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A REVIEW ON "THE GLOBAL IMPACT OF RESPIRATORY SYNCYTIAL VIRUS (RSV): A COMPREHENSIVE REVIEW OF CURRENT KNOWLEDGE"

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ABSTARCT

Respiratory Syncytial Virus (RSV) is a form of a single-stranded RNA virus that has two subtypes; A and B, and it affects the respiratory tract which results in the patient spending time in a hospital. Those who are more vulnerable to this illness are the elderly, infants, and those suffering from weak immune symptoms. It accounts for bronchiolitis in young children and respiratory complications in older adults. One of the key characteristics of RSV is its capability of escaping any long-term immunity which results in continuous reinfections through different stages of life. As for subtype A, it is more virulent than B which means that RSV infection will be more severe. The replication cycle of RSV G and F must happen within the host cell membrane where they are released into the cytoplasm. Once it occurs, the virus can replicate and make new viral components. G and F serve as RSV surface glycoproteins which aid in the attachment of the host cell. At present, there are no direct antiviral therapies for RSV, and the treatment is primarily supportive, managing oxygen needs and providing mechanical ventilation in extreme cases. Developing types of treatment such as nebulized hypertonic saline, Heliox therapy, and

monoclons including Nirsevimab have the likely potential to reduce symptoms and provide infection prophylaxis to certain high-risk groups. The use of other antiviral agents such as Ribavirin is controversial because of possible toxic effects and alleged low efficacy. Additional treatment with corticosteroids and bronchodilators is of questionable clinical usefulness. Diagnostic techniques for RSV consist of antigen testing, PCR, and viral cultures, with PCR testing offering the best sensitivity and specificity. With all the advancements in scientific research, RSV is still a worldwide health issue, especially in less economically developed countries, and remains one of the leading causes of respiratory tract infections across the globe.

KEYWORDS: Respiratory syncytial virus, Viral cultures, Molecular testing, Antigen testing, Wheezing, Single-Stranded RNA, Respiratory infections, Hematopoietic stem cell, Immunoglobulins, Orthopneumovirus, Polymerase Chain Reaction.

INTRODUCTION

A communicable pathogen Respiratory Syncytial Virus (RSV), otherwise known as human respiratory syncytial virus (hRSV) and human orthopneumovirus, infects the respiratory tract. It is classified under negative-sense, single-stranded RNA viruses. 'Syncytial' comes from the Greek term "*syncytia*," meaning to spell out as formed from a union of cells. RSV is one of the most important causes of respiratory infection, particularly in younger children, the elderly population, and those with weakened immune systems.[1]

Respiratory syncytial virus (RSV) causes a large proportion of hospital admissions for children's respiratory problems. While re-infection is common throughout one's life, it is less severe in older children and adults. People of all ages can be affected by RSV, but infection is most prevalent in colder weather. It is a well-known cause of bronchiolitis in children, whereas adults usually present it as a cold. However, it may still lead to more severe complications in older adults or those with weakened immune systems, such as pneumonia.[2]

RSV can cause an outbreak in non-hospitals, hospitals and is highly contagious. The virus gets into the body through the eyes and/or the nose and has an effect on the epithelial cells in the airways (above and below) first. After this, airway inflammation, damage to cells, and an obstruction results. Viral cultures, molecular testing, and antigen testing can all help in RSV diagnosis.[3]

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Good hygiene, being away from individuals who have an infection, as well as vaccination helps in preventing RSV.[6] It has been proven that the virus mainly spreads through droplets and is released into the environment while talking, breathing, or coughing.[7] This means that using infection control measures that help prevent airborne transmission might be the answer. The first RSV vaccines were approved by the FDA in 2023, which includes Arexvy (GSK) and Abrysvo (Pfizer). Also, at risk infants can be protected from getting infected with RSV using monoclonal antibodies such as palivizumab and nirsevimab.[4] [5]

For critical RSV infection, treatment is primarily supportive, such as administering oxygen or, in some cases, employing continuous positive airway pressure (CPAP) or high-flow nasal oxygen. Some issues, like respiratory failure, may require intubation and ventilatory support.[8]

