



INTERNATIONAL JOURNAL OF PHARMACEUTICAL RESEARCH AND BIO-SCIENCE

A REVIEW ON HERPES ZOSTER

AMAR PP¹, AJINKYA C², TOHID NB², ROHIDAS P², AVINASH C²

1. Assistant Professor, Department of Pharmacology, Sant Gajanan Maharaj College of Pharmacy, Mahagaon M. S. India.

2. Student at Sant Gajanan Maharaj College of Pharmacy, Mahagaon M. S. India.

Accepted Date: 17/05/2014; Published Date: 27/06/2014

Abstract: Herpes zoster (HZ), also known as shingles, is a painful vesicular rash resulting from reactivation of the virus that also causes chickenpox – Varicella zoster virus (VZV). Typically, the rash runs its course in a matter of 4-5 weeks. The pain, however, may persist months, even years, after the skin heals. This phenomenon is known as postherpetic neuralgia (PHN). Often described as an intense burning, itching sensation, this pain can be significant to the point of being debilitating, and as such can greatly affect quality of life. Although shingles is generally regarded as a self-limited condition, the fact it can take several weeks to resolve and has the potential for development of complications such as PHN presents a challenge to clinicians. Many treatment options are available, each offering variable levels of efficacy. Conventional therapies include prescription antivirals, analgesics, both oral and topical. Other considerations include use of over-the-counter anti-inflammatory agents, vaccines, herbal drug treatment. This article reviews herpes zoster and postherpetic neuralgia, and presents the most effective conventional treatment options currently available, as well as select botanical, drug, and other considerations that may be beneficial in the management of this condition.

Keywords: Herpes Zoster, Chicken Pox, Shingles

Corresponding Author: MR. AMAR P. PATIL



PAPER-QR CODE

Access Online On:

www.ijprbs.com

How to Cite This Article:

Amar PP, Ajinkya C, Tohid NB, Rohidas P, Avinash C; IJPRBS, 2014; Volume 3(3): 123-127

INTRODUCTION

Varicella zoster is the etiological agent of two diseases namely; varicella (chicken pox) and zoster (shingles). Herpes zoster is also called as shingles. It is a viral infection caused by a varicella-zoster virus (VZV). It occurs when reactivation of VZV after infection with chicken pox. After acute infection the virus goes into the dormant condition in sensory dorsal root ganglia.

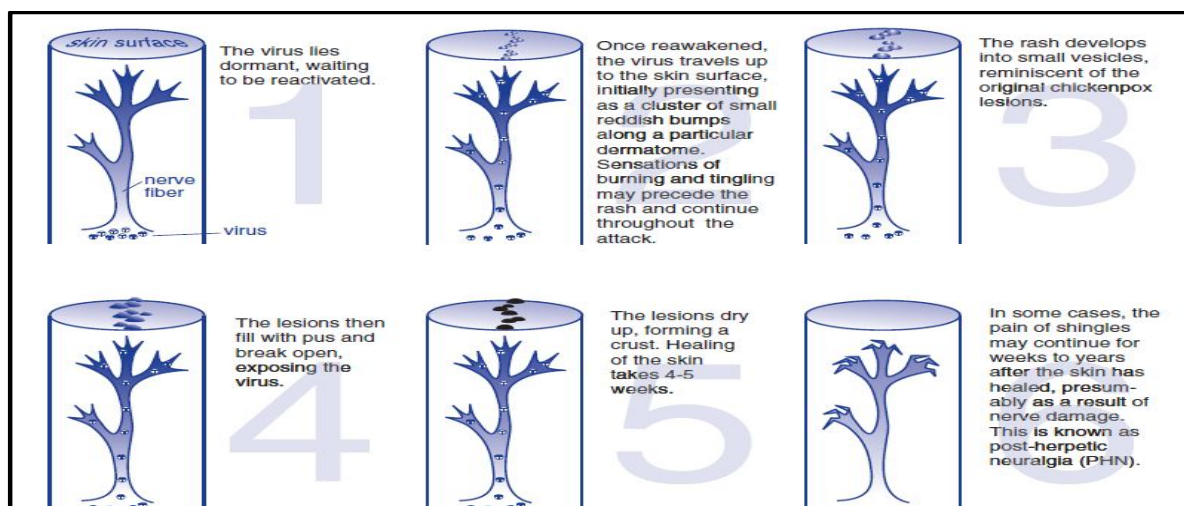
The cause for reactivation is unclear, but the decrease in cell mediated immunity with age, immunosuppressant treatment or infection such as HIV which causes reactivation of the virus. Herpes zoster infection arises rarely before the age of 50.

