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Measles in Children

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Abstract

Rubeola, often known as measles, is a highly contagious viral infection. Measles is caused by infection with the measles virus. The measles virus is a non-segmented negative sense RNA virus that belongs to the morbillivirus genus of the paramyxoviridae family. It is spread through the air via respiratory droplets or aerosolized particles, and symptoms include fever, cough, coryza, and conjunctivitis, followed by an erythematous maculo-papular rash. Many organ systems are affected by measles complications, and pneumonia accounts for the majority of measles-related morbidity and mortality. Clinical presentation and laboratory test results, including the presence of anti IGM antibodies and/or viral RNA, are used to confirm a case. Vitamin A is given to measles patients as part of their treatment. Measles vaccines comprise of a live attenuated measles virus strains and great advance has been made to increase vaccination coverage in the world to decrease the incidence of measles from infection with measles virus.

Keywords: Measles; Measles virus; Fever; Vitamin A; Measles vaccine.

Introduction

Measles is a contagious disease that begins with catarrhal signs and progresses to a characteristic rash. Despite the availability of a reliable vaccination, measles remains a leading cause of illness and mortality in young children around the world. The measles virus causes the disease. The measles virus is a single-stranded RNA virus with a lipid envelope that belongs to the genus Morbillivirus and the family Paramyxoviridae.[1] The particle of the measles virus is pleomorphic. Two surface glycoproteins, fusion (F) and hemagglutinin (H), form a multimeric complex in the virus particle that promotes viral entrance.[2] Transplacental antibodies normally protect infants until they reach 9 months of age. Measles is characterised by a high fever and a rash on the skin, and it is frequently accompanied by cough, coryza, and conjunctivitis. The hallmark of measles, according to Briggita et al[3] is transitory immunological suppression, which increases vulnerability to opportunistic infections.

Epidemeology

Measles is a disease that spreads both epidemically and endemically. Incidence is highest in the winter and spring. A single measles infection provides immunity for the rest of one's life. Measles is the most common vaccine preventable disease in the world, accounting for 38 percent of the disease burden. From 1997 to 2005, the number of reported cases decreased, but then increased dramatically in 2006. In India, the WHO began a measles surveillance effort in 2007. According to a WHO

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report published in 2008, there were 60751 reported cases of measles in India in 2006 and 52454 in 2005. According to an EPI data sheet, India's measles vaccination coverage was 67 percent in 2003 among children under the age of one year.

According to global estimates for the year 2013, measles was responsible for over 0.14 million deaths, or nearly 16 deaths each hour. According to research conducted by Anand et al[4] and Kumar R et al[5], India alone accounted for more than half of all global measles-related deaths. Sudfeld et al[6] conducted a systematic review of studies published over four decades in 12 Indian states and found that the median case fatality ratio was 1.63 percent. Bose AS et al[7] stated that furthermore, the higher case fatality ratio was reported among under-five children and children from the backward class. The number of measles cases has come down in 2019 to24076 cases from 69391 in 2018 according to WHO.

Transmission

The measles virus enters the body through the respiratory tract or conjunctiva after coming into touch with big droplets or small droplet aerosols containing the virus. From three days before the rash and four days after the rash , patients are infectious.[7-8]

Muhlebachet al.[9] found that Tracheobronchial epithelial cells are sensitive to Measles Virus infection, which is linked to bronchial and bronchiole epithelial damage.

Pathology

The measles infection results in respiratory tract epithelial necrosis and a lymphocytic infiltration. On the skin and oral mucosal membranes, measles causes a small vessel vasculitis. Intracellular oedema and dyskeratosis are seen on histology of the rash and exanthem. Within these massive cells, viral particles have been discovered. Multinucleated giant cells, the warthinfinkedly gigantic cells that are pathognomic of measles, are formed when infected cells fuse.[10] According to Briggita et al[11], Measles Virus replication in B-cell follicles was found in lymphoid tissues of experimentally infected Non-human Primates.

Pathogenesis

There are four stages in the measles infection. The incubation period, prodromal sickness, exanthematous phase, and recovery are all factors to consider. Measles virus migrates to regional lymph nodes during incubation. A primary viremia develops, allowing the virus to spread throughout the reticuloendothelial system. A secondary viremia is when the virus spreads to the surface of the body. The prodromal disease follows secondary viremia and is characterised by epithelial necrosis and the production of large cells in bodily tissues. In the prodromal phase, viral shedding begins.Antibody production begins with the onset of the rash, and viral replication and symptoms begin to fade. Yanagi et al[12] found that alveolar macrophages, dendritic cells, and lymphocytes are the first targets for the measles virus, which are infected via CD150. Attachment to PVRL4 receptor (nectin 4), which is expressed on cells in the trachea, oral mucosa, nasopharynx, and lungs, is the mechanism of infection of respiratory tissues.

Clinical Manifestations

High fever, exanthem, cough, coryza, conjunctivitis, are all symptoms of measles infection. The prodromal phase begins with a slight fever, followed by conjunctivitis with photophobia, coryza, a pronounced cough, and a growing fever after an incubation period of 8 to 12 days. Koplik spot, is a pathognomonic symptom of measles that appears 1 to 4 days before the rash Rash appears on the forehead, behind the ears, and on the upper neck. In up to 50% of patients, it spreads throughout the chest and extremities, reaching the palms and soles. Symptoms of the rash tend to fade as soon as it appears. The rash dissipates in about 7 days, following the same pattern as it appears. Cough is the most persistent of the primary symptoms in measles, lasting up to ten days. Generalized lymphadenopathy may be observed in severe cases, with the cervical and occipital lymph nodes being particularly prominent. According to a recent modelling study by Mina et al[13], higher susceptibility to infections can last for up to 3 years after measles.

