ULCERATIVE, VESICULAR AND BULLOUS LESIONS

1. Macules. Well-circumscribed, flat lesions that are noticeable because of their change from normal skin color. They may be red due to the presence of vascular lesions or inflammation, or pigmented due to the presence of melanin, hemosiderin, and drugs.

2. Papules. Solid lesions raised above the skin surface that are smaller than 1 cm in diameter. Papules may be seen in a wide variety of diseases including erythema multiforme, rubella, lupus erythematosus, and sarcoidosis.

3. Plaques. Solid raised lesions that are over 1 cm in diameter; they are large papules

4. Nodules. These lesions are present deep in the dermis, and the epidermis can be easily moved over them.

5. Vesicles. Elevated blisters containing clear fluid that are under 1 cm in diameter.

6. Bullae. Elevated blisterlike lesions containing clear fluid that are over 1 cm in diameter.

7. Erosions. Moist red lesions often caused by the rupture of vesicles or bullae as well as trauma.

8. Pustules. Raised lesions containing purulent material.

9. Ulcers. A defect in the epithelium; it is a well-circumscribed depressed lesion over which the epidermal layer has been lost.

10. Purpura. Reddish to purple flat lesions caused by blood from vessels leaking into the subcutaneous tissue. Classified by size as petechiae or ecchymoses, these lesions do not blanch when pressed.

11. Petechiae. Purpuric lesions 1 to 2 mm in diameter. Larger purpuric lesions are called ecchymoses

Classification According to Burket's 11th Ed

•THE PATIENT WITH ACUTE MULTIPLE LESIONS

Herpesvirus Infections-Cytomegalovirus infectionsCoxsackievirus Infections- Stevens Johnson Syndrome &TENVaricella-Zoster Virus Infection- ANUGErythema Multiforme- Contact Allergic StomatitisPrimary Herpes Simplex Virus Infections

•THE PATIENT WITH RECURRING ORAL ULCERS

Recurrent Aphthous Stomatitis

Behçet's Syndrome

Recurrent Herpes Simplex Virus Infection

•THE PATIENT WITH CHRONIC MULTIPLE LESIONS

Pemphigus Vulgaris, P Vegetans,
Subepithelial Bullous Dermatoses,
Mucous Membrane pemphigoidParaneoplastic Pemphigus
Bullous PemphigoidEpidermolysis Bullosa AquisitaLinear IgA Disease
Chronic Bullous Ds of Childhood
Herpes Simplex Virus Infection in Immunosuppressed Patients

•THE PATIENT WITH SINGLE ULCERS

Histoplasmosis Blastomycosis Mucormycosis Traumatic Injuries Causing Solitary Ulcerations Traumatic Ulcerative Granuloma

Fitz Patrick classification of Vesiculbullous Lesions

1.According to anatomical plane:a. Intra epidermal blister granular layer

1.Pemphigus Foliaceous 2.Frictional blister

3. Staphylococcus scalded

syndrome

b. Spinous layer

1. Eczematous Dermatitis

- 2. Secondary to heat and cold
- 3. Herpes virus infection

4. Familial Benign Pemphigus

2. Dermal-epidermal junction zone A. Lamina Lucida

- 1. Bullous Pemphigoid
- 2. Cicatrical Pemphigoid
- 3. Epidermolysis Bullosa Dystrophica

c. Suprabasal

Pemphigus vulgaris
 Pemphigus vegetans
 Darier's disease

d. Basal layer

- 1. Erythema Multiforme
- 2. Toxic epidermal necrolysis
- 3. Lupus erythematosis
- 4. Lichen planus
- 5. Epidermolysis bullosa

B. Below Basal Lamina

- 1. Erythema Multiforme
- 2. Epidermolysis Bullosa Dystrophica

Classification of Ulcers

1. MICROBIAL ORIGIN

A.BACTERIAL

1.Streptococcal 2.Tuberculosis 3.Syphilis 4.Scarlet fever 5.Diphtheria 6.Typhoid 7. Noma

B. FUNGAL

- Histoplasmosis
 Blastomycosis
 Paracoccidiomycosis
 Coccidiomycosis
- 5. Cryptococcus6. Zygomycosis7. Aspergillosis

C.VIRAL

1.Herpes 2.HIV 3.Pox virus

D. PROTOZOAL

1.Entamoeba histolytica 2.Leishmaniasis 3. Toxoplasmosis

2. PHYSICAL ORIGIN

- 1. Cheek bite(morsicatio buccorum)
- 2. Traumatic (TUGSE)
- 3. Thermal
- 4. Electrical
- 5. Osteoradionecrosis
- 6. Anaesthetic

