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MEDICINE

For AYUSH Students

(Ayurveda, Yoga & Naturopathy, Unani, Siddha and Homeopathic Students)



R Alagappan

JAYPEE

Contents

1. Introduction to Clinical Medicine 1
2. Nutrition 33
3. Infectious Diseases 55
4. Clinical Pharmacology 81
5. Oral Manifestations in Systemic Disorders 115
6. Cardiovascular System 123
7. Respiratory System 209
8. Abdomen 263
9. Haematology 309
10. Nephrology 339
11. Endocrine and Metabolic Disorders 355
12. Central Nervous System 419
13. Substance Abuse 521
14. Imaging Modalities in Internal Medicine 531

Appendix *551*

Laboratory Reference Values *553*

Index *555*

Chapter 3
Infectious Diseases

HISTORY

Age Group

Demographic details

Occupational Exposure

Nature of Symptomatology

General Systemic Enquiry

Recent travel

Exposure to Pets/Animals/ Insect bites

Recent Dietary Intake

Sexual Habits

Previous Treatment (Eg. Blood Transfusion)

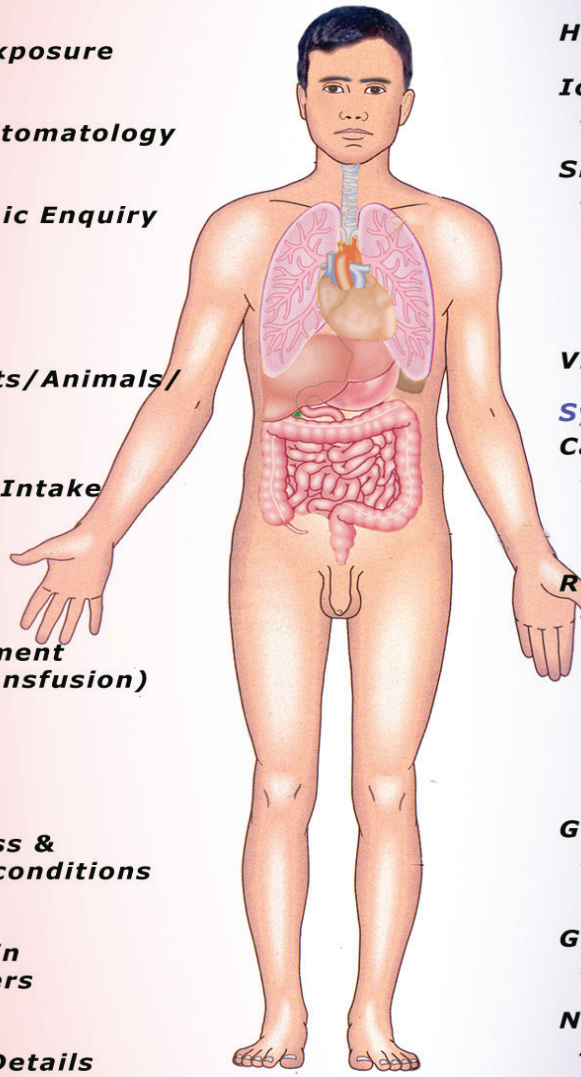
Addictions

Comorbid Illness & Predisposing conditions

Similar illness in Family members

Immunisation Details

Menstrual History (In Reproductive Age Group)



CLINICAL EXAMINATION

General Examination

Mental Changes

Temperature Pattern

Hydration

Icterus/Clubbing/Cyanosis/ Lymphadenopathy

Skin Lesions
- *Site*
- *Distribution*
- *Pattern of Appearance & Disappearance*
- *Secondary Changes*

Vital Signs

Systemic Examination

Cardiovascular System
- *Hemodynamic Status*
- *New onset murmurs*
- *Pericardial Rubs*

Respiratory System
- *Signs of Respiratory Distress*
- *Upper Airways(including ear)*
- *Lower Airways*
  - *Bronchial Sounds*
  - *Added Sounds*
  - *Diminished/Absent sounds*
  - *Chest wall Tenderness*

Gastrointestinal System
- *Focal/Generalised Tenderness*
- *Organomegaly*

Genitourinary System
- *Discharge/Ulcers*

Nervous System
- *Altered sensorium*
- *Nuchal rigidity*
- *Focal Deficits*

Skin,Soft tissue & Skeletal Examination

Appropriate Investigations & Specific Treatment

TYPHOID AND PARATYPHOID FEVER (ENTERIC FEVER)

Enteric fever is a prolonged, continuous or remittent fever caused by *Salmonella typhi* (typhoid) and *Salmonella paratyphi* A and B (paratyphoid). The food or water contaminated by excreta of typhoid patients or convalescing/chronic carriers of typhoid are the main source of infection. The incubation period is 3 to 21 days.

Chronic carriers excrete the bacilli intermittently in the stool and less commonly in the urine for more than one year. Carrier State is more common in females above the age of 50 and they often have gallstones.

Pathogenesis

The orally ingested bacilli invade the small bowel and they multiply intracellularly to produce transient and asymptomatic bacteremia. When the number of intracellular bacteria increases, secondary bacteremia occurs with fever and other clinical signs. It results in the invasion of gallbladder and Peyer's patches of the small intestine. These lesions ulcerate and heal. Sometimes it may bleed and perforate.

