Measles – Resurgence of an Old Foe

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KEYWORDS

• Measles • Rash • Vaccine • Prevention • Immunity

KEY POINTS

- Measles is extremely contagious viral infection with an attack rate of 90% in nonimmune hosts.
- Complications including pneumonia, diarrhea, dehydration, secondary bacterial infections, and encephalitis are common.
- Treatment is largely supportive, but vitamin A supplementation can help reduce mortality in young children.
- Population coverage of at least 95% with 2 doses of vaccine is critical for herd immunity.

INTRODUCTION

Measles, an acute systemic infection caused by the rubeola virus, is a major cause of morbidity and mortality, particularly in children, around the globe. Humans are the only known reservoir and an asymptomatic carrier state has not been described.

Measles has been a longstanding scourge of human history with the first detailed description of the infection including its differentiation from smallpox by the Persian physician Rhazes, in the 10th century.[1] In the Middle Ages, measles was widespread throughout Europe and Asia. It, along with other diseases such as smallpox, was introduced to the Americas during European colonization and led to the death of large numbers of indigenous people.[1]

Measles continued to be a major cause of global morbidity and mortality in the prevaccination modern era with higher burden of disease in the developing world with millions of infections and deaths annually. In 1963, the first measles vaccine was approved in the United States, developed by Dr John F. Enders and Dr Thomas C. Peebles from a virus cultured from a patient in Massachusetts.[1,2] The live virus was further attenuated in 1968 and continued to be used today.[2]

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Abbreviations	
ADEM	acute disseminated encephalomyelitis
EIA	enzyme immunoassay
HIV	human immunodeficiency virus
Ig	immunoglobulin
R_0	reproductive number
RT-PCR	reverse transcription polymerase chain reaction
SSPE	subacute sclerosing panencephalitis
WHO	World Health Organization

Less than 50 years after development of the vaccine, measles was declared eliminated in many parts of the world including the Americas and parts of Europe thanks to massive vaccination programs targeting young children, meticulous surveillance of cases, and aggressive outbreak management.[3] Unfortunately, largely due to suboptimal vaccination rates and global mobility, measles cases have been increasing and vaccination rates have been decreasing around the world, including in the United States and other regions who had previously achieved elimination.[4] It is imperative that frontline providers recognize the signs and symptoms in susceptible hosts in order to stop transmission to others. This article will describe the epidemiology, clinical symptoms, treatment, and prevention of measles.

VIROLOGY

The rubeola virus is part of the *Morbillivirus* genus and *Paramyxoviridae* family. It is an enveloped, non-segmented, single stranded, negative sense RNA virus that measures 100 to 300 nm in diameter. There are 2 surface proteins important for pathogenesis: the hemagglutinin protein is responsible for binding of the virus to host cell receptors and the fusion protein is responsible for fusion of virus and host cell membranes, viral penetration, and hemolysis. Humans are the only natural host for rubeola, but primates may also be infected.[5]

EPIDEMIOLOGY

Measles is an infection that has affected every country on the globe. In the pre-vaccine area, endemic measles was characterized by annual outbreaks with longer epidemic cycles every 2 to 5 years as new susceptible birth cohorts were introduced to the population.[6] As a result, nearly every individual entered adulthood with natural immunity to measles after childhood infection.

In the pre-vaccine era, the World Health Organization (WHO) estimated measles caused 30 million infections and more than 2 million deaths annually.[7] In the United States, an estimated 3 to 4 million people were infected each year resulting in 48,000 hospitalizations, 1000 chronic disabilities, and 400 to 500 deaths.[2,8]

