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A Review on Viral Diseases Prevention and Management in the Community

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A B S T R A C T
Viral infections are important causes of disease of the respiratory tract. The common cold is the most frequently encountered infectious syndrome of humans, while influenza continues to be a major cause of mortality and serious morbidity worldwide. Respiratory viral infections frequently complicate the course of patients with chronic obstructive pulmonary disease and asthma. Immunocompromised persons in the population has increased, infections due to cytomegalovirus and other herpes viruses, adenoviruses, and paramyxoviruses. A proper understanding of the biological mechanisms underlying RNAi lags behind the movement to apply this technology to human diseases such as viral infections. Despite increasing availability of rapid nucleic acid amplification assays for laboratory diagnosis, effective antivirals, and safe vaccines, the control of most viral infections depends on time-honored surveillance and infection control measures. Moreover most viruses can be readily destroyed by common disinfectants and proper follow of infection preventive mesures colud lower the viral diseases spreading count in the community.

Keywords: Viral infections, Respiratory tract, Cytomegalovirus, Biological mechanisms, infection control measures.

A R T I C L E I N F O

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C O N T E N T S

1. Introduction

A landmark in the battle against viral infectious diseases was made in 1798 when Jenner first inoculated humans against smallpox with the less virulent cowpox. For about two centuries since then, humans relied almost exclusively on vaccines for protection against viruses. To differentiate them from the conventional vaccines that prevent viral infection by boosting immune system, we refer the new antiviral approaches as "Biochemical Prevention and Treatment". Biochemical Prevention and Treatment, as an alternative to vaccines and chemical compound based antiviral drugs, may prove to be particularly valuable in the areas where vaccines and/or chemical drugs cannot be generated or have not been successful in human, including diseases caused by some common pathogenic viruses, such as HIV, hepatitis C virus (HCV), RSV and human rhinovirus (HRV) etc.

Biochemical Prevention and Treatment via Protein targeting: Among the biochemical therapeutics currently in clinical trials, the majority consists of monoclonal antibodies (MAbs). Soluble receptor drug candidates have gradually lost favor over the past several years due to issues relating to low potency and cost. Peptide-based drug candidates are limited by insufficient efficacy and unfavorable pharmacokinetics. MAbs have increasingly gained favor in large part because of the development of chimeric, humanized, and human antibodies have reduced the immunogenicity of antibody therapies.

Prevention of Human Rhinovirus infections

Human rhinovirus (HRV) causes over 80% of the common cold in the fall. Developing vaccines against HRV is unfeasible because HRVs have at least 115 antigenically distinct serotypes. One of the proven methods to prevent and inhibit viral infections is to block host cell receptors that are used by viruses to gain cell entry. Receptor blockage is commonly achieved via application of MAbs that bind to specific epitopes on the receptor molecules. A plethora of *in vitro* studies have reported effective viral inhibition by receptor-blocking MAbs. However, these works have not yielded yet any approved drug on the market[1].

Biochemical Prevention and Treatment via targeting on viral mRNA: Targeting viral mRNA is one of the most active areas of research and development. Several strategies have emerged over the years and are being tested pre-clinically and clinically. They include: antisense-oligonucleotides (AS-ONs), ribozymes, and recently, RNA interference (RNAi). All these strategies share the features of conceptual simplicity, straightforward drug design and quick route to identify drug leads. However, the challenges have been to improve potency, pharmacokinetics and, most importantly, intracellular delivery of the drug candidates.

Antisense-oligonucleotides:

Antisense-oligonucleotides are short synthetic oligonucleotides that form complementary pair with specific viral mRNA targets. Since the discovery of viral inhibition effect of AS-ONs by Zamecnik and Stephenson in 1978, antisense technology has been developed as a powerful tool for target validation and therapeutic purposes. Phosphorothioate (PS) oligodeoxynucleotides are the *'first generation'* DNA analogs. The *'second generation'* ONs contain nucleotides with alkyl modifications at the 2' position of the ribose. For instance, peptide nucleic acids and their analogs display superior sequence specificity and are resistant to nuclease degradation. These third generation AS-ON have limited non-specific interactions with other genes and, therefore, have shown great potentials in clinical trials.

Ribozymes

Ribozymes (Rz) are catalytically active ONs that both bind and cleave target RNAs. They were discovered after the AS-ON technology. Initial findings on ribozymes raised the hope that they may offer a more potent alternative to AS-ONs. Many cell based and animal tests have performed on anti-viral effects of ribozymes, including HIV, hepatitis B, hepatitis C, influenza, etc[2-6].

