Journal of Medical Bacteriology

An Overview of Viral Respiratory Infections

Elahe Derakhshan-Nezhad

Student Research Committee, Mashhad University of Medical Sciences, Mashhad, Iran.

ARTICLE INFO

Article type:
Review Article

Article history:

Received	24	Nov	2024
Revised	17	Dec	2024
Accepted	21	Dec	2024
Published	16	Feb	2025

Keywords:
Complication, LRTIs, Manifestation, Transmission, URTIs.

*Corresponding Authors: Elahe Derakhshan-Nezhad: Student Research Committee, Mashhad University of Medical Sciences, Mashhad, Iran. *Tel*: +98-51-53336689, *E-mail:* Ederakhshan38@gmail.com

ABSTRACT

Background: Various viruses and bacteria are involved in the incidence of respiratory tract infections (RTIs), and viruses account for the majority of RTIs. Given the high economic and therapeutic burden of RTIs, the current review aimed to present a brief but comprehensive overview of the most important viral etiologies of RTIs along with their complications, manifestations, and transmission routes. A comprehensive literature search was performed in electronic databases including Google Scholar, Scopus, PubMed, and Web of Science to find articles related to the scope of this research, published in English. Eligible studies providing evidence on viral respiratory tract infections, their clinical complications, symptoms, and causes were included in this review.

Conclusion: Considering the diversity and prevalence of viral RTIs and the economic and therapeutic burden imposed on health systems, it is necessary to raise public awareness about their transmission methods and preventive measures to control and manage these infections.

- ***Please cite this paper as:*** Derakhshan-Nezhad E. An Overview of Viral Respiratory Infections. J Med Bacteriol. 2025; 13 (1): pp.60-68. DOI: 10.18502/jmb.v13i1.18050

Introduction

Various viruses and bacteria are involved in the incidence of respiratory tract infections (RTIs). Unlike specific pathogens confined to tropical regions, respiratory pathogens are globally distributed, effectively spread between humans, and affect people of all ages (1). Respiratory infections could vary from mild or asymptomatic illnesses to severe and life-threatening diseases, thus posing a serious health threat to immunocompromised individuals, the elderly, and young children. Respiratory infections account for the majority of mortality and morbidity worldwide and impose a high global economic and therapeutic burden on health systems (2). Children have the greatest burden of viral respiratory diseases (3). Studies have shown that viral RTIs are the main cause of mortality and morbidity in children less than 5 years old (4). Respiratory pathogens are transmitted through various ways, including direct or indirect contact, aerosols, and respiratory droplets. Viral respiratory pathogens commonly infect both the lower and upper respiratory tract. These infections could be clinically categorized either by the causative pathogen (e.g., influenza) or the resulting complication (e.g., bronchiolitis). Specific pathogens usually cause distinct clinical complications along with a variety of general viral RTI symptoms; for instance, respiratory syncytial virus is usually considered as the causative agent of bronchiolitis, while rhinovirus is usually considered as the causative agent of the common cold. Although specific respiratory viruses are significantly related to specific complications, it has been indicated that each virus may be involved in the incidence of many complications (3).

Viral respiratory diseases vary greatly in severity. The elderly and infants are more susceptible to severe diseases. Complications may be directly due to viral infection or indirectly because of exacerbation of preexisting diseases or bacterial coinfection of respiratory organs (3).

Some respiratory viruses are well known as important etiological agents of lower respiratory tract infections (LRIs), including human coronaviruses (HCoVs) 229E and OC43, parainfluenza virus (PIV), influenza viruses (IFVA and IFVB), adenovirus (AdV), respiratory syncytial virus (RSV), and enterovirus (EV) (5). Until the emergence of SARS-CoV (severe acute respiratory syndrome coronavirus), MERS-CoV (Middle East respiratory syndrome coronavirus), and SARS-CoV-2, the pathogenicity of HCoVs and their importance and role in causing LRIs was not well recognized, and OC43 and 229E were the only coronaviruses most frequently associated with LRIs. The emergence of new coronaviruses has marked a new epoch of international research in the field of science and medicine. In the past few decades, in addition to HCoVs, a number of new respiratory viruses causing human diseases have been detected. While the pathogenicity of some has been well characterized, the pathogenicity of others has not yet been elucidated.

