Viral infections in children

Department of Pediatrics

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Measles

Measles:- is an acute viral infection caused by Measles virus which is an RNA virus of the genus Morbillivirus in the family Paramyxoviridae.

Transmission:-

Measles is highly contagious disease. The portal of entry of measles virus is through the respiratory tract or conjunctivae following contact with large droplets or small- droplet aerosols in which the virus is suspended. Patients **are infectious from 3 days before to up to 4-6 days after the onset of rash.**

Infants acquire immunity transplacentally from mothers. this immunity is usually complete for the first 4-6 month of life.

Measles

Clinical Manifestations :-

Measles has three clinical stages :-

1- An incubation period stage.

2- Prodromal stage.

3- Final stage with maculopapular rash.

The incubation period lasts about 8-12 days to the first prodromal symptoms.

The prodromal stage begins with a mild fever, followed by the onset of conjunctivitis with photophobia, coryza, a prominent cough, and increasing fever. **Koplik spots** represent the enanthem and are the pathognomonic sign of measles, appearing 1-4 days prior to the onset of the rash.

Measles

KOPLIK SPOTS :- are grayish white dotes, as small as grains of sands, that have reddish areolae, they tend to occur on the inner aspects of the cheeks at the level of premolars, but may spread over the rest of buccal mucosa. They disappear with in 12- 18 hr.

The conjunctival inflammation and photophobia may suggest measles before koplik spots appear.

Symptoms increase in intensity for 2-4 days until the first day of the rash. The temperature rises abruptly as the rash appears. **The rash** begins on the forehead, behind the ears , and on the upper neck as a red maculopapular eruption. It then spreads downward to the torso and extremities, reaching the palms and soles in up to 50% of cases. The exanthem frequently becomes confluent on the face and upper trunk. The rash fades over about 7 days in the same progression as it evolved, often leaving a fine desquamation of skin in its wake. The severity of disease is directly related to the extend and confluence of the rash.

Clinical manifestations of measles

- In more sever cases, generalized lymphadenopathy may be present, with cervical and occipital lymph nodes especially prominent.
- Otitis media ,bronchopneumonia and GIT symptoms such as diarrhea and vomiting are more common in infant and small children.

Differential diagnosis :-

1- Rubella

2- Roseola infantum

3- Scarlet fever.

4- Infectious mononucleosis.

5- meningococcemia.

6- Infection result from Adeno and enteroviruses.

7- Drug rash.

Measles

Diagnosis :-

Diagnosis is usually apparent from the characteristic clinical picture, laboratory confirmation is rarely needed.

The Lab investigations include :

1- Measurement of Measles IgM antibodies titer.

IgM antibody appears in serum 1-2 days after the onset of the rash and remains for about 1 month.

2- Isolation of measles virus from blood, urine, nasopharyngeal secretions .

3- WBC tends to be low , with lymphocytes decreased more than neutrophils.

Measles

Treatment :-.

Treatment is supportive by

1- anti pyretics (acetaminophen) for fever.
2- Bed rest.
3- Maintenance of adequate fluid intake.
4- Humidification may alleviate symptoms of laryngitis or irritating cough.
5- patient with photophobia should be protected from exposure to strong light.
6- Prophylactic antimicrobial therapy to prevent bacterial infection is not indicated.

7-Vitamin A therapy is indicated for all patients with measles , vitamin A should be administered once daily for 2 days at doses of 200,000 IU for children 12 mo of age or older.100,000 IU for infants 6 mo through 11 mo of age , and 50.000 IU for infants younger than 6 mo of age.

Measles

Complications :-

1- Otitis media.

2- Pneumonia: it is either interstitial pneumonia caused by measles virus(giant cell pneumonia) . Or it is caused by bacterial super infection (bronchopneumonia) with pneumococcus ,group A streptococcus or staph.

3- Croup,tracheitis,and bronchiolitis are common complications in infants.

4- Myocarditis.

5- Exacerbation of underlying mycobacterium tuberculosis.

6- Neurological complications ,the most common is **encephalitis** which occur 2-5 days after appearance of rash.

Measles

Complications of measles :-

Other neurological complications include

Guillain- Barre syndrome,hemiplegia and cerebralthrombophlebitis.

7- Sub acute sclerosing panencephalitis: It is a chronic encephalitis caused by persistent measles virus infection of the central nervous system.

Prevention :-

1-Patients should be isolated

2- Vaccine:- The initial measles immunization usually as measles – mumps-rubella (MMR) vaccine is recommended at 12- 15 mo of age . A second immunization is recommended at 4-6 yr.

Measles

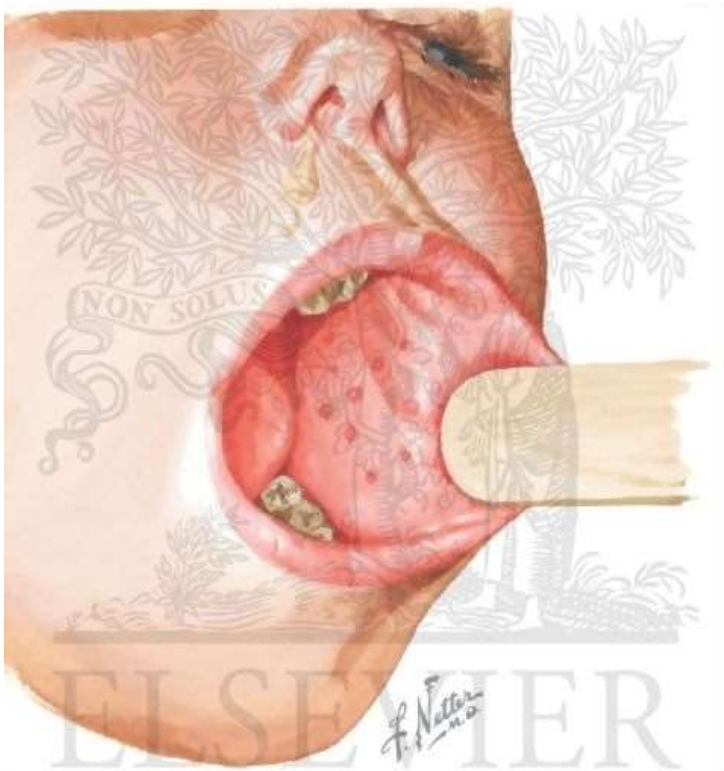
Prevention of measles :-

3- Post exposure prophylaxis :

Susceptible individuals exposed to measles may be protected from infection by either vaccine administration or immunization with immune globulin. The vaccine is effective in prevention or modification of measles if given within 72 hr of exposure. Immune globulin may be given up to 6 days after exposure to prevent or modify infection. Immunocompetent children should receive 0.25 ml/ kg intra muscularly, and immunocompromised children should receive 0.5ml/kg .

Immune globulin is indicated for susceptible household contacts of measles patients, especially infants younger than 6 months of age, pregnant women, and immunocompromised persons.