RNA Interference (RNAi)

RNA interference, or RNAi, is the inhibition of expression of specific genes by double-stranded RNAs (dsRNAs). It is becoming the method of choice to knockdown gene expression rapidly and robustly in mammalian cells.

Classification: Virus classification depends in part on the type and configuration of the nucleic acid in the viral genome, the characteristics of the viral structural proteins, and the presence or absence of an envelope surrounding the virus particle. The number of distinct antigenic types within each of the virus families also varies.

Transmission

The routes by which the different respiratory viruses spread from person to person are variable and include combinations of contact, droplet, and aerosol transmission. For example, rhinovirus and *respiratory syncytial virus* (RSV) are primarily spread by direct contact with contaminated skin and environmental surfaces followed by self-inoculation of virus onto the nasal mucosa or conjunctiva.

Pathogenesis of Infection

The initial sites of infection and pathogenesis differ for the various virus groups. Some, such as rhinovirus, are associated mainly with upper respiratory tract involvement. Others, such as influenza, commonly invade the lower airways and sometimes pulmonary parenchyma in addition to causing upper airway disease. The viruses also differ in the relative contributions to the clinical manifestations of disease from damage due to direct viral mechanisms and damage due to host immune responses and inflammation.

Clinical Syndromes

Similarly, a particular syndrome can result from infection with different viruses. Moreover, infection with a single virus may cause disease at multiple levels of the respiratory tract.

Common Cold

Systemic complaints are absent or modest in severity and fever is unusual. Allergic diseases of the upper airway often have clinical manifestations similar to those of colds. Colds are frequently associated with involvement of the middle ear, likely due to eustachian tube dysfunction.

Pharyngitis

In some cases, pharyngeal symptoms predominate to a degree that overshadows other complaints. The kinins are potent stimulators of pain nerve endings, and high levels of bradykinin and lysylbradykinin are present in nasal secretions of patients with rhinovirus-induced colds.

Acute Bronchitis

The diagnosis of acute bronchitis is usually applied to cases of acute respiratory disease with severe and prolonged cough that continues after other signs and symptoms of the acute infection have subsided. Adenovirus infections characteristically involve the tracheobronchial tree, with resultant bronchitis that, in military populations, is part of the syndrome of acute respiratory disease.

Influenza-Like Illness

The clinical syndrome of influenza is characterized by the rapid onset of constitutional symptoms, including fever, chills, prostration, muscle ache, and headache, concurrent with or followed by upper and lower respiratory tract symptoms. Photophobia, excess tearing, and pain with eye movement are common early in the illness. Mild conjunctivitis, clear nasal discharge without obstruction, pharyngeal injection, and small tender cervical lymph nodes are frequently present. Fever may peak at 39° C to 40° C or higher and can last for 1 to 5 days. Persistent nonproductive cough, easy fatigability, and asthenia are common in the second week of illness. However, the syndrome can also be seen in association with infection by other viruses, including adenovirus, parainfluenza, and RSV[7-9].

Bronchiolitis

Bronchiolitis is an acute inflammatory disorder of the small airways characterized by obstruction with “air trapping,” hyperinflation of the lungs, and atelectasis typically seen in children younger than 2 years.

Pneumonia

Viruses are important causes of pneumonia in both adults and children. These figures may underestimate the importance of viral infections as a cause of pneumonia, particularly in outpatients, because of the insensitivity of viral diagnostic methods and because of the lack of chest radiographs in many patients with acute viral infections.

Immunocompromised Host

Viral pneumonia can be an important problem for the increasing number of persons in the population who have deficiencies in immunity as the result of cytotoxic chemotherapy, organ transplantation, and the *acquired immunodeficiency syndrome*. The major respiratory viruses that affect normal persons may also cause pneumonia in impaired hosts; severe and prolonged pneumonias due to adenovirus, respiratory syncytial, influenza, measles, or parainfluenza virus can develop in such patients.

2. Major Viral Pathogens

Adenovirus

Currently, 47 antigenic types of adenovirus are associated with human infection, although not all types have been associated with human disease. The protein coat of the virus is composed of 252 hexagonal and pentagonal capsomeres in an icosahedral array with long projecting fibers at each vertex. These fibers are thought to be the site of host cell attachment. Adenoviruses type 2 and 5 and coxsackie B viruses use the same receptor, designated the *coxsackie virus and adenovirus receptor* whose usual function is to mediate cell interactions with extracellular matrix proteins.

