BDS Year 4 Regular & Casual batch

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Subject: Oral Medicine and Radiology

Topic: VESICULOBULLOUS AND ULCERATIVE LESIONS-III

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CHRONIC ULCERS

Pemphigus

- Pemphigus is a rare group of blistering autoimmune diseases that affect the skin and mucous membranes.
- In pemphigus, autoantibodies form against desmoglein.
- Desmoglein forms the "glue" that attaches adjacent epidermal cells via attachment points called desmosomes.
- When autoantibodies attack desmogleins, : acantholysis occurs.
- This causes blisters which break and leave ulcers

Major variants:

- Pemphigus vulgaris
- Pemphigus vegetans
- Pemphigus foliaceus
- Pemphigus erythematosus
- Paraneoplastic pemphigus
- Drug related pemphigus

Pemphigus Vulgaris

Clinical features:

Age: Usually occurs between 50-60 years

Gender: No gender predilection

Site: Skin & mucosa

Symptoms:

-Chronic, multiple blistering of skin would be the presenting complaint

-Denudation and burning sensation

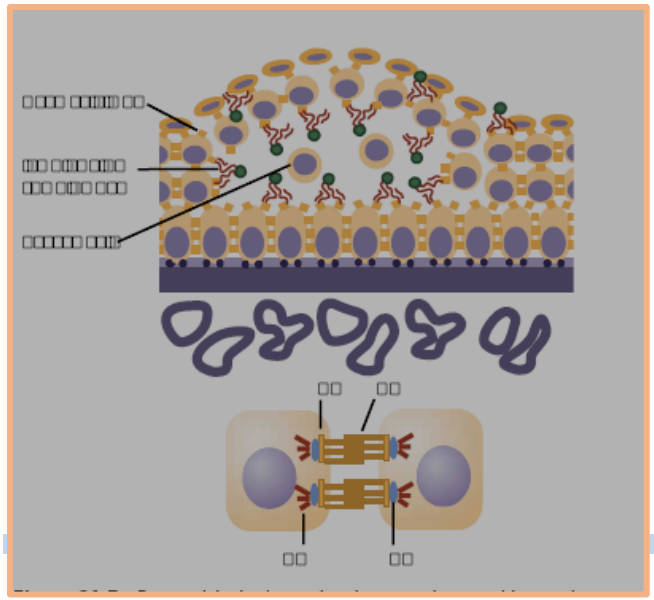
-Difficulty in chewing and swallowing

Pathophysiology:

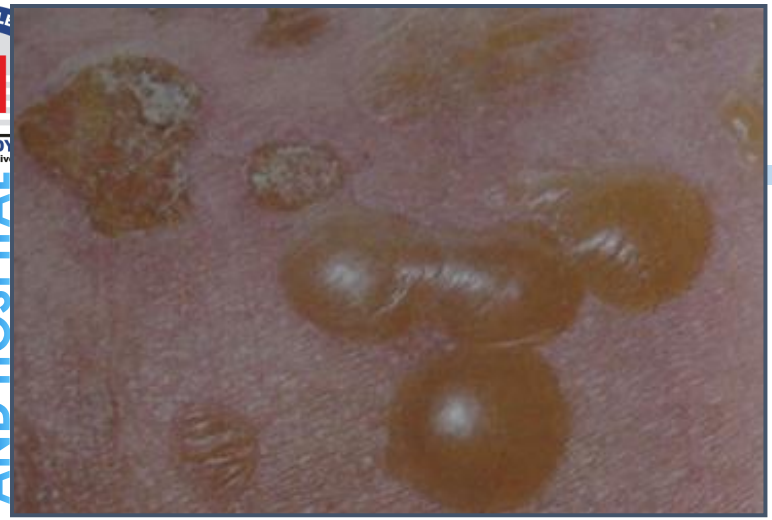
-Intraepithelial lesion is formed when Ig G Abs target 2 structural proteins of desmosomes, desmoglein3 (Dsg3)

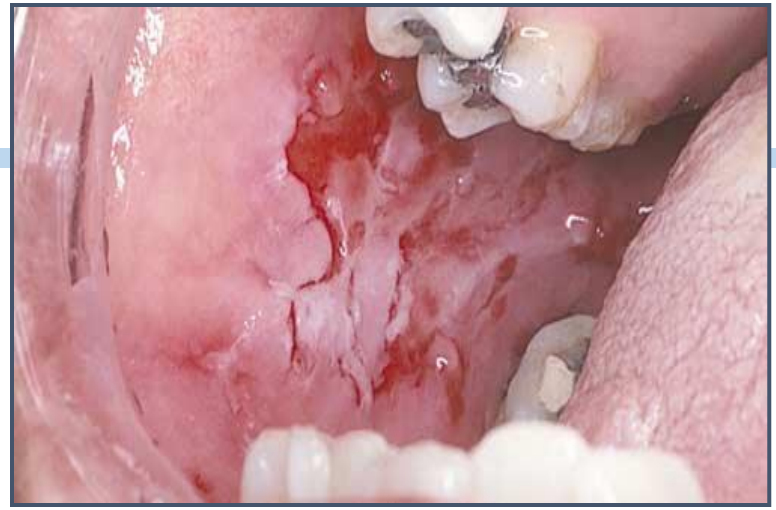
-Loss of cell to cell adhesion, Acantholysis, occurs in lower layers of stratum spinosum

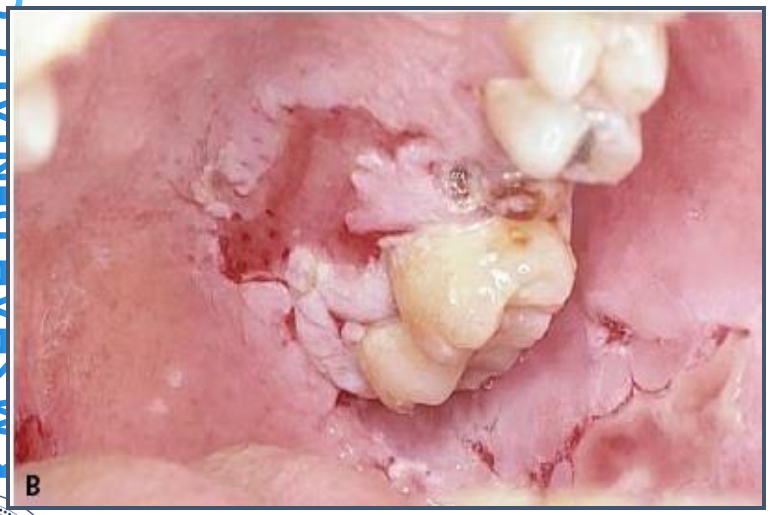
-Results in classic “suprabasilar bulla” involving increasingly greater areas of skin & mucosa

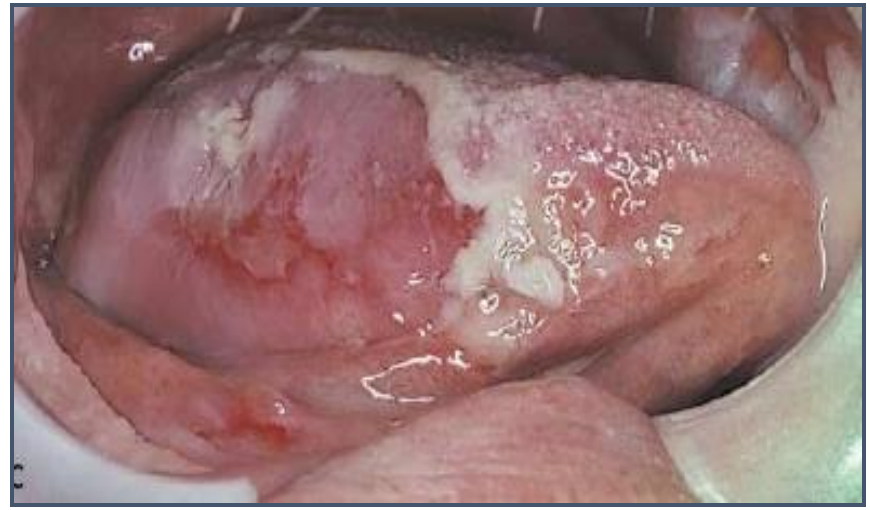


(Nishikawa T et al: J Dermatol Sci 1996; 12: 1-9)



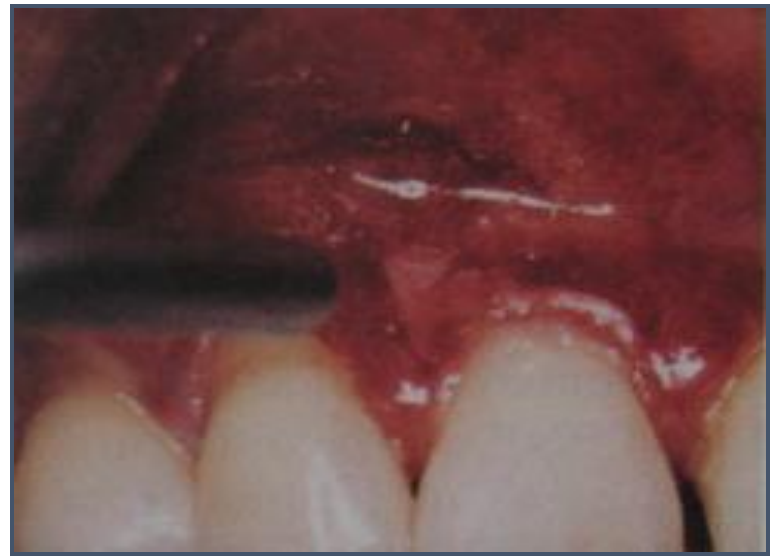






Nikolsky's sign is positive:

- Application of pressure on unaffected skin leads to bulla formation
- Application of pressure on intact bulla leads to its lateral spread



Pemphigus vegetans

-Is a relatively benign variant of pemphigus vulgaris

-2 forms:

a) Neumann type: Large bullae & denuded areas which attempt healing by developing vegetations of hyperplastic granulation tissue

b) Hallopeau type: Less aggressive with pustules being the initial lesions followed by verrucous hyperkeratotic vegetations

Oral manifestations:

-Gingival lesions may be lace-like ulcers with a purulent surface on a red base or have a granular or cobblestone appearance

-Cerebriform tongue: Sulci & gyri on dorsum

Pemphigus foliaceus (Fogo selvagem)