Discovery and History Pneumoviruses of RSV

The discovery of Respiratory Syncytial Virus (RSV) dates all the way back to 1956, when researchers isolated a virus from a chimp suffering from a respiratory illness, naming it Chimpanzee Coryza Agent (CCA). Then, in 1957, Robert M. Chanock discovered the same virus in children suffering from respiratory infections, leading to further research which confirmed RSV to be a commonplace infection among children.[9] The older name was changed to Human Orthopneumovirus, later known as Human Respiratory Syncytial Virus (hRSV).[10] Bovine Respiratory Syncytial Virus (BRSV) is the most similar virus, sharing around 80% of its genetic makeup with hRSV. Unlike hRSV, though, BRSV primarily infects calves less than six months of age, causing severe respiratory disease. BRSV strikes a striking resemblance to hRSV; hence, it serves as an important animal model for studying RSV pathogenesis and developing potential treatments and vaccines.[11] [12]

MECHANISMS

Transmission of RSV

Respiratory Syncytial Virus (RSV) is a highly infectious disease that can spread rapidly in the community or within healthcare services. One RSV infected individual can, on average, infect anything from 5 to 25 other individuals.[13] The primary route of transmission of RSV is through coughing and sneezing which releases droplets containing the virus. These droplets can enter an individual's body through the mouth, nose or even the eyes. Recent studies, however, suggest that RSV could also be transmissible through the air in fine aerosols that are produced while breathing, talking, or even singing.[7]

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The virus is capable of persisting on various surfaces for a prolonged duration, being active for around 25 minutes on skin (hands) and hours on items such as doorknobs and countertops. The incubation period for RSV is usually from 2 to 8 days, and it can stay contagious for another 3 to 8 days. However, in infants and immunocompromised individuals, this duration may increase to four weeks of continuous spread potential even when they show no symptoms.[14]

Pathogenesis of RSV Infection

Following entry through the nose or eyes, RSV primarily infects the ciliated columnar epithelial cells of the upper and lower airways. Within these cells, the virus replicates over an approximately eight-day period.[3] During this time, the cells gradually lose their shape and function. These cells, once damaged, disassociate from the border and swim down into the smaller bronchioles of the lower respiratory tract. It is believed that this process aids RSV's progression from the upper to lower airways.

As a response to RSV, the lungs undergo almost complete inflammation. As a result, the immune system's monocytes and T-cells get activated and start to infiltrate the area. This response is further accompanied by some degree of epithelial cell disruption, edema, and heightened mucosal secretion. RSV is not like other types of infections which show a more homogenous pattern of lung damage. Patchy areas of destruction are the hallmark of the infection. The accumulation of sloughed epithelial cells, mucus, and immune cells form the mucus plug, and in the bronchi, these partially obstructing structures are referred to as bronchiolar "mucus plugs," which is a major factor in decreased airflow and increased work of breathing for these patients.[1]

Reinfection

Post recovery from the RSV infection, the adaptive immune system fails to mount an enduring immune response, and so lifelong RSV reinfection is common.

This happens as a consequence of RSV preventing the body from forming the essential immunological memory, leading to weak antibody and cell mediated immune responses. Research indicates that approximately 36% of people suffer at least one reinfection during the winter months.[15]

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Reinfections of RSV add to seasonal infectious outbreaks, especially during the winter and early spring in temperate areas, although they can differ depending on the region. During the cold season healthy adults experience mild upper respiratory infections and accompanying symptoms. Infants and younger children on the other hand have a significantly greater chance of suffering from severe lower respiratory infections due to small airway narrows that easily get inflamed, swollen and clogged by mucus. Furthermore, early life RSV reinfection may affect the child's lung function later in life and contribute to the development of constant wheezing and asthma.[16]

Immune Evasion

RSV has developed different genetic strains that help it to avoid detection by the immune system, preventing the body from establishing a solid protective defence. Alterations of the bacterial infection surface proteins G, SH and F completely modifies the virus's relation to the immune system.