Epidemiology and Transmission

The adenoviruses that cause human disease do not have nonhuman reservoirs, although nonhuman adenoviruses are found in other species.

Clinical Illness

Adenovirus Respiratory Disease.

The nonpneumonic respiratory syndromes associated with adenovirus infection include acute respiratory disease of military recruits and pharyngoconjunctival fever of civilians, which have similar characteristics. Adenovirus respiratory disease typically involves the pharynx as a moderate to severe, sometimes purulent, pharyngitis. Conjunctivitis is not a feature of infection with the other major respiratory viruses and therefore, when present, is a useful diagnostic finding in adenovirus respiratory disease. Fever, chills, myalgia, and prostration are prominent features of adenovirus infection, so it is often perceived by the patient as a "flulike" illness or an unusually severe cold.

Adenovirus Pneumonia.

Adenovirus was first recognized as a cause of viral pneumonia in military recruits and has since been recognized as a rare cause of pneumonia in civilian adults and children. The clinical characteristics of adenovirus pneumonia are similar to those of other pneumonias, so it is difficult to make an accurate etiologic diagnosis on the basis of clinical features. In fatal cases, there has been extensive pulmonary damage, with death intervening 2 to 3 weeks into the illness. Intravascular coagulopathy has also been a late feature of some cases, and a septic shock picture has been described.

Coronaviruses

Coronaviruses are enveloped viruses containing a single-stranded, positive-sense *ribonucleic acid* (RNA) genome of approximately 29,000 nucleotides. Distinctive club-shaped projections are present on the virus surface, giving the appearance of having a crown or corona, from which it derives its name. Coronaviruses are classified into four genera: alpha, beta, gamma, and delta.

Epidemiology and Transmission

Human coronaviruses OC43 and 229E have been recognized as causes of the common cold for many years and cause frequent reinfections throughout life. In adults, these viruses account for 4% to 15% of acute respiratory disease annually and up to 35% during peak periods.

Pathogenesis

Conventional coronavirus antigen has been detected in epithelial cells shed from the nasopharynx of infected volunteers and, during experimental infection, nasal airway resistance, mucosal temperature, and the albumin content of nasal secretions increase. However, relatively little is known about the pathogenesis of the common cold induced by conventional human coronaviruses[10-12].

Clinical Illness

Common symptoms on presentation are fever, chills and/or rigors, myalgias, and occasionally diarrhea. Cough and dyspnea are the predominant respiratory symptoms but may not be present initially.

Diagnosis

Common findings on chest CT include unilateral or bilateral areas of ground-glass opacifications and interlobular septal and intralobular interstitial thickening. In most patients, peripheral involvement in the lower lung zones has been observed. In some cases, after recovery from acute illness, pulmonary fibrosis has developed. Clinical features predictive of poor outcomes included the presence of bilateral disease at presentation, markedly elevated lactate dehydrogenase, older age, and other comorbid conditions.

Treatment and Prevention

Immunity against coronaviruses appears to be short-lived. Epidemiologic studies of coronavirus infection have demonstrated high reinfection rates. In human volunteer experiments, infection with a 229E-like coronavirus only induced effective immunity short-term because rechallenge with homotypic virus, the 229E serotype, resulted in infection and illness.

Cytomegalovirus: *Cytomegalovirus* (CMV) is a member of the gammaherpesvirus subfamily of the herpes viruses and has the same structural and biochemical characteristics, which include an internal core containing linear double-stranded DNA, an icosadeltahedral capsid containing 162 capsomeres, and an envelope derived from the host-cell nuclear membrane.

Epidemiology and Transmission

Infection with CMV, whether symptomatic or not, is followed by prolonged excretion of virus in urine, saliva, stool, tears, breast milk, vaginal secretions, and semen. Virus shedding persists for years in children with congenital and perinatal CMV infections.

Pathogenesis

In human fibroblast cell cultures, CMV produces a slowly progressive lytic infection. Infected cells contain large

irregular basophilic intranuclear inclusions and also eosinophilic inclusions in paranuclear areas.

Clinical Illness

The risk of CMV pneumonia is greatest between 30 and 90 days after bone marrow transplant. However, late-onset CMV syndromes, at more than 180 days posttransplantation, have been increasingly recognized with effective control of earlier-onset disease.

