

Research Article

Assessment of Nurses Knowledge about Children Measles

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ABSTRACT

Although life-threatening, measles (Rubeola) still generates epidemics. Refusing vaccination leaves vulnerable groups exposed to the virus upon importation from endemic nations. Children and other susceptible people may get the infection from these people.

Rubella, a mild, self-limiting illness with fever and rash, is usually acquired postnatally. Coughing and sneezing aerosols spread the virus 7 days after the rash. CRS, the primary cause of birth abnormalities and miscarriages, is a significant pregnancy condition. The disease is worse in the first 12 weeks of pregnancy. Vaccination may prevent the illness despite its severe consequences and irreparable abnormalities. This review discusses rubella vaccination's role in eradicating this dangerous illness.

1. INTRODUCTION

Measles is an acute viral disease caused by a virus belonging to the Paramyxovirus family and the Morbillivirus genus. Measles is marked by an initial phase of fever (reaching up to 105°F), malaise, cough, coryza, and conjunctivitis, followed by a maculopapular rash. Measles is a very contagious and infectious illness. The measles virus infection is a significant infectious illness in humans, having resulted in millions of fatalities since its origin as a zoonotic infection millennia ago. The rise in measles incidence is attributed to the continuous decline in vaccination coverage. This occurrence has elicited public and scientific curiosity. Consequently, we examined the pathophysiology of measles infection, emphasizing the methods by which the virus disseminates systemically throughout the host body. Measles creates an immunosuppressive state by accessing lymphocytes in the airways via a "Trojan horse" mechanism. H and f glycoproteins, both present in the envelope, facilitate the virus's attachment to host cells and its dissemination across cells by interacting with various receptors. The severity of the illness is contingent upon the patient's age, preexisting diseases, and the social and health milieu surrounding the development of epidemics. It is often encumbered by sequelae and problems that may arise many years post-infection. The elimination of measles is deemed technically achievable due to the following reasons: the current vaccines exhibit high efficacy; measles predominantly affects humans, with no non-human reservoirs; there is an absence of a carrier state and a minimal number of asymptomatic cases; and immunity to measles, whether obtained through vaccination or natural infection, is long-lasting, potentially lifelong.

- The thesis aimed to:

The intent of the thesis was to This analysis aims to assess the significance of the measles virus and its impact on children as a risk factor for the diseases it induces, with a particular focus on the importance of regular screening and appropriate health protection measures.

2. LITERATURE REVIEW:

Measles is an acute, viral, and infectious disease. We can find references to measles as early as the 7th century. In the 10th century, the Persian physician Rhazes described the disease as more dreaded than smallpox. In 1846, Peter Panum described the incubation period of measles and lifelong immunity after recovery from the disease. John Enders and Thomas Chalmers Peebles isolated the virus in human and monkey kidney tissue culture in 1954.

Severe forms with non-pathognomonic clinical features may occur, especially in individuals with compromised or deficient cellular immunity, such as those being treated for malignant disease, transplanted, individuals with acquired immunodeficiency syndrome (AIDS), or any form of congenital immunodeficiency [1].

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Over 30 million new cases and 700,000 fatalities occur yearly in low-income nations, according to WHO estimates. Measles complications including pneumonia, diarrhea, and starvation kill many children in these countries. Health care personnel may misdiagnose a child's illness as "pneumonia" or "diarrhoea," unaware knowing it is measles.

Post-measles consequences last years. Chronic lung sickness, malnutrition (marasmus or kwashiorkor), stunted growth, recurrent infections, and measles (which blinds half of African children) are examples. After one infection, measles doubles the risk of sickness and death for months. This slide shows a measles-blind youngster. Measles, vitamin A deficiency, or subsequent illness may cause blindness [2]. Measles incubates for 4 days before and after rash. More droplets and viral transmission result from violent coughing with higher viremia. Since it cannot infect animals or reservoirs, the virus can only live in humans via unbroken transmission chains. Easy diseases may heal rapidly and without problems.[3]

The absence of therapy for children with measles heightens the risk of exacerbating complications and mortality. Despite the presence of medical facilities, several parents delay seeking treatment for their ill children until it is too late. Vitamin A insufficiency compromises the immune system and damages gastrointestinal and pulmonary cells. Damage hinders infection control and preventive efforts. A deficit in Vitamin A elevates the risk of complications and death associated with measles. A regression analysis indicates the relationship between vitamin A deficiency and mortality associated with measles [4]. In children under two years of age, the mortality risk from measles infection increased with very low vitamin A levels. In the United States, where clinical vitamin A insufficiency is rare, measles cases in children with diminished vitamin A levels correlate with a heightened risk of hospitalization and severe illness. Vitamin A treatment helps alleviate difficulties associated with respiratory ailments, including cough, pneumonia, and diarrhea. Children who use vitamin A supplements had reduced hospitalizations [5].

