IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

REVIEW: HERPES

[1]Pravin B.Solanki, [2]Azhar C. Shaikh, [3]Nasir S. Tamboli, [4]Suhas J. Khandagle, [5]Bhikkan I. Basale

[1]B PHARM, [2]B PHARM, [3]B PHARM, [4]B PHARM, [5]B PHARM.

AAEMF's Delight College of pharmacy, Pimple jagtap road,Koregaon Bhima, Maharashtra 412216

Abstract: The herpes in one of the deadly and painful disease present in the world there are mainly 2 types of herpes oral one and the genital one can result in clinically significant neurologic impairment or death as the death rate of herpes is as much as 78 percent people didn't treat it as dangerous disease or people are ashamed to go to doctor for the future treatment of disease due to the society fear they just left it untreated and death occurs in this article we help people to understand what herpes is how it can be treated properly

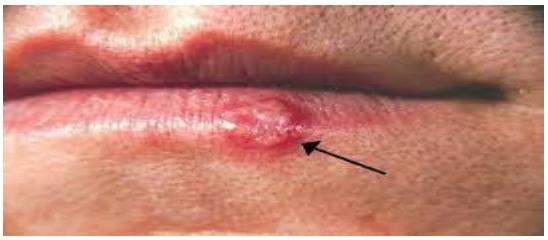
I. INTRODUCTION

Herpes simplex is infection caused by the herpes simplex virus Infections are differentiate on the part of the body infected. Oral herpes involves the face or mouth. It may result in small blisters in groups often called cold sores or fever blisters or may just cause a sore throat. Genital herpes, often simply known as herpes, may have minimal symptoms or form blisters that break open and result in small ulcers. These typically heal over two to four weeks. Tingling or shooting pains may occur before the blisters appear. Herpes cycles between periods of active disease followed by periods without symptoms. The first episode is often more severe and may be associated with fever, muscle pains, swollen lymph nodes and headaches. Over time, episodes of active disease decrease in frequency and severity. Other disorders caused by herpes simplex include: herpetic whitlow when it involves the fingers, herpes of the eye, herpes infection of the brain, and neonatal herpes when it affects a newborn, among others.

The herpes is really painful worldwide rates of either HSV-1 or HSV-2 are between 60% and 95% in adults HSV-1 is usually acquired during childhood. Rates of both increase as people age Rates of HSV-1 are between 70% and 80% in populations of low socioeconomic status and 40% to 60% in populations of improved socioeconomic status.An estimated 536 million people worldwide (16% of the population) were infected with HSV-2 as of 2003 with greater rates among women and those in the developing world. Most people with HSV-2 do not realize that they are infected The name is from Greek: ἕρπης herpēs, which is related to the meaning "to creep", referring to spreading blisters. The name does not refer to latency.

A new study has opened the door to a new approach to attacking herpesviruses. The study demonstrated that targeting 2 metal ion-dependent enzymes of human herpesviruses with 2 compounds, AK-157 and AK-166, can inhibit the replication of the virus.25-Jan-2022

Early efforts to produce a protein-based vaccine for herpes failed. But a new mRNA approach has outperformed the efficacy of the past vaccines in preclinical trials and is expected to be introduced in clinical trials in the second half of 2022, investigators say.01-Mar-2022



DEFINITION: Herpes is an infection that is caused by a herpes simplex virus (HSV). Oral herpes causes cold sores around the mouth or face. Genital herpes affects the genitals, buttocks or anal area. Genital herpes is a sexually transmitted disease (STD).

HISTORY:

Hippocrates is known to have described the cutaneous spreading of herpes simplex lesions and scholars of Greek civilization define the greek word "herpes" to mean "to creep or crawl" in reference the spreading nature of the herpetic skin lesionsAnd in 1919, Lowenstein confirmed experimentally the infectious nature of HSV that Shakespeare had only suspected. In the 1920's and 1930's, the natural history of HSV was widely studied and it was found that HSV not only infects the skin, but also the central nervous system.

Herpes was not found to be a virus until the 1940s. Herpes antiviral therapy began in the early 1960s with the experimental use of medications that interfered with viral replication called deoxyribonucleic acid (DNA) inhibitors.

Herpes simplex has been infecting hominids for millions of years, but it wasn't until 1967 that scientists first distinguished between the HSV-1 and HSV-2 types, effectively creating the concept of "genital herpes.