Measles (Koplik spots)



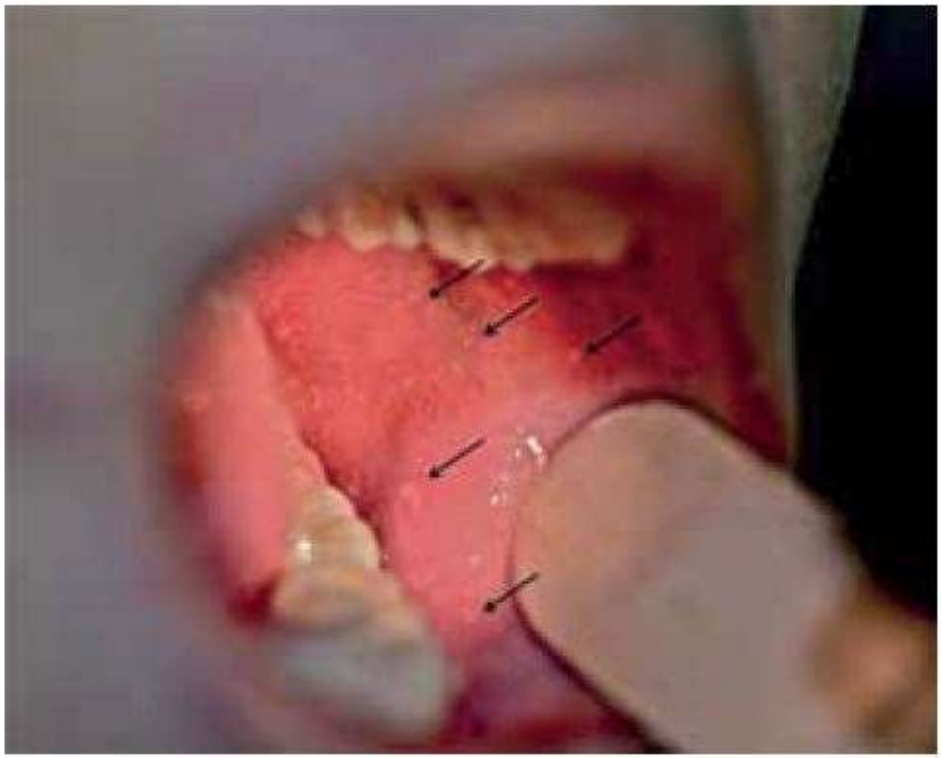
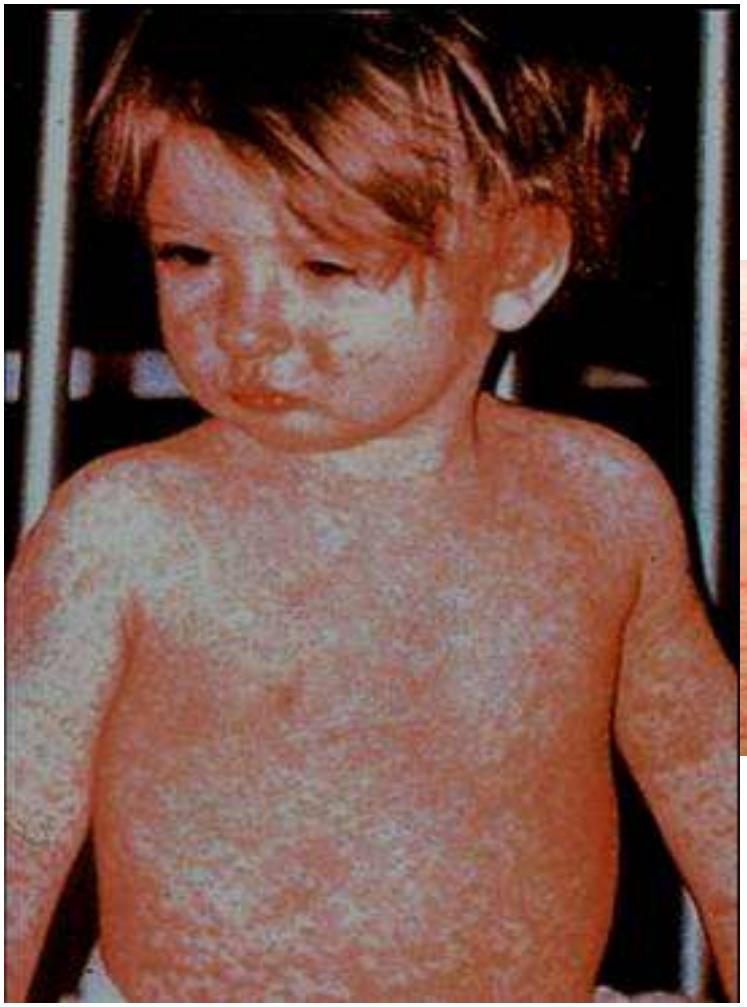
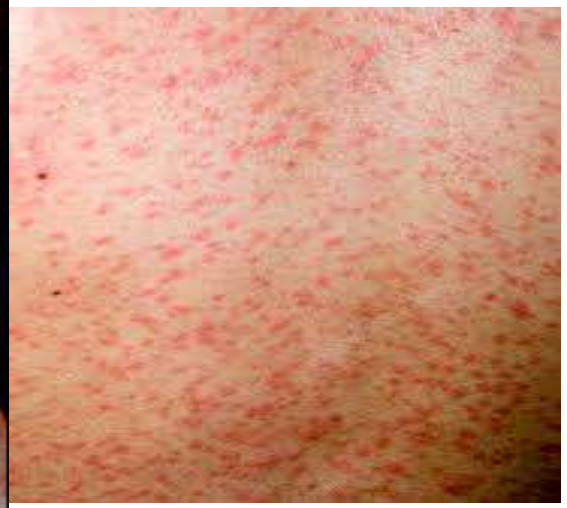


FIG 1. Koplik spots (arrows) presenting as bluish-white papules on the buccal mucosa opposite the molars

Measles rash





Rubella

Rubella (German or three day measles)

Etiology :-

Rubella virus, the cause of rubella is a single- stranded RNA virus of the genus Rubivirus in the family togaviridae.

Epidemiology :-

- Humans are the only natural host of rubella virus, which is spread either by respiratory droplet or transplacentally to the fetus causing congenital infection.
- The period of highest communicability is from 5 days before to 6 days following appearance of the rash.

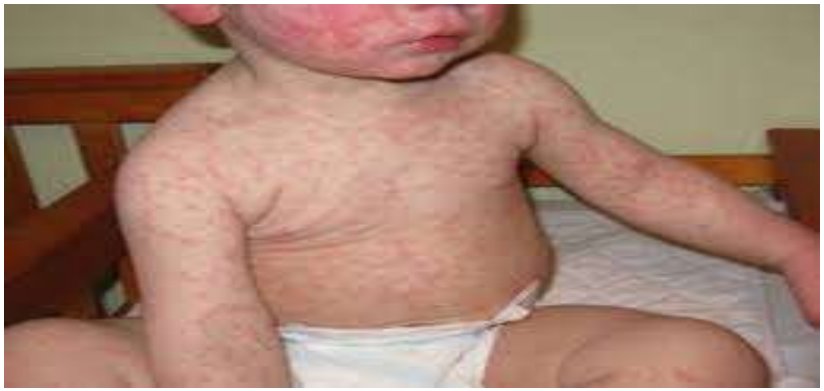
Rubella

Clinical manifestations :-

- The incubation period is 14- 21 days.
- The prodromal symptoms are mild. It consisting of low –grade fever, sore throat, red eyes, headache , malaise and anorexia.
- The most characteristic sign of rubella is retro auricular, post cervical and post occipital lymph adenopathy .
- The lymph nodes are tender and appear 24 hr before the rash start, they may remain for one week.
- An enanthem appears just before the onset of the rash .It consists of rose colored spots (Forchheimer spots)on the oropharynx, or petechial hemorrhages on the soft palate.
- The exanthem (the rash) begins on the face and spread quickly. It may be fade on the face by the time it appears on the trunk.

Rubella

- The rash is maculopapular with flushing.
- The rash spreads over the entire body with in 24 hr.During the second day the rash assume a pinpoint appearance, mild itching may occur. The rash usually clears by third day .
- There is no photophobia , fever is low grade during the rash and persist for 1-3 days .The spleen is often slightly enlarged. In older girls polyarthritis with arthralgia may occur .



