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Oral mucosa Ulceration:- is a common reason for patients to seek medical or dental advice, and because many oral ulcerative or vesiculobullous disease have a similar clinical appearance the clinician must be taken a careful and detailed history to provide a much particular information to categorize a patient's disease and simplify the diagnosis.
The most important information are:

- Length of time the lesions have been present.
- Past history of similar lesions (primary, recurrent or persistent episodes).
- Number of lesions present (single or multiple).

Dermatological lesions are classified according to their clinical appearance into:-

Macules:- well-circumscribed, flat lesions that are noticeable because of their change from normal skin or mucosa colour (red or pigmented).

Papules:- Solid lesions raised above the skin or mucosal surface, < (1 cm) Ø.

Plaque:- Solid raised lesions, > (1 cm) Ø; they are large papules.

Nodules:- Lesions are present deeper in the dermis or mucosa.

Vesicles:- Elevated blisters containing clear fluid , < (1 cm) Ø.

Bullae:- Elevated blisters containing clear fluid, > (1 cm) Ø.

Pustules:- Blisters containing purulent material.

Erosions:- Red lesions often caused by the rupture of vesicles or bullae or trauma and are generally moist on the skin.

Ulcer:- Well-circumscribed, often depressed lesions with an epithelial defect that is covered by a fibrin clot, causing a yellow-white appearance.

Purpura:- reddish to purple bruises caused by blood from vessels leaking into the connective tissue. These lesions do not blanch when pressure is applied and are classified by size as Petechiae (< 0.5 cm) or Ecchymosis (> 0.5 cm).

Principal Causes of oral mucosal ulceration include:-

1- Trauma.
2- Recurrent ulceration (RAS, BS).
3- Microbial infections (primary and secondary Herpes simplex, zoster, hand food mouth disease, NUG or NUP, syphilis, and TB.
4- Mucocutaneous diseases. (Pemphigus, Pemphigoid, EM, Linear IgA Disease, Epidermolysis Bullosa Aquisita).
5- Drug therapy.
6- Systemic disorders. (Hematological, GIT).
7- Squamous cell carcinoma.

Trauma

The most common cause of single ulcers on the oral mucosa is trauma.

Aetiological factors:

1- Physical:-

a. Mechanical.

Due to sharp, broken teeth, orthodontic and prosthetic (ill-fitting denture) appliances, numb lips or tongue being bitten after a local anaesthetic injection, malocclusion, overzealous tooth brushing and flossing, and self-injurious habits .

b. Thermal.

Due to ingesting hot foods and beverages such as hot pizza or coffee). An iatrogenic cause of thermal injury is from a heated dental instrument inadvertently contacting the mucosa, the burn is usually more serious if the mucosa has been anaesthetized and there is prolonged contact.

c. Electrical.

Due to inadvertently chew on electrical wiring that occur especially in children.

2- Chemical.

Due to aspirin placed directly on the oral mucosa, as a remedy for toothache, can cause a (chemical burn), use of denture cleansers as an oral rinse, and prolonged contact of methacrylate monomer on the mucosa may also lead to necrosis of the mucosa.

Clinical features and Oral findings:
Sore, painful to touch, irregular border with erythematous margins and a yellow base, during the healing phase they frequently develop a 'keratotic halo'.

Differential diagnosis:
- Aphthous ulcer. – Syphilis. – Tuberculosis. - Squamous-cell carcinoma.

Management:
- Elimination of suspected cause.
- Antiseptic mouthwash (e.g. 0.2 per cent chlorhexidine)

Or

- Simple covering agent such as Orabase.
- If it persists for more than 10-14 days after elimination of the aetiological factor, biopsy should be carried out to exclude squamous cell carcinoma.

Recurrent Ulceration

⊠ Recurrent aphthous stomatitis (RAS)

It's the most common oral mucosal diseases affecting humans. Most patients with RAS have between two and six ulcers at each episode and with several episodes (not less than three) in a year.

Aetiology:
The causative factors of RAS are unknown but there are many **predisposing factors** that lead to the RAS such as:-

1. Anxiety and Stress.
2. Menstruation.
3. Microbial.
4. Food hypersensitivity.
5. Genetic factors.
6. Immunologic abnormalities.
7. Hematologic and Nutritional deficiency (serum iron, folate, or vitamin B12).
8. Trauma.