The virus originated in chimpanzees, jumping into humans 1.6 million years ago.

Researchers at the University of California, San Diego School of Medicine have identified the evolutionary origins of human herpes simplex virus (HSV) -1 and -2, reporting that the former infected hominids before their evolutionary split from chimpanzees 6 million years ago while the latter jumped from ancient

Herpes has been known for at least 2,000 years. Emperor Tiberius is said to have banned kissing in Rome for a time due to so many people having cold sores. In the 16th-century Romeo and Juliet, blisters "o'er ladies' lips" are mentioned. In the 18th century, it was so common among prostitutes that it was called "a vocational disease of women".The term 'herpes simplex' appeared in Richard Boulton's A System of Rational and Practical Chirurgery in 1713, where the terms 'herpes miliaris' and 'herpes exedens' also appeared. Herpes was not found to be a virus until the 1940s.

Herpes antiviral therapy began in the early 1960s with the experimental use of medications that interfered with viral replication called deoxyribonucleic acid (DNA) inhibitors. The original use was against normally fatal or debilitating illnesses such as adult encephalitis, keratitis,] in immunocompromised (transplant) patients, or disseminated herpes zoster. The original compounds used were 5-iodo-2'-deoxyuridine, AKA idoxuridine, IUdR, or(IDU) and 1-β-D-arabinofuranosylcytosine or ara-C, later marketed under the name cytosar or cytarabine. The usage expanded to include topical treatment of herpes simplex, zoster, and varicella. Some trials combined different antivirals with differing results. The introduction of 9-β-D-arabinofuranosyladenine, (ara-A or vidarabine), considerably less toxic than ara-C, in the mid-1970s, heralded the way for the beginning of regular neonatal antiviral treatment. Vidarabine was the first systemically administered antiviral medication with activity against HSV for which therapeutic efficacy outweighed toxicity for the management of life-threatening HSV disease. Intravenous vidarabine was licensed for use by the U.S. Food and Drug Administration in 1977. Other experimental antivirals of that period included: heparin, trifluorothymidine (TFT),] Ribivarin, interferon, Virazole, and 5-methoxymethyl-2'-deoxyuridine (MMUdR). The introduction of 9-(2-hydroxyethoxymethyl)guanine, AKA aciclovir, in the late 1970s] raised antiviral treatment another notch and led to vidarabine vs. aciclovir trials in the late 1980s. The lower toxicity and ease of administration over vidarabine has led to aciclovir becoming the drug of choice for herpes treatment after it was licensed by the FDA in 1998. Another advantage in the treatment of neonatal herpes included greater reductions in mortality and morbidity with increased dosages, which did not occur when compared with increased dosages of vidarabine. However, aciclovir seems to inhibit antibody response, and newborns on aciclovir antiviral treatment experienced a slower rise in antibody titer than those on vidarabine.

Early 20th century public health legislation in the United Kingdom required compulsory treatment for sexually transmitted diseases but did not include herpes because it was not serious enough. As late as 1975, nursing textbooks did not include herpes as it was considered no worse than a common cold.[24] After the development of acyclovir in the 1970s, the drug company Burroughs Wellcome launched an extensive marketing campaign that publicized the illness, including creating victim's support groups.

II. PATHOPHYSIOLOGY:

HSV-I and HSV-2 are characterized by the following unique biological properties

- Neurovirulence (the capacity to invade and replicate in the nervous system)
- Latency (the establishment and maintenance of latent infection in nerve cell ganglia proximal to the site of infection): In orofacial HSV infections, the trigeminal ganglia are most commonly involved, while, in genital HSV infection, the sacral nerve root ganglia (S2-S5) are involved.
- Reactivation: The reactivation and replication of latent HSV, always in the area supplied by the ganglia in which latency was established, can be induced by various stimuli (eg, fever, trauma, emotional stress, sunlight, menstruation), resulting in overt or covert recurrent infection and shedding of HSV. In immunocompetent persons who are at an equal risk of acquiring HSV-I and HSV-2 both orally and genitally, HSV-I reactivates more frequently in the oral rather than the genital region. On the other hand, HSV-2 reactivates 8-10 times more commonly in the genital region than in the orolabial regions. Reactivation is more common and severe in immunocompromised individuals.

