floor of the mouth should also be inspected carefully. If any growth or ulceration or white lesion is present, it must be thoroughly checked. Openings of Wharton's duct in the floor of mouth and of Stensen's duct in cheek mucosa opposite the upper molars should be checked for normal secretions, inflammation, etc.

b. Hard tissue examination includes inspecting the overall status of the patient's dentition. The teeth should be examined for their total number, presence of any developmental anomalies, hypoplasia, caries, discoloration, wasting diseases such as attrition, abrasion, erosion and abfraction.

9. Examination of the Area of Chief Complaint

The area of chief complaint must be examined in detail. Presence of any soft tissue swelling, pus discharge, bony expansion must be noted. The teeth in the area must be evaluated for dental caries and periodontal disease. The teeth may be gently percussed by tapping the occlusal surface lightly with the blunt end of the probe. Pain elicited on percussion indicates tenderness which may be a sign of periapical disease. The teeth should be checked for abnormal mobility and vitality.

10. Provisional Diagnosis and Investigations

After taking a good history and performing the clinical examination, the clinician must arrive at a provisional diagnosis. Also the possible differential diagnosis should be charted so that appropriate investigations can be advised to the patient. Commonly advised investigations are an IOPA radiograph, occlusal or extraoral radiographs, blood investigations such as CBC, etc. Advanced investigations such as CT, MRI, scintigraphy may be required in certain cases. Tests which are not likely to yield any diagnostic information for that particular case must not be advised to the patient.

11. Final Diagnosis

Once the reports of the investigations are available, the case should be reviewed to correlate the clinical and investigatory findings to arrive at a final diagnosis. When a tumor or cyst is suspected, biopsy may be required to arrive at the final diagnosis.

12. Treatment Plan

Once a case has been diagnosed, the next crucial step is to chalk out a treatment plan for the patient. If the patient is suffering from pain or has signs of infection, antibiotic and anti-inflammatory drugs must be prescribed as an emergency measure. Appropriate treatment must then be planned for the patient after obtaining necessary consultation from the other specialists. While treating patients who are medically compromised, the necessary precautions and treatment modification must be incorporated in the treatment plan.

TERMINOLOGIES USED IN CLINICAL EXAMINATION OF LESIONS

Clinical examination is by far the most important step in the Diagnostic sequence. It is essential to identify various lesions which can be manifested clinically and further analyze and correlate their significance.

Terminologies

- Lesion may be described as a visible change in the normal anatomical structure caused by a pathological process.
- Primary lesion is the first pathological change manifested clinically, e.g. vesicle in herpes simplex.
- Secondary lesion is the altered primary lesion, e.g. ulceration caused by the rupture of the vesicle in herpes simplex.
  Lesions may be flat such as a macule, raised such as vesicle or depressed such as an ulcer.
- Vesicle is a circumscribed elevated serous fluid filled blister not more than 1 cm in diameter with a thin covering of epithelium, e.g. HSV infection, herpes zoster (Fig. 1.1).

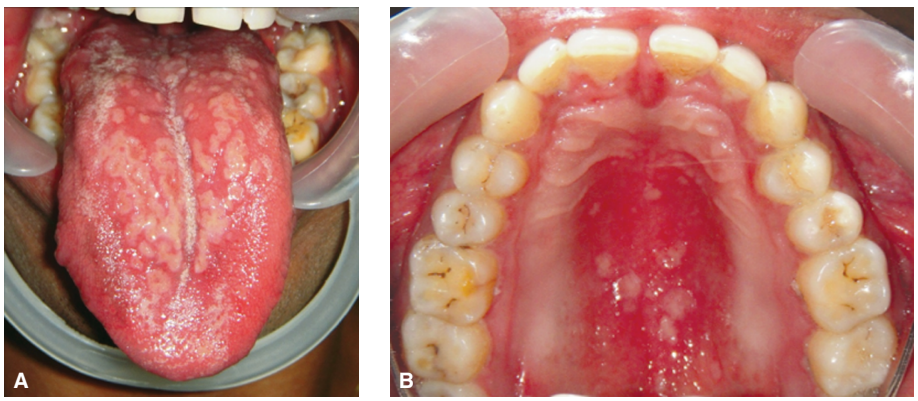

Fig. 2.2: Primary HSV infection—geometric glossitis and palatal lesions

formation and there is always a danger of viral infections spreading rapidly when the immune reaction is suppressed. Currently HSV associated erythema multiforme cases have been reported and in such cases suppression of erythema multiforme can be achieved with the help of acyclovir and steroids will be contraindicated in such cases.

