DESQUAMATIVE GINGIVITIS

Dr.Hema.P
Senior Lecturer
Department of Periodontics

Learning Objectives:

- Introduction
- Definition
- History
- Epidemiology
- Classification
- Conditions associated with desquamative gingivitis and their management
- Special considerations in periodontal care
- Diagnostic Algorithm
- Summary

- Desquamation - latin word *dequamare (scraping fish flakes)*
- Desquamation – loss of epithelium in small & large amounts, peeling of skin & exfoliation.
- Chronic Desquamative gingivitis

DEFINITION

"Desquamative gingivitis" is a descriptive term, that indicates the presence of erythema, desquamation, erosion,and blistering of the attached gingiva and marginal gingiva.

Prinz 1932

Unusual, unique, nonplaque associated gingivitis characterized by intense diffuse erythema and desquamation (peeling) of surface epithelium. It is often associated with ulceration of the marginal and attached gingiva. It is not a specific disease entity but a clinical manifestation of a variety of conditions.

- Carranza 12th edition

HISTORY

First recognised and reported by **Tomes and Tomes in 1894**

Term Chronic Desquamative gingivitis coined by **Prinz** in 1932
" peculiar condition characterised by intense erythema, desquamation & ulceration of free & attached gingiva."

Early investigators – single cause (estrogen deficiency, hypothyroidism, nutritional deficiency).

Foss et al and **Glickman & Smulow** – degenerative disease

McCarthy et al 1960- Not a specific disease entity but a gingival response associated with a variety of conditions.

Epidemiologic features

- Highest prevalence - lichen planus and mucous membrane pemphigoid

Disorder	Number of cases of desquamative gingivitis
Mucous membrane pemphigoid	35-48%
Oral lichen planus	24- 45%
Pemphigus vulgaris	3-15%

GENERAL EPIDEMIOLOGICAL FEATURES

4^{TH} & 6^{TH} DECADE OF LIFE

FEMALE PREDILICTION
(EM exception)

CLINICAL APPEARANCE

- Fierry red & Glazed
- B > L
- Atrophic/Eroded
- Loss of stippling
- Desquamate with minimal trauma
- Painful/burning sensation - exacerbated – spicy food
- Attached gingiva
- Clinical appearance - Not altered by conventional trt

CLASSIFICATION

BASED ON ETIOLOGY(McCarthy et al)

1) Dermatoses

- a) Benign mucous membrane pemphigoid
- b) Pemphigus
- c) Lichen Planus
- d) Erythema Multiformae
- e) Lupus Erythematosis
- f) Linear IgA disease

2) Hormonal influence

- a) Estrogen deficiency following oophorectomy & in post menopausal women
- b) Testosterone imbalance
- c) Hypothyroidism

3) Abnormal response to irritation

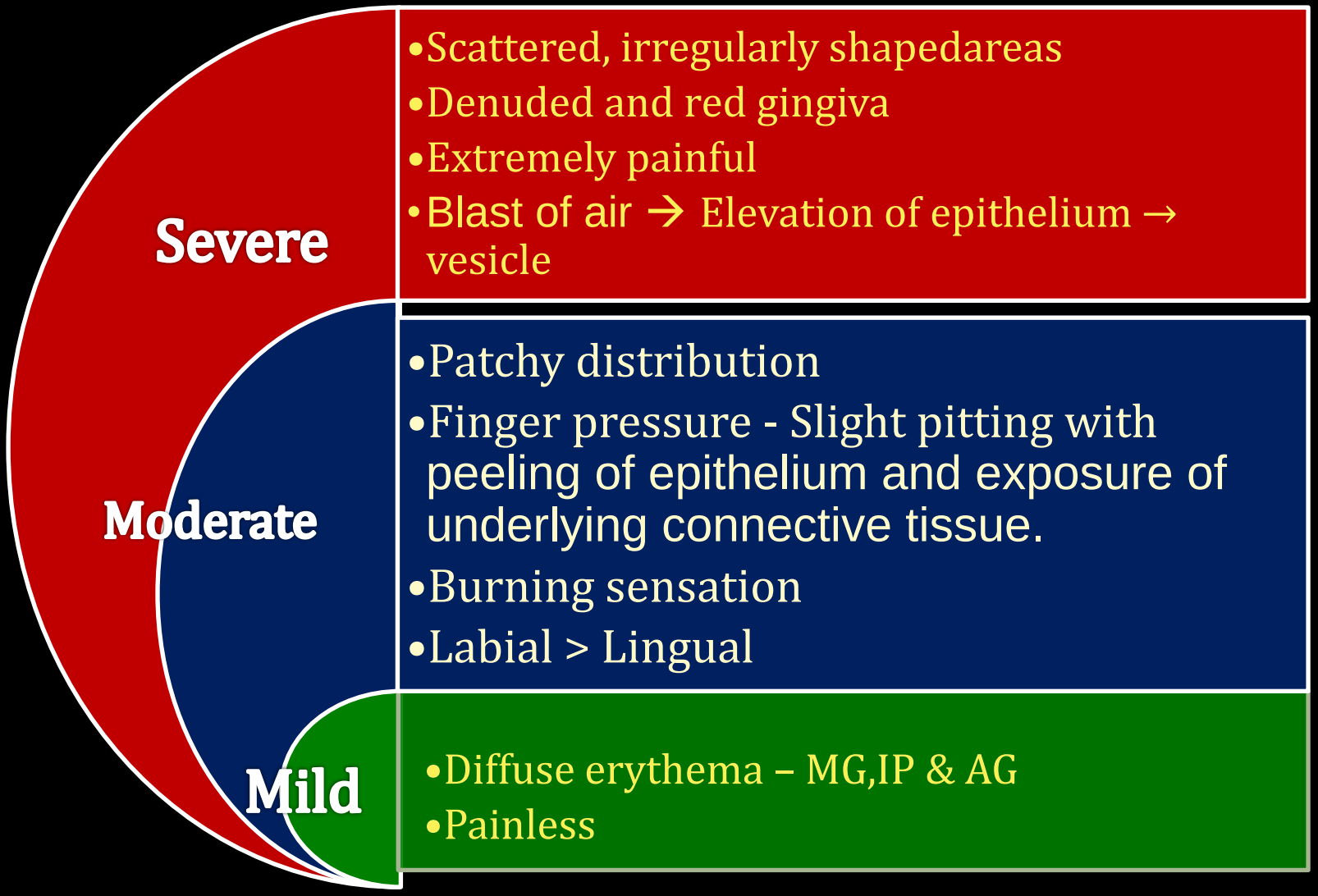
4) Chronic infection

- a) Tuberculosis
- b) Chronic candidiasis
- c) Histoplasmosis

5) Idiopathic causes

6) Aging

BASED ON CLINICAL FEATURES: (GLICKMAN AND SMULOW)



CONDITIONS ASSOCIATED WITH DESQUAMATIVE GINGIVITIS

- Oral lichen planus
- Phemphigus vulgaris
- Mucous membrane phemphigoid
- Bullous Pemphigoid
- Erythema multiforme
- Lupus erythematosus
- Chronic ulcerative stomatitis
- Linear IG A disease
- Dermatitis herpetiformis
- Epidermolysis bullosa
- Graft versus host disease

J Periodontol 2003;74:1545-1556.

LICHEN PLANUS

- Lichen planus (LP) - a chronic inflammatory mucocutaneous disease.

- First described in 1869 by the British physician Erasmus Wilson

- It is primarily a disease affecting the skin and mucosal surfaces including the oral cavity

- Can also involve other sites like the scalp and nails.

Approx 0.1 – 4% of population.

Prevalence of OLP in India - 2.6%.

40% of lesions occur on both oral and cutaneous surfaces,

35% occurs on cutaneous surfaces alone

25% occur on oral mucosa alone

- Chronicity
- Symmetrical appearance
- Multi – site involvement

Burkets oral medicine- 12th edition,
Carranza 10th edition

PATHOGENESIS

Expression of a keratinocyte-derived antigen

Induced by systemic drugs, contact allergens, physical trauma, viral or bacterial

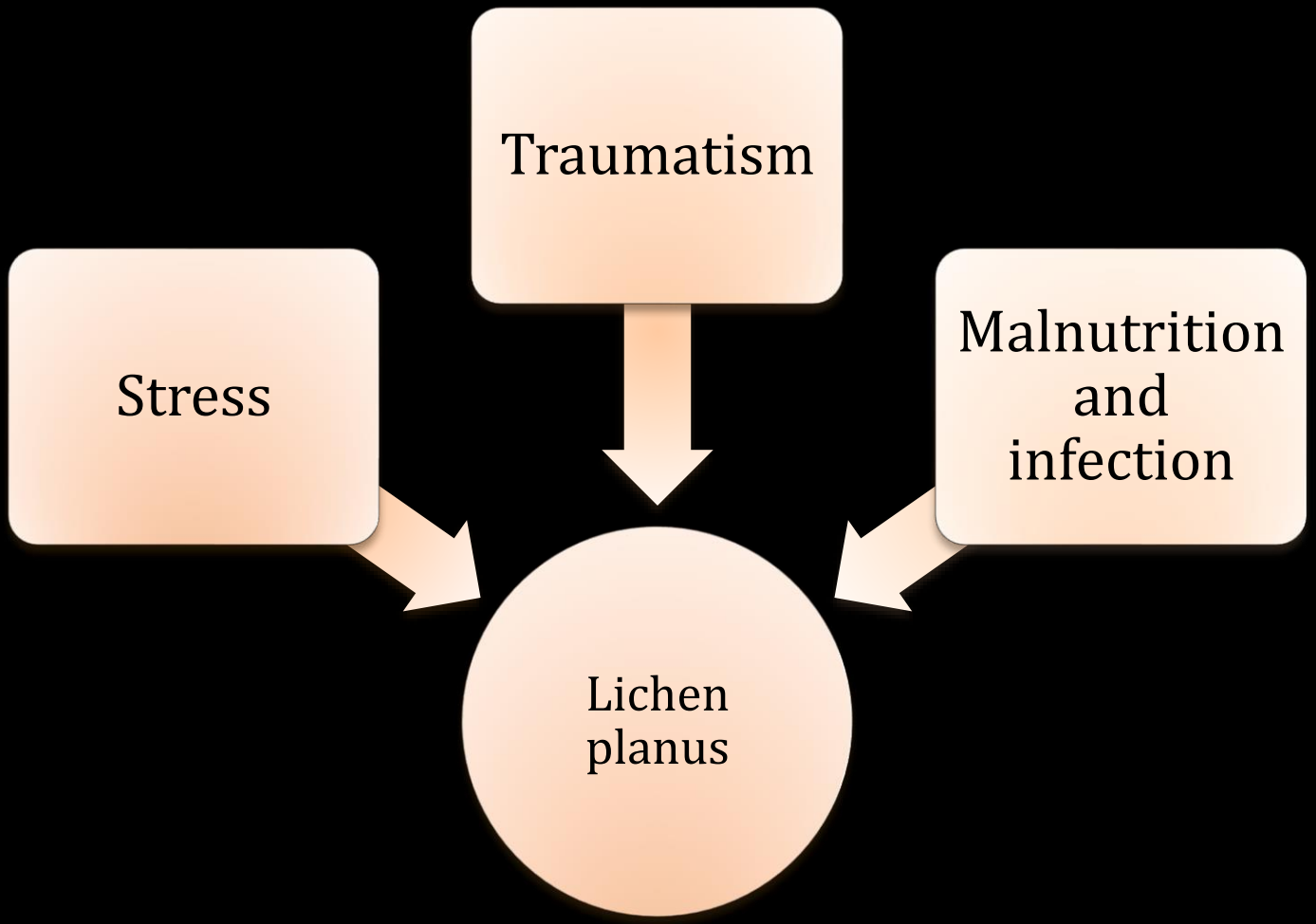
T-cell recognition

cytokine expression, including TNF-alpha, IL, and upregulation of MMP

apoptosis of keratinocytes

keratinocyte death

- Exacerbation



ORAL LICHEN PLANUS

T cell mediated auto immune response

inflammatory mediator nuclear factor kappaB

transforming growth factor control pathway

the hyperproliferation of keratinocytes,

white lesions in OLP

T cell mediated auto immune response

autocytotoxic CD8+ Tcells

apoptosis of oral epithelial cells

Chronic inflammation

Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation. J Oral Sci 2007;49:89106

