BDS Year 4 Regular & Casual batch

Academic Year 2023-2024

Subject: Oral Medicine and Radiology

Topic: VESICULOBULLOUS AND ULCERATIVE LESIONS-II

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SUMANDEEP VIDYAPEETH
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NECROTISING ULCERATIVE GINIGVITIS/PERIODONTITIS-

SUMANDEEP VIDYAPEETH
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Estd. 2007
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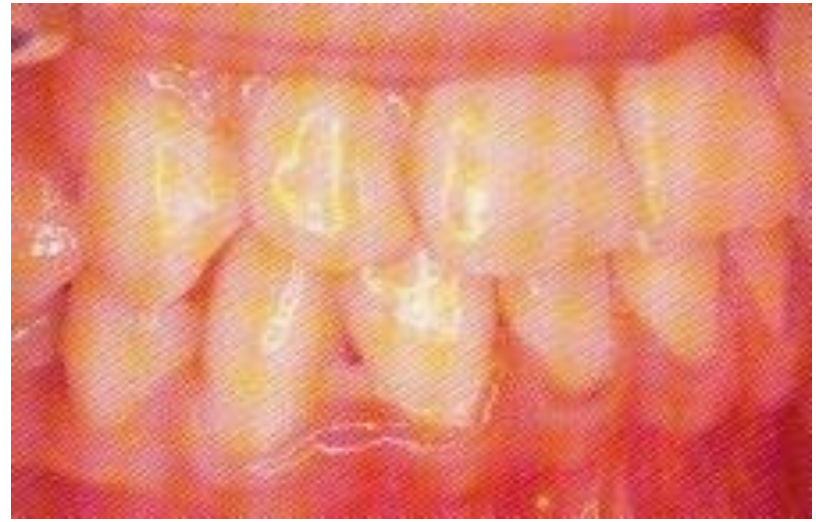
NUG/NUP

- Acute ulcerative inflammatory destructive conditions of the gingiva and periodontium respectively.
- Trench mouth.



Etiology

- **“the specific cause of ANUG has not been established. The prevalent opinion is that it is produced by a complex of bacterial organisms but requires underlying tissue changes to facilitate the pathogenic activity of the bacteria.**

Etiology

- **Predisposing factors**
  - **Local**
    - **Pre-existing gingivitis**
    - **Smoking**
    - **Injury to the gingiva**
  - **Systemic Predisposing Factors**
    - **Nutritional deficiency**
    - **Debilitating disease eg. Syphilis, cancer, ulcerative colitis, leukemia, anemia, AIDS**
    - **Psychosomatic factors**
      - **Stress situations eg.school examinations**

Epidemiology and Prevalence

- Epidemic pattern
- Highest incidence reported btw: 15 – 20yrs and 20-30 yrs
- Prevalence high amongst children under 10 yrs old from low socioeconomic backgrounds and underdeveloped countries

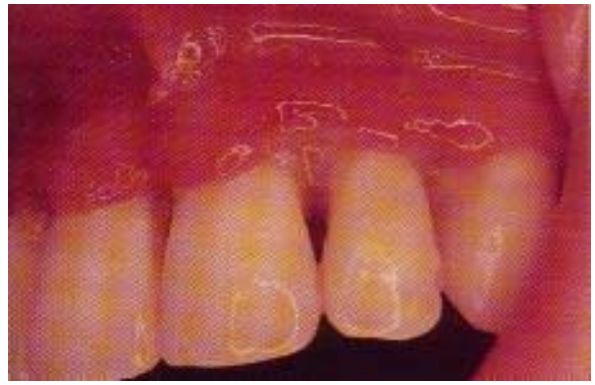
Microorganisms Involved

- **Zone 1: Bacterial Zone:**
  - Cocci, fusiform bacilli, Spirochetes
  - Treponema,prevotella intermedia,F.nucleatum,Peptostreptococccus,P.gingivalis.
- **Zone 2: Neutrophil-rich Zone**
  - Numerous leukocytes
  - Bacteria: fusiform bacilli, spirochetes
- **Zone 3:Necrotic Zone**
  - Disintegrated tissue cells
  - Fibrillar material
  - Remnants of collagen fibers
  - Spirocetes and other microorganisms
- **Zone 4: Zone of spirochetal infiltration**
  - Well preserved tissue
  - Infiltrated by spirochetes

- Tissue destruction is due to production of endotoxins/immunologic activation.
- Reduced chemotaxis and phagocytosis, resulting in poor control of infection.
- Underlying systemic disease –cancrum oris,noma/gangrene.
- F.necrophorum-dermonecrotoxin,hemolysin,leukotoxin and proteolytic enzymes.

Clinical Features of ANUG

- Sudden onset
- Usually associated with history of acute respiratory tract infection or debilitating disease, a change in living habits, psychological stress
- Appears as punched out crater like depressions at the crest of the interdental papilla, extending to marginal gingiva
- Ulcers covered my gray pseudomembranous slough.



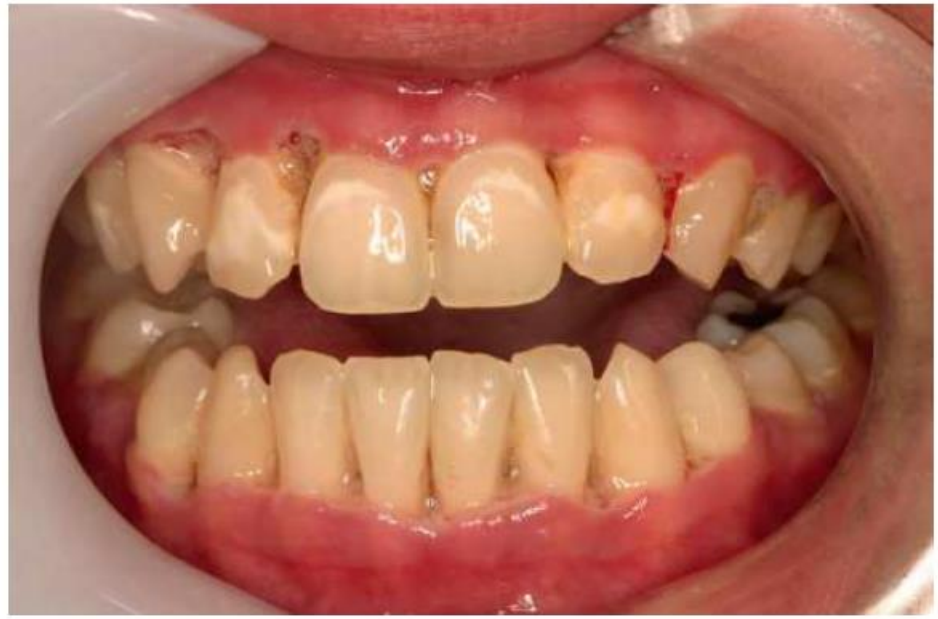


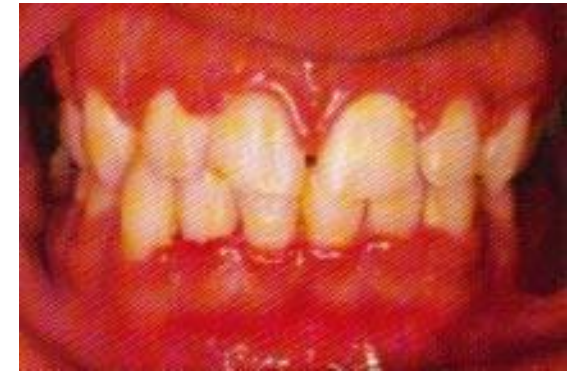
Figure 3-13 Necrotizing ulcerative gingivitis with typical punched out, necrotic, and ulcerated interdental papillae.

