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Clinical Medicine for Dental Students



New Features

- As per ***DCI*** syllabus
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- System-based assessment for developing clinical skills
- **Flowcharts and illustrations** for understanding the complicated concepts
- **Oral manifestations** of medical conditions
- **Special boxes**, i.e., candle box and stethoscope box carrying clinically relevant information
- **Latest definitions and guidelines**
- Updated epidemiological data
- **Sample assessment charts.**

R Alagappan

4th Edition

JAYPEE

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CHAPTER 3

Infectious Diseases

History

Age group

Demographic details

Occupational exposure

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General systemic enquiry

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Recent dietary intake

Sexual habits

Previous treatment (e.g., blood transfusion)

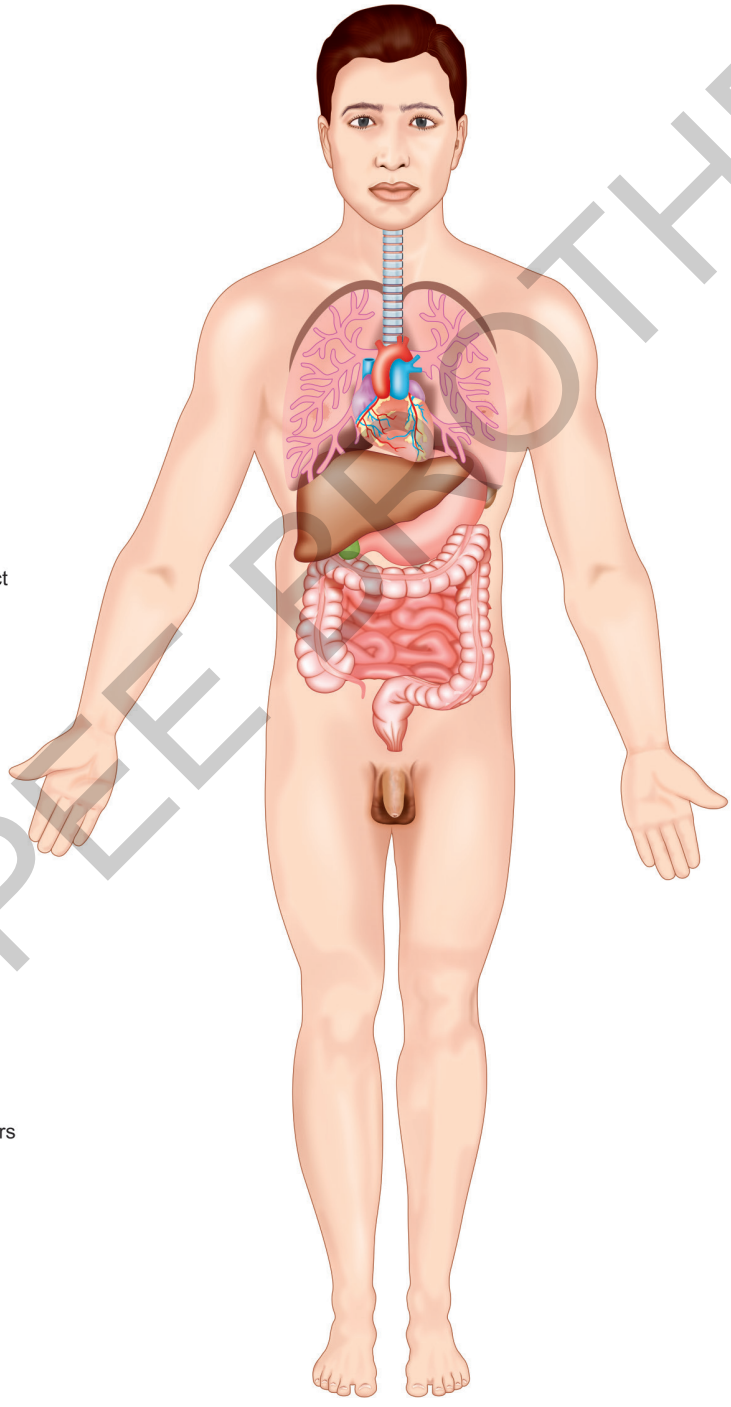
Addictions

Comorbid illness and predisposing conditions

Similar illness in family members

Immunisation details

Menstrual history (in reproductive age group)



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Nervous system

Altered sensorium
Nuchal rigidity
Focal deficits

Skin, soft tissue and skeletal examination

Appropriate investigations and specific treatment

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VIRAL EXANTHEMS

INTRODUCTION

Viral infections producing maculopapular skin eruptions are called viral exanthems. The lesions that occur in the mucous membrane are called enanthems. Virus-induced maculopapular exanthems (not vesicular) spare the palms and soles. In drug reactions and non-viral (bacterial, mycoplasma, rickettsial) exanthems, the palms and soles are involved. Maculopapular exanthems are caused by both viral and non-viral diseases. Viral causes include infectious mononucleosis, measles, rubella, varicella-zoster virus (VZV), adenovirus, reovirus, arbovirus while non-viral causes include syphilis, typhoid fever, rickettsia, mycoplasma, drug eruption.

MEASLES (RUBEOLA)

Measles is an acute febrile illness with maculopapular eruption caused by ribonucleic acid (RNA) paramyxovirus. It is a highly contagious disease. The rate of secondary infection is high (90%).

PATHOGENESIS

- It involves the respiratory mucous membrane or conjunctivae and is transmitted either directly or by airborne droplet. Infectivity is greatest in first 3 days before the onset of rash.
- Measles virus basically targets epithelial, reticuloendothelial and white blood cells. It also declines the CD-4 count resulting in suppression of delayed-type hypersensitivity.
- The patient gets symptomatic after 5 days of exposure till 5 days after the appearance of skin lesions.
- Children between the ages of 5 years and 15 years are commonly affected.
- The incubation period is 10 days.