In the United States, the incidence of measles declined from 22,000 to 75,000 cases annually after the implementation of a single vaccine dose program in the 1960s and 1970s. In 1989, the Advisory Committee on Immunization Practices recommended a second dose of measles vaccine in response to sporadic outbreaks with the goal to induce immunity in the estimated 15% of children who did not mount an appropriate immunologic response and work toward the goal of elimination.[9] Many countries, including the United States, launched comprehensive vaccination programs with the goal of elimination (the absence of endemic transmission for at least 12 months). These programs included provision and administration of vaccines, enforcement of

vaccine requirements for public school, rigorous surveillance for new cases, and outbreak response.[10] As a result, measles was declared eliminated from the United States in 2000 and from the WHO Region of the Americas in 2002 and the only new cases reported were from travelers who were infected abroad.[9]

Elimination status in the United States was threatened by 2 prolonged outbreaks in 2019 among communities with low vaccination rates.[11] The outbreaks were linked to international travel and importation of the virus. There were 1274 confirmed cases in 31 states in 2019, the highest number of cases since the 1992.[12] Sporadic outbreaks have continued in the United States, largely affecting individuals who are not vaccinated.

Globally, measles incidence increased 18% in 2022 compared to 2021 and deaths increased 43% in 2022 compared to 2021.[4] In 2022, the WHO reported more than 200,000 cases of measles around the world.[13] This number is likely underestimated. There were more than 136,000 reported deaths due to measles, mostly in unvaccinated or under vaccinated children less than 5 years of age in countries with low per capita incomes.[13]

TRANSMISSION

Measles is one of the most contagious infections affecting humans with an attack rate of up to 90% in which 9 out of 10 susceptible individuals who are exposed to the rubeola virus will develop disease.[9] The contagiousness can also be expressed by the basic reproductive number (R_0), which is the expected number of secondary cases infected from the index case. The R_0 of an infection depends on the population immunity, frequency of social contacts, and the transmissibility of the pathogen. Measles has an estimated R_0 of 9 to 18, which is much higher than smallpox or influenza, 5 to 7 and 2 to 3, respectively.[6]

Transmission can occur via airborne spread or direct person-to-person contact with large respiratory droplets. Infectious respiratory aerosols can remain suspended in the air for several hours.[5] Transmission of measles has been documented in several public areas including aboard international flights, within airport terminal waiting areas, theme parks, and international sporting events.[14–17] Perinatal measles can be transmitted via transplacental infection or post-natally via respiratory droplets from an infected mother.[18]

In temperate zones, transmission tends to occur in late winter-spring, but can be year-round in warmer climates.[2]

CLINICAL PRESENTATION

The incubation period of measles from exposure to rash averages 14 days (range of 7 – 21 d). The prodrome can start 11 to 12 days after exposure and is characterized by high fever and the “3 C’s” of measles: cough, coryza (acute inflammation of the mucous membranes of the nasal cavities) and conjunctivitis[2] (**Fig. 1**). Koplik spots are a classic diagnostic finding in measles and may be easy to overlook. They are small white, raised lesions on the buccal mucosa opposite of the first or second molars with an erythematous base and have been described as grains of salt on a red background (**Fig. 2**). They typically appear 1 day prior to rash.[19]

The rash of measles is the major hallmark of the disease. The classic rash consists of erythematous macules and papules that start at the hairline and spread downward and outwards to the hands and feet and can involve the palms and soles. The erythema may not be as pronounced on individuals with darker skin pigmentation (**Figs. 3–5**). The macules and papules will often become confluent starting at the

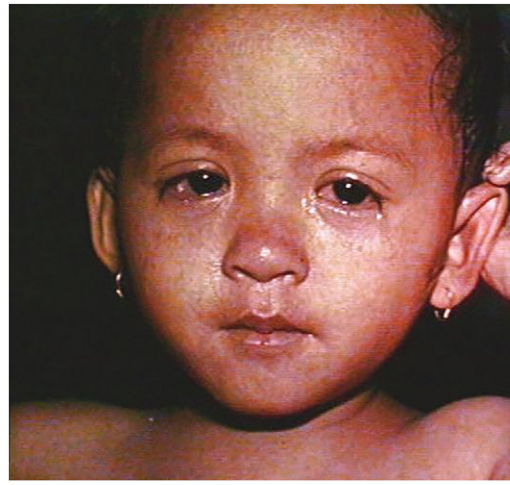

Fig. 1. Young child with macular rash and conjunctivitis—characterized by erythema of conjunctival membrane and marked tear production. (CDC/Barbara Rice.)

head and spreading down the body. The rash typically lasts 5 to 6 days and then will fade in the same order of spread and peel. Patients are contagious 4 days before and 4 days after rash onset.