Clinical features and Oral findings:
The first episodes of RAS most frequently begin during the second decade of life. The lesions are confined to the oral mucosa and begin with burning or pricking sensation for a short period (from 2 to 48 hours) before an ulcer appears (prodromal stage). Following this phase, a localized area of erythema develops (initial period). After that within hours, a small white papule forms, ulcerates, and gradually enlarges over the next 48 to 72 hours.
The individual lesions are round or ovoid, painful, symmetric, and shallow (similar to viral ulcers), but no tissue tags are present from ruptured vesicles (helps distinguish RAS from diseases that start as vesicles such as pemphigus and pemphigoid).

Multiple lesions are often present, but the number, size, and frequency vary considerably. The most common affected area are buccal and labial mucosa (non-keratinized), but the less common are palate and gingiva (keratinized), if the tongue is involved it cause difficulty during eating or speaking. Lymph node enlargement is seen only as a response to secondary infection in severely affected patients.

Types of RAS:

There are a differences in the clinical presentations; According to this differences it divided in to three types:-

1- Minor recurrent aphthous stomatitis (**MiRAS**).

   It's the most common form of RAS and approximately 80% of patients have lesions of this type. The characteristic form presents the picture of a recurrent, small ulcers single or multiple (1-5 ulcers), size is < (1cm) Ø, duration between 7-14 days and heal without scar formation. The site of this type is the nonkeratinized mucosa especially (labial/buccal mucosa, dorsum and lateral borders of tongue).

2- Major recurrent aphthous stomatitis (**MjRAS**).

   The characteristic form presents the picture of a recurrent, small ulcers single or multiple (1-3 ulcers), size is > (1cm) Ø, duration between 2 weeks to 3 months and heal with scar formation. All the oral cavity are effected with this type (keratinized plus nonkeratinized mucosa), particularly (soft palate).

   Note):- the long period of time involved when the single ulcer is seen in isolation, may lead to the suspicion that the lesion is malignant.

3- Herpetiform ulceration (**HU**).

   It's the less common form of RAS.The characteristic form presents the picture of a recurrent, small multiple ulcers (5-20 (up to 100 ulcers), with small size (1-2mm), duration between 7-14 days, the site of this type is the nonkeratinized mucosa, but particularly (floor of the mouth and ventral surface of tongue). And heal without scar formation unless a number of ulcers coalesce "Where many ulcers are present they coalesce to form larger confluent areas of ulcer, usually with marked erythema".

Differential diagnosis:

- Herpes simplex. - Hand-foot-and mouth disease. - Behcet's disease. - Cyclic neutropenia.
- Erythema multiforme. - Squamous cell carcinoma.

Management:

- Remove all predisposing factors.

- Orabase - Topical antiseptic. - Topical analgesics (diclofenac). - Topical antibiotic (tetracycline). - Topical steroids (steroid gel two to three times a day after meals and at bedtime).

- In severe cases: Intralesional steroid injection or systemic steroids in low doses (10-20 mg prednisone) for four to eight days can reduce the symptoms dramatically.

******((The choice of therapy depends on the severity and frequency of ulceration but the objectives of treatment are to relieve discomfort, reduce secondary infection, promote healing of existing ulceration, and prevent new ulcers occurring))

⊠ Behcet's disease (Behcet's syndrome) "BD"

Behcet 's (pronounced ' Betchet's ') syndrome was first recognized in turkey and was originally thought to be a disease of Mediterranean origin.

Clinical manifestations:

Women are more commonly affected than men and the highest incidence in young adults between the ages of 25 and 40. It's characterized by a classical triad of RAS 'any of the three clinical variants', recurring genital ulcers, and inflammatory eye lesions). Other manifestations include skin, joint, neurological, vascular, and intestinal disorders.

The most common site of involvement is oral mucosa and the second most common site involvement is genital area, the ocular involvement is usually late, occurring sometimes after many years of intermittent oral and genital ulceration. The eye lesions consist of uveitis, retinal vasculitis, optic atrophy, and conjunctivitis. And the most common complication of the eye lesions in late episodes are permanent damage by scar formation or, even, to "Blindness".

Diagnosis:

Because the signs and symptoms of BD overlap with several other diseases, so a set of diagnostic criteria of BD was developed that includes RAS occurring at least three times in one year period plus any two of the followings:-

1- Recurrent genital ulceration.
2- Eye lesions.(uveitis or retinal vasculitis)
3- Skin lesions. (Include erythema nodosum, papulopustular lesions, or acneiform nodules in postadolescent patients not receiving corticosteroids).