CLINICAL FEATURES

Catarrhal Stage (Prodromal Symptoms)

Initial symptoms are similar to common respiratory illness such as fever, malaise, irritability, conjunctivitis with photophobia, running nose and cough. The stage lasts for 3–4 days.

Exanthematous Stage

On the 4th day, maculopapular rash appears over the back of the ears, forehead and then spreads downwards over the face, neck, trunk and limbs, where they frequently get confluent. The maculopapular rash fades and leaves behind brown discolouration of the skin following which desquamation of maculopapular rash occurs. The total duration of the rash is 6 days **(Fig. 3.1).**

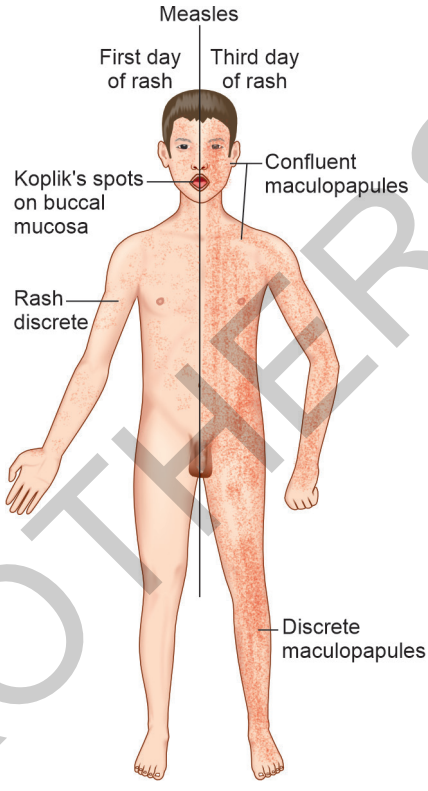

Fig. 3.1: Clinical features of measles.

Koplik's Spots

These spots are small, red and irregular lesions with bluish-white centres which appear on the mucous membrane of the mouth and occasionally on the conjunctivae, or intestinal mucosa. They are best seen at the opening of parotid duct (upper second molar teeth) **(Fig. 3.2).**

COMPLICATIONS

It is a benign self-limiting disease but as it targets multiple organs, complications may occur. These are given in **Table 3.1.**

- **Pregnancy:** It is not known to cause congenital abnormalities to the foetus. It may induce abortion or premature delivery and may cause severe disease

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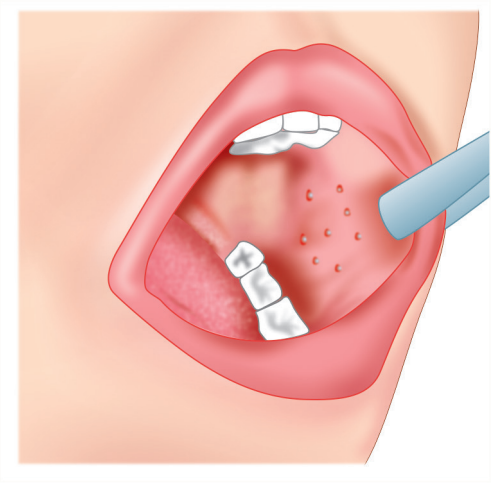

Fig. 3.2: Koplik's spots on buccal mucosa.

Table 3.1: Complications of measles.

Respiratory system	Bronchitis, bronchiolitis, pneumonia
Eyes	Conjunctivitis, corneal ulceration, keratitis, blindness
Gastrointestinal system	Diarrhoea, stomatitis noma, involvement of mesenteric lymph node, appendix with peritonitis
Nervous system	Postviral encephalitis, subacute sclerosing panencephalitis (SSPE), Raye's syndrome, convulsions, rarely transverse or ascending myelitis
Haematologic	Thrombocytopaenic purpura, disseminated intravascular coagulation
Cardiovascular	Myocarditis, pericarditis
Other complications	Otitis media, myocarditis, glomerulonephritis, death of foetus in 20% of pregnancy, exacerbation of existing tuberculosis, thrombocytopaenia, kwashiorkor and marasmus

Sub-acute sclerosing panencephalitis (SSPE) is a very late CNS complication 5–15 years after the infection. The measles virus acts as a slow virus to produce CNS degenerative disease after many years. The clinical manifestations are—personality changes, developmental retardation, dementia, myoclonic jerks, cerebellar ataxia and various degree of motor disabilities. Measles virus has been isolated from the brain tissue.

in mother. Infants can be passively immunised with immunoglobulin at birth.

- **Vitamin A:** There is inverse correlation between serum retinol concentration and measles severity. Measles is a serious disease in adults, malnourished, vitamin deficient, and immunocompromised individuals. Give vitamin A single dose 1 lac IU orally for children <1 year and 2 lac IU orally for children >1 year.

INVESTIGATIONS

- **Complete blood count:** There is leukopenia in early phase. Leucocytosis, if present, denotes secondary bacterial infection. Lymphopaenia less than 2,000/μL indicates poor prognosis.
- **Cell cultures:** Multinucleated giant cells in the stained smears obtained from sputum or nasal secretions show measles virus.
- **Demonstration of viral antigen:** It is done in the stained respiratory or urinary epithelium cells by fluorescent antibody test.
- **Enzyme-linked immunosorbent assay (ELISA):** Rising specific antibody levels are demonstrated by ELISA (by immunofluorescent technique) and complement fixation tests.

MANAGEMENT

The treatment is supportive and symptomatic. Antibiotics may be given in case of secondary bacterial infections. Immunoglobulins may be given prophylactically to prevent the disease, if administered within 6 days of exposure.