Considering that most RTIs are of viral origin, the current review aimed to present a brief but comprehensive overview of the most important viral etiologies of respiratory tract infections along with their complications, clinical manifestations, and transmission routes, including new and old HCoVs, IFVs (A and B), PIV, RSV, metapneumovirus (MPV), rhinoviruses (RV), AdV, and EV. The information presented in this article is expected to raise public awareness about their manifestations and transmission routs to prevent their widespread prevalence.

Seaech strategy

A comprehensive literature search was performed in electronic databases including Google Scholar, Scopus, PubMed, and Web of Science to find articles related to the scope of this research, published in English. Eligible studies providing evidence on viral respiratory tract infections, their clinical complications, symptoms, and causes were included in this review.

Human coronaviruses (HCoVs)

Coronaviruses are known as a large family of spherical or pleomorphic, single-stranded, positive-sense, enveloped, RNA viruses with distinct virion morphology. The seven HCoVs identified until now are as follows: HCoV-229E in 1965, HCoV-OC43 in 1967, SARS-CoV in 2002–2003, HCoV-NL63 in 2004, HCoV-HKU1 in 2005, MERS-CoV in 2012, and SARS-CoV-2 in 2019. These viral pathogens are grouped into four categories, including α, β, γ, and δ (6). Human coronaviruses NL63 and 229E are grouped in type α, while SARS-CoV-2, MERS-CoV, SARS-CoV, HKU1, and OC43 are grouped in type β (7).

The four coronaviruses HKU1, OC43, NL63, and 229E are globally distributed, spread via coughing and sneezing, induce mild to moderate upper RTIs, and are frequently accompanied by common colds in adults. These infections might also occasionally cause severe pneumonia and bronchiolitis in immunocompromised individuals, the elderly, young children, and infants (8). Differentiation of these infections from other URIs is clinically difficult, and their coinfection with other respiratory pathogens like HRV, EV, and HPIV makes their detection even more challenging but does not typically aggravate the infection (8). These infections may also be accompanied by chronic bronchitis, wheezing, and asthma exacerbation in children (9). Clinical manifestations of HCoV-229E are as follows: general malaise, sore throat, sneezing, nasal discharge, headache, and sometimes cough and fever (8). HCoV-OC43 is another HCoV that manifests as fever, coryza, cough, and sore throat in children as well as nasal symptoms and cough in older patients (10). HCoV-OC43 has neuroinvasive properties and could affect neuronal cells and induce permanent infections and encephalitis in human brain (8). HCoV-OC43 and HCoV-NL63 are the causes of most respiratory illnesses that lead to hospital admissions (8). HCoV-NL63 often presents with comparatively mild upper RTI symptoms, including rhinitis, sore throat, cough, and fever (8); however, it could also cause febrile seizures, asthma exacerbation, and croup in children. People of all ages are infected with HCoV-NL63 (11). HCoV-HKU1 as another HCoV manifests as tonsillar hypertrophy, rhinorrhea, postnasal discharge, nasal congestion, chills, sore throat, sputum, fever, cough, and febrile seizures (8).

