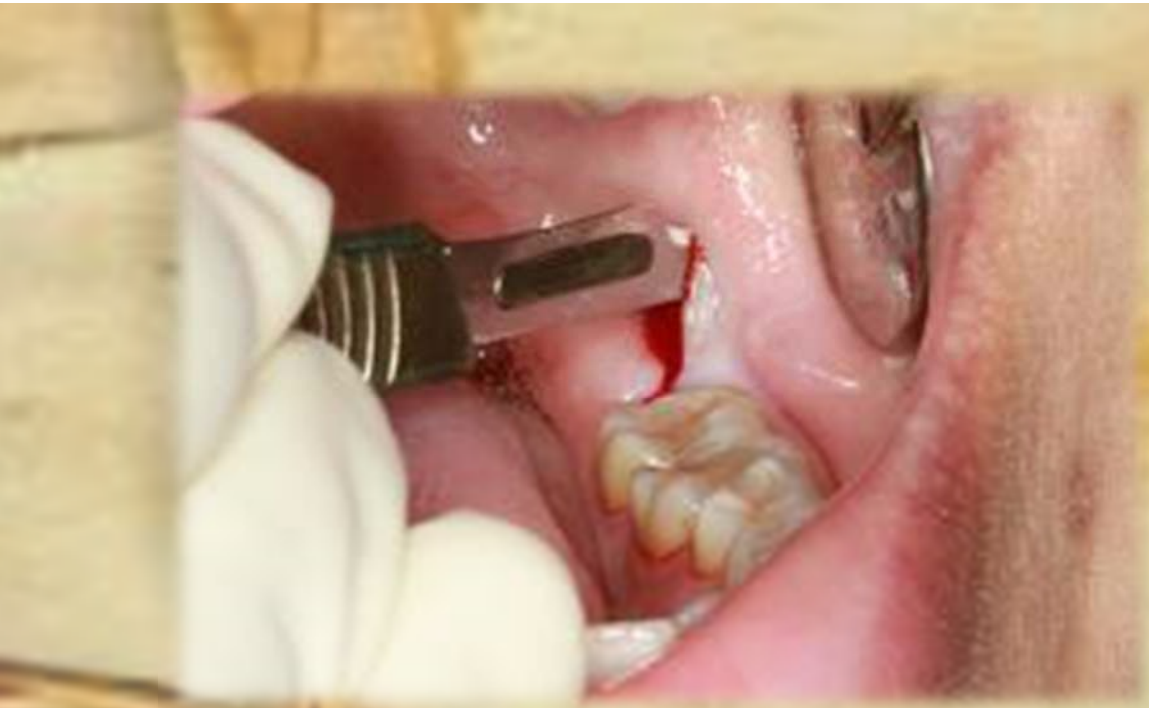
- To reduce the risk of bone loss around 2nd molars, partially or completely impacted 3rd molars should be extracted as early as possible.

- If the decision is made to retain the tooth, the pericoronal flap is removed using peroidontal knives or electrosurgery.

- **It is necessary to remove the tissue distal to the tooth, as well as the flap on the occlusal surface.**

- **Incising only the occlusal portion of the flap leaves a deep distal pocket, which invites recurrence of acute pericoronal involvement.**






Before
After
Before
After


Pericoronitis


THANK
YOU