PREVENTION

Active Immunisation

Live-attenuated measles vaccine along with mumps and rubella vaccine (MMR) in a dose of 0.5 mL is given subcutaneously at the age of 9 months. Two dose schedule is followed for MMR vaccine and second dose is given at the age of 4–6 years. MMR vaccine is contraindicated in pregnant women, patients with leukemia, lymphoma, children with untreated tuberculosis and immunocompromised patients.

Passive Immunisation

Human normal immunoglobulin at a dose of 250 mg (children <1 year) and 500 mg (children >1 year) is given intramuscularly. Passive immunisation is given to contacts under 18 months of age and to those in whom, vaccine is contraindicated. It should be given within 6 days of exposure.

RUBELLA

Rubella is also known as German measles or 3-day measles. It is an infectious benign febrile exanthem caused by RNA—toga virus.

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PATHOGENESIS

Rubella spreads by respiratory droplets. After replication in respiratory tract it spreads via the bloodstream. Viraemia is present 8 days before and 2 days after the appearance of rash. The incubation period is 14–21 days. The congenital malformations that can develop at various stages of gestation after the infection are listed in **Table 3.2.**

CLINICAL FEATURES

Prodromal Stage

It lasts for 1–7 days. Symptoms include headache, malaise, fever, conjunctivitis and lymphadenopathy (suboccipital).

Infective Period

Patient remains infective 1 week prior and 1 week after the onset of rashes.

Exanthematous Stage

This stage is characterised by pink macular rash which first appears on the forehead and face and then passes downwards to the trunk and limbs. The maculopapular rash is of lighter hue than that of measles and disappears in 3 days. There may be enlarged, tender lymph nodes, especially posterior cervical group with splenomegaly.



Forchheimer Spots

These are petechial lesions which appear in the soft palate. They may be associated with mild coryza and conjunctivitis.

COMPLICATIONS

- Congenital rubella syndrome (CRS)
- Post-infectious encephalitis
- Spontaneous abortion
- Thrombocytopaenia

Table 3.2: Rubella—risk of congenital malformation.

Stage of gestation	*Incidence in %*	*Type of malformations*
4–8 weeks	65–85	Multiple defects, spontaneous abortion
12 weeks	30–35	Single defect—deafness or congenital heart disease
16 weeks	10	Deafness or congenital defects
>20 weeks		Occasional deafness

CONGENITAL RUBELLA SYNDROME

It results from transplacental transmission of the virus. The virus can persist in the foetus during intrauterine life and up to 1 month after birth. There is high risk of congenital malformations in the developing foetus.



Congenital Malformations in CRS

- ❑ **Heart:** Patent ductus arteriosus, ventricular septal defect, pulmonary stenosis and myocarditis
- ❑ **Eyes:** Corneal clouding, cataract, chorioretinitis, microphthalmia, glaucoma
- ❑ **Nervous system:** Mental retardation, deafness
- ❑ **Others complications:** Microcephaly, skeletal abnormalities, intrauterine growth retardation, arthritis, orchitis, thrombocytopaenia, encephalomyelitis

Immune-mediated arthritis involves fingers, wrist and knees in 30% of patients. Later complications can be diabetes mellitus and panencephalitis. The risk is more when the maternal infection occurs in the first trimester of pregnancy, especially initial 3–6 weeks.

Diagnosis

The World Health Organization (WHO) diagnostic classification of CRS.

- **Suspected congenital rubella syndrome:** Infant less than 11 months presenting with heart diseases, suspicion of deafness with eye symptoms or positive history of mother.
- **Clinically-confirmed CRS:** Infants less than 1 year with two or more complications.
  - Cataract, congenital glaucoma, congenital heart disease, loss of hearing.
  - Purpura, splenomegaly, jaundice within 24 hours
- **Laboratory confirmed CRS:** Infant more than 1 year with positive blood test for rubella-specific immunoglobulin M (IgM).

Treatment

- No specific treatment, only symptomatic
- Consider termination of pregnancy to prevent CRS.

Prevention

Measles, mumps and rubella-vaccine as for measles confers lifelong immunity.

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MUMPS

Mumps is an acute infectious disease caused by single-stranded RNA paramyxovirus. It is a systemic disease that causes painful enlargement of salivary glands, especially parotids. It might involve meninges, pancreas, gonads, lacrimal glands, thymus, thyroid gland, breasts, liver and kidneys.

PATHOGENESIS

Human beings are the only reservoir of infection. Transmission of infected droplets occurs via the upper respiratory tract and cervical lymph nodes where the virus replicates. It is then disseminated through the bloodstream to the target organs like parotids and meninges. Single attack confers lifelong immunity. The incubation period varies between 12 days and 25 days.

CLINICAL FEATURES

- Anorexia, sore throat, malaise and fever with painful enlargement of one or both parotids occur in mumps. The angle of the jaw may be obliterated.
- Parotid enlargement is unilateral in 25% of cases and bilateral in the remaining 75% of cases. Submaxillary and sublingual glands are involved in 10% of cases. The orifice of Stensen duct is red and swollen. Trismus may occur.
- Main complaints are difficulty in eating, swallowing and talking. Saliva is infectious 7 days prior and after parotitis.

Epididymo-orchitis

- It occurs in 25% of postpubertal males during the 2nd week.
- Along with high fever there is unilateral or bilateral testicular swellings with pain and tenderness. It subsides after 1 week. Atrophy of testicle occurs in 50% of cases. Sterility is unusual but it can occur in bilateral testicular atrophy. Rarely painful priapism may occur in orchitis.

Oophoritis

It presents with lower abdominal pain and fever in postpubertal females.

Pancreatitis

It presents as upper abdominal distention along with fever, nausea, vomiting, pain and elevated level of serum amylase. Rarely, there may be brittle diabetes following pancreatitis.