Clinical Features of ANUG

- Lesions may be denuded of the surface pseudomembrane, exposing the gingival margin which is red, shiny, and hemorrhagic
- There is progressive destruction of the gingiva and periodontal tissues
- Spontaneous pronounced gingival bleeding
- Fetid odor
- Increased salivation

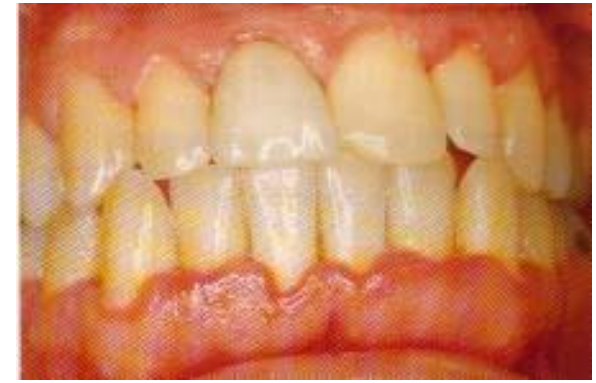
Clinical Features of ANUG

- Lesions extremely sensitive to touch
- Pain
- Mettalic foul taste
- Excessive amounts of pasty saliva
- Local lymphadenopathy
- Slight elevated temperature



Clinical Features of ANUG

- Severe cases present with
  - High fever
  - Increased pulse rate
  - Leukocytosis
  - Loss of appetite
  - General lethargy
- Children
  - Tend to have more severe systemic involvement including:
    - Insomnia
    - Constipation
    - GI disorders
    - Headache
    - Mental depression



ANUG

- Sequelae
  - Noma or gangrenous stmatitis
  - Fusospirochetal meningitis
  - Peritonitis
  - Pulmonary infections
  - Toxemia
  - Fatal brain abscess

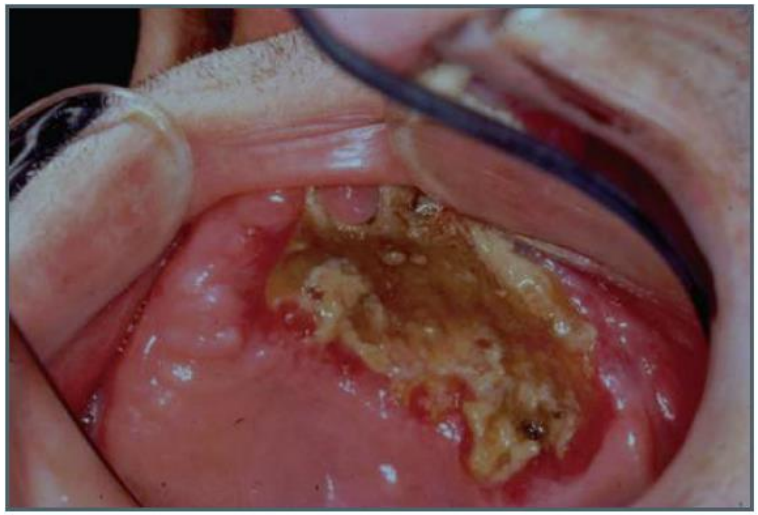


Figure 3-14 Fusospirochetal palatal lesions in a neutropenic patient.

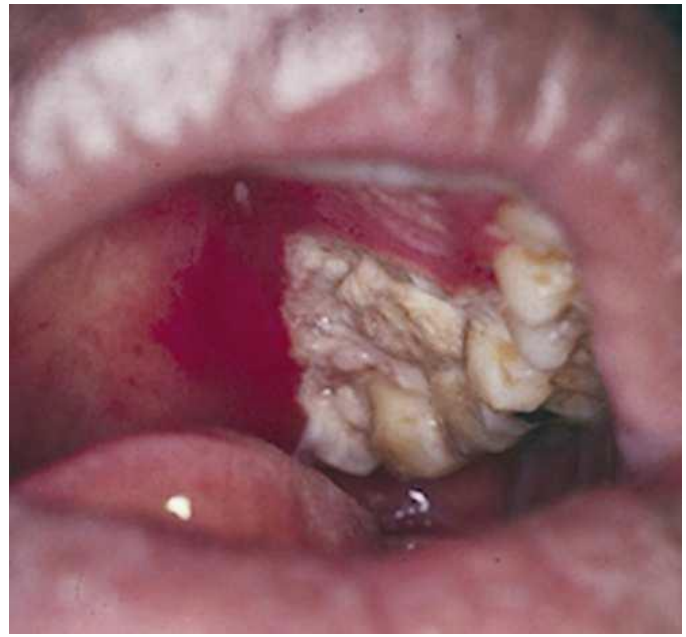


Figure 3-15 Necrotizing ulcerative periodontitis with osteonecrosis in a patient with AIDS.

Histopathology

- Involves both the stratified squamous epithelium and the underlying connective tissue
- Surface epithelium is destroyed and replaced by a pseudomembranous meshwork of fibrin, necrotic epithelial cells, PMN's and microorganisms
- Connective tissue is hyperemic, with numerous engorged capillaries and dense infiltration of PMN's
- Numerous plasma cells appear at the periphery of the infiltrate- chronic marginal gingivitis

Histopathology

- Epithelium and connective tissue have decreasing alterations in appearance as the distance from the necrotic gingival margin increases
- The microscopic changes are non-specific and similar changes occur after trauma, chemical irritation and application of escharotic drugs
- ANUG does not involve formation of a conventional periodontal pocket
- Necrotic changes involve the junctional epithelium
- When supporting structures are involved the condition is called Necrotising ulcerative periodontitis

Differential Diagnosis

- Acute herpetic gingivostomatitis
- Chronic periodontal pockets
- Desquamative gingivitis
- Streptococcal gingivostomatitis
- Aphthous stomatitis
- Gonococcal gingivostomatitis
- Diptheritic and syphilitic lesions
- Tuberculous gingival lesions
- Candidiasis
- Agranulocytosis
- Dermatoses(pemphigus, erythema multiforme, lichen planus)
- Stomatitis venenata

Communicability

- ANUG is transmissable but not contagious or communicable
- A predisposed host and the presence of appropriate bacteria are necessary for the production of this disease

- **In patients with AIDS, the prevalence is 6% and is strongly predictive of CD4 count below 200cell/mm3**

LAB TESTING

- Culture
- Biopsy
- HIV TEST

Treatment

Local

- Debridgement, ultrasonic scaling,
- Rinses of chlorhexidine
- 3% hydrogen peroxide+saline- 12 times a day
- Topical povidone iodine

Systemic antibiotics-

- Penicillin-500mg qid for 5 days
- Tetracycline-250-500mg 6 hrly
- Metronidazole-200-400mg tds

15mg/kg body wt 6 hrly I.V. Eliminating predisposing factors

Maintainance of oral hygiene

Prognosis

- Excellent

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Erythema Multiforme

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Definition

- ***Erythema Multiforme*** **(EM) is an acute disorder of the skin and mucosal membranes manifesting in the oral cavity as polymorphic erosive, ampullar and erythematous lesions, and bloodstained crusts preferentially located in the non-keratinized mucosa.**

Classified within this group are

- Erythema multiforme minor (emm),
- Erythema multiforme major (EMM),
- Stevens–johnson syndrome (SJS), and
- Toxic epidermal necrolysis (TEN).

Etiology

- The etiology is unclear.
- The etiology of EM is unclear, although a type IV cytotoxic immune reaction is known to be involved, mediated by T lymphocytes that react to antigens (viral, bacterial, pharmacological, or chemical).
- Cytotoxic immune complexes are formed; these in turn affect the keratinocytes, causing important intra- and subepithelial damage.
- The keratinocytes show intra- and extracellular edematous phenomena, necrosis and apoptosis-mediated cell death
- However, an immunologically mediated process triggered ***by herpes simplex*** or *Mycoplasma pneumoniae*, drugs, radiation, or malignancies, is probable.

