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Overview on Shingles (Herpes Zoster)

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ABSTRACT

The Varicella-zoster virus (VZV) or human herpes virus 3 is a neurotropic human alpha herpes virus responsible for chickenpox/varicella and shingles/Herpes zoster (HZ).HZ represents a reactivation of VZV in the host and has gained interest because of variable clinical presentation, which is important in the differential diagnosis of diseases. Furthermore, HZ complications are potentially life-threatening. HZ reactivation has been reported as a possible adverse event after COVID-19 vaccination. HZ reactivation has recently been observed after COVID-19 vaccination. The disease shows different clinical stages. A person's immune system weakens as they age. The more the immune system weakens, the less likely it is to prevent the virus from reactivating. Hence, older people are more at risk from Shingles.

The varicella zoster virus is responsible for chickenpox (also known as herpes zoster). After a person has chickenpox, the virus stays in their body and becomes inactive. The virus can become active again years later and cause Shingles. Scientists aren't exactly sure what causes the virus to become active again. However, there may be multiple factors.

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INTRODUCTION

WHAT CAUSES SHINGLES ?

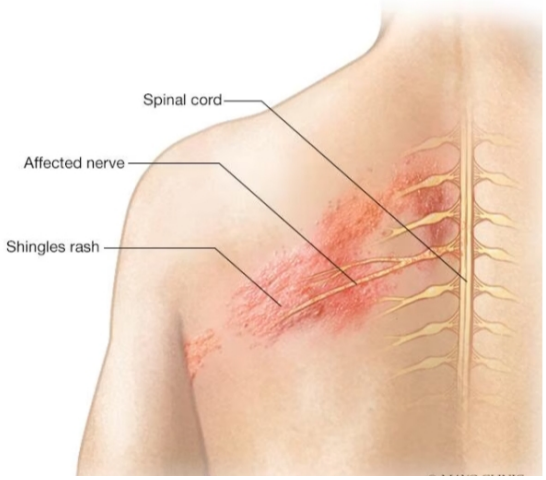
The varicella zoster virus is responsible for chickenpox (also known as herpes zoster). IT is a common condition characterised by nerve damage and the painful skin rash. The first exposure to the virus usually during childhood leads to chicken pox after the primary infection results moves to sensory ganglia of the spinal and cranial nerves where it becomes inactive against chicken pox suppresses viral replication keeping it dormant for decades reactivation of the virus occurs when the immunity weakens and fails to contain the virus, there may be multiple factors. A persons immune system weakens as they age. The more the immune system weakens, the less likely it is to prevent the virus from reactivating. Hence, older people are more at risk from Shingles .higher risk after age 50 and above anyone who has had chickenpox already has the virus that can cause Shingles. Some people have had chickenpox and don't remember it or might not have realised it. Either way, Sthey can develop Shingles if the virus reactivates, despite how healthy they may feel.

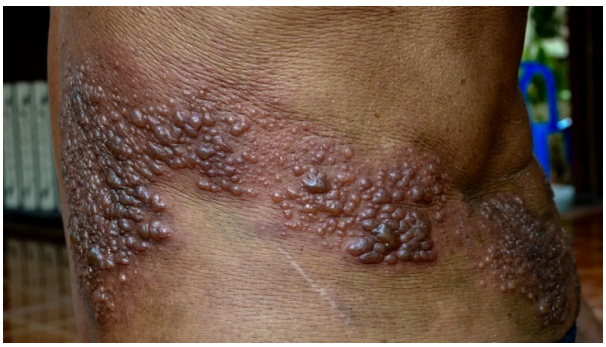
People with low immunity are at a higher risk of developing Shingles. And since the immune system naturally weakens over time with age, people are at a higher risk after age 50. The virus that causes Shingles is already present in the body from when you are infected with chickenpox. It goes dormant until reactivated. Hence, you cannot pass it on to another.