4. METABOLIC ULCERS

- 1. Diabetes
- 2. Uremia
- 3. Neutropenia
- 4. Sickle cell anemia
- 5. Agranulocytosis
- 6. Crohn's disease

3. CHEMICAL ORIGIN

- 1.Phenol
- 2. Silver nitrate
- 3. Hydrogen peroxide
- 4. Aspirin

5. NON SPECIFIC

- 1. HIV ulcers
- 2. Graft vs host reaction
- 3. Necrotizing sialometaplasia
- 4. Reynaud's phenomenon
- 5. Bacterial angiomatosis

5. IMMUNOLOGICAL

1. Behcet's Syndrome

2. Reiter's Syndrome

3. Erythema Multiforme

4. Erosive Lichen Planus

(secondary ulcer)

5. Lupus Erythematosus

6. Sarcoidosis

7. Cyclic Neutropenia

8. Ulcerative Colitis

9. HIV

10. Pemphigus

11. Epidermolysis Bullosa

6. NEOPLASTIC Squamous cell carcinoma

Herpes simplex virus: (HSV)

HSV is a ubiquitous virus, linear DNA virus of herpesviridae family of viruses contains nine different viruses that are pathogenic in humans





Immunopathogenesis

HSV infects epithelial cells \Box sensory neurons \Box nerve cell body in sensory ganglion(latent infection) \Box Reactivation when host immunity goes down.

•Damage to prickle cells leads to acantholysis and vesicle formation.

•HSV induces cells locally to fuse and form giant cells/polykaryons (Tzanck cells).

•Intranuclear inclusion bodies known as Lipschutz bodies then appear.

•There is cell-to-cell spread resulting in viraemia.

•Common sites of HSV infection are oral, genital and eye.

Clinical Manifestations

- •1-3 day viral Prodrome of fever . Loss of appetite, malaise, and myalgia that may be accompanied by headache and nausea.
- •Most cases of primary HSV-1 infections are subclinical and generally occur in children and teenagers.
- •Dentists may get Herpetic whitlow as an occupation hazard



Oral findings

•Erythema and clusters of vesicles and/or ulcers appear on

- Keratinised mucosa
- •Non-keratinised mucosa

•Vesicles breakdown to form ulcers

Primary Herpetic Gingivo Stomatitis

Tiny numerous ulcers may coalesce to form larger ulcers with scalloped borders and marked surrounding erythema.
Gingiva – FIERY RED in colour and mouth extremely painful

Acute marginal gingivitis characteristic of primary A 12-year-old female with prima herpetic gingivostomatis causing discrete vesicles and ulce surrounded by inflammation

Fiery Red gingiva



Recrudescent oral HSV infection

- •Reactivation of HSV may lead to asymptomatic shedding of HSV.
- •It may also cause ulcers
- •Asymptomatic shedding is not associated with systemic signs and symptoms and occurs in 8 to10% of patients.
- •Important trigg
- Reoccurance of
- labialis.
 - -occur in 20 -associated
 - -Pain genera
- 1—5mm single erythematous b

ent herpes

gling or burning . 2 days. h bright

Cold Sore

HSV in Immunocompromised patients

Patients who are undergoing

- •Chemotherapy
- •Undergone organ transplantation
- •Immune deficiency syndrome (AIDS)
- •Recurrent intraoral HSV (RIH)
- •Most common sites of involvement were

Buccal/labial mucosa - 27%

Tongue -25%

Gingiva -18%

Differential diagnosis

Primary herpetic gingivostomatitis is usually apparent from clinical features

The sys signs and symptoms coupled with oral ulcers may require to differentiate from

- 1. Streptococcus pharyngitis
- 2. Erythema multiforme
- 3. Coxsackievirus infection
- 4. Acute necrotizing ulcerative gingivitis
- 5. Apthous ulcers
- 6. Traumatic lesions

Laboratory diagnosis
1.Cell culture
2. PCR
3. HSV can be identified from scrapings from the base of the lesions.
4. A smear similar preparation can be used for direct

fluorescent antigen detection test – Direct fluorescent

antigen testing.

5. Primary HSV i of immunoglobul titers.

Recurrent infec antibody. 6. Biopsy.



