

Measles

*Professor Dr.Dawood Al-Azzawi

Measles is highly contagious. Due to widespread vaccination, endemic transmission has been interrupted . In some areas of the world, measles remains a serious threat to children.

Etiology

Measles virus is a single-stranded lipid enveloped RNA virus in the family Paramyxoviridae and genus Morbillivirus. humans are the only host of measles virus...

Epidemiology;

The measles vaccine has changed the epidemiology of measles dramatically. Morbidity and mortality associated with measles decreased prior to the introduction of the vaccine due to improved health care and nutrition. The attack rate fell from 313 cases/100,000 population in 1956–60 to 1.3 cases/100,000 in 1982–88.

The portal of entry of measles virus is through the respiratory tract or conjunctivae.

Patients are infectious from 3 days before the rash up to 4–6 days after its onset.

Approximately 90% of the exposed susceptible individuals develop measles. Secondary cases have been reported in physicians' offices and in hospitals by spread of aerosolized virus.

Pathology and pathogenesis;

Measles infection causes necrosis of the respiratory tract epithelium and an accompanying lymphocytic infiltrate. Measles produces a small vessel vasculitis on the skin and on the oral mucous membranes.

Measles consists of 4 phases: incubation period, prodromal illness, exanthematous phase, and recovery. During incubation, measles virus migrates to regional lymph nodes. A primary viremia ensues that disseminates the virus to the reticuloendothelial system. A secondary viremia spreads virus to body surfaces. The prodromal illness begins following the secondary viremia and is associated with epithelial necrosis and giant cell formation in body tissues.

Clinical manifestation;

Measles is a serious infection characterized by high fever, an enanthem, cough, coryza, conjunctivitis, and a prominent exanthem. After an incubation period of 8–12 days, the prodromal phase begins with a mild fever followed by the onset of conjunctivitis with photophobia, coryza, a prominent cough and increasing fever. The enanthem, **Koplik spots**, is the pathognomonic sign of measles and appears 1 to 4 days prior to the onset of the rash. They first 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. They may spread to involve the lips, hard palate, and gingiva. They also may occur in conjunctival folds and in the vaginal mucosa. Koplik spots have been reported in 50–70% of measles cases but probably occur in the great majority.

Symptoms increase in intensity for 2–4 days until the 1st day of the rash. The rash begins around the forehead (around the hairline), 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.

*Consultant Pediatrician CABP DCH MBChB
Diyala Medical College

The exanthem frequently becomes confluent on the face and upper trunk. With the onset of the rash, symptoms begin to subside and the rash fades over about 7 days in the same progression as it evolved, often leaving a fine desquamation of skin in its wake. Of the major symptoms of measles, the cough lasts the longest, often up to 10 days. In more severe cases, generalized lymphadenopathy may be present, with cervical and occipital lymph nodes especially prominent

In individuals with passively acquired antibody, such as infants or recipients of blood products, a **subclinical form of measles may occur**. The rash may be indistinct, brief, or, rarely, entirely absent. Likewise, some individuals who have received vaccine when exposed to measles may develop a rash but few other symptoms. Persons with inapparent or subclinical measles do not shed measles virus and do not transmit infection to household contacts.

Children who had received the original formalin-inactivated measles vaccine at times developed a more severe form of disease called **atypical measles**. Patients had onset of high fever and headache followed by the appearance of a maculopapular rash on the extremities that become petechial and purpuric and progressed in a centripetal direction. The illness was frequently complicated by pneumonia and pleural effusions. It is thought that atypical measles was caused by development of circulating immune complexes that formed due to an abnormal immune response to the vaccine.

Laboratory finding;

The diagnosis of measles is almost always based on clinical and epidemiologic findings. Laboratory findings in the acute phase include reduction in the total white blood cell count, with lymphocytes decreased more than neutrophils. Absolute neutropenia has been known to occur, however. In measles not complicated by bacterial infection, the erythrocyte sedimentation rate and C-reactive protein levels are normal.

Diagnosis;

In the absence of a recognized measles outbreak, confirmation of the clinical diagnosis is often recommended. Serologic confirmation is most conveniently made by identification of immunoglobulin M (IgM) antibody in serum. IgM antibody appears 1–2 days after the onset of the rash and remains detectable for about 1 mo. Serologic confirmation may also be made by demonstration of a 4-fold rise in IgG antibodies in acute and convalescent specimens taken 2–4 wk later. Viral isolation from blood, urine, or respiratory secretions can be accomplished by culture. Molecular detection by polymerase chain reaction is possible but is a research tool.

Differential diagnosis;

Typical measles is unlikely to be confused with other illnesses, especially if Koplik spots are observed. Measles in the later stages or inapparent or subclinical cases may be confused with a number of other exanthematous immune-mediated illnesses and infections, including rubella, adenoviruses, enteroviruses, and Epstein-Barr virus. Exanthem subitum (in infants) and erythema infectiosum (in older children) may also be confused with measles. Mycoplasma pneumoniae and group A streptococcus may also produce rashes similar to measles. Kawasaki syndrome can manifest many of the same findings as measles but lacks discrete intraoral lesions (Koplik spots) and a severe prodromal cough, and typically has elevated neutrophils and acute-phase reactant levels. In addition, the characteristic thrombocytosis of Kawasaki syndrome is absent in measles. Drug eruptions may occasionally be mistaken for measles.