This characteristic makes the G protein more susceptible to conformational changes that can prevent the triggering of an immune response. These changes also assist RSV in evading capture and reinfection.

Novel RSV genotypes like ON1 and BA9 have appeared with new features, specifically, changes to the G protein which may improve immune evasion. Additionally, the F protein, a predominant neutralizing target, is known to change and also reduce the effectiveness of antibody targeting such as Palivizumab. Therefore, RSV tends to elicit immune responses that are tailored to specific genotypes, making it difficult to achieve universal immunity across all strains.

Apart from genetic changes, RSV has many other methods of modulating the immune response to disable the host's antiviral defences. Over 50% of its proteins manipulate immune functions by antagonizing the cytokine signalling and the activation of immune cells. This capacity to avoid immune detection explains RSV's ability to inflict only mild symptoms on the general population, while becoming extremely dangerous to infants, the elderly, and those with weakened immune systems, potentially resulting in life-threatening lung diseases.[17]

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VIROLOGY

****Taxonomy of the Respiratory Syncytial Virus (RSV)****

The respiratory syncytial virus (RSV) is classified as a single stranded RNA virus that possesses a negative sense. Its scientific name is human orthopneumovirus, which is at times used interchangeably with (hRSV)—better known as RSV.[18] This virus is placed under the genus Orthopneumovirus, with the family Pneumoviridae and order Mononegaviral. The naming of the term "respiratory syncytial virus" has further been defined due to its distinctive feature of fusing the membranes of adjacent cells to form large multinucleated cells referred to as syncytia. F proteins that are on the virus surface cause this fusion.[3]

Antigenic Subtypes of RSV

RSV is divided into two antigenic subtypes A and B based on the differences in the F (fusion) and G (attachment) proteins of the virus' surface when exposed to monoclonal antibodies. Both subtypes circulate at the same time within a region in times of RSV outbreak, but RSV subtype A (RSVA) tends to be more predominant which is considered to be more virulent in nature than RSV subtype B (RSVB).[19] RSVA tend to have a greater viral load which leads to more rapid spread.[3]

So far researchers have noted 16 clades of RSVA and 22 clades of RSVB. For RSVA, the clades of GA1, GA2, GA5 and GA7 are most frequently found, while GA7 only in United States. For RSVB, BA clade appears to be most common across the globe.[1]

Genomic Structure of RSV

The genome of the Respiratory Syncytial Virus (RSV) is segmented into an approximate length of 15,000 nucleotides, single-stranded RNA (negative sense type) and encodes eleven proteins via 10 genes.

The gene sequence of RSV includes the following: NS1-NS2-N-P-M-SH-G-F-M2-L. NS1 and NS2 function as non-structural structural components of the virus, serving for regulating the replication process and immune system avoidance, also known as immune evasion.[20]

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[Figure 1].

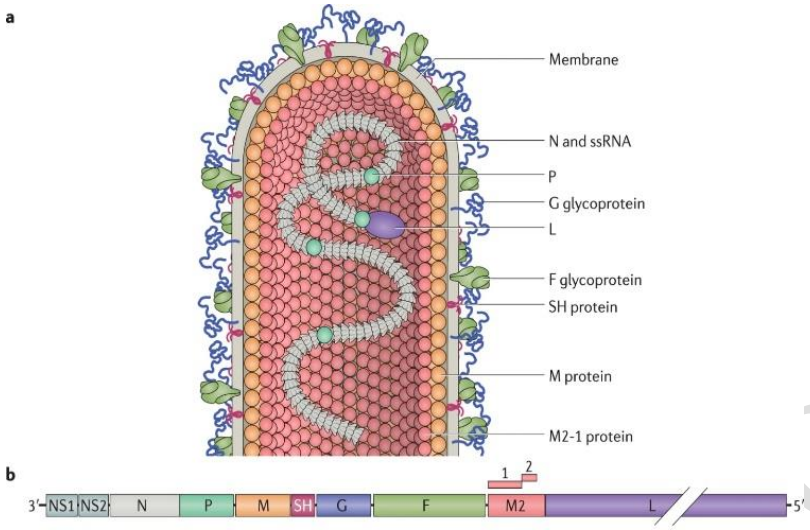

Figure 1. Schematic structure and genome of RSV.