Rubella

Diagnosis of rubella :-

A specific diagnosis of rubella is important for epidemiologic reasons, for diagnosis of infection in pregnant women, and for confirmation of diagnosis of congenital rubella.

1- Leukopenia, neutropenia, and mild thrombocytopenia.

2- The most common diagnostic test is rubella immunoglobulin (Ig) M enzyme immunosorbent assay.

3-IgM capture assay, reverse transcriptase polymerase chain reaction test , or viral culture should be performed for confirmation of diagnosis in congenital infection.

Rubella

Differential Diagnosis:-

1- Measles

2- Roseola infantum.

3- Scarlet fever.

4- Infectious mono nucleosis.

5- Drug rash .

Complications :-

1- Encephalitis.

2- Thrombocytopenic purpura.

3- Arthritis.

4- Progressive rubella pan encephalitis.

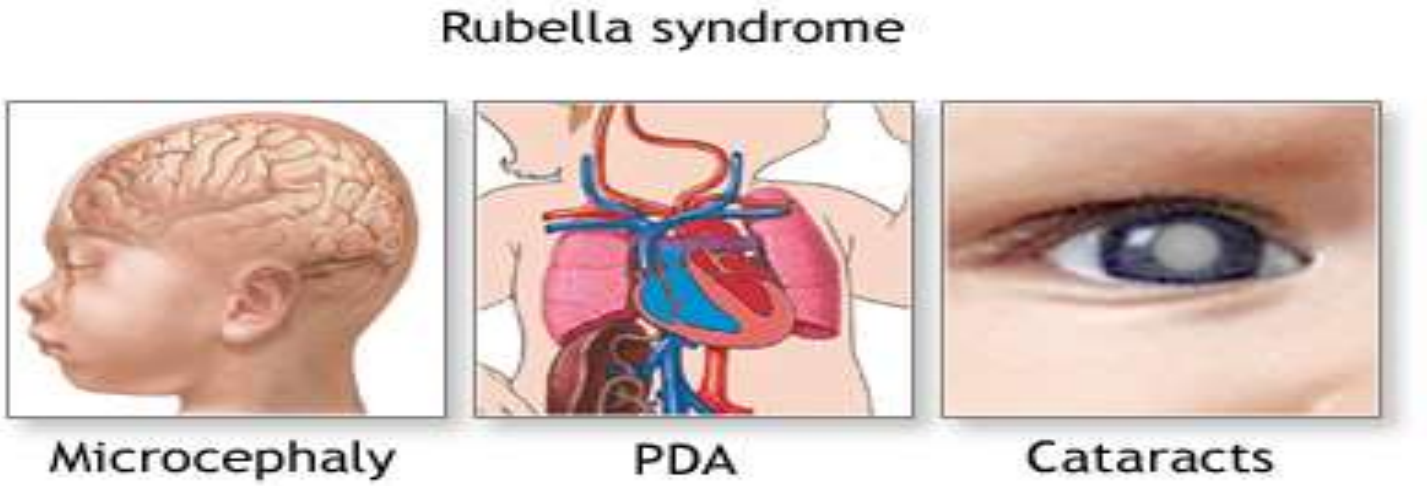
Rubella

Congenital rubella syndrome :- it affects all organ systems, the most common manifestation is intrauterine growth retardation. Other common findings include salt and pepper retinopathy which are the most common ocular abnormality, cataract, and microphthalmia. Myocarditis, structural cardiac defect(**patent ductus arteriosus** which is the most frequently reported cardiac defect, or pulmonary artery stenosis). Sensorineural deafness, meningoencephalitis and blueberry muffin skin lesion. Persistent infection lead to pneumonia, hepatitis, anemia and thrombocytopenic purpura. Patient may have enlargement of liver and spleen. Later sequelae include motor and mental retardation.

Congenital rubella syndrome

Systems	Findings
Ophthalmology	Cataracts, congenital glaucoma, microphthalmos, pigmentation retinopathy
Cardiac	Patent ductus arteriosus, peripheral pulmonary artery stenosis
Hematology	Thrombocytopenia, hemolytic anemia, petechiae/purpura, dermal erythropoiesis causing blueberry muffin rash
Neurology	Behavioral disorders, meningoencephalitis, microcephaly, mental retardation, autism,
Hearing	Sensorineural hearing loss

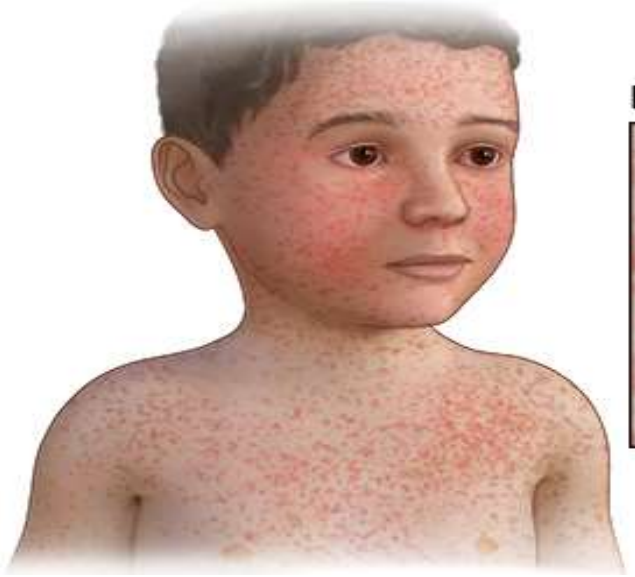
Congenital rubella syndrome

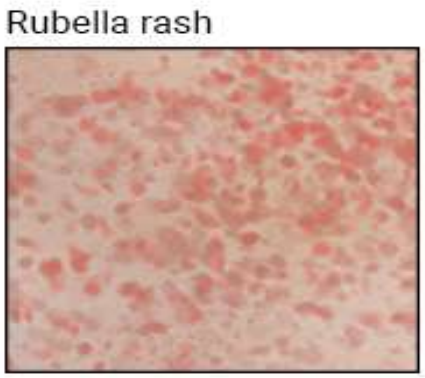


A.D.A.M.

Rubella

Treatment :- There is no specific treatment available for either acquired rubella or CRS.Postnatal rubella is a mild illness require antipyretics and analgesics. Management of children with CRS is more complex and requires pediatrics, cardiac, audiologic, ophthalmologic, and neurologic evaluation and follow-up.





Rubella

Prevention of rubella :-

The period of highest communicability is from 5days before the rash to 6 days following appearance of the rash.

- **P**atients with postnatal infection should be isolated from susceptible individuals for 7 days after onset of the rash.
- Prevention of rubella is by vaccination.

The initial rubella immunization, usually as measles – mumps rubella (MMR) vaccine .It is recommended at (12 -15 mo) of age .A second immunization also as MMR is recommended at age 4-6 yr .children who have not received this second dose should be immunized by 11-12 yr of age .

Mumps

Etiology :-

-Mumps is an acute self-limited infection.

-Mumps virus is in the family Paramyxoviridae and the genus Rubulavirus.It is a single –stranded pleomorphic RNA virus.

-Mumps occurred in young children between the age of 5-9 years.

Epidemiology :-

The virus is spread from person to others by respiratory droplets. Maximum transmission of the virus occur 1-2 days before the appearance of the swelling until 5 days after parotid swelling.

Mumps infection occurred more often in the winter and spring months.

Clinical Manifestations :-

- The incubation period is (12- 25) days.
- About 30- 40 % of infection is sub clinical.