Cellular immunity is an important defense against herpes simplex. Dissemination of herpes simplex infection can occur in people with impaired T-cell immunity, such as in organ transplant recipients and in individuals with AIDS. Herpes simplex infection can also complicate burn wounds or damaged skin such as in atopic dermatitis or other allergic dermatoses.

HSV is distributed worldwide. Humans are the only natural reservoirs, and no vectors are involved in transmission. Endemicity is easily maintained in most human communities owing to latent infection, periodic reactivation, and asymptomatic virus shedding. HSV is transmitted by close personal contact, and infection occurs via inoculation of virus into susceptible mucosal surfaces (eg, oropharynx, cervix, conjunctiva) or through small cracks in the skin. The virus is readily inactivated at room temperature and by drying; hence, aerosol and fomite spread are rare.

III. TYPES:

1. ORAL HERPES:

Oral herpes causes blisters, sometimes called fever sores or cold sores, to develop in or around the lips and mouth. Sometimes these blisters form elsewhere on the face or on the tongue, and more rarely on other areas of skin.
The sores usually last 2–3 weeks at a time.

2. GENITAL HERPES:

These sores tend to develop on the penis, around or inside the vagina, on the buttocks, or on the anus, though they can form on other areas of skin.

Herpes can also cause pain when urinating and changes in vaginal discharge.
The first time a person develops the sores, they may last 2–6 weeks.
Soon after this initial outbreak, symptoms may recur frequently. Over time, outbreaks may occur less often and the symptoms tend to become less severe

IV. CAUSES:

When HSV is present on the skin, it can easily pass from person to person through contact with the moist skin of the mouth and genitals, including the anus.

The virus may also spread through contact with other areas of the skin and the eyes.
A person cannot contract HSV by touching an object or a surface, such as a washbasin or towel.
Infection can occur in the following ways:

- having vaginal or anal sex without using barrier protection, such as a condom
- sharing sex toys
- having any other oral or genital contact with a person who has herpes

The virus is most contagious between the time when symptoms first appear and when they heal. Less commonly, a person can transmit the virus when symptoms are not present.

If a woman with genital herpes has sores while giving birth, the virus can pass on to the baby.

V. SIGNS AND SYMPTOMS:

Herpes type 1 (HSV-1), or oral herpes infection is asymptomatic in most of the cases even most people with HSV-1 infection are unaware they are infected. Symptoms of Herpes type 1 (HSV-1), or oral herpes include painful blisters or open sores called ulcers in or around the mouth. Sores present at the lips are commonly referred to as “cold sores.” Infected persons will often experience a tingling, itching or burning sensation around their mouth, before the appearance of sores. After initial infection, the blisters or ulcers can periodically recur. Genital herpes caused by HSV-1 can be asymptomatic or can have mild symptoms that go unrecognized. When symptoms do occur, genital herpes is characterised by 1 one or more genital or anal blisters or ulcers. After an initial genital herpes episode, which may be able to be severe, symptoms may recur. However, genital herpes caused by HSV-1 typically does not recur frequently, unlike genital herpes caused by herpes simplex virus type 2 (HSV-2)

VI. DIAGNOSIS:

1. **Viral culture**:
This test involves taking a tissue sample or scraping of the sores for examination in the laboratory.

2. **Polymerase chain reaction (PCR) test**.
PCR is used to copy your DNA from a sample of your blood, tissue from a sore or spinal fluid. The DNA can then be tested to establish the presence of HSV and determine which type of HSV you have.

3. **Blood test**:
This test analyzes a sample of your blood for the presence of HSV antibodies to detect a past herpes infection.

4. Direct tests:

It endeavor to demonstrate the presence of HSV in a suspicious lesion or in genital secretions. Ideally, the sample should be taken from a vesicular lesion that has been present for less than 24 h because once the lesion has begun to crust, the test sensitivity will decline. If multiple vesicles are present, more than one lesion should be sampled. In addition, test sensitivity is lower in patients with recurrent lesions than in those with first episodes

5. Viral isolation:

Standard viral culture: Tube culture isolation is the traditional gold standard for HSV detection and the reference method against which all other tests are measured (16,17). While the test has 100% specificity for HSV-1 or HSV-2, the sensitivity depends on the stage of the lesion at the time of specimen collection. The sensitivity also varies from 75% for first episodes to 50% for recurrence