SARS-CoV-2, MERS-CoV, and SARS-CoV are members of the Betacoronavirus (βCoV) genus in the Coronaviridae family. These infections are associated with severe upper and lower RTIs, have similar symptoms, and are mainly transmitted via respiratory droplets, direct contact, and fecal–oral routes. SARS-CoV infection manifests as shortness of breath or acute respiratory distress syndrome (ARDS), high fever, chills, cough, malaise, myalgia, hypoxia, and cyanosis (12). MERS-CoV infection, like SARS-CoV, is associated with serious LRIs, extra-pulmonary involvement, and high mortality (13). Clinical symptoms include shortness of breath, cough, chills, fever, myalgia, stiffness, discomfort, diarrhea, and vomiting. Pneumonia is a frequently reported complication. Serious infections along with shock, acute respiratory failure, or renal failure are more prevalent among adults (14). Similarly, SARS-CoV-2 infection presents with sore throat, headache, dry cough, chills, fever, fatigue, myalgia, loss of taste or smell, sputum, hemoptysis, dyspnea, and vomiting and diarrhea (15). Like MERS and SARS, the infection may lead to ARDS in some patients (16). Around 81% have only mild illnesses, while 14 and 5% have severe and critical conditions, respectively (17). Problems such as anorexia and sore throat are more common in older ICU (intensive care unit) patients with preexisting diseases like diabetes and high blood pressure (15).

Respiratory syncytial virus (RSV)

There viruses are pleomorphic, negative-strand, non-segmented, enveloped, RNA viruses in the Orthopneumovirus genus of the recently-defined Pneumoviridae family. RSV is highly contagious, has global distribution, and could be transmitted

through close contact, airborne particles (via coughing and sneezing), or fomites (via contaminated hands) (1). RSV could induce a wide range of illnesses from modest URIs to serious LRIs, including croup, tracheobronchitis, bronchiolitis, and pneumonia (18). In adults, infections are cold-like and self-limiting. The most common RSV-induced URI in young children and neonates is otitis media along with fever. RSV is also the primary cause of LRIs in young children and neonates, including bronchiolitis, which is preceded by URI symptoms and followed by air trapping, expiratory wheezing, cough, dyspnea, tachypnea, and in some severe cases, cyanosis and intercostal muscle retractions. About 50% of infants experience fever (18). Infants with underlying heart or lung diseases, premature infants, and immunocompromised individuals of any age are at risk of serious and life-threatening infection. The most common symptoms in children > 3 years and older patients include exacerbation of chronic lung disease, coryza, hoarseness, sore throat, cough, mild fever, and wheezing (18). RSV is one of the main causes of aggravation of COPD (chronic obstructive pulmonary disease) and asthma in people suffering from obstructive pulmonary diseases. In general, RSV infections are more prevalent among people with preexisting chronic lung diseases (19).

Human metapneumovirus (HMPV)

There viruses are pleomorphic or spherical, filamentous, single-stranded, enveloped, RNA viruses in the Metapneumovirus genus of the Pneumoviridae family (20). Transmission occurs via close contact with respiratory secretions or contaminated objects and surfaces. HMPV is responsible for both lower and upper RTIs (21). Infection usually occurs during childhood. Coinfection with other respiratory pathogens seems to aggravate the infection (22). Similar to RSV, HMPV causes bronchiolitis, pneumonia, croup, and mild URIs with flu-like symptoms such as vomiting, myalgia, and fever (23, 24). The most common symptoms include sore throat, rhinitis, wheezing, dyspnea, cough, and fever (21). Patients with comorbidities, immunocompromised individuals, the elderly, and very young patients are at risk of more serious infections (21). HMPV in adults may manifest as the common cold, acute bronchitis, or a flu-like infection (21).

Influenza viruses (IFVs)

IFVs are negative-strand, segmented, enveloped, pleomorphic, RNA viruses in the Orthomyxoviridae family. These viral pathogens are grouped into four distinct categories, A, B, C, and D. While the host of IFV A could be a variety of organisms, the main hosts of IFV B and C are humans, and the hosts of IFV D are mostly pigs, goats, and cattle (25, 26). Avian influenza A (H5N1) virus, which has recently been identified as a highly pathogenic agent in Asia, could occasionally cause human diseases (27). These pathogenic viruses are disseminated through large airborne particles, respiratory droplets, and presumably fomites (via contaminated hands) (1). They are highly contagious, globally distributed, and associated with RTIs with high seasonal mortality and morbidity, especially in elderly patients and infants (1). Influenza causes both lower and upper RTIs with various clinical manifestations such as pneumonia, tracheobronchitis, sinusitis, and otitis media. Young children may also develop croup and bronchiolitis. Other complications include congestive heart failure, chronic bronchitis, and asthma exacerbation. Clinical symptoms include fever, chills, nasal congestion, myalgia, headache, malaise, mild rhinorrhea, sore throat, prostration, and nonproductive cough. Frail elderly patients may also experience gastrointestinal problems, low fever, confusion, lethargy, and lassitude (28). Immunodeficiency, human immunodeficiency virus (HIV) infection, and pregnancy are risk factors for more serious infection and subsequent problems (28).