3. IDENTIFICATION OF THE MAIN VARIETIES OF MEASLES THAT IMPACT PEOPLE AND THEIR ETIOLOGICAL FACTORS.

Measles is a highly infectious viral illness characterized by elevated fever, widespread rash, rhinorrhea, cough, and conjunctivitis without exudate. It may result in serious problems, including mortality. Measles exists in two forms, each attributable to a distinct virus. Nevertheless, both variants of measles display similar symptoms, induce a rash and fever, and represent distinct illnesses. When the majority of individuals use the word measles, they are alluding to the first ailment listed below.

3.1 First type Standard measles:

Commonly referred to as red measles or hard measles, it is caused by the Rubeola virus. The rubeola virus is responsible for "red measles," sometimes referred to as "hard measles" or just "measles." While most of individuals recuperate without complications, rubeola may result in otitis media, pneumonia, or encephalitis.

3.1.1 Measles virus (MV):

Measles virus (MV) belongs to the genus *Morbillivirus* of the family *Para-myxoviridae*. It is an enveloped, no-segmented, single-stranded, negative-sense RNA virus, and its genome encodes at least six structural proteins. MV (also known as rubeola virus) causes measles, an acute, highly contagious infection usually seen in children. Recovery from measles is the rule, but severe complications may develop in some cases [6].



Fig. 1. depicts a child with red eyes. Conjunctivitis eye or measles [7]

3.2 Second type German measles:

The rubella virus induces a distinct sickness referred to as rubella. The rubella virus induces "German measles," often referred to as "three-day measles" and "complicated measles." This sickness is less severe than red measles. An infected pregnant woman may transmit the virus to her unborn child, potentially resulting in severe birth abnormalities [8].

3.2.1 Rubella virus:

The term Rubella is derived from the Latin word rubella, meaning "little red." The Rubella virus, the only member of the Rubi virus family within *Togaviridae*, is responsible for Rubella.

The rubella virus is somewhat spherical, measuring 40–60 nm in diameter, and has a positive-sense single-stranded RNA genome encased inside an icosahedral lipid capsid. The virus comprises three structural proteins: two envelope proteins, glycoproteins E1 and E2, and one core protein, C, encasing the DNA at its 3' terminus. The E1 proteins manifest as surface spikes measuring 6 nanometers in length. E1 is believed to regulate the immune system's response to structural proteins and is associated with both neutralizing and hemagglutinating factors. The envelope includes the E2 glycoprotein [9].