Clinical Features

It presents with fever, malaise, headache, centrally coated tongue, relative bradycardia, constipation/diarrhoea and hepatosplenomegaly.

Pale red blanching, slightly raised macules known as "Rose spots" appear over the chest and abdomen at the end of the first week.

Abdominal pain, distension and epistaxis may occur.

Signs of meningism, delirium, other complications including coma and death can occur at the end of second week.

PARATYPHOID FEVER

It is caused by *S. paratyphi* A and B. The incubation period and the course is shorter. It runs a milder course with abundant rash and less frequent intestinal complication.

Investigations

1. Blood—Normocytic normochromic anaemia and leucopenia (Leucocytosis in the presence of severe septicaemia and other complications)
2. Positive blood culture—(First week)
3. Positive stool/urine culture (second and third week)
4. Positive marrow culture (highest yield—even in partially treated cases)
5. Widal test is not confirmatory but only suggestive.
6. LFT – Aminotransferase, alkaline phosphotase and lactate dehydrogenase

Widal Test

- It detects antibodies to the causative organisms.
- It should be interpreted with caution in typhoid vaccinated patients.
- Patients develop agglutinating antibodies to O, H, and Vi antigens.
- The widal test detects and measures these antibodies.
- O-antibody represents soma and specific.
- H-antibody represents flagella and is nonspecific.
- Rise of Vi antibodies occur four weeks after illness and it may be useful to detect carrier state.
- Either a fourfold rise in titer or high titer of antibody to 0 antigen (1:640) is suggestive and not specific.
- To detect fourfold rise in titer, the base line sample should be obtained earlier.
- In Indian population among unvaccinated patients a titer of 1:200 or more is suggestive.

Complications

General

Toxemia and typhoid state, hyperpyrexia, peripheral circulatory failure, disseminated intravascular coagulation, deep vein thrombosis and thrombophlebitis.

Gastrointestinal

Haemorrhage, perforation, paralytic ileus, peritonitis, parotitis, toxic hepatitis.

CNS

Meningism, meningitis, encephalitis, meningovasculitis.

Other Systems

Myocarditis, pyelonephritis, glomerulonephritis, osteomyelitis, arthritis, cholecystitis, bronchitis, pneumonia, orchitis and rarely alopecia.

Management

1. Bed rest, feeding, nursing and maintenance of fluid and electrolyte balance.
2. Several antibiotics are effective in enteric fever and any one of the antibiotics can be given.
   A. Ciprofloxacin 500-750 mg 12-hourly
   B. Co-trimoxazole 2 to 3 tablets 12-hourly
   C. Ampicillin/Amoxycillin 1g 6th hourly
   D. Chloramphenicol 500 mg 6th hourly

E. Ceftriaxone - 4 gm daily for first two days followed by 2 gm daily for 3 days.
Or Ceftriaxone – 1-2 g IV for 10 days
F. Azithromycin – 1g PO daily for 5 days

Antibiotics must be continued either for 14 days or 5 to 7 days after the temperature settles down to normal level.

Management of Complications

Typhoid state: Due to severe toxemia, patient is in a state of delirium, tremulousness of the hands, carphalogy (picking movements of the hands), and coma vigil (stage of coma with eyes open).

In addition to parenteral antibiotic, give a dexamethasone 3 mg/kg IV stat, then 1 mg/kg 6th hourly for two days.

Intestinal haemorrhage: Unexplained tachycardia, fall in BP and temperature during the end of second week denotes intestinal haemorrhage. The patient might pass large amounts of clotted and fluid blood. Blood transfusion and early surgical intervention is necessary.

Intestinal perforation: Abdominal pain, tenderness, and rigidity denote this complication. If the general condition permits, early surgical intervention is ideal.

Relapse: Mostly it occurs in partially treated cases, one or two weeks after recovery. Use the same antimicrobial in adequate dose.

Chronic Carrier State

1. Ciprofloxacin 500 mg bid for four weeks
2. Ampicillin/Amoxycillin 100 mg/kg/day with probenecid 30 mg/kg/day for six weeks.
3. Cotrimoxazole (800+160) bid plus Rifampin 600 mg once daily for six weeks.
4. Cholecystectomy may be needed, if any one of the above regimen fails to eradicate the carrier state.

Prevention

1. Proper sanitation and protected water supply
2. Vaccination

A. *TAB vaccine:* This contains heat killed and phenol preserved
S. typhi 1000 million and *S. paratyphi* A and B 750 million each/ml. Give 0.5 ml IM on two occasions with a week interval. Booster dose has to be given every year.

B. *Typhoid Vi Polysaccharide vaccine*: 0.5 ml IM single dose gives protection for three years.

C. *Live oral vaccine (Ty21a):* Three doses are to be taken. One capsule 1 hr prior to meal on every alternate days.

Moderately enlarged firm spleen is against the diagnosis of enteric fever, Mildly enlarged soft spleen is suggestive of enteric fever.