-loss of intercellular adhesion of keratinocytes in upper part of epidermis, resulting in formation of superficial blisters

-Chronic course with little or no mucous membrane involvement

-6 subtypes:

a) Pemphigus erythematosus

b) Pemphigus herpetiformis

c) Endemic pemphigus foliaceus

d) Immunoglobulin A pemphigus

e) Paraneoplastic pemphigus foliaceus

f) Drug induced pemphigus foliaceus

Pathogenesis:

IgG Abs against desmoglein 1 (Dsg1) expresses mainly in granular layer

of epidermis

Clinical features:

Age: Older adults

Appearance: Early bullous lesions which rupture rapidly & dry to leave masses of flakes or scales

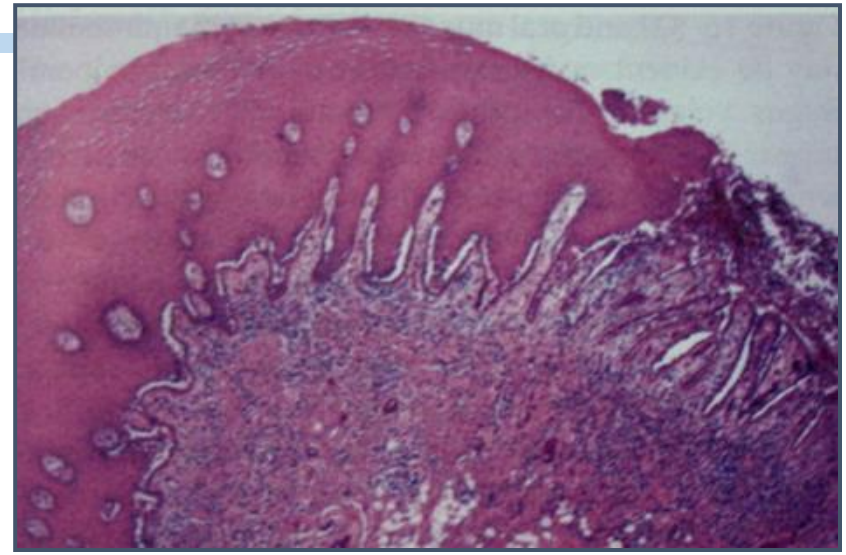
Brazilian pemphigus: Is a mild endemic form of PF found in tropical regions, particularly Brazil, that occurs often in children & frequently in family groups

Pemphigus erythematosus

-Is characterized by occurrence of bullae & vesicles with concomitant appearance of crusted patches resembling seborrheic dermatitis or lupus erythematosus

Investigations & diagnosis:

a) Nikolsky's sign



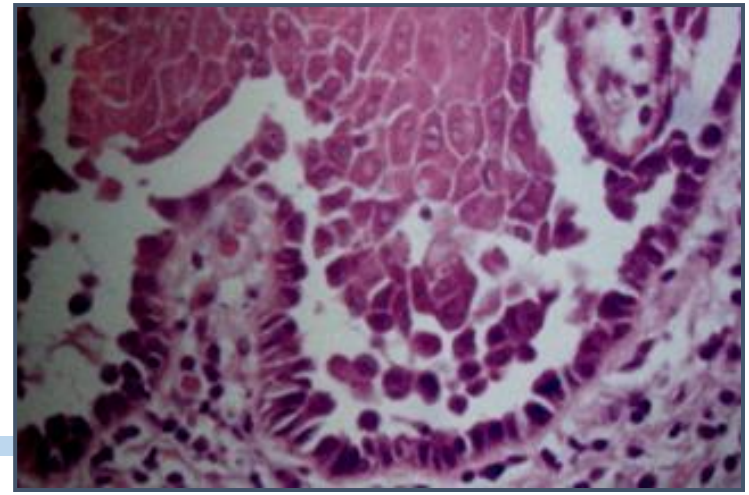
b) Histological examination

-Formation of a vesicle or bulla intraepithelially, just above basal layer producing the distinctive suprabasilar split

-Loss of cohesiveness or acantholysis

-Clumps of epithelial cells lying free within vesicular space: Tzanck cells

-Scarcity of inflammatory cell infiltrate in vesicular fluid & at base of vesicle or bulla

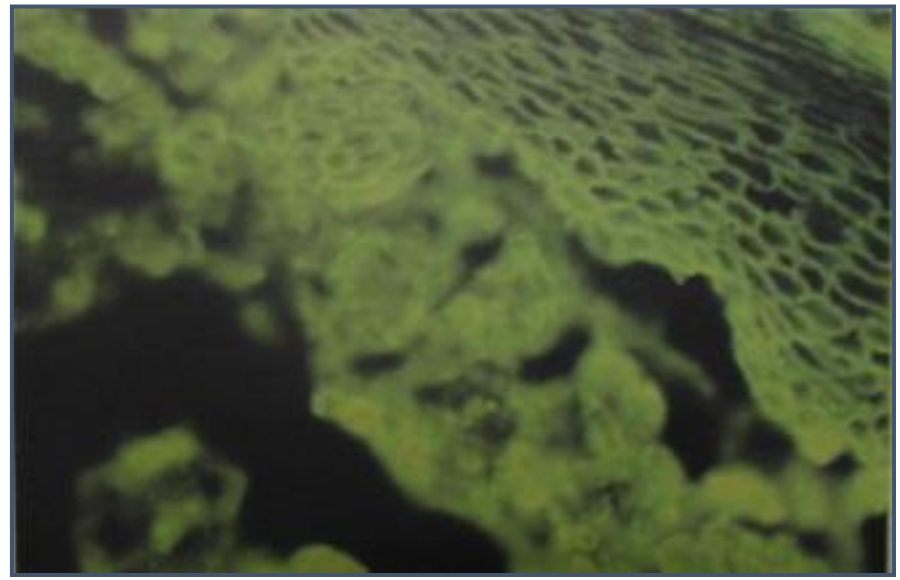


c) Tzanck test

d) Immunofluorescence:

-DIF: Presence of IgG predominantly with C3, IgA & IgM in intercellular spaces or substance in epithelium (fishnet pattern in pemphigus vulgaris)

-IIF: Antikeratinocyte Abs against intercellular substances



e) ELISA: For direct measurement of Dsg1 and Dsg3 antibodies in serum

Differential diagnosis:

* Erosive lichen planus
* Benign mucous membrane pemphigoid
* Recurrent aphthous stomatitis
* Acute viral stomatitis
* Bullous pemphigoid
* Pyostomatitis vegetans
* Wegener's granulomatosis
* Erythema multiforme
* Dermatitis herpetiformis

Erythema multiforme v/s Pemphigus

Features	Erythema multiforme	Pemphigus
Age	Primarily in young adults	Older patients
Duration	Acute disease	Chronic disease
Appearance	Erosive crusty changes	Pustulopapular vegetation
Skin changes	Target lesions	No target lesions
Nikolsky's sign	Negative	Positive

Pemphigus v/s Benign mucous membrane pemphigoid

Features	Pemphigus	BMMP
Vesicles	Intraepithelial	Subepithelial
Sites	Oral mucosa & skin	Oral mucosa & eyes
Skin changes	Common	Infrequent
Mucosal lesions	Thin-walled	Thick-walled
Scarring	Absent	Present

Management:

Guidelines: British Association of Dermatologists (2003)

- Systemic steroids
  - ✓ Mild cases: prednisolone 40–60 mg day
  - ✓ More severe cases: prednisolone 60–100 mg day

    Tapered by 5–10 mg weekly
- Pulsed i.v. steroids (severe or recalcitrant disease, particularly if unresponsive to high oral doses)
  - ✓ Intermittent administration of high doses of i.v. methylprednisolone (250–1000 mg)
  - ✓ Dexamethasone (250–1000 mg) given on 1-5 consecutive days
- Adjuvant drugs (in conjunction with CS)
  - ✓ Azathioprine 3.5–4 mg kg

- ✓ Cyclophosphamide 50–200 mg day
- ✓ Mycophenolate mofetil 2–2.5 g/day in 2 divided doses with prednisolone
- ✓ Gold 50 mg/week
- ✓ Methotrexate12 mg weekly with 40–240 mg/day of prednisolone
- ✓ Cyclosporin 5 mg/kg
- ✓ Tetracyclines 1.5 gm & Nicotinamide 2 gm
- ✓ Dapsone
- ✓ Chlorambucil 4 mg/day

- **Topical therapy**

✓ **Analgesics or anaesthetics: benzydamine hydrochloride 0.15%**

✓ **Antiseptic mouthwashes: chlorhexidine gluconate 0.12%,**
1 : 4 hydrogen peroxide solution

✓ **Topical CS:**
-Betamethasone sodium phosphate 0.5 mg tablet dissolved in 10 ml water 4 times daily for 5 min
-Triamcinolone acetonide 0.1%
-2.5 mg hydrocortisone lozenges

❖ **Others:**
-Intravenous immunoglobulin: Doses of 1.2– 2 g/kg divided over 3–5 days infused every 2–4 weeks for 1–34 cycles
-Plasmapheresis

Evidence

Authors	Martin LK, Werth VP, Villaneuva EV, Murrell DF.
Title	**A systematic review of randomized controlled trials for pemphigus vulgaris and pemphigus foliaceus.** J Am Acad Dermatol. 2011 May;64(5):903-8. Level 1a
Aim	We sought to evaluate the safety and efficacy of interventions for pemphigus vulgaris and pemphigus foliaceus.
Results	Eleven studies with a total of 404 participants were identified. Interventions assessed included prednisolone dose regimen, pulsed dexamethasone, azathioprine, cyclophosphamide, cyclosporine, dapsone, mycophenolate, plasma exchange, topical epidermal growth factor, and traditional Chinese medicine. We found some interventions to be superior for certain outcomes, although we were unable to conclude which treatments are superior overall.
Interpertation	Since pemphigus has autoimmune etiology, the above mentioned immunosupperssive therapies can be used.