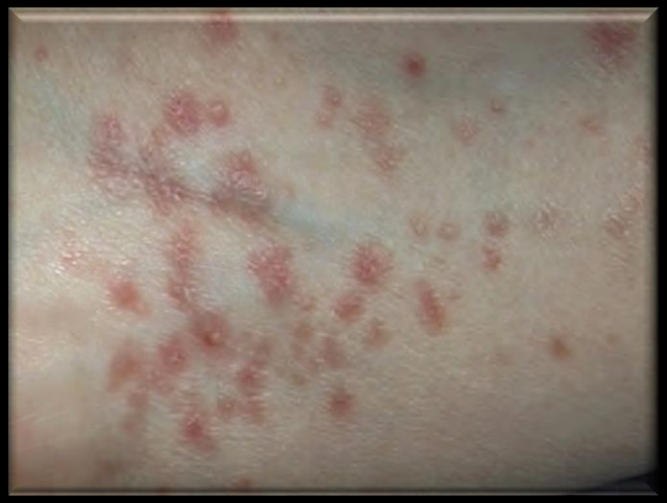
CLINICAL FEATURES

SKIN LESION:

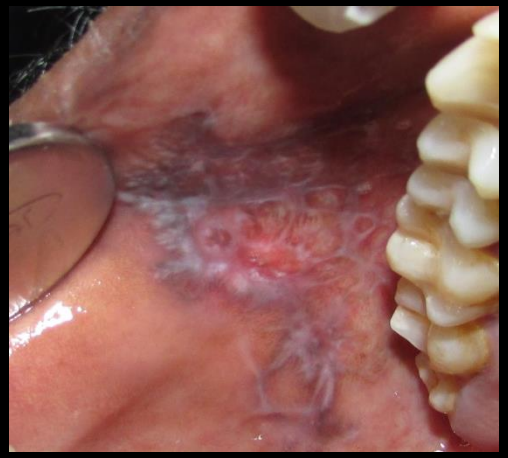
- Classic appearance-
  - pruritic erythematous to violaceous papules that are flat topped that have a predilection for the trunk and flexor surfaces of arms and legs

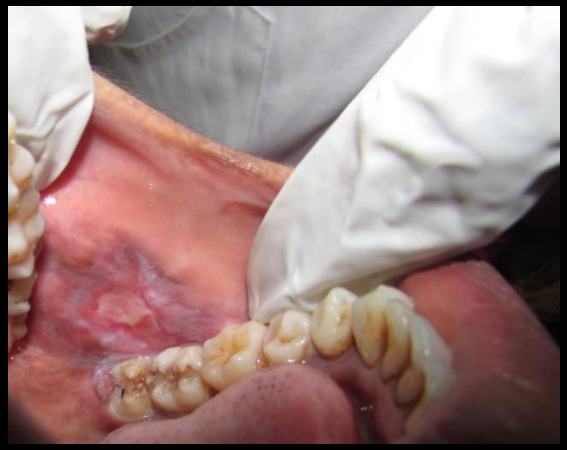
Other sites :

- nails, scalp, genital mucosa

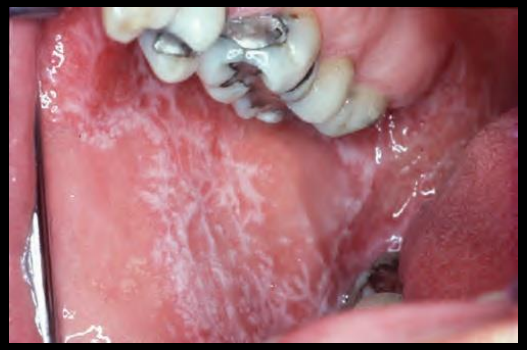


- ORAL LESION:
- Majority of the patients- Oral Lichen Planus precede Cutaneous LP
- 65% women affected in the age range of 40 -70 yrs.
- common site: buccal mucosa & lateral borders of tongue & Gingiva
- Appear as radiating , white / grey velvetty thread like papule- Bilaterally symmetric
- At the intersection of this white line, tiny white elevated dots are frequently present-WICKHAMS STRIAE
- Presenting symptom- Burning sensation.

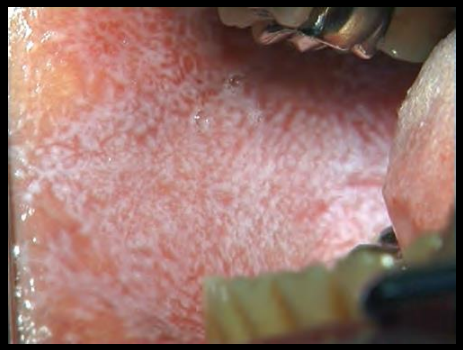




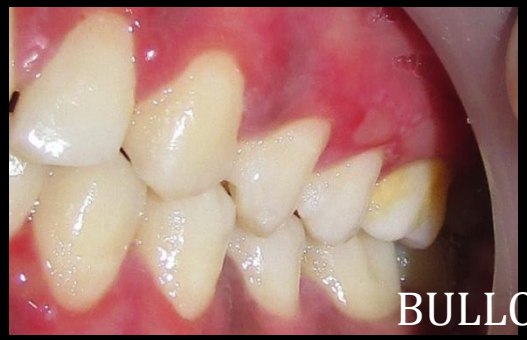
6 CLINICAL SUB TYPES OF OLP



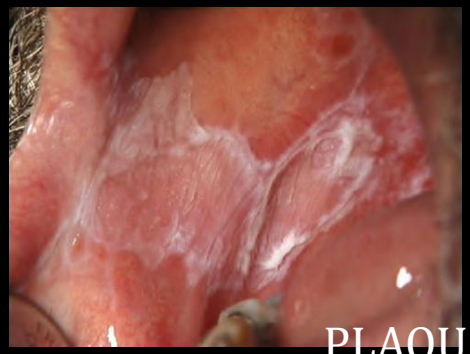
RETICULAR



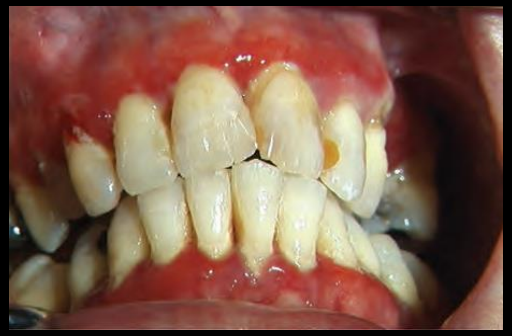
PAPULAR



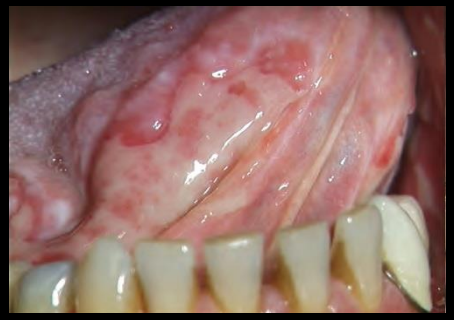
BULLOUS



PLAQUE- like



ERYTHEMATOUS



ULCERATIVE

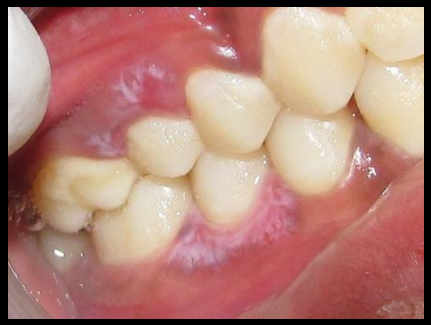
Andreasen (1968)

- Reticular
- Papular
- Plaque – like
- Bullous
- Erosive / Ulcerative
- Atrophic/ Erythematous

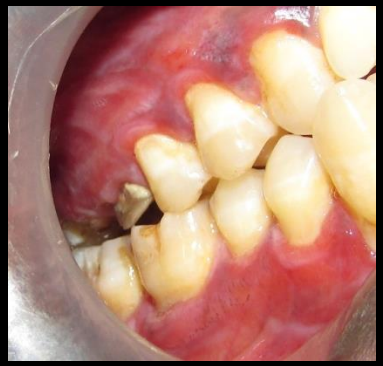
Eisen 1993

GINGIVAL LESIONS

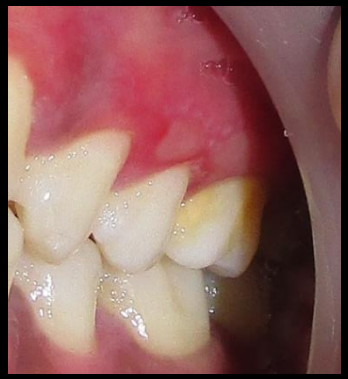
- Upto 10% of patients with oral lichen planus have lesions restricted to gingival tissue
- 4 distinctive patterns noted:
  - Keratotic lesions
  - Erosive or ulcerative lesions
  - Vesicular or bullous lesions
  - **Erythematous/Atrophic lesions**



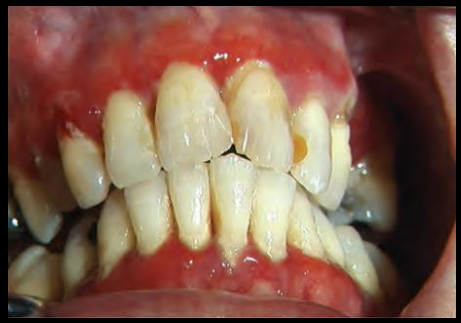
KERATOTIC



EROSIVE



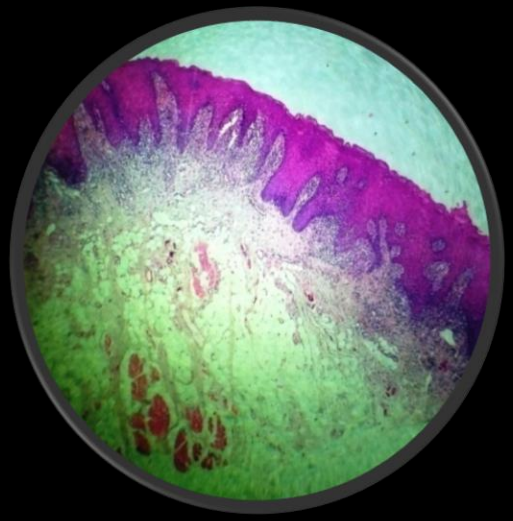
BULLOUS



ATROPHIC

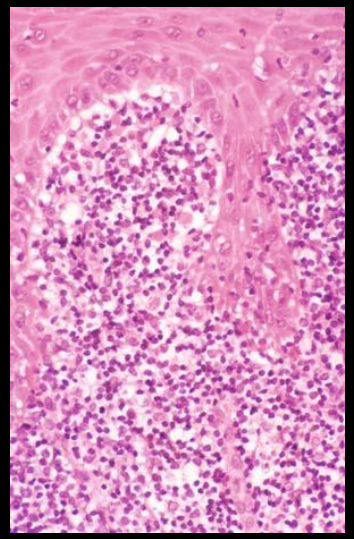
- Gingiva : erythematous, occasional areas of erosion, white striae at periphery
- Pt C/O persistent soreness
- Worsened by spicy food/ daily oral hygiene procedure
- c/f confused with PI gingivitis
- DG due to LP does not result in clinical attachment loss & periodontitis
- Diagnosis : difficult if gingiva is the only site of involvement

HISTOLOGICAL FEATURES



- Hyperparakeratosis or Hyperorthokeratosis.
- Acanthosis with intercellular edema
- Thickening of granular cell layer.
- Saw tooth appearance of rete peg.
- gingival lesion – thinning / flattening of epithelium
- Liquefaction degeneration/ necrosis of basal layer, often replaced by an eosinophilic band & dense subepithelial band of lymphocytes.
- Characteristic presence of **Civette Bodies**

(Krammer & Griffin)



Electron microscopic studies shows

1. Irregularity of the nuclear membrane
2. Increased thickening and granularity of tonofibrils

Treatment

Keratotic, asymptomatic lesions – no R_x. Regular follow-up

eryhtematous/bullous/ ulcerative lesions - topical steroids such as *0.1%* triamcenalone acetonide, 0.05% fluocinonide ointment, 0.05% clobetasol propionate

Alternative to Corticosteroids -
- Cyclosporine
- Levamisole
- Topical human interferon – β
- Systemic isotretinoin
- Dapsone
- PUVA

Non-Drug Therapy
- Surgical excision
- Cryosurgery
- CO_2 laser
- Ultraviolet radiation

In more severe cases,

Intralesional injections of triamcinolone acetonide (10mg/ml or 40mg/ml)

- Topical Pimecrolimus- 1%
- Topical tacrolimus- 0.1%

Systemic:

40 mg of prednisone OD for 5 days, followed by 10-20 mg OD for an additional 2 weeks

Elimination of mechanical trauma or irritants such as sharp filling margins

Superimposed fungal infection-
Clotrimazole 1%

Dental Clinics of North America 2013;57(4)
J Periodontol 2008;79:4-24.