DIPHTHERIA

Diphtheria is an acute infectious disease caused by nonmotile, nonsporing, generally aerobic, gram-positive rods *Corynebacterium diphtheriae*. The diagnostic feature is the formation of bluish-white or grayish-green pseudomembrane at the site of infection. The organisms release soluble exotoxin, which is toxic to myocardium and nervous system.

Pathology

- The disease is usually spread by droplet infection from infected patients or carriers.
- The common site of infection is upper respiratory tract.
- The infection may also occur in other sites like nose, ears, conjunctiva, genital tract and skin.
- The incubation period is 2-4 days. At the site of infection there is hyperaemia followed by pseudo-membrane formation.
- The membrane has well-defined edge and is firmly adherent. It bleeds on forcible removal.
- Regional lymph nodes are enlarged.
- The exotoxin released can cause damage to the myocardium, kidneys, adrenal glands, cranial and peripheral nerves.

Clinical Features

1. Sore throat
2. Low grade fever
3. Headache
4. Malaise
5. Membrane formation over the tonsils and pharynx
6. Gross cervical lymphadenopathy (Bull neck).

Nasal Diphtheria

Foul smelling serosanguinous discharge, epistaxis.

Laryngeal Diphtheria

Airway obstruction due to membrane, hoarseness of voice, stridor, dyspnoea, cyanosis.

Cutaneous Diphtheria

The organisms enter through the existing wounds, abrasions, burns, since the intact skin does not permit invasion. It causes punched out ulceration covered with a pseudomembrane.

Complications

1. Airway obstruction is more with lesions of larynx, and tracheobroncheal tree.
2. *Myocarditis:* 50 to 70% patients have myocarditis during the acute phase.
   Clinical manifestations occur only in 25% of cases.
   Diminished intensity of heart sounds, tachycardia, gallop-rhythm, systolic murmurs, cardiac failure.
   ECG shows ST-T wave changes, varying degrees of arrhythmias and heart block, atrial fibrillation, ventricular ectopic beats, ventricular tachycardia, ventricular fibrillation, sudden death.
3. *Polyneuritis*

- Cranial polyneuritis—It occurs in 10% of cases.
- Palatal and pharyngeal paralysis occurs during the first two weeks.
- Cranial nerves commonly affected are 3rd, 6th, 7th, 9th and 10th.
- Difficulty in swallowing, nasal regurgitation, hoarseness of voice, and loss of accommo–dation and ophthalmoplegia are the clinical manifestations of cranial nerve palsies.
- Peripheral polyneuritis: It begins 1 to 3 months after the illness.
- Proximal weakness of extremities with subsequent spread distally.
- Respiratory failure occurs in severe form of diphtheria.
- Paraesthesias—Glove and stocking distribution.
- Polyneuritis resolves completely.

4. Pneumonia: Fifty percent of the fatal cases develop pneumonia.
5. Less common complications
   - Encephalitis
   - Cerebral infarction
   - Pulmonary embolism
   - Bacteremia
   - Endocarditis
   - Renal failure.

Differential Diagnosis

Acute follicular tonsillitis, ulceration of tonsils in agranulocytosis and acute leukemia.

	Acute follicular tonsillitis	*Diphtheria*
Membrane	Confined to tonsil	Extensive
Colour	Yellowish	Bluish-white/ greenish-grey
	Loosely adherent	Firmly adherent
	Easily removable	Bleeds on forcible removal
Fever	High grade	Low grade
Regional lymph nodes	Mildly enlarged	Markedly enlarged (Bull-neck)

Diagnosis

1. Clinical diagnosis is based on the presence of pseudo-membrane.
2. Staining of infected material to demonstrate the organisms.
3. Culture using Loeffler's medium.
4. Antigen antibody tests.

Management

1. Administer antitoxin diphtheritic serum (ADS) on making clinical diagnosis without waiting for lab datas. The toxin once fixed to the tissue cannot be neutralized.
2. Dose of ADS depends on site, duration and severity of infection
   a. One tonsil—20,000 units
   b. Both tonsils—48 hours-40,000 units
   c. Pharyngeal/laryngeal—72 hours - 80,000 units
   d. Extensive—more than 72 hours - 1,20,000 units.
   Give ADS in normal saline infusion IV over 1 hour.
   Be prepared to tackle hypersensitivity reactions with epinephrine, steroids and antihistamine.
   Give a test dose of 4000 units IM before IV therapeutic dose.
   Adapt de-sensitization procedure for patients with adverse reaction.
3. Notify public health authorities.
4. Antibiotics to eradicate *C. diphtheriae* in patients and carriers. It has no action on exotoxin.
   Penicillin G, erythromycin, rifampin or clindamycin is useful. Procaine penicillin G, 600,000 units bid. 14 days or Erythromycin 500 mg 6th hourly or Rifampin 600 mg OD for 14 days.
   Amoxycillin - 500 mg 8th hourly for 14 days.
   Antibiotics are also useful in preventing transmission of diphtheria from patient to susceptible contacts.

5. Treatment of complications: Airway obstruction—Tracheostomy.
   Myocarditis—Monitor and treat arrhythmias and failure.

Prevention

Immunize all children.

Three doses of DPT at the age of 3rd, 4th and 5th month and booster doses at one year and school going age.

Treat all contacts with erythromycin and give a booster dose of toxoid.