Clinical manifestations of mumps

- In children prodromal manifestations lasting 1-2 days and consisting of fever, headache and pain in the neck.
- The involvement of salivary glands are characterized by pain and swelling in one or both parotid glands.
- The parotid swelling first fills the space between the post boarder of the mandible and the mastoid and then extends down ward and fore ward, the swollen tissues push the ear lobe up ward and outward .
- The swollen area is tender and painful, pain being elicited by testing sour liquids such as lemon juice.
- Edema of the homolateral pharynx and soft palate may occur with displacement of the tonsil medially.

Clinical manifestations of mumps

- One parotid gland usually swells a day or two before the other. The opening of the Stensen duct mat be red and edematous.
- The parotid swelling peaks in approximately 3 days then gradually subsides over 7 days.
- Although the parotid glands alone are affected but swelling of submandibular glands and less commonly sublingual glands may occur.

Diagnosis :-

The diagnosis of mumps is usually apparent from the clinical symptoms and examination. routine laboratory tests are non specific , usually leucopenia is present with relative lymphocytosis. An elevation of serum amylase level is common .

Mumps

Diagnosis :-

The microbiologic study include:

- Serology :- Enzyme immunoassay for mumps immunoglobulin IgG, IgM antibodies are commonly used for diagnosis.
- Isolation of the virus in cell culture, detection of viral antigen by direct immunofluorescence , or identification of nucleic acid by reverse transcriptase polymerase chain reaction.

Virus can be isolated from upper respiratory tract secretions, CSF, or urine during the acute illness.

D. diagnosis :-

1- Other viral causes of parotitis ,include HIV, influenza, parainfluenza viruses.

2- Acute suppurative parotitis by staphylococcus aureus.

3- Salivary calculus obstruction.

Mumps

Complications :-

1- Meningoencephalitis :- it is the most common complications ,the cerebrospinal fluid examination may show increase cell count (less 500 cells / mm) mainly lymphocyte, protein 50-200 mg/dl. Glucose usually normal but may decrease to 40 mg/dl.

2- Orchitis and Epididymitis :- it is usually follows parotitis with in 8 days. The onset is abrupt with fever ,nausea and lower abdominal pain, the affected testis become swollen and tender.

3- Pancreatitis.

4- Myocarditis.

5- Arthritis.

6- Thyroiditis.

7- Deafness

9- Oophoritis.

Mumps

Treatment :-

- There is no specific antiviral therapy , treatment is supportive.

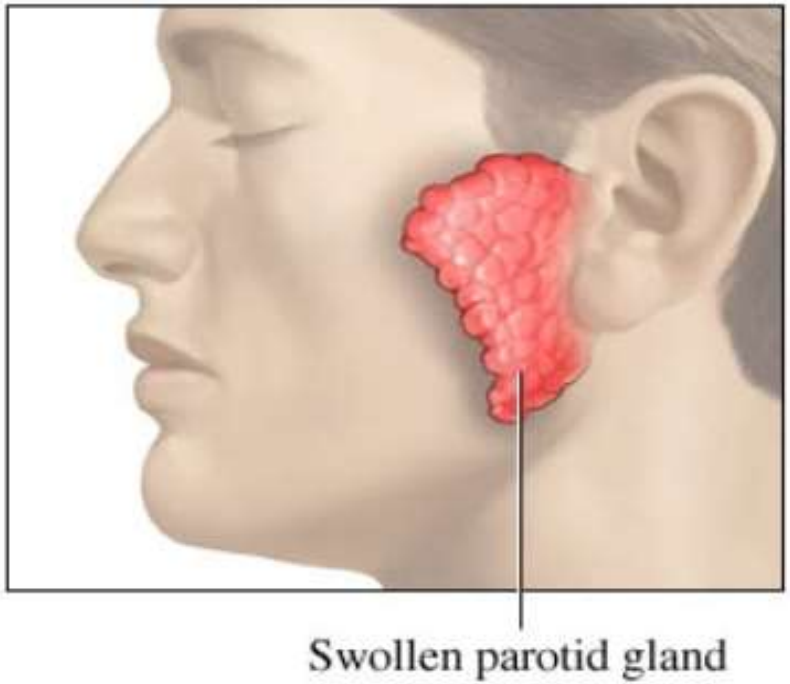
- Antipyretics are indicated for fever.

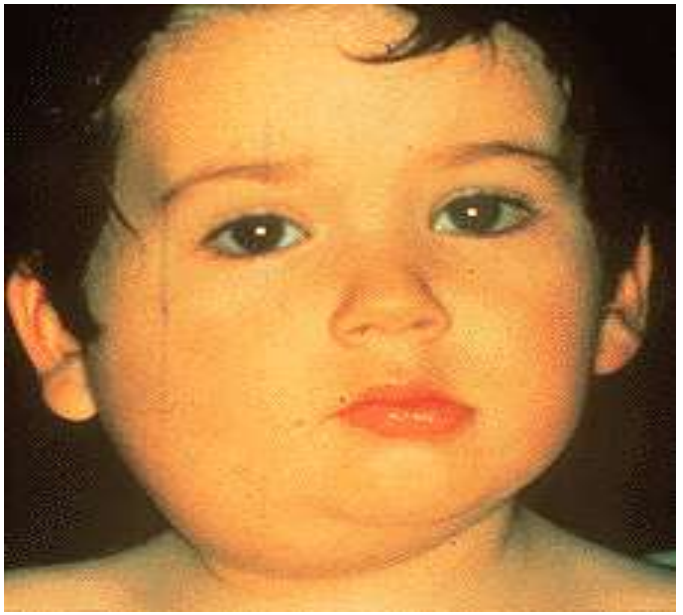
- Bed rest.

- The diet should be adjusted to the patient ability to chew.

- Orchitis should be treated with local support and bed rest.

Mumps





Varicella

Varicella – zoster virus (VZV) causes primary, latent, and recurrent infections.

The primary infection is manifested as **Varicella (chickenpox)** and result in establishment of a lifelong latent infection of sensory ganglion neurons .Reactivation of the latent infection causes herpes zoster **(shingles).**

Herpes zoster because it is due to the reactivation of latent VZV so it is uncommon in childhood .

Epidemiology :-

Patients with varicella are contagious from 24- 48 hr before the rash appears and until the vesicles are crusted , usually 3-7 days after onset of rash.

Varicella

VZV is transmitted in respiratory secretions and in the fluid of skin lesions either by airborne spread or through direct contact.

Clinical Manifestations :-

- The incubation period range from 10- 21 days.
- Prodromal symptoms include fever, malaise, anorexia , headache , and occasionally mild abdominal pain may occur 24-48 hr before the rash appears.
- Varicella lesions appear first on the scalp ,face or trunk. The initial exanthem consists of intensely pruritic erythematous macules that evolve through the papular stage to form clear fluid-filled vesicles. While the initial lesions are crusting , new crops form on the trunk and then the extremities.

Varicella

- The simultaneous presence of lesions in various stages of evolution is characteristic of varicella.
- The distribution of the rash is predominantly central . Many children have vesicular lesions on the eyelids and conjunctivae, but corneal involvement is rare. Hypopigmentations or hyper pigmentation of lesions sites persist for days or weeks but sever scaring is unusual unless the lesions were secondary infected. Ulcerative lesions involving the oropharynx are also common.

Herpes zoster is caused by the reactivation of latent VZV; It is not common in childhood. It is manifested as vesicular lesions clustered within one or two adjacent dermatomes. Zoster in children is frequently associated with localized pain, hyperesthesia, pruritus, low grade fever, the rash is mild. Symptoms of acute neuritis are minimal and complete resolution occurs within 1-2 weeks.