Evidence

Authors	Singh S
Title	**Evidence-based treatments for pemphigus vulgaris, pemphigus foliaceus, and bullous pemphigoid: a systematic review.** Indian J Dermatol Venereol Leprol. 2011 Jul-Aug;77(4):456-69. **Level 1a**
Aim	To summarize evidence-based treatments for these diseases by performing a systematic review.
Results	No randomized controlled trials of interventions in pemphigus vegetans, pemphigus erythematosus, and epidermolysis bullosa acquisita could be found. After the second-stage screening, 12 randomized controlled trials were analyzed, which included patients with pemphigus vulgaris or pemphigus vulgaris and pemphigus foliaceus, and 7 which included patients with bullous pemphigoid.
Interpretation	Oral corticosteroid along with a steroid-sparing agent appears to be the most effective treatment for pemphigus. Azathioprine may be most effective as a steroid-sparing agent

Paraneoplastic pemphigus (Neoplasia induced pemphigus)

-Is a severe variant of pemphigus that is associated with an underlying neoplasm- non-Hodgkin's lymphoma, chronic lymphocytic leukemia or thymoma

Pathophysiology:

-Abs against Dsg3, Dsg1 & plakin proteins

-Pathologic Ags derived from associated malignancy may stimulate generation of Abs that cross-react with normal epithelial proteins

(Nikolskaia OV et al: Br J Dermatol 2003; 149(6): 1143-51)

-Some suggest the phenomenon of epitope spreading resulting from necrotic keratinocytes that enhance autoimmune response

(Bowen GM: Arch Dermatol 2000; 136(5): 652-6)

Clinical features:

Age: age at onset is 60 years (7-76 years)

Gender: M:F=1:1

Symptoms:

-Painful eroded areas

-Skin eruptions

-Pruritis

-Denudation & burning sensation

-Difficulty in chewing & swallowing

Signs:

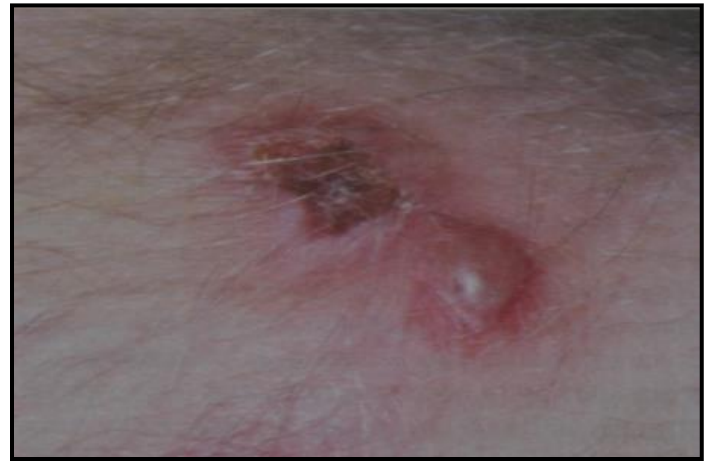
-Painful oral erosions accompanied by a generalized cutaneous eruption

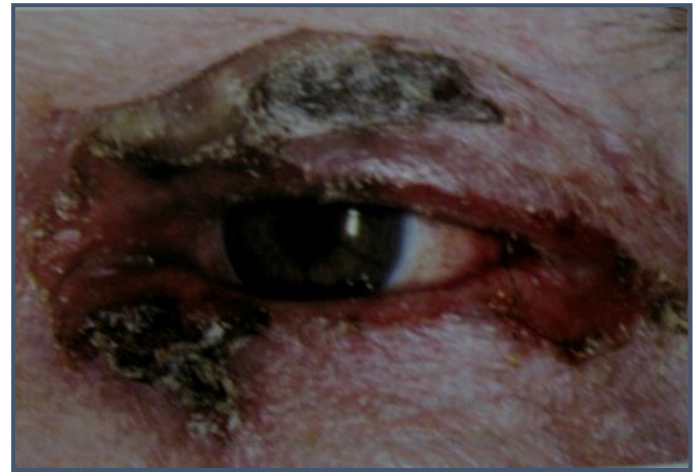
-Morbilliform, urticarial, bullous, papulosquamous, lichen planus-like or EM-like eruption

-Nose, pharynx & tonsils can be affected

-Genital mucosa may be involved

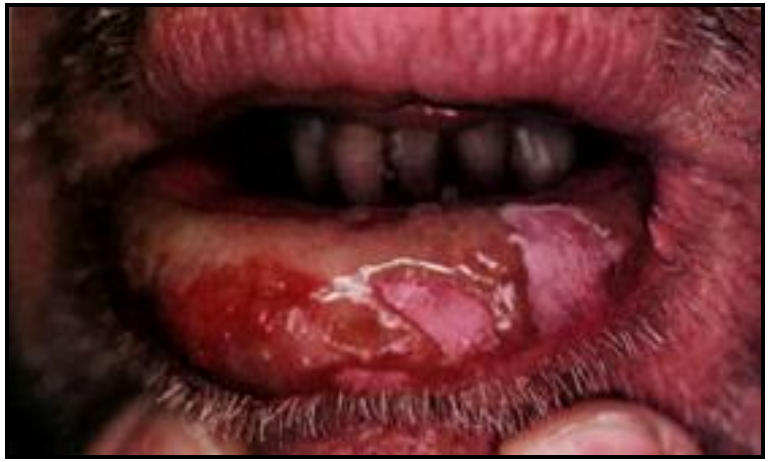
-Conjunctival involvement with scarring

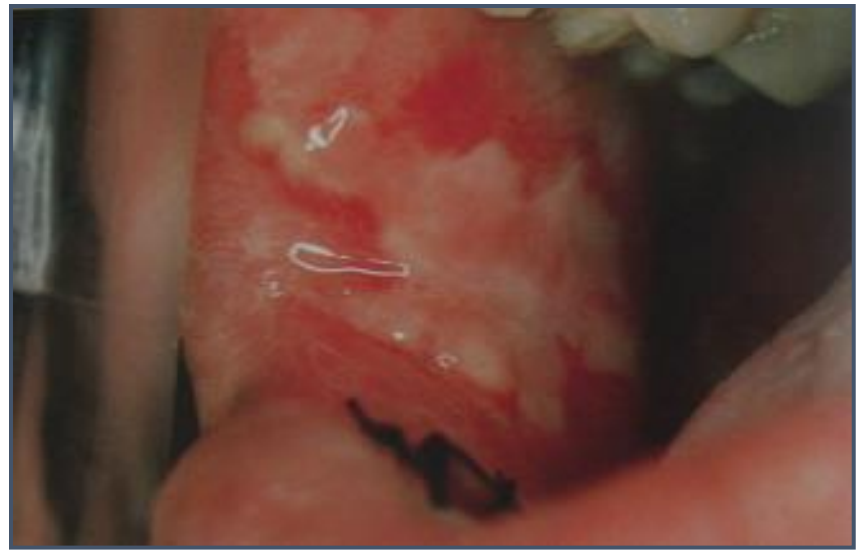




-Oral erosions affecting all surfaces of oropharynx & involves lateral borders of tongue & vermillion border of lips

-Hemorrhagic crusting of lips





-Associated malignancy: Non-Hodgkin's lymphoma, chronic lymphocytic leukemia, thymoma, giant cell lymphoma, bronchogenic squamous cell carcinoma, Waldenstrom macroglobulinemia, follicular dendritic cell sarcoma

-30 to 40% of cases develop pulmonary involvement: dyspnea, reduced pulmonary functions, radiographic signs of disease

-Inflammation & acantholysis of bronchial respiratory epithelium: Bronchiolitis obliterans

Investigations & diagnosis:

a) 4 diagnostic criteria by Anhalt (2004):

(i) Painful, progressive stomatitis, with preferential involvement of tongue

(ii) Histologic features of acantholysis or lichenoid or interface dermatitis

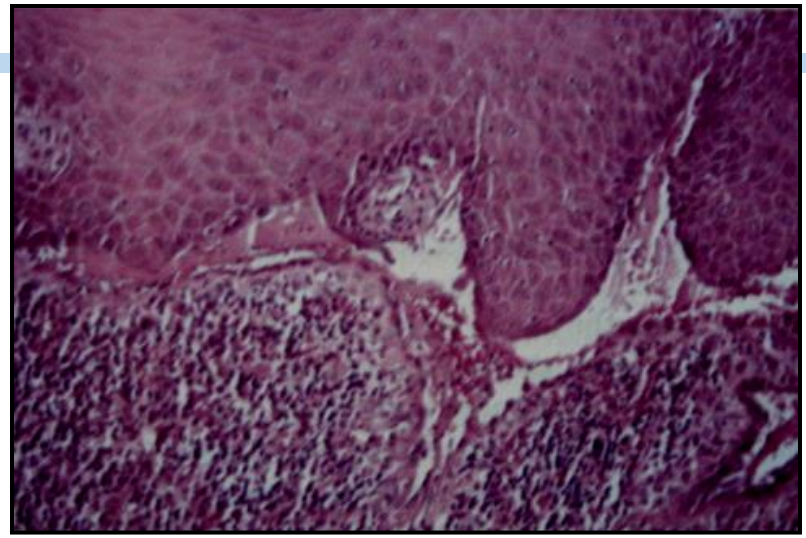
(iii) Demonstration of antiplakin autoAbs

(iv) Demonstration of underlying lymphoproliferative neoplasm

(Anhalt GJ: J Investig Dermatol Symp Proc 2004; 9(1): 29-33)

b) Histologic features:

- -Epidermal necrosis
- -Suprabasal acatholysis
- -Dyskeratotic keratinocyte
- -Vacuolar interface dermatitis
- -Lymphocytic infiltration



c) Immunofluorescence:

- -DIF shows deposition of IgG & complement along basement membrane & inter-cellularly
- -IIF demonstrates Abs that bind to epithelium, liver, heart & bladder tissues

Management:

-Warm compress, non-adherent wound dressings

-Topical antibiotic ointment

-Corticosteroids, cyclophosphamide, cyclosporin, azathioprine, gold, dapsone, high-dose IV Igs

-Plasmapheresis may be useful in controlling severe disease in initial period by reducing amount of circulating Abs