ETIOLOGY AND PATHOLOGY

It is mainly occur due to infection of varicella zoster virus. The virus enter by respiratory system multiplies in an unclear site, penetrates the reticuloendothelial system and passes into the blood.

The usual incubation period for varicella zoster is about 14-16 days. An individual can no longer transmit VZV infection after the skin lesions are destructed. Then the VZV enters in the sensory dorsal root ganglia where it can lie dormant for many years. Years or decades after the initial infection, the virus may break out of nerve cell bodies and travel down nerve axons to cause viral infection of the skin in the region of the nerve. The virus may spread from one or more ganglia along nerves of an affected segment and infect the corresponding dermatome (an area of skin supplied by one spinal nerve) causing a painful rash. Fig.1

Fig.1 Pathology and Progress of HZV



Revised from: www.fda.gov/fdac/features/2001/301_pox.html (accessed 5march 2014)

SIGNS AND SYMPTOMS

The typical demonstration of HZV starts with a prodrome of mild-to-moderate burning or itchy in or under the skin of a given dermatome, often supplemented by stomach upset, fever, chills, headache, and general malaise. In 48-72 hours after the prodrome, an erythematous, maculopapular rash forms singly along the dermatome and quickly develops into vesicular lesions evocative of the unusual chickenpox outbreak. The pain associated with shingles differs in intensity from mild to severe, such that even the smallest amount touch or draft can provoke excruciating spasms. The lesions usually initiate to dry and scab 4-5 days after appearing. Total period of the disease is generally between 7-10 days; yet, it may take several weeks for the skin to return to normal.

Development of the shingles rash

Day 1



Day 2



Day 5



Day 6



COMPLICATIONS

Post herpetic neuralgia is the most common serious complication of herpes zoster. It is a neuralgic pain that continues for more than 3 months after the healing of the rash. An additional possible complication of HZV includes encephalitis, myelitis, peripheral nerve palsies, and forms of contralateral hemiparesis.

INCIDENCE RATE

Throughout the world, the incidence rate of herpes zoster every year ranges from 1.2 to 3.4 cases per 1,000 healthy individuals, increasing to 3.9–11.8 per year per 1,000 individuals among those older than 65 years. Over a lifetime, a large fraction of people develop herpes zoster, though usually only once; a 1965 16-year British study proposed that, of those individuals living to age 85, 50% would likely have had at least one attack, and 1% had at least two attacks.

DIAGNOSIS OF SHINGLES

Shingles is generally diagnosed on the basis of a clinical evaluation, mainly once the rash seems. However, conditions such as HSV infection, eczema herpeticum, impetigo, contact dermatitis and others can be wrong for shingles. Laboratory authorization can be obtained by taking a sample from the base of the skin lesions and carrying out a nucleic acid detection test (PCR) or

direct-fluorescent antibody test (DFA). Other techniques, such as viral culture, are less sensitive and take longer to complete.