Varicella

Differential diagnosis :-

1- vesicular lesions by herpes simplex virus.

2- staphylococcus aureus skin lesion.

3- contact dermatitis.

4- drug rash.

5- insect bites.

Diagnosis :

Diagnosis is usually depend on clinical examination and lab evaluation is only important in high risk patients.

1- VZV can be identified by direct fluorescence assay (DFA) of cell from cutaneous lesion and by PCR amplification testing .

2- virus can be recovered by using tissue culture methods.

Varicella

3- VZV immunoglobulin (IgG) can be detected , and 4- folds rise of the IgG level confirm the diagnosis of acute infection.

4- Leukopenia is typical during the first 72 hr after onset of rash; it is followed by a relative and absolute lymphocytosis.

Complications of varicella:-

1- **Secondary bacterial infections of the skin** usually by streptococcus and staphylococcus.

2- **Encephalitis and cerebellar ataxia**.

Neurological symptoms usually begin 2-6 days after the onset of the rash but may occur during the incubation period or after resolution of the rash.

Varicella

Complications of varicella :-

3- **pneumonia .** Varicella pneumonia is a sever complication . The respiratory symptoms of cough, dyspnea, cyanosis and pleuritic chest pain usually begins within 1-6 days after the onset of the rash.

4- **Progressive varicella** : with visceral organ involvement , coagulopathy, sever hemorrhage, and continued vesicular lesion development after 7 days, is a sever complication of primary VZV infection.

5- Hepatitis.

6- Thrombocytopenia.

7- Other rare complications include nephritis, nephrotic syndrome,arithritis, myocarditis, pericarditis, pancreatitis and orchitis.

Varicella

Treatment :-

Symptomatic treatment of varicella includes antipyretics, cool baths, and careful hygiene. Routine oral administration of antiviral drug (acyclovir) is not recommended in otherwise healthy children with varicella.

Acyclovir or valacyclovir may be considered in those at risk of sever varicella such as unvaccinated persons older than 12 years, those with chronic cutaneous or pulmonary disease; receiving short course, intermittent or aerosolized corticosteroids; or receiving long term salicylate therapy. Early therapy with antiviral in immunocompromised persons is effective in preventing sever complications.

Varicella

Prevention:-

1-isolation of infected patient.

2- Vaccine : Varicella is a vaccine –preventable disease. Varicella vaccine contains live, attenuated VZV and is indicated for subcutaneous administration, in 2 doses at age 12-15 mo and 4-6 yr.

3- post exposure prophylaxis:- varicella–zoster immunoglobulin (VZIG) post exposure prophylaxis is recommended for immunocompromised patient, pregnant women and new-born exposed to maternal varicella .

Varicella

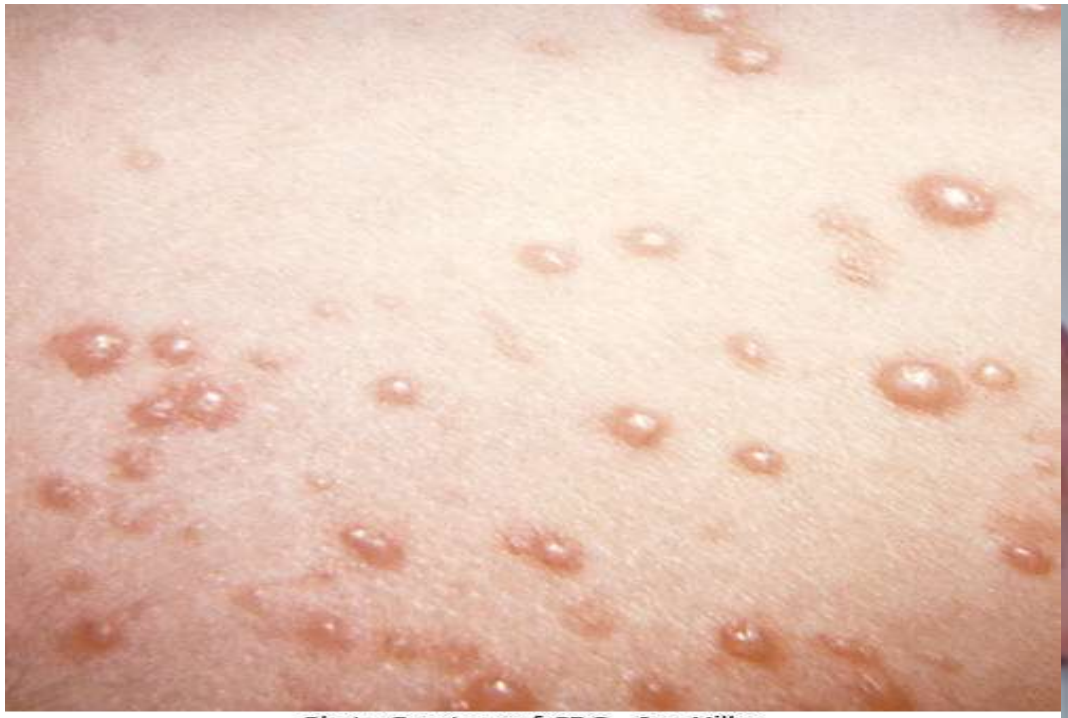
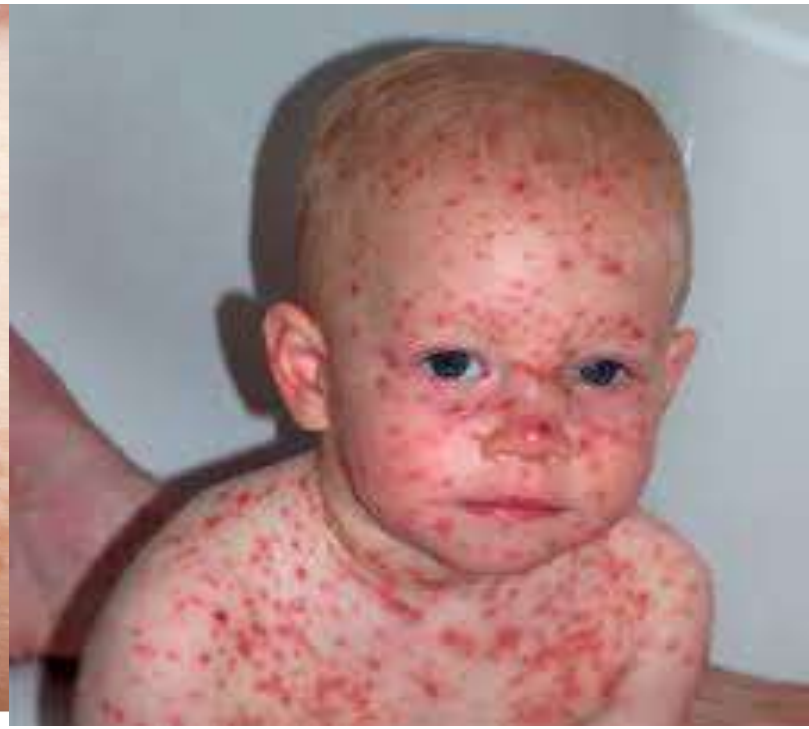


Photo Courtesy of CDC - Joe Miller



Roseola

Roseola infantum (exanthema subitum or sixth disease)

Etiology :- it is caused by human herpes virus-6 (HHV-6 and less frequently by human herpes virus -7 (HHV-7)

Epidemiology:-

Roseola can develop in children year around but higher incidence occur during spring and fall months. Peak acquisition of primary HHV-6 infection from 6-15 month of age.