- It is believed that 15–70% of all cases of recurrent EM are associated to previous or concomitant outbreaks of herpes simplex virus (HSV) infection.
- Drugs, including particularly antibiotics, anticonvulsivants, antifungals, or sulfa drugs, are related to 40–50% of all outbreaks of EM.
- There have been recent reports of other triggering drug substances such as valdecoxib , oral contraceptives , or metoprolol

- **Three diagnostic categories were established:**

(i) *Minor forms*, in the presence of typical rosette-like skin lesions covering less than 10% of the total body surface, affecting a single mucosa (oral), and with erythema, erosions, ulcers and lip crusts;

(ii) *Major Forms*, when in addition to skin involvement (less than 10% of the total surface) at least one additional mucosal membrane other than the oral mucosa is affected – the oral lesions being extensive and severe; and

(iii) *Stevens–Johnson syndrome (SJS).*

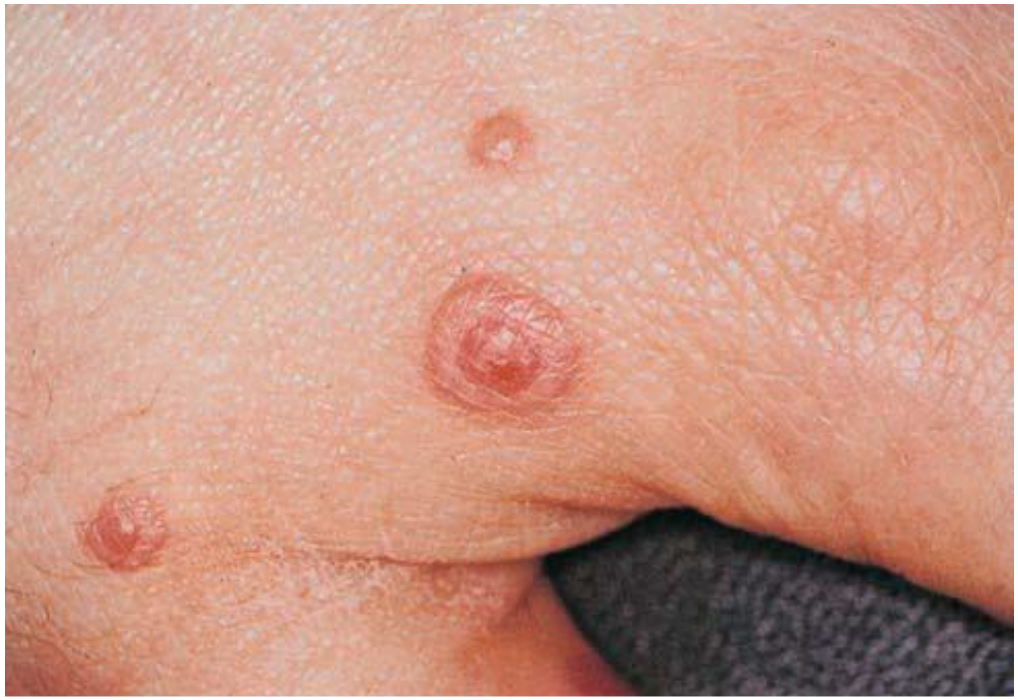
CLINICAL FEATURES

- Erythema multiforme typically affects adults under 50 years of age.
- Patients with recurrent EM have an average of 6 episodes a year.
- There is a prodrome of fever malaise,headache,sore throat,rhinorrhea and cough.

Clinical features

- The skin manifestations consist of erythematous, flat, round macules, papules, or plaques, usually in a symmetrical pattern.
- They start primarily in the hand and moving centripetally in the trunk in a symmetric pattern.
- The most common sites are upper extremeties,face and neck.

- The skin lesions take on several forms- ***MULTIFORME.***
- The classic skin lesions consist of a central blister or necrosis with concentric rings of variable color around it called **target- or iris-like lesions.**
- Skin bullae may occasionally be seen.
- Recurrences are common.



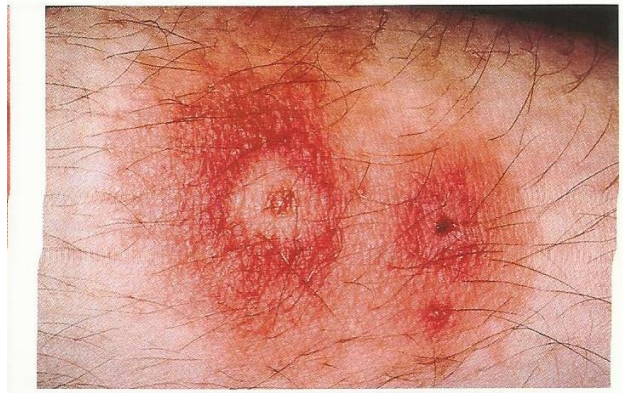
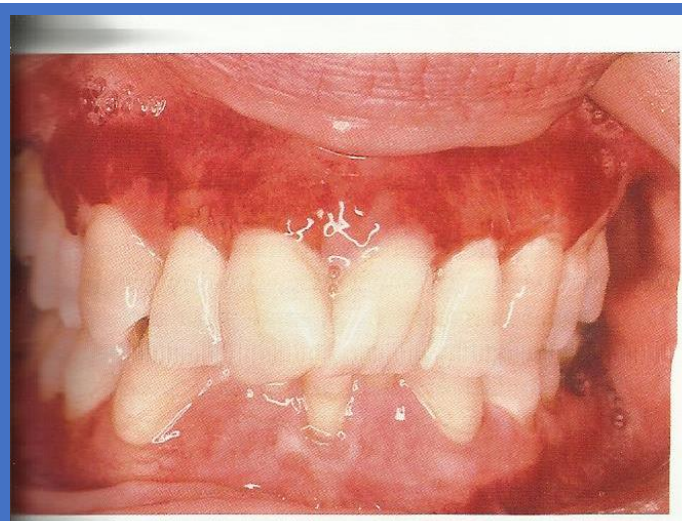


Fig. 75.2. **Erythema multiforme:** classic target skin lesions.

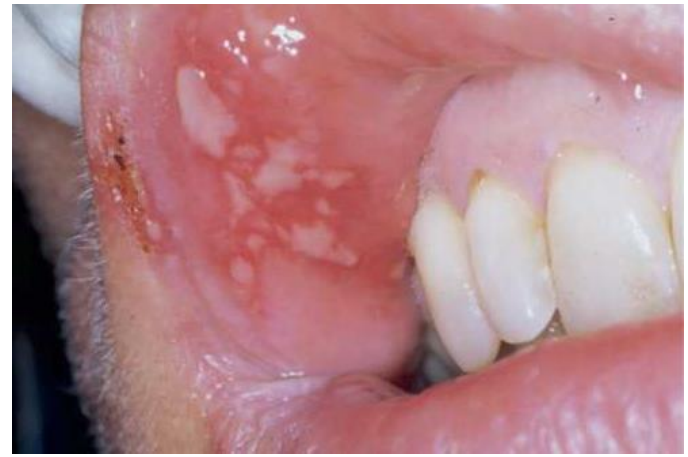
Erythema multiforme: typical target- or iris-like lesions of the skin.