Diagnosis

1. Clinical picture—classically a young patient presenting with H/o fever, malaise multiple vesicles, acute marginal gingivitis and lymphadenopathy gives a clue.
   - Cytologic smear from the base of freshly opened vesicle stained with Giemsa stain (also Wright and Papanicolau's stain) shows multinucleated giant cells or intranuclear inclusion bodies (Lipschultz bodies) with ballooning degeneration of nuclei and syncytium formation. (Syncytium—multinucleated protoplasmic aggregation of cells without apparent cell outlines). Ballooning degeneration is absent in RAS, EM and allergic stomatitis.
2. Fluorescent staining of cytological smear.
3. Conclusive evidence of primary HSV includes testing for complement fixing or neutralizing antibody in acute and convalescent sera—4 fold increase in convalescent serum is diagnostic in primary. HSV isolation can be done on chorioallantoid membrane of chick embryo or kidney of rabbit.
4. Antibody titre is raised. To differentiate between recurrent and primary attack, acute and convalescent serum is collected. In primary attack, only the convalescent serum shows raised titre, as during the active stage antibodies are not formed. In the recurrent attack, both the active and convalescent serums show raised titre, as the defense mechanism has been previously exposed to the virus and produces large number of antibodies.

Differential Diagnosis

Other conditions presenting as acute multiple ulcers are included in the d/d. The ulcers associated with primary HSV are seen in younger patients and are small round symmetrical and shallow with associated marginal gingivitis and H/o prodromal symptoms.

1. ANUG older patients, punched out necrotic ulcers on interdental papilla and marginal gingiva, tender submandibular lymph nodes and H/o fever.
2. Erythema multiforme older patients with allergic background, acute explosive onset with no H/o prodromal symptoms and