Strict isolation until 3 swabs 24 hours apart are culture negative.

VIRAL EXANTHEMS

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, rickettsia) exanthems, the palms and soles are involved.

MEASLES (RUBEOLA)

Measles is an acute infectious fever with maculopapular eruption caused by RNA-paramyxovirus.

It occurs only in human beings.

Pathogenesis

- It spreads by droplet infection.
- It involves the respiratory mucous membrane or conjunctivae.
- The person is infectious 5 days after exposure and until 5 days after the appearance of skin lesions.
- Children between the age of 5-15 years are commonly affected.
- The incubation period is 10 days.
- One attack confers life-long immunity and the affected mother confers passive immunity on to her infant for first six months.

Clinical Features

A. *Catarrhal stage:* Fever, malaise, irritability, and conjunctivitis with photophobia, running nose and cough for the first two days.
   Koplik's spots: On the third day small, red, irregular lesions with bluish-white centers appear on the mucous membrane of the mouth, and occasionally on the conjunctivae, or intestinal mucosa. They first appear on the buccal mucosa opposite the upper molar teeth.

B. *Exanthematous stage:* On the fourth day maculopapular rash appears over the back of the ears, forehead and then spreads downwards over the face, neck, trunk and limbs. Brown discolouration, desquamation of maculopapular rash with disappearance in the same order as they appeared. The total duration of the rash is 6 days.

Complications

Respiratory system Bronchitis, bronchiolitis, pneumonia.

Eyes Conjunctivitis, corneal ulceration, keratitis, blindness.

Gastrointestinal Stomatitis, involvement of mesenteric lymphnode, appendix with peritonitis.

Nervous system Postviral encephalitis, sub-acute sclerosing panencephalitis, rarely transverse or ascending myelitis.

Other complications Otitis media, myocarditis, glomerulonephritis, death of fetus in 20% of pregnancy, exacerbation of existing tuberculosis, thrombocytopenia, kwashiorkor and marasmus.

Investigations

1. Leukopenia in early phase—leukocytosis denotes secondary bacterial infection—Lymphopenia less than 2000/microliter indicate poor prognosis.
2. Virus culture and isolation.
3. Demonstration of viral antigen in the infected cells.
4. Rising specific antibody levels by ELISA (by immunofluorescent technique) and complement fixation tests.
   Clinical hallmark—Koplik's spots followed by maculopapular rash.

Management

1. No specific treatment—symptomatic treatment.
2. Antibiotic for secondary bacterial infection.
3. Isolation— feeding - nursing.

Prevention

- Active immunization—Live attenuated measles virus - Measles, mumps and rubella vaccine (MMR) given at 15 months of age confers lifelong immunity.
- Second dose is given at 12 years.
- Immunize all HIV infected individuals against measles.

Contraindication for MMR Vaccine

1. Pregnant women.
2. Children with untreated tuberculosis.
3. Patients with leukemia, lymphoma.
4. Immunocompromised patients.

Passive Immunization

For contacts under 18 months of age and for those in whom, vaccine is contraindicated.

It should be given within 6 days of exposure.

SMALLPOX (Variola Major)

Smallpox is a highly infectious disease caused by DNA - variola virus. It is characterized by vesicular and pustular eruption and the morbidity and the mortality are greater.

The official announcement of global eradication of smallpox was made in the year 1979. This achievement was made possible by intensive compulsory mass vaccination.

Only two laboratories (USA and Russia) maintain live smallpox virus for research purpose. The staffs over there only have to be vaccinated.

Pathogenesis

- The virus spreads by respiratory route.
- Nasopharyngeal secretions, skin lesions and scabs contain the virus.
- The virus can remain viable in the dried scab for more than one year.
- The incubation period is 14 days.

Clinical Features

It existed in two forms.

- Variola major—serious illness - mortality more than 20%.
- Variola minor—mild form - mortality less than 1%.

Prodromal stage: 2 to 4 days. Fever, headache, backache, malaise, abdominal pain, vomiting and occasionally diarrhoea.

Exanthematous stage

- Skin rash progresses from macule to papule, vesicle, pustule and scab formation.
- It progresses roughly in 48 hours from one stage to another.
- The skin lesion is deep seated, multiloculated with central umbilication.
- The rash is distributed in centrifugal pattern.
- The face, arms, legs, palms and soles are more affected than the trunk.
- Fever once again appears in the pustular stage.
- Intraoral rash (enanthem) when it breaks down releases large amount of virus.
- After 14 days crusts heal and fall, leaving depigmented scars.

Investigations

1. Blood—Leukopenia, thrombocytopenia, lymphocytosis, leukocytosis in the stage of pustules.
2. Isolation of virus from skin lesions, oropharynx, urine.
3. Rising antibody titers—Haemagglutination, complement fixation test, neutralization and gel precipitation.

Complications Bacterial pneumonia, keratitis, corneal ulceration, arthritis, encephalitis, osteomyelitis, orchitis, etc.

Management

1. No specific treatment. Only symptomatic.
2. Antibiotics for secondary bacterial infection.
3. Good nursing care.
4. Vaccination and immunotherapy with vaccinia immune globulin during the first week of incubation period modified the course of the disease.