Patients may also experience anorexia, malaise, lymphadenopathy, nausea, vomiting, and diarrhea. Laboratory findings are notable for leukopenia and thrombocytopenia. Chest radiography may reveal interstitial infiltrates.

Patients with severe immunosuppressive conditions, such as advanced human immunodeficiency virus (HIV), acute leukemia, lymphoma, or other cell mediated immune deficiencies, may have an atypical presentation. Kaplan and colleagues reports the absence of rash in up to 40% of cases in immunocompromised patients.[20] Complication rates were higher than the general population at 80% and case fatality rates were 70% and 40% for patients with malignancies (majority with acute lymphoblastic leukemia) and HIV, respectively.[20]

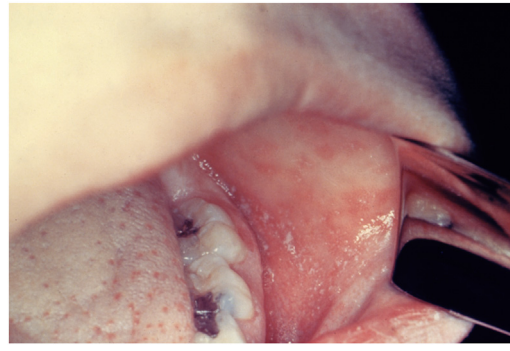

Fig. 2. Koplik spots on buccal mucosal surface near the first and second M. Punctate white spots on erythematous background, often described as grains of salt on a red background. (CDC/Public Health Image Library.)

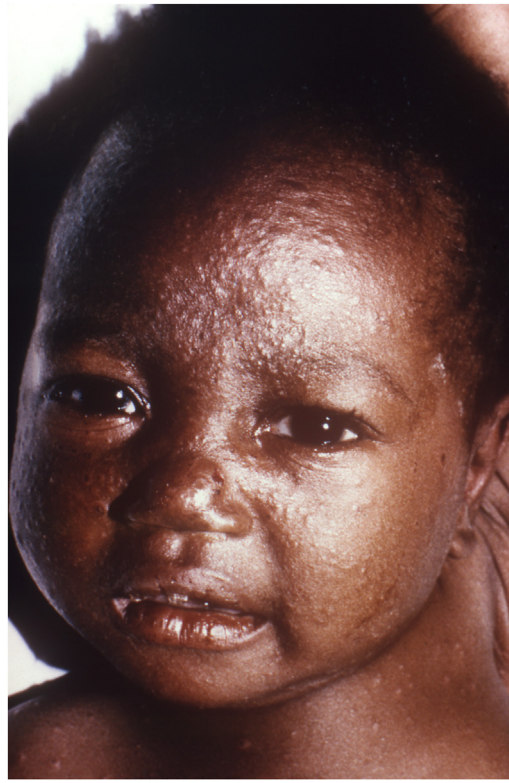

Fig. 3. Maculopapular rash on young child with darker skin pigmentation. Rash is becoming confluent on the forehead. (CDC/Public Health Image Library.)

COMPLICATIONS

More than 30% of patients with measles will experience a complication of various organ systems[2] (**Table 1**). Children less than 5 years of age, immunocompromised patients, individuals with vitamin A deficiency and pregnant women are at highest risk of severe measles and its complications.[5]

Pneumonia, either direct viral invasion or secondary bacterial infection, is the leading cause of death in infants with measles—accounting for 60% of deaths in this age group.[5] In a case series of children with measles in the Philippines, the most common bacterial pathogens were *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Haemophilus influenzae,* and *Staphylococcus aureus*. Viral co-infection was common as well with parainfluenza, adenovirus, cytomegalovirus, enterovirus, influenza, and respiratory syncytial virus.[21] Measles can also cause laryngotracheobronchitis, also known as croup, and bronchiolitis, which can cause significant morbidity and mortality in young children. The development of bronchiectasis has also been described, which can have long-lasting effects on lung function.[22]

