

Current Status of Treatment for Respiratory Viral Infections

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Abstract:

Respiratory virus infections are widely prevalent worldwide and pose a serious threat to human health. Common respiratory viruses include influenza virus, respiratory syncytial virus, parainfluenza virus, human rhinovirus, etc. At present, antiviral drugs such as neuraminidase inhibitors can effectively alleviate symptoms and shorten the course of influenza virus infection in the early stages of onset, but the problem of drug resistance is gradually becoming prominent. In terms of respiratory syncytial virus infection, although drugs such as ribavirin have been used, there is controversy over their efficacy and safety, and supportive care remains the main treatment method. The current treatment of respiratory viral infections still faces many challenges, such as difficulties in diagnosis and treatment caused by viral mutations, antibiotic resistance issues, and limitations in some treatment methods. In depth research on the treatment methods of respiratory viral infections is of great significance for improving clinical efficacy and reducing disease burden.

Keywords:

Respiratory Virus Infection, Antiviral Treatment, Virus Infection

1. Introduction

Respiratory tract infection refers to a series of diseases caused by pathogens invading the respiratory mucosa. Viral upper respiratory infection is a common respiratory disease caused by a variety of viruses. It has a high incidence rate in clinical practice and occurs in all ages [1]. As a type of non cellular microorganism, viruses have a simple structure and only contain one type of nucleic acid. They must parasitize in living cells and proliferate through replication. Since Russian scientist Ivanovsky first discovered tobacco mosaic virus in 1892, virological research has made rapid progress. Worldwide, influenza virus causes approximately 39.1 million acute lower respiratory infections and 58200 deaths annually, while respiratory syncytial virus causes approximately 24.8 million infections and 76600 deaths annually. Today, viruses have been identified as one of the main pathogens of human infectious diseases, posing a serious threat to human health and social development, and causing widespread concern in society. Studying the current status of treatment for respiratory viral infections has important practical significance and urgency. From

the perspective of public health, an in-depth understanding of the effectiveness of current treatment methods and existing problems will help to develop more scientific and reasonable prevention and control strategies, improve the prevention and control of viral infectious diseases, reduce the incidence rate and mortality of diseases, and ensure public health. From a clinical treatment perspective, studying the current treatment status can provide clinical doctors with more comprehensive and accurate treatment information, guide them to choose a reasonable treatment plan, improve treatment effectiveness, reduce the occurrence of complications, and improve patient prognosis.

2. Research Objective

This study aims to comprehensively and systematically review the current treatment status of respiratory viral infections, analyze the mechanism of action, clinical application effects, and existing problems of treatment methods, in order to provide scientific basis for clinical treatment. Specifically, by analyzing the treatment methods for different types of viral infections, we summarize the advantages and disadvantages of existing treatment plans, explore how to optimize treatment strategies, improve treatment effectiveness, and reduce the harm caused by viral infections, and contribute theoretical support to promoting progress in the field of viral infection treatment.

3. The Concept of Viral Infection and Antiviral Therapy

3.1. Definition of viral infection

Viral infection refers to the process in which viruses invade the body through different pathways and proliferate in susceptible cells. As a non cellular microorganism, viruses lack a metabolic system and must parasitize within living cells, relying on the host cell's substances and energy to complete their own replication. When a virus enters the human body, it first adsorbs onto the surface of the host cell, binds to specific receptors on the cell surface, and then invades the interior of the cell. Inside the cell, the virus utilizes various enzymes, nucleic acids, proteins, and other substances of the host cell to replicate extensively according to its own genetic information, producing progeny viruses. These progeny viruses can continue to release and infect surrounding cells, causing a series of pathological and physiological changes, leading to various symptoms and diseases in the body. For example, influenza virus mainly invades respiratory epithelial cells, replicates extensively within cells, causing cell damage and death, and subsequently triggering a series of pathological changes.