Human parainfluenza viruses (PIVs)

PIVs are classified into two groups in the Paramyxoviridae family (1). HPIV types 1 and 3 are members of the Respirovirus genus, whereas HPIV types 2 and 4 are members of the Rubulavirus genus. PIV type 1 is mostly associated with croup epidemics, while PIV type 2 is often associated with milder infection but could also cause croup (29). PIV type 3 is the main cause of LRIs, often causing croup, pneumonia, and bronchitis in patients under one year of age, while patients with immunodeficiency are more at risk of morbidity and mortality due to pneumonia (30). PIV types 1 and 3 cause LRIs in patients with immunodeficiency, chronically ill patients, children, and the elderly (29). PIV type 4 is only occasionally recognized and is mainly accompanied by URI symptoms in children and older patients (29). Generally, HPIVs are the primary cause of croup in children and infants and the second most common cause of LRIs in infants worldwide after RSV (18). Similar to RSV, PIV infection first manifests as mild nasal congestion and coryza, which progresses toward LRT and causes cough as the main symptom (30). HPIVs are mainly transmitted through large droplets and fomites (18). About two-thirds of all PIV illnesses in children lead to febrile URIs, and the remaining one-third result in bronchiolitis, pneumonia, or croup (31, 32).

Adenoviruses (AdV)

Adenoviruses are double-stranded, non-enveloped, icosahedral, non-segmented, DNA viruses in the Mastadenovirus genus of the Adenoviridae family, which cause a variety of clinical complications in humans (33). There are 70 or more distinct adenovirus serotypes categorized into seven (A to G) species. Various serotypes are associated with various complications. Infection may be airborne or waterborne (33). Respiratory transmission occurs through respiratory droplets, aerosols, fomites, or fecal-oral route at all ages but is most common among children and those living in close quarters and proximity, with clinical manifestations ranging from common colds to severe pneumonia (33, 34). Ocular transmission occurs through swimming pools and medical facilities where hygiene principles are not observed (33, 34). Adenoviruses are globally distributed, highly contagious, and usually asymptomatic, infect all parts of the respiratory tract, and persist for a long time (33-35). Clinical complications are different depending on adenovirus type, age, and immunological status. Most adenovirus infections manifest as febrile colds, and children may have high and prolonged fever. Adenoviruses cause both lower and upper RTIs. The most frequent URT complications are common cold, pharyngitis, and otitis media, followed by pharyngeal and tonsillar exudate, granular mucus, and anterior cervical adenopathy. Adenovirus-induced LRIs include pneumonia and bronchitis (35). Adenoviral pneumonia progresses similar to that detailed for PIV and RSV, but the infection could be much more serious and last for a long time. Children, immunocompromised individuals, and people with preexisting diseases are more at risk of severe potentially lethal infection (36). Typically, clinical symptoms include fever, upper respiratory symptoms, malaise headache, chest pain, and cough.

Rhinoviruses

Rhinoviruses are small, single-stranded, positive-sense, non-enveloped, RNA viruses in the Enterovirus genus of the Picornaviridae family, with nearly 170 genotypes (37). HRVs are disseminated via close contact and respiratory droplets or micro-droplets (38). HRVs are traditionally considered as the causative agents of URIs, otitis media, rhinosinusitis, and sinusitis. However, recent advancements in diagnostic methods have greatly contributed to the identification of HRVs as LRT pathogens, especially in infants, the elderly, asthmatic patients, and immunocompromised individuals. HRVs are currently associated with pneumonia,

bronchiolitis, asthma exacerbation, and several forms of severe respiratory diseases. (39, 40). HRVs are considered as the primary etiological agents of common colds (41). Rhinoviral colds could not be easily distinguished from those of other viral pathogens. The most frequent clinical manifestations of HRVs are exacerbations of asthma and COPD (42). Clinical symptoms include fever, sore throat, sneezing, nasal obstruction, nasal discharge, facial/ ear pressure, hoarseness, otalgia, pharyngitis, cough, and headache (41).