Structure of RSV

As for the Respiratory Syncytial Virus (RSV), it is classified as an enveloped viral medium sized, with a diameter that measures about 150 nanometres. Majority of RSV particles hold a spherical shape but can also appear resemble in a filamentous form.

The viral genome consists of a helical nucleocapsid which synthesizes a hallowed midsection that is uncaptured in a matrix look like protein. Such feature is surrounded by a lipid envelope containing the viral glycoproteins crucial for host cell consummation aiding in permission attachment and infection.[21]

Role of the G Glycoprotein in RSV Infection

In RSV, the host cell attachment is primarily carried out through the action of G glycoprotein which binds membrane to the cells. It takes on two distinguishing approaches between different strains of RSV, existing as a bound form and a secret one bound to a membrane.

Entry of the virus into the host cell is facilitated by the membrane-bound G protein that binds glycosaminoglycans (GAGs) like heparan sulphate on the cell surface. The decoy form of the protein inhibits antigen-presenting cells mediating neutralization which helps the virus avoid destruction by antibodies.[3]

Moreover, the G protein possesses CX3C fractalkine motif which binds with CX3C chemokine receptor 1 (CX3CR1) on ciliated bronchial cells. This bond has the potential to

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impair the positively-charged white blood cells' trafficking to the lungs, thus restricting entry to the lungs even when there is infection. Also, the G protein can further allow RSV to escape defences by suppressing immune response through TLR signalling, which is especially TLR4, and RSV is able to avoid the defences of the host.[22]

Function of F protein in RSV infection

Both F and G proteins perform their own unique functions and have specific structures G protein is has the most distinctive roles in attachment while the fusion F (fusion) protein enable the RSV to enter the host to fuse their cellular membranes with those of the host, G protein facilitates entry of RSV.[19]

This protein displays a notable degree of preservation on RSV strains and can be found in several form conformational states. The F protein is in a pre-fusion (Pre F) state and is formed as a trimeric structure. Pre F contains the major antigenic site, Ø, which is the predominant neutralizing antibody target held by the defender system. The virus binds with a receptor on the host cell and during this step a ligand is unknown. In this process, Pre F is said to change and as a result, Ø which is absent is released. This change enables the protein to perform transverse into the host cell membrane, which is said to induce fusion of the viral and host membranes. The last step is then reaching a more stable elongated form of post-fusion (PostF) at which point the protein has rearranged itself.

The F protein instead of doing what the G protein does, which is blocking some immune scallops, makes active efforts to find and attach to TLR 4. This binding results to triggering signalled actions and inflammation which are parts of innate immunity.[22]

Replication Cycle of RSV

As soon as both the host and the viral membranes become one, the viral nucleocapsid that contains am RSV genome and a viral polymerase which is used for transcribing purposes are set free and can be found in the cytoplasm of the host cell. The intercellular machinery furnished by the cell is employed for transcription and translation, these procedures are carried out in the cytoplasm.

After the virus attaches itself to the host's cellular machinery, its membrane fuses with the host's, allowing the RSV genome and the associated viral polymerase trapped within the viral

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nucleocapsid to be set free into the host cell's cytoplasm. The host 's machinery fulfils its duties within the cytoplasm, executing both transcription and translation.

An RNA-dependent RNA polymerase (RdRp) transforms the negative-sense RNA genome into ten fragments of messenger RNA (mRNA). These mRNA fragments serve as templates and are transformed into a variety of structural proteins that are essential for the virus assembly.