4- Positive pathergy test. (Which is performed by placing a 20-gauge needle 5mm into the skin forearm. The test is positive if an indurated papule or pustule greater than 2mm in diameter forms within 24-48 hours).

Differential diagnosis:
- RAS. - Ulcerative colitis. - Erythema multiforme. - Syphilis.

Management:-
- Local management of RAS
- Systemic steroids
- Azathioprine
- Anti-TNFα therapy and mycophenolate.

Microbial infections

☒ Necrotizing ulcerative gingivitis (NUG)

Acute ulcerative-inflammatory conditions of gingiva and periodontium, respectively, that are associated with smoking, stress, poor oral hygiene, local trauma, immune suppression disease.

Aetiology:
Tissue destruction is most probably a result of the production of endotoxins and/or immunologic activation and subsequent destruction of the gingiva and adjacent tissues.
The infections (NUG and NUP) can spread rapidly from the gingiva to the periodontium and into the soft tissues if the patients underlying systemic illness (with severe immunodeficiency or malnutrition).
Fusobacterium necrophorum play an important role in the progression of NUP to cancrum oris. A tissue destruction occurs because this organism produces a dermonecrotic toxin, hemolysin, leukotoxin, and proteolytic enzymes.

Clinical features and Oral findings:
Characterized by malaise, submandibular lymphadenopathy. Intra orally it occur usually on the interdental papillae, although any part of the marginal gingiva may be affected.
The 1st symptoms include:-
Oral Medicine lecture
1- Excessive salivation. 2- Metallic taste. 3- Sensitivity of the gingiva.

And then rapidly develop into
1- Painful. 2- Erythematous gingiva with scattered punched-out ulcerations.

The most common compilations are malodour and gingival bleeding.
Gingiva may rise or progress to noma (cancrum oris or orofacial gangrene). The overlying skin becomes discolored, and perforation of the skin ensues. The orofacial lesions are cone-shaped, with the base of the cone within the oral cavity and the tip at the skin aspect. There is sloughing of the oral mucosa followed by sequestration of the exposed, necrotic bone and teeth.

Differential diagnosis:

- Herpetic gingivitis. - Desquamative gingivitis (such as mucous membrane (cicatricial) pemphigoid, pemphigus vulgaris, lichen planus, and hypersensitivity reaction). - Leukemia.

Management:

- Systemic metronidazole and oxygen-releasing agents topically

followed by - a mechanical gingival treatment (scaling and root planning).

- Periodontal surgery to correct gingival and periodontal defects.

☒ Hand-Foot-and-mouth disease

A very common enterovirus infection (coxsackievirus A10 or A16), which may occur in mild epidemic proportion, chiefly in children younger than 10 years in summer. It characterised by low-grade fever, sore mouth and skin lesion especially on the palmar, plantar surface of hands and feet consist of a red macular rash, each macule surrounding a deep- seated vesicle.
Oral mucosal lesions begin as erythematous macules that become vesicles and quickly break down to ulcers it usually located on the tongue, hard and soft palate, and buccal mucosa but can present on any oral mucosal surface. The symptoms resolve in a week

Diagnosis:

1- Concomitant oral and cutaneous lesions.
2- Skin lesions commonly involve hands, feet and may involve buttocks.
3- Antibody-titer increase measured between acute and recovery phases.

Differential diagnosis:

- Aphthous ulcers. - Herpes simplex infection. - Herpangina

Management:

- Symptomatic treatment only

Note ** [Patient should be cautioned against the use of aspirin to manage fever ((Avoided for children with viral infections)); because when the aspirin given during (viral infections) has been associated with an increased incidence of Reye syndrome (fatal, fulminant hepatitis with cerebral edema)].

☒ Primary herpetic gingivostomatitis

Herpes simplex virus (HSV) is a DNA virus, divided in to two types:

- Herpes simplex type 1: in general it occur above the waist and typically that affect the oral mucosa, pharynx and skin (Oro-facial infection).
- Herpes simplex type 2: it present below the waist and predominantly involves the genitalia (genital infection).

It's a relatively common viral infection of mouth and it affects mainly young children and young adults but in early childhood the infection may pass unnoticed (subclinical).