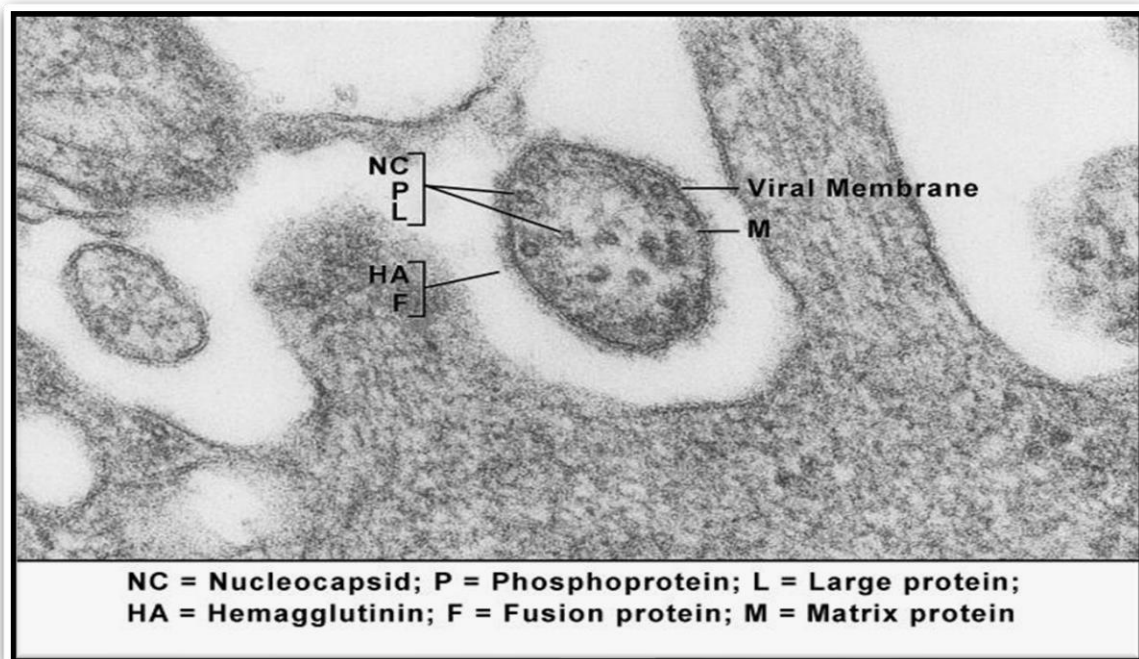


Fig. 2. Measles virus, mosaic

4. PATHOGENESIS OF MEASLES VIRAL INFECTION

The pathogenesis of MeV begins with the virus's entrance into the host's respiratory system by the inhalation of infectious droplets [10].

MeV first infects epithelial cells in the respiratory tract, where it multiplies and disseminates to regional lymph nodes [11]. MeV targets dendritic cells and macrophages, which promote viral propagation to other organs [12]. During the early phase of infection, MeV suppresses the host's innate immune response by inhibiting interferon production and reducing the expression of major histocompatibility complex (MHC) classes I and II molecules [13]. This allows the virus to evade detection and clearance by the host's immune system. As the infection gets worse, MeV triggers a strong T cell response, which makes cytokines that cause inflammation, like interferon-gamma, tumor necrosis factor-alpha, and interleukin-6. These cytokines promote the recruitment of additional immune cells to the site of infection and contribute to the development of the characteristic measles rash. In addition to its effects on the immune system, MeV also has direct cytopathic effects on infected cells. MeV induces syncytia, in which infected cells fuse together to form multinucleated giant cells [14]. This process can lead to tissue damage and contribute to the development of MeV-associated complications such as encephalitis.

4.1. Virulence factors of Measles

1. Portal of entry: respiratory mucous membrane. Initially, it infects the respiratory mucosa, then disseminating via the lymphatic system and circulation, potentially infecting the conjunctiva, respiratory tract, urinary tract, gastrointestinal tract, endothelial cells, and central nervous system.[15]

2. Attachment: Hemagglutinin is a glycoprotein that is present on the surface of most cells. It is an integral membrane protein that is located on the surface of the measles virus. Hemagglutinin binds to CD46, which is a cluster of differentiation glycoprotein. Specifically, CD46 binds to C3b and C4b and then cleaves them, so protecting host cells from being destroyed by autoimmune processes.
3. Evade the immune system: Immunosuppression. The measles virus blocks the T proliferation response to IL-2. And the measles's hemagglutinin protein and fusion proteins bind to lymphocytes and interrupt IL-2 cell signaling. [16]
4. Destruction of tissue: a serious febrile illness. Immune T-cells target the infected endothelial cells of the small blood vessels, causing the maculopapular rash, which starts at the hairline and spreads over the whole body. T-cell-deficient individuals do not have the rash but do have an uncontrolled disease that usually results in death. The immune system most likely causes the damage and controls the disease [19]

5. RISK FACTORS FOR INFECTION AND ITS TRANSMISSION:

Contact with measles puts non-immune people at risk. The most effective way to prevent measles is through vaccination with the measles, mumps, and rubella (MMR) vaccine, which provides lasting protection against measles [17]. other risk factors for measles infection include: Visit regions experiencing active measles epidemics

1. Overcrowded living environments, including refugee camps or slums.
2. Inadequate health care. Inaccessibility to healthcare, which may postpone diagnosis and treatment.
3. Immunocompromising: illnesses or therapies that increase the likelihood of serious disease and consequences.
4. Vitamin A deficiency: Individuals who are low in vitamin A are critically susceptible to severe corneal melting (keratomalacia, corneal ulceration). Corneal ulceration/keratomalacia (X3B): This results from severe vitamin A insufficiency. The beginning is often abrupt, and the cornea may deteriorate rapidly, sometimes within a few hours (keratomalacia). This phenomenon is mostly seen in young children. The youngster shown on the bottom left has severe corneal ulcers advancing towards keratomalacia. As seen in Figure 3, it is crucial to understand that not all children who are vitamin A deficient and at risk of blindness will have conspicuous ocular symptoms. Identifying keratomalacia or corneal ulceration in one kid suggests that additional children within the same family and community are also vitamin A deficient, despite the absence of overt symptoms. A youngster may possess enough vitamin A levels yet have little hepatic reserves. a youngster gets measles.