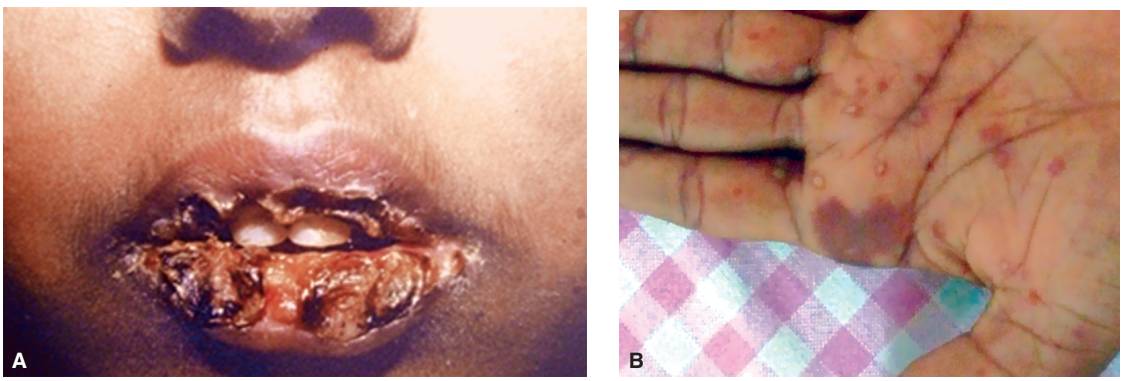

Fig. 2.7: Erythema multiforme—lip lesions and target lesions on the palms

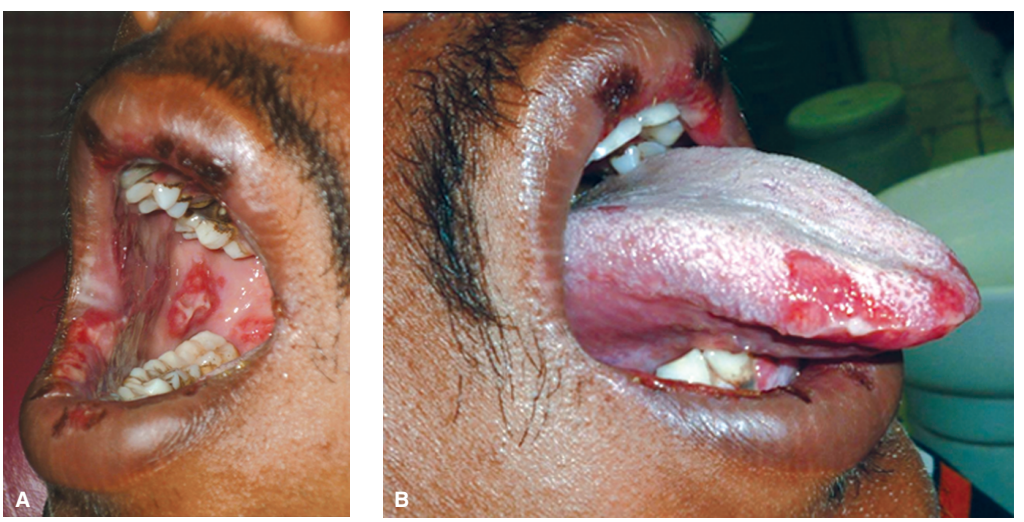

Fig. 2.8: Erythema multiforme showing bloody crustations on lip and ulcerations on tongue

9. Gingiva rarely affected.
10. Stevens-Johnson syndrome [SJS]—Severe form of EM affecting skin, oral mucous membrane, eyes (keratoconjunctivitis, corneal ulcerations) (Fig. 2.9), genitalia (balanitis, urethritis, vaginitis). In the severe form, Nikolsky's sign may be positive. This condition, if untreated, may lead to infection, electrolyte imbalance and death.
11. TEN is the most severe form of EM, secondary to drug reaction, it results in sloughing of skin and mucosa in large sheets. Death may be due to secondary infection; fluid, electrolyte imbalance or involvement of lungs, liver or kidneys. Patients are best managed in burn centers where necrotic skin is removed and healing takes place under sheets of porcine xenografts.
12. *Recent concept*: SJS is less severe form of TEN and both are separate from EM. Skin lesions of SJS and TEN are more severe and arise on chest, called atypical targets (erythematous, purpuric macules). SJS associated with drug allergy and mycoplasma and EM with HSV infection.

Diagnosis is based on—history, clinical characteristics, histopathology and biopsy of intact bulla.

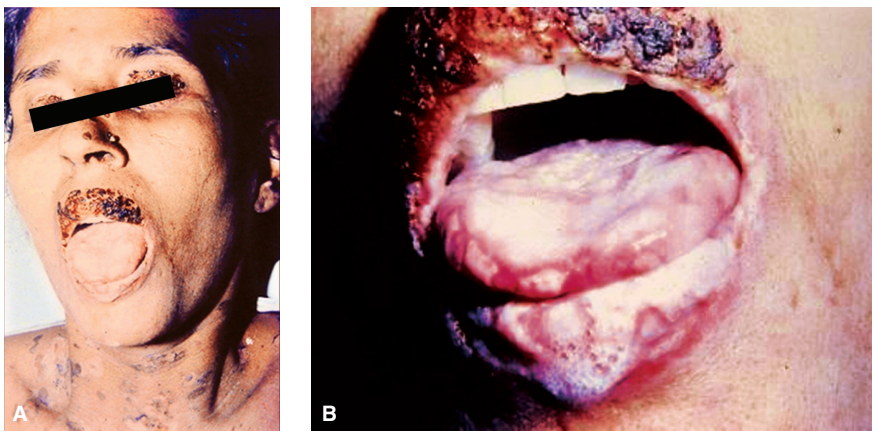

Fig. 2.9: Stevens-Johnson syndrome

Histopathology: Shows intraepithelial lesions which may form subepithelial lesions, liquefactive degeneration of upper layers of epithelium, thinning or absence of basement membrane and inflammation of coreum. It is not specific.

D/D

1. *Allergic stomatitis*: Difficult to differentiate, erythema, vesiculations, ulcerations with a positive history of allergy helps, however, both these conditions are similar pathologically and clinically.
2. *HSV*: Primary HSV with prodromal symptoms, small round symmetrical shallow ulcers as opposed to EM with acute explosive onset and large irregular deep bleeding ulcers. HSV associated with marginal gingivitis, lymphadenopathy which is not seen in EM.
3. *Herpes zoster*: Unilateral distribution is pathognomonic of HZ with the lesions abruptly stopping in the midline, severe pain, EM bloody crustations on both the sides of the lip.
4. *Herpangina*: Lymphadenopathy, lesions affecting posterior part of oral cavity.
5. *Pemphigus vulgaris*: Patient gives history of chronic lesions (H/o 2–3 months), EM is of acute duration, i.e. very short duration history of few days.