VII. TREATMENT:

1. UNANI:

- Evacuation of yellow bile and sanguine (Tanqiya-e- Safra wa Dam) through purgation (Ishaal) for bilious part and bloodletting (Fasd) for sanguineous part, respectively
- To normalize the heat of sanguine (Itfa-e- Hiddat-e- Dam)
- Topical cooling (Tabreed-e- Mmaqaami) to relieve the symptoms
- Local desiccation (Tajfeef-e-maqaami)

Pharmacotherapy (Ilaj Bil Dawa):
Oral administration of Ma-ul- Fawaakih with Saqmuniya (Convolvulus scammonies Linn.) for purgation of yellow bile (Ishaal-e-Safra).Oral administration of "Joshanda-e- Halaila" for purgation of yellow bile (Ishaal-e- Safra).
Local application of paste prepared with Arminium earth (Gil-e- Armani), Berberis aristata DC. (Rasaut) and Camphor (Kaafoor) mixed with egg white.

Local application of ointment prepared with unripe Quercus infectoria Oliv. (Maazu Kham), dried leaves of Myrtus communis Linn. (Barg-e- Aas Khushk), Roghan-e- Gul and Wax (Mom) 3/4 of all the contents.
Local application of paste prepared with Arminium earth (Gil-e- Armani), Santalum album Linn. (Sandal Safed), Rosa damascene Mill. (Gul-e- Surkh), flower of Punica granatum Linn. (Gulnar) mixed with juice of leaves of Solanum nigrum Linn. (Aab-e- Inab-us- Sa'lab)

2. AYURVEDA:

Treatment plan was done considering **Vatpitta Dosha, Rakt Dhatu, Tvacha Sthan**. Removal of Dushta Rakta along with Shaman through internal medicines was considered. Involvement of Ambu (~Kled) is also considered as important factor during planning the treatment Ayurveda Treatment was planned considering Vyadhi Sankar of Prameha and Visarpa. S. Table 3 summarizes various properties of internal medicines mentioned in Ayurveda . Easy availability of these medicines at our hospital and Ayurveda description of the medicines both were given importance to choose particular medicines. Treatment of existing type 2 diabetes was continued as per allopathic doctors.

First setting of leech therapy was done at the site of blister immediately after examination of patient and ensuring negative for HIV and Hepatitis B on the same day. Total three leech application seating's were required to achieve complete recovery. Three leeches of medium size were used in each seating. Standard Operating Protocol was followed as mentioned by Kumar et al. Same leeches were used for all three seating's undertaken.

Powders of I Shatavari 2gm (Asparagus racemosus) þ Gokshur 2 gm (Tribulus terestris) þ Lodhra (Symplococus racemosus) 1gm was given internally considering their Pittaghna, Rasayan and Kledghna properties respectively as well as availability at our hospital. Fisillax ointment, that is easily available in our hospital pharmacy was given for local application for 8 days. This ointment is mainly indicated in burning and wound healing. On day-9 Narikel Mashi was added along with other medications. Details of content of fissilax are given in Table 1.
During the course of treatment antiviral, analgesics and other treatment for herpes were stopped. Treatment for Ischemic heart disease and hypertension was continued. Antiplatelet medicines were withheld on the day of application of leech

3. ALLOPATHY:

There is no cure for herpes simplex. The good news is that sores often clear without treatment. Many people choose to treat herpes simplex because treatment can relieve symptoms and shorten an outbreak.

Most people are treated with an antiviral medicine. An antiviral cream or ointment can relieve the burning, itching, or tingling. An antiviral medicine that is oral (pills) or intravenous (shot) can shorten an outbreak of herpes.

Prescription antiviral medicines approved for the treatment of both types of herpes simplex include:

- Acyclovir
- Famciclovir
- Valacyclovir

Taken daily, these medicines can lessen the severity and frequency of outbreaks. They also can help prevent infected people from spreading the virus.

4. HOME REMEDIES:

The following could help relieve herpes symptoms for some people:

- dabbing cornstarch onto the affected area
- squirting water from a bottle onto blisters to ease pain while urinating
- applying aloe vera gel to sores
- However, no research indicates that these remedies work.