VARICELLA-ZOSTER VIRUS INFECTIONS

It causes two types of clinical disorders.

1. Chickenpox is a benign, infectious febrile illness of childhood, characterized by exanthematous vesicular rash.
2. Herpes zoster (Shingles): It presents as dermatomal painful vesicular rash due to reactivation of latent varicella-zoster virus. Children coming in contact with herpes zoster patients, can develop chickenpox and not *vice-versa.*

Chickenpox (Varicella)

Varicella-zoster virus is a DNA virus.

It spreads by droplet infection or contact with skin lesions of chickenpox patients or contact with herpes-zoster lesions. After a period of viremia, vesicles develop in the skin and mucous membrane.

Patient is infective two days prior to rash and until all the vesicles are crusted. The incubation period is 14 days.

Clinical Features

Prodromal stage 2-3 days. Low-grade fever, headache, malaise, backache, and sore throat.

Exanthematous stage
- Enanthem earlier than exanthem.
- Maculopapules first appear over the palate and pharyngeal mucosa.
- The skin lesions appear in crops maculo-papules, vesicles, and scabs in varying stages of evolution—Pleomorphic.
- Vesicles are on the skin rather than in the skin and unilocular.
- It collapses on puncture and there is no central umbilication.
- The rash distribution is centripetal—Maximally over the trunk, axillae, sparing the distal parts of the extremities.
- The vesicles become pustular in 24 hours.
- Secondary bacterial (*Streptococcus pyogenes*, *Staphylococcus aureus*) super-infections of the skin lesions are common.

Complications

1. *Nervous system*
   Acute cerebellar ataxia
   Aseptic meningitis
   Encephalitis
   Transverse myelitis
   Ascending polyneuritis
   Optic neuritis
   Reye's syndrome.

Reye's syndrome
- A serious complication of Varicella-zoster infection and influenza B and to lesser extent influenza A infection.
- Children between the age of 2 and 16 are affected after several days of viral infection.
- Central nervous system symptoms follow nausea and vomiting of one or two days.
- Lethargy, delirium, seizures, coma, and hepatomegaly with elevated AST, ALT and LDH levels.
- Elevated ammonia and bilirubin level with hypoglycemia.
- Lumbar puncture reveals elevated pressure of CSF with normal chemistry, indicating encephalopathy rather than encephalitis.
- Exact pathogenesis is unknown and Aspirin therapy for viral infection may precipitate this syndrome.

2. *Varicella pneumonia*

Most serious complications following chickenpox.

Clinical features
- Tachypnea
- Cough
- Dyspnea
- Pleuritic chest pain
- Cyanosis
- Haemoptysis
- X-ray chest—nodular infiltrates and interstitial pneumonitis.

3. *Other complications*
- Corneal lesions
- Myocarditis
- Nephritis
- Hepatitis
- Arthritis
- Bleeding diatheses.

Perinatal varicella: Newborn does not receive transplacental protective antibodies and has an immature immune system and so, the mortality rate is high when maternal infection occurs, 5 days prior to delivery and 48 hours after delivery.

Varicella embryopathy—a rare cause for congenital limb defects.

Table 3.1: Difference between chickenpox and smallpox

Features	*Chickenpox*	*Smallpox*
Prodrome	Mild	Severe
Distribution	Centripetal i.e. more on the body and less on face, limbs	Centrifugal less on the body more on face and limbs
Nature of skin lesion	Superficial	Deep in the skin
Axilla	Involved	Not involved
Appearance of rash	Multiple crops	Single crop
Stages of rash	Different stage (Pleomorphic)	Same stage (Monomorphic)
Vesicle	Unilocular No-umbilication	Multilocular Umbilication
On Puncture	Collapses	Does not collapse
Scar	Superficial-temporary	Deep-permanent

Investigations

1. Isolation of virus
2. Demonstration of eosinophilic—intranuclear inclusion bodies
3. Immunofluorescent detection of antibody
4. Other serological tests—ELISA, complement fixation.
5. Aspiration of vesicular fluid for PCR or tissue culture.

Management

1. Isolation—feeding-nursing
2. Symptomatic treatment
3. Acyclovir—20 mg/kg or maximum of 800 mg PO five times daily for 5 to 7 days.

In immunocompromised patients with pneumonia and encephalitis—IV acyclovir 10 mg/kg 8th hourly.
4. Acyclovir - eye application.
5. Oral famciclovir - 500 mg tds for one week.
6. Oral valacyclovir - 1000 mg tds for one week.

Prevention

Live attenuated varicella-zoster virus vaccine.

Varilrix: 0.5 cc SC 12 months to 12 years - single dose. 13 years and above - 2 doses with an interval of 8 weeks.

Passive immunization: Varicella-zoster immune globulin for contacts especially immunocompromised, pregnant women, neonates, premature infants, and newborn infants of mothers with chickenpox. Give 0.5 to 1 ml/kg within 72 hours of exposure.

RUBELLA
(German Measles—3 Day Measles)

Rubella is an infectious benign febrile exanthem caused by RNA - Toga virus.

Transplacental transmission of virus in affected mother causes congenital rubella with varying degrees of foetal anomalies.