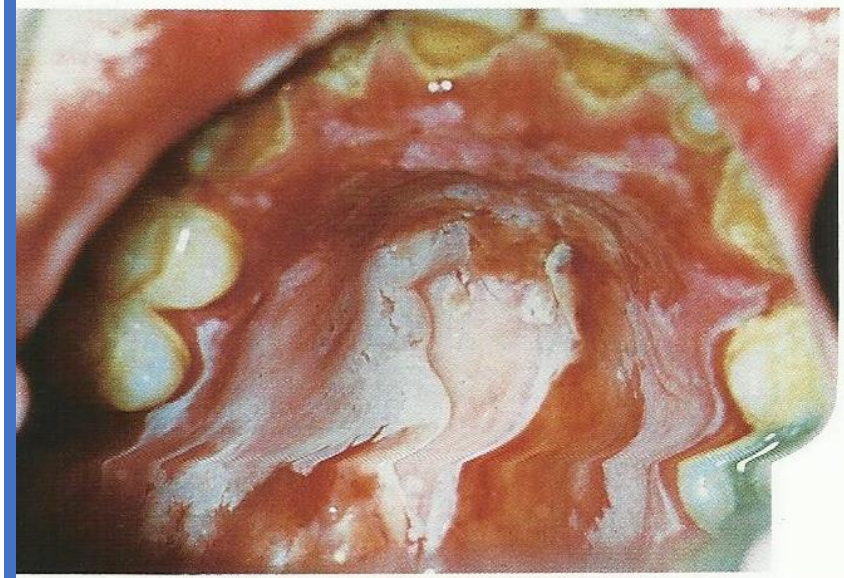
Clinical features

- The oral lesions present as coalescing small vesicles that rupture within two or three days, leaving irregular, painful erosions covered by a necrotic pseudomembrane (Fig)
- The lips, buccal mucosa, tongue, soft palate, and floor of the mouth are most commonly involved.



Oral **erythema multiforme:** gingival involvement.





Erythema multiforme: erythema and ulceration.

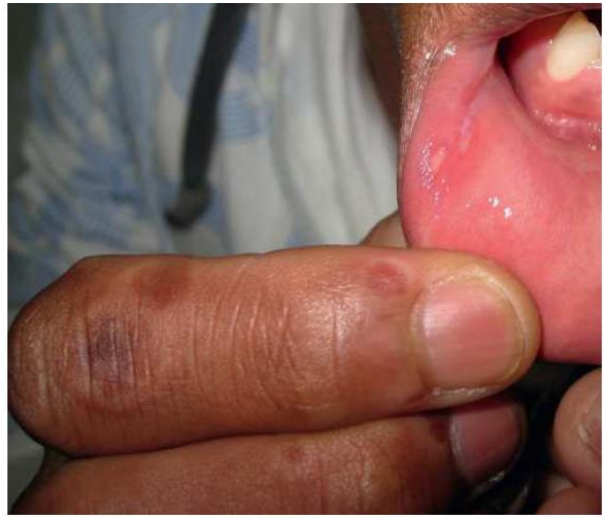


Figure 3-18 Erythema multiforme with target lesions on the skin of the fingers and intraoral ulcers. *Source:* Courtesy of Dr. Adam Lipworth, Boston, MA.

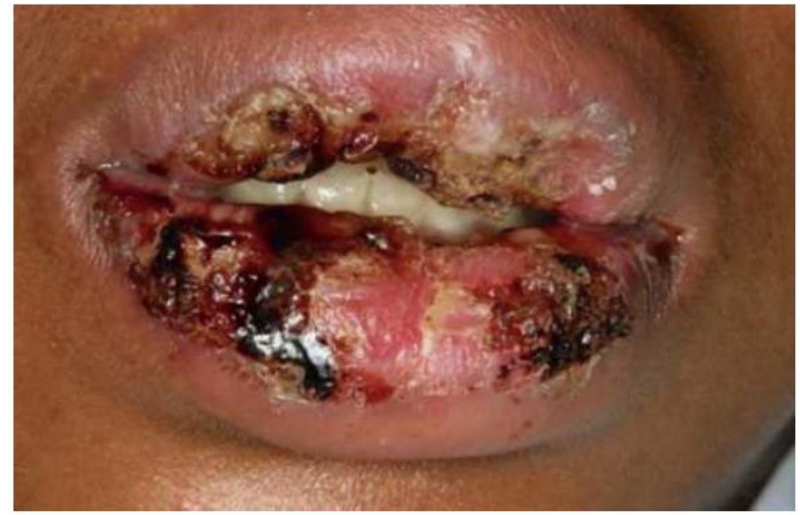
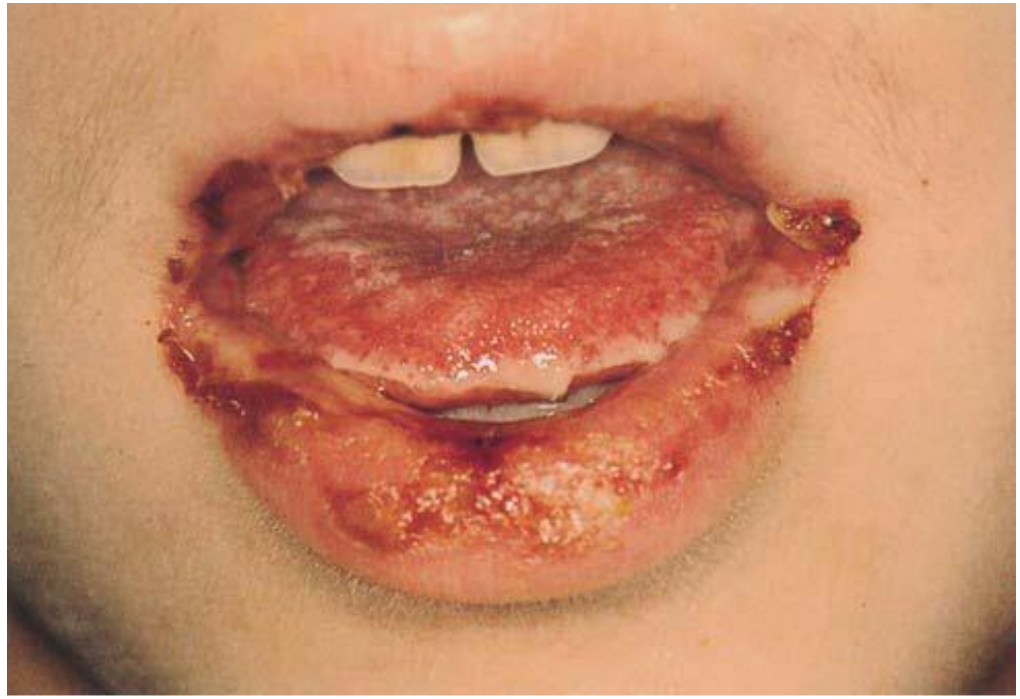


Figure 3-19 Erythema multiforme with hemorrhagic crusts of the lips.



Erythema multiforme: multiple erosions on the lips and tongue.

- When severe,ulcers may be large and confluent,causing difficulty in eating,drinking and swallowing and may drool blood tinged saliva.

Laboratory tests

- **Histopathological examination.**

Differential diagnosis

- Primary herpetic gingivostomatitis,
- Aphthous ulcers,
- erosive lichen planus,
- pemphigus vulgaris,
- pemphigoid.