A person might also try:

- taking pain relief medication, such as acetaminophen or ibuprofen
- bathing in lightly salted water or soaking in a warm sitz bath
- applying petroleum jelly to the affected areas
- wearing loose clothing to avoid irritation
- refraining from sexual activity, even with protection, until symptoms have gone
- applying a cream or lotion to the urethra before urinating, such as one that contains lidocaine

VIII. PREVENTION:

The following strategies can reduce the risk of developing or passing on herpes:

- using barrier protection, such as condoms, when having sex
- voiding sex while symptoms are present
- avoiding kissing and oral sex when there is a cold sore around the mouth
- washing the hands thoroughly, especially after touching the affected area, during an outbreak

IX. CONCLUSION:

The herpes is one of the most ignored type of the disease with high death toll one should not take it lightly this article is about to create awareness among the people about the causes diagnosis and treatment of this disease so the safety of people with proper treatment maintain.

X. **ACKNOWLEDGMENTS**:

Pravin, Azhar ,Nasir ,Suhas , Bhikkan we like to express special thanks to respected principal sir Dr. A.M.Shaikh and Head of department Mrs. Mangal S. Gaikwad mam who inspired us to do this article on such interesting topic and guided thought the information collection.

REFERENCE:

[1] Gnann JWJr, Whitley RJ. Herpes zoster, N Engl J Med, 2002, vol. 347 (pg. 340-6)

[2] Dubinsky RM, Kabbani H, El-Chami Z, Boutwell C, Ali H. Practice parameter: treatment of postherpetic neuralgia: an evidence-based report of the Quality Standards Subcommittee of the American Academy of Neurology, Neurology, 2004, vol. 63 (pg. 959-65)

[3] Hempenstall K, Nurmikko TJ, Johnson RW, A'Hern RP, Rice AS. Analgesic therapy in postherpetic neuralgia: a quantitative systematic review, PLoS Med, 2005, vol. 2 pg. e164

[4] Ku CC, Zerboni L, Ito H, Graham BS, Wallace M, Arvin AM. Varicella-zoster virus transfer to skin by T cells and modulation of viral replication by epidermal cell interferon-α, J Exp Med, 2004, vol. 200 (pg. 917-25)

[5] Chen JJ, Gershon AA, Li ZS, Lungu O, Gershon MD. Latent and lytic infection of isolated guinea pig enteric ganglia by varicella zoster virus, J Med Virol, 2003, vol. 70 Suppl 1(pg. 71-8)

[6] Gershon AA, Sherman DL, Zhu Z, Gabel CA, Ambron RT, Gershon MD. Intracellular transport of newly synthesised varicella-zoster virus: final envelopment in the trans-Golgi network, J Virol, 1994, vol. 68 (pg. 6372-90)

[7] Cole NL, Grose C. Membrane fusion mediated by herpesvirus glycoproteins: the paradigm of varicella-zoster virus, Rev Med Virol, 2003, vol. 13 (pg. 207-22)

[8] Wang ZH, Gershon MD, Lungu O, Zhu Z, Gershon AA. Trafficking of varicella-zoster virus glycoprotein gI: T(338)-dependent retention in the trans-Golgi network, secretion, and mannose 6-phosphate-inhibitable uptake of the ectodomain, J Virol, 2000, vol. 74 (pg. 6600-13)

[9] Ragozzino MW, Melton LJIII, Kurland LT, Chu CP, Perry HO. Population-based study of herpes zoster and its sequelae, Medicine, 1982, vol. 61 (pg. 310-6)

[10] ullooly JP, Riedlinger K, Chun C, Weinmann S, Houston H. Incidence of herpes zoster, 1997–2002, Epidemiol Infect, 2005, vol. 133 (pg. 245-53)

[11] rogan PM, Gronseth GS. Practice parameter: steroids, acyclovir, and surgery for Bell's palsy (an evidence-based review), Neurology, 2001, vol. 56 (pg. 830-6)

[12] Echevarria JM, Casas I, Martinez-Martin P. Infections of the nervous system caused by varicella-zoster virus: a review, Intervirology, 1997, vol. 40 (pg. 72-84)

[13] Moriuchi H, Rodriguez W. Role of varicella-zoster virus in stroke syndromes, Pediatr Infect Dis J, 2000, vol. 19 (pg. 648-53)