Enteroviruses (EVs)

These pathogens are small, single-stranded, positive-sense, RNA viruses that belong to the Enterovirus genus of the Picornaviridae family. Transmission occurs mainly through the fecal-oral transmission route and vice versa. Transmission from animals to humans or vice versa has also been documented (42). Non-rhinovirus enteroviruses manifest as chest pain, sore throat, sneezing, cough, altered mental status, weakness, lymphadenopathy, malaise, fever, and gastrointestinal problems. Patients with underlying diseases, including diabetes, hematologic disease, immunodeficiency, chronic lung disease, preterm birth, and BPD (bronchopulmonary dysplasia), are more susceptible to serious infections. Most enteroviral illnesses are mild and self-limiting. Clinical complications are as follows: exacerbation of preexisting conditions, destruction of lung tissue, irreversible paralysis, progression to persistent or chronic diseases, and the incidence of secondary infections. Enteroviruses are the most prevalent viruses that could affect the CNS (central nervous system). Viremia leads to disseminated diseases, such as pancreatitis and myocarditis, which in turn could lead to a second, stronger viremia, possibly causing clinical diseases and CNS involvement (42).

Herpesviruses

Herpesviridae are a family of large, double-stranded, enveloped, DNA viruses causing various infections. The nine human herpesviruses are as follows: human cytomegalovirus (HCMV), varicella zoster virus (VZV), herpes simplex virus 1 (HSV-1), HSV-2, Epstein–Barr virus (EBV), Kaposi sarcoma herpesvirus (KSHV), human herpesvirus 6A (HHV6A), HHV6B, and HHV7. Herpesviruses are globally distributed and cause lifelong latent infections that could be reactivated periodically (43). All herpesviruses could induce LRIs in immunocompromised individuals and spread hematogenously. Most human herpesviruses are transmitted through direct contact when a susceptible person has direct physical contact with an infected person; otherwise, they are transmitted through airborne particles. Among the known members of the Herpesviridae family, EBV, HSV, CMC, and VZV are the most common and ubiquitous pathogens.

Most HCMV infections are asymptomatic (44). Although the initial infection in children is often asymptomatic, but in later stages of life, it could lead to severe problems for fetal development in pregnant women, patients with immunodeficiency or underdeveloped immunity, and transplant recipients (43). At-risk individuals are more prone to develop serious disease after initial infection or reactivation of latent virus (44, 45). Transmission occurs through close contact via infected secretions. The most frequent clinical presentation of HCMV is a self-limiting febrile infection resembling infectious mononucleosis caused by EBV (46). Clinical symptoms include fatigue, headache, myalgia, malaise, and fever (44, 46). CMV pneumonitis is usually characterized as a pulmonary infection with progressive pulmonary distress, tachypnea, and fever.

HSV is involved in a wide variety of illnesses (like RTIs) and symptoms such as localized lymphadenopathy, gingivostomatitis, sore throat, and fever (47). HSV pneumonitis is accompanied by ARDS, long-term ventilator use, and increased morbidity and mortality in severely-ill cases (48).

Transmission occurs through close contact. Immunodeficiency of any type at any age is a risk factor for HSV pneumonitis. Clinical symptoms of HSV infections are as follows: herpes simplex encephalitis, meningitis, disseminated disease in neonates, eczema herpeticum, herpes stromal keratitis (HSK), genital herpes, and cold sores (49). VZV infection causes various complications, among them pneumonia is the most important and frequent. VZV is transmitted through respiratory route and close contact (50). Varicella pneumonitis is one of the clinical manifestation of VZV infection. Immunodeficiency and infancy are two risk factor for fatal infection. Clinical presentations of VZV infection are as follows: otalgia, eye swelling and redness, facial paralysis, fever, sensitivity to light, headache, rash with fluid-filled blisters, and burning pain in the skin.