Treatment

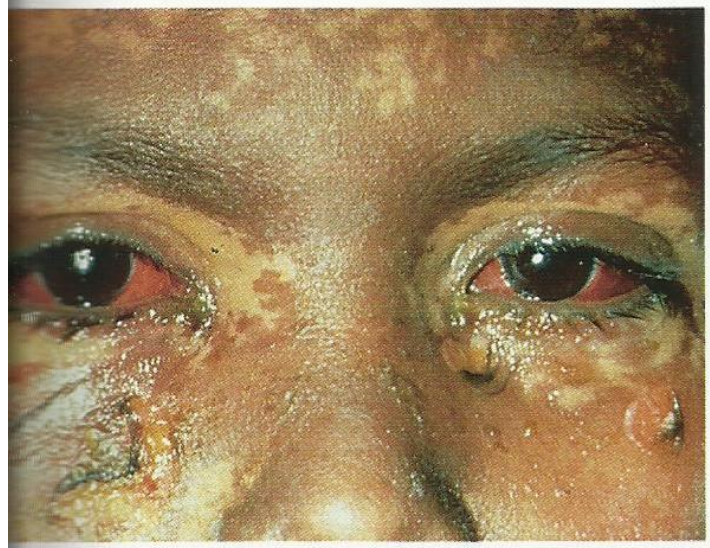
- Mild EM-SYSTEMIC /TOPICAL analgesics
- Severs EM-Systemic steroids.
- Acyclovir may be helpful in cases of recurrence. 400mg tid

Title	Triggers, clinical manifestations, and management of pediatric erythema multiforme: A systematic review
Authors	Zoghaib S, Kechichian E, Souaid K, Soutou B, Helou J, Tomb RJ Am Acad Dermatol. 2019 Sep;81(3):813-822.
Objective	To investigate the triggers, clinical manifestations, and treatment of pediatric EM
	The mean age was 5.6 years, ranging 0.1-17 years. Infectious agents were the main triggers: herpes simplex virus (HSV) in 104 patients (17.9%) and Mycoplasma pneumoniae in 91 patients (15.7%). In total, 140 cases (24.1%) were drug-related and 89 cases (15.3%) had other triggers, such as vaccines (19 patients, 3.2%). In total, 229 patients had EM major (39.5%). Treatment was supportive care only (180 patients, 31.1%), systemic corticosteroids (115 patients, 19.8%), antivirals (85 patients, 14.6%), and antibiotics (66 patients, 11.3%), mostly macrolides (45 patients, 7.7%). Long-term sequelae were rare (1.3%). Pediatric EM was reported in 19 infants (3.2%). The main trigger was vaccination (9 patients). Infantile EM was EM major in 2 cases and EM minor in 17. Infants were less prone to develop EM major than older children (P < .01). Pediatric EM was recurrent in 83 cases (14.3%), which was triggered by HSV in 36 patients (61%). Recurrence affected older children.
	This condition can affect all mucosal surfaces, most commonly the oral mucosae. The diagnosis is clinical, and management relies on supportive care. Vaccines are a particular trigger in infants. Recurrent cases are most commonly linked to HSV. Dermatologists and pediatricians should be aware of this potentially recurrent and

STEVEN JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS

- They are more severe and tend to arise on the chest rather than the extremities on erythematous and purpuric macules.
- More likely associated with infection with Mycoplasma pneumonia and drugs like
- NSAIDS
- Anticonvulsants
- Sulfonamides

- The mucosal surfaces of the eye, genitalia and mouth are almost always severely affected by SJS/TEN, always with skin involvement.
- The typical oral manifestation is extensive oral ulceration with haemorrhagic crusts on the vermillion



Stevens-Johnson syndrome: eye, skin involvement.

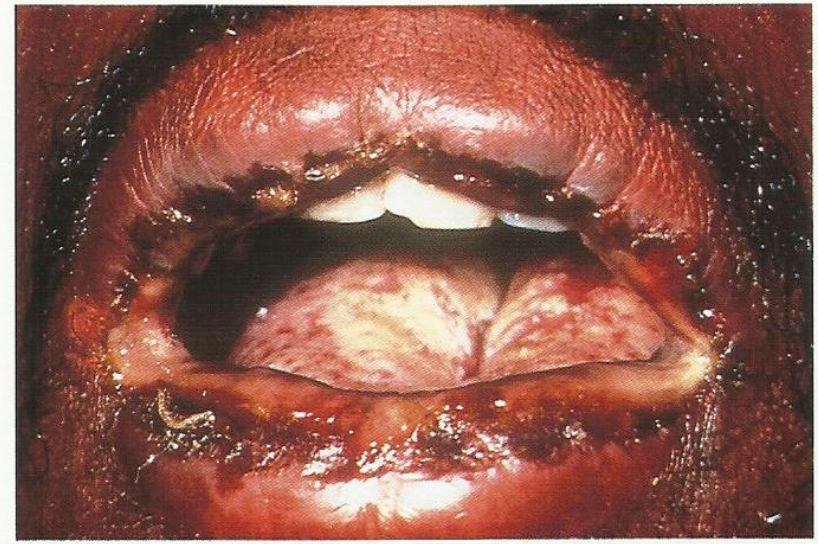
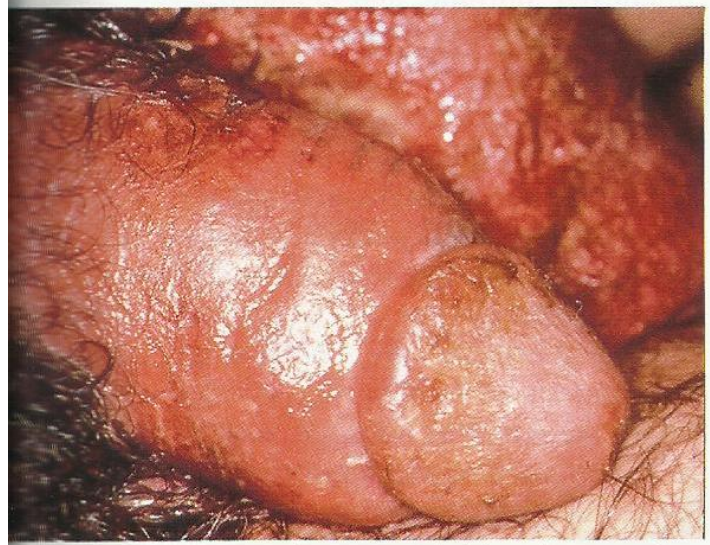


Fig. 75.6. Stevens-Johnson syndrome: blood-crusted lips.



Stevens-Johnson syndrome: genital involvement.*

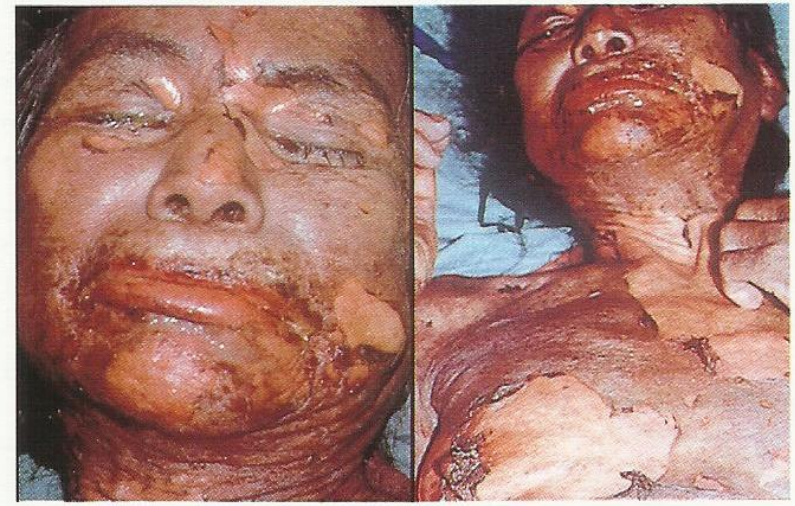


Fig. 75.8. Toxic epidermal necrolysis: life threatening.

- HP –most of the disease is localized in the dermis.