Most adult excrete HHV-6 in saliva and may serve as primary sources for viral transmission to children. There is evidence that HHV-6 can be transmitted in utero but this is rare and no malformations have been noted.

Roseola

Clinical Manifestations :-

- Roseola is a mild febrile illness occurring during infancy with peak at 6- 15 month of age. Transplacental antibodies protect most infant until 6 month of age.
- The incubation period about (5- 15)days.
- The prodromal period include mild rhinorrhea, slight pharyngeal inflammation, mild conjunctival redness and mild cervical or occipital lymphadenopathy.
- Patient usually develops high fever, the temperature range from (37.9-40c) Some children may become irritable and anorexic . **Seizures** may occur in 5-10 % of children during this febrile period.
- Fever persists for 3-5 days, and then typically resolves abruptly or gradually diminished over 24-36 hr.A rash appears with in 12-24 hr of fever resolutions.

Roseola

- The Roseola rash begins as discrete ,small, slightly raised pink lesions on the trunk then spreads to neck, face and proximal extremities, the rash is not pruritic. The rash of roseola is rose colored , as the name implies.
- After 1-3 days the rash fades.

Diagnosis :-

The most important reasons for establishing the diagnosis of Roseola is to differentiate this mild illness from other serious rash like measles. The diagnosis usually depends on history and clinical findings.

-**Laboratory findings include** :- during the period of exanthem the WBC decrease with relative lymphocytosis.

-Specific tests for diagnosis of HHV-6 infection include serology, virus culture, antigen detection, and polymerase chain reaction(PCR).

Roseola

D.Diagnosis :-

1- Measles :- the appearance of rash in measles occur with the height of fever , as well as the presence of cough, conjunctivitis, and koplik spots.

2- Rubella.

3- Scarlet fever.

4- Enteroviruses.

5- Drug hypersensitivity.

Treatment :-

1- Supportive treatment in form of acetaminophen, for fever, and adequate fluid intake.

Roseola

Treatment :-

2- Referral to hospital should be considered in those with serious complications like encephalitis, pneumonia, and hepatitis.

3- The generally benign nature of Roseola precludes utilization of antiviral therapy, but this therapy may be considered in children with neurological complication, and immunocompromised patient.HHV-6 infection inhibited by ganciclover, cidofovir, and foscarnet.

Complications:-

1- **Convulsions** are the most common complications of roseola and are recognized in up to one third of patients.

Roseola

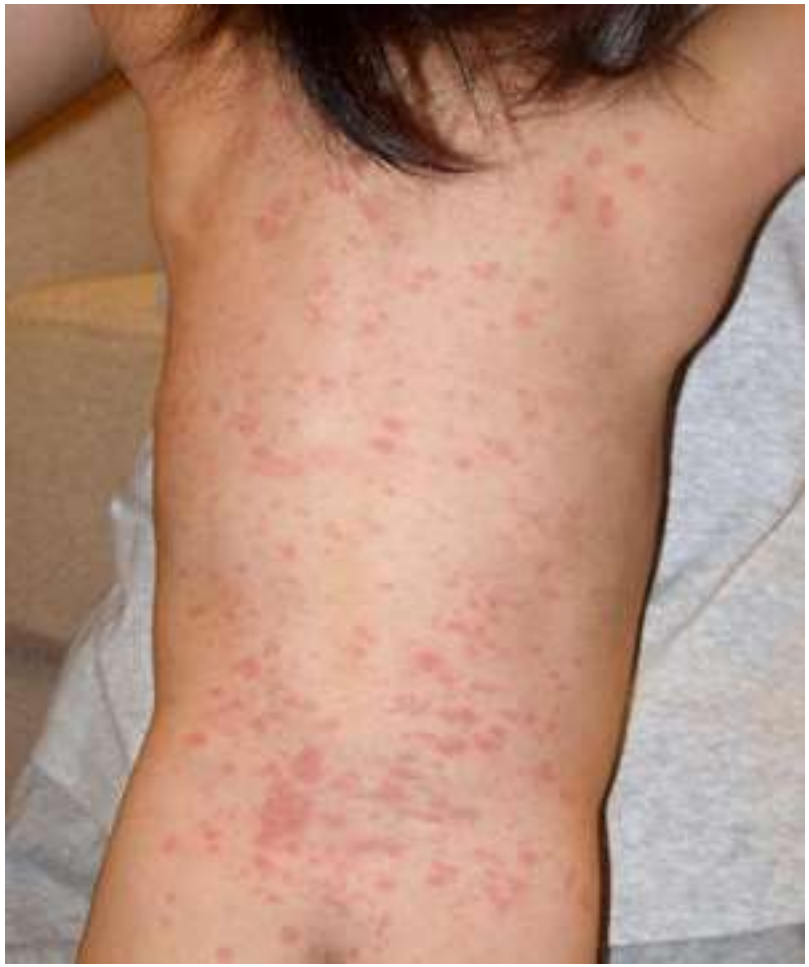
Complications :-

2- Encephalitis

3- Hepatitis

4- Myocarditis

5- Pneumonia



Infectious mononucleosis (glandular fever)

- Epstein- Barr virus (EBV) causes more than 90% of infectious mononucleosis cases.
- Among children, transmission may occur by exchange of saliva from child to child.

Clinical Manifestations :-

The incubation period of IMN in adolescents is 30-50 days. In children it may be shorter. The majority of cases of EBV infection in infants and young children are clinically silent.

Patients may complain of malaise, fever, fatigue, headache, sore throat, abdominal pain and myalgia.This prodromal manifestations may last 1-2wk

The complaints of sore throat and fever gradually increase until patients seek medical care.

Infectious mononucleosis

Clinical Manifestations :-

The physical examination is characterized by generalized lymphadenopathy, splenomegaly and hepatomegaly. Lymphadenopathy occurs most commonly in the anterior and posterior cervical lymph nodes.Epitrochlear lymphadenopathy is suggestive of IMN.Splenomegaly to 2-3 cm below costal margin is typical . Massive enlargement is uncommon. Symptomatic hepatitis or jaundice is uncommon. The sore throat is often associated with pharyngitis, tonsillar enlargement occasionally with exudates.Petechiae at the junction of the hard and soft palate are frequently seen.

Other clinical findings may includes skin rash and edema of the eyelids.