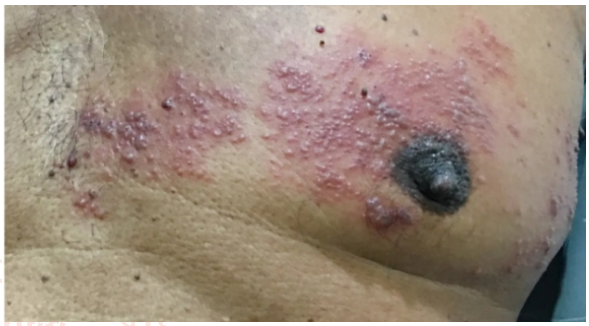
Chickenpox is a very contagious disease that causes a blister-like rash typically all over the body, itching, and fever. The chickenpox virus can reactivate, causing Shingles. People with Shingles may have pain, itching, tingling, and blisters in one area of the body that can last for weeks. Shingles typically produces a painful rash that often blisters, and scabs over in 10 to 15 days and clears up within 2 to 4 weeks. It usually appears on one side of the body or face. 48-72 hours before the rash appears, people may experience pain, itching, tingling, or numbness in the area where the rash will develop. Shingles Prevention Options

Shingles is caused by the reactivation of the virus that remains in the body after chickenpox. So, if a person hasn't had chickenpox, ask them to avoid contact with people who have chickenpox or Shingles. Also,

ensure that they follow all hand and cough hygiene to reduce the risk of developing chickenpox.







Most people get Shingles once in a lifetime, but it is even possible to get infected more than once.

ETIOLOGY

The VZV is a neurotropic human herpes virus belonging to the genus alpha herpesviridiae.

The VZV genome consists of about 125,000 base pairs of linear double-stranded DNA, and its nucleocapsid consists of 162 capsomers. The virus is highly cell-associated and only infects human cells, such as epithelial cells, T lymphocytes, and ganglionic neurons [1] Virus entry into neural cells is mediated by heparan sulfate proteoglycan and the glycogen synthase kinase 3 (GSK-3) pathway [2] Viral core glycoproteins B, H, and L participate in the core fusion complex. New virus particles can be released as soon as 9 to 12 h after cellular entry [3] The incubation period for varicella varies between 10 and 21 days. Varicella is contagious from 1 to 4 days before the cutaneous rash and until all vesicular cutaneous lesions have dried up [4]

Varicella infection in pregnancy can spread via placenta, leading to fetal infection. Fetal varicella infection leads to disseminated life-threatening diseases. Vaccination protects the fetus . [5]

While HZ is uncommon among children, the HZ risk could be diminished by 64% in children after vaccination for varicella, as shown in a Canadian study. Varicella vaccination in the youth does not seem to reduce HZ risk during [6]

Clinical Stages

Clinical symptoms appear in three stages—pre-eruptive, acute exudative, and chronic [7]. The pre-eruptive stage presents with burning or pain within the affected dermatome at least 2 days prior to cutaneous eruptions. Noncutaneous symptoms such as experiencing headaches, general malaise, and photophobia may also be present.[8]

In the acute eruptive phase, multiple umbilicated and painful vesicles develop. The vesicles often burst, ulcerate, and eventually dry out. This is the most contagious stage. Pain is often severe and unresponsive to nonsteroidal pain medications. The acute eruptive phase may last 2–4 weeks. Pain can continue longer.[8]

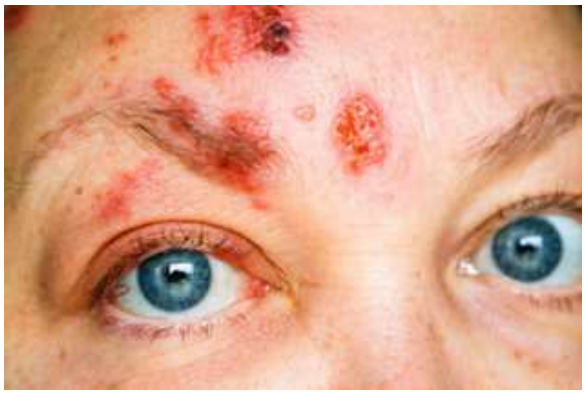
Chronic HZ infection is characterized by severe pain that lasts >4 weeks. Patients experience dysesthesias, paresthesias, and sometimes shock-like sensations. The pain is disabling and may last for several months.[8]

In most patients, diagnosis is made clinically. Due to variable clinical presentation and atypical cases, the diagnosis of HZ may be challenging in some patients [9]

chain reaction (PCR) is useful for confirmation of suspected HZ-type pain without a rash.