Gingiva

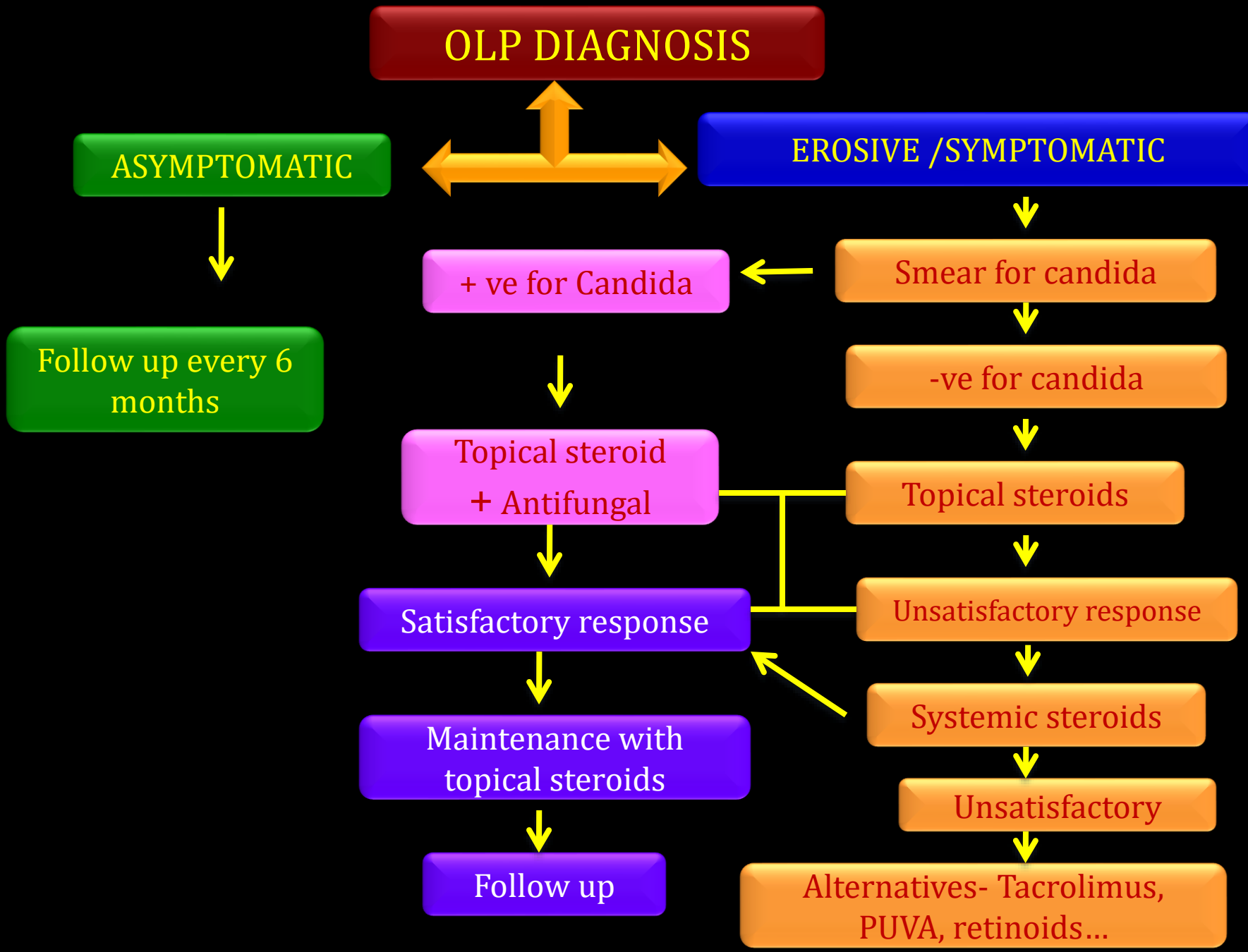
- atraumatic oral hygiene
- Topical corticosteroids – main stay of trt
- Vacuum formed custom trays / gingival veneers
- Applied to the fitting surface – worn overnight

POTENTIAL NEW TREATMENT:

- Tacrolimus capsule 1mg dissolved in 1000 ml sterile water
  - Rinse with 1 tsp 4 times daily
  - One report describes 20% complete remission, 70% improvement

Valeric et al *Arch Dermatol 2002*

Gould A. Am Acad Oral Max-Fac Path 2012 (Oral presentation)


OLP DIAGNOSIS
ASYMPTOMATIC
EROSIVE /SYMPTOMATIC
Follow up every 6 months
+ ve for Candida
Smear for candida
-ve for candida
Topical steroid + Antifungal
Topical steroids
Satisfactory response
Unsatisfactory response
Systemic steroids
Maintenance with topical steroids
Unsatisfactory
Follow up
Alternatives- Tacrolimus, PUVA, retinoids…

PEMPHIGUS

Pemphigus

- *Greek pemphix, meaning bubble or blister*
- Group of auto immune disorders characterized by autoantibodies targeting the desmosomes.

Types

- Pemphigus Vulgaris
- Pemphigus Vegetans
- Pemphigus Foliaceous
- Para Neoplastic Pemphigus

Pemphigus Vulgaris

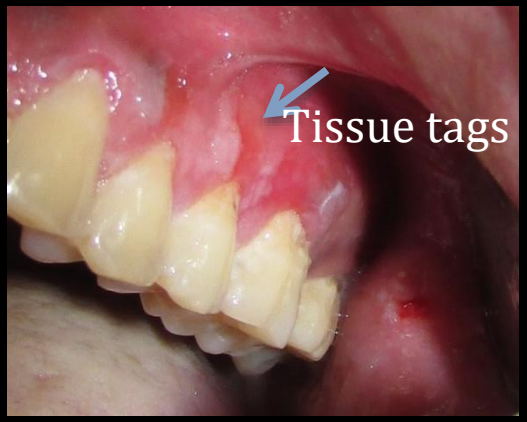
- 3rd -5th decade.
- M=F.

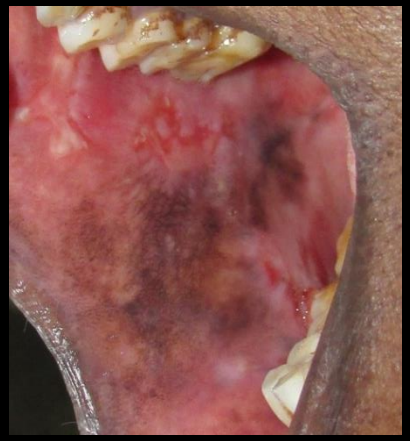
80-90% PV pts develop oral lesion

60% cases,
oral lesions – first sign

Early lesions- classic bulla – noninflamed base

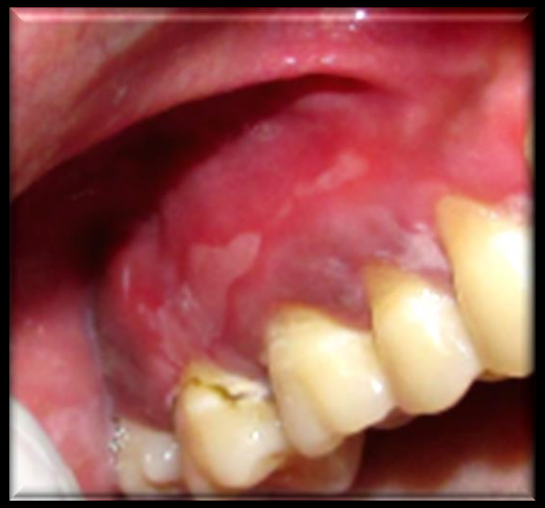
rupture easily -ragged erosions which ulcerates.





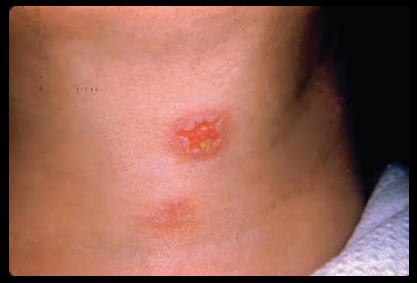
Epithelium peels in irregular pattern leaving a denuded base

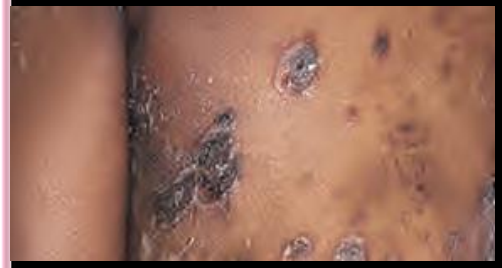
- Common site : buccal mucosa often on areas of trauma
  - Palate
  - Gingiva : Desquamation
- Pain,
- difficulty in eating,
- ↑ tendency for bleeding, bad breadth.
- New lesions may develop following trauma.



Asboe-Hansen sign

Nikolsky's sign.

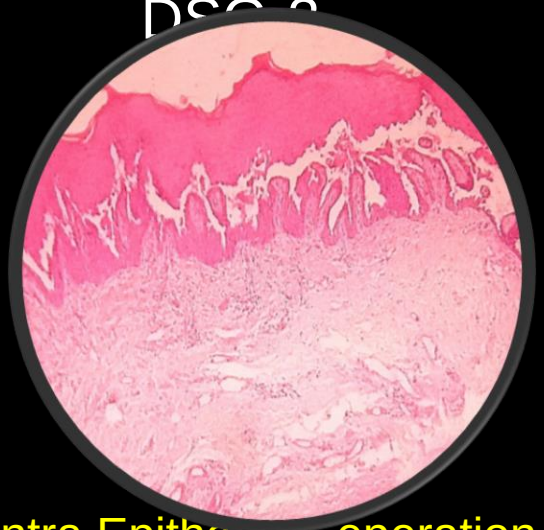




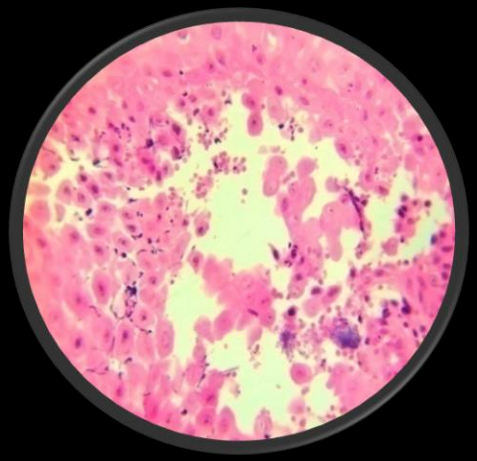
Skin lesions appear initially on head and neck and later spreads to involve trunk and extremities.