TREATMENT

Antiviral drug therapy

Antiviral drug treatment hastens the healing of skin lesions and lessens the duration of pain, probably by limiting the magnitude of damage done to the involved sensory nerves by the replicating of VZV and by restriction the duration of new lesion formation. Three oral antiviral drugs are approved in the U.S. for treatment of herpes zoster in immunocompetent patients: acyclovir, valacyclovir and famciclovir. Valacyclovir and famciclovir are now preferred in practice because they have a simplified dosing schedule and improved pharmacokinetic characteristics compared with acyclovir. Valacyclovir and famciclovir are therapeutically equivalent for treatment of herpes zoster in the normal host, and valacyclovir has been found to be more cost effective.

Narcotic analgesics

The pain associated with HZ covers a broad spectrum of intensity. Generally, individuals with mild-to-moderate pain find sufficient relief via over-the-counter topical or oral analgesics and anti-inflammatory agents, such as aspirin, acetaminophen, or ibuprofen.

Botanical drugs

Capsaicin, licorice, Madonna lily, reishi mushroom, honey, bi phaya Yaw, aloe etc.

Vaccine

A zoster vaccine, known as Zostavax® (CSL Biotherapies/Merck & Co. Inc.) was licensed in Australia in 2006 and has been available via private purchase since early 2008. Zostavax® is a live attenuated viral vaccine formulated from the same VZV vaccine strain (Oka-derived) as both currently licensed varicella (chickenpox) vaccines (Varivax® and Varilrix®) but is of a higher potency, containing, on average, at least 14 times more plaque forming units of vaccine virus per dose. This higher viral potency is required to yield a satisfactory boost in the immune response in older adults.

CONCLUSION

Many options are available to the clinician for the treatment of HZ and PHN, albeit with variable degrees of success. Antiviral agents, such as acyclovir, valacyclovir, and famciclovir, have been shown to reduce both the pain and healing time of skin lesions associated with HZ, but have

marginal success in preventing and treating PHN. Corticosteroids may be used for pain management in HZ, but do not seem to be effective in prevention of PHN. Analgesics provide effective temporary pain relief for both HZ and PHN. Nerve block injections offer more long-term pain relief in both conditions, provided they are administered early in the course of the disease.

REFERENCES

1. National Institute of Allergy and Infectious Disease, 2003. <http://content.nhiondemand.com/psv/HC2.asp?objID=100635&cType=hc> Accessed April 27, 2014
2. Gnann JW Jr, Whitley RJ. Clinical practice. Herpes zoster. *N Engl J Med* 2002; 347:340-346.
3. National Institute of Neurological Disorders and Stroke (NINDS), 1999. <http://content.nhiondemand.com/psv/HC2.asp?objID=100635&cType=hc> Accessed April 27, 2014
4. Hope-Simpson RE. The nature of herpes zoster: a long term study and a new hypothesis. *Proc R Soc Med* 1965; 58:9-20.
5. Katz J, Cooper EM, Walther RR, et al. Acute pain in herpes zoster and its impact on health-related quality of life. *Clin Infect Dis* 2004; 39:342-348.
6. Whitley RJ. Varicella-zoster virus infections. In: Fauci AS, Braunwald E, Isselbacher KJ, et al. eds. *Harrison's Principles of Internal Medicine*. 14th ed. New York, NY: McGraw Hill; 1998:1086-1089.
7. Berkow R, Fletcher AJ, eds. Chickenpox (varicella). *The Merck Manual*. 16th ed. Rahway, NJ: Merck Research Laboratories; 1992:2175.
8. Samuelson J, von Lichtenberg F. Infectious diseases. In: Cotran R, Kumar V, Robbins S, eds. *Robbins Pathologic Basis of Disease*. 5th ed. Philadelphia, PA: WB Saunders; 1994:349.
9. Schmader KE. Epidemiology and impact on quality of life of postherpetic neuralgia and painful diabetic neuropathy. *Clin J Pain* 2002; 18:350-354.
10. Gnann JW, Whitley RJ. Natural history and treatment of varicella-zoster in high-risk populations. *J Hosp Infect* 1991; 18:317-329.