3.2. Concept of antiviral therapy

Antiviral therapy refers to the treatment process of using various methods, including chemotherapy, immunotherapy, biologics, etc., to inhibit or eliminate viruses in the body, reduce the damage caused by viral infections to the body, and restore health. Its main mechanism of action is to inhibit virus replication and prevent virus invasion into host cells.

4. Classification and Therapeutic Mechanism of Viruses

4.1. Classification of viruses

DNA virus: The genetic material of this type of virus is DNA, which can be double stranded or single stranded. Double stranded DNA viruses are more common, such as EB virus, cytomegalovirus, herpes simplex virus, etc. Single stranded DNA viruses are relatively rare, and parvovirus is one of their representatives. After infecting host cells, these viruses need to use the host cell's DNA polymerase to double their single stranded DNA for subsequent replication and transcription processes. **RNA virus:** A virus whose genetic material is RNA, which can be further classified into various types based on the characteristics of its RNA. Double stranded RNA viruses, such as rotavirus, have genomes composed of double stranded RNA, and their replication process is relatively complex. They rely on their own RNA polymerase to transcribe and replicate in host cells. Justice single stranded RNA viruses, whose RNA can be directly translated as mRNA to synthesize viral proteins, such as influenza viruses and rhinoviruses, belong to this category. Anti sense single stranded RNA viruses, such as measles, mumps, etc., have complementary RNA and mRNA, which need to be transcribed into mRNA by the virus's own RNA polymerase before protein synthesis can proceed. Single stranded RNA retroviruses, represented by human immunodeficiency virus (HIV), have two single stranded RNAs. After infecting host cells, they need to be reverse transcribed into cDNA by reverse transcriptase, and then integrated into the host cell genome for assembly and replication in a manner similar to DNA viruses. Compared with DNA viruses, RNA viruses are more prone to mutations during replication due to the lack of effective error correction mechanisms, resulting in changes in the virus's antigenicity, pathogenicity, etc. This also poses greater challenges for the treatment and prevention of RNA viruses [5].

4.2. Pathogenic mechanisms of different types of viruses

4.2.1. Pathogenic mechanism of DNA virus

It mainly includes three aspects; ① Cell receptor binding and invasion of DNA viruses have specific protein structures on the surface, which can specifically bind to receptors on the host cell surface and then enter the cell through phagocytosis or membrane fusion. For example, adenovirus binds to the Cocksackievirus adenovirus receptor on the surface of host cells through its fibrin, and then enters the cells through endocytosis. ② After genome replication and transcription enter the cell, viral DNA is released into the nucleus or cytoplasm, utilizing host cell enzymes and other molecular mechanisms for genome replication and transcription. Like herpes simplex virus, after its DNA enters the nucleus, it synthesizes viral mRNA through the host cell's transcriptional enzymes, which then translates viral proteins and replicates viral DNA. ③ Protein synthesis and assembly of viral mRNA translate various viral proteins, including structural and non structural proteins, onto the ribosomes of host cells. Structural proteins participate in the assembly of viral particles, while non structural proteins play a role in virus replication, regulation of host cell functions, and other aspects. Taking human papillomavirus as an example, its late proteins L1 and L2 assemble into the viral capsid, which wraps around the viral DNA and forms complete viral particles.

4.2.2. Pathogenic mechanism of RNA viruses

It mainly includes three aspects: ① Virus adsorption and invasion: Glycoproteins or other structures on the surface of RNA viruses can bind to specific receptors on the surface of host cells, and this process has high specificity. For example, the