If not treated promptly it may be fatal due to loss of fluids and secondary infection.

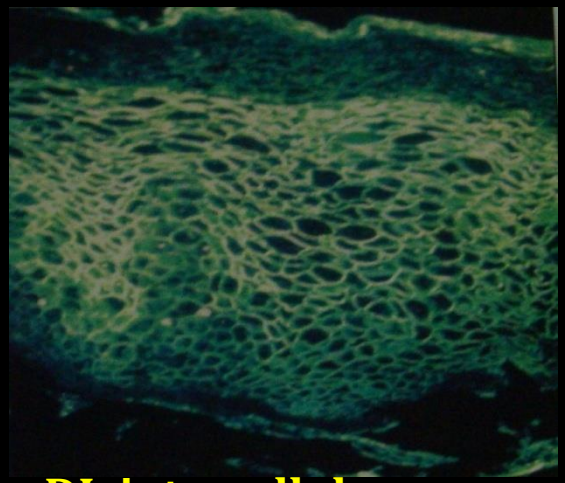
- Diagnosed by
- Cytology- Tzank smear- Acantholytic cells
- Antibody Titre evaluation to monitor the progress of treatment.
- ELISA-To distinguish bet. anti DSG-1 antibodies and anti DSG-3



Intra Epithelial Seperation with basal cells attached to the basement membrane



Acantholytic cells in tzank smear



DI :intercellular deposits of IgG - fish net pattern

Treatment

- PV death rate: 65-90%
- With syst corticosteroids reduced by 10%
- Current mortality reduced by 6.2%

Systemic Corticosteroids	**T. Prednisolone**	**0.5-1mg/kg/d.Dose tapered by 25%biweekly**
Topical Corticosteroids	Clobetasol-0.05%, Fluocinonide- 0.05%, Triamcinolone- 0.1%,	Applied thin bid
Calcineurin inhibitor	Topical Pimecrolimus- 1% Topical tacrolimus- 0.1%	
Immuno Suppressives	T.Methotrexate T.Mycophenalate mofetil T.Dapsone	10-20 mg/week 2g/d 100mg/d
Monoclonal anti-CD20 antibody (recalcitrant cases)	T.Rituximab	1000mg IV/2weeks
Intravenous Immunoglobulins		2 g/kg/cycle. IV administered over 2–5 consecutive days, monthly.
Plasmapheresis	plasma exchange-selective removal of Ig's	
Immunoadsorbtion	Rapid removal of circulating autoantibodies	

Dental Clinics of North America 2013; 57(4)
J Periodontol 2008;79:4-24.

- Application of topical steroids : safe and effective
- initial prednisolone dose should not exceed 0.75 mg/kg/d

- Monotherapy of pemphigus with oral corticosteroids causes frequent side effects, including systemic infections
  - (25% are lethal),
  - diabetes mellitus(45%),
  - osteoporosis (30%),
  - thromboses (15%), and
  - gastrointestinal ulcers (15%)
- Thus, in the treatment of pemphigus, systemic corticosteroids are given in combination with other immunosuppressive drugs such as azathioprine, mycophenolate mofetil, mycophenolate sodium, cyclophosphamide and methotrexate

Dexamethasone Cyclophosphamide Pulse Therapy:

Defn: A short, intensive course of pharmacotherapy, given at intervals such as weekly or monthly

- Phase I: Dexamethasone 100 mg in 5% dextrose slow IV infusion over 2 hours for three consecutive days along with Cyclophophamide 500 mg infusion on one of the days. Such pulses are repeated every 28 days till no new lesions are seen between pulses. Cyclophosphamide 50 mg/day given orally.
- Phase II: Patients were in remission but monthly DCP therapy and daily oral cyclophosphamide were continued for 9 months.

- Phase III: Only oral cyclophosphamide 50 mg/day given for 1 year
- Phase IV: All the drugs are withdrawn and the patient followed as long as possible.

Kaur S, Kanwar AJ. Dexamethasone cyclophosphamide pulse therapy in pemphigus. Int. J Dermatol 1990; 29: 371-74.

PEMPHIGOID

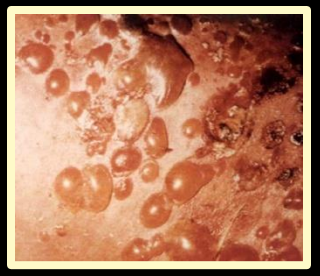
PEMPHIGOID

- The term ‘ PEMPHIGOID’ applies to a number of cutaneous, immune mediated, subepithelial bullous disease.
- Includes: MUCOUS MEMBRANE PEMPHIGOID
  BULLOUS PEMPHIGOID

Mucous membrane pemphigoid (MMP)

- Heterogeneous group of chronic, autoimmune subepithelial blistering diseases that predominantly involves the mucous membranes and occasionally the skin.

MMP -heterogeneous -several different antigens implicated



PATHOGENESIS

Ag-Ab complexs IgG BM zone

↓

directed against hemidesmosomes & basement membrane zone (BP 180, BP230,laminin 5)

↓

Complement activation

↓

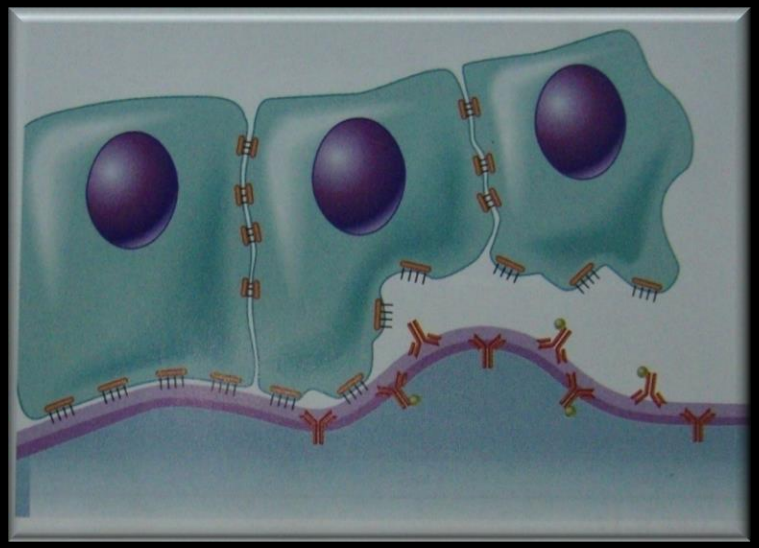
Leukocyte recruitment

↓

Proteolytic enzymes released

↓

Dissolves / cleaves BM



MUCOUS MEMBRANE PEMPHIGOID

Also called CICATRICIAL PEMPHIGOID

Chronic vesiculobullous autoimmune disorder.

Unknown cause.

Predominantly affects Women.

5^{th} decade of life.

Involves oral cavity, conjunctiva, & mucosa of nose, vagina, rectum, esophagus and urethra.

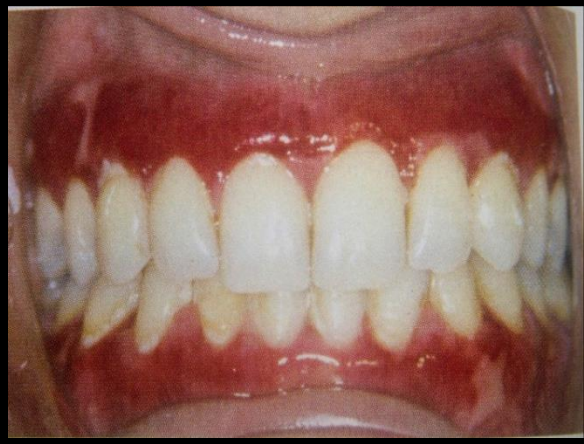
Skin involvement– 20% of patient

ORAL LESIONS

Oral lesion : 90% with MMP

DESQUAMATIVE GINGIVITIS

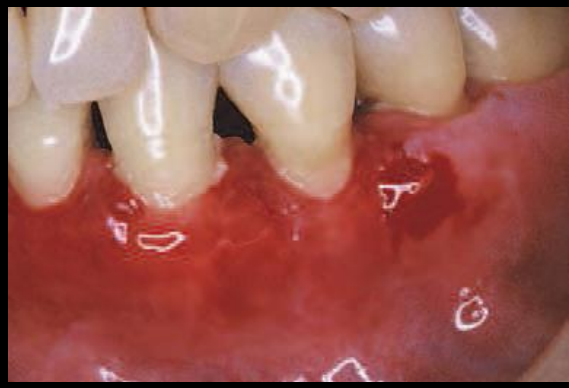
Erythema, **desquamation,** ulceration & vesiculation of attached gingiva.



Bulla has thick roof.

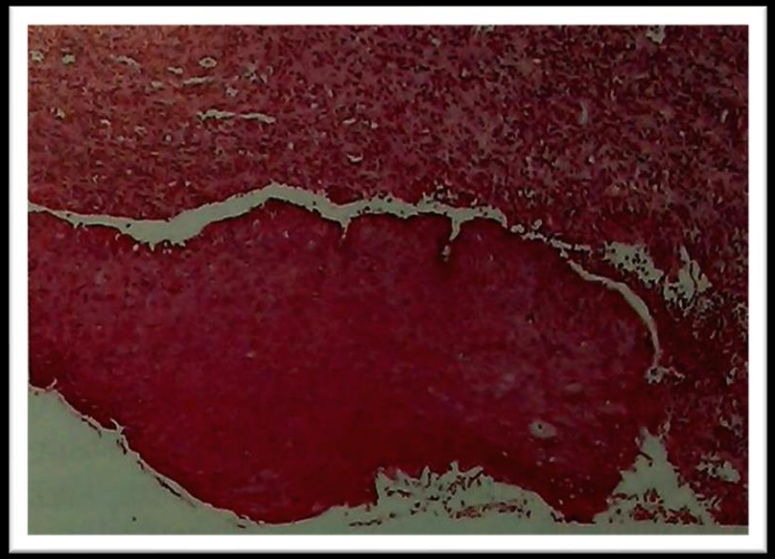
Ruptures in 2-3 days → irregular shaped ulcers.

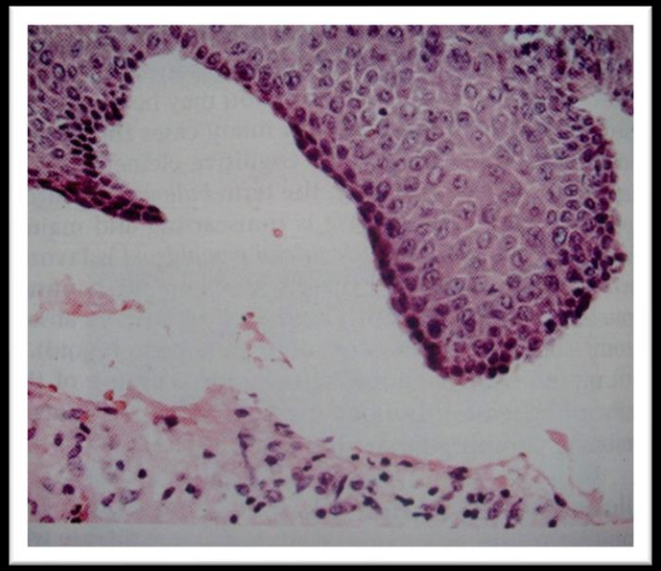
Heals in 3 weeks/ longer duration.