Management

Usually Self limiting but if required

Acyclovir – 15mg/kg five times a day

pain control and supportive care measure

2% viscous lidocaine (swish and spit out 5ml 4-5times/day Liquid diphenhydramine (swish and spit out 5ml 4-5times/day 0.1% dicyclomine hydrochloride Systemic analgesia Supportive care and Hydration Ice chips/popsicles Soft bland diet Antipyretics such as ibuprofen

Varicella zoster virus (VZV) infection

- It contains a double-stranded DNA genome & structurally very similar to HSV
- During disease process VZV may progress along sensory nerves to sensory ganglia where it resides latent and later forms Zoster



Varicella (chickenpox)

- •Transmission of varicella is believed to be predominantly thru' respiratory tract and leads to a viremia and subsequent mucocutaneous lesions.
- •Incubation period is 14-21 days.
- •Disease begins with low grade fever, malaise and headache, anorexia, irritability followed by vesicles and mouth ulcers which resembles those of herpetic stomatitis but there is no gingivitis typical of herpetic stomatitis; oral lesions affect mainly the palate and tongue

Vesicles and Ulcers can be seen on the palate



Herpes zoster

•Basically a condition of older adult population and individuals.

•Involvement of various branches of trigeminal nerve



, facial and ocular lesions. fever or lymphadenopathy days by appearance of crops

unilate distrik scabs nerve.

Sheldon Mintz DDS

Differential diagnosis

 Pulpitis
 Herpes simplex virus
 Pemphigus or pemphigoid
 ANUP
 Coinfection cytomegalovirus
 Bisphosphonate associated, radiation

Diagnosis

- 1. Cell culture
- 2. Direct fluorescent antibody testing
- 3. PCR
- 4. HZI causes transient rise in IgM and IgG titre
- 5. Biopsy

Treatment

Primary VZV: Acyclovir – 800mg 5 times for 7 days Valacyclovir- 1000mg 3 times/day for 7 days Famiciclovir- 500mg 3 times/day for 7 days

Pain control particularly Prevention of post herpetic neuralgia Supportive care Hydration Definitive treatment: Aspirin use contraindicated and ibuprofen is preferred analgesic.

Recurrent Aphthous Stomatitis (RAS)

- RAS is a disorder characterized by recurring ulcers confined to oral mucosa in patients with no other signs of disease.
- Affects 20% of population
- RAS is classified according to clinical characteristics as
- 1. Minor ulcers
- 2. Major ulcers
- 3. Herpetiform ulcers

Etiology

- a. Genetic factors
- b. Hematological deficiencies- Fe, Vit B₁₂ Folate
- c. Immunological abnormalities
- d. Local factors Smoking Trauma
 - e. Other factors include
 - Anxiety
 - Periods of psychological stress
 - Localized trauma to the mucosa
 - Menstruation
 - Upper respiratory infections
 - Food allergy

- 1. First episodes of RAS most frequently begin during the second decade of life.
- 2. Lesion begin with prodromal burning any time 2-48 hrs before an ulcer develop.
- 3. Individual lesions are round, symmetric and shallow.
- 4. RAS commonly involves buccal and labial mucosa, less common on heavily keratinized palate or gingiva.
- 5. There may be 2-6 ulcers /episode
- 6. 3-4 episodes a year

1. Minor RAS :

- size of ulcer is 0.3-1cm in diameter and heal without scarring within 10-14 days.
- 1 to 5 lesions may be present during each episode.



2. Major RAS :

Develop deep lesions that are larger then 1cm in diameter and heal in 2 to 6 weeks, and may cause scarring.
1to10 lesions may be present during each episode.



3. Herpetiform type RAS :

- Ulcerations demonstrates the greatest number of lesions and the most frequent recurrences.

- The individual lesions are small, averaging 1 to 3 mm in diameter, and as many as 100 may be present in a single recurrence.

- Ulcerations heal within 7-10 days but recurrence tend to

be closely spaced.



Differential diagnosis

- I. Primary herpetic gingivostomatitis
- 2. Mucous membrane pemphigoid and pemphigus
- 3. Erythema multiforme
- 4. Atrophic candidiases
- 5. Traumatic ulcer

Laboratory findings Complete Blood count Biopsies

Management

- 1. Protective Emolient- Orabase or Zilactin
- 2. Pain Relief- Mucopain, Dentogel
- 3. Topical Steroid
 - 1. Fluocinonide 0.05% cream QID
 - 2. Clobetasol 0.05% cream QID
- 4. Systemic Steroid- Prednisolone 20-40mg
- 5. Others
 - 1. Thalidomide 100-300 mg OD with water at bedtime
 - 2. Azathioprine 1-3 mg/kg/d
 - 3. Colchicine 500 μ g three times daily
 - 4. Pentoxifylline 400 mg three times daily