Management

- High doses of systemic steroids
- IV immunoglobulins
- Thalidomide

ORAL HYPERSENSITIVITY REACTIONS

- EM
- LICHENOID LESIONS
- FIXED DRUG ERUPTIONS
- PLASMA CELL STOMATISTIS
- ANGIOEDEMA
- ORAL ALLERGY SYNDROME

THE PATIENT WITH RECURRING ORAL ULCERS

RAS

BEHCET SYNDROME

RHL

REM

RECURRENT APHTHOUS STOMATITIS

- Recurrent aphthous stomatitis **(RAS; aphthae; canker sores)** is a common condition which is characterized by multiple recurrent small, round or ovoid ulcers with circumscribed margins, erythematous haloes, and yellow or grey floors, appearing first in childhood or adolescence.

Epidemiology

- Depending upon the group examined, RAS may affect 5–60%.
- The highest incidence (60%) was found in female student nurses, male student dentists(56%) and
- Professional school students (55%).
- There may be female predominance of RAS in adults.

Aetiopathogenesis

- A genetic predisposition is present, as shown by an increased frequency of certain human leukocyte antigen (HLA) types, and a positive family history in some patients with RAS.

- Haematinic deficiency is found in up to 20% of patients,
- cessation of smoking may precipitate or exacerbate RAS in some cases, and
- stress may underly RAS in some individuals.
- It has been suggested in some, but not all, studies that sodium lauryl sulphate (SLS) detergent in some oral healthcare products may give rise to ulceration akin to that of RAS.

- Ulcers similar to RAS may be seen in human immunodeficiency virus (HIV) and some other immune defects, and drugs, especially non-steroidal anti-inflammatory drugs (NSAIDS) and nicorandil may produce lesions clinically similar to RAS in appearance, but not periodicity.

Genetic basis

- At least 40% of RAS patients have a vague familial history of RAS.
- There is evidence that patients with RAS have changes in cell-mediated immunity.
- Patients with RAS may have increased levels of peripheral blood CD8+ T lymphocytes and/or decreased CD4+ T lymphocytes.

Microbial aspects of RAS

- Oral streptococci were previously suggested as important in the pathogenesis of RAS, either as direct pathogens or as antigenic stimuli in the genesis of antibodies that may conceivably cross-react with the oral mucosa.
- S. sanguis and S. Mitis
- An association between RAS and Helicobacter pylori has also been suggested, but the evidence suggests such a link is unlikely.
- IgM and IgG antibodies to VZV may be elevated in some RAS patients

Local factors predisposing to RAS

- Local, physical trauma may initiate ulcers in susceptible people.
- A significantly reduced frequency of tobacco smoking in individuals with RAS compared with an appropriate control group.
- Allergy to metals, nuts, chocolates and dairy products.
- Dentifrice use

Table 1 Some disorders with similar characteristics to recurrent aphthous stomatitis

Disease	*Comment*
Behcet's disease	RAS-like ulceration is a cardinal feature of Behcet's disease. The ulceration may be more severe, and more likely to comprise major and/or herpetiform ulcers from RAS. Patients with Behcet's disease also have recurrent genital ulceration, cutaneous disease (usually papulopustular lesions or erythema nodosum), ocular disease (typically posterior uveitis) and a range of other gastrointestinal, neurological, renal, joint and haematological abnormalities
MAGIC syndrome	Comprises major aphthae and generalized inflamed cartilage. A variant of Behcet's disease
Sweet's syndrome	Also termed acute neutrophilic dermatosis. Affected patients have superficial ulceration similar to RAS. In addition, there is sudden onset fever, leucocytosis and well demarcated cutaneous, plum-coloured papules or plaques. Usually arises in middle-aged females. In 50% of patients there is an associated malignancy (e.g. acute myeloid leukaemia)
PFAPA syndrome	Comprises periodic fever, aphthae like oral mucosal ulceration, pharyngitis and cervical adenitis. Although rare, PFAPA tends to occur in young children. Tends to be self-limiting, and non-recurrent. May respond to cimetidine (via suppression of T lymphocyte function)
Cyclic neutropenia	Cyclic reduction in circulating levels of neutrophils about every 21 days. Affected patients develop oral ulceration, fever, cutaneous abscesses, upper respiratory tract infections and lymphadenopathy. Other oral complications include severe gingivitis and aggressive periodontitis. Treated with recombinant granulocyte colony stimulating factor (rG-CSF). Other neutropenias (e.g. chronic neutropenia) can give rise to superficial oral mucosal ulceration without any significant periodicity
HIV disease	Aphthous-like ulceration may occasionally arise in HIV disease. However, it remains unclear, if there is a significantly raised frequency of recurrent idiopathic oral ulceration in HIV disease

- Haematinic (iron, folic acid or vitamin B12) deficiencies may be twice as common in some groups.
- Inflammatory bowel disease.

Table I. Suggested causes of recurrent aphthous stomatitis.

Local/oral factors
Trauma
Salivary gland dysfunction
Microbial
Bacterial: streptococci
Viral: varicella zoster, cytomegalovirus
Systemic factors
Behçet's disease
Crohn's disease
Ulcerative colitis
Cyclic neutropenia
Mouth and genital ulcers with inflamed cartilage syndrome
HIV infection
Stress
Nutritional
Gluten sensitive enteropathy
Iron, folic acid, zinc deficiencies
Vitamin B1, B2, B6, B12 deficiencies
Genetic
Immunologic
Localized T-cell dysfunction
Antibody-dependent cellular cytotoxicity

- RAS comprises recurrent bouts of one or several rounded, shallow, painful ulcers at intervals of a few months to a few days.
- RAS has three main presentations

minor (MiRAS), major (MaRAS) or herpetiform (HU) ulcers.

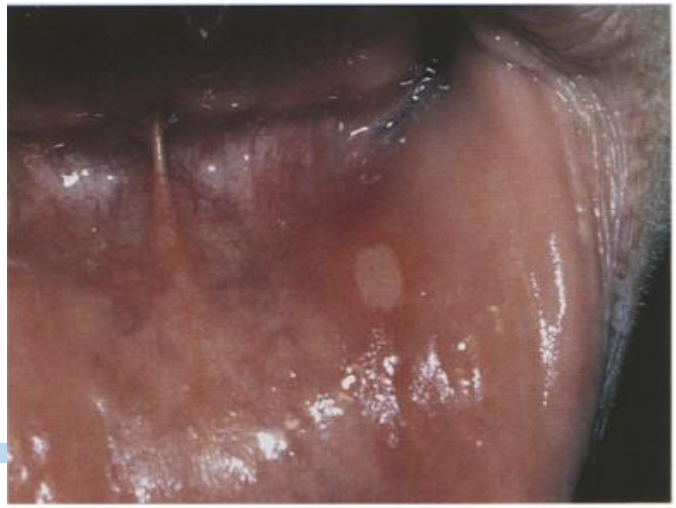
- The MiRAS is the most common, affecting about 80% of patients with RAS: ulcers are round or oval usually <5 mm in diameter with a grey white pseudo membrane and an erythematous halo.

