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PATHOGENESIS, IMMUNITY, PATHOLOGY AND CLINICAL MANIFESTATIONS OF MEASLES

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Annotation

Measles (rubeola) is a highly contagious, acute, exanthematous respiratory disease with a characteristic clinical picture and a pathognomonic enanthem: Koplik's spots, an eruption on the buccal mucous membranes. Measles virus is the only member of the genus Morbillivirus that infects humans. Part of the family Paramyxoviridae, it is related to viruses causing similar infections in other mammals: distemper, rinderpest, morbilli, and peste des petits ruminants. There is only one antigenic type. Virions—pleomorphic spheres with a diameter of 100–250 nm—consist of six proteins. The inner capsid is composed of RNA and three proteins. The outer envelope consists of a matrix protein bearing short surfaceglycoprotein projections or peplomers, one a hemagglutinin (H) and the other a fusion (F) protein.

Keywords: reticuloendothelial system, paramyxoviridae, Warthin-Finkeldey cells, pneumonia, otitis media, malaise, cough, coryza, conjunctivitis, lacrimation, nasal discharge, fever.

Measles virus invades the respiratory epithelium and spreads via the bloodstream to the reticuloendothelial system, from which it infects white blood cells, thereby establishing infection of the skin, respiratory tract, and other organs. Both viremia and viruria develop. Multinucleated giant cells with inclusion bodies in the nucleus and cytoplasm (Warthin-Finkeldey cells) are found in respiratory and lymphoid tissues and are pathognomonic for measles. Direct invasion of T lymphocytes and increased levels of suppressive cytokines (e.g., interleukin 4) may play a role in the temporary depression of cellular immunity that accompanies and transiently follows measles. The major infected cell in the blood is the monocyte. Infection of the entire respiratory tract accounts for the characteristic cough and coryza of





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measles and for the less frequent manifestations of croup, bronchiolitis, and pneumonia. Generalized damage to the respiratory tract, with loss of cilia, predisposes to secondary bacterial infections such as pneumonia and otitis media. Specific antibodies are not detectable before the onset of rash. Cellular immunity (consisting of cytotoxic T cells and possibly natural killer cells) plays a prominent role in host defense, and patients who are deficient in cellular immunity are at high risk for severe measles. Children with isolated agammaglobulinemia are not at increased risk. Immune reactions to the virus in the endothelial cells of dermal capillaries play a substantial role in the development of Koplik's spots (the pathognomonic enanthem) and rash; in immunodeficient hosts, measles may be severe despite the absence of these manifestations. Measles antigens have been demonstrated in involved skin during early stages of the illness. Pathologic changes in measles encephalitis include focal hemorrhage, congestion, and perivascular demyelination. Measles virus is rarely isolated from cerebrospinal fluid (CSF) in cases of encephalitis, which are thought to be due to the interaction of virusinfected cells with local cellular immune factors.

Measles begins with a 2- to 4-day respiratory prodrome of malaise, cough, coryza, conjunctivitis with lacrimation, nasal discharge, and increasing fever [with temperatures as high as 40.6°C (105°F), probably reflecting secondary viremia]. At this stage of the illness, in which the rash has not yet developed, influenza may be suspected. Just before rash onset, Koplik's spots appear as 1- to 2-mm bluewhite spots on a bright red background. Without adequate illumination for examination, they may be overlooked. Koplik's spots are typically located on the buccal mucosa, alongside the second molars, and may be extensive; they are not associated with any other infectious disease. The spots wane after the onset of rash and soon disappear. The entire buccal and inner labial mucosa may be inflamed, and the lips may be reddened.

The characteristic erythematous, nonpruritic, maculopapular rash of measles begins at the hairline and behind the ears, spreads down the trunk and limbs to include the palms and soles, and often becomes confluent. At this time, the patient is at the most severe point of the illness. By the fourth day, the rash begins to fade in the order in which it appeared. Brownish discoloration of the skin and





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desquamation may occur later. Fever usually resolves by the fourth or fifth day after the onset of rash; prolonged fever suggests a complication of measles. Lymphadenopathy, diarrhea, vomiting, and splenomegaly are common features. The chest x-ray may be abnormal, even in uncomplicated measles, because of the propensity of measles virus to invade the respiratory tract. The entire illness, which usually lasts ~10 days, tends to be more severe in adults than in children, with higher fever, more prominent rash, and a higher incidence of complications.

Milder forms of the illness with less intense symptoms and a milder rash, termed modified measles, may occur in individuals with preexisting partial immunity induced by active or passive vaccination. These patients include infants <1 year of age who retain some proportion of passively acquired maternal antibodies. On occasion, individuals with a history of immunization may develop modified measles.

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