In ruder to replicate the genome, RdRp synthesizes an antecedent strand termed the antigenome, which is a positive-sense complement. This strand is later used to synthesize new genomic RNA, which renders the virus nucleate red capsids, now containing newly synthesized viral genomes, placed into the plasma membrane where they get assembled into new virus particles. The obliteration of the host cell is then followed by RSV's readiness to infect new cells.[22] [Figure.2]

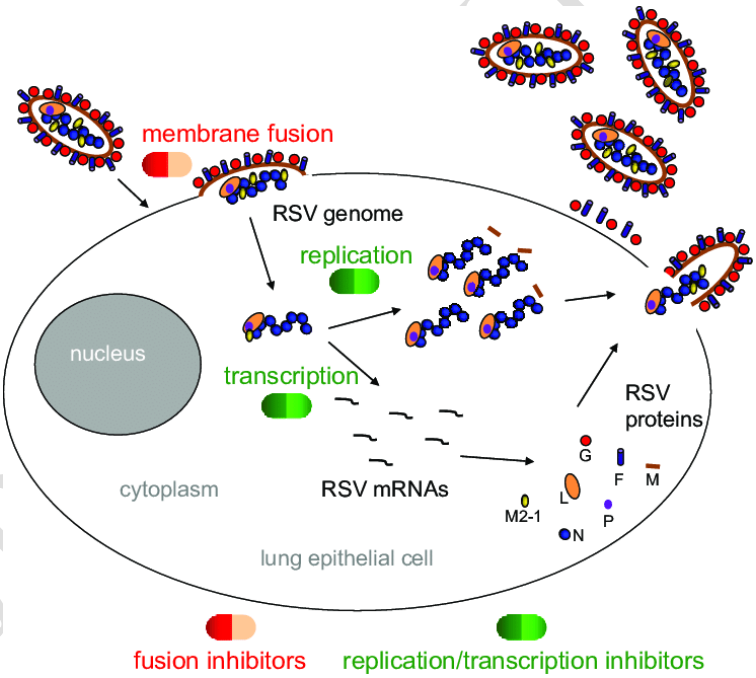

Figure.2 RSV Replication cycle.

EPIDERMIOLOGY

RSV in Infants and Children

As regions of the world Respiratory Syncytial Virus (RSV) remains the most prevalent cause behind bronchiole inflammation and pneumonia in young children and infants globally. Children below the age of five are the most affected demographic. Being the critical point in

life within the initial six months, a whopping two to three percent of newly infected infants suffering from severe bronchiolitis requiring hospitalization are admitted within the first year of life.[23]

Worldwide, RSV is linked to an estimated 30 million acute respiratory infections and more than 60,000 childhood fatalities each year. Infants are almost universally infected with RSV by the time they turn three, with an expected outbreak of 87% by 18 months. In the US, RSV is associated with up to 20% of hospital admissions for acute respiratory infections in children aged under five. The majority of RSV related fatalities occur in economically disadvantaged nations with limited medical resources.[24]

Palivizumab and nirsevimab can be used as preventative care against RSV infection in infants, providing passive immunity and minimizing the chances of severe disease and hospitalization.

A study conducted in 2024 by JAMA Open raised the possibility of a link between the RSV surge during the years of 2021 and an increase in SUID, or sudden unanticipated infant death. Looking through CDC data, researchers analysed over 14,000 SUID cases, noting that there was a rise in the SUID rate from 2019 to 2021, going from 10% per 100,000 live births to 100,000 live births. The surge in the unexplained SUIDs occurred between June to December of 2021, coinciding with an atypical surge in RSV cases usually observed in winter, following the COVID-19 pandemic.[25]

RSV in Adults

For healthy young adults, RSV infections are often mild and seldom require hospitalization. Nevertheless, the virus presents a serious concern for some other adult populations, especially older adults and those suffering from chronic heart or lung diseases. The consequences of RSV in the elderly are considered similar to the impacts of influenza as it contributes heavily to morbidity and mortality in this population.