HISTOPATHOLOGY

- Subepithelial vesiculation.
- Epithelium separated from underlying lamina propria.
- Separation occurs at BM.
- Mixed inflammatory infilterate in CT.



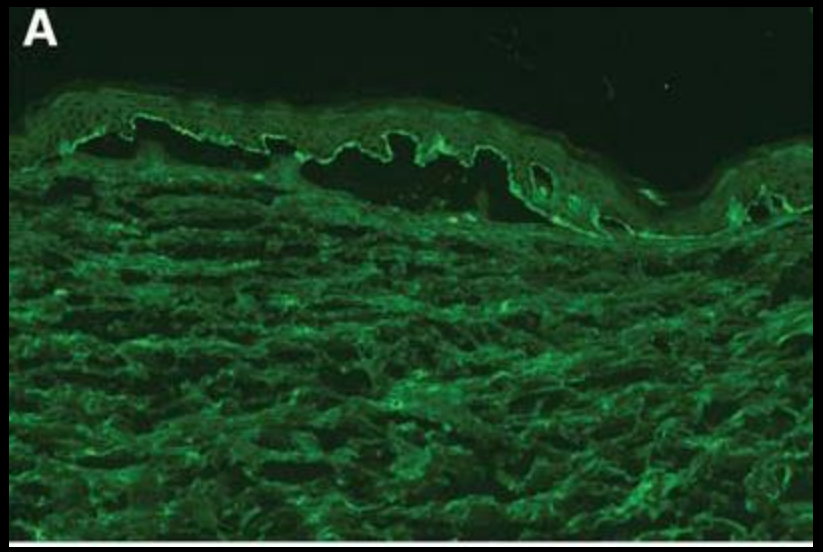


Mucous membrane pemphigoid

Direct immunfluorescence	Indirect immunofluorescence	Autoantigens
Linear IgG, and C3 at the dermal–epidermal junction	Epidermal or dermal IgG – basement membrane zone	BP180, Laminin 5

DI

IDI



Mild cases

1)Topical steroids-Fluocinonide (0.05%)

Clobetasol propionate (0.05%) -3 times a day- 6 months.

2) Tetracycline 1500-200mg/day or minocycline- 50 to 100mg/day.

Can be combined with nicotinamide 500-2500mg/kg/d

3) Dapsone- initial dose 50mg, increase by 25 mg every 7 days upto

100 to 200 mg/day

4) Systemic steroid- Prednisolone 0.5-1mg/kg/day

Severe disease

1) Systemic steroid : Prednidolone- 1-2mg/kg/day or Dexamethasone 100mg/day for 3 days(Pulse therapy) or i.v pulse therapy 0.5 to 1gm for 3days.

2) Mycophenolate mofetil 35-45mg/kg/d

3)Azathioprine- Initially 1-2mg/kg/day, can be raised upto 5mg/kg/d

4)i.v immunoglobulin 2mg/cycle every 4 weeks

5) others: Methotextrate, cyclosporin,

Gingival lesion

In lesion confined to gingiva – topical corticosteroid in vacuum formed custom trays / veneers.

Bullous Pemphigoid

- Sub epithelial blistering disease
- Self limited

PATHOGENESIS

- Autoantibodies of IgG type (and less commonly
- IgA, IgM and IgE) attack components of the adhesion complex of the basement membrane zone (BMZ) and result in subepidermal blistering.
- The two main autoantigens are BP230 (BPAg1) and BP180 (BPAg2, collagen XVII)

Occurs chiefly in adults over 60 years of age.

Skin lesion : blister on an inflammed base. involves scalp arms legs axilla & groin

Tense subepithelial bullae - affects whole thickness of epithelium- persists longer.

Oral lesions smaller,less painful.

Desquamation of gingiva – COMMON FINDING

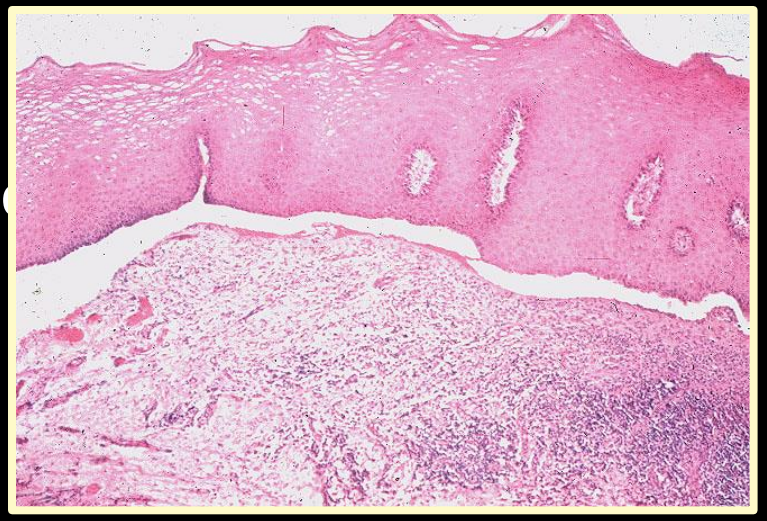
Can sometimes be the only oral finding

The gingival lesions consist of generalized edema, inflammation, and desquamation with localized areas of discrete vesicle formation.

Bulla do not extend peripherally to form large denuded areas like pemphigus.

HISTOLOGICAL FEATURE

- Bullae are subepidermal.
- No acantholysis.
- Vesicle contain fibrinous exudate admix with occasional inflammatory cell.

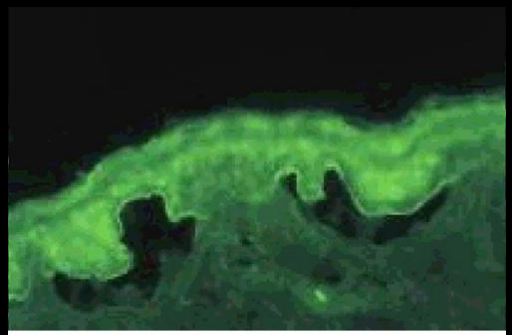


ELECTRON MICROSCOPY:

- Split beneath basement menbrane
- Blood vessels show alteration in their permeability.
- BM show thickening.

IMMUNOFLUORESENCE

- DIRECT:
- Tissue bound anti- basement membrane zone antibodies IgG class.
- INDIRECT:
- Positive in 80% of the patient.



Bullous pemphigoid

Figure 19-09 B. Bullous pemphigoid. Indirect immunofluorescence study performed on salt-split normal human skin substrate with the serum from a patient with bullous pemphigoid detects immunoglobulin G (IgG) class circulating autoantibodies that bind to the epidermal (roof) side of the skin basement membrane.

British Association of Dermatologists' guidelines for the management of bullous pemphigoid 2012

- Topical corticosteroids
- use of topical steroids as first-line treatment for both localized and moderate disease

British Association of Dermatologists' guidelines for the management of bullous pemphigoid 2012

- Systemic steroids are the best established treatment for BP : prednisolone of 0.75-1mg/kg daily in widespread BP are effective within 1–4 weeks in about 60–90% of cases.

- It is not possible to identify a starting dose of prednisolone (or prednisone) that would be maximally effective and minimally toxic for all patients with BP.
- Doses which might meet these criteria for a majority of patients are:
- 0.75-1mg/kg -severe cases
- 0.5 mg/kg : for moderate cases
- 0.3 mg/kg for mild or localized disease.

- If new inflammatory or blistered lesions are few or absent within 4 weeks - gradually reduced
- Reduction of the daily dose of prednisolone -about 1/3rd to 1/4 down to 15 mg daily, then down to 10 mg daily, is suggested.

- The dose could then be reduced by 1 mg each month

- In about 50% of cases relapse will occur at some point during the dose-reduction period, indicating that the previous dose is likely to be the minimal effective dose for that patient

ERYTHEMA MULTIFORME

Acute self limiting inflammatory mucocutaneous disease.

Erythema multiforme minor <10% skin

Erythema multiforme major (extensive skin +oral)

Fulminant forms.

Stevens-Johnson syndrome (SJS)

Toxic epidermolysis necrosis syndrome (TENS)

Erythema multiforme usually occurs in adults 20 to 40 years of age

Hypersensitivity to infectious agents such as **HSV,** drug reaction – NSAIDs /anticonvulsants

EM with HSV : 65-70%

HSV Ag inciteT-cell mediated delayed hypersensitivity reaction.

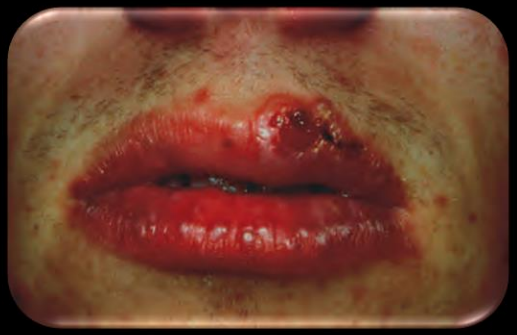
Cytotoxic T cells,natural killer cells and cytokines destroy epithelial cells.

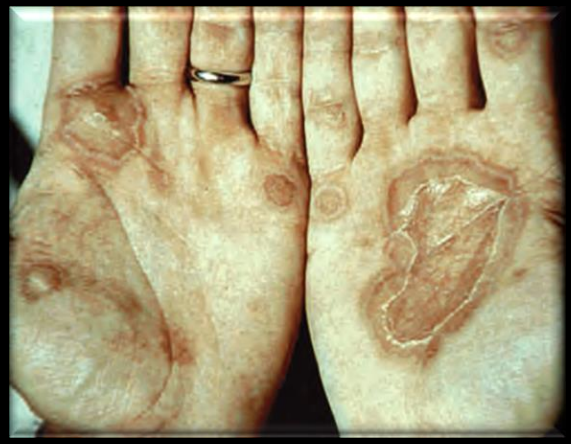
Erythema Multiforme

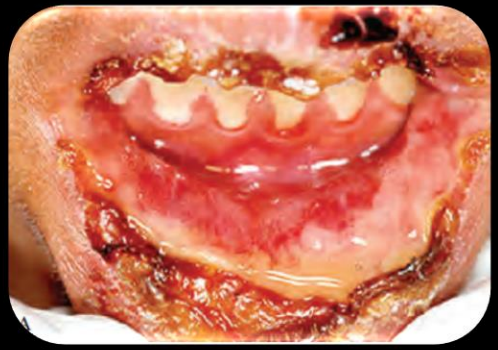
- Skin lesion – multiforme
- Target or iris lesion

Oral involvement 70%

- vesicles or bullae -rupture - thick white or yellow exudates.
- The lips may exhibit ulceration with bloody crusting
- Severe form : large ulcer- coalesce
  - Difficulty in eating,swallowing
  - Drool blood tinged saliva







- Erythema multiforme resolves spontaneously in three to five weeks without sequelae, but it may recur
- Erythema multiforme is diagnosed clinically.
- In patients who have target lesions with a preceding or coexisting HSV infection, the diagnosis can be made easily

KEY RECOMMENDATIONS FOR PRACTICE

- Oral acyclovir (Zovirax) should be given early in herpes-associated outbreaks of erythema multiforme to lessen the number and duration of lesions

MANAGEMENT	Drug	Dosage
HSV associated EM	T.Acyclovir T.Valacycovir T. Famcyclovir	400mg/bd 500 mg/bd 250mg/bd
Mild EM	Systemic, Topical Analgesics, supportive care	
Severe EM Top. Corticosteroids	Clobetasol, Fluocinonide, Triamcinolone, Fluocinolone, Hydrocortisone acetate	0.05% 0.05% 0.1% 0.05%
Systemic Corticosteroids	T. Prednisolone	1-2mg/kg/day
Mouth wash	•Equal parts of viscous lidocaine 2%, diphenhydramine-12.5mg/5ml •Maalox- Aluminium hydroxide and magnesium hyroxide mixture	Swish and spit 4 times/day
Recurrent EM	T. Dapsone Azathioprine	100-150mg/day 100-150 mg/day

LUPUS ERYTHEMATOSIS

- Heterogeneous autoimmune disorder
- Involves the oral cavity along with the skin and internal organs
- Wide spectrum of symptoms

PATHOGENESIS

Lupus is strongly associated with defects in apoptotic clearance.