Diarrhea is the most common complication of measles. In a recent outbreak of measles amongst children in central Ohio in 2022, 26% of patients experienced diarrhea and 52% of the patients required hospitalization with dehydration being the most common admitting diagnosis.[23]

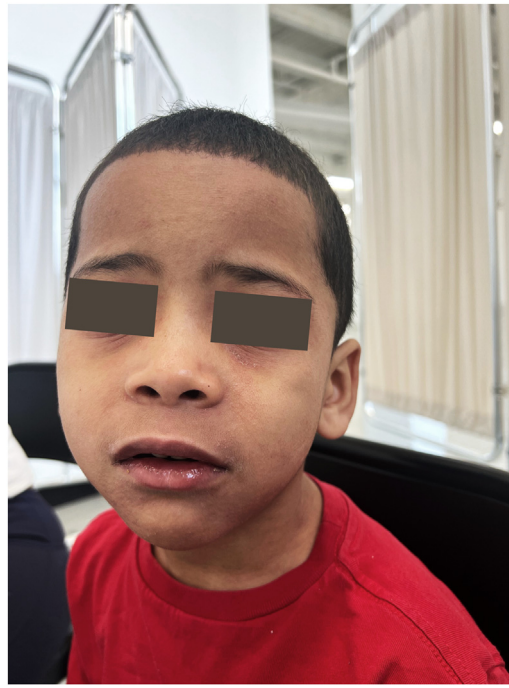

Fig. 4. Macular rash on forehead of child. (CDC/Kimberly Gressick, MD, MPHC.)

Neurologic complications of measles are common and can have long lasting impact. They are divided into 3 entities that have similar presentations, but variable temporal relationships to the index measles infection.

- *Acute encephalitis*: Occurs in 1 per 1000 measles cases. Symptoms develop within a few days of rash and include fever, headache, vomiting, nuchal rigidity, encephalopathy, coma, and seizure. Cerebrospinal fluid typically shows a lymphocytic pleocytosis, elevated protein, and normal glucose. Many patients who survive are left with permanent neurologic sequelae.[5]

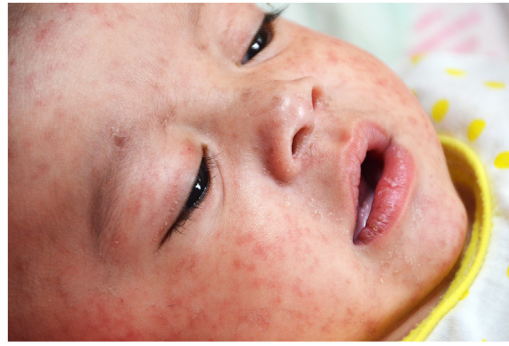

Fig. 5. Macular rash on young child with lighter skin pigmentation. (CDC/James Goodson, M.P.H.)

Table 1
Complications of measles infection

Organ System	Complication
Pulmonary	Pneumonia Laryngotracheobronchitis (Croup) Bronchiolitis Bronchiectasis
Gastrointestinal	Diarrhea Gingivostomatitis Gastroenteritis Hepatitis Mesenteric lymphadenitis Appendicitis
Neurologic	Encephalitis ADEM SSPE
Ocular	Keratitis Corneal ulceration
Cardiac	Myocarditis Pericarditis