Clinical feature and Oral findings:

Firstly start with the prodromal phase that expected for one to two days it characterized by the initial symptoms such as high fever, malaise, tiredness, generalized muscle aches, sore throat and lymphadenopathy (submandibular lymph nodes are enlarged and tender)) and is followed by the appearance of oral and sometimes circumoral lesions.

Oral and circumoral lesions: groups of vesicles (numerous coalescing vesicles) which rapidly rupture to produce painful small, round, shallow ulcers covered by yellow fibrin but sometimes the groups' confluent with each other that give a result in the formation of large areas of ulceration. Any of the oral mucosal surfaces are involved and whole of the oral mucosa are a bright-red and sore, the incubation period is about 5-7 days.

Encephalitis and Meningitis are a complications that occur when the primary infection become widespread and disseminated throughout the body.

✓ *Secondary (Recurrent) herpes simplex infection **or** Secondary (Recurrent) herpetic stomatitis:

It's a common oral and perioral disease that is due to reactivation of HSV-1. The lesions almost occur in any site on the face but the most common (usual) site is on or near the lips and the lesion is known as 'herpes labialis' or a 'cold sore' and it less in skin and mucosa of the nose.

The provoking factors for recurrence include sunlight, mechanical trauma, cold, heat, fever, emotional stress.

It start by Prodromal symptoms include itching, tingling, or burning followed by the lesions present as multiple small vesicles or papules that leaving a crusted ulcers it heal spontaneously within 6–10 days.

Differential diagnosis:

Aphthous ulcers, hand-foot-and-mouth disease, herpangina, acute necrotizing ulcerative gingivitis, erythema multiforme,

Management:

- Supportive measures.
- -Antipyretics/Analgesics (systemic).
- -Antiseptic/Analgesic mouthwashes.
- -Antiviral drug.(Topical – aciclovir and penciclovir cream used for herpes labialis or Systemic – used for primary herpetic gingivostomatitis).

☒ Varicella Zoster Virus infection

The Primary infection with VZV is called a (chicken pox) the virus then becomes latent, usually in the dorsal root ganglia or ganglia of the cranial nerves.

The Reactivation produces herpes zoster infection (HZI), commonly called shingles. The incidence of HZI increases with age and the degree of immunosuppression.

This virus is cytopathic to the epithelial cells of the skin and mucosa, causing blisters and ulcers. Transmission is usually by the respiratory route.

Clinical feature and Oral findings:

- Primary VZV infection (chicken pox):- it occurs in the first two decades of life characterized by a low-grade fever, malaise, and the development of an intensely pruritic, maculopapular rash, followed by vesicles that have been described as "dewdrop-like." These vesicles turn cloudy and pustular, burst, and scab, with the crusts falling off after 1 to 2 weeks.

Lesions begin on the trunk and face and spread centrifugally. The complications are central nervous system involvement may result in cerebellar ataxia, encephalitis, pneumonia, myocarditis, and hepatitis.

- HZI (shingles):- is more common in adults and starts with a prodrome of deep, aching, or burning pain and little to no fever or lymphadenopathy, this is followed within 2 to 4 days by the appearance of crops of vesicles in a dermatomal or "zosteriform" pattern. This pattern characterized by unilateral, linear, and clustered distribution of the vesicles, ulcers, and scabs in a dermatome supplied by one nerve.

One of the most important complications of HZI is post-herpetic neuralgia, defined as pain that lingers for 30 days or 120 days after the onset of the acute rash and the Predisposing factors for post-herpetic neuralgia include older age, prodromal pain, and more severe clinical disease during the acute rash phase.

The unilateral location of the lesions is a typical pattern of herpes zoster and the oral manifestations occur only when the second and third branches of the trigeminal nerve are involved

- The V2 affected that lead to lesions on the midface and upper lip with ulcers (rarely vesicles, because it break down quickly) on the hard palate or even buccal gingiva.
- The V3 affected that lead to lesions on the lower face and lower lips with blisters and ulcers on the mandibular gingiva and tongue.

But the most common one is the V1 and leads to lesions on the upper eyelid, forehead, scalp and if the corneal involvement may lead to blindness.

And when the geniculate ganglion involving that called Ramsay Hunt syndrome which characterized by:-

- Bell's palsy. - Vesicles of the external ear. - Loss of taste sensation in the anterior 2/3 of the tongue.