Pathogenesis

- It spreads by droplet infection.
- After replication in respiratory tract it spreads via the blood stream.
- Viremia is present 8 days before and 2 days after the appearance of rash.
- In affected fetus, the virus multiplies in the liver, spleen, bone marrow and other tissues
- Leading to various fetal malformations.
- The incubation period is 14-21 days.

Clinical Features

1. *Prodromal stage:* 1 to 7 days—headache, malaise, fever, conjunctivitis and lymphadenopathy.
   Infective period – Patient is infective one week before and one week after the onset of rash.
2. *Exanthematous stage:* The rash begins in the forehead and face and spreads downward to the trunk and limbs.

   The maculopapular rash of lighter hue than those of measles and disappear in three days time.

   Enlarged, tender lymphnodes especially posterior cervical group with splenomegaly occurs in the early phase of the disease.

Forchheimer Spots–'Petichial lesions appear in the soft palate' associated with mild corysa and conjunctivitis. Fever occurs only on the first day of rash.

Complications

Congenital rubella syndrome: The virus can persist in the fetus during intrauterine life and upto one month after birth. It can cause the following defects.

Heart Patent ductus-arteriosus, ventricular septal defect, pulmonary stenosis, and myocarditis.

Eyes Corneal clouding, cataract, chorioretinitis, microophthalmia, glaucoma.

Nervous system Mental retardation, deafness.

Others complications Microcephaly, skeletal abnormalities, intrauterine growth retardation.

Arthritis, orchitis, thrombocytopenia, encephalomyelitis.

Immune mediated arthritis involves fingers, wrist and knees in 30 % of patients.

Later complications can be diabetes mellitus, panencephalitis.

The risk is more when the maternal infection occurs in the first trimester of pregnancy especially first 3 to 6 weeks.

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 CHD
16 weeks	10	Deafness or congenital defects
> 20 weeks		Occasional deafness

Diagnosis

1. Isolation of virus
2. Demonstration of viral antigen
3. Presence of specific antibodies

Detection of rubella specific IgG with absent IgM denotes previous infection.

Detection of rubella specific IgM denotes recent infection.

Table 3.2: Differential diagnosis between rubella and measles

Features	*Rubella*	*Measles*
Prodrome	Mild	Severe
Rash	Discrete, light tint	Confluent, Pinkish
Fever	Mild	Severe
Koplik's spots	Nil	Hallmark
Lymphnodes	Retroauricular, Suboccipital Posterior cervical	Not characteristic

Management

1. No specific treatment—only symptomatic.
2. Consider termination of pregnancy to prevent congenital rubella syndrome.

Prevention

MMR-vaccine as for measles confers lifelong immunity.

HERPES ZOSTER (SHINGLES)

Herpes zoster occurs as a result of reactivation of latent virus from dorsal root ganglia. Reactivation may be spontaneous affecting middle aged or elderly or may be due to immuno-suppression as in HIV, Hodgkin's disease, Non-Hodgkin lymphoma and other malignancies (Fig. 3.1).



Figure 3.1: Herpes zoster—cutaneous vesicles

Clinical Features

- Unilateral vesicular lesion confined to a dermatome with pain.
- Dermatomes from T3 to L3 are more frequently affected.
- Trigeminal ganglion—Vth cranial nerve- ophthalmic division more commonly than other divisions (Herpes zoster ophthalmicus).
- Geniculate ganglion (Ramsay Hunt syndrome), vesicles in the auricle, palate with facial palsy and impairment of taste.
- In HIV—Several dermatomes are involved.
- Disseminated—generalized herpes can occur in leukemia, lymphoma and immunocompromised individuals.
- Pregnancy—The foetus may be affected- congenital malformations.
- Pain may precede or follow skin lesions.
- The total duration of the rash is 7 to 10 days.
- Pain during acute phase due to neuritis is common.

Postherpetic Neuralgia

- Pain may persist for many months.
- It is uncommon in young individuals.
- 50% of patients over the age of 50 suffer.
- Sensory changes either hypo- or hyperesthesia is common.

Diagnosis

1. Smear study
2. FAMA test—Fluorescent antibody to membrane antigen test
3. ELISA test

Complications

- Common in immunocompromised with disseminated herpes.
- Meningoencephalitis, pneumonitis, hepatitis, transverse myelitis, Motor paralysis of involved segment may occur rarely.

Management

1. Oral acyclovir 800 mg five times daily for 1 week.
   Valacyclovir - 1000 mg tds for one week.
2. Severe infection or immunocompromised patients—5-10 mg IV infusion/kg 8th hourly for 7 to 10 days.
3. Topical acyclovir for skin lesions and eye lesions.
4. Postherpetic neuralgia: Analgesics—NSAIDs
   Amytriptyline 25 to 100 mg daily
   Diphenylhydantoin 200 to 600 mg/day or Carbamazepine 200-600 mg/day
   Gabapentin 300 mg od or bid
   The above drugs act by modifying neurotransmission.
   Transcutaneous nerve stimulation
   A short course of glucocorticoids like prednisolone in elderly in the early phase may prevent postherpetic neuralgia and it is controversial.
5. Antibiotics to prevent secondary bacterial infection of skin lesions.