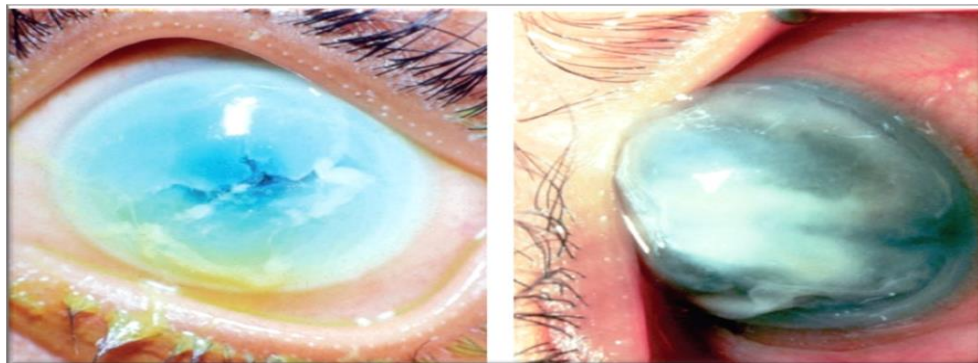


Fig. 3. keratomalacia infection [18]

6. MANIFESTATION AND CLINICAL FEATURE:

a. Differential Symptoms:

- Prodromal and general manifestations. Measles infection presents with a 2- to 3-day prodrome marked by fever, lethargy, cough, and rhinorrhea. Conjunctivitis and bronchitis are often seen. Although a rash was not seen initially, the patient is disseminating the virus and is highly contagious. A pronounced, unproductive cough is seen throughout the febrile phase, persists for 1–2 weeks in uncomplicated cases, and often represents the last symptom to abate. Generalized lymphadenopathy often presents in newborn neonates. Older children may exhibit photophobia and, sometimes, arthritis.
- Koplik's Spots. Koplik's spots may be seen on the buccal mucosa in more than 80% of instances if meticulous daily inspections are conducted immediately before to the development of the rash. Koplik's spots are elevated white lesions about 2-3 mm in diameter on an erythematous background, as seen in figure 3. Initially, there are typically one to five lesions; however, as the commencement of the rash nears, the number may increase to several hundred.

- Rash : A distinctive rash including extensive, irregular red patches first manifests behind the ears and on the skin of the face 2 to 4 days after the onset of prodromal symptoms. A high fever concurrently manifests. The rash intensifies after 2–3 days, being most pronounced on the trunk and upper extremities. The density of the rash may fluctuate.

b. Differential Diagnosis:

Fever, rash, and an array of vague symptoms are associated with several diseases. When assessing for measles, it is essential to consider the presence of rubella and scarlet fever. Additionally, other disorders may manifest similarly, such as enterovirus or adenovirus infections, Kawasaki disease, toxic shock syndrome, rickettsial diseases, and medication hypersensitivity responses. Modified forms of measles, characterized by often mild symptoms, tend to occur in newborns retaining some immunity from maternal antibodies, and infrequently in individuals who have acquired incomplete vaccination protection.

6.1 Differential Signs and symptoms of red measles and German measles

Symptoms and indications often manifest after 8 to 12 days following infection with the rubeola virus. The incubation period refers to the duration between exposure to the measles virus and the manifestation of first symptoms. Symptoms and signs present in two stages. Distinct symptoms and signs manifest in measles infections; these signals are crucial for differential diagnosis between the two forms of measles. I will outline the distinctive symptoms of both types.

- **Slandered Measles (red measles)**

1. People with measles may develop Koplik spots, and rash.
2. Malnourished, chemotherapy-treated, and HIV-positive persons have weaker immune systems, making red measles worse.
3. Encephalitis diseases: is defined as inflammation of the brain that causing by Rubeola (Standard measles).

- **Rubella (German Measles; 3-Day Measles):**

- Common complications: There are a number of common complications that may arise from measles, such as:
 1. Vomiting and diarrhoea, which can lead to dehydration
 2. Eye infection, conjunctivitis
 3. Ear infection, otitis media, which can lead to earache
 4. Inflammation of the voice box, laryngitis
 5. While these symptoms can be distressing, they are not usually serious. However, affected people should see a doctor if they are concerned.
- Uncommon complications: More uncommon complications from measles may include:
 1. A squint, if the measles virus affects the nerves or muscles of the eye
 2. Fits caused by fever, such as febrile seizures
 3. Inflammation of the liver, known as hepatitis
 4. Infection of the airways and lungs, such as pneumonia, bronchitis or croup, which can be very serious

7. MEASLES IMMUNE RESPONSE:

Recent viral illnesses like measles typically manifest as fever, cough, coryza, and conjunctivitis, followed by the development of a maculopapular rash. The respiratory system is the principal site of infection for the measles virus, which then disseminates to the regional lymph nodes. The virus selectively targets immunological cells, including dendritic cells and T cells, inside these lymph nodes [19]. The measles virus triggers an immune response that includes both innate and adaptive immune systems. The development of lasting immunity is essential for protection against subsequent infections.