Diagnosis

Cytomegalovirus pneumonia should be in the differential diagnosis for any immunosuppressed patient with unexplained lower respiratory complaints or pulmonary opacities.

Treatment and Prevention

Once CMV pneumonitis is established, particularly in allogeneic bone marrow transplant patients, poor outcomes are common. Ganciclovir is highly active against CMV in vitro, but monotherapy is not effective in pneumonitis in bone marrow/stem cell transplant recipients.

Hantaviruses

Hantaviruses are members of the Bunyavirus family and include a number of genetically diverse viruses. The hantavirus responsible for an outbreak of severe pulmonary disease in the southwestern United States, Sin Nombre virus, is roughly spherical, with a mean diameter of 112 nm. The genome consists of negative-sense single-stranded RNA arranged in three physically discrete gene segments[13-17].

Epidemiology and Transmission

The *hantavirus pulmonary syndrome* (HPS) is a zoonosis in which humans experience severe, often fatal disease. Each of the individual hantavirus strains appears to be associated with a specific rodent host (e.g., Sin Nombre virus with the deer mouse, Bayou virus with the rice rat, *Black Creek Canal virus* (BCCV) with the cotton rat, and New York virus with the white-footed mouse). Transmission to humans is presumed to result from contact with infected rodent excreta. Hantaviruses are stable and can persist in the environment for 10 to 15 days without loss of viability.

Pathogenesis

Infection with Sin Nombre virus or other agents of HPS have relatively long incubation periods (median 14 to 17 days; range 1 to 51 days), and antibody and cellular responses in humans are usually detectable at the time of presentation.

Clinical Features: Presentation of HPS begins with a prodrome of fever, chills, and myalgias, occasionally accompanied by abdominal discomfort and GI symptoms, and generalized malaise.

Diagnosis

Chest radiographs are typical of pulmonary edema, without consolidation. In the absence of immunodeficiency, patients universally have detectable serum *immunoglobulin M* (IgM) and IgG antibody at the time of admission, and serologic techniques are the mainstay of diagnosis. In low-prevalence areas, a positive IgM is diagnostic.[107] Virus can also be detected in blood by *reverse transcriptase polymerase chain reaction* (RT-PCR) during the first 10 days of illness.

Treatment and Prevention

It has been suggested that high-dose steroid therapy may be useful because of the pathogenesis of the disease and potential utility of steroids in systemic capillary leak syndrome.

Herpes Simplex Virus

Both *herpes simplex virus* (HSV) types 1 and 2 belong to the alphaherpesvirus subfamily of herpesviruses and share the same basic structural features. HSV-1 is most commonly associated with respiratory infection, whereas HSV-2 is more commonly associated with genital infection.

Epidemiology and Transmission

Humans are the reservoir for HSV-1 and HSV-2 viruses. With primary infection, infectious virus is produced in the skin and mucous membranes, being present in vesicle fluid and cellular debris from herpetic ulcers. After establishment of latency in nerve ganglia, virus is intermittently shed in respiratory, vaginal, and urethral secretions in the absence of clinical disease. Asymptomatic respiratory tract shedding can be detected in about 1% to 2% of seropositive children and adults.

Pathogenesis

Primary HSV infection has a mean incubation period of approximately 1 week. Primary infection begins at a local site, with viral replication in parabasal and intermediate epithelial cells and resultant cell destruction and initiation of host inflammatory responses. Cells containing characteristic nuclear inclusions and sometimes multinucleation may be observed in lesions. In immunocompetent individuals, regional lymph nodes may be involved during primary infection, but the disease is usually contained at the primary site by innate antiviral responses. Cellular immunity is of primary importance in controlling HSV infection; studies in patients with AIDS and severe mucocutaneous HSV indicate that both CD4 and CD8 T cells contribute to control of viral replication and spread.

Clinical Illness

Acute Gingivostomatitis and Pharyngitis.

Fever, malaise, and reduced oral intake may add to the overall severity of these illnesses, which last up to 2 weeks[18-22].

Chronic Ulcerative Pharyngitis and Laryngotracheitis

may spread to the esophagus and lower airway, possibly facilitated by instrumentation such as orotracheal intubation or bronchoscopy, resulting in the development of similar lesions at these sites.