Among nursing home residents, RSV is diagnosed in 5-10% of individuals each year and often leads to serious complications such as pneumonia or death. Notably, RSV accounts for 2-5% of community-acquired pneumonia cases in adults, which underscores the significance of this virus as an illness beyond childhood infections.[19]

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RSV in Compromised Immune System Patients

Children and adults with compromised immune systems are more susceptible to the severe complications of RSV. When considering Paediatric patients with HIV, acute illness is much more common; recoverable children with HIV are 3.5 times more likely to be hospitalized than children without the disease. Among patients with bone marrow transplants, the risk is particularly high pre-engagement as RSV is the culprit for almost half of the viral infections in this population. The mortality rates when RSV is the cause of pneumonia can be as high as 80%.

The majority of respiratory syncytial virus (RSV) infections are acquired in community settings, but 30–50% of RSV cases are thought to be hospital-acquired in people with weakened immune systems, underscoring the need for stringent infection control in those settings.[19]

Seasonality

Each subtropical and tropical region of the world has its peak season for RSV outbreaks. It peaks in January in colder regions, likely as a result of increased indoor crowding and the virus's stability in lower temperatures. In arctic and tropical regions, outbreaks are unpredictable and usually occur during the rainy season.

The emergence of multiple viral strains each year causes subtype A and B RSV epidemics. Both subtypes A and B viruses are present in a given region, but subtype A is usually dominant.

Respiratory syncytial virus RSV is one of the leading causes of respiratory infections for young children, older adults, and those with weak immune systems. There is no direct antiviral medication for RSV, only supportive care. In this article, the different strategies for supportive RSV infection management, both traditional and new, will be presented.[3]

General Care Measures that Provide Support

Supportive RSV infection care highly emphasizes on symptom alleviation as well as making sure of adequate oxygen supply. Such care entails the following measures:

Airway Mobilization: Infants may have improved breathing after suctioning of nasal and upper airway secretions.

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Oxygen Therapy: Patients in oxygenated distress may need oxygen supplementation via nasal cannula or face mask to during respiratory clearance.

Mechanical Ventilation: In some instances, with extreme respiratory failure, intubation and mechanical ventilation might be needed for respiration support.

Fluid Management: Maintaining fluid balance is essential while hydration is provided either orally or through IV if dehydration is present.[26]

Another Care Treatment Currently Under Research

In attempts to change the prognosis of hospitalized patients suffering from RSV bronchiolitis, some additional therapies have been developed.

Nebulized Hypertonic Saline

The use of nebulized hypertonic saline among infants with viral bronchiolitis may aid in decreasing the hospitalization period and overall severity of the illness. The principal mechanism of this kind of treatment is thought to be obstruction relief by reduction of airway swelling and mucus blockage.[27] [28]

Heliox Therapy

Inhaling helium-oxygen mixture, also known as Heliox, offers some respite from breathing difficulty as it relaxes the airways and decreases the work of breathing. Although helpful in the short term, studies suggest that within one hour of treatment, no significant change in the disease outcome is noted on the disease's course.[29]

Chest Physiotherapy

The effectiveness of chest physiotherapy on RSV management is still not established. Some studies suggest that combined physiotherapy techniques do not considerably alleviate the severity of the disease. Other forms of physiotherapy such as instrumental physiotherapy and the Rhino pharyngeal Retrograde Technique (RRT) need more research. There is some evidence, albeit weak, that has shown modest to significant benefits from passive slow-expiratory techniques in hospitalized infants, but in outpatient settings, the results remain ambiguous.[30]

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Inhaled Recombinant Human DNase (rhDNase)

This enzyme that helps liquefy mucus by hydrolysing extracellular DNA does not appear to offer notable clinical advantages for patients with RSV infection.

Viral-Specific Treatments for RSV Infection

Ribavirin: An Approved but Controversial Treatment

Children suffering from RSV infections can be treated with ribavirin, an antiviral medication. Its effectiveness is the same as that of guanosine cap's formation capping and viral RNA replication blockade. While the drug was approved in 1986, it remains a controversial topic to this day because of concerns regarding its efficacy, the probable toxic effects of aerosolized ribavirin on the nursing personnel, and its exorbitant price. Due to all these factors, current treatment guidelines do not recommend ribavirin for Paediatric RSV infections.