Neurological Symptoms

- About 10% of cases develop meningitis with stiff neck and headache. The cerebrospinal fluid (CSF) reveals a lymphocytic pleocytosis or less commonly neutrophils.
- Encephalitis can occur with seizures, headache and unconsciousness.
- Rarely, transverse myelitis, motor polyneuritis or cerebellar ataxia can occur.

DIFFERENTIAL DIAGNOSIS

Parotitis (pyogenic), salivary duct calculus and sarcoidosis.



Other Rare Complications

Myocarditis, nephritis, hepatitis, thyroiditis, deafness, etc.

INVESTIGATIONS

Diagnosis of mumps is mainly based on clinical grounds.

- Isolation of virus from saliva, blood, throat swab and CSF.
- Detection of viral antigen directly from oropharyngeal cells with the help of immunofluorescence.
- Demonstration of specific antibodies (ELISA) and rise in titres is diagnostic.
- There may be mild leucopenia with relative lymphocytosis.
- Serum amylase and lipase level is elevated in pancreatitis.

MANAGEMENT

The treatment is entirely symptomatic.

- Analgesics are given and local fomentation is done to relieve symptoms.
- Arthritis: Ibuprofen and prednisolone may be given.
- Prednisolone can also be given to relieve discomfort of arthritis (60 mg/day for 7 days).
- **Orchitis:** Support the testes with suspensory bandage. NSAIDs are useful to relieve pain and if pain is not relieved, inject 10 mL of 1% procaine solution into the spermatic cord at the site of external inguinal ring. Hydrocortisone sodium succinate 100 mg IV followed by 20 mg qid PO for 3 days. Interferon alfa 2b may be useful in preventing testicular atrophy.

PREVENTION

Live-attenuated mumps virus vaccine can be given to children at the age of 15 months alongside MMR vaccines.

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It confers lifelong immunity. It is also advised for human immunodeficiency virus (HIV) infected patients.

Viral shedding is short lived and patient should be isolated for first 5 days following swelling.

HERPES ZOSTER (SHINGLES)

VARICELLA ZOSTER INFECTIONS

Varicella zoster virus (VZV) is HHV-3. Disease manifestations include chickenpox (varicella) and shingles (herpes zoster). Chickenpox is a disease of childhood. Humans are the only reservoir of VZV. Transmission occurs by the respiratory route and subsequent replication of the virus presumably in the nasopharynx leads to seedling of the reticuloendothelial system and viraemia.

The VZV infects dorsal root ganglia during chickenpox, where it remains latent until reactivated.

Herpes zoster (shingles): It results from reactivation of latent VZV from dorsal root ganglia. It is a unilateral vesicular eruption confined to a single dermatome. Multiple dermatomes are involved in immunocompromised persons.

PATHOGENESIS

Herpes zoster occurs as a result of reactivation of latent virus from dorsal root ganglia following chickenpox in early life. Reactivation may be spontaneous effecting middle age or elderly or due to immunosuppression as in HIV, Hodgkin's disease, non-Hodgkin lymphoma and other malignancies.

CLINICAL FEATURES (FIG. 3.3)

- Burning discomfort or pain are the first most common manifestation in the affected dermatome. Dermatomes from T3 to L3 are more frequently affected.
- Unilateral vesicular lesions appear within 3–4 days. Pain during acute phase due to neuritis persists as long as vesical remain.
- Involvement of ophthalmic division of the trigeminal nerve may lead to corneal ulceration. The condition may lead to blindness if appropriate treatment is not taken.
- Geniculate ganglion involvement causes Ramsay hunt syndrome characterised by vesicles in the auricle, pallate along with facial palsy and impairment of taste.



Fig. 3.3: Herpes zoster.

- Disseminated and multidermatomal involvement occurs in leukaemia, lymphoma, HIV and immunocompromised individuals.
- Appearance of only prodromal symptoms without characteristic rashes is referred to as "zoster sine herpete".

Postherpetic Neuralgia

Patients over the age of 50 years generally suffer from pain which may persists for many months. Sensory changes either hypo- or hyperaesthesia are common.

DIAGNOSIS

Clinical diagnosis should be made by visual examination as few diseases follow a dermatome pattern.

INVESTIGATIONS

- Smear studies, the Tzanck smear diagnoses acute infection with the herpes virus, but does not distinguish between herpes simplex virus (HSV) and varicella zoster virus
- Fluorescent-antibody-to-membrane-antigen test
- ELISA test, qualitative polymerase chain reaction (PCR).

COMPLICATIONS

Meningoencephalitis, pneumonitis, hepatitis, transverse myelitis, motor paralysis of involved segment may occur rarely.

MANAGEMENT

Topical Therapy

Topical idoxuridine 5% lotion or acyclovir for local relief is given.

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Systemic Therapy

Antivirals

Oral acyclovir 800 mg 5 times daily or valacyclovir 1,000 mg TDS for 1 week are given in early stage of disease. In cases of severe infection or immunocompromised patients 5–10 mg IV infusion per kg 8 hourly for 7–10 days is administered.

Analgesics

Pain is managed with analgesics. Nonsteroidal anti-inflammatory drugs (NSAIDs) are used in postherpetic neuralgia as follows:

- Amitriptyline 25–100 mg daily
- Diphenylhydantoin 200–600 mg/day
- Carbamazepine 200–600 mg/day
- Gabapentin 300 mg OD or BID

Corticosteroids

- A short course of glucocorticoids like prednisolone in elderly in the early phase may prevent postherpetic neuralgia.
- **Vaccine:** There are two vaccines—older live attenuated VZV vaccine 'Zostavax' and the new 'Varivax'. Two doses of vaccine given two months apart had 97% efficacy in preventing herpes zoster. It is recommended for persons above 60 years. The zoster vaccine cannot be used in children. It can be given even if the person had a prior episode of herpes zoster.