Complications

- ***Gastrointestinal:*** Diarrhoea is the most prevalent gastrointestinal problem, affecting about 8% of patients. Gingivostomatitis, gastroenteritis, hepatitis, mesenteric lymphadenitis, and appendicitis are some of the other gastrointestinal problems. Measles-induced stomatitis and diarrhoea, according to Demmelon Harrison et al, can lead to

nutritional deficiency in resource-limited situations.

- ***Pulmonary:*** Pneumonia is the most prevalent cause of measles-related death in children, accounting for about 6% of all cases. Bronchopneumonia, laryngotracheobronchitis (croup), and bronchiolitis are pulmonary consequences of measles virus infection. Measles has also been linked to the development of bronchiectasis, a lung disease that can lead to recurrent respiratory infections. In one retrospective analysis of measles mortality in South Africa, 85 percent of cases were ascribed to pneumonia, indicating that bacterial superinfection may occur in up to 5% of cases (due to viral or bacterial infection). Streptococcus pneumoniae, Streptococcus pyogenes, Haemophilus influenzae, and Staphylococcus aureus were found in a series of 182 cases with measles-associated pneumonia, according to Halonen et al.[14]
- ***Neurological:*** Neurologic Encephalitis, acute disseminated encephalomyelitis, and subacute sclerosing panencephalitis are among the neurologic sequelae linked with measles. Acute measles-induced encephalopathy in the setting of human immunodeficiency virus infection was described by Ross et al.[14]
- ***Other Complications:*** Keratitis (a common cause of blindness) and corneal ulceration are two ocular consequences of measles.
- Myocarditis and pericarditis are two cardiac consequences of measles.
- Haemorrhagic measles, often known as black measles, is a severe form of measles that is rarely encountered nowadays. It appears as a haemorrhagic skin eruption and is frequently lethal.

Clinical Variants

- ***Modified measles:*** Modified measles is an attenuated infection that occurs in people who already have immunity to measles (either via wild-type disease or vaccination). The clinical signs are often milder, and the incubation period is longer than with classic measles (17 to 21 days). Modified measles patients are not particularly contagious.[15]
- ***Atypical measles:*** This term refers to measles virus infection in people who were inoculated with the killed virus vaccine in the United States between 1963 and 1967; atypical measles is now uncommon. The dead virus vaccine made the recipient sensitive to measles virus antigens but did not provide complete protection.[15]

Laboratory Findings:

- Clinical and epidemiological findings are usually often used to diagnose measles. In the acute phase, laboratory findings include a fall in total white blood cell count, with lymphocytes decreasing more than neutrophils. ESR and CRP levels are normally normal in cases of measles that are not exacerbated by bacterial infection.

Diagnosis

In a patient with a febrile rash illness and clinically compatible symptoms (e.g. cough, coryza, and conjunctivitis), the diagnosis of measles should be considered, especially if there has been recent exposure to someone with a febrile rash illness or travel to a high-measles-prevalence area, especially in the absence of measles immunity. Patients who are being tested for measles should be kept apart.

A significant rise in measles IgG antibody between acute and convalescent titres, isolation of measles virus in culture, or detection of measles virus RNA by reverse transcription polymerase chain reaction are all used to diagnose measles virus infection (RT-PCR). Depending on the incidence of measles in a given region, several approaches to diagnosis are used. The anti-measles virus IgM assay should be read with caution, according to Ross et al, as both false-positive and false-negative results have been observed.

Treatment

Because there is no specific antiviral drug approved for the treatment of measles, management is supportive. The goals of therapy are to maintain hydration, oxygenation, and comfort. Antipyretics are helpful in reducing fever. Airway humidification and supplementary oxygen may be beneficial for patients with respiratory tract involvement. Respiratory failure caused by croup or pneumonia may necessitate the use of a ventilator.In most situations, oral rehydration is sufficient, but severe dehydration may necessitate intravenous therapy. Antimicrobial prophylaxis to prevent bacterial infection is not recommended. In

immunocompromised persons, measles infection is highly fatal. In vitro, ribavirin is effective against the measles virus.

Vitamin A

Vitamin A deficiency has been linked to an increased risk of death from a range of infectious diseases, including measles, in children in underdeveloped nations. Vitamin A therapy is recommended for all measles patients. Vitamin A should be given once a day for two days at doses of 200,000 IU for children 12 months and older, 100,000 IU for infants 6 months and older, and 50,000 IU for infants younger than 6 months.

Prevention

The measles vaccine is an effective way to prevent the disease. Transplacentally acquired maternal antibodies protect the newborn baby. Beginning at the age of six months, the antibodies begin to diminish, and the infant becomes susceptible to measles. The majority of newborns become sensitive between the ages of 9 and 12 months. Three doses are recommended; one as MR/MMR at the age of 9 months, second as MMR at 15 months, and third as MMR at school entry (4-5) year or at any time 8 weeks after the previous dose.

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