Pneumonia

Herpes simplex virus causes pneumonia in neonates with congenital and peripartum infections and in patients with malignancy, burns, organ transplantation, and other conditions associated with impaired immunity.

Diagnosis

The clinical features of herpetic gingivostomatitis are sufficiently characteristic to permit accurate diagnosis in most cases. Other conditions with similar oral lesions are limited and include herpangina, aphthous stomatitis, Steven-Johnson syndrome, and other enanthems resulting from infection and drug sensitivities.

Treatment and Prevention

No vaccines of proven value are currently available. Primary HSV gingivostomatitis in immunocompetent persons responds to oral acyclovir treatment. Specific therapy of herpes simplex pneumonia has not been

evaluated in controlled trials, but most clinicians would use intravenous acyclovir.

Influenza Virus

Influenza viruses belong to the family Orthomyxoviridae and are classified into three distinct types: influenza A, influenza B, and influenza C virus. All three viruses share the presence of a host cell–derived envelope, envelope glycoproteins important for entry and egress from cells, and a segmented negative-sense, single-stranded RNA genome.

Epidemiology and Transmission

Influenza virus infection is acquired by transfer of virus-containing respiratory secretions. Both small particle aerosols and droplets probably play a role in this transmission, but for infection control purposes influenza is generally considered to be transmitted by droplets.

Pathogenesis

Infection with influenza virus in humans is generally limited to the respiratory tract. After inoculation, the incubation period is thought to be from 18 to 72 hours depending in part on the inoculum dose. Virus shedding is maximal at the onset of illness and may continue for 5 to 7 days or longer in children.

Measles Virus

Measles virus is classified in the *Morbillivirus* genus of the Paramyxoviridae family and is structurally similar to parainfluenza virus and RSV. Its surface glycoproteins include a hemagglutinin responsible for attachment to cells, a *fusion* (F) protein responsible for cell membrane fusion and virus penetration of cells, but no neuraminidase.

Epidemiology

Before vaccine use, measles arose in epidemics of 3 to 4 months in duration every 2 to 5 years in temperate regions. Measles virus infection is highly contagious and can spread despite high levels of acquired immunity in the population. Airborne transmission via small-particle aerosols and possible spread by fomites appear to account for its high communicability.

Pathogenesis

The respiratory tract and possibly the conjunctival epithelium are the portals of entry and initial sites of replication of measles virus, as well as subsequent target organs of disease expression. An initial viremic phase leads to infection of mononuclear phagocytes including dendritic cells, and a second phase of viremia, corresponding to the prodromal stage of illness, results in dissemination of virus to the epithelial cells of the skin, respiratory tract, gut, bile duct, and bladder and to lymphoid organs.

Clinical Illness

Typical Measles.

The typical prodrome of measles lasts 2 to 8 days and is characterized by fever, malaise, anorexia, cough, coryza, and conjunctivitis. Koplik spots, which are erythematous macular lesions with central white-yellow or gray puncta, appear on the buccal or labial mucous membranes toward the end of the prodromal period.

Atypical Measles

The illness begins abruptly, with high fever, headache, myalgia, vomiting, abdominal pain, and nonproductive cough. Respiratory symptoms, including dyspnea, coryza, sore throat, and pleuritic chest pain, are common. A polymorphous eruption, which may include vesicles, petechiae, purpura, and urticarial lesions, begins typically on the distal extremities and spreads proximally over 3 to 5 days[23=29].

Diagnosis

The diagnosis of measles is most readily confirmed in immunocompetent patients by detecting measles virus–specific IgM by ELISA. In immunodeficient patients, detection of measles virus by nucleic acid amplification of urine or of samples obtained by throat or nasopharyngeal swab is sensitive and specific; samples can be sent to the U.S. Centers for Disease Control.

Metapneumoviruses

The *human metapneumoviruses* (hMPVs) are pleomorphic particles with short envelope projections, resembling other paramyxoviruses. These viruses are closely related to the pneumoviruses (of which RSV is the human example), differing only by the absence of two nonstructural proteins and a slightly different arrangement of gene order on the negative-sense, single-stranded RNA genome.

Epidemiology and Transmission

hMPV infections are distributed worldwide and have been documented in both the outpatient and inpatient setting. Recent estimates of disease burden based on PCR diagnostics suggest that hMPV results in 1 to 1.2 hospitalizations, 13 emergency department visits, and 55 outpatient visits per 1000 children younger than 5.