hemagglutinin protein of influenza virus can bind to sialic acid receptors on the surface of host respiratory epithelial cells. After binding, the virus enters the cell through endocytosis and forms an endosome. Under the acidic environment of the endosome, the viral envelope fuses with the endosome membrane, releasing the viral genome into the cytoplasm. ② Genomic replication and transcription: After entering the cell, RNA viruses utilize their own RNA dependent RNA polymerase (RdRp) and host cell related substances to replicate and transcribe the genome. For positive stranded RNA viruses, the genome itself can be directly translated as mRNA to synthesize viral proteins. At the same time, negative stranded RNA is replicated under the action of RdRp, and more positive stranded RNA genomes are synthesized using negative stranded RNA as a template. Negative stranded RNA viruses need to first use their own genome as a template to synthesize positive stranded RNA under the action of RdRp for translation and replication. Like rabies virus, its negative stranded RNA genome is transcribed into mRNA under the action of RdRp carried by viral particles, and then translated into viral proteins. ③ Protein synthesis and viral assembly: Virus mRNA translates various viral proteins, including structural and non structural proteins, onto the ribosomes of host cells. Structural proteins such as capsid proteins and envelope proteins are involved in the assembly of viral particles. Non structural proteins play a role in virus replication, regulation of host cell functions, and other aspects. For example, the structural proteins of poliovirus assemble into a capsid, wrapping around the virus genome, forming complete virus particles, and then released from the host cell.

5. Common Drugs and Their Mechanisms of Action for Viral Infections

5.1. Nucleoside antiviral drugs: Ribavirin

Ribavirin is a broad-spectrum antiviral drug that has inhibitory effects on various DNA and RNA viruses, such as respiratory syncytial virus, influenza virus, adenovirus, etc. Its mechanism of action is relatively complex and currently not fully understood. According to existing research, ribavirin mainly exerts antiviral effects through the following pathways: firstly, it inhibits the activity of inosine-5'-phosphate dehydrogenase (IMPDH), thereby suppressing the synthesis of guanosine monophosphate and blocking the synthesis of viral nucleic acids; Secondly, the phosphorylation products of ribavirin can serve as substrate analogs for viral RNA polymerase, which can be incorporated into viral RNA, leading to errors in viral RNA synthesis and affecting virus replication and transmission; Thirdly, ribavirin can enhance the body's immune response, promote the proliferation and differentiation of T cells and B cells, and improve the body's antiviral ability [6].

5.2. Protease inhibitor: Nimatevir tablets

After COVID-19 infects human cells, it will use the ribosomes of host cells to synthesize polyprotein, which is necessary for virus replication and assembly. The 3CL protease is a key protease in the replication process of COVID-19. It can recognize and cut these poly proteins to form functional viral proteins, thus promoting the replication and assembly of the virus. The main component of Nimatevir tablets is Nimatevir, which is a peptide like compound with a structure similar to the natural substrate of 3CL protease. Nimatevir can enter the active site of 3CL protease and form stable covalent bonds with the enzyme's active center, irreversibly inhibiting the

activity of 3CL protease. When the activity of 3CL protease is inhibited, the viral polyprotein cannot be correctly cleaved, and functional viral proteins cannot be formed, thereby blocking the replication and assembly process of the virus and achieving antiviral effects [7].

5.3. Neuraminidase inhibitor: Oseltamivir

After influenza infects the human body, it replicates and assembles within host cells to form new viral particles. These new viruses need to be released from the surface of infected cells in order to continue infecting surrounding cells and expanding the scope of infection. Neuraminidase is a glycoprotein on the surface of influenza virus, which can catalyze the hydrolysis of sialic acid residues at the end of the glycoprotein on the host cell surface, allowing the virus to be released from the cell surface. The metabolic product of oseltamivir in the body, oseltamivir carboxylate, can tightly bind to the active site of neuraminidase, forming a stable complex that inhibits the activity of neuraminidase. The newly generated virus particles cannot be released from the surface of infected cells, thus blocking the transmission and spread of the virus in the body.

5.4. Non nucleoside antiviral drugs: Acyclovir

After entering virus-infected cells, acyclovir is converted into acyclovir monophosphate by the action of thymidine kinase encoded by the virus. Subsequently, under the action of intracellular guanylate kinase and other enzymes, it is further converted into acyclovir diphosphate and acyclovir triphosphate. Among them, acyclovir triphosphate is the main form that exerts antiviral effects. It can bind to viral DNA polymerase and inhibit its activity. Viral DNA polymerase is a crucial enzyme in the process of viral replication, responsible for synthesizing new DNA strands using viral DNA as a template. Acyclovir triphosphate competes with natural deoxyribonucleoside triphosphate for the active site of viral DNA polymerase and is incorporated into the synthesized viral DNA strand. Due to the lack of the necessary 3'-OH group for DNA strand extension in acyclovir triphosphate, the synthesis of DNA strands is terminated, thereby blocking the replication of viral DNA [8].