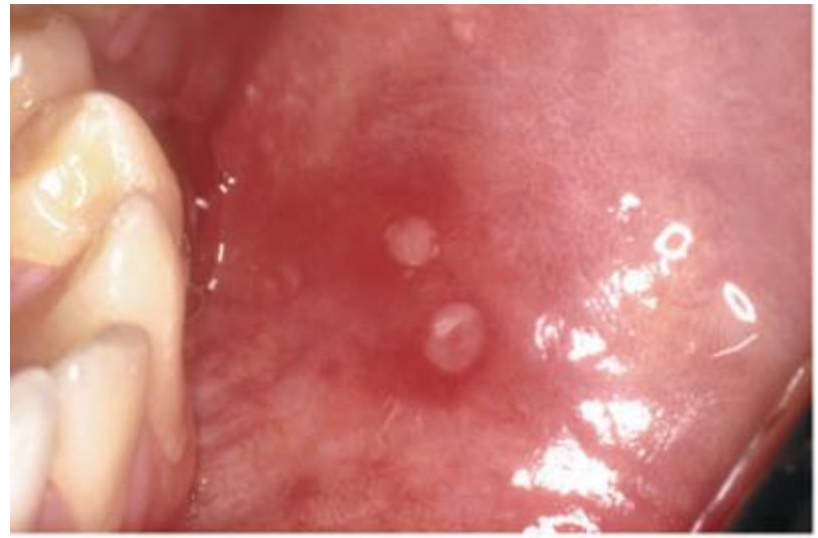
TABLE II. *Characteristics of recurrent aphthous stomatitis*[a]

Characteristic	MiAU	MjAU	HU
Female-male ratio (% females)	1.3:1 (56)	0.8:1 (44)	2.6:1 (73)
Age at onset (yr)	10–19	10–19	20–29
Lesions			
Size (mm)	<10	>10	1–2
Site	"Moveable oral mucosa"		Entire oral mucosa
	Lips, cheeks, tongue	Lips, cheeks, tongue, palate, pharynx	
Number	1–5	1–10	10–100
Healing with scarring (%)	8	64	32

MINOR RAS

- MiRAS usually occur on the labial and buccal mucosa and floor of mouth, but are uncommon on the gingiva, palate or dorsum of the tongue.
- The ulcers heal within 10–14 days without scarring.

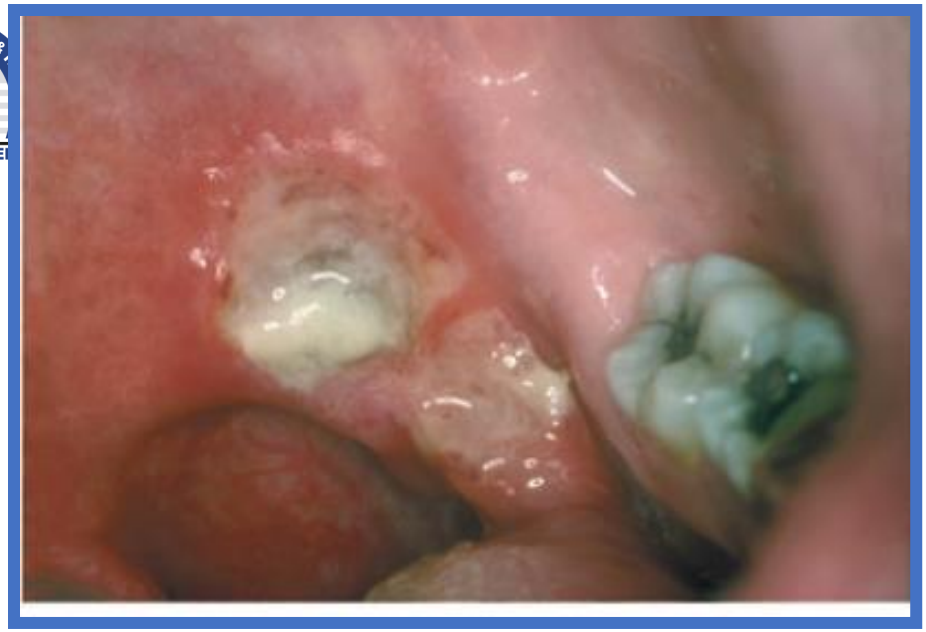


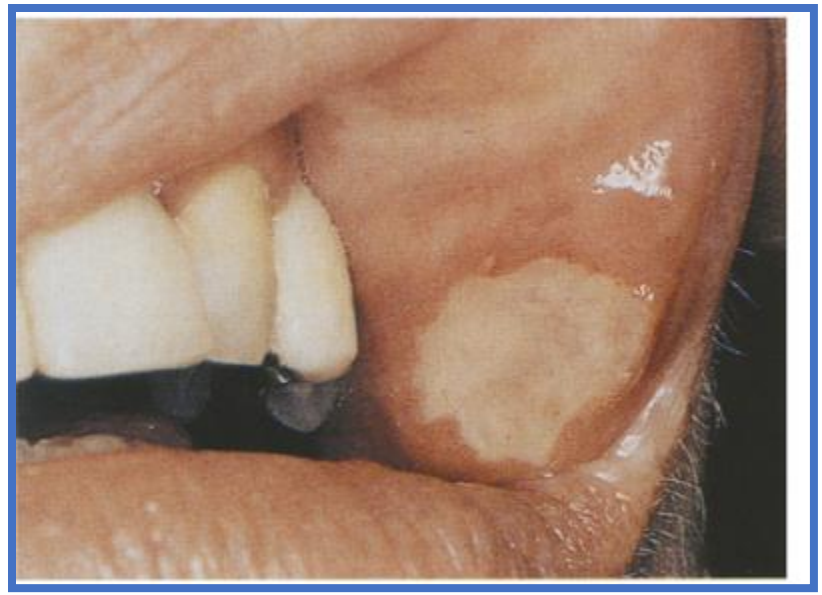


gure 4 Typical minor aphthae (buccal mucosa, 22-year-old male)

MAJOR RAS

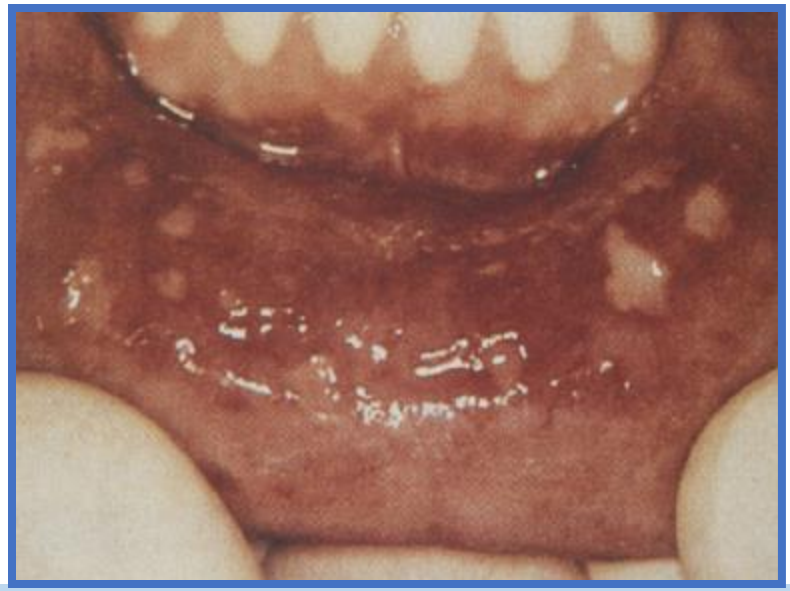
- Major RAS is a rare, severe form of RAS, sometimes termed **periadenitis mucosa necrotica recurrens**
- These ulcers are oval and may exceed 1 cm in diameter and have a predilection for the lips, soft palate and fauces.
- The ulcers persist for up to 6 weeks and often heal with scarring.
- MaRAS usually has its onset after puberty and is chronic, persisting for up to 20 or more years.





HERPETIFORM ULCERATION

- Herpetiform ulceration is the least common variety and is characterized by multiple recurrent crops of widespread small, painful ulcers.
- As many as 100 ulcers may be present at a given time, each measuring 2–3 mm in diameter ,although they tend to fuse producing large irregular ulcers.
- HU may have a predisposition for women and have a later age of onset than other types of RAS



DIFFRENTIAL DIAGNOSIS

- Connective tissue disorders-SLE
- Granulomatous disorders- TB, Sarcoidosis
- Pemphigus pemphigoid

INVESTIGATIONS

- Haematological investigations- CBC-Hb, peripheral smear.
- Serum iron, folate and Vitamin B12
- Rule out malabsorption syndromes
- Patch test
- Granulomatous diseases- TB & sarcoidosis
- HIV test CD4 count
- Biopsy

MANAGEMENT

- Removal of etiological factors
- Mild cases-supportive treatment-emollient-orabase ,topical anaesthetics
- Severe cases- high potency topical steroids.