EBV is also involved in various clinical complications and life-long illnesses, such as CNS diseases, infectious mononucleosis, nasopharyngeal carcinoma, and malignant lymphoproliferative problems. Primary EBV infection may be accompanied by pleural effusion, pneumonia, and pneumonitis, but these complications are rare (51). Transmission occurs through close contact and saliva. Clinical symptoms of EBV infection may be as follows: rash, swollen liver, enlarged spleen, swollen lymph nodes in the neck, fever, fatigue, and sore throat. EBV pneumonitis does not have specific clinical symptoms and usually presents with fever, expectoration, cough, etc (51).

Conclusion

Considering the diversity and prevalence of viral RTIs and the economic and therapeutic burden imposed on health systems, it is necessary to raise public awareness about their transmission methods and preventive measures to control and manage these infections.

Funding Information

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics approval and consent to participate

Not applicable. This article does not contain any studies with human or animal subjects, performed by the author.

Conflict of interest

Elahe Derakhshan-Nezhad declares that she has no conflict of interest.

References

1. Arruda E, Cintra OA, Hayden FG, 2006. Respiratory tract viral infections. In: Guerrant, R.L., Walker, D.H., Weller, P.F. (Eds), Tropical Infectious Diseases. 2nd ed. Saunders Elsevier, p. 637-59.
2. Zhang S, Akmar LZ, Bailey F, et al. Cost of respiratory syncytial virus-associated acute lower respiratory infection management in young children at the regional and global level: A systematic review and meta-analysis. *J Infect Dis* 2020; **222**(Suppl 7):S680-7.
3. Pavia AT. Viral infections of the lower respiratory tract: Old viruses, new viruses, and the role of diagnosis. *Clin Infect Dis* 2011; **52** (Suppl 4):S284-9.
4. Lafond KE, Nair H, Rasooly MH, et al. Global role and burden of influenza in pediatric respiratory hospitalizations, 1982-2012: A systematic analysis. *PLoS Med* 2016; **13**(3): e1001977.
5. Ruuskanen O, Lahti E, Jennings LC, et al. Viral pneumonia. *Lancet* 2011; **377**(9773):1264-75.
6. Su S, Wong G, Shi W, et al. Epidemiology, genetic recombination, and pathogenesis of coronaviruses. *Trends Microbiol* 2016; **24**(6): 490-502.
7. Zolfaghari Emameh R, Nosrati H, Taheri RA.