HERPES SIMPLEX

Herpes simplex is a viral disease caused by HSV (herpes virus hominis). HSV is a deoxyribonucleic acid (DNA) virus and there are two strains of HSV (HSV-1 and HSV-2). ***HSV-1*** infection occurs in childhood and 90% of adults have antibodies to HSV-1. It involves mucocutaneous surfaces of head and neck. ***HSV-2*** causes genital lesions and are transmitted by sexual contact.

PATHOGENESIS

- Herpes simplex virus enters through the mucosal surfaces or abraded skin and the initial replication takes place in cells of epidermis and dermis **(Fig. 3.4)**.
- Then, it infects sensory or autonomic nerve endings.
- Virus is transported intra-axonally to the nerve cells in ganglia.
- After replication, it spreads to mucosal skin surfaces through centrifugal migration.
- It remains dormant in the skin and nerve ganglia and is reactivated by fever, physical or mental stress, sunlight,

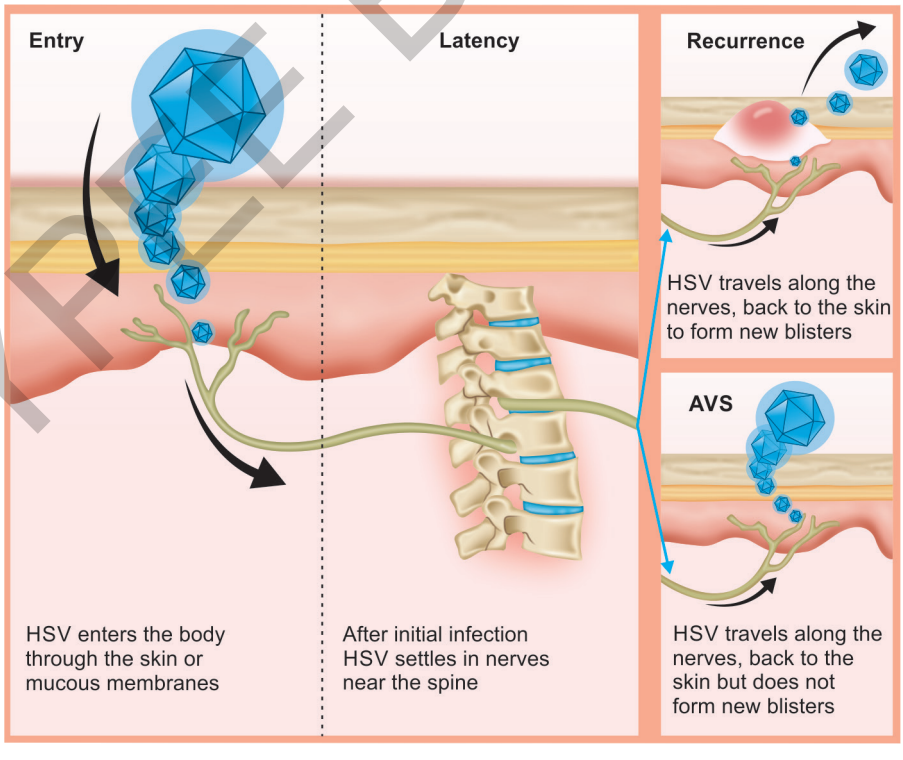

Fig. 3.4: Pathway of a herpes infection.
(HSV: herpes simplex virus; AVS: asymptomatic viral shedding)

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immunosuppression, trauma to skin producing recurrent form of disease called herpes labialis (cold sore).

CLINICAL FEATURES

- The incubation period is 7–21 days.
- Primary infection with HSV-1 or 2 is more likely to be symptomatic later in life.
- A crop of vesicles appears with tingling pain and paraesthesia and healing starts after a week.
- Healing is complete by the fall of crusts by 3 weeks.
- Recurrent attacks occur throughout life, generally in association with concomitant medical illness, menstruation, immunosuppression, etc.

TYPES OF HERPES SIMPLEX

Gingivostomatitis and Pharyngitis

It is the most common clinical manifestation of first episode HSV-1 infection. Typically lesions are found in hard palate, soft palate, gingiva, tongue, lip and facial area.

Genital Herpes

Genital herpes can be caused by both HSV-1 and HSV-2 viruses. Recurrence is more common with HSV-2 than HSV-1 infection. Lesions occur over the external genitalia with vaginal and urethral discharge. Cervicitis, endometritis, salpingitis and prostatitis can occur.

Rectal and Perianal

Herpes generally affects distal 10 cm of rectal mucosa and perianal region.

Herpetic Whitlow

It is a complication of primary oral or genital herpes. An inoculation lesion on the finger gives rise to a paronychia which is termed as "whitlow". It is commonly seen in contacts of patients with herpetic lesions. It was earlier seen in dentists and healthcare workers but now use of protective gloves has led to decreased incidence.

Herpes Gladiatorum (Traumatic Herpes)

It refers to lesions that occur in any area of the skin due to trauma. This condition is generally seen in wrestlers—thorax, ears, face and hands.

Herpetic Keratitis

It is characterised by pain, chemosis, conjunctivitis and dendritic corneal ulcer which may lead to blindness.

Herpes Encephalitis

Meninges and brain can be invaded either as a primary infection or due to reactivation.

Eczema Herpeticum

Herpetic lesions occurring on eczematous skin are termed as eczema herpeticum.

Visceral Infections

These types of disseminated lesions are common in immunocompromised individuals. During viraemia, multiple organs might get involved.

Neonatal Herpes Simplex Virus Infection

It occurs in neonates less than 6 weeks of age. HSV-2 infection is more common than HSV-1. The neonate contracts the infection via genital secretions during delivery or contact with herpes lesions. Systemic infection is common with central nervous system (CNS) involvement. Mortality is high if not treated early.