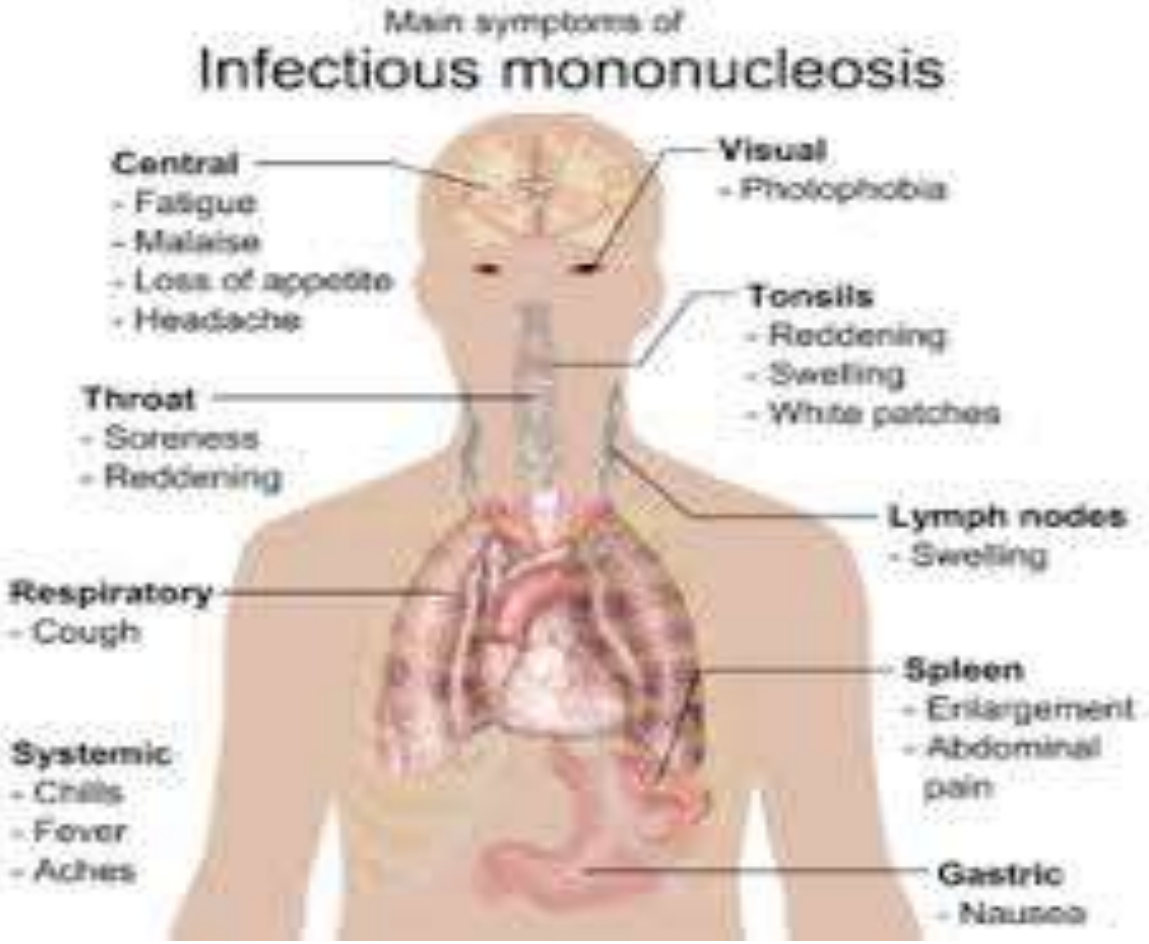
Infectious mononucleosis

Clinical manifestations:-

The rash is maculopapular and reported in 3-15% of patients. Up to 80% of patients of IMN develop ampicillin rash if treated with ampicillin or amoxicillin . This rash is immune mediated and resolve with out treatment. The major symptoms typically last 2-4 weeks.

EBV was the first human virus to be associated with malignancy (nasopharyngeal carcinoma, Burkitt lymphoma, Hodgkin disease, lymphoproliferative disorders, and leiomyosarcoma in immunodeficient states).

Infectious mononucleosis



Infectious mononucleosis

Diagnosis :-

A presumptive diagnosis can be made by the presence of typical clinical symptoms with atypical lymphocytosis in the peripheral blood. The diagnosis can be confirmed by serologic testing.

1- **Routine laboratory tests:-**

C.B.P reveals leukocytosis of 10000-20000cells/mm3. of which at least two third are lymphocytes. Atypical lymphocytes account for 20-40%of the total number.

The atypical cells are mature T-lymphocytes that have been antigenically activated. Compared with regular lymphocytes microscopically , atypical lymphocytes are larger overall, have larger eccentrically placed nuclei with lower nuclear to cytoplasm ratio. Platelet count may decrease.

Mild elevation of hepatic transaminase occur in 50% of the cases.

Infectious mononucleosis

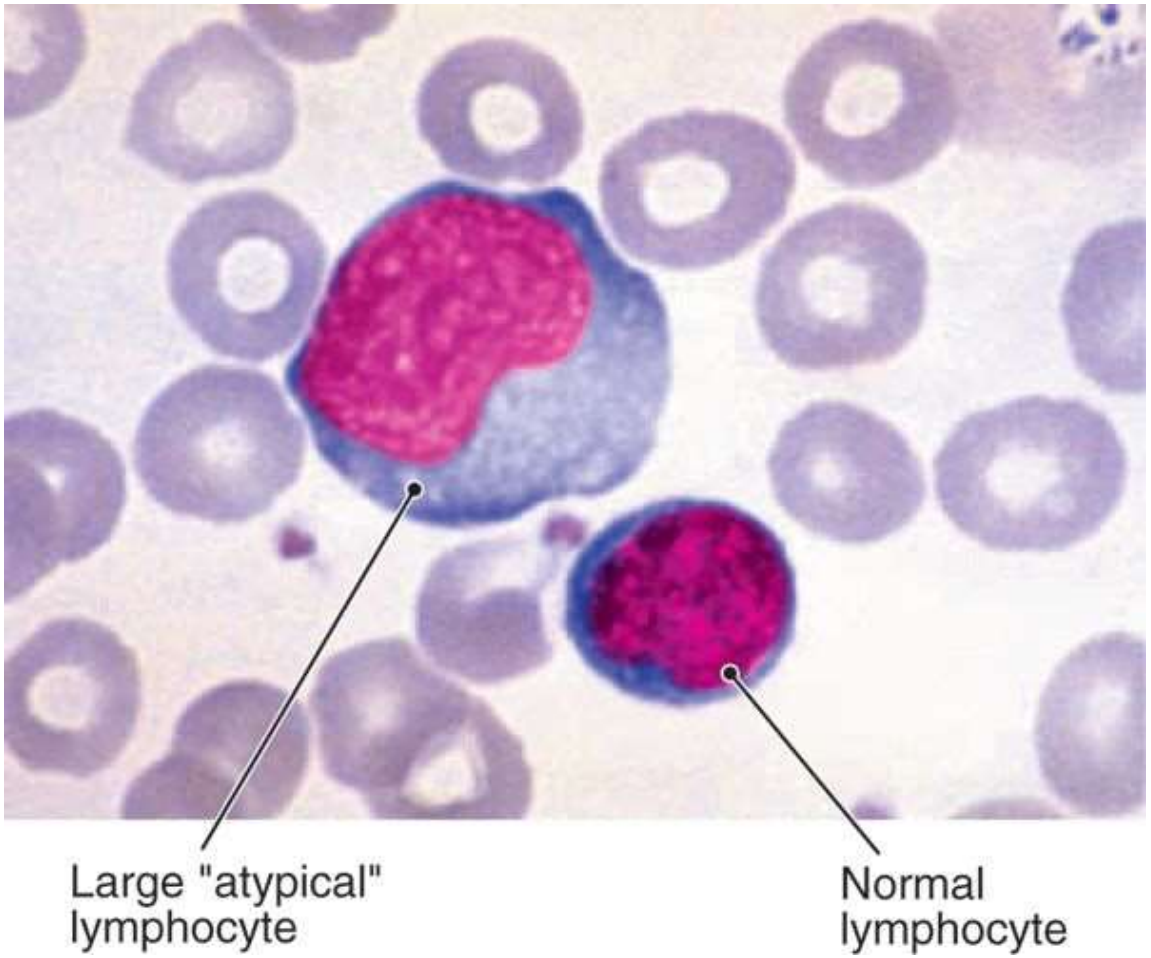
Diagnosis :-

2-The diagnosis is usually confirmed by serologic testing , either for heterophile antibody (IgM antibodies) or specific EBV antibodies

EBV specific antibodies testing is useful to confirm acute and past infection.

The EBNA (nuclear antigen), EA (early antigen) , and VCA (viral capsid antigen), these are most useful for diagnosis. The detection of IgM antibody to VCA is the most valuable and specific serologic test for the diagnosis of acute infection.