- *Acute disseminated encephalomyelitis (ADEM)*: Occurs in 1 per 1000 cases. ADEM presents shortly after recovery of measles—typically within 2 weeks of the rash. ADEM is a post-infectious autoimmune demyelinating disorder that is incompletely understood. Symptoms include fever, headache, nuchal rigidity, seizure, and encephalopathy. Cerebrospinal fluid shows a lymphocytic pleocytosis and elevated protein. Morbidity, including cognitive deficits and recurrent seizures, is common and mortality is estimated at 10% to 20%.[24]
- *Subacute sclerosing panencephalitis (SSPE)*: A rare complication with a variable incidence of 0.06 to 8.5 cases per million cases of measles.[25] SSPE is a fatal and progressive degenerative process of the central nervous system that occurs 7 to 10 years after measles infection. The pathogenesis of SSPE is not well-understood. The clinical features and progression are summarized in **Table 2**. Seizures can occur in any stage. Cerebrospinal fluid shows elevated protein and detectable anti-measles antibody. SSPE is uniformly fatal.

Measles can also manifest in ocular complications such as keratitis and corneal ulceration and is a major cause of childhood blindness in the developing world.[26]

Table 2
Stages of subacute sclerosing panencephalitis

Stage	Symptoms	Duration
I	Insidious onset of personality changes, lethargy, behavior changes, difficulty in school	Wk – y
II	Worsening dementia, myoclonus	3–12 mo
III	Neurologic deterioration ending with flaccidity or decorticate rigidity. Autonomic dysfunction Myoclonus is absent	Variable
IV	Vegetative state	Variable

Measles infection can affect both cell-mediated and humoral immunity leading to lymphopenia, suppression of cellular immunity to secondary infections, and dysregulation of immune signaling.[27] Concomitant viral infection has been well-described and secondary bacterial infections such as otitis media, pneumonia, gastroenteritis, and bacteremia have been reported along with high hospitalization rates in recent outbreaks.[23,28]

Measles infection during pregnancy has been associated increased rates of spontaneous abortion and premature labor.[18]

DIAGNOSIS

The differential diagnosis of measles includes Kawasaki syndrome, scarlet fever, rickettsial infections, meningococcemia, or other viral exanthems including rubella, roseola, mononucleosis, enterovirus, or adenovirus.

Diagnosis of suspected measles typically can be confirmed by serologic tests or by detection of viral RNA via reverse transcription polymerase chain reaction (RT-PCR). Available serologic tests include serum measles immunoglobulin (Ig) M or paired acute and convalescent titers of IgG. Anti-measles IgM by enzyme immunoassay (EIA) is the most common way to diagnose acute infection and is detectable 3 days after the appearance of rash and can persist for 30 days. Most available tests report sensitivity 83% to 89% and specificity 95% to 100% after the first week of rash.[29] It is important to note that serologic testing may be negative on the first day of the rash.[29] A 4-fold increase in IgG titer in acute and convalescent serum samples is considered diagnostic.[5] The IgG antibody is typically negative until 7 days after rash onset.[29] Serology may be negative in severely immunocompromised hosts despite active infection.

Direct detection of viral RNA via RT-PCR can be performed on heparinized blood, nasopharyngeal aspirates, throat swabs, and urine for approximately 3 days after rash onset. The Centers for Disease Control and Prevention recommends laboratory confirmation of all suspected cases of measles with both.

- Measles IgM EIA in serum
- Measles RNA via RT-PCR in a respiratory specimen (throat swab or nasopharyngeal swab). Urine samples can also be collected.

Health care personnel are required to report all suspected cases of measles to the local health department and molecular testing is performed by public health laboratories. In addition to detecting viral RNA, public health laboratories can determine the genotype in order to link cases in a potential outbreak. It is important to note that recent receipt of measles, mumpls, and rubella (MMR) vaccine can lead to positive serologic and molecular testing. Genotyping is the only way to determine if viral RNA is from wild-type measles versus recent vaccination.[30]

TREATMENT

The mainstay of treatment is supportive care, including fluids and anti-pyretics. Most healthy hosts will recover after approximately 1 week of illness. Levels of vitamin A fall during measles infection and pre-existing deficiency can worsen severity and increase risk of complications.[31] Vitamin A supplementation has been shown to reduce mortality in children less than 2 years of age.[32] The WHO recommends 2 doses of oral vitamin A supplementation for children under 5 years of age with acute measles. Children with evidence of severe vitamin A deficiency, such as xerophthalmia, including Bitot's spots and corneal ulceration, should receive a third dose 4 to 6 weeks later.