DIFFERENTIAL DIAGNOSIS

- *Candida balanitis*
- Chancroid
- Multiple syphilis chancres

INVESTIGATIONS

Infection is confirmed by:

- Rising antibody titres—confirmatory test
- Direct immunofluorescence or viral culture
- Polymerase chain reaction (PCR) techniques

TREATMENT OF HERPES SIMPLEX VIRUS INFECTIONS

The acyclic antivirals are the treatment of choice for HSV infection.

- **Primary HSV:** Famciclovir 250 mg 8 hourly or valacyclovir 500 mg 12 hourly or acyclovir 200 mg 5 times daily for 10–14 days.
- **Severe form:** In patients with severe form of HSV where oral intake of medicine is difficult, acyclovir 5–10 mg IV 8 hourly is administered.
- **Recurrent episode (HSV-1 and HSV-2):** Oral acyclovir 200 mg 6 hourly along with acyclovir ointment topical application 3–5 times daily or famciclovir 250 mg 12 hourly or valacyclovir 500 mg daily for 5–7 days.

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- **Immunocompromised individuals:** Acyclovir 400 mg 6 hourly or famciclovir 500 mg 12 hourly or valacyclovir 1,000 mg 12 hourly for 14 days. Acyclovir-resistant HSV cases are treated with IV foscarnet 40 mg/kg 8 hourly for 14 days.
- **Genital HSV:** Treatment is similar as for primary HSV and recurrent episode; the duration of therapy is limited to 5–7 days.
- **Central nervous system HSV/severe complications:** Acyclovir IV 10–20 mg/kg 8 hourly for 10 days.
- **Visceral HSV infections (oesophagitis/pneumonitis):** Acyclovir 15 mg/kg/day IV for 10 days. In milder cases oral therapy with valacyclovir or famciclovir is effective.
- **Bell's palsy:** Valacyclovir 1 g od/day for 5 days PO (but not acyclovir) along with prednisolone 25 mg bid PO for 10 days is beneficial if started within 7 days of onset.
- **Acyclovir resistant HSV infections:** Foscarnet 40 mg/kg tid IV/day should be given until lesions heal.
- **Newer drugs under study:** Amprenavir, fosamprenavir and pritelivir
- **Neonatal HSV:** Oral acyclovir 60 mg/kg/day in three divided doses for 21 days or IV acyclovir.
- **Paediatric HSV:**
  - **3 months to 11 years:** Initial episode, 10–20 mg/kg orally 4 times a day for 7–10 days. American Academy of Pediatric recommends 40–80 mg/kg orally per day in 3–4 divided doses for 5–10 days. Maximum dose 1 g.
  - **12 years or older, over 40 kg:** Initial episode, severe initial episode, and recurrent episode: Adult dose.

PREVENTION

In caretakers of the patients suppressive antiviral chemotherapy is administered as acyclovir 400 mg BID or 800 mg OD for 5–10 days. Famciclovir 250 mg 12 hourly or valacyclovir 500 mg daily for 5–10 days can also be used for disease prevention or suppression.

Prophylaxis

Prophylactically suppressive antiviral therapy is provided to the caretaker to prevent the spread of the diseases.

IMPORTANT QUESTIONS

1. Discuss in detail pathogenesis, clinical features, management and prevention of measles.
2. Discuss in detail pathogenesis, clinical features, management and prevention of rubella.
3. Discuss in detail pathogenesis, clinical features, management and prevention of mumps.
4. Discuss in detail pathogenesis, clinical features, management and prevention of herpes zoster.
5. Discuss in detail pathogenesis, clinical features, management and prevention of herpes simplex virus.

HIV AND AIDS

INTRODUCTION

HIV can be elaborated as human immunodeficiency virus that causes slow and constant damage of the immunity consequently taking over the entire immune system. Acquired immunodeficiency syndrome (AIDS) is a clinical condition that is manifested due to severe HIV infection that results in low CD4 count. It makes the body susceptible to opportunistic infections due to low immunity. There are two types of HIV virus: HIV-1 and HIV-2.

Definition According to World Health Organisation

Human Immunodeficiency Virus

- **Adults and children 18 months or older**
  - Human immunodeficiency virus infection is diagnosed based on: positive HIV antibody testing (rapid or laboratory-based enzyme immunoassay). This is confirmed by a second HIV antibody test (rapid or laboratory-based enzyme immunoassay) relying on different antigens or of different operating characteristics; and/or positive virological test for

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HIV or its components [HIV-ribonucleic acid (RNA) or HIV-deoxyribonucleic acid (DNA) or ultrasensitive HIV p24 antigen] confirmed by a second virological test obtained from a separate determination.

- **Children younger than 18 months**
  - Human immunodeficiency virus infection is diagnosed based on: positive virological test for HIV or its components (HIV-RNA or HIV-DNA or ultrasensitive HIV p24 antigen) confirmed by a second virological test obtained from a separate determination taken more than 4 weeks after birth.

Positive HIV antibody testing is not recommended for definitive or confirmatory diagnosis of HIV infection in children until 18 months of age.

Acquired Immunodeficiency Syndrome

Acquired immunodeficiency syndrome is a term which applies to the most advanced stages of HIV infection. It is defined by the occurrence of any of more than 20 opportunistic infections or HIV-related cancers.

PATHOGENESIS

The hallmark of HIV infection is the reverse transcription of genomic RNA to DNA by the enzyme called reverse transcriptase. HIV binds to CD4 receptors on helper T-lymphocytes and monocytes **(Fig. 3.5).** The double-stranded DNA gets integrated to the host cell chromosomes through the action of an enzyme—integrase. The proviral DNA has the ability to transcribe new viral RNA copies and hence the name—retrovirus. After a long latent period of 5–10 years CD4 cell counts begin to decline and the acquired deficiency increases the risk of many opportunistic infections and tumours.