-Solid neoplasms: Curative resection

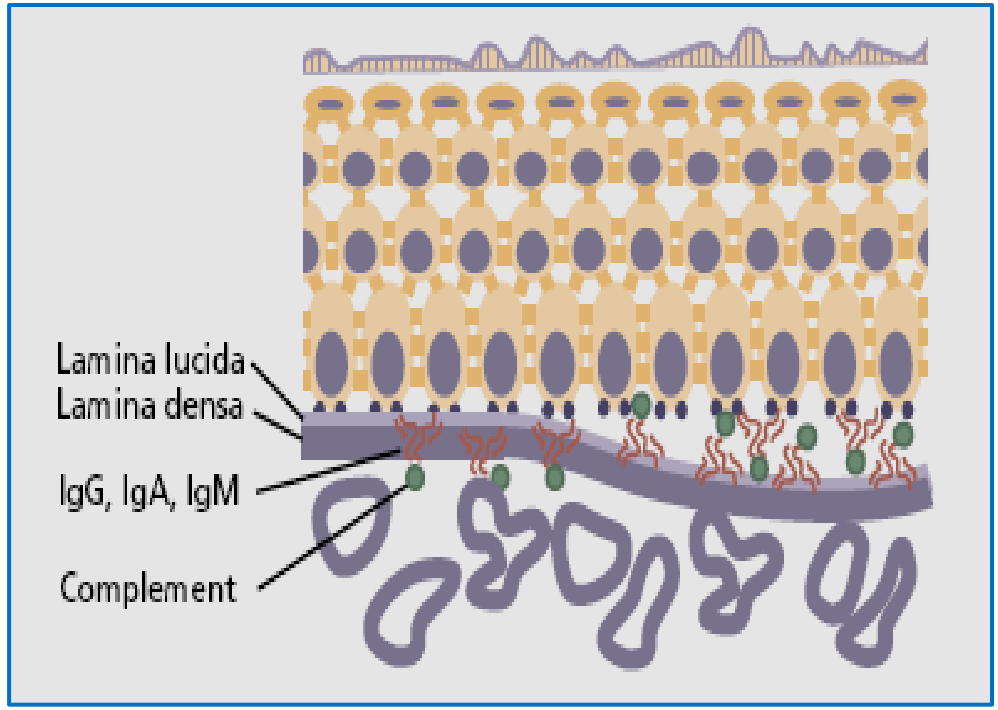
BENIGN MUCOUS MEMBRANE PEMPHIGOID/ CICATRICIAL PEMPHIGOID

-Is a chronic autoimmune subepithelial disease that primarily affects the mucous membranes, resulting in mucosal ulceration & subsequent scarring

-MMP occurs up to 3 times more frequently than pemphigus

Pathogenesis:

-Primary lesion occurs when autoantibodies IgG & A are directed against proteins-BPAG2, BPAG1, Laminin-5, α6ß4 integrin in basement membrane zone



Pathogenesis of BMMP

Clinical features:

Age: Over 50 years

Gender: Female predominance, F:M=2:1

Sites: Oral cavity, eyes, pharyngeal & laryngeal mucosa, nasal, esophageal & vaginal mucosa, 30% of cases: skin

Symptoms:

-Oral pain caused by ulcers

-Inability to effectively clean the dentition

-Bleeding gums

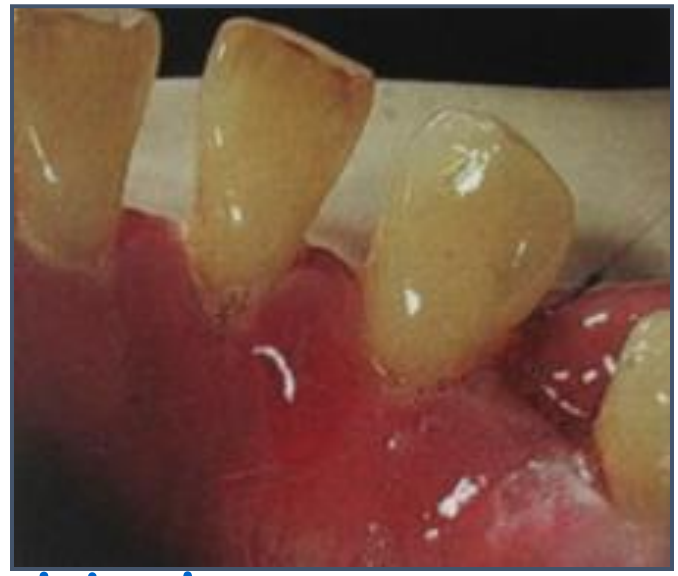
-Halitosis

-Hoarseness of voice & difficulty in breathing

-Dysphagia

Signs:

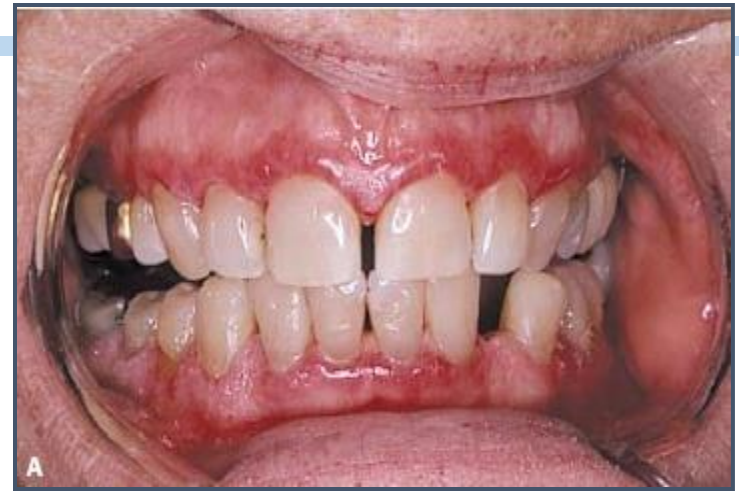
-Intact vesicles (clear fluid or blood filled) are rare in oral cavity



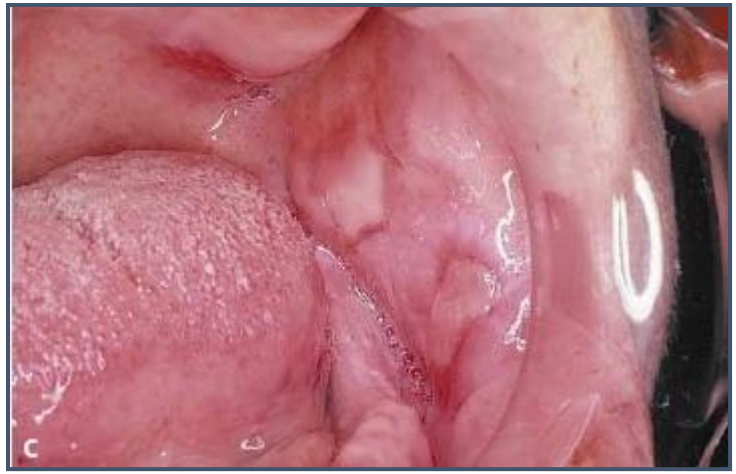
-Rupturing of vesicles into ulcer

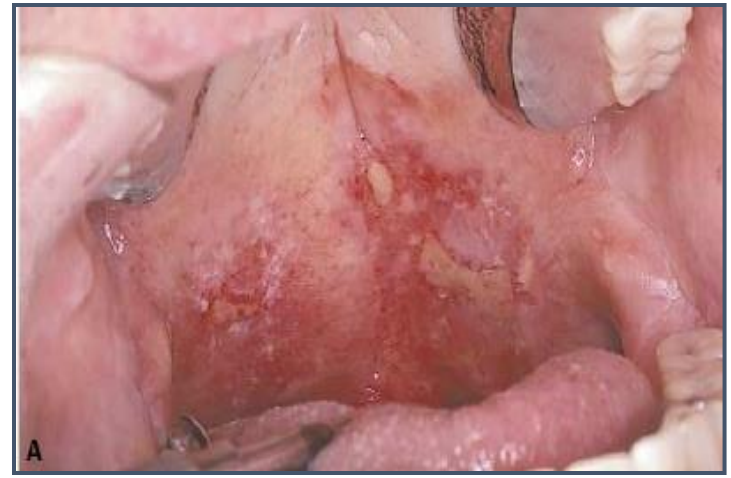
-An oral ulcer presents with a pseudomembrane

-Desquamative gingivitis with severe gingival erythema & frank ulceration



-Ulceration of buccal & labial mucosa, palate, tongue





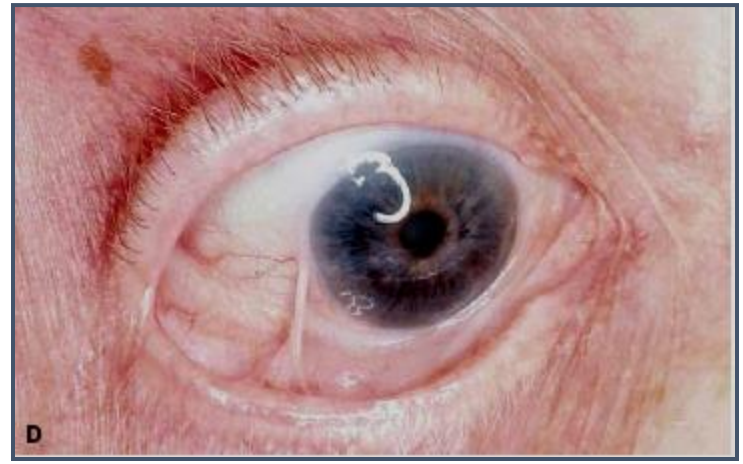
Extraoral manifestations: -

Involvement of conjunctiva, genitalia, esophagus, trachea & larynx

-Scarring & adhesions developing between bulbar & palpebral conjunctiva: Symblepharon

-Corneal damage, scarring lead to blindness

-Skin lesions in 20-30% cases



Differential diagnosis:

* Pemphigus vulgaris
* Erosive lichen planus
* Bullous pemphigoid
* Erythema multiforme
* Behçet's syndrome

Pemphigus v/s Benign mucous membrane pemphigoid

Features	Pemphigus	BMMP
Vesicles	Intraepithelial	Subepithelial
Sites	Oral mucosa & skin	Oral mucosa & eyes
Skin changes	Common	Infrequent
Mucosal lesions	Thin-walled	Thick-walled
Scarring	Absent	Present

Investigations & diagnosis:

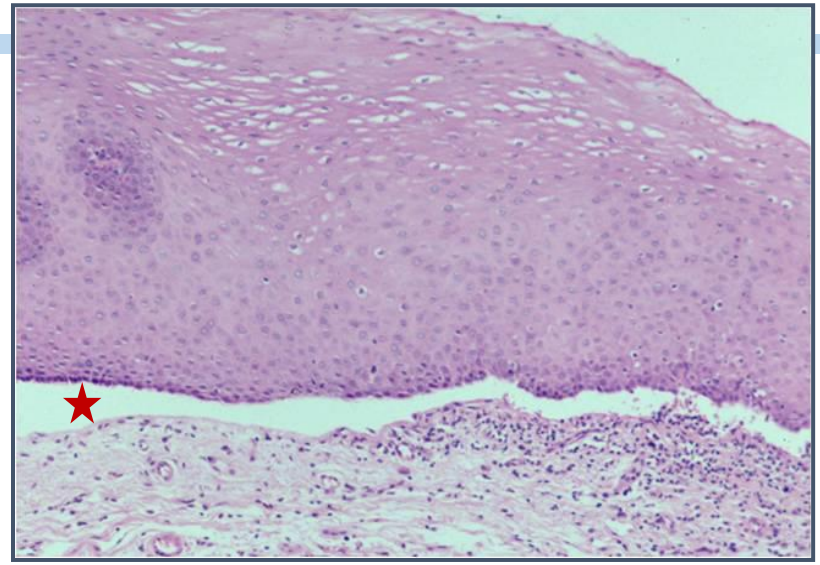
a) Histologic features:

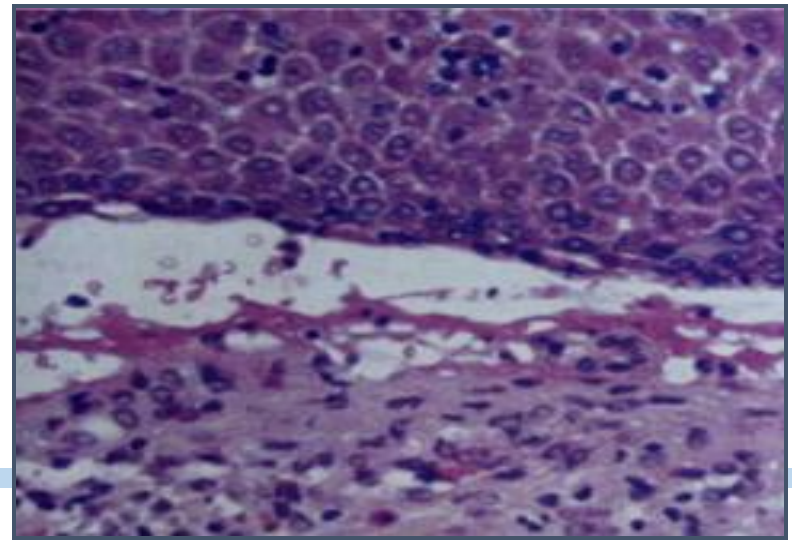
-Vesicles & bullae are subepidermal

-No acantholysis

-Basement membrane structure appears to detach with epithelium from underlying connective tissue

-Non-specific chronic inflammatory infiltrate in connective tissue, lymphocytes, plasma cells & eosinophils

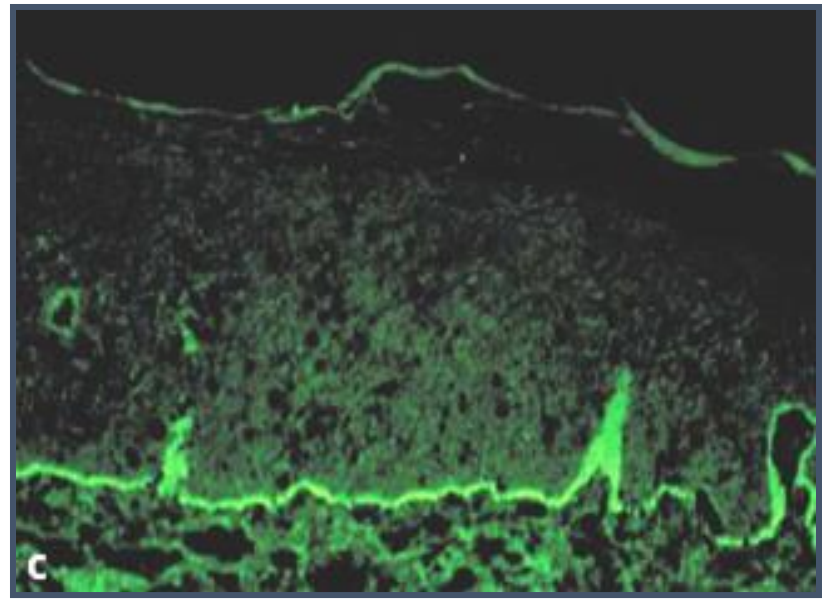




b) Immunofluorescence:

-DIF shows a uniform, apple-green, linear deposition of IgG & complement along basement membrane zone

-IIM demonstrates presence & titres of circulating IgG & IgA autoAbs to BMZ Ags



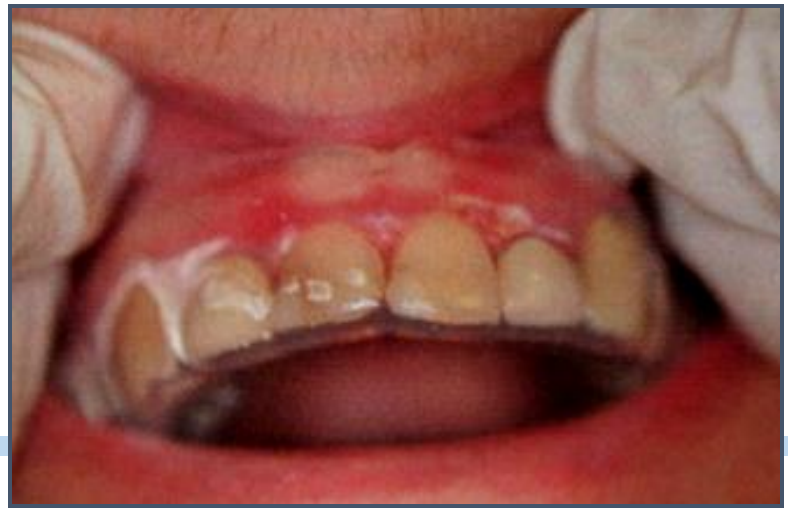
Management:

Mild oral lesions:

-Topical high-potency or ultra-high-potency steroids

- Fluocinonide 0.05% (Lidex)
- Clobetasol propionate 0.05% (Temovate)
- Betamethasone dipropionate 0.05% (Diprosone)

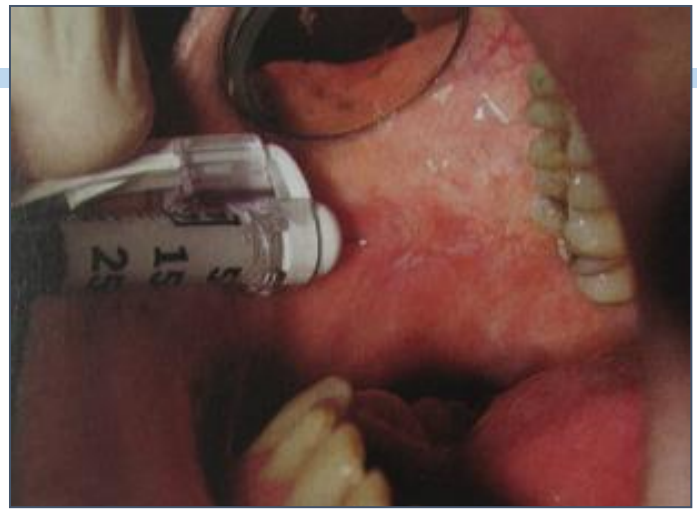
-Desquamative gingival lesions: Topical steroids in a soft occlusal splint covering involved gingiva



Recalcitrant cases:

-Intralesional corticosteroids

- ✓ Triamcinolone diacetate 25 mg/ml (Aristocort)
- ✓ Betamethasone sodium phosphatefoetamethasone acetate 6mg/ml (Celestone)



Extraoral lesions:

-Systemic steroids

- ✓ Prednisone 1 mg/kg/day tapered each following week by 10 mg & discontinued within 8-12 wk

-Dapsone 100 mg/day, azathioprine, mycophenolate mofetil, methotrexate, allopurinol, nicotinamide, minocycline

BULLOUS PEMPHIGOID (PARAPEMPHIGUS)

-Is a chronic, autoimmune, subepidermal, blistering skin disease that rarely involves mucous membranes

Pathogenesis:

IgG autoAbs against hemidesmosomal bullous pemphigoid Ags BP230 & BP180 results in defect in lamina lucida region of basement membrane

Clinical features:

Sites: Scalp, arms, legs, axilla, grion

Symptoms:

-Generalized skin rash, Pruritis

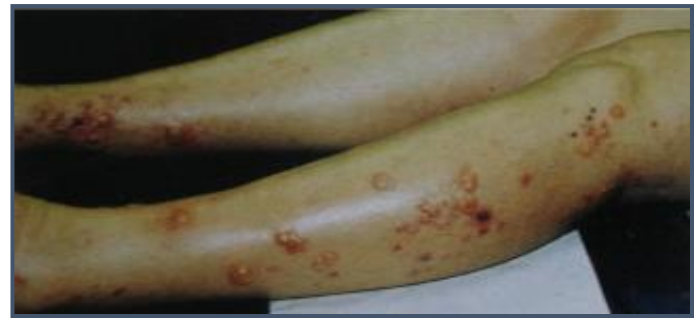
-Appearance of blisters

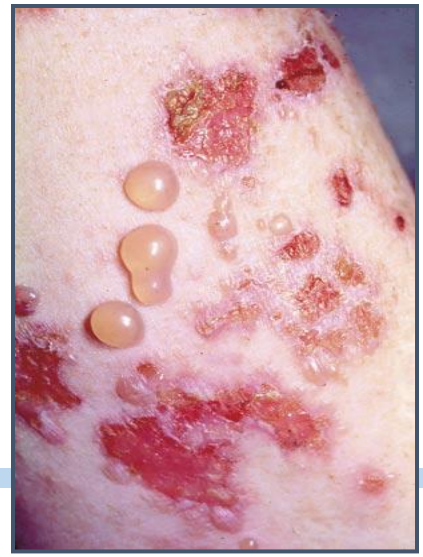
Signs:

-Urticarial or erythematous rash on limbs persisting for several weeks to months

-Vesicles & bullae in the prodromal skin lesions & in normal skin

-Ruptured vesicles leave a raw, eroded area

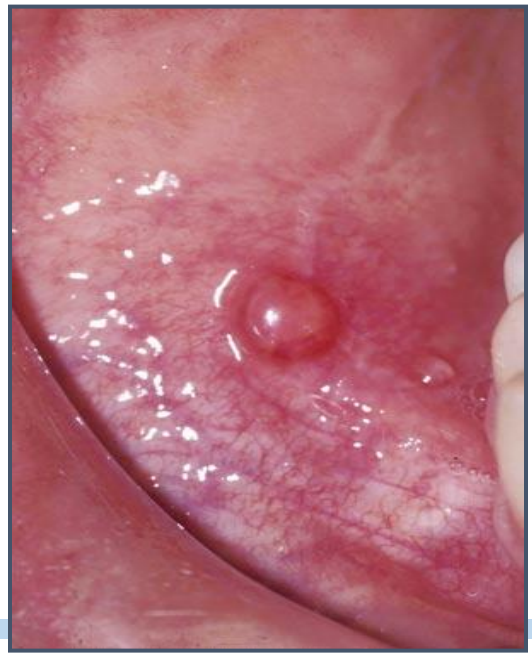


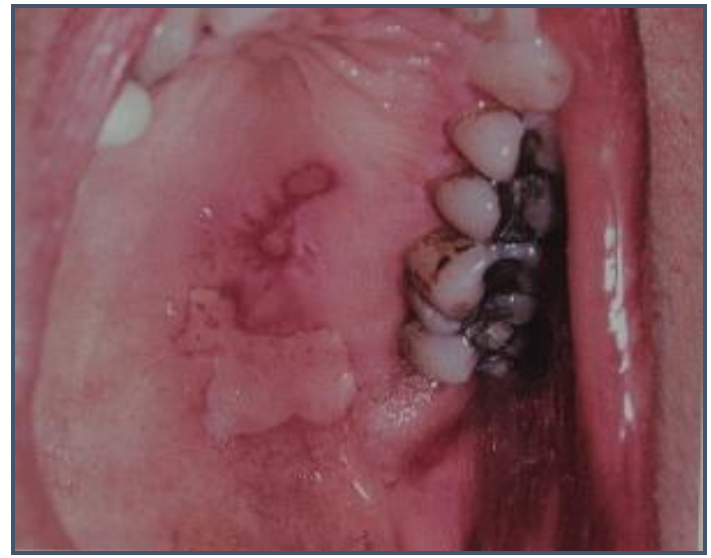


-Oral lesions are vesicles, areas of erosion & ulceration

-Involvement of buccal mucosa, palate, floor of mouth & tongue

-Gingiva: Erythematous & may desquamate





Histologic features:

-Vesicle & bullae are subepidermal & non-specific

-No acantholysis

-Epithelium appears relatively normal

-Basement membrane remains attached to the connective tissue rather than to overlying separated epithelium

-Vesicle contains fibrinous exudate admixed with occasional inflammatory cells

Immunofluorescence:

-DIF demonstrates IgG bound to basement membrane zone

-IIF demonstrates circulating IgG Abs against BM Ag

Differential diagnosis:

* Erosive lichen planus
* Mucous membrane pemphigoid
* Pemphigus vulgaris
* Erythema multiforme

Management:

- Localized lesions: High-potency topical steroids
- Moderate cases: Dapsone; combination of tetracycline 1.5gm & nicotinamide 2gm
- Severe cases: Systemic steroids, immunosuppressive agents (azathioprine, cyclophosphamide, mycophenolate)

SOLITARY ULCERS

TRAUMATIC ULCER

-Trauma is the most common cause of ulceration of the oral mucosa

-May be either factitial or iatrogenic in their origin

-May result from physical, chemical, electrical or thermal insults to tissue

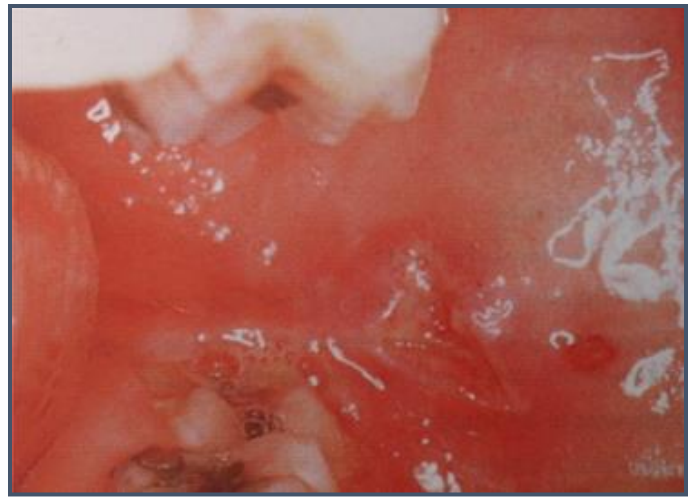
-A traumatic ulcer in anterior portion of tongue of infants with natal teeth: Riga-Fede disease

Clinical features:

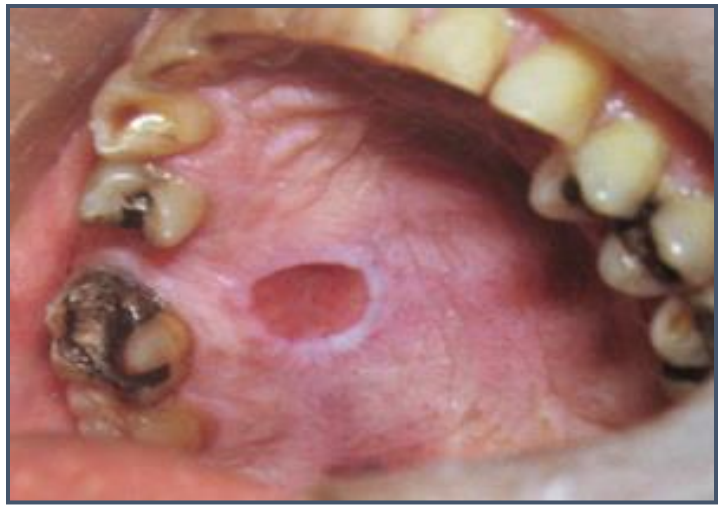
Traumatic ulcer

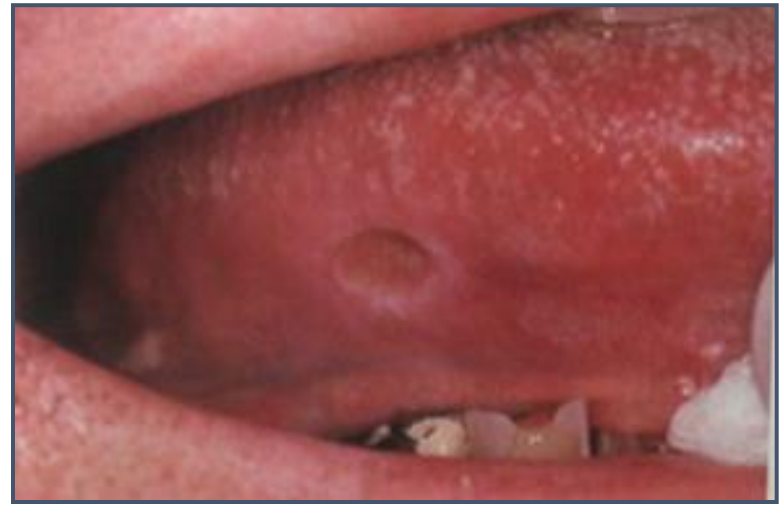
Acute ulcer **Chronic ulcer**

- **Acute**



- **Chronic ulcer:**





Histoplasmosis

- fungus *Histoplasma capsulatum*
- a dimorphic fungus that grows in the yeast form in infected tissue.
- Infection results from inhaling dust contaminated with droppings, particularly from infected birds or bats.
- mild, manifesting as a self-limiting pulmonary disease that heals to leave fibrosis and calcification similar to tuberculosis.

- progressive disease results in cavitation of the lung and dissemination of the organism to the liver, spleen, adrenal glands, and meninges
- develop anemia and leukopenia secondary to bone marrow involvement
- ORAL MANIFESTATIONS: may appear as a papule, a nodule, an ulcer, or a vegetation. If a single lesion is left untreated, it progresses from a firm papule to a nodule, which ulcerates and slowly enlarges.
- The cervical lymph nodes are enlarged and firm.

Blastomycosis

- ***Blastomyces dermatitidis.*** **This dimorphic organism can grow in either a yeast or as a mycelial form.**
- **The organism is found as a normal inhabitant of soil**
- **by inhalation: this causes a primary pulmonary infection**

-Is a relatively uncommon disease caused by dimorphic fungus, Blastomyces dermatitidis

Inhalation of spores

↓

Grow as yeasts in lungs

↓

Infection contained in lungs

or Hematogenous dissemination

-Occurrence is rare in immunocompromised patients

Clinical features:

Age: Middle age group

Gender: Male predilection, M:F= 9:1

Sites: Lungs

Dissemination sites: Skin, bone, prostate, meninges, oropharyngeal mucosa & abdominal organs

Symptoms:

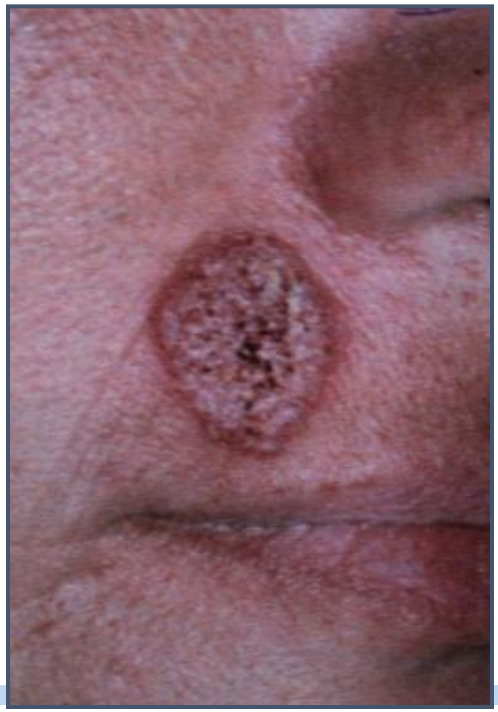
-Asymptomatic

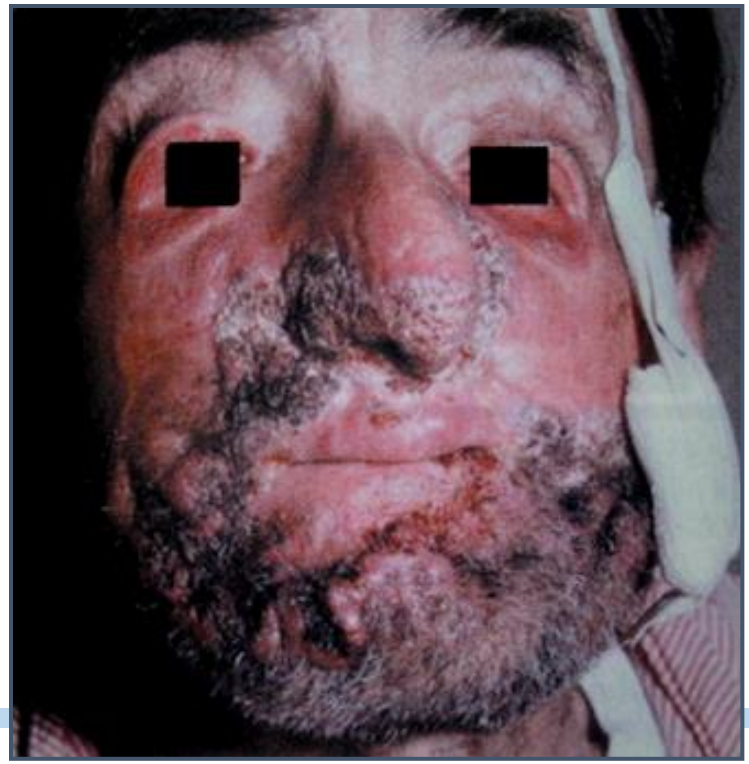
- **Acute blastomycosis: High fever, chest pain, malaise, night sweats, productive cough with mucopurulent sputum**
- **Chronic blastomycosis: Low-grade fever, night sweats, weight loss, productive cough**

Signs:

Cutaneous lesions:

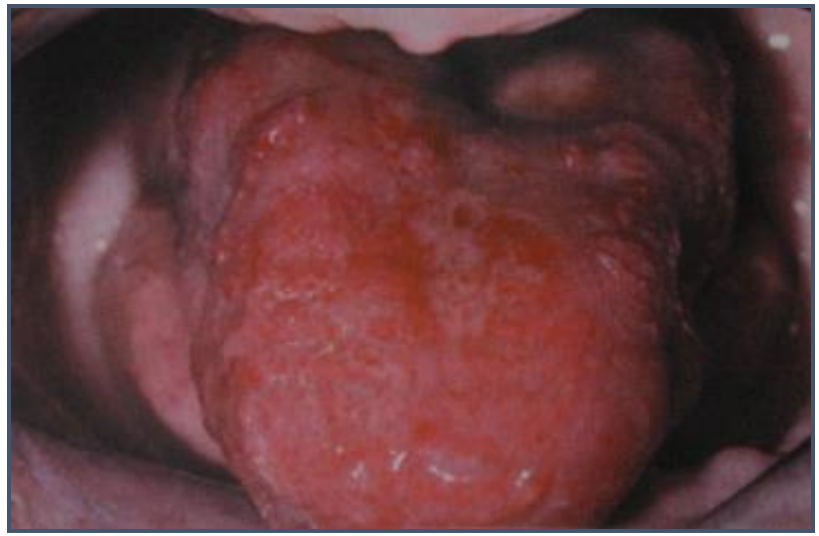
Begin as erythematous nodules that enlarge, becoming verrucous or ulcerated





Oral lesions:

-Result from either extrapulmonary dissemination or local inoculation with the organism



-Have an irregular, erythematous or white intact surface or appear as painful ulcerations with irregular rolled borders

Management:

- Symptomatic acute blastomycosis

Systemic amphotericin B administration is indicated if patient:

a) Is seriously ill

b) Is not improving clinically

c) Is ill for more than 2 or 3 weeks

- Chronic blastomycosis

-Itraconazole or ketoconazole (mild to moderate) for 6-12 months

-Amphotericin B (severe) for 10 weeks

Differential diagnosis:

* Squamous cell carcinoma
* Tuberculosis
* Histoplasmosis
* Mucormycosis
* Cryptococcosis

Mucormycosis(phycomycosis)

-Is an opportunistic, frequently fulminant, fungal infection that is caused by Zygomycetes

(saprophytic fungus that normally occurs in soil or as a mold on decaying food.)

-Occurs more commonly in uncontrolled Insulin-dependent

diabetics with ketoacidosis & immunocompromised patients

Clinical features:

-Rhinocerebral form is most common

Symptoms:

-Nasal obstruction

-Bloody nasal discharge

-Facial pain or headache

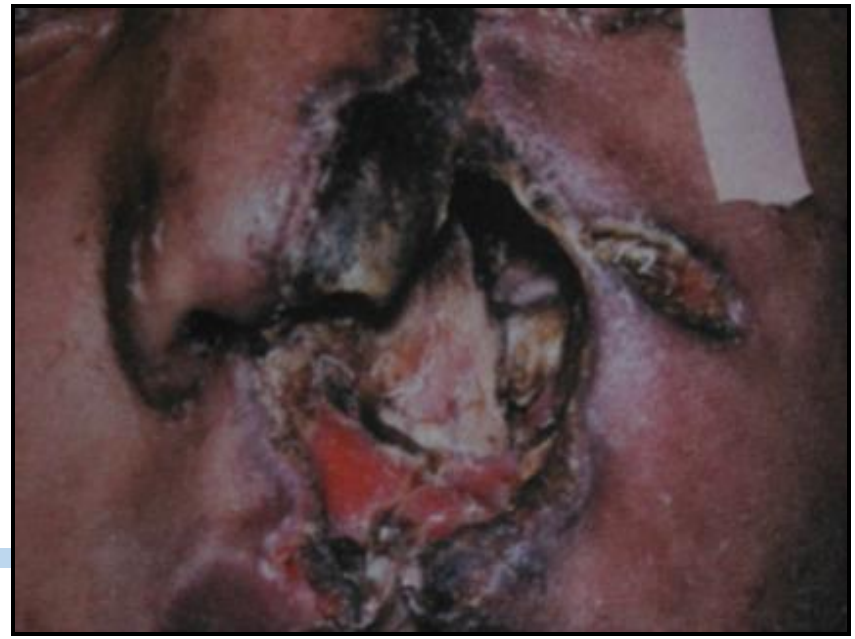
-Facial swelling

-Visual disturbances

Signs:

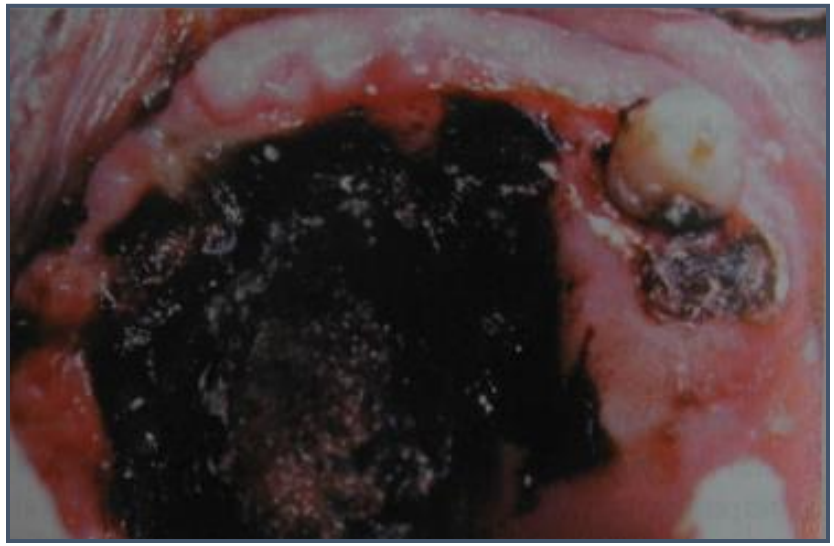
-Cranial nerve involvement

-With disease progression, disease of cranial vault, blindness, lethargy, seizures

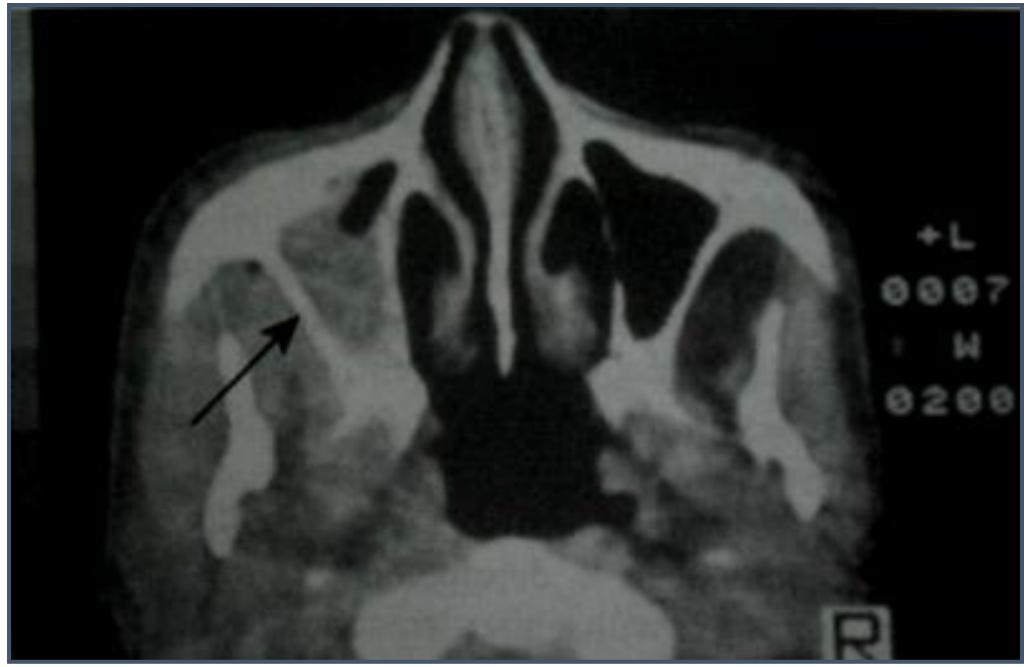


-Maxillary sinus involvement: Intraoral swelling of mx alveolar process, palate or both