7.1 Innate Immune Response

The first line of defense against invading pathogens, including the measles virus, is the innate immune response. Pattern recognition receptors (PRRs), including Toll-like receptors (TLRs) and RIG-I-like receptors (RLRs), are essential in the innate immune response to the measles virus. These receptors particularly recognize viral components, such as viral RNA [20]. This initiates the synthesis of type I interferons (IFNs). These interferons initiate antiviral signaling pathways and induce the expression of several interferon-stimulated genes (ISGs) [21]. The production of ISGs induces an antiviral response in both infected cells and adjacent cells, therefore inhibiting viral propagation and facilitating clearance.

Measles-specific T cells are essential for the adaptive immunological response to the measles virus. Measles virus-specific T cells may recognize and eliminate infected cells, either by direct destruction or by secreting cytokines that activate other immune cells [22].

T cells that target the measles virus are essential for establishing enduring immunity by differentiating into memory T cells, which can promptly respond to future measles infections.

7.2 Adaptive Immune Response:

The adaptive immune response to the measles virus entails the activation of B and T cells, which collaborate to eradicate the virus and create enduring immunity [23].

In the adaptive immune response, the measles virus envelope glycoprotein is the principal target of neutralizing antibodies. This glycoprotein is also the primary target of the defensive immunological response. B cells are able to identify and attach to the envelope glycoprotein, which ultimately results in the generation of antibodies that are unique to the virus. These antibodies have the ability to neutralize the virus by preventing it from entering host cells. Additionally, they have the ability to assist the clearance of virus-infected cells by activating the complement system and attracting immune cells.

Additionally essential to the adaptive immune response against the measles virus are T cells that are unique to the measles virus. T cells that are specific to the measles virus may either destroy virus-infected cells directly or secrete cytokines that stimulate other immune cells. These T cells are able to identify and eradicate virus-infected cells.

8. COMPLICATIONS

- **Diarrheal Illness.** A significant proportion of babies and toddlers in underdeveloped nations have diarrhea both during and after acute measles infection.
- **Respiratory Infections.** Respiratory infections are the most common cause of significant morbidity and mortality in infants and children with measles. Pneumonia may be due to the measles virus alone or to secondary infection with other viral agents-especially herpes simplex and adeno-viruses or bacterial organisms [22].
- **Malnutrition Diarrhea:** is one of the major factors contributing to the adverse impact of measles on the Nutri already malnourished. Moreover, measles may exacerbate malnutrition because of decreased food intake due to malaise, increased metabolic requirements in the presence of fever, or the mistaken belief of parents and health practitioners that a child's food should be withheld during an acute illness.
- **Neurological Complications.:** These cases manifest in 1–4 out of every 1,000 children who are affected. Febrile convulsions are the primary cause of mortality and typically do not result in long-lasting residual consequences. Encephalitis, also known as post infectious encephalopathy, it is caused by the continued presence of the measles virus in the central nervous system. Chronic measles can manifest several years following an initial measles infection [23].
- **Febrile seizures:** Febrile seizures can occur in children during the acute phase of measles infection. They are usually self-limiting and do not lead to long-term neurological damage
- **Thrombocytopenia:** Measles can cause a decrease in platelets, which are responsible for blood clotting. This can lead to bleeding disorders and may require hospitalization
- **Otitis media:** Measles can lead to otitis media, which is an infection of the middle ear. It can cause pain, fever, and hearing loss [24].
- **Death:** Measles may be lethal, particularly in infants under five and people over twenty. The death rate may reach 30% in some populations, particularly in regions with inadequate healthcare facilities and low immunization rates. [25].

9. VACCINE

Multiple forms of measles vaccinations exist; the nanocomponent is used in the majority of countries. The MMR vaccination, which combines mumps, measles, and rubella, is used throughout the remainder of Europe and North America. A quadrivalent vaccination for mumps, measles, rubella, and varicella (MMRV) is available in the United States and demonstrates safety and immunogenicity comparable to the MMR vaccine. [26], The risk of febrile convulsions is increased in comparison to the trivalent vaccine.