MODES OF TRANSMISSION

Human immunodeficiency virus is transmitted by:

- Sexual intercourse:
  - Vaginal
  - Anal
  - Homosexual
  - Heterosexual
- Infected blood and blood products, i.e., by needle-stick injury or blood transfusion
- By infected mother to infant intrapartum, perinatally, or via the breast milk.
- Multiple injections.
- Mosquito bite does not transmit HIV.

Saliva, tears, sweat and urine are usually not infective.

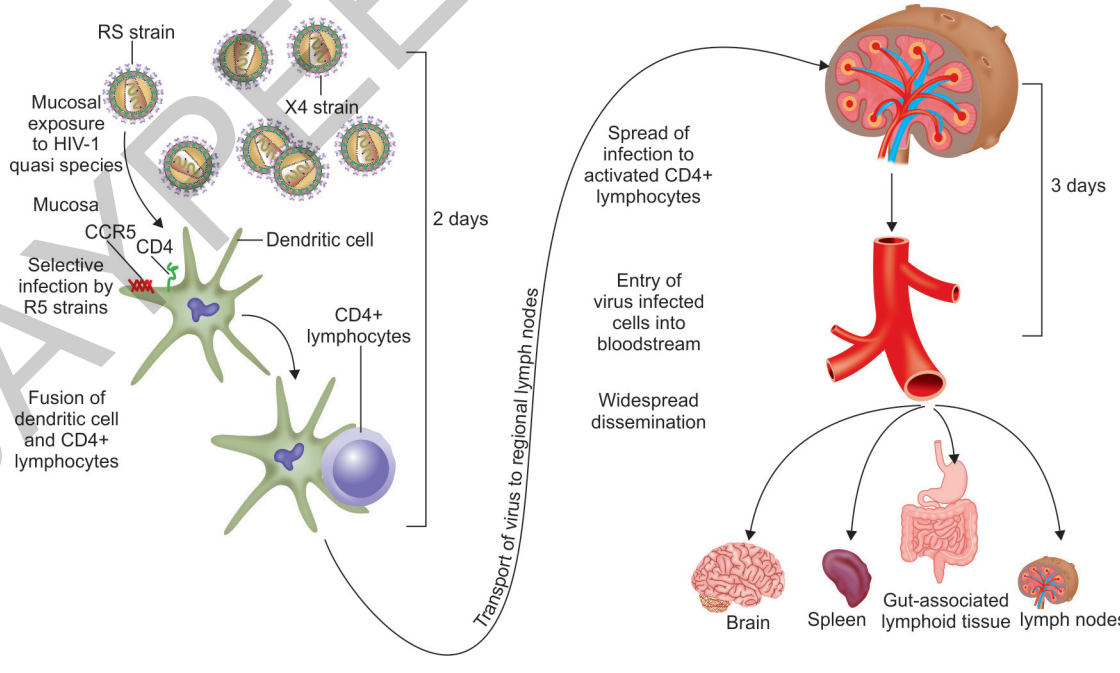

Fig. 3.5: Pathogenesis and immunopathogenesis of human immunodeficiency virus (HIV).

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CLINICAL STAGING

World Health Organisation (WHO) clinical staging of HIV/AIDS for adults and adolescents with confirmed HIV infection is shown in **(Table 3.3)**.

Stage 1

- Asymptomatic
- Persistent generalised lymphadenopathy.

Stage 2

- Moderate unexplained weight loss (<10% of presumed or measured body weight)
- Recurrent respiratory tract infections (sinusitis, tonsillitis, otitis media and pharyngitis)
- Herpes zoster
- Angular cheilitis
- Recurrent oral ulceration
- Papular pruritic eruptions
- Seborrhoeic dermatitis
- Fungal nail infections

Stage 3

- Unexplained severe weight loss (>10% of presumed or measured body weight)
- Unexplained chronic diarrhoea for longer than 1 month
- Unexplained persistent fever (above 37.6°C intermittent or constant, for longer than 1 month)
- Persistent oral candidiasis
- Oral hairy leucoplakia (OHL)
- Pulmonary tuberculosis (current)
- Severe bacterial infections (pneumonia, empyema, pyomyositis, bone or joint infection, meningitis or bacteraemia)
- Acute necrotising ulcerative stomatitis, gingivitis or periodontitis
- Unexplained anaemia (<8 g/dL), neutropaenia (<0.5 per litre) or chronic thrombocytopaenia (<50 × 109 per litre).

Stage 4

- HIV wasting syndrome
- Pneumocystis pneumonia
- Recurrent severe bacterial pneumonia
- Chronic herpes simplex infection (orolabial, genital or anorectal of more than 1 month's duration or visceral at any site)
- Oesophageal candidiasis (candidiasis of trachea, bronchi or lungs)
- Extrapulmonary tuberculosis
- Kaposi's sarcoma
- Cytomegalovirus (CMV) infection
- Central nervous system toxoplasmosis
- HIV encephalopathy
- Extrapulmonary cryptococcosis including meningitis
- Disseminated non-tuberculous mycobacterial infection
- Progressive multifocal leucoencephalopathy (PML)
- Chronic cryptosporidiosis (with diarrhoea)
- Chronic isopsoriasis
- Disseminated mycosis (coccidiomycosis or histoplasmosis)
- Recurrent non-typhoidal *Salmonella bacteraemia*
- Lymphoma (cerebral or B-cell non-Hodgkin) or other solid HIV-associated tumours
- Invasive cervical carcinoma
- Atypical disseminated leishmaniasis
- Symptomatic HIV-associated nephropathy or cardiomyopathy

Table 3.3: WHO clinical staging of established HIV infection.