5.5. Immunomodulatory antiviral drugs: immunoglobulin

Specific antibodies in immunoglobulin can recognize and bind to antigens on the surface of the virus, forming antigen antibody complexes. This binding can prevent the virus from binding to receptors on the surface of the host cell, thereby blocking the virus from entering the cell, making it lose infectivity, and unable to replicate and spread in the body. Immunoglobulin can also enhance the phagocytic activity of phagocytic cells towards viruses. After the antibody binds to the virus, its Fc segment can bind to the Fc receptor on the surface of phagocytic cells, making the virus more easily recognized and engulfed by phagocytic cells, thereby accelerating virus clearance. Especially for children with immunodeficiency, they are more susceptible to RSV invasion and death [9]. Immunoglobulin can regulate the immune function of the body, interact with relevant receptors on the surface of immune cells, affect the activation, proliferation, and differentiation of immune cells, thereby enhancing the body's antiviral immune response.

6. Problems in the Treatment of Viral Infections

6.1. Variation and Drug Resistance of Viruses

6.1.1. Causes and mechanisms of viral mutations

Virus mutations mainly stem from changes in its genetic material, and RNA viruses are more prone to mutations during replication due to the lack of effective error correction mechanisms. COVID-19 is a typical RNA virus. Its genome is single stranded positive strand RNA. When replicating, RNA polymerase lacks the proofreading function. Each replication will introduce about 1-2 nucleotide mutations. This high frequency mutation makes COVID-19 constantly produce new variants in the process of transmission, such as Delta variants and Omicron variants. These mutant strains have undergone multiple amino acid mutations in key areas such as spike proteins, resulting in changes in their transmission ability, pathogenicity, and immune escape ability. The mutation of viruses is closely related to the selection pressure of the external environment. When viruses are exposed to antiviral drugs, vaccines, or the host immune system, they are subject to selection pressure. For example, under the action of antiviral drugs, the virus undergoes adaptive mutations in order to survive and continue to spread, leading to the development of drug resistance. Taking influenza virus as an example, long-term use of neuraminidase inhibitors (such as oseltamivir) for treatment can lead to mutations in the neuraminidase gene of influenza virus, resulting in a decrease in the binding ability between the drug and the enzyme, thereby reducing the efficacy of the drug. In the environment of vaccination, viruses may also mutate to evade vaccine induced immune responses. In addition, the continuous attacks of the host immune system can also cause the virus to mutate in order to evade recognition and clearance by host immune cells [10].

6.1.2. The emergence and impact of drug resistance

Drug resistance is an adaptive change gradually developed by viruses under drug selection pressure, which has a serious impact on treatment efficacy and disease control. When the virus is exposed to antiviral drugs for a long time, its genome undergoes mutations, causing changes in the target of drug action, making it unable to effectively bind to the target and lose its inhibitory effect on the virus. The spread of drug-resistant viruses can form a reservoir of drug-resistant viruses in the population, increasing the difficulty of treating viral infections. Once drug-resistant viruses spread among the population, ordinary antiviral drugs will be difficult to exert their effects, making the epidemic difficult to control. In the treatment of influenza, the spread of drug-resistant influenza viruses can greatly reduce the effectiveness of influenza treatment, increasing the hospitalization and mortality rates of patients. For example, in some regions, due to the prevalence of drug-resistant influenza viruses, the therapeutic effect of commonly used anti influenza drugs such as oseltamivir has significantly decreased. Doctors have to use other drugs or combination therapy to treat influenza patients, which not only increases treatment costs but may also bring more adverse drug reactions. Therefore, the emergence and spread of drug resistance pose a huge challenge to the treatment and control of viral infections, and effective measures need to be taken to prevent and respond.