Complications;

There are **several factors** that make complications more likely.

- 1)- Morbidity and mortality from measles are greatest in patients <5 yr of age (especially <1 yr of age) and those >20 yr of age.
- 2)- In developing countries, higher case-fatality rates have been associated with crowding, which is possibly attributable to a larger inoculum dose following household exposure.
- 3)- Severe malnutrition in children results in suboptimal immune response and higher morbidity and mortality with measles infection.
- 4)- Low serum retinol levels in children with measles have been shown to be associated with higher measles morbidity and mortality. Measles infection lowers serum retinol, so subclinical cases of hyporetinolemia may be made symptomatic during measles.
- 5)- Measles infection in immunocompromised persons is associated with increased morbidity and mortality.
- 6)- Pneumonitis occurs in 58% of patients with malignancy infected with measles, and **encephalitis** occurs in 20%.

The complications are :

- 1)-Pneumonia is the most common cause of death in measles. It may manifest as **giant cell pneumonia** caused directly by the viral infection or as superimposed bacterial infection.
- 2)-Croup, tracheitis, and bronchiolitis are common complications in infants and toddlers with measles.
- 3)-Acute otitis media is the most common complication of measles. Sinusitis and mastoiditis also occur as complications. Viral and/or bacterial tracheitis are seen and can be life threatening. Retropharyngeal abscess has also been reported.
- 4)-Measles infection is known to suppress skin test responsiveness to purified tuberculin antigen. There may be an increased rate of activation of pulmonary tuberculosis in populations of individuals infected with Mycobacterium tuberculosis.
- 5)-Diarrhea and vomiting are common symptoms associated with acute measles, and the gastrointestinal tract has diffuse giant cell formation in the epithelium. Dehydration is a common consequence, especially in young infants and children. Appendicitis may occur due to obstruction of the appendiceal lumen by lymphoid hyperplasia.
- 6)-Febrile seizures occur in <3% of children with measles.
- 7)-Encephalitis following measles has been a long-associated complication, often with an unfavorable outcome. Rates of 1–3/1,000 cases of measles have been reported, with greater numbers occurring in adolescents and adults than in preschool or school-aged children. This is a postinfectious immunologically mediated process rather than due to a direct effect by the virus. Clinical onset begins during the exanthem and presents with seizures (56%), lethargy (46%), coma (28%), and irritability (26%). Findings in cerebrospinal fluid include lymphocytic pleocytosis in 85% and elevated protein concentration. Approximately 15% of patients die, and 20–40% suffer long-term sequelae, including mental retardation, motor disabilities, and deafness.
- 8)-A severe form of measles rarely seen now is **hemorrhagic or “black measles.”** It presented with a hemorrhagic skin eruption and was often fatal.
- 9)-Keratitis, appearing as multiple punctate epithelial foci, resolved with recovery from the infection.

10)-Thrombocytopenia sometimes occurred following measles.

11)-Myocarditis is a rare complication

12)- Miscellaneous bacterial infections have been reported, including bacteremia, cellulitis, and toxic shock syndrome.

13)- Measles during pregnancy has been associated with high maternal morbidity, fetal wastage and stillbirths, and congenital malformations in 3% of live born infants.

14)-Subacute Sclerosing Parencephalitis (SSPE).

SSPE is a chronic complication of measles with a delayed onset and an outcome that is nearly always fatal. After 7–10 yr the virus apparently regains virulence and attacks the cells in the CNS that offered the virus protection. Most die within 1–3 yr of onset from infection or loss of autonomic control mechanisms.

Treatment;

Management of measles is supportive. Antiviral therapy is not effective in the treatment of measles in otherwise normal patients. Maintenance of hydration, oxygenation, and comfort are goals of therapy. Antipyretics for comfort and fever control are useful. For patients with respiratory tract involvement, airway humidification and supplemental oxygen may be of benefit. Respiratory failure due to croup or pneumonia may require ventilatory support. Oral rehydration is effective in most cases, but severe dehydration may require intravenous therapy. Prophylactic antimicrobial therapy to prevent bacterial infection is not indicated.

Measles infection in immunocompromised patients is highly lethal. Ribavirin is active in vitro against measles virus.. However, no controlled trials have been performed, and ribavirin is not licensed in the United States for treatment of measles.

VITAMIN A

Vitamin A deficiency in children in developing countries has long been known to be associated with increased mortality from a variety of infectious diseases, including measles. In the United States, studies in the early 1990s documented that 22–72% of children with measles had low retinol levels

Prognosis;

In the early 20th century, deaths due to measles varied between 2,000 and 10,000, or about 10 deaths per 1,000 cases of measles. With improvements in health care and antimicrobial therapy, better nutrition, and decreased crowding, the death to case ratio fell to 1 per 1,000 cases.

Prevention;

Patients shed measles virus from 7 days after exposure to 4–6 days after the onset of rash. Exposure of susceptible individuals to measles patients should be avoided during this period.

Post exposure prophylaxis

Susceptible individuals exposed to measles may be protected from infection either by vaccine administration or immunization with immunoglobulin. 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 following exposure to prevent or modify infection. Immunocompetent children should receive 0.25 mL/kg intramuscularly and immunocompromised children should receive 0.5 mL/kg. Immune globulin is indicated for susceptible household contacts of measles patients, especially infants <6 mo of age, pregnant women, and immunocompromised persons.