-Palatal ulceration with black & necrotic surface



-Radiographically, opacification of sinuses with patchy effacement of bony walls of sinuses

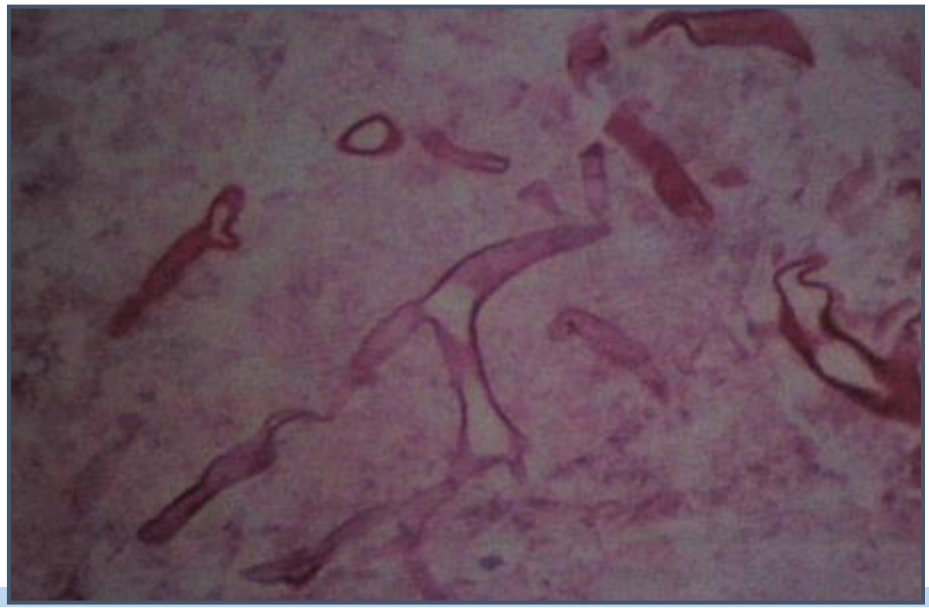


Histologic features:

-Extensive necrosis with numerous large (6-30 µm) branching, non-septate hyphae at the periphery

-Hyphae tend to branch at 90°

-Neutrophilic infiltrate predominates in viable tissue



Differential diagnosis:

* Squamous cell carcinoma

* Necrotizing sialometaplasia

* Aphthous Ulcer

Management:

-Radical surgical debridement of infected, necrotic tissue

-Systemic administration of amphotericin B up to 3 months

-Control of underlying systemic disease

-Prosthetic obturation for palatal defects

MIDLINE LETHAL GRANULOMA

-Also known as Malignant granuloma, Lethal granuloma, Midline lethal granulomatous ulceration

-Is an idiopathic progressive destruction of nose, paranasal sinuses, palate, face & pharynx

Pathogenesis:

-Is due to a dysfunction of the immune mechanisms normally responsible for granuloma formation (Williams HL et al 1950)

-A vascular allergy occurs, either Arthus phenomenon or periarteritis nodosa

-Hyperimmune tissues become necrotic

Clinical features:

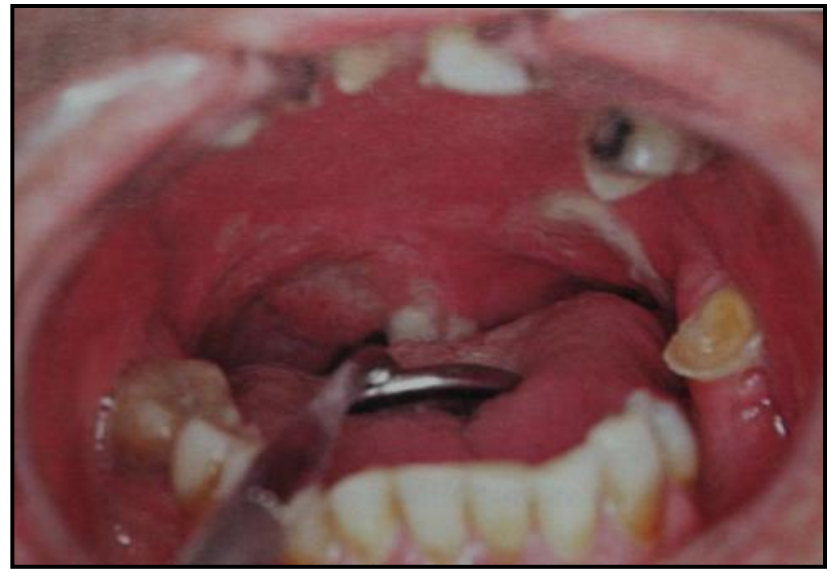
Site: Middle of oronasal region

Symptoms:

-Ulcers of palate

-Stuffiness of nose

-Discharge from eyes & nose

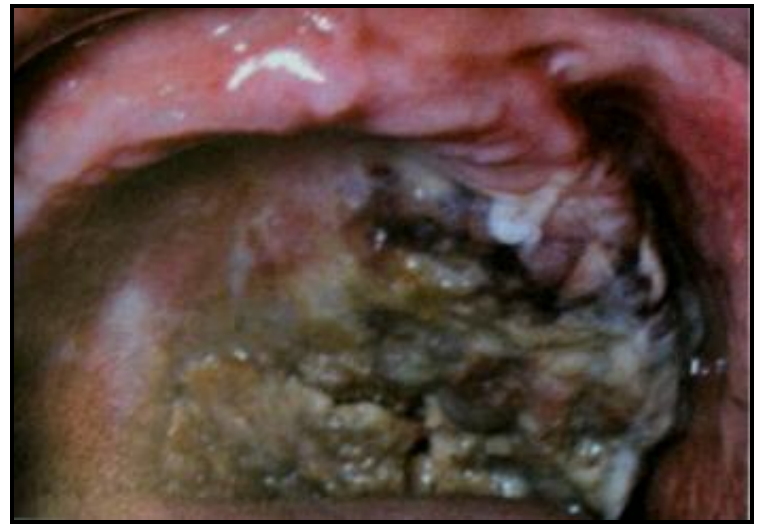


Signs:

-Superficial ulceration of palate or nasal septum

-Ulceration spreads from palate to inside of nose

-Palatal, nasal & malar bones may be involved, undergo necrosis & sequestrates



-Extensive destruction

-Perforating sinus may develop

-Sloughing of soft tissue of face

-Secondary infection

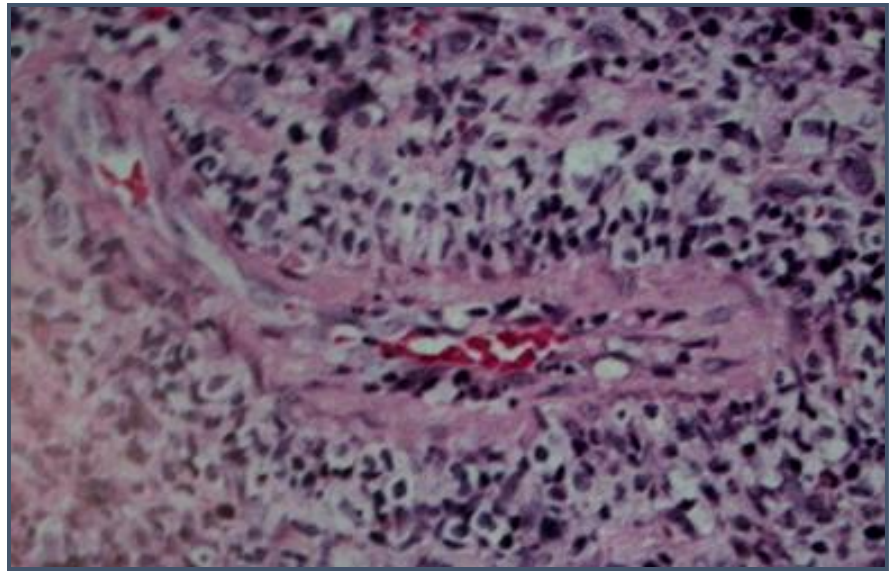
-Life-threatening hemorrhage

Histologic features:

-Extensive necrosis with infiltration of some inflammatory cells

-Formation of occasional new capillaries

-Angiocentric inflammation is common



Differential diagnosis:

*** Wegener's granulomatosis**

*** Noma**

Management:

-High-dose radiation therapy

-Corticosteroids & antibiotics

REFERENCES

- Martin S. Greenberg, Michael Glick. Burket's Oral Medicine: Diagnosis and Treatment. 10th ed. Hamilton: BC Decker Inc; 2003.
- Soames J V, Southam J C. Oral Pathology. 2nd ed. New York: Oxford Medical Publications; 1993.
- Brad W. Neville, Douglas D. Damm, Carl M. Allen, Jerry E. Bouquot. Oral and Maxillofacial Pathology. Philadelphia: W. B. Saunders Company;1995.
- Gary C. Coleman, John F. Nelson. Principles of Oral Diagnosis. St.Louis: Mosby-Year Book;1993.
- Joseph A. Regezi. James J. Scuibba. Richard C.K. Jordan. Oral Pathology: Clinical Pathological Correlations. 4th ed. St.Louis: Saunders;2003
- Singh s et,al.Evidence-based treatments for pemphigus vulgaris, pemphigus foliaceus, and bullous pemphigoid: a systematic review. Indian J Dermatol Venereol Leprol. 2011 Jul-Aug;77(4):456-69.
- Martin L.K. et,al.A systematic review of randomized controlled trials for pemphigus vulgaris and pemphigus foliaceus. J Am Acad Dermatol. 2011 May;64(5):903-8.

MCQs

CCES

1. Suprabasilar split is seen in
   a. Pemphigus
   b. Cicatrical Pemphigoid
   c. Both of the above
   d. None of the above

2. Neumann & Hallopeau type lesions are types of
   a. pemphigus vulgaris
   b. cicatrical pemphigoid
   c. pemphigus vegitans
   d. bullous pemphigoid

3. Desquamative gingivitis is a feature of

a. pemphigus vulgaris

b. pemphigoid

c. Erosive lichen Planus

d. All of the above

4. Traumatic ulcer can be due to

a. sharp cusps of teeth

b. heat

c. chemical

d. all of the above

5. Scarring is a feature of

a. pemphigus vulgaris

b. Lichen planus

c. Cicatrical pemphigoid

d. None of the above