The role of ribavirin in severe measles is controversial. There are case series of successful use of ribavirin in immunocompromised adults and children with measles, but data are very limited.[33,34]

PREVENTION

The most effective form of measles prevention is completion of the 2 doses series of the live attenuated measles vaccine with the first dose administered at age 12 to 15 months and the second dose at age 4 to 6 years.[9] The live vaccine incites both humoral and cellular immunity against the virus. The vaccine effectiveness after 1 dose in individuals aged 12 months or older is approximately 94% and after 2 doses is greater than 99%.[9] Immunity after natural infection and vaccination is long-lasting and considered to be life-long.

Herd immunity occurs when a large proportion of a population is immune from an infection and makes further spread of the disease highly unlikely. This disruption on viral transmission is the key factor in protecting those individuals who are ineligible for vaccination due to infancy or immunocompromising conditions. This threshold is variable for different infections. In order to disrupt measles transmission and achieve herd immunity, vaccination coverage in a population must be above 95%.

Contraindications to measles vaccine include anaphylactic allergic reaction to any component of the vaccine, pregnancy, and severe immunosuppression. These individuals should be given Ig within 6 days of exposure to measles.[9] Non-immune individuals who are eligible to receive the vaccines should be vaccinated as soon as possible after exposure.[9] Infants born to mothers with active measles should be given Ig at birth and start the recommended vaccine series at 15 months.[5] Infants younger than 12 months are not routinely vaccinated against measles as the immune response may be suppressed by residual maternally acquired antibodies.[5] Infants, as young as 6 months, may receive the vaccine if there is high risk of exposure to measles, but additional doses may be required later in life.[35]

Since the development of the vaccine in the mid-twentieth century, public health organizations have been working toward universal 2 dose vaccination coverage with the ultimate goal of global elimination. On a global scale, vaccination coverage had been increasing in the early 2000s to a peak of 86% coverage for the first dose of measles vaccine in 2019. Largely due to missed vaccination during the COVID-19 pandemic and increasing vaccine hesitancy, first dose coverage rates dropped to 81% in 2021, which was the lowest rate since 2008.[4]

In the United States, vaccine campaigns were successful in achieving greater than 95% 2-dose coverage, but rates have been decreasing over the last several years. In 2019 to 2020, 95.2% of kindergartners were fully vaccinated compared to 92.7% in the 2023 to 2024 school year.[12]

SUMMARY

Measles was a major cause of childhood morbidity and mortality throughout human history. The introduction of vaccination in the mid-twentieth century drastically reduced the burden of disease and saved millions of lives. Measles outbreaks are resurging given suboptimal population vaccination coverage, global mobility, and the transmissibility of the virus. Complication rates are high with infection and treatment is largely supportive. Vitamin A supplementation is beneficial in children with severe measles. More data are needed on the efficacy of ribavirin in immunocompromised patients. Complete vaccination with 2 doses of vaccine is highly effective in preventing measles infection,

but achieving the 95% population coverage that is needed for elimination is an ongoing challenge throughout the world.

CLINICS CARE POINTS

- Measles outbreaks continue to occur around the world given suboptimal vaccine coverage, global mobility, and very high transmissibility.
- The initial symptoms can overlap with other common childhood infections and clinicians must have a high index of suspicion for measles in susceptible populations.
- Both serum IgM and PCR of a respiratory specimen should be sent on all suspected cases of measles. The serology may be negative on the first or second day of the rash.
- Complications and need for hospitalization is common in patients with measles. Vitamin A supplementation is recommended for children with severe measles.
- Vaccination coverage of 95% is critical to achieve herd immunity in a population and maintain elimination of measles.
- Cases of measles are expected to increase in the United States and around the world.

DISCLOSURES

The author has nothing to disclose.

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