Differential diagnosis:

- Herpes simplex. - Erythema multiforme

Management:

- Primary VZV infection acyclovir (800 mg five times a day) acyclovir has poor bioavailability. Or
- Valacyclovir (1,000 mg 1x3) or famcyclovir (500 mg 1x3) for 7 days is effective in treating HZI and should be started within 72 hours of disease onset. These drugs also reduce the incidence of postherpetic neuralgia when compared with acyclovir.

The first line of treatment for postherpetic neuralgia is gabapentin and 5% lidocaine patch, and the second line of treatment is with opioid analgesics and tricyclic antidepressants

Mucocutaneous diseases

☒ Pemphigus

Pemphigus is an autoimmune disease causing vesicles or bullae on skin and mucous membranes and a very serious condition usually fatal if untreated
. It's a diseases of middle age with most patients between the ages of 40-60 years and it affects both sexes but female is a most common one.
There are four major types of pemphigus: vulgaris, vegetans, foliaceus, and erythematosus.
Pemphigus vulgaris is the most common one and accounts for about 70% of all cases.

Clinical feature and Oral findings:

Skin lesions: A characteristic signs of disease clinically is the Nikolsky sign which is the ability to form or extend a bulla by the:-

1. Application of pressure to an intact bulla, the bulla enlarges by extension to an apparently normal skin surface.

Or

2. Application of pressure to an apparently normal area results in the formation of a new bulla.

Oral lesions: present in 80-90% of patients during the course of the disease, and in 60% of the cases, the oral lesions are the first sing because it present for months before the skin lesion appear. The bulls and vesicles are fragile and infrequently seen intact in the mouth because it rapidly break so the clinician sees shallow, irregular, painful, tender, and ragged edges ulcers with thin layer of epithelium peels away in an irregular pattern, leaving a denuded base as a

result of the split and fragile epithelium. It's occur in any site within the mouth and oropharynx but the lesions start most commonly on the buccal mucosa, palate and gingiva respectively.

Diagnosis:

1- Positive Nikolski sign in clinical examination
2- Incisional biopsy: intraepithelial vesicle or bulla therefore lie above the basal and.
3- Smear from bulla fluid to see Tzank cells
4- DIF: IgG autoantibodies on the intercellular substances
5- IIF (indicates the progress of the disease): +ve IgG auto-antibodies in 90% of patients.

Differential diagnosis:

Pemphigoid, linear IgA disease, Epidermolysis bullosa acquisita, Primary herpetic gingivostomatitis, Erythema multiforme, Erosive lichen planus, Aphthous ulcer

Management:

Management varies according to several factors, including the severity of the disease and the speed at which the disease progresses.

In severe cases

- Systemic corticosteroids (Prednisone), usually given in dosages of 1 to 2 mg/kg/d.

But patients with only oral involvement also may need lower doses of prednisone for shorter periods of time.

Immunosuppressive drugs such as mycophenolate mofetil, azathioprine, or cyclophosphamide to reduce the complications of high dose of steroid.

☒ Pemphigoid

Is an autoimmune subepidermal (subepithelial) blistering disease that typically affects the elderly but may rarely present in children and younger adults. 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.

There are two broad clinical subtypes of pemphigoid include:

1- Bullous pemphigoid (**BP**)

It’s the most common one, occurs in main, elderly, most being over the age of 60 years; it's rarely life threatening, self –limited and may last from a few months to 5 years and it particularly conjunction with other diseases or drug therapy such as multiple sclerosis malignancy and diuretics. Caused by the Binding of antibody to antigen activates both leukocytes and complement, causing localized damage to the basement membrane, resulting in vesicle formation in the subepithelial region

Clinical feature and Oral findings:

Skin lesions: are a blister on inflamed base that involves the scalp, arms, legs, groin; and the common feature is a pruritis which initially present as macules and papules.

Oral lesions: 30-50% of patients with oral involvement. Desquamative gingivitis is the most common oral manifestation and the gingival lesions is the only site of oral involvement that consist of generalized edema, inflammation, and desquamation with localized areas of discrete vesicle formation.