Endogenous nuclear antigens are characteristic of SLE

Autoantigens released by apoptotic cells are presented by dendritic cells to T cells

Failure of phagocytes to remove apoptotic material efficiently

Fragments of nuclear particles -captured by antigen presentingcells

Interact with T and B cells

Development antinuclear antibodies that are typical of the disease.

Murphy, Grainne, Larissa Lisnevskaia, and David Isenberg. "Systemic lupus erythematosus and other autoimmune rheumatic diseases: challenges to treatment." The Lancet 382.9894 (2013): 809-818.

Oral lesion : 40% of pts

Prevalence in Indian population – 3.2%

F > M F:M =10:1

30 to 40 yrs

Type III hypersensitivity- immune complex IgG and IgM

Types:

Cutaneous LE,
Discoid LE,
Systemic LE

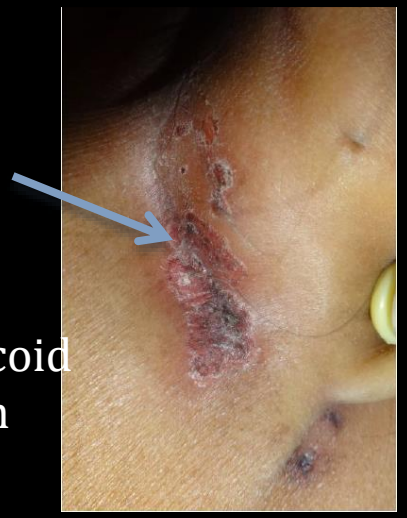
- *Kuhn, Annegret, et al. "The diagnosis and treatment of systemic lupus erythematosus." Deutsches Ärzteblatt International 112.25 (2015): 423.*
- *Malaviya, A. N., et al. "Prevalence of systemic lupus erythematosus in India." Lupus 2.2 (1993): 115-118.*

- Oral lesions are characterized by the presence of a central erythematous erosion or ulceration surrounded by a white rim with radiating keratotic striae "brush border"

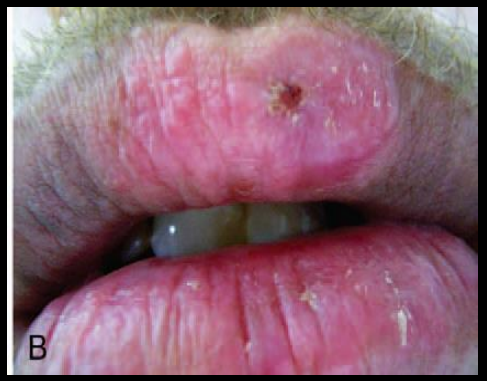
- Frequent sites : hard and soft palate, buccal mucosa, and the vermillion border of the lips.

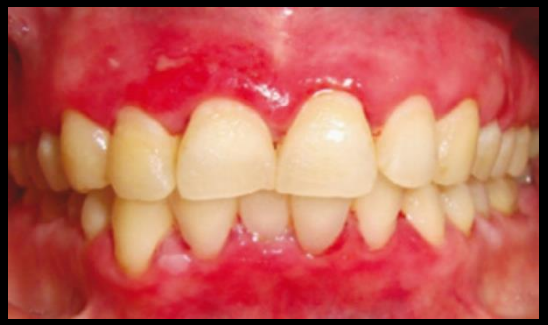
- The gingiva : desquamative appearance,
- burning or soreness.

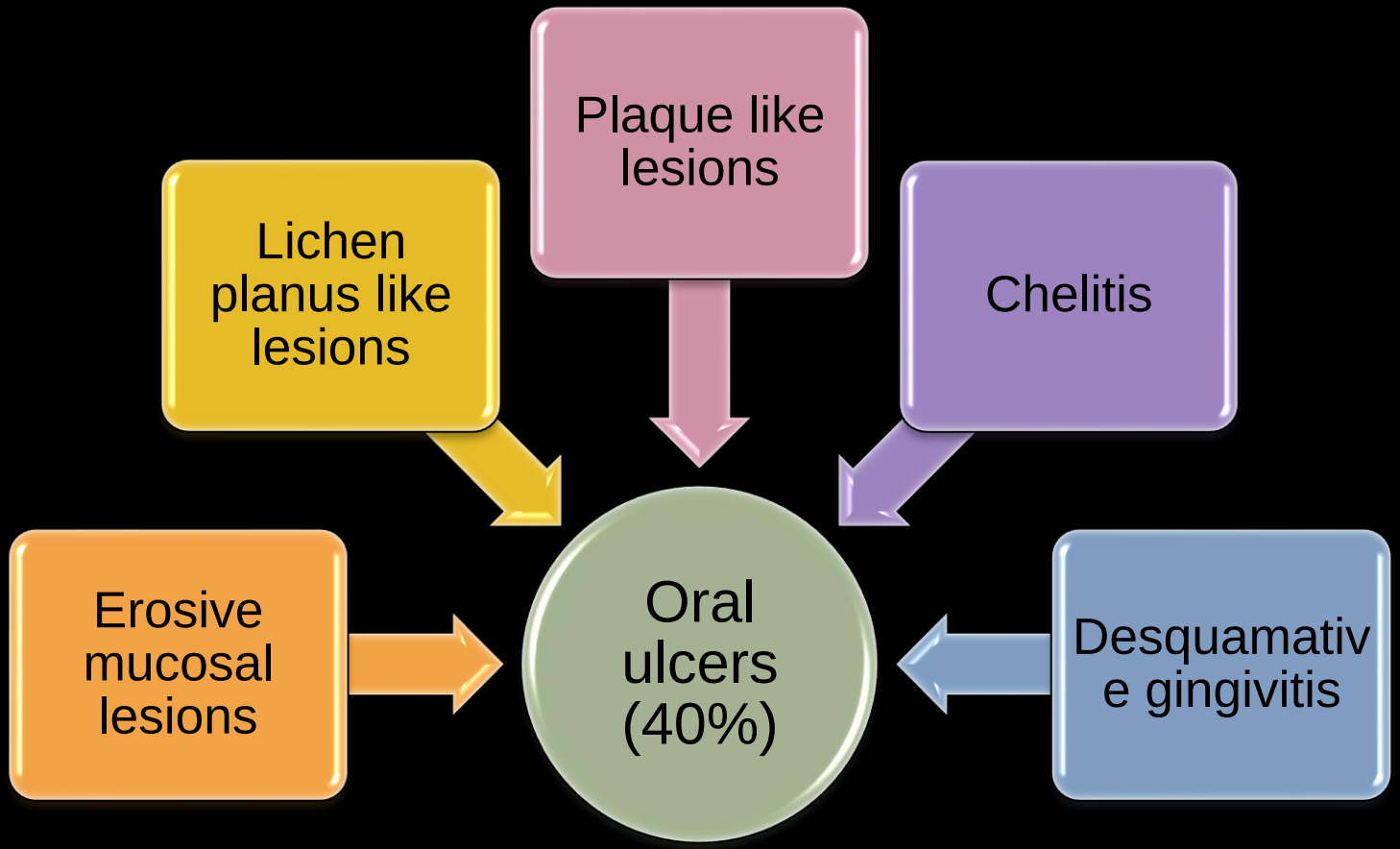
- Other mucosal surfaces :oropharyngeal mucosa,nares, larynx, and epiglottis



Discoid rash







Hard palate (89%)>Buccal mucosa> Lips> Gingiva

- *Murphy, Grainne, Larissa Lisnevskaia, and David Isenberg. "Systemic lupus erythematosus and other autoimmune rheumatic diseases: challenges to treatment." The Lancet 382.9894 (2013): 809-818.*
- *Brennan, Michael T., et al. "Oral manifestations of patients with lupus erythematosus." Dental Clinics of North America 49.1 (2005): 127-141.*

DIAGNOSIS

Box 1. American College of Rheumatology criteria for systemic lupus erythematosus*

1. Malar rash
2. Discoid lesions
3. Photosensitivity
4. Presence of oral ulcers
5. Nonerosive arthritis of two joints or more
6. Serositis
7. Renal disorder
8. Neurologic disorder (seizures or psychosis)
9. Hematologic disorder (hemolytic anemia, leukopenia, lymphopenia, or thrombocytopenia)
10. Immunologic disorder (anti-DNA, anti-Sm, or antiphospholipid antibodies)
11. Antinuclear antibody

---

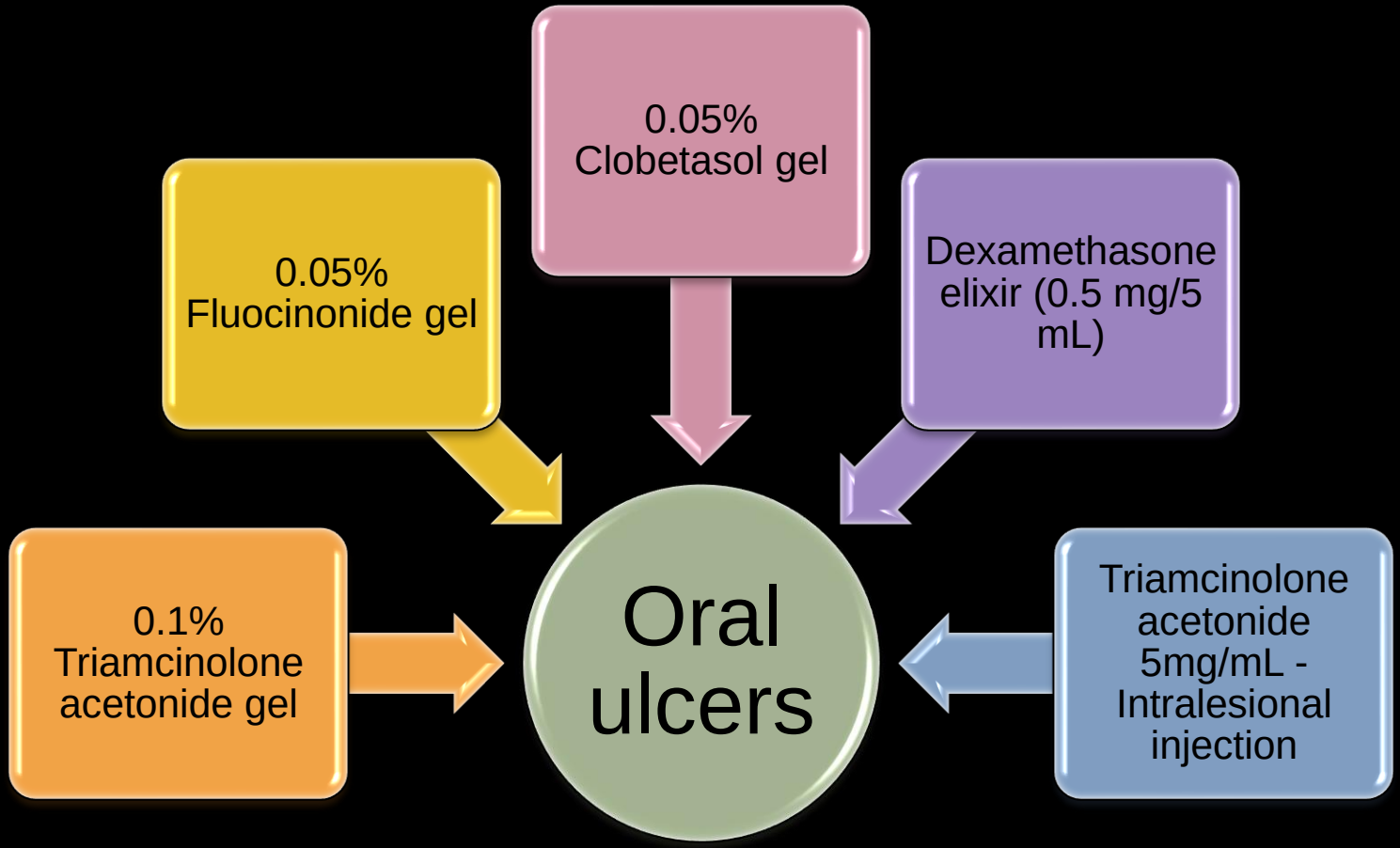
* SLE diagnosis with 4 or more of 11 criteria present at any time.