All of these vaccinations are attenuated viral vaccines that reproduce inside the host to elicit protective immunity. The World Health Organization recommends the first dosage at 12 months and the subsequent dose at 15 to 18 months [27]. The CDC advises the first dosage be given at 12 to 15 months and the subsequent dose at 4 to 6 years in the United States. The WHO advises the administration of the first dose of the measles-containing vaccine (MCV1) at 9 months of age in certain circumstances when measles is prevalent. In exceptional circumstances such as epidemics, we should expect to deliver the first dosage at 6 months, which is especially relevant for displaced people or children with HIV infection [28]. The function of B cells in vaccination response remains incompletely understood. Memory B cells are essential for initiating a rapid humoral response upon exposure to the wild virus. Nonetheless, the activation is presumably predominantly induced by plasma cells stimulated by the antigen in the bone marrow, rather than by memory B cells [29].

9.1 What Is MMRV Vaccine?

The MMRV vaccination provides protection against four diseases: measles, mumps, rubella, and varicella (chickenpox). This vaccination is only for children aged 12 months to 12 years. [30]

The CDC advises that children get one dose of the MMRV vaccination between 12 and 15 months of age, followed by a second dose between 4 and 6 years of age [31].

Children may get the second dose of the MMRV vaccination prior to the age of 4–6 years. The second dose of the MMRV vaccination may be administered three months after the first dose. A physician may assist parents in determining whether to provide this vaccination or the MMR vaccine. [32] MMRV may be administered by injection concurrently with other vaccinations.

10. DIAGNOSIS OF MEASLES VIRUS:

The most common method of diagnosis is the identification of the measles virus in clinical specimens, which may include swabs taken from the throat, nasal passages, or urine samples. Using tests conducted in the laboratory. An assay known as the reverse transcription-polymerase chain reaction (RT-PCR) is a laboratory test that is often used for the purpose of diagnosing the measles virus. The test that was proposed is capable of identifying viral RNA in clinical samples, and it has a significantly high level of both sensitivity and specificity.

Furthermore, in addition to enzyme-linked immunosorbent assays (ELISA) and plaque reduction neutralization tests (PRNT), there are other laboratory procedures that may be used for the purpose of identifying the measles virus. Serum samples may be analyzed using these methods in order to discover antibodies that are specific to the measles virus. When it comes to diagnosing measles, the presence of clinical symptoms including fever, cough, rhinorrhea, and dermatitis, in addition to laboratory testing, may be helpful in determining the existence of the measles virus.

11. TREATMENT OF MEASLES VIRUS INFECTION”:

The primary method for treating an illness caused by the measles virus is to provide assistance, with a particular emphasis on the proper treatment of problems and the reduction of the risks of complications from subsequent infections. Vitamin A supplementation is recommended by the World Health Organization for all children who have been diagnosed with measles. This is due to the fact that vitamin A has been demonstrated to be useful in reducing the risk of sequelae, such as pneumonia and diarrhea.

There is ongoing debate on whether or not antiviral therapy with ribavirin and interferon is effective in treating severe instances of the virus. The use of immunoglobulin therapy has been shown to be successful in patients with weakened immune systems; however, the efficacy of this treatment in immunocompetent individuals is still limited.

12. CONCLUSION

The measles virus is an RNA virus that is extremely contagious and has the potential to cause severe morbidity and death, especially in communities that have a low percent of vaccines administered. The virus is characterized by its pleomorphic form and its intricate genomic organization, which functions to encode six structural proteins.

The interaction of the viral H protein with cellular receptors is the mechanism by which the measles virus enters host cells. This is then followed by the replication and assembly of new virus particles for the virus. When it comes to managing the illness and providing protection over the long term, the immunological response to the measles virus is very important.

The detection of the measles virus in clinical specimens by the use of laboratory testing is required for the diagnosis of measles virus infection. Clinical symptoms are also very helpful in making the diagnosis. Death, pneumonia, and encephalitis are just some of the serious complications that may arise from an infection caused by the measles virus. The vaccination of people, the maintenance of a high level of immunity throughout the community, and the early detection and isolation of sick persons are all key components of measles virus infection prevention. Despite the fact that there is a vaccination that is effective against measles, the virus continues to pose a substantial risk to public health. Recent outbreaks have been recorded in a number of different places throughout the world. There is presently no particular antiviral therapy available for the treatment of measles virus infection; hence, the primary treatment offered is supportive care.

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Conflicts of Interest:

The authors declare no potential conflicts of interest.