Ribavirin is not officially recognized as a treatment for RSV in adults; however, it is occasionally prescribed off-label. Its use is largely restricted to someone who is immunosuppressed, like a patient recovering from a hematopoietic stem cell transplant, as they stand to incur severe complications from RSV.[31]

Presatovir: An Emerging Antiviral Therapy

Presatovir is a candidate antiviral agent which is undergoing further development as part of clinical trials. It works as a fusion inhibitor, blocking RSV entry into cells by targeting the RSV F protein. Further studies are needed to determine the safety and effectiveness of presatovir, and in the interim, it is not approved for medical use.[32]

The Use of Immunoglobulins in Managing RSV

In the past, there were considerations both for RSV-specific and non-specific immunoglobulins to be utilized in treating RSV infection. Nevertheless, it seems that modern research does not offer adequate backing for the use of these immunoglobulins in Paediatric RSV cases. There are instances where they have been used, but in general, their routine use remains unsupported.[33]

Anti Inflammatory Therapies

There is no evidence of corticosteroids, whether inhaled or systemic, reducing the severity of RSV bronchiolitis or shortening the duration of hospitalization. Moreover, their application might extend the period of viral shedding which is dangerous due to the increase in infectious

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period. Hence, corticosteroids are not recommended as a matter of practice for RSV treatment. On the contrary, the use of oral corticosteroids is still commonplace in the management of RSV exacerbation in patients with chronic lung diseases.[34]

Leukotriene receptor antagonists, like montelukast, have been investigated for use in the treatment of bronchiolitis for young children. However, the current body of research does not support this claim, and there is no conclusive evidence available that supports the idea of them being effective in the treatment of respiratory symptoms due to RSV.[35]

Bronchodilators in RSV Treatment

For the treatment of bronchial asthma, bronchodilators are commonly used. These medications are sometimes prescribed to manage RSV-related wheezing as well. Albuterol (salbutamol) and other beta-agonists are prescribed in these situations because they widen muscle spasms of the airways to allow greater movement of air. Despite the use of these bronchodilators, there is no marked improvement in clinical outcomes, hospitalization rates, or RSV infection severity. Due to the lack of benefits, bronchodilators are not routinely recommended in the treatment of RSV bronchiolitis.[34]

The Role of Antibiotics

Antibiotic therapy has no effect on RSV infection as antibiotics only work on bacterial diseases. Thus, antibiotic therapy for bronchiolitis RSV or viral pneumonia is not indicated. However, if a secondary bacterial infection such as bacterial pneumonia or an ear infection is present, then antibiotics will be prescribed. In some children suffering from RSV bronchiolitis, there can be associated secondary ear infections for which the treatment of choice is oral antibiotics.[36]

Preventing RSV with Monoclonal Antibodies

Apart from vaccines, monoclonal antibodies have come forth as a RSV infection preventive strategy. Provided monoclonal antibodies by AstraZeneca and Sanofi, Nirsevimab has shown 75% efficacy in preventing RSV infections in infants below one year of age. It has regulatory approval in Europe as of November 2022 and FDA approval in July 2023. Furthermore, Merck's monoclonal antibody, clesrovimab, currently in the later stages of clinical trials, may serve as an additional preventive measure in the coming future.[37]

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DIAGNOSIS

A number of laboratory tests can be performed to identify RSV infection. As noted by the American Academy of Paediatrics, routine laboratory testing for RSV bronchiolitis is generally not indicated because the management is supportive (i.e., no specific treatment). However, testing may be appropriate for lower-risk patients when the outcome will change management. Common RSV identification methods include antigen detection tests, molecular testing (PCR), and viral culture.[6]