Adapted from Tan EM, Cohen AS, Fries JF, et al. The 1982 revised criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 1982;25:1271–7; with permission.

Treatment recommendations for systemic lupus erythematosus (SLE) with no, mild, and/or moderate organ manifestations (e.g., skin, joints, serositis),

First line and basic treatment	**Hydroxychloroquine or Chloroquine Initial nonsteroidaL anti-inflammatory Drugs and/or glucocorticoids**
If no response	Azathioprine or methotrexate or mycophenolate mofetil

- *Kuhn, Annegret, et al. "The diagnosis and treatment of systemic lupus erythematosus." Deutsches Ärzteblatt International 112.25 (2015): 423.*
- *Weening, Jan J., et al. "The classification of glomerulonephritis in systemic lupus erythematosus revisited." Kidney international 65.2 (2004): 521-530*



Brennan, Michael T., et al. "Oral manifestations of patients with lupus erythematosus." Dental Clinics of North America 49.1 (2005): 127-141.

CHRONIC ULCERATIVE STOMATITIS

- Rare mucocutaneous disease
- Primarily involves mucosal surfaces.

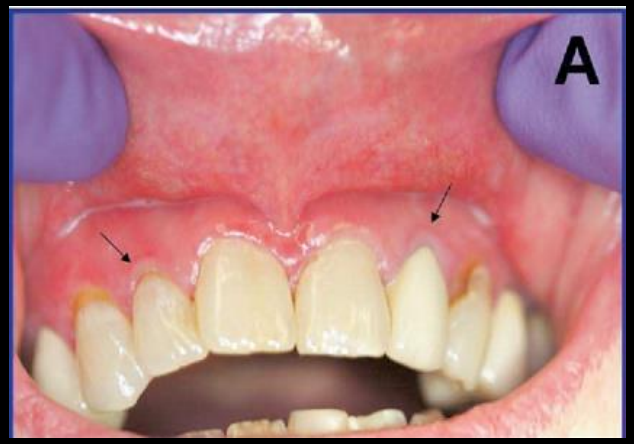
First reported in 1990. (Jaremko et al)

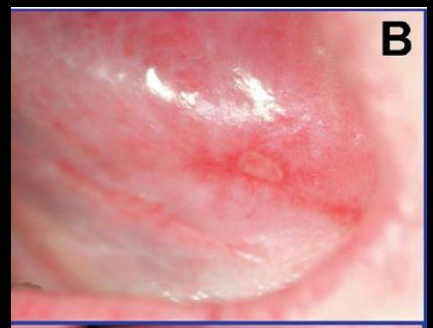
Predilection for women.

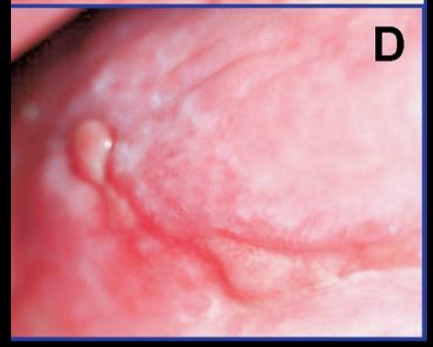
Lesions predominantly seen in oral cavity.

ORAL LESION: - Painful solitary, small blister & erosion with surrounding erythema.

- Tongue
-Buccal mucosa
-Gingiva - desquamation







- TREATMENT

  Mild case: Topical steroid

  Topical tetracycline

  Severe: High doses of systemic corticosteroid

Treatment of choice for complete long lasting remission:
Hydroxychloroquine 200 – 400 mg/ day

Dental Clinics of North America 2013; 57(4)
J Periodontol 2008;79:4-24.

Linear IgA Disease

Sub Epithelial disease with deposition of IgA in the basement membrane.

Affects children below the age of 10.

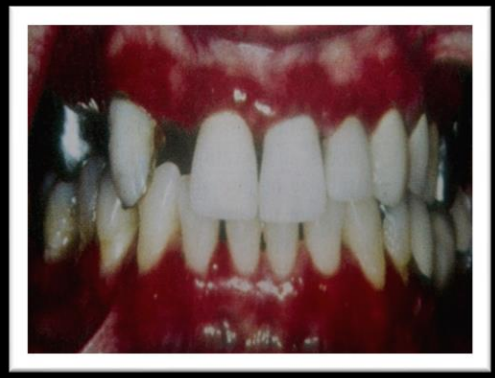
Etiolgy - Unknown.

1)Might be drug induced (ACE inhibitors)

2)systemic disease (hematological malignancies, dermatomyositis

Skin Lesions- Annular pruritic papules and blisters “cluster of jewels” appearance.

Oral lesions are common- Blisters,erosions and ulcers of oral mucosa with **Desquamative Gingivitis.**



H/P- Sub Epithelial Clefting.

DIF-Linear deposition of IgA.

TREATMENT

1)Combination of sulfone & dapsone.

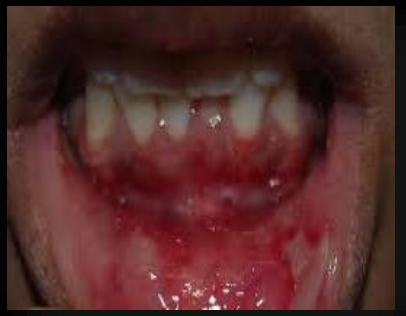
2) Prednisone 10-30mg – if initial response inadequate.

3)Tetracycline (2g / day) + Nicotinamide (1.5g / day)

Epidermolysis bullosa

Blistering of skin & mucous membrane.

Acquired subepithelial fragile blisters affecting skin and mucosa.



Painful ulcerations preceded by collapse of vesicles/bullae.

Resembles bullous pemphigoid.

Ig GAntibodies against Type VII Collagen

DIF-Linear IgG and C3 basement membrane zone

Treatment:

High dose of Corticosteroids.

Immunosuppressants

DRUG ERUPTIONS

Eruptive skin and oral lesions are attributed to drugs acting as an allergen, either alone or in combination → sensitizing the tissues and causing allergic reaction.

Eruption in oral cavity from sensitivity to drugs taken by mouth/ parenterally →

STOMATITIS MEDICAMENTOSA

Local reaction →

STOMATITIS VENENATA / CONTACT STOMATITIS.

Due to: Irritating local action

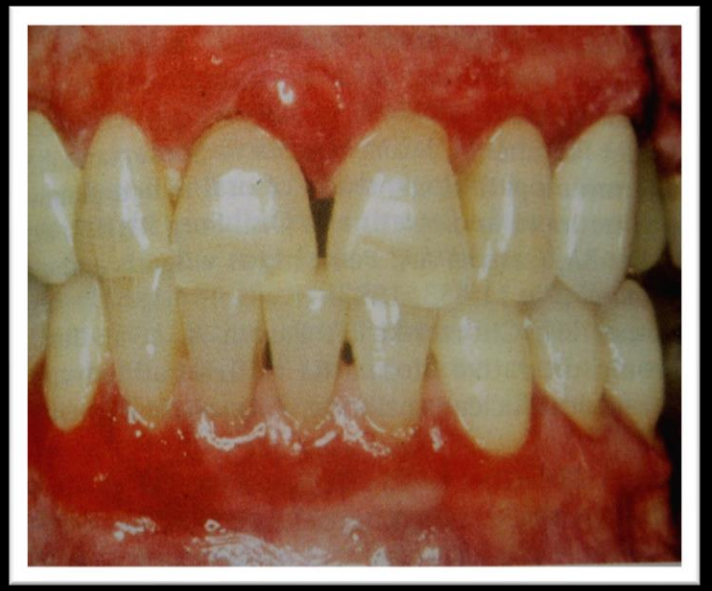
Drug sensitivity

Lesion Morphology	Drugs
Lichenoid lesions	Antimalarials (chloroquine, hydroxychloroquine, quinacrine, quinidine), antihypertensives (β-blockers), NSAIDs, methyldopa, penicillamine, lithium, lorazepam, isoniazid, ketoconazole, amphotericin B, carbamazepine, streptomycin, tetracycline, cimetidine, flunarizine*
Pemphigoid-like	Antirheumatics (ibuprofen, penicillamine, and phenacetin), captopril, carbamazepine, furosemide, clonidine, practolol, antibiotics (penicillins and sulfonamides)
Pemphigus-like	Penicillamine, captopril, thiol drugs (containing sulfhydryl radical: -SH), diclofenac, ibuprofen, piroxicam, propranolol, ACE inhibitors, theobromine, ampicillin, rifampin, interferon
EM	NSAIDs, allopurinol, barbiturates, penicillin, cephalosporins, sulfonamides, phenytoin, quinolones, carbamazepine, furosemide
Lupus-like	Hydantoins, carbamazepine, lithium, chlorpromazine, ethosuximide, reserpine, griseofulvin, methyldopa, isoniazid, procainamide, quinidine, primidone, streptomycin, thiouracils, trimethadione

DRUGS ASSOCIATED WITH DESQUAMATIVE GINGIVITIS

- Appear as either:
  - Vesicle or bullae
  - Pigmented / nonpigmented macule
  - Erosions
  - Ulceration with purpuric lesion

- Gingival lesions can be seen due to:
  - Mercurial compounds
  - Pyrophosphates & flavoring agents
  - Cinnamon compounds

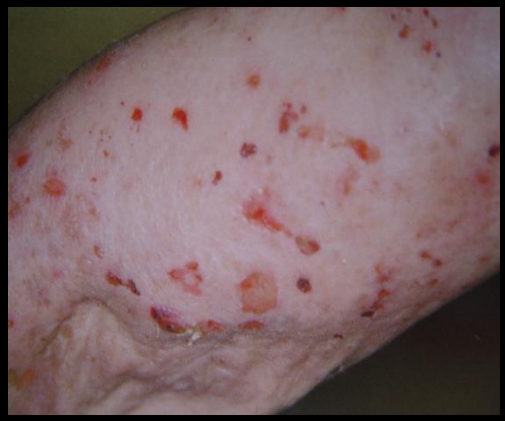


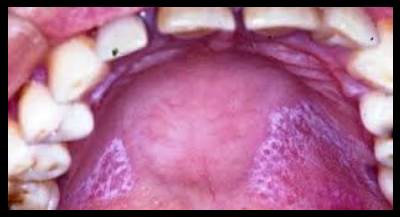
TREATMENT

- Elimination of offending agent
- Oral antihistamines
- anti-inflammatory mouth rinse of benzydamine hydrochloride
- severity of the lesion - steroids

DERMATITIS HERPETIFORMIS

- Chronic condition
- Young adults (20-30 years)
- Slight predilection for males.
- Bilateral and symmetric pruritic papules/vesicles





EXTRAORAL: skin , GI tract , gluten sensitivity

EXTRAORAL: extremely pruritic erythematous , urticarial plaques papules with vesicles .

CLINICAL COURSE : chronic

INTRAORAL: range from painful ulcerations, preceded by the collapse of vesicles or bullae to erythematous lesions.

