

CLINICAL PHARMACOLOGY OF ANTIVIRAL DRUGS

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ANNOTATION

This article discusses the clinical pharmacology, mechanism of action, time of action, types and composition of antiviral drugs. The article introduces new antiviral drugs being developed in the pharmaceutical industry, their pharmacotherapeutic groups, international non-patented names, and the country of manufacture. Nowadays, the need for antiviral drugs is increasing. Because it is the time when seasonal viral diseases and allergies are on the rise and immunity decreases to a certain extent. Therefore, we will get acquainted with the new anti-viral drugs included in the State Register.

Keywords: antiviral drugs, integrase inhibitors, mechanism of action, nucleoside and nucleotide reverse transcriptase inhibitors, protease inhibitors, viral infections.

INTRODUCTION

Viruses are major pathogenic agents causing a variety of serious diseases in humans, other animals, and plants.

Viruses are one of the most widespread of all organisms and are capable of infecting every species of animal from mammals down to insects, plants, and even bacteria. It seems there are more species of viruses in the world than of all other creatures put together.

The most common viral infections are respiratory (infections of the nose, throat, upper airways, and lungs); gastrointestinal (gastroenteritis); liver (hepatitis); and skin (warts or other blemishes, rashes).

Viral diseases include influenza (causing fever, severe aching, and catarrh, often occurring in epidemics); severe acute respiratory syndrome (a form of pneumonia); chickenpox (disease caused by the herpes zoster virus, which manifests in a mild fever and a rash of itchy inflamed blisters); herpes (herpes simplex or herpes zoster, causing the eruption of small blister-like vesicles on the skin or mucous membranes); hepatitis (a disease characterized by inflammation of the liver); cold sores (diseases affecting mouth or genitals); measles (disease causing fever and a red rash on the skin, typically occurring in childhood); shingles (painful inflammation of the nerve ganglia, with a skin eruption often forming a girdle around the middle of the body); poliomyelitis (disease that affects the central nervous system that can cause temporary or permanent paralysis); acquired immunodeficiency syndrome (AIDS) caused by human immunodeficiency virus (HIV) (disease includes dry cough or shortness of breath, difficult or painful swallowing, diarrhea, white spots or unusual blemishes in and around the mouth, pneumonia-like symptoms, fever, vision loss, nausea, abdominal cramps, and vomiting); smallpox (disease started from fever, overall discomfort, headache, severe fatigue, severe

back pain, vomiting. A few days later, flat, red spots appear on whole trunk, which become lesions. Occur first in the mouth and spread to the face, then to entire body); rabies (a contagious and fatal viral disease that causes madness and convulsions); dengue (jungle fever) causing sudden fever and acute pains in the joints); Ebola (fatal disease marked by fever and severe internal bleeding); and Lassa (fever with headaches, mouth ulcers, muscle aches, hemorrhages under the skin, heart and kidney failure, and a high mortality rate).

Antiviral medication and its mechanism of action

Acyclovir

Acyclovir is the basis of 2'-deoxiguanosin which applies antiviral effects after manipulation on acyclovir triphosphate. The hidden development of this methodology, an increase in acyclovir monophosphate, is catalysed by thymidine kinase caused by cells contaminated by herpes simplex infection^{11,12} or varicella zoster infection or phosphotransferase made by cytomegalovirus. Cellular protein then adds phosphate to produce acyclovir diphosphate and acyclovir triphosphate. Acyclovir triphosphate slows the mixing of viral DNA by countering 2'-deoxy guanosin triphosphate as a substrate for viral DNA polymerase.^{11,12} After acyclovir (not 2'-deoxiguanosin) was implanted in a duplicate of viral DNA, fusion stopped.

Valacyclovir

Valacyclovir, L-valyl ester from acyclovir, is also available in oral form. After swallowing, drug is immediately changed to acyclovir by the substance valacyclovir hydrolase in the digestive tract and liver. The original bioavailability is three to several times that of acyclovir.¹⁹ Valacyclovir has proven exceptional in treatment of pollution obtained by the herpes simplex virus and varicella-zoster virus and in prophylaxis against cytomegalovirus. Ganciclovir, which starts overseeing the Journal late, contrasts with acyclovir by extending a hydroxymethyl group in position 3' from a non-cyclic side chain. The assimilation and arrangement of its action are similar to acyclovir, on the other hand, it actually has carbon 3' with a hydroxyl package that can allow the widening of the foundation design similar to levelled DNA chain terminators.

Penciclovir

Penciclovir is basically like ganciclovir, in contrast only by replacing the methylene connection for oxygen either in the non-cyclic ribose portion of the particle. Its digestive component and activity are similar to acyclovir, so again, it is only a DNA chain terminator that is bound. The inhibitory effect of in vitro penciclovir on herpes simplex 1 and 2 types and varicella-zoster infection is alike to acyclovir.²² Now, it has claimed only as topical plan for the treatment of cold sores. Intravenous preparations are considered as treatment for mucocutaneous herpes in immunocompromised patients.

Famciclovir

Famciclovir is a simple diacetyl-6-deoxy from penciclovir. All this is assimilated after oral organisation and is quickly used for penciclovir by deacetylation in digestive tract, blood and liver, next it is oxidised by liver in position 6 of purine cycle. Half of the presence of a dynamic intracellular drug, penciclovir triphosphate, is very long, offering the possible for a dose once a day. Famciclovir works against genital herpes and the shingles virus.²³

Conclusion

The fight between human and viruses in on and both are rapidly improving the strategies of attacking and defence. In recent years, there has been tremendous progress in understanding the

genetic basis and molecular mechanism of diseases. Various new drugs have been formulated and the development of a lot more is in underway. Though, the new infectious diseases caused by viruses such as COVID-19 remain a challenge. Furthermore, the drugs failure in human trials is a general process that requires to be worked out and addressed. The promising results are expected through the emergence of many new technologies.

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