Infectious mononucleosis



Infectious mononucleosis

Treatment :-

1- therapy with high dose of acyclovir decrease viral replication and oropharyngeal shedding but not reduce the severity or duration of symptoms.

2- Bed rest and symptomatic therapy are the mainstays of management.

3- Because blunt abdominal trauma may predispose patients to splenic rupture so it is important to advise against participation in contact sports and strenuous athletic activities during the first 2-3 wk of illness.

4- Short courses of corticosteroids (less than 2 wk) may be helpful for complications of IMN.

Infectious mononucleosis

Treatment:-

The indications for steroid therapy include :-

A- incipient upper air way obstruction.

B- thrombocytopenia

C- autoimmune hemolytic anemia.

D- seizures and meningitis.

A recommended dose is prednisone 1mg/ kg / 24 hr for 7 days and tapered over another 7 days.

Infectious mononucleosis

Complications :-

1- The most feared complication is sub- capsular splenic hemorrhage or splenic rupture, which occur most frequently during the second week of the disease.

2- Swelling of the tonsils and oropharyngeal lymphoid tissue may cause air way obstruction

3- Neurological complications :- include headache, seizures, ataxia,meningitis,encephalitis and facial nerve palsy.

4- Hemolytic anemia.

5- A plastic anemia.

6- Thrombocytopenia and neutropenia.

7- Myocarditis.

8- Interstitial pneumonia.

9- Other rare complications include Pancreatitis, parotitis and Orchitis.

Polioviruses

Etiology :- polioviruses are RNA viruses belong to the bicornaviridea family in the genus Enterovirus and include three serotypes (type 1,2,3). Polioviruses spread from the intestinal tract to the central nervous system, where they cause aseptic meningitis and poliomyelitis or polio.

Transmission :-

Humans are the only known reservoir for the viruses. Polio virus has been isolated from the feces more than two weeks before paralysis to several weeks after the onset of symptoms.

The disease is spread by contaminated feces (fecal- oral spread).

Polio

Pathogenesis :-

Poliovirus enters the body via the oral route , it multiply in the tonsillopharyngeal tissue then it pass to regional lymph nodes.

The virus reach the to the intestinal tract and multiply there. It pass to blood stream then it reach to the CNS,the main pathological lesion in the CNS involve motor neuron cells in the spinal cord (the anterior horn cells) and the medulla oblongata (the cranial nerve nuclei).

Clinical Manifestations :-

The incubation period of poliovirus is 8-12 days. with range of(5-35 days).

Polio

Clinical manifestations:-

Poliovirus infections may follow one of several courses :-

1- in apparent infection. 2- abortive polio.

3- non paralytic polio. 4- paralytic polio.

1- in apparent infection :- occurs in 90-95% of cases and causes no disease and no sequelae.

2- abortive poliomyelitis :- occurs in 5% of cases. The patient develops fever, malaise, anorexia and headache, there may be sore throat, abdominal pain and vomiting. The physical examination is normal, recovery is complete. The duration of illness is 2-3 days.

Polio

Clinical Manifestations :-

3- Non paralytic polio :- occurs in 1% of cases, signs of abortive poliomyelitis are presented and as well as pain and stiffness of the posterior muscle of the neck, trunk , and limbs.

4- Paralytic Polio:- develops in 0.1% of cases, causing 3 clinically recognizable syndromes. These are

A- spinal paralytic polio. B- bulbar polio. C- polio encephalitis.

A- spinal paralytic polio:- may occur as the 2nd phase of a biphasic illness, the 1st phase of which corresponds to abortive polio. The patient then appears to recover and feels better for 2-5 days, after which sever headache and fever occur with exacerbation of the previous systemic symptoms. Sever muscle pain is present , and sensory and motor phenomena (e.g., paresthesia,hyperesthesia, fasciculation and spasms) may develop .

Spinal paralytic Polio

Within 1-2 days , **asymmetric** flaccid paralysis or paresis occur. Involvement of one leg is most common, followed by involvement of one arm. Initially hyperactive deep tendon reflexes (for a short period) followed by absent or diminished reflexes. In the spinal form, there is weakness of muscle of the neck, abdomen , trunk, diaphragm and thorax. Sensation is intact. Paralysis of the lower limbs is often accompanied by bowel and bladder dysfunction.

- The return of strength and reflexes is slow and may continue to improve as long as 18 months after the acute disease.
- Lack of improvement from paralysis with in the first several weeks or months (about 6 months) after onset is usually evidence of permanent paralysis.

Polio

B-Bulbar polio :-

It may occur as a clinical entity with out apparent involvement of the spinal cord.

The clinical findings seen with bulbar polio include :-

1- nasal twang to the voice caused by palatal and pharyngeal weakness.

2- inability to swallow smoothly.

3- accumulation of pharyngeal secretions which may cause irregular respiration.

4- absence of effective coughing.

5- nasal regurgitation of saliva and fluids as a result of palatal paralysis.

6- involvement of vital centers in the medulla which are manifested by irregularity in rate, and rhythm of respiration. and cardiovascular alteration include increase in blood pressure. and cardiac arrhythmias.

7- paralysis of one or both vocal cords causing hoarseness of voice , aphonia, and asphyxia.

Polio

C- Polio encephalitis :- it is a rare form of the disease in which higher centers of the brain are severely involved. Irritability , seizure, coma, and spastic paralysis, with increase reflexes may be observed. Peripheral and cranial nerves paralysis may occur . Hypoxia and hypercapnia may caused by inadequate ventilation.

Differential Diagnosis :-

Differential diagnosis of Acute Flaccid Paralysis include:

- Neurotropic viruses(rabies virus, varicella-zoster virus and Japanese encephalitis virus).
- Guillain-Barre syndrome.

Polio

- ❖ Acute traumatic sciatic neuritis.
- ❖ Diseases of the neuromuscular junction (myasthenia gravis).
- ❖ Metabolic disorders (hypokalemic periodic paralysis).

Diagnosis :-

- In suspected cases of acute flaccid paralysis 2 stool specimens should be collected 24 -48 hr apart if the diagnosis of polio is suspected. Polio virus can be isolated from stool sample especially during the first week after the onset of paralysis.
- The CSF is normal during minor illness but with CNS involvement there is pleocytosis between 20-300 cells mainly lymphocytes. Protein is elevated to 50- 100mg/dl.
- Serologic test demonstrate increase in antibody titer during acute phase of illness and 3-6 wk later.

Polio

Treatment:-

There is no specific antiviral treatment for poliomyelitis and the management is supportive.

Abortive polio : supportive treatment with analgesics, sedative, good nutrition, and bed rest.

Nonparalytic polio treatment is similar to that for abortive form.

Paralytic poliomyelitis : most patient require hospitalization with complete physical rest.

- Suitable body alignment is necessary to prevent skeletal deformity.
- Moist hot packs may relieve muscle pain and spasm.
- A proper diet and high fluid intake should be started.

Polio

- If permanent paralysis of the limb occur this may need a skilled orthopedic advice. and regular physiotherapy.

Treatment of Bulbar poliomyelitis:

1- maintain the air way and avoid all risk of inhalation of saliva, food and vomitus .

2- frequent suction of nasopharyngeal secretions.

3- intravenous infusion of fluid and electrolytes.

4- close observation for respiratory insufficiency and frequent monitoring of blood pressure.

5- some patients may need tracheostomy because of vocal cord paralysis.

6- mechanical ventilation may be needed.

Polio

Prevention :-

1- proper hygienic measures.

2- Vaccination :- both the inactivated polio vaccine (IPV) and the live attenuated orally administered polio vaccine (OPV) have established efficacy in preventing poliovirus infection.

Polio