DESQUAMATIVE GINGIVITIS

Treatment:

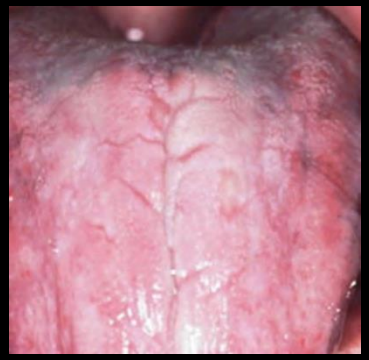
1)Dapsone

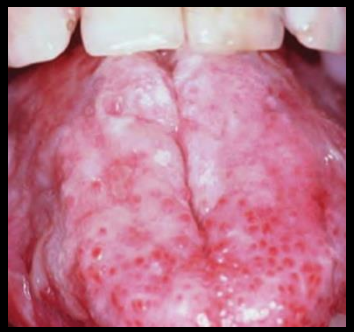
2)Gluten free diet

GRAFT vs HOST DISEASE

Etiology- HSCT

Oral manifestation: Any oral site, Lesions similar to OLP. Manifest as lichenoid Reaction





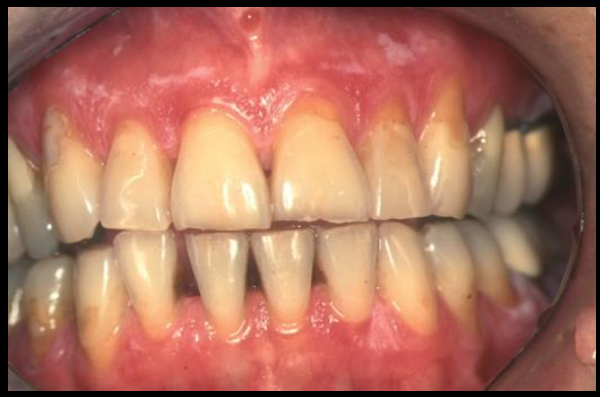
Skin

mild maculopapular rash

Severe-skin sloughing

Clinical course:

- Acute: within 100 days after transplantation
- Chronic: >100 days. Relapsing, remitting course.



Treatment:

- Same as olp
- Topical steroids
- Oppurtunistic infections mgt

SPECIAL CONSIDERATIONS IN PERIODONTAL CARE

- Non traumatic periodontal supportive care.
- Plaque control
- Recognition of early signs of disease onset.
- Surveillance for relapses
- Avoid triggering factors

BIOPSY SITE SELECTION

Choose an area of intact epithelium

Include perilesional tissue- H&E

Select normal appearing tissue for immunofluorescence testing.

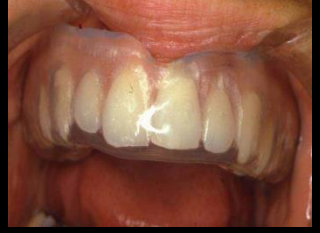
If lesion is also present elsewhere in the mucosa, avoid gingival biopsy and prefer mucosal biopsy.

TRANSPORT

•**Formaldehyde** for histopathologic evaluation

•Ambient temperature transport media **(Michelle's solution)** for DIF

Steroid Carrier Trays/ customised carrier trays



Soft maxillary and mandibular trays usually made of silicone.

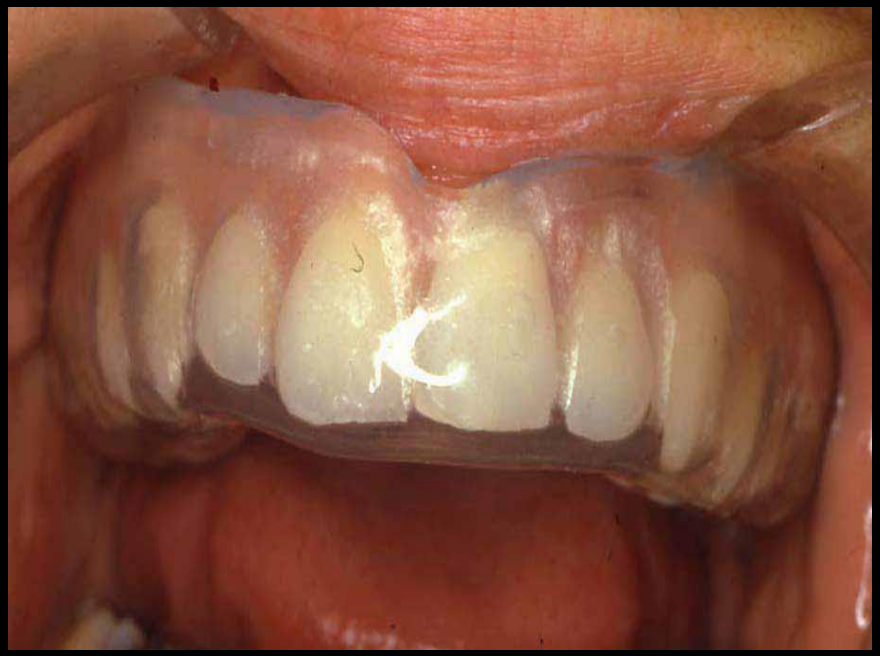
Customised to the patients dental arch.

For **High potency steroids** and **immunomodulators** when used for topical application

INSTRUCTIONS:
Coat all the inner surface of tray with topical medicament
Insert the tray 3 times a day(After breakfast, lunch and dinner).
Tray to be worn 20 minutes each time
Ask the patient to expectorate excess saliva after application and not to swallow atleast for 1 hour..
Continue the procedure for 2 weeks.
If lesion subsides, taper the dose to alternative days for the next week and then discontinoue

Aufdemorte TB et al- 1985

Steroid Carrier Trays/ customised carrier trays



ADVANTAGES:

- Increased period of contact of drug with the lesion

DISADVANTAGES:

- Insertion and removal may initiate gingival desquamation
- Risk of increased systemic uptake
- Risk of gingival epithelial thinning

ALGORITHM

PATIENT WITH DESQUAMATIVE GINGIVITIS

SPECIFIC MUCOCUTANEOUS VESICULOBULLOUS/EROSIVE DERMATOSES OTHER ORAL&/EYE&/SKIN LESIONS

HORMONAL/ IDIOPATHIC (NO SKIN, EYE OR MUCOSAL LESIONS)

BIOPSY WITH OR WITHOUT IMMUNOFLUORESCENCE

BIOPSY WIYH OR WITHOUT IMMUNOFLUORESCENCE

EYE &/OTHER MUCOSAL LESIONS

SUBBASAL SPLIT; LINEAR BASEMENT MEMBRANE DEPOSITS OF IgG,C3

BENIGN MUCOUS MEMBRANE PEMPHIGOID

TREAT WITH TOPICAL &/TRANSIENT SYSTEMIC STEROIDS

SKIN&/OTHER ORAL MUCOSAL LESIONS

SUBBASAL SPLIT; BAND LIKE LYMPHOCYTIC INFILTRATE; GLOBULAR BASEMENT MEMBRANE DEPOSITS & COMPLEMENT C3 AND FIBRINOGEN

EROSIVE/ BULLOUS LICHEN PLANUS

TREAT WITH TOPICAL STEROIDS

SKIN&/OTHER ORAL MUCOSAL LESIONS

SUPRAABASAL EPITHELIAL SPLIT; INTRACELLULAR IgG DEPOSITS

PEMPHIGUS VULGARIS

TREAT WITH LONG TERM SYSTEMIC STEROIDS &/ IMMUNOMODULATOR Y AGENTS

SKIN, LIPS&/OTHER ORAL MUCOSAL LESIONS

SUBBASAL AND SUPRABASAL SPLIT (NON SPECIFIC); NEGATIVE IMMUNO-FLUORESCENECE

ERYTHEMA MULTIFORME

TREAT WITH SHORT TERM SYSTEMIC STEROIDS &/

SKIN,FACE,EYES, PERINEUM LIPS&/OTHER ORAL MUCOSAL LESIONS

SUBBASAL SPLIT; LINEAR BASEMENT MEMBRANE DEPOSITS OF IgA AND FIBRIN

LINEAR IgA DISEASE

TREAT WITH LONG TERM SULFAPYRIDINE WITH OR WITHOUT SYSTEMIC STEROIDS

GINGIVAL LESIONS ONLY

SUBBASAL SPLIT; NEGATIVE IF

NON SPECIFIC CHRONIC DESQUAMATIVE GINGIVITIS

ESTROGENS &/ SYMPTOMATIC TREATMENT ONLY

Decision making in Periodontology- Walter B Hall- 3rd edition

- Detailed history
- Systemic symptoms
- Presence of similar lesions at other sites
- Medication used
- Contact with chemical material
- Family history

- Suspicious of allergy : patch test against dental materials
- Definitive diagnosis : HP, IF, + auto Ab in blood

Summary…

List common and rare disorders that encompass this term

Review the clinical, histological and serological findings commonly associated with Desquamative Gingivitis

Identify treatments suggested for disorders associated with Desquamative Gingivitis.

References

- *Burket LW, Greenberg MS, Glick M, Ship JA. Burket's oral medicine. PMPH-USA; 2008.*
- *Newman MG, Takei HH, Klokkevold PR, Carranza FA. Clinical Periodontology 10th Edition, chapter-31.*
- *Newman MG, Takei H, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology: Expert Consult: Online. Elsevier Health Sciences; 2014 Sep 18.*
- *Neville BW, Damm DD, Chi AC, Allen CM. Oral and maxillofacial pathology. Elsevier Health Sciences; 2015.*
- *Decision making in Periodontology- Walter B Hall- 3rd edition*
- *Froum, Scott, Naomi Marie Ramer, and Molly Cohen. "Etiology, Diagnosis, and Treatment of Desquamative Gingivitis." (2015).*

- *Endo H, Rees TD. Diagnosis and management of desquamative gingivitis. INTECH Open Access Publisher; 2011.*
- *Argiriadou AS, Sardella A, Demarosi F, Carrassi A. Gingival lesions in a patient with chronic oral graft-versus-host disease: a case report. Journal of clinical periodontology. 2003 Apr 1;30(4):375-8.*
- *Robinson, N. A., and D. Wray. "Desquamative gingivitis: A sign of mucocutaneous disorders-a review." Australian dental journal 48.4 (2003): 206-211.*
- *American Academy of Periodontology. "Oral features of mucocutaneous disorders (position paper)." J Periodontol. 74 (2003).*
- *Lo Russo, Lucio, et al. "Diagnostic pathways and clinical significance of desquamative gingivitis." Journal of Periodontology 79.1 (2008): 4-24.*
- *Williams, D. M. "Non-infectious diseases of the oral soft tissue: a new approach." Advances in dental research 7.2 (1993): 213-219.*

- *Kranti K, Seshan H, Juliet J. Discoid lupus erythematosus involving gingiva. Journal of Indian Society of Periodontology. 2012 Jan;16(1):126.*
- *Islam, M. N., et al. "Chronic ulcerative stomatitis: diagnostic and management challenges. Four new cases and review of literature." Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology 104.2 (2007): 194-203.*
- *Lo Russo, Lucio, et al. "Epidemiology of desquamative gingivitis: evaluation of 125 patients and review of the literature." International journal of dermatology 48.10 (2009): 1049-1052.*
- *McCarthy, Francis P., Philip L. McCarthy, and Gerald Shklar. "Chronic desquamative gingivitis: A reconsideration." Oral Surgery, Oral Medicine, Oral Pathology 13.11 (1960): 1300-1313.*
- *Rogers, R. S., Sheridan, P. J., & Nightingale, S. H. (1982). Desquamative gingivitis: clinical, histopathologic, immunopathologic, and therapeutic observations. Journal of the American Academy of Dermatology, 7(6), 729-735.*
- *Aufdemorte, Thomas B., Richard L. De Villez, and Stephen M. Parel. "Modified topical steroid therapy for the treatment of oral mucous membrane pemphigoid." Oral surgery, oral medicine, oral pathology 59.3 (1985): 256-260.*

THANK YOU!!