1) Common Viral Infections in Pediatric Age Group – Dr. Bakr

Measles mumps and Rubella

**MEASLS**

- RNA paramyxovirus.
- Incubation period is (8-12) days.
- Highly contagious disease.
- Transmitted by droplet through respiratory tract or conjunctivae. Face to face contact is not necessary because viable virus may remain suspended in air for up to 1hr after a source case leaves a room.
- Contagious 3 days before the appearance of rash up to 4-6 days after its onset.
- Most infants are protected by transplacental antibodies till towards the end of first year.
- Subclinical cases are few.
- It is more common in winter and spring.

**Pathogenesis**

- Measles infection causes necrosis of the respiratory tract epithelium with lymphocytic infiltrate.
- Measles produces a small vessel vasculitis on the skin and oral mucus membrane.
- There is fusion of infected cell which results in multinucleated giant cells, the WARTHIN-FINKELDEY giant cells that are pathognomonic for measles.
- The giant cell contains viral particles.

**Clinical manifestations**

- Measles consists of 4 phases: incubation period, prodromal illness, exanthematous phase, and recovery.
- Prodromal phase: begins with a mild fever, followed by Cough, Coryza, Conjunctivitis with photophobia, and increasing fever, then pathognomonic Koplik spots that last for 1-4days before the rash.
- Koplik spots: they appear as discrete red lesions with bluish white spots in the center on the inner aspects of the cheeks at the level of the premolars.
- Rash phase: this phase is often accompanied by high grade fever (40-40.5 c) . The maculopapular rash begins on the head often above the hair line around the forehead, behind the ears, and on the upper neck and spreads all over the body with in 24 hrs in a descending fashion. It fades in the same manner.
- The severity of the illness is related to the extent of rash.
- The rash may be petechial or hemorrhagic (black measles).
- As the rash fades in about 7 days it undergoes desquamation and brownish discoloration.
- Cervical lymphadenitis, splenomegaly, and mesenteric lymphadenitis (with abdominal pain) may be noted.
Complications

1. Otitis Media: is the most common complication of measles.
2. Interstitial pneumonia by measles virus or by secondary bacterial infection is the most common cause of death in patient with measles. Brochilolithis and croup may occur.
3. Activation of latent tuberculosis.
4. Mesenteric adenitis, abdominal pain, diarrhea, vomiting within dehydration.
5. Myocarditis.
7. Subacute sclerosing panencephalitis.
   - **Encephalitis** occur in 1-3/1000 cases of measles, more common in adolescents. This is a postinfectious immunologically mediated process rather than due to direct effect by the virus. Clinical onset begins during the exanthem and present with seizure, lethargy, coma, and irritability.
   - CSF analysis shows lymphocytosis, with elevated protein.
   - Approximately 15% die, 20-40% suffer long term sequelae like mental retardation, motor disability, & deafness.
   - In immunocompromised patient encephalitis may occur due to direct damage of brain tissue by the virus.
   - **Subacute sclerosing panencephalitis:** This is a late neurological complication occurring years after the acute illness, usually in adolescence. After 7-10 years the virus regains virulence and attacks the cells in the CNS resulting to neurodegenerative disease caused by persistent infection of the brain by an altered form of the measles virus.
   - It is a rare complication; measles at an early age favors the development of SSPE.
   - Clinical presentations: it manifest with subtle changes in behavior, irritability, reduced attention span, impaired school performance, myoclonus jerk, gradually progress to choreoathetosis, immobility, dystonia, dementia, stupor, coma and death.
   - Diagnosis done by detecting measles antibody in CSF, EEG, brain biopsy.

Diagnosis

1. Clinical presentation: the diagnosis of measles is almost always based on clinical and epidemiologic finding.
2. Koplik spots are pathognomonic.
3. Serological test: diagnosis can be done by detecting measles IgM antibody, which appear 1-2 days after the appearance of the rash, and persiste for about 1 month. Serologic confirmation may also be made by demonstration of a 4 fold rise in IgG antibodies in acute and convalescent specimen taken 2-4 wk later.
4. Virus isolation and culture from blood, urine, or respiratory secretions.
5. PCR.

Differential diagnosis

1. Rubella
2. Adenoviruses
3. Enteroviruses
4. EBV
5. Roseola
6. Mycoplasma pneumoniae
7. Group A streptococcus
8. Kawasaki
9. Drugs

Treatment

- Supportive Such as antipyretic, oxygen, intravenous fluid in case of dehydration, antibiotics in case of secondary bacterial infection.
- Vitamin A: vitamin decrease mortality and morbidity in patient with measles, indicated for those above 6 months with sever measles.
- Prevention: Vaccination
Mumps

- Mumps is an acute self-limited infection, previously was common, but now unusual in developed countries because of widespread use of vaccination.
- Mumps is a single stranded RNA virus of the family Paramyxoviridae.
- Incubation period 12-25 days.
- Humans are the only host.
- Most frequent in winter and spring.
- Spread by droplets, and contact or fomites.
- It’s contagious from 1-2 days before to 5 days after the parotid swelling.
- Infection is associated with lifelong immunity.
- Transplacental antibodies protect the infant in first 6 months.

Pathogenesis:

- Mumps virus affects the salivary glands, CNS, tests, and to a lesser degree thyroid gland, ovaries, heart, kidney, liver, and joint synovia.
- Virus is acquired through infected respiratory secretions.
- Replicates in the URT & regional L.N.
- Seeds in the salivary glands.
- Mumps virus Cause necrosis of infected cells with inflammatory lymphocytic infiltration and edema.