6.2. Limitations of Antiviral Drugs

6.2.1. Drug side effects

Antiviral drugs often cause many side effects and even liver function damage during the treatment of viral infections, posing certain risks to patients. For example, the nucleoside antiviral drug ribavirin commonly has side effects such as anemia, fatigue, nausea, vomiting, etc. This is because ribavirin not only inhibits viral nucleic acid synthesis, but also has a certain impact on the nucleic acid metabolism of normal cells in the human body, leading to a decrease in red blood cell production and causing anemia symptoms. The non nucleoside antiviral drug amantadine may also have some side effects when treating influenza A. Common side effects include central nervous system symptoms such as dizziness, difficulty falling asleep, and gastrointestinal discomfort. The central nervous system side effects of amantadine may be related to its impact on the dopaminergic and cholinergic systems of the central nervous system. In some elderly individuals or patients with renal insufficiency, the side effects of amantadine may be more pronounced, and may even lead to serious psychiatric symptoms such as hallucinations and delirium. Therefore, when using amantadine, the dosage needs to be adjusted according to the patient's age, renal function, and other conditions to reduce the occurrence of side effects.

6.2.2. Narrow scope of drug application

An antiviral drug often only works against specific viruses, which makes its scope of application relatively narrow and limited when facing multiple viral infections. For example, acyclovir is mainly used to treat herpes virus infections such as herpes simplex virus, varicella zoster virus, etc. This is because acyclovir can be specifically phosphorylated by thymidine kinase encoded by herpes virus, converting it into active triphosphate acyclovir, thereby inhibiting herpes virus DNA polymerase and blocking virus DNA synthesis. However, for other types of viruses, such as influenza virus and hepatitis B virus, because they do not have the thymidine kinase encoded by herpes virus, acyclovir cannot be activated, so it cannot play an antiviral role. In the treatment of influenza, neuraminidase inhibitors such as oseltamivir mainly target the influenza virus. The neuraminidase on the surface of influenza virus plays a crucial role in the release and transmission of the virus. Neuraminidase inhibitors can bind to neuraminidase, inhibit its activity, and thus suppress the release of the virus from infected cells, reducing the spread of the virus. However, these drugs have almost no therapeutic effect on other respiratory viruses such as respiratory syncytial virus, adenovirus, etc. Because the infection and transmission mechanisms of these viruses are different from influenza viruses, they do not rely on neuraminidase for release and transmission, so neuraminidase inhibitors cannot act on them.

7. Prospects for Antiviral Therapy

7.1. Development Trends of New Antiviral Drugs

7.1.1. Targeted therapy drugs

With the continuous deepening of research on the pathogenic mechanism of viruses, the development of drugs targeting specific virus targets has become an important trend. For example, for the treatment of COVID-19, researchers focus on the key proteins of the virus, such as spike protein, main protease (Mpro) [11] and RNA dependent RNA polymerase (RdRp). By analyzing the structure and function of these targets, drugs that can specifically inhibit virus replication and transmission have been

developed. For example, Nematvir/Ritonavir tablets, in which the 3CL protease of COVID-19 can be inhibited by it, blocking the cutting and processing of viral polymeric proteins, thereby inhibiting the replication of the virus; Litonavir inhibits the cytochrome P450 enzyme system, slows down the metabolism of remdesivir, increases its blood concentration, and enhances its antiviral effect [12]. Clinical studies have shown that the use of nimativir/ritonavir tablets in the early stage of COVID-19 infection can effectively reduce the viral load of patients, alleviate symptoms, and reduce the risk of severe disease and death [13]. In the field of influenza treatment, there are also new drug developments targeting specific targets of influenza virus. For example, Mabaloxavir is a novel anti influenza virus drug that targets the influenza virus with endonucleases, inhibiting the synthesis of viral mRNA and thus preventing virus replication. Compared with traditional neuraminidase inhibitors, balosavir ester has a unique mechanism of action and advantages. It only requires a single oral administration to rapidly inhibit virus replication and shorten the duration of influenza symptoms. Clinical studies have shown that balosavir ester has good efficacy and safety in treating influenza, especially for high-risk populations such as children [14].