HIV-associated symptoms	*WHO clinical stage*
Asymptomatic	1
Mild symptoms	2
Advanced symptoms	3
Severe symptoms	4

IMMUNOPATHOGENESIS

Profound immunodeficiency occurs due to deficiency of CD4 counts of T lymphocytes **(Figs. 3.5 and 3.6)**.

- **Stage of primary infection:** Viral replication takes place in lymphoid organs and leads to viraemia.
- **Stage of clinical latency:** Lymphadenopathy is a sign of immune response, which leads to curtailment of viraemia by efficient trapping of virions with the help of follicular dendritic cells (follicular dentritic cells) residing in the germinal centres of lymph nodes. As the disease progresses, disruption of architecture of germinal centre with dissolution of Follicular dentritic cell network leads to loss of trapping function of lymph nodes with accelerated viraemia that leads to the final stage of advanced HIV disease.

Effect of Disease

$CD4^+$ T Cells

- Defective T cell cloning and colony-forming efficiencies
- Reduction in number
- Reduced responses to antigen/mitogen
- Impaired expression of interleukin 2 (IL-2) receptors—defective IL-2 production
- Decreased interferon-gamma production

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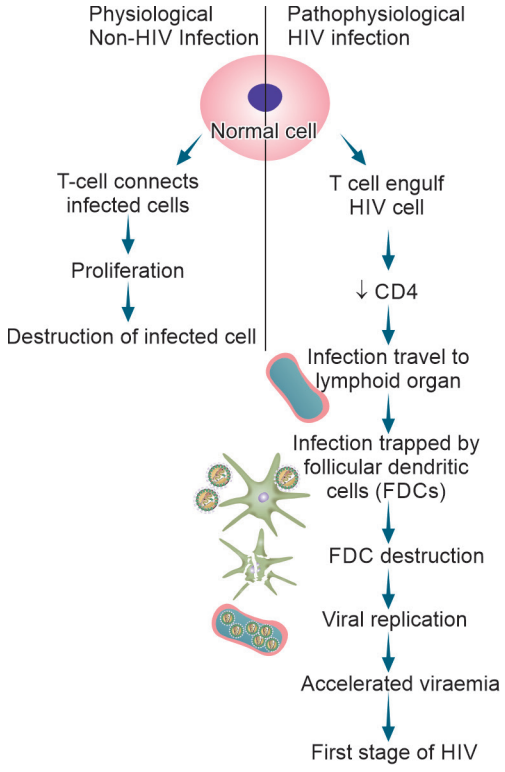

Fig. 3.6: Response of immune cells in non-HIV-infected and HIV-infected patients.

CD4⁺ T Cell Levels

- **Stage of primary infection:** More than 500/μL (prior to significant immunosuppression)
- **Intermediate stage:** 200–500/μL
- **Advanced stage:** Less than 200/μL.

CD8⁺ T cells: Loss of cytolytic activity and the count may be normal, elevated or decreased.

B-Lymphocytes

- Number increases by proliferation
- Increased secretion of tumour necrosis factor-alpha (TNF-α) and IL-6
- Reduced responses to specific antigen/mitogen
- Increased production of immunoglobulins—hypergammaglobulinaemia.

Monocytes/Macrophages

- They are normal in number
- Defective chemotaxis and defective antigen presentation
- Defective secretion of cytokine: IL-1
- Defective phagocytosis and killing

Natural Killer Cells

Defective natural killer (NK) cell activity with normal count.

Humoral and Cell-mediated Immunity

- Multiple antigens of HIV induce formation of different types of antibodies
- Antibodies appear within 2–8 weeks after primary infection
- Various types of antibodies—binding, neutralising, protective, pathogenic and enhancing antibodies are formed and their functional significance is not clear.
- Gradual reduction of CD4⁺ T cells during the course of HIV disease and CD8⁺ T cells are generated which have a protective role in containing the infection in the initial stages.

CLINICAL FEATURES

Group I (Acute Seroconversion Illness)

- It occurs 3–6 weeks following primary infection.
- Patient is highly infectious during this stage characterised by markedly depleted CD4 count.
- Anorexia, nausea, vomiting, headache, body-pain, arthralgia, fever, pharyngitis, weight loss and diarrhoea
- Meningitis, encephalitis, myelopathy, peripheral neuropathy.
- Maculopapular rash, mucocutaneous ulceration.

Group II (Asymptomatic Infection)

- They remain symptom free for 10 years.
- During symptom-free period, patients are monitored by laboratory tests, i.e., CD4, CD8, CD4: CD8 ratios, serum and urinary neopterin, p_{24} antigen and antibody titre.
- Towards the end of this stage, skin lesions like seborrhoeic dermatitis, folliculitis, OHL with CD4⁺ T cell count falling below 400/μL.

Group III (Persistent Generalised Lymphadenopathy) (Fig. 3.7)

- This stage in the past was known as pre-AIDS syndrome or AIDS-related complex (ARC).
- Nodes more than 1 cm in diametre, in two or more extrainguinal sites for more than 3 months without an obvious cause.
- The lymph nodes are symmetrical, firm, discrete and non-tender.
- Constitutional symptoms like fever, weight loss can occur.

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Clinical Medicine for Dental Students

It is my privilege and honor to present the fourth edition of *Clinical Medicine for Dental Students*. The basic principles of clinical examination do not change. All the chapters have been revised and updated. Many newer definitions and classifications have been added in the respective disorders. The national protocol in the management of tuberculosis, especially drug-resistant tuberculosis, is included. In infectious diseases, chapter "COVID-19" has been incorporated. The newer drugs in the management of many disorders, especially hepatitis, epilepsy, and diabetes mellitus, have been added.



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