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Reference:

- [1] S. R. Permar, W. J. Moss, J. J. Ryon, M. Monze, F. Cutts, T. C. Quinn, and D. E. Griffin, "Prolonged measles virus shedding in human immunodeficiency virus-infected children, detected by reverse transcriptase-polymerase chain reaction," *J. Infect. Dis.*, vol. 183, pp. 532–538, 2001.
- [2] B. D. Dalziel, O. N. Bjørnstad, W. G. van Panhuis, D. S. Burke, C. J. E. Metcalf, and B. T. Grenfell, "Persistent chaos of measles epidemics in the prevaccination United States caused by a small change in seasonal transmission patterns," *PLoS Comput. Biol.*, vol. 12, no. 2, pp. e1004655, 2016.
- [3] N. López-Perea, A. Fernández-García, J. E. Echevarría, F. De Ory, M. Pérez-Olmeda, and J. Masa-Calles, "Measles in vaccinated people: Epidemiology and challenges in surveillance and diagnosis in the post-elimination phase. Spain, 2014–2020," *Viruses*, vol. 13, no. 10, pp. 1982, 2021.
- [4] S. K. Kasundriya, M. Dhaneria, A. Mathur, and A. Pathak, "Incidence and risk factors for severe pneumonia in children hospitalized with pneumonia in Ujjain, India," *Int. J. Environ. Res. Public Health*, vol. 17, no. 13, pp. 4637, 2020.
- [5] D. Manolescu, B. Timar, F. Bratosin, O. Rosca, C. Citu, and C. Oancea, "Predictors for COVID-19 complete remission with HRCT pattern evolution: A monocentric, prospective study," *Diagnostics*, vol. 12, no. 6, pp. 1397, 2022.
- [6] M. J. Mina, T. Kula, Y. Leng, M. Li, R. D. de Vries, M. Knip, H. Siljander, M. Rewers, D. F. Choy, M. S. Wilson, *et al.*, "Measles virus infection diminishes preexisting antibodies that offer protection from other pathogens," *Science*, vol. 366, pp. 599–606, 2019.
- [7] M. I. Toker, H. Erdem, H. Erdogan, M. K. Arici, A. Topalkara, O. S. Arslan, and A. Pahsa, "The effects of topical ketorolac and indomethacin on measles conjunctivitis: Randomized controlled trial," *American Journal of Ophthalmology*, vol. 141, no. 5, pp. 902–905.e1, 2006, doi: 10.1016/j.ajo.2005.12.004.
- [8] H. Fu *et al.*, "Impact and cost-effectiveness of measles vaccination through microarray patches in 70 low-income and middle-income countries: Mathematical modelling and early-stage economic evaluation," *The Lancet Global Health*, vol. 8, no. 11, 2023.
- [9] M. De Santis, A. F. Cavaliere, G. Straface, and A. Caruso, "Rubella infection in pregnancy," *Reproductive Toxicology*, vol. 21, no. 4, pp. 390–398, 2006, doi: 10.1016/j.reprotox.2005.01.014.
- [10] D. E. Griffin, W.-H. Lin, and C.-H. Pan, "Measles virus, immune control, and persistence," *FEMS Microbiology Reviews*, vol. 36, no. 3, pp. 649–662, 2012, doi: 10.1111/j.1574-6976.2012.00330.x.
- [11] R. D. de Vries, A. W. Mesman, T. B. H. Geijtenbeek, W. P. Duprex, and R. L. de Swart, "The pathogenesis of measles," *Curr. Opin. Virol.*, vol. 2, no. 3, pp. 248–255, 2018.
- [12] T. Nakayama, R. Fujisawa, and Y. Izumi, "Measles virus selectively blind to signaling lymphocyte activation molecule as a novel oncolytic virus for treatment of breast cancer," *Breast Cancer Res.*, vol. 20, no. 1, pp. 70, 2018.
- [13] M. J. Mina, T. Kula, Y. Leng, M. Li, R. D. de Vries, M. Knip, H. Siljander, M. Rewers, and D. F. Choy, "Measles virus infection diminishes preexisting antibodies that offer protection from other pathogens," *Science*, vol. 366, no. 6465, pp. 599–606, 2019, doi: 10.1126/science.aay6485.
- [14] S. Schneider-Schaulies and J. Schneider-Schaulies, "Measles virus-induced immunosuppression: From effectors to mechanisms," *Med. Microbiol. Immunol.*, vol. 199, no. 4, pp. 227–237, 2020.
- [15] J. B. Patterson, D. Thomas, H. Lewicki, M. A. Billeter, and M. B. Oldstone, "V and C proteins of measles virus function as virulence factors in vivo," *Virology*, vol. 267, pp. 80–89, 2000.
- [16] C. Escoffier, S. Manie, S. Vincent, C. P. Muller, M. Billeter, and C. Gerlier, "Nonstructural C protein is required for efficient measles virus replication in human peripheral blood cells," *J. Virol.*, vol. 73, pp. 1695–1168, 1999.
- [17] J. C. Bester, "Measles and measles vaccination," *JAMA Pediatrics*, vol. 170, no. 12, pp. 1209–1215, 2016, doi: 10.1001/jamapediatrics.2016.1787.
- [18] C. Gilbert, "The eye signs of vitamin A deficiency," *Community Eye Health*, vol. 26, no. 84, pp. 66–67, 2013.
- [19] R. Nwalozie, M. Uzoechi, R. K. Esiere, and B. A. Nnokam, "Biology of measles virus: Epidemiology and clinical manifestations," *International Journal of Pathogen Research*, vol. 12, no. 4, pp. 1–10, 2023, doi: 10.9734/IJPR/2023/v12i4231.
- [20] W. J. Bellini, J. S. Rota, and P. A. Rota, "Virology of measles virus," *The Journal of Infectious Diseases*, vol. 170, suppl. 1, pp. S15–S23, 1994, doi: 10.1093/infdis/170.Supplement_1.S15.
- [21] M. J. Mina, C. J. Metcalf, R. L. de Swart, A. D. Osterhaus, and B. T. Grenfell, "Long-term measles-induced immunomodulation increases overall childhood infectious disease mortality," *Science*, vol. 348, no. 6235, pp. 694–699, 2018.
- [22] L.-T. Lin *et al.*, "The Host Cell Receptors for Measles Virus and Their Interaction with the Viral Hemagglutinin (H) Protein," *Viruses*, vol. 8, no. 9, p. 250, 2016, doi: 10.3390/v8090250.
- [23] R. T. Perry and N. A. Halsey, "The clinical significance of measles: A review," *J. Infect. Dis.*, vol. 189, no. 1, pp. 4–16, 2018.
- [24] W. J. Moss and D. E. Griffin, "Global measles elimination," *Nat. Rev. Microbiol.*, vol. 4, pp. 900–908, 2006, doi: 10.1038/nrmicro1550.
- [25] H. A. Gans, A. M. Arvin, J. Galinus, L. Logan, R. DeHovitz, and Y. Maldonado, "Deficiency of the humoral immune response to measles vaccine in infants immunized at age 6 months," *JAMA*, vol. 280, pp. 527–532, 1998.
- [26] A. Radbruch, G. Muehlinghaus, E. O. Luger, A. Inamine, K. G. Smith, T. Dörner, and F. Hiepe, "Competence and competition: The challenge of becoming a long-lived plasma cell," *Nat. Rev. Immunol.*, vol. 6, pp. 741–750, 2006.
- [27] J. C. Bester, "Measles and measles vaccination: A review," *JAMA Pediatr.*, vol. 170, pp. 1209–1215, 2016.