HERPES SIMPLEX

Herpes simplex virus (herpes virus hominis) is a 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 causes commonly herpes labialis and herpes keratitis.

HSV-2 cause genital lesions and are transmitted by sexual contact.

Pathogenesis

- HSV enters through the mucosal surfaces or abraded skin and the initial replication takes place in cells of epidermis and dermis.
- 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 reactivated by fever, physical or mental stress, or sunlight, immunosuppression or trauma to skin.
- The incubation period is 7 to 21 days.

Clinical Features

- Both viral subtypes can cause genital and orofacial infections.
- 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 three weeks.

Various Types of Primary Infection can occur

1. *Gingivostomatitis and pharyngitis*
   - Most common clinical manifestation of first episode HSV-1 infection.
   - Lesions involve hard and soft palate, gingiva, tongue, lip, and facial area.
   - Fever, malaise, myalgias, inability to eat and cervical adenopathy.
2. *Genital herpes*
   - HSV type 1 and 2 can cause the infection.
   - 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, prostatitis can occur.
3. *Rectal and perianal*
   Distal 10 cm of rectal mucosa and perianal region are affected.
4. *Herpetic whitlow*
   - Complication of primary oral or genital herpes.
   - Digital infection occurs through a breach in the skin.
5. *Herpes gladiatorum:* (traumatic herpes) lesions occur in any area of the skin due to trauma as in wrestlers—thorax, ears, face, hands.
6. *Herpetic keratitis:* Pain, chemosis, conjunctivitis, dendritic corneal ulcer—blindness.
7. *CNS:* HSV meningitis and encephalitis can occur either as a primary infection or due to reactivation.
8. *Eczema herpeticum:* Lesions occur on eczematous skin.
9. *Visceral infections*
   - During viremia, multiple organs might get involved—esophagus, lung, liver, adrenals, kidneys.
   - These types of disseminated lesions are common in immunocompromised individuals—so called systemic infection.
10. Neonatal HSV infection
    - Neonates less than 6 weeks of age.
    - HSV-2 infection more common than HSV-1.
    - Contact via infected genital secretions during delivery or contact with herpes lesions.
    - Systemic infection is common with CNS involvement.
    - Mortality is high if not treated early.

Investigations

1. Rising antibody titres.
2. Viral culture.
3. Demonstration of HSV antigen from lesions.
4. HSV PCR techniques may be more sensitive.

Management of HSV Infections

1. *Primary HSV:* Famaciclovir 250 mg 8th hourly or Valacyclovir 500 mg 12th hourly or Acyclovir - 200 mg 5 times daily for 10 to 14 days.
2. *Severe form:* Preventing oral intake – Acyclovir 5-10 mg IV 8th hourly.
3. *Recurrent Episode(HSV 1&2):* Oral acyclovir 200 mg 6th hourly along with acyclovir ointment topical application 3-5 times daily or Famaciclovir 250 mg 12th hourly or Valacyclovir 500 mg daily for 5-7 days.
4. *Immunocompromised individuals:* Acyclovir 400 mg 6th hourly or Famaciclovir 500 mg 12th hourly or valacyclovir 1000 mg 12th hourly for 14 days.
5. *Genital HSV:* Treat as for primary HSV and for recurrent episode limit the duration of therapy to 5-7 days.
6. *CNS HSV/Severe Complications:* Acyclovir IV 10-20 mg/kg 8th hourly for 10 days.
7. *Neonatal HSV:* Oral acyclovir 60 mg/kg/day in three divided dose for 21 days or IV acyclovir
8. *Acyclovir Resistant HSV:* IV foscarnet 40 mg/kg 8th hourly for 14 days.

Prevention

Suppressive antiviral chemotherapy—Acyclovir 400 mg BID or 800 mg OD for 5 to 10 days.

Famaciclovir 250 mg 12th hourly or valacyclovir 500 mg daily for 5-10 days can also be used for disease prevention or suppression.

MUMPS

Mumps is an acute infectious disease caused by single stranded RNA Paramyxovirus. It causes painful enlargement of salivary glands especially parotids. It might involve meninges, pancreas, gonads, lacrimal glands, thymus, thyroids, 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 lymphnodes where the virus replicates. Then, it is disseminated through the blood stream to the target organs like parotids and meninges.
- The incubation period varies between 12 to 25 days.
- One attack confers life-long immunity.

Clinical Features

- Anorexia, sore throat, malaise, fever, tenderness at the angle of the jaw with painful enlargement of one or both parotids.
- Bilateral parotid enlargement occurs in 60% of the cases.
- Main complaints are difficulty in eating, swallowing and talking.
- Saliva is infectious 7 days prior and after parotitis.
- Diagnosis is often made only on clinical grounds.

Epididymo-orchitis:

- Occur in 25% of postpubertal males, during the second week.
- Unilateral or bilateral testicular swelling with pain and tenderness.
- It subsides after one week.
- Atrophy of testicle 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: Upper abdominal pain with elevated serum amylase. Rarely brittle diabetes may occur following pancreatitis.

CNS

- 10% of cases develop meningitis with stiff neck and headache.
- Encephalitis can occur with seizures, headache and unconsciousness.
- Rarely transverse myelitis, motor polyneuritis or cerebellar ataxia can occur.