7.1.2. Immunotherapy drugs

Immunotherapy drugs have shown broad prospects in antiviral therapy, opening up new avenues for the treatment of viral infections. By activating or regulating the body's immune system, immunotherapy drugs can enhance the body's immune response to viruses and improve antiviral ability. For respiratory syncytial virus (RSV) infection, pembrolizumab is a commonly used monoclonal antibody drug that can bind to the F protein on the surface of RSV, block the binding of the virus to cell receptors, and prevent and treat RSV infection. In high-risk infants and young children, prophylactic treatment with pembrolizumab can significantly reduce the incidence and severity of RSV infection [15].

7.2. Challenges and Opportunities of Future Antiviral Therapy

7.2.1. Challenge

The continuous mutation of viruses and the continuous development of drug resistance have brought severe challenges to antiviral therapy. Viruses have very high variability, especially RNA viruses, such as influenza virus, COVID-19, and so on. Their genetic material is prone to mutation in the process of replication. These mutations may cause changes in the antigenicity of the virus, rendering existing vaccines and antiviral drugs ineffective. For example, the influenza virus undergoes varying degrees of mutation every year, and each mutation leads to an increase in the virus's transmission ability [16]. During the global pandemic, COVID-19 also appeared several variants, such as Delta variant and Omikron variant, which have significantly changed in transmission capacity, pathogenicity and other aspects, bringing great difficulties to epidemic prevention and control and treatment [17]. Drug resistance is another major challenge faced by antiviral therapy. Long term use of antiviral drugs can greatly increase one's own drug resistance. Once the virus develops drug resistance, its replication becomes increasingly difficult to suppress, leading to treatment failure. In addition, the spread of drug-resistant viruses can also form a pool of drug-resistant viruses in the population, further exacerbating the difficulty of antiviral treatment. The high cost and long cycle of antiviral drug development also limit the speed of new drug launches [18]. The development of a

new antiviral drug requires multiple stages, including basic research, drug design, clinical trials, etc. Each stage requires significant investment of funds and time. This poses enormous risks and pressures for pharmaceutical companies in the research and development of antiviral drugs, limiting the speed of new drug development and market launch.

7.2.2. Research Shortcomings and Prospects

Although this study provides a comprehensive analysis of the current status of treatment for viral infections, there are still certain limitations. During the research process, the acquisition of some data is limited by geography and time, which may not fully reflect the true situation of viral infection treatment worldwide. The depth and breadth of analysis in the treatment research of some novel viral infections need to be further strengthened. In addition, when exploring the prospects of antiviral therapy, although an analysis of new technologies and trends has been conducted, these contents still have a certain degree of foresight, and the actual application effects and problems faced still need further research and verification. In the future, the field of viral infection treatment has broad research space and development prospects. We should strengthen research on the mechanisms of virus mutation and drug resistance, providing a theoretical basis for the development of new antiviral drugs and treatment strategies. Increase investment in the research and development of new antiviral drugs, especially drugs targeting specific virus targets and immunotherapy drugs, to improve treatment efficacy and safety. Further optimize the combination therapy and personalized treatment strategies, develop precise treatment plans based on the patient's condition, improve the effectiveness of treatment and the patient's quality of life, and accelerate the innovation and development of antiviral therapy technology.

8. Conclusions

The field of respiratory virus infection treatment is constantly developing and changing, and the current treatment status presents multidimensional characteristics. From the perspective of treatment methods, symptomatic treatment is still a basic and widely used strategy. In terms of treatment effectiveness, early standardized treatment can achieve good results for some viral infections with specific antiviral drugs. However, the overall treatment of upper respiratory viral infections still faces many challenges. In the future, it is necessary to further strengthen basic research to develop more specific antiviral drugs, optimize clinical treatment plans, improve comprehensive treatment levels, and more effectively respond to the threat of respiratory virus infections to human health.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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