RSV Diagnosis Utilizing Antigen Tests

An antigen test is performed to diagnose RSV by identifying specific viral antigen pieces that are usually obtained from a nasopharyngeal swab or aspirate. RSV can also be diagnosed by direct fluorescence assay (DFA); where examination is done through a microscope, fluorescently containing antigens are labelled to determine if they are present. Another approach is through a rapid antigen detection test (RADT) which uses commercially available kits for faster results.[3]

This method of testing works particularly well in younger children because sensitivity rests on 80-90%. In older children and adults, the reliability decreases because there is lower viral shedding. Furthermore, during off peak hours of RSV season, there is a greater chance the antigen tests will provide false positive results. In such cases, much more accurate methods of diagnosis like viral culture or NAAT (nucleic acid amplification testing) may be used to accurately confirm an RSV infection.[17]

Molecular Testing

Molecular methods of testing, such as nucleic acid amplification tests (NAATs), are very sensitive and are used to identify a viral fragment's genetic material often found in specimens such as nasopharyngeal swabs or aspirates. Unlike antigen tests which search for viral proteins, NAATs such as Polymerase chain reaction (PCR) tests identify the genetic material of a virus. These tests are known to have a high degree of sensitivity and specificity, (often) almost 100%. However, they are more expensive and require advanced equipment, thus, their use is not feasible in low-resource settings.

However, routine molecular testing for RSV is not recommended for all patients with respiratory symptoms but is often considered for those with a high risk of complications like infants, elders, or patients suffering from chronic illnesses.

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Among NAATs, one of the best-known examples is RT-PCR (Reverse Transcription PCR) which has a sensitivity of 90–95% and specificity of 98–99%. Another technique, which is also highly accurate but less well known, is called LAMP (Loop-mediated Isothermal Amplification), which can have a sensitivity between 95-100% and specificity between 99-100%.

PCR detects infections by taking small amounts of DNA or RNA from a virus and multiplying it into millions of identical copies that are easier to detect. This is more sensitive than viral cultures or antigen tests, detecting infections in individuals with lower viral loads like older children or adults. In high-risk patients, such as those who are hospitalized or immunocompromised, PCR does a better job of early detection because traditional tests may not be able to detect the low viral load these patients have. Due to the nature of the test, PCR can further be sensitive enough to detect traces of the virus in asymptomatic individuals or after the infection has resolved.[38]

Viral Culture

This technique involves an introductory step where a suspected sample containing a virus is put into specific cell lines. Traditional methods of laboratories allow a virus to replicate so that they can be studied in more detail later during the research. Unlike other modern techniques, viral culture can enable the genetic characterization of a virus and check for varying strains as well as test how effective antiviral drugs are with the virus.

Viral culture has its perks, yet it is not widely adopted in routine clinical practice because of its slow turnaround time of 3 to 7 days. This lag means it is largely confined to research settings rather than providing timely diagnosis and management for patients.[3]

Serologic testing

Serologic testing's application in RSV diagnostics tends to be non-existent as it generally leads to unfavourable treatment outcomes. The reason lies in the time it takes for the body to mount an antibody response, which is often impractical in 'time-critical' scenarios.

Interestingly, roughly 30% of patients who have been diagnosed with RSV infection will still be serologically tested negative. That said, the lack of versatility with such a test does raise eyebrows, as serological testing is primarily reserved for research epidemiology or monitoring population trends.[38]

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CONCLUSION

In closing, RSV (Respiratory Syncytial Virus) continues to endanger public health on a global scale, especially for at-risk populations like babies, elderly individuals, and those with weak immune systems. RSV still poses a threat for severe respiratory illnesses and recurrent infections due to the diverse immune-evasive strategies, genetic variability, and multifaceted development pathways. The factors responsible for the virus's evasion of immune surveillance through squamous structure modifications alongside nutrient stasis add to the difficulty in controlling the virus's spread. Although supportive care remains the most commonly used treatment, more research RSV related illness focused on antiviral medications, better vaccines and monoclonal antibody treatments may ease the associated burden with RSV. The RSV research aimed at creating more effective preventative and treatment solutions needs further development due to barriers like seasonal inconsistency and absence of targeted antiviral medications.

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