Clinical Features

Human metapneumoviruses appear to be responsible for a spectrum of acute respiratory illnesses ranging from mild or asymptomatic infection to severe bronchiolitis and pneumonitis. The clinical picture most closely resembles that of RSV, and bronchiolitis is the major manifestation in children.

Pathogenesis

Relatively little is known regarding the pathogenesis of this disease. In hospitalized children with hMPV, levels of nasal secretion RANTES have been reported to be suppressed, while levels of nasal IL-8 were increased.

Diagnosis: Viral culture is slow and has low sensitivity. Most infections have been detected by nucleic acid amplification techniques, which are available in panels to detect and identify respiratory viruses.

Prevention and Treatment

No antiviral agents or vaccines are currently licensed for treatment or prevention of hMPV infections, and this is unlikely to change in the near future. Ribavirin is as active in vitro against hMPV as it is against hRSV, but there are no data to support the therapeutic efficacy of this drug.

Parainfluenza Viruses

Parainfluenza viruses belong to the *Paramyxovirus* genus of the Paramyxoviridae family, which includes mumps virus and important veterinary pathogens. This group of medium-sized (150 to 200 nm), pleomorphic, enveloped viruses has a nonsegmented, single-stranded RNA genome contained in a helical nucleocapsid.

Epidemiology and Transmission

Parainfluenza viruses have a worldwide distribution, and almost all persons are infected initially during childhood. Parainfluenza type 3 virus may cause infection in infancy, whereas infections by type 1 and 2 viruses appear to be prevented by maternal antibody and usually arise later.

Pathogenesis

Although viremia has been described, replication of the virus is generally restricted to the respiratory tract mucosa. The quantity of virus shed in respiratory secretions tends to parallel the severity of illness. Virus shedding commonly continues for periods of 8 to 10 days in initial infections but may last for 3 weeks or longer. Prolonged shedding (months) of parainfluenza virus type 1 or 3 has been reported in apparently normal hosts, as well as in immunodeficient children.

Clinical Illness

Primary infections are usually symptomatic and are associated with the most severe forms of illness. Initial infections with parainfluenza virus types 1 to 3 cause febrile rhinitis, pharyngitis, laryngitis, and bronchitis in children. Depending on the serotype causing infection, 50% to 80% of primary infections are associated with fever, and up to one third of children have evidence of lower respiratory tract involvement.

3. Diagnosis

Rapid diagnosis of parainfluenza infection can be made by detection of viral antigen or RNA in respiratory secretions obtained using throat or nasopharyngeal swabs. Detection of parainfluenza Respiratory secretions contain the virus at the time of symptom onset. Viral culture is also sensitive, and parainfluenza viruses can be isolated as early as 3 days and usually within 10 days after inoculation of cell culture with specimens from infants and children.

Treatment and Prevention

There are currently no available antiviral agents of proven effectiveness against parainfluenza virus. Ribavirin is active against parainfluenza viruses in vitro and would theoretically be expected to be active in vivo as well.

Respiratory Syncytial Virus

RSV is classified in the *Pneumovirus* genus of the Paramyxoviridae family. Similar in structure to parainfluenza viruses, RSV is a pleomorphic (150 to 300 nm), enveloped virus with a single-stranded, nonsegmented RNA genome. The surface proteins include the F protein responsible for fusion of the viral envelope with the host cell membranes and formation of syncytium, and the G protein, a heavily glycosylated protein responsible for attachment to cells. Antibodies against the F and G protein neutralize RSV in vitro, but antibodies against the G do not prevent syncytium formation.

Epidemiology and Transmission

RSV is worldwide in distribution and, in temperate climates, causes annual outbreaks of infection in the late fall, winter, or spring. Epidemics are associated with increases in pediatric hospitalizations and deaths due to lower respiratory tract illness in infants and young children. Nearly 50% of children are infected within the first year of life, and almost all have been infected by 3 years of age.

Pathogenesis: Viral replication generally begins in the upper respiratory tract with gradual (4- to 5-day) progression to involve the lower respiratory tract. In children with normal immunity, the duration of viral shedding ranges from 1 to 3 weeks. Clinical signs of bronchiolitis include airway trapping and wheezing.

Clinical Illness

The clinical manifestations of infection depend on both the age and immunologic state of the host. RSV is the major cause of lower respiratory tract illness in infants and young children and accounts for 45% to 90% of bronchiolitis, up to 40% of pneumonia, and smaller proportions of croup and bronchitis cases in this age group[30-33].