Diagnosis:

1- Incisional biopsy: subepithelial bulla
2- DIF: Autoantibodies (mainly IgG), C3 in a linear distribution at the basement membrane zone.
3- IIF : +ve IgG auto-antibodies in 75% of patients but it not a reliable test for BP, used only to distinguish BP from Epidermolysis bullosa aquisita (EBA)

2- Mucous membrane pemphigoid (**MMP**) [Cicatricial Pemphigoid]

It's a chronic disease primarily affects any mucosal surface, but oral mucosa is the most common affected site and the second most common site is the conjunctiva. Occurs twice as frequently in women over the age of 50 years. Lesion occurs when autoantibodies directed against proteins in the basement membrane zone, acting with complement (C3) and neutrophils, causing subepithelial split and subsequent vesicle formation.

Clinical feature and Oral findings:

Skin lesions: are present in 20-30% usually in head and neck region of patients.

Oral lesions: over 90% of patients with oral involvement. Desquamative gingivitis is the most common manifestation and the only manifestation of the disease appearing bright red. The lesions present as intact vesicles of the gingival or other mucosal surfaces, but more frequently they appear as nonspecific-appearing erosions and heals by scaring. The erosions typically spread more slowly than pemphigus lesions and are more self-limiting.

Diagnosis:

1- Incisional biopsy: subepithelial bulla.
2- DIF: Autoantibodies (mainly IgG), IgA, C3 in a linear distribution at the basement membrane zone.
3- IIF : +ve IgG autoantibodies in 75% of patients and IgA 50%.

Management:

Management varies depends on the severity of symptoms and site of involvement.

- Topical, Intralesional or systemic corticosteroids
-In severe oral lesions, conjunctival or laryngeal involvement, dapsone therapy is recommended as the next choice before considering long-term systemic steroids or immunosuppressive drug therapy. But the patient's hemoglobin must be closely monitored because the side effect of dapsone are hemolysis, methemoglobinemia, glucose-6-phosphate dehydrogenase deficiency.
- Desquamative gingivitis most be treated.

Differential diagnosis:

Pemphigus, pemphigoid (PB or MMB), Linear IgA disease, Dermatitis herpetiformis, Epidermolysis bullosa acquisita, Erosive lichen planus, Discoid lupus erythematosus, Chronic ulcerative stomatitis.

☒ Erythema multiforme (EM)

Is an acute vesiculobulous disease of mucous membranes and skin (mucocutaneous disease) and the term "multiforme" because it have a wide range of clinical presentations. The patients are predominantly young adults between the ages of 20 and 40 years and more in males than females.

It's classified as

1- EM minor (EM) if there is less than 10% of skin involvement and there is minimal to no mucous membrane involvement.
2- EM major (Stevens-Johnson Syndrome (SJS)) if there are skin, oral mucosa and other mucous membranes affected.[is a severe form of EM that predominantly affects the mucous membranes]

There is unknown exact aetiology but there are many precipitated factors including:

1. Infections (particularly herpes simplex viral).
2. Drugs (sulfonamide and anticonvulsants).
3. Pregnancy.

Clinical feature and Oral findings:

Skin lesions: Erythematous, flat, round macules, papules, or plaques, and the classic skin lesion consists of a central blister or necrosis with concentric rings of variable color around; usually in a symmetrical pattern this patterns have a characteristic target or iris like appearance that known as a (target skin lesions or iris skin lesion).

Oral lesions: present as coalescing small vesicles that rupture within two or three days, leaving irregular, painful erosions, or ulcers covered by a necrotic pseudomembrane and surrounded by extensive areas of inflammation (Sudden development of widespread erosions). The erosions on the lips (especially the lower lip) are accompanied by crusting and bleeding. The most commonly affected sites are lips, buccal mucosa, tongue, soft palate, and floor of the mouth, respectively.

Diagnosis:

1- Lips involvement (strong indication of diagnosis)
2- Presents a target skin lesions or iris skin lesion as a clinical examination.
3- Biopsy: non-specific histological picture

Differential diagnosis:-

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

Management:

- Mild EM : systemic or topical analgesics for pain and supportive care since the disease is self-limiting and resolves within a few weeks
- More severe: Topical steroids or systemic corticosteroids, antibiotics (azathioprine 100–150 mg/d), if considered necessary in severe cases.
- if associated with HSV: should be treated with antiviral medications(acyclovir 400 mg 1x2 or valacyclovir 500 mg 1x2)

☒ Dermatitis herpetiformis

It is an uncommon chronic autoimmune blistering disorder that is often associated with a gluten-sensitive enteropathy (GSE) that affects the skin and rarely oral cavity. The pruritic eruption classically seen on the buttocks and the extensor surfaces of the arms and legs.