Combination of biodata mining and computational modelling in identification and characterization of ORF1ab polyprotein of SARS-CoV-2 isolated from oronasopharynx of an Iranian patient. *Biol Proced Online* 2020; **22**: 1-8.
8. Liu DX, Liang JQ, Fung TS, 2021. Human coronavirus-229E,-OC43,-NL63, and-HKU1 (Coronaviridae). In: Bamford, D.H., Zuckerman, M. (Eds), Encyclopedia of virology. 4th ed. Elsevier, pp. 428-40.
9. Boncristiani HF, Criado MF, Arruda E, 2009. Respiratory viruses. In: Schaechter, M. (Ed), Encyclopedia of microbiology. Elsevier, pp. 500-518.
10. Hendley JO, Fishburne HB, Gwaltney JM. Coronavirus infections in working adults: Eight-year study with 229E and OC43. *Am Rev Respir Dis* 1972; **105**(5):805-11.
11. Esper F, Weibel C, Ferguson D. Evidence of a novel human coronavirus that is associated with respiratory tract disease in infants and young children. *J Infect Dis* 2005; **191**(4):492-8.
12. Lam CW, Chan MH, Wong CK. Severe acute respiratory syndrome: Clinical and laboratory manifestations. *Clin Biochem Rev* 2004; **25**(2): 121-32.
13. Chan JF, Lau SK, To KK, et al. Middle east respiratory syndrome coronavirus: another zoonotic betacoronavirus causing SARS-like disease. *Clin Microbiol Rev* 2015; **28**(2):465-522.
14. Baharoon S, Memish ZA. MERS-CoV as an emerging respiratory illness: A review of prevention methods. *Travel Med Infect Dis* 2019; **32**:101520.
15. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *JAMA* 2020; **323**(11):1061-9.
16. Zumla A, Hui DS, Azhar EI, et al. Reducing mortality from 2019-nCoV: Host-directed therapies should be an option. *Lancet* 2020; **395** (10224):e35-6.
17. Wu Z, McGoogan JM. Characteristics of and important lessons from the coronavirus disease 2019 (COVID-19) outbreak in China: Summary of a report of 72 314 cases from the Chinese Center for Disease Control and Prevention. *JAMA* 2020; **323**(13):1239-42.
18. Piedra PA, Englund JA, Glezen WP, 2002. Respiratory syncytial virus and parainfluenza viruses. In: Richman, D.D., Whitley, R.J., Hayden, F.G. (Eds), Clinical virology. ASM Press, Washington, p. 763.
19. Nam HH, Ison MG. Respiratory syncytial virus infection in adults. *BMJ* 2019; **366**:l5021.
20. van den Hoogen BG, Bestebroer TM, Osterhaus AD, et al. Analysis of the genomic sequence of a human metapneumovirus. *Virology* 2002; **295**(1):119-32.
21. Boivin G, Abed Y, Pelletier G. Virological features and clinical manifestations associated with human metapneumovirus: A new paramyxovirus responsible for acute respiratory infections in all age groups. *J Infect Dis* 2002; **186**(9):1330-4.
22. Semple MG, Cowell A, Dove W. Dual infection of infants by human metapneumovirus and human respiratory syncytial virus is strongly associated with severe bronchiolitis. *J Infect Dis* 2005; **191**(3):382-6.
23. Williams JV, Harris PA, Tollefson SJ. Human metapneumovirus and lower respiratory tract disease in otherwise healthy infants and children. *N Engl J Med* 2004; **350**(5):443-50.
24. Mejias A, Chavez-Bueno S, Ramilo O. Human metapneumovirus: A not so new virus. *Pediatr Infect Dis J* 2004; **23**(1):1-7.
25. Long JS, Mistry B, Haslam SM, et al. Host and viral determinants of influenza A virus species specificity. *Nat Rev Microbiol* 2019; **17**(2):67-81.
26. Zhai SL, Zhang H, Chen SN, et al. Influenza D virus in animal species in Guangdong province, southern China. *Emerg Infect Dis* 2017; **23**(8): 1392-6.
27. Kim JH, Cho CH, Shin JH, et al. Highly sensitive and label-free detection of influenza H5N1 viral proteins using affinity peptide and porous BSA/MXene nanocomposite electrode. *Anal Chim Acta* 2023; **1251**:341018