Diagnosis

The most rapidly and readily available approach to establishing RSV infection is by rapid antigen detection.

Treatment and Prevention

Correction of hypoxemia is the most important aspect of managing RSV lower respiratory tract disease. Ribavirin is highly active against RSV in vitro, and aerosolized ribavirin has been shown to reduce viral shedding and shorten the course of illness in some but not all studies.

Rhinovirus

Rhinoviruses (RVs) are species in the *Enterovirus* genus in the Picornaviridae family. The RV virion is a nonenveloped particle 30 nm in diameter with four major structural proteins.

Epidemiology and Transmission

RV is believed to produce even higher infection rates in children, leading to acquisition of antibody to the different RV types throughout childhood and adolescence, with peak antibody prevalence in young adults.

Pathogenesis

Approximately two thirds of both natural and experimental RV infections result in overt illness. The incubation period of RV colds is usually 2 days but may be up to a week. Symptoms begin within 1 day following experimental infection. Small doses of RV instilled into the nose or eye of susceptible volunteers regularly lead to infection, indicating that mucociliary clearance is not effective against the virus.

Clinical Illness: RV colds vary in severity from mild episodes characterized by 1 to 2 days of coryza or scratchy throat to full-blown illnesses with profuse and prolonged rhinorrhea, pharyngitis, and bronchitis.

Diagnosis

Rapid tests for detecting RV nucleic acid are available in respiratory virus panels (see Chapter 17); they generally do not distinguish rhinoviruses from other enteroviruses. RVs can be isolated in cell culture, usually within 2 to 7 days after inoculation.

Treatment and Prevention

The only effective therapy for RV colds currently available is symptomatic treatment of individual complaints. Remedies recommended for such treatment are described in the section on common cold in this chapter.

Varicella-Zoster Virus

Varicella-zoster virus (VZV) is an enveloped double-stranded DNA virus with a large genome (≈125,000 bp). Varicella is a highly contagious, childhood disease that typically causes community outbreaks in late winter and early spring months in temperate regions. Varicella spreads rapidly to household contacts, with an attack rate of nearly 90% within 2 weeks.

Pathogenesis

The incubation period of varicella averages 2 weeks, and almost all cases of varicella develop within 11 to 20 days

after exposure. Reactivation of virus replication and centrifugal spread along sensory nerves lead to the unique dermatomal distribution of shingles (zoster)[34-38].

Clinical Illness

Varicella.

The exanthem typically begins around the scalp and head, with subsequent involvement of the trunk and extremities. Lesions progress through various stages (erythematous macules, vesicles, pustules, crusts), so an area will have lesions in different stages of evolution.

Herpes Zoster.

Zoster represents reactivation of latent virus along one to three dermatomes and, in adults, is usually associated with pain. The thoracic dermatomes are involved in about one half of cases. Prolonged severe pain, or postherpetic neuralgia, can be a serious complication, with increased frequency in those older than 50.

Diagnosis

A rapid diagnosis of herpes group infection can be established by cytologic examination of lesion scrapings which has a sensitivity of 70% to 85% when lesions are in the vesicular stage. Direct immunofluorescence for VZV antigen in lesions is the most sensitive rapid laboratory test.

Treatment for viral infections

One of the main antibiotics used in the repositioning with antiviral activity are macrolides and especially azithromycin, which has been shown to be safe, have good antibacterial activity and a long half-life. Various studies have shown that macrolides have antiviral properties in vitro against various viruses. In several cases, AZM constantly emerges as a possible antiviral drug candidate against respiratory viruses and there are promising signs of its possible use in the clinic. Another property for its use in some viral infections is its anti-inflammatory activity, which may be important to reduce immunopathology, such as against pandemic beta-coronaviruses, where elevated inflammatory processes seem to be associated with mortality[39].

4. Conclusion

The regular laboratory diagnostic tests and specific antimicrobial agents are generally available for the treatment of bacterial, fungal, and parasitic infections, we are just entering the stage when rapid nucleic acid tests and a greater array of antiviral agents are available for tackling viral infections[40]. Relay on novel emerging viruses are causing major epidemics from time to time especially in densely populated areas where human populations have close contact with wild animals (wildlife markets) and food animals (wet markets and abattoirs). Therefore, prevention of viral infections by implementing effective infection control and vaccination is of utmost importance to contain these virus infections.

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