Treatment

1. In severe cases, it is best to hospitalize the patient to maintain electrolyte balance and to prevent secondary infection. Drug of choice is corticosteroids (prednisonolone—30–50 mg, metamethasone or dexamethasone—2–5 mg more potent) which is given in tapering dose.
2. Topical steroids in orabase—Betamethasone with neomycin, fluocinolone N, triamcinolone acetonide.
3. Cases suspected to be HSV associated are treated with 400 mg acyclovir bid which prevents the development of EM.
4. For non-HSV related EM—Azathioprine (100–150 mg/day), Dapsone (100–150 mg/day), antimalarials are partially successful in preventing recurrent outbreaks.
5. Local anesthetic mouthwashes prior to meals as lesions are painful.
6. Local as well as systemic effect by use of 0.5 mg tab Betamethasone (tab Betnesol) – crush the tablet + water and hold in mouth for 5–10 mins swish and swallow. This regimen lasting 20 days includes giving

   Betamethasone 0.5 mg 1 tab 4 times a day for 5 days
   3 times a day for next 5 days
   2 times a day for next 5 days
   Once a day for next 5 days

It can be idiopathic or after contact with the allergen. It can also be classified as—hereditary, non-hereditary, or allergic.

Angioedema of larynx and posterior tongue causes asphyxia which can be life-threatening. There is respiratory distress.

Generalized anaphylaxis:

- Allergic emergency
- No time to call a consultant
- *Mechanism*—reaction of IgE antibodies with an allergen to cause release of histamine, bradykinin and SRSA. These chemical mediators cause contraction of smooth muscles of respiratory and intestinal tract as well as increased vascular permeability.
- *Factors for increased risk of anaphylaxis*:
  1. History of allergy to food and drug
  2. History of asthma
  3. Family history of allergy
  4. Parenteral administration of drug
  5. High-risk drug like penicillin

Generalized anaphylaxis involves 4 systems:

1. CVS
2. GIT
3. Respiratory system
4. Skin

First signs seen on the skin are—localized anaphylaxis, erythema, angioedema, urticaria, pruritus.

Pulmonary—dyspnoea, wheezing, asthma.

Gastrointestinal—vomitting, cramps, diarrhoea.

If these are untreated, there is hypotension due to loss of intravascular fluid. If untreated, shock occurs. Patients with generalized anaphylactic reaction may die from respiratory failure, hypotensive shock or laryngeal edema. Most important treatment is administration of epinephrine—aqueous epinephrine 1:1000 in sterile easily accessible syringe 0.5 ml im or sc. Epinephrine will usually reverse all severe signs of generalized anaphylaxis. If no improvement in 10 mins re-administer epinephrine.

1. *For bronchospasm*: Slow iv aminophylline 250 mg over 10 mins. If given too rapidly, it can lead to fatal cardiac arrhythmias. Inhalation of sympathomimetics. Oxygen to prevent or manage hypoxia.
2. *For laryngeal edema*: Establish airway by endotracheal intubation. Cricothyroidotomy may be necessary.

Ulcers Secondary to Chemotherapy

Cancer chemotherapeutic drugs are classified into 4 groups:

1. *Alkylating agents*: Cyclophosphamide, Chlorambucil
2. *Alkaloids*: Vincristine, Vinblastine
3. *Antibiotics*: Adriamycin, Actinomycin D, Bleomycin
4. *Antimetabolites*: 6 Mercaptopurine, Methotrexate

These are either used singly or in combination. As these are potent drugs expected to kill the cancer cells, some damage to healthy cells also is unavoidable. One of the side effects is multiple oral ulcers. Mechanism of ulcer formation:

1. *Indirect*: Depression of bone marrow and immune response caused, e.g. by vinblastine leading to invasive infection of the oral mucosa which can be bacterial, viral or fungal.
2. *Direct*: Methotrexate causes oral ulcers by direct effect on replication and growth of epithelial cells by interfering with nucleic acids and protein synthesis leading to thinning and ulceration of oral mucosa.

Clinical Features

1. History of chemotherapy.
2. Deep, large, necrotic ulcers without erythematous halo and without tissue tags.

Differential Diagnosis

1. *HSV*: H/o chemotherapy is absent, ulcers are small, shallow round and show tissue tags, erythematous halo.
2. *EM*: No H/o chemotherapy, bleeding ulcers involving lips (bloody crustation), tissue tags present, skin lesions present.

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