28. Hayden FG, Palese P, 2002. Influenza virus. In: Richman, D.D., Whitley, R.J., Hayden, F.G. (Eds), Clinical virology. ASM Press, Washington, p. 891.
29. Henrikson KJ. Parainfluenza viruses. *Clin Microbiol Rev* 2003; **16**(2):242-64.
30. Elizaga J, Olavarria E, Apperley JF. Parainfluenza virus 3 infection after stem cell transplant: Relevance to outcome of rapid diagnosis and ribavirin treatment. *Clin Infect Dis* 2001; **32**(3):413-8.
31. Gbadero DA, Johnson ABR, Aderele WI. Microbial inciters of acute asthma in urban Nigerian children. *Thorax* 1995; **50**(7):739-45.
32. Psarras S, Papadopoulos NG, Johnston SL. Pathogenesis of respiratory syncytial virus bronchiolitis-related wheezing. *Pediatr Respir Rev* 2004; **5**(Suppl A):S179-84.
33. Arnold A, MacMahon E. Adenovirus infections. *Medicine* 2017; **45**(12):777-80.
34. Kampf G. Efficacy of ethanol in hand hygiene against adenoviruses. *Am J Infect Control* 2016; **44**(11):1429.
35. Rhee Elizabeth G, Barouch Dan H, 2015. Adenoviruses. In: Bennett, J.E., Dolin, R., Blaser, M.J. (Eds), Mandell, Douglas, and Bennett's principles and practice of infectious diseases. 8th ed. Elsevier Saunders, Philadelphia, PA.
36. Lynch III JP, Kajon AE. Adenovirus: Epidemiology, global spread of novel serotypes, and advances in treatment and prevention. *Semin Resp Crit Care Med* 2016; **37** (4): 586-602.
37. Simmonds P, Gorbalenya AE, Harvala H, et al. Recommendations for the nomenclature of enteroviruses and rhinoviruses. *Arch Virol* 2020; **165**:793-7.
38. Giardina FA, Piralla A, Ferrari G, et al. Molecular epidemiology of rhinovirus/enterovirus and their role on cause severe and prolonged infection in hospitalized patients. *Microorganisms* 2022; **10**(4):755.
39. Zhao Y, Shen J, Wu B, et al. Genotypic diversity and epidemiology of human rhinovirus among children with severe acute respiratory tract infection in Shanghai, 2013–2015. *Front Microbiol* 2018; **9**:1836.
40. Ljubin-Sternak S, Meštrović T, Ivković-Jureković I, et al. The emerging role of rhinoviruses in lower respiratory tract infections in children—Clinical and molecular epidemiological study from Croatia, 2017-2019. *Front Microbiol* 2019; **10**: 2737.
41. Gern JE. The ABCs of rhinoviruses, wheezing, and asthma. *J Virol* 2010; **84**(15):7418-26.
42. Sinclair W, Omar M, 2024. Enterovirus. StatPearls Publishing, Treasure Island (FL).
43. Connolly SA, Jardetzky TS, Longnecker R. The structural basis of herpesvirus entry. *Nat Rev Microbiol* 2021; **19**(2):110-21.
44. Mocarsk ES, Shenk T, Griffiths PD, Pass RF, 2013. Cytomegaloviruses. In: Knipe, D.M., Howley, P.M. (Eds), Fields virology. 6th ed. Lippincott Williams & Wilkins, Philadelphia, pp. 1960–2014.
45. Britt W, 2008. Manifestations of human cytomegalovirus infection: Proposed mechanisms of acute and chronic disease. In: Stinksi, M.F., Shenk, T. (Eds), Human cytomegaloviruses. Springer, Berlin, pp. 417–470.
46. Bravender T. Epstein-Barr virus, cytomegalovirus, and infectious mononucleosis. *Adolesc Med State Art Rev* 2010; **21**(2):251-64.
47. Hraiech S, Azoulay E, Papazian L, 2021. Herpesviruses in critically ill patients with ARDS. In: Janes, S.M. (Ed), Encyclopedia of respiratory medicine. Elsevier, pp. 373–85.
48. Tuxen DV, Cade JF, McDonald MI, et al. Herpes simplex virus from the lower respiratory tract in adult respiratory distress syndrome. *Am Rev Respir Dis* 1982; **126**(3):416-9.
49. Zhu S, Viejo-Borbolla A. Pathogenesis and virulence of herpes simplex virus. *Virulence* 2021; **12**(1):2670-702.
50. Mirouse A, Vignon P, Piron P, et al. Severe varicella-zoster virus pneumonia: A multicenter cohort study. *Crit Care* 2017; **21**:1-10.
51. Niazi MR, Iqbal QZ, Mishiyev D, et al. Epstein-Barr virus (EBV) induced pneumonitis in an immunocompetent adult: A case report. *Respir Med Case Rep* 2020; **31**:101262.