Clinical feature and Oral findings:

Skin lesions characterized by grouped excoriations, erythematous, urticarial plaques, and papules with vesicles. Due to the extremely pruritic; the vesicles are often excoriated to erosions by the time of physical examination. The classic location for lesions is on the extensor surfaces of the elbows, knees, buttocks, and back and severe cases involve larger surface areas. But in an unusual case of dermatitis herpetiformis, starting with oral lesions 6 months prior to the onset of skin lesions, is reported

Oral lesions in dermatitis herpetiformis have been rarely described. These may comprise oral mucosal vesicles, blood-filled blisters, irregular ulcers and desquamative gingitivitis. Linear IgA disease may likewise give rise to blood-filled vesicles or bullae, irregular ulcers and desquamative gingivitis.

Diagnosis:-

1- DIF: Autoantibodies (granular IgA), C3 deposits in to the tips of dermal papillae, (A vertically oriented fibrillar staining pattern is noted in a subset of patients, with immune deposits along dermal microfibrils, creating a characteristic "picket fence" pattern of immunofluorescence).
2- IIF : -ve for epithelial autoantibodies.
3- Although most patients are asymptomatic, greater than 90% have an associated gluten-sensitive enteropathy upon endoscopic examination.

Management:

1- Sulfones and sulfapyridines.
2- Patients with celiac disease treated by dapsone and a gluten-free diet.

☒ Linear IgA disease

Is a subepithelial disease characterized by the deposition of IgA in the basement membrane. It affects female more than male and the patients are predominantly in preschool aged and most common in the nonreproductive years. But it's a rare bullous disease of skin when compere with dermatitis herpitiformis and bullous pephigoid.
According to the age there are two types

1- Chronic bullous disease: affected the children
2- Adult linear IgA disease: affected the adults

Skin lesions characterized by pruritic papules and blisters at sites of trauma such as the knees and elbows so its groups known as "rosettes"
Oral lesions appear to be uncommon but are similar to those seen in pemphigoid

Diagnosis:

1- Incisional biopsy: subepithelial bulla
2- DIF: Auto-antibodies (mainly IgA), C3 in a linear distribution at the basement membrane zone.
3- IIF : +ve IgA auto-antibodies in 30% of patients

Management:

Dapsone is a drug of choice

☒ Epidermolysis Bullosa Aquisita (EBA)

Is a heterogeneous autoimmune subepidermal bullous disorder involving the skin and mucous membranes and the aetiology is genetic factor. There are two clinical forms or types:
1st one is an inflammatory type resemble of BP
2nd one is a mechanobullous type: it occur in response to trauma and lesions are therefore most prominent on the knees elbows, hands, and feet.
Oral lesions it vary in severity from mild desquamative gingivitis to severe generalized ulcerative and often scarring mucosal involvement.

Diagnosis:

1- DIF: (+ve IgG and C3 in a linear distribution at the basement membrane zone).
2- IIF: Salt-split skin (circulating IgG antibodies bind to the base of the split corresponding with the target antigen, type 7 collagen)

Management:

The treatment is similar as described for MMP, with the therapy depending upon the extent and severity of the clinical lesions.

☒ Angina Bullosa Haemorrhagica (ABH)

It is a rare acute and benign blood blistering or vesicles oral disorder and the exact aetiology is unknown. However, there are many causes play as predisposing factors such as mild trauma mainly during food ingestion particularly sharp foods or following dental treatment, chronic use of steroid inhalers seem to play an important role in the development of the lesions and Diabetes Mellitus; Both middle-aged and elderly individuals are affected but more common in middle-aged women

Oral lesions: appears as single or multiple hemorrhagic bullae these blisters present a color ranging from dark red to purple that rupture spontaneously within hours or 1–2 days, that lead to some discomfort and pain when their content is spilled over the oral cavity and leaving superficial ulcerations that heal without scarring in 5–10 days. The sites of predilection are soft palate, buccal mucosa, and tongue.

Diagnosis:

1- Mainly based on the history and the clinical presentation.
2- Laboratory tests necessary to rule out any other bullous diseases (Histopathological examination and immunofluorescent tests)

Management:

1- Symptomatic treatment.
2- In patients who use steroid-based inhalers regularly, water gargling following medicine application can be an effective way to prevent ABH.