Other rare complications: Myocarditis, nephritis, and hepatitis

Investigations

- Blood—mild leucopenia with relative lymphocytosis.
- Serum amylase and lipase level elevated in pancreatitis.
- Isolation of virus from saliva, blood, throat swab and CSF.
- Detection of viral antigen directly from oropharyngeal cells with the help of immuno-fluorescence.
- Demonstration of specific antibodies (ELISA) and rise in titers.

Differential Diagnosis

Parotitis (pyogenic), salivary duct calculus, sarcoidosis.

Management

1. No specific treatment
2. Analgesics and local fomentation
3. Support the testes to relieve pain and prednisone 60 mg/day for 7 days.
4. Arthritis—Ibuprofen and prednisone.

Prednisolone can also be given to relieve discomfort of arthritis.

Prevention: Live attenuated mumps virus vaccine can be given to children above the age of one year. It confers life-long immunity. It is also advised for HIV infected patients.

SEXUALLY TRANSMITTED DISEASES

Sexually transmitted diseases are either caused by bacteria or viruses and also due to protozoa or fungi. Transmission is predominantly by sexual intercourse. Men and Women between the ages of 15 and 30 are commonly affected. High-risk group includes commercial sex workers, army services personnel, travellers and homosexual men and women with multiple partners.

1. STDs due to bacterial infection
   - *Treponema pallidum*—Syphilis
   - *Neisseria gonorrhoeae*—Gonorrhoea
   - *Haemophilus ducreyi*—Chancroid
   - Donovania granulomatis—Granuloma inguinale
   - *Chlamydia trachomatis*—Urethritis, cervicitis, lymphogranuloma venereum.

2. STDs due to viral infection
   - Human immunodeficiency viruses (HIV-1 and 2)—AIDS
   - Herpes simplex virus type 2 (HSV-2)— Herpes genitalis
   - Human papillomavirus—several types— Genital warts
   - Molluscum contagiosum virus— Molluscum contagiosum
   - Human T-cell Lymphotropic virus (HTLV-1) — Tropical spastic paraparesis
   - Other viruses: Hepatitis B, C, D viruses, and cytomegalovirus.
3. STDs due to other causes
   - *Trichmonas vaginalis* (Protozoa)—Trichomonas vaginitis
   - *Candida albicans* (Fungi)—Genital candidiasis
   - *Phthirus pubis*—Pubic lice infestations
   - *Sarcoptes scabiei*—Scabies.

Common STD Syndromes

Genital ulcers—T.Pallidum, H.ducreyi, HSV-1 and 2, C.granulomatis, C.trachomatis (LGV).

Urethritis and epididymitis—N. gonorrhoeae, C. trachomatis, HSV.

Cervicitis and vulvovaginitis—C. trachomatis, N. gonorrhoeae, C. albicans.

SYPHILIS

Syphilis is a chronic systemic disease caused by Treponema pallidum.

The incubation period is 3 weeks.

It marches through various stages from the stage of primary lesion (localized), a secondary bacterimic stage with diffuse mucocutaneous lesions and generalized lymphadenopathy—(4 to 8 weeks after chancre), Tertiary stage (2 to 20 years later with progressive destructive lesions — Gummas involving many organs, and quarternary stage with involvement of CVS and Nervous system.

Pathogenesis

T. pallidum invades the intact mucous membrane or abraded skin, enters the blood or lymphatics to produce systemic infection.

The blood even during incubation period is infectious.

The primary lesion occurs at the site of inoculation, after 2 to 3 weeks and it heals spontaneously in 2 to 6 weeks time.

It then marches through various stages.

Classification of Syphilis

I. Acquired
   1. Early syphilis
      a. Primary syphilis
      b. Secondary syphilis
      c. Early latent (WHO - less than 1 year, International less than 2 years).
   2. Late syphilis
      a. Late latent (WHO-more than 1 year, International more than 2 years)
      b. Tertiary syphilis (benign gummatous lesions)
      c. Quarternary syphilis (cardiovascular and neuro-syphilis).

II. Congenital syphilis
   a. Early—appear within first 2 years of life and infectious.
   b. Late—appear after 2 years and noninfectious.
   c. Residual stigmata.

Primary Syphilis

Primary chancre appears as a single painless papule, which ulcerates and coppery-red in colour with indurated cartilaginous consistency of base and margin.

Site of lesion

- Heterosexual men—glans penis
- Homosexual men—anal canal, rectum, mouth, external genitalia
- Women—cervix, labia.
- Other sites – finger, lip, tongue, tonsil, nipple, arms

Regional Lymphadenopathy

- Appear within 1 week after the chancre.
- Painless, discrete, non-suppurative, firm, bilateral inguinal or other regions depending on the site of lesion.
- Primary chancre heals within 4 to 6 weeks and lymphadenopathy may persist for many months (Fig. 3.2).

Differential Diagnosis of Primary Chancre

1. Chancroid
2. Herpes genitalis
3. Lymphogranuloma venereum
4. Traumatic ulcer
5. Reiter's disease
6. Stevens-Johnson syndrome
7. Scabies
8. Early carcinoma
9. Behcet's syndrome (Figs 3.3 and 3.4).

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