Table II. Currently recommended treatments of recurrent aphthous stomatitis

- Topical therapies*
  - Glucocorticoid creams/ointments
    - Triamcinolone acetonide (Kenalog 0.1% or 0.5%, or Kenalog in orabase)
    - Fluocinonide 0.05% gel or ointment (Lidex)
    - Betamethasone valerate 0.1% (Valisone)
    - Clobetasol propionate 0.05% cream or ointment (Temovate)
  - Glucocorticoid elixirs
    - Dexamethasone elixir 0.5 mg/5 ml (Decadron)
  - Glucocorticoid injections
    - Triamcinolone diacetate 25 mg/ml (Aristocort-Intralesional)
    - Betamethasone sodium phosphate/betamethasone acetate 6 mg/ml (Celestone Soluspan)
  - Antimicrobials
    - Chlorhexidine gluconate 0.12% (Peridex)
  - Analgesics
    - Dexamethasone elixir 0.5 mg/5 ml (Decadron)
    - Diphenhydramine HC1 elixir 12.5 mg/5 ml (Benadryl)
    - Dyclonine HC1 0.5% or 1.0% (Dyclone)
    - Lidocaine HC1 viscous 2% (Xylocaine)
    - All can be combined in a 50% elixir with sucralfate, Kaopectate, or Maalox
- Systemic therapies†
  - Medications
    - Glucocorticoids
      - Prednisone: 60 mg qd or qod for two doses decreasing to 40 mg, 30 mg, 20 mg, 10 mg, 5 mg (each qd or qod for two doses)
    - Immunomodulators
      - Azathioprine 50 mg bid (Imuran)
  - Nutritional replacements
  - Avoidance of allergens
  - Stress reduction

Table 2 Some reported therapies for recurrent aphthous stomatitis (RAS)

Local physical treatment	Surgical removal
	Debridement
	Laser ablation
	Low dense ultrasound
	Chemical cautery (e.g. silver nitrate sticks)
	Physical barriers (e.g. cyanoacrylate adhesives)
Antimicrobials	Chlorhexidine gluconate (mouthrinse)
	Triclosan (mouthrinse)
	Topical tetracyclines (e.g. aureomycin, chlortetracycline, tetracycline)
Topical corticosteroids	Hydrocortisone hemisuccinate (pellets)
	Triamcinolone acetonide (in adhesive paste)
	Flucinonide (cream)
	Betamethasone valerate (mouthrinse)
	Betamethasone-17-benzoate (mouthrinse)
	Betamethasone-17-valerate (mouthrinse)
	Flumethasone pivolate (spray)
	Beclomethasone diproprionate (spray)
	Clobetasol proprionate (cream)

Topical analgesics	Benzydamine hydrochloride (spray or mouthrinse)
	Topical anaesthetics (gel)
Other topical anti-inflammatory agents	Amlexanox
	Sodium cromoglycate (lozenges)
	Carbenoxolone sodium mouthrinse
	Azalestine
	Human alpha-2-interferon (cream)
	Ciclosporin (mouthrinse)
	Deglycirrhizinated liquorice
	Topical 5-aminosalicylic acid
	Prostaglandin E2 (gel)
	Topical granulocyte-macrophage colony-stimulating factor
	Aspirin mouthrinse
	Diclofenac in hyaluronase
	Sucralfate
Systemic immunosuppression	Prednisolone
	Azathioprine
	Levamisole
	Colchicine
	Thalidomide
	Pentoxifylline
	Dapsone
	Cimetidine

Title	Low-Level Laser Therapy and Topical Medications for Treating Aphthous Ulcers: A Systematic Review
Authors	Khaleel Ahmed M, Jafer M, Nayeem M, Hussain Moafa I, Quadri MFA, Gopalaiah H, Ali Quadri MF. J Multidiscip Healthc. 2020
Objectives	The study compares low-level laser therapy with topical medications for treating aphthous ulcers.
	A search of articles in this systematic review was completed in six databases. Treatment and comparative groups comprised of patients subjected to laser therapy and topical medications, respectively. Two different treatment outcomes were considered; pain and size of the lesion.
Results	The overall sample comprised of 98 males and 232 females, with a mean age of 32.4 years. The laser therapies in each included study had different active media and varying wavelengths. Topical medication used in the comparative group were triamcinolone acetonide, amlexanox, granofurin, and solcoseryl. Findings showed that patients who reported lower pain and decreased aphthous ulcer lesions were more in the laser therapy group than in the topical medication group.
Conclusion	Low-level laser therapy was better in treating aphthous ulcer lesions in comparison to topical medications, and all laser wavelengths in the included reports were seen to be effective

Conclusion

- Although common, RAS remains an elusive disorder, as the precise aetiology and long-term behaviour of disease are ill understood.
- No therapies are available to stop the bouts of ulceration and not give rise to adverse side-effects.
- The management of RAS is thus presently directed towards lessening physical trauma to the oral mucosa, and reducing the inflammatory response.

BEHCETS SYNDROME

- It was initially described by a turkish dermatologist- Hulusi Behcet.
- Eastern Asia,Middle East and Mediterranean- Turkey and Japan

ETIOLOGY

- Immune dysregulation of circulating immune complexes,autoimmunity,cytokines.
- HLA-B51
- A bacteria /virus triggers an immune reaction in a genetically predisposed individual.

CLINICAL FEATURES

- TRIAD
- Recurring oral ulcers
- Recurring genital ulcers
- Eye lesions

Young adults -25-50 years

Oral mucosa most comon next is genital region.

DIAGNOSTIC CRITERIA

- **RECURRENT oral ulceration occuring in atleast 3 times in one 12 month period plus 2 of the following:**
- **Recurring genital ulcers**
- **Eye lesions- uveitis/retinal vasculitis**
- **Skin lesions-erythema nodusum, pseudofolliculitis, papulopustular lesions or acneiform nodules in post adolescent patients receiving corticosteroids.**

- Positive ***Pathergy*** test- placing a 20 gauge needle 5 mm into the skin of the forearm. The test is positive if an indurated papule or pustule greater than 2mm in diameter forms within 24 hrs.

LAB FINDINGS

- LAB TESTS TO rule out other diseaes like LE and blood dyscrasias

MANAGEMENT

- Depends on the severity of the disease
- Prednisone with immunosuppressive drugs- Azathioprine, Dapsone ,Colchicine and Thalidomide.
- Anti TNF – Infliximab and etanercept.

1. Sutton’s disease is
    1. Minor aphthae
    2. Major aphthae
    3. Herpetiform aphthae
    4. Reiter’s disease

2.Necrotic punched out ulcers are characteristic of

a) Bechet's disease

b) Erythema multiforme

c) ANUG

d) Hand ,foot and mouth disease

3.Hyper-reactivity to intracutaneous injection or a needle stick is found in patients with

a) Pemphigus
b) HSV infection
c) Behcets disease
d) Erythema multiforme

4.Antiviral prophylaxis is mandatory in recurrent cases of HSV for prevention of which disease

a) Herpes zoster

b) Herpetic neuralgia

c) Erythema multiforme

d) All of the above

5.Target lesions are seen in

a) Herpangina

b) Hand foot and mouth disease

c) Erythema multiforme

d) Behcet syndrome