- [28] E. Ortac Ersoy, M. D. Tanriover, S. Ocal, L. Ozisik, C. Inkaya, and A. Topeli, "Severe measles pneumonia in adults with respiratory failure: Role of ribavirin and high-dose vitamin A," *Clin. Respir. J.*, vol. 10, pp. 673–675, 2016.
- [29] I. H. Haralambieva, I. G. Ovsyannikova, V. S. Pankratz, R. B. Kennedy, R. M. Jacobson, and G. A. Poland, "The genetic basis for interindividual immune response variation to measles vaccine: New understanding and new vaccine approaches," *Expert Rev. Vaccines*, vol. 12, pp. 57–70, 2013.
- [30] L. M. Nic Lochlainn, B. de Gier, N. van der Maas, R. van Binnendijk, P. M. Strebel, T. Goodman, H. E. de Melker, W. J. Moss, and S. J. M. Hahne, "Effect of measles vaccination in infants younger than 9 months on the immune response to subsequent measles vaccine doses: A systematic review and meta-analysis," *Lancet Infect. Dis.*, vol. 19, pp. 1246–1254, 2019.
- [31] G. De Serres, N. Boulianne, F. Defay, N. Brousseau, M. Benoit, S. Lacoursiere, F. Guillemette, J. Soto, M. Ouakki, B. J. Ward, *et al.*, "Higher risk of measles when the first dose of a 2-dose schedule of measles vaccine is given at 12–14 months versus 15 months of age," *Clin. Infect. Dis.*, vol. 55, pp. 394–402, 2012.
- [32] A. L. Forni, N. W. Schluger, and R. B. Roberts, "Severe measles pneumonitis in adults: Evaluation of clinical characteristics and therapy with intravenous ribavirin," *Clin. Infect. Dis.*, vol. 19, pp. 454–462, 1994.