Clinical manifestations

- Mumps virus infection may be asymptomatic, nonspecific symptoms, or typical.
- 30-40% of cases are subclinical.
- In symptomatic cases presents with a prodrome lasting 1-2 days consisting of fever, muscle pain, head ach, vomiting malaise and pain, parotitis then appears and may be unilateral initially but became bilateral in 70% of cases swelling in the parotid glands lasting 3-7 days. The parotid gland is tender, ingestion of acidic foods or liquids may enhance pain in the parotid area. As the swelling progress the angle of jaw is obscured and the ear lobe may be lifted upward and outward.
- Swelling of the submandibular glands may accompany the parotid swelling, and in 10-20% of cases may be the only gland involved.
- Sublingual glands are involved less commonly.
- Edema over sternum may occur.

Complications

1. Meningoencephalomyelitis: 65% of patients with parotitis have CSF pleocytosis, and more than 10% have clinical manifestations of meningoencephalitis. The meningoencephalitis may occur before, with or after parotitis, most commonly appears 5 days after parotitis.
2. Orchitis, Epididymitis: 15-35% of adolescents and adults, it is rear in prepubertal children. They become manifested at the end of the first week with moderate to high fever, chills, swelling with severe testicular pain. In less than one third it is bilateral. Atrophy is a common squally but infertility is rare.
3. Pancreatitis: Pancreatitis may occur without parotitis manifest as epigastric pain, tenderness and vomiting. Serum amylase may be elevated in mumps with or without pancreatitis.
4. Rare complications may occur like myocarditis, arthritis, thyroiditis.
5. Mumps virus infection during first trimester of pregnancy may cause abortion, no fetal malformation has been reported.
Diagnosis

- Clinical presentation.
- Isolation of the virus from saliva, urine, CSF and blood.
- Serological tests; a rise serum antibody (IgM) to mumps is also diagnostic.

Differential diagnosis

- Parotitis caused by other viruses: influenza, parainfluenza, Enterovirus, CMV, AIDS
- Seropurpurative parotitis: Usually a result of S. aureus infection, in this case purulent material can be expressed through Stensen duct, the swelling is unilateral and is severely painful.
- Salivary calculus,
- Recurrent parotitis due to Sjogren syndrome, SLE
- Salivary gland tumors, lymphoma
- Cervical lymphadenitis

- **Treatment:** Just supportive care.
- **Prevention:** Vaccination

Rubella

- Called 3 day measles or German measles.
- A single strand RNA virus of the family Togaviridae.
- Generally occurs in the spring.
- Humans are the only host.
- In unvaccinated population illness occurs in 5-14 year olds.
- In vaccinated population it occurs more commonly in teenagers and young adults.
- Vaccination and infection does not provide lifelong immunity.

Pathogenesis
Clinical Manifestations

- Rubella is a mild, often exanthemata's disease especially in infant and children not easily distinguishable from other viral illness.
- The incubation period is 14-21 days.
- It begins with a prodromal phase of low grade fever, sore throat with red eye. Subclinical infection is common, and 25-40% of children may not have rash.
- In children the first manifestation is usually the rash which is variable and not distinctive consist of small, irregular pink macules that coals, it begins on face and neck and spreads centrifugally to involve the torso and extremities it lasts for 3 days then it fades from the face as it extend to the rest of body so that the whole of the body may not involve at one time.
- About the time of onset of the rash tiny, rose colored lesions or petechial hemorrhages on the soft appears.
- Retro auricular, anterior cervical and occipital lymphadenopathy occurs.

Diagnosis

- Clinical presentation is nonspecific.
- Rubella IgM immunoglobulin enzyme immunosorbent assay.
- PCR; polymerase chain reaction.
- Viral culture

Complications

1. Thrombocytopenia; usually 2 wks after the onset of the rash, it is more common in children and in girls ,It is usually self-limited.
2. Arthritis; more common in adults especially women.
3. Encephalitis; 2 forms, post infectious syndrome it appears within 7 days following the appearance of the rash with headache, seizure, confusion ,coma with focal neurological signs and ataxia, most patient recover completely. A rear progressive rubella pan encephalitis (PRP) manifests years after the infection, it is slowly progressive neurodegenerative disease and death occurs after2-5 years.
5. Peripheral neuritis.

Differential Diagnosis:

- Mild measles, Roseola, Infectious mononucleosis, Drug eruption

Treatment:

- Supportive, no antiviral therapy is available for rubella.
**Congenital Rubella syndrome**

This is the most serious complication of rubella infection occur due to transplacental transmission of the virus from the mother to the fetus in first trimester. The most important risk factor for severe congenital defects is the stage of gestation at the time of infection. The risk of congenital defect is 90% if maternal infection occurs in the first trimester, while congenital defect is rare after 16 wk. of gestation even if fetal infection occur. Nearly all systems will affect.

- Cataract unilateral or bilateral is the most serious eye finding.
- Congenital heart disease occurs and the most common lesion is PDA.
- Mental retardation, microcephaly.
- Hearing loss is the most common finding
- IUGR is common with post natal growth retardation and short stature
- Late onset complications may occur like thyroid dysfunction, DM, PRP, autism with visual abnormalities.