SPECIAL STAGES

- HZ Ophthalmicus (HZO
- HZO is an ophthalmologic emergency. There is a risk of vision loss. The main risk factors for HZO include age >50 years and immunosuppression[13, 18]
- Orbital phlegmon with a risk of secondary blindness [10]
- Acute retinal necrosis with possible visual loss[11]
- Anterior uveitis;
- Epithelial punctate keratitis[10]
- Oculomotoric nerve palsy [11]
- Superior orbital fissure syndrome [13]
- Orbital apex syndrome [14]
- Corneal inflammation and opacification with visual impairment



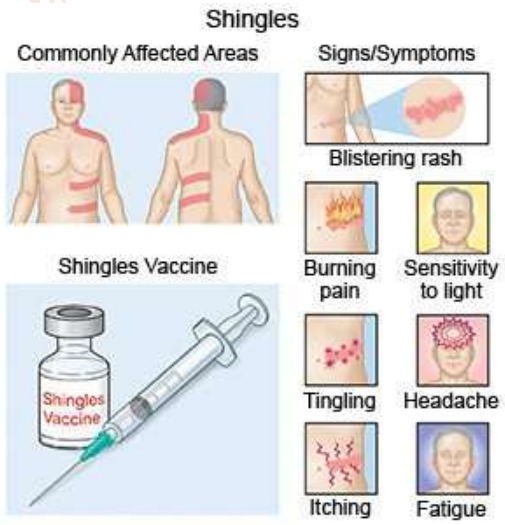
- HZ may affect the central nervous system (CNS). Immunocompromised patients, such as AIDS/ HIV patients, are prone for CNS manifestation. [17]
- Complications from shingles can include:

Postherpetic neuralgia. For some people, shingles pain continues long after the blisters have cleared. This condition is known as postherpetic neuralgia. It occurs when damaged nerve fibers send confused and exaggerated messages of pain from your skin to your brain.

Vision loss. Shingles in or around an eye (ophthalmic shingles) can cause painful eye infections that may result in vision loss.

Neurological problems. Shingles may cause inflammation of the brain (encephalitis), facial paralysis, or problems with hearing or balance.

Skin infections. If shingles blisters aren't properly treated, bacterial skin infections may develop.



Symptoms

Shingles symptoms usually affect only a small section on one side of your body. These symptoms may include[22]:

- Pain, burning or tingling
- Sensitivity to touch
- A red rash that begins a few days after the pain
- Fluid-filled blisters that break open and crust over
- Itching

Some people also experience:

- Fever
- Headache
- Sensitivity to light
- Fatigue
- Depending on the location of the pain, it can sometimes be mistaken for problems with the heart, lungs or kidneys. Some people experience shingles pain without ever developing the rash.[22]

Most commonly, the shingles rash develops as a stripe of blisters that wraps around either the left or right side of the torso. Sometimes the shingles rash occurs around one eye or on one side of the neck or face.

TREATMENT

VACCINATION-

HZ vaccines aim to prevent activation of HZ and the development of PHN. Currently, two HZ vaccines are available for healthy older adults, a live attenuated VZV vaccine (Zostavax; Merck, Kenilworth, NJ, USA) and a recombinant adjuvanted VZV glycoprotein E subunit vaccine (Shingrix, GlaxoSmithKline, London, UK). Live attenuated vaccine had been the standard vaccine for years. The safety and efficacy of both vaccines has been demonstrated in clinical trials in immunocompetent healthy adults, in selected immunocompromised patients, and in patients with immune disorders. Recombinant HZV vaccine is more effective for prevention of HZ

compared to live attenuated HZV vaccine. Recombinant HZV vaccine is nonreplicating and is therefore safe also for immunocompromised persons.[20, 21]

Table 1 Medical treatment of HZ

Drug	Dosage	Remark	METHOD
ACYCLOVIR	ADULT: 5X 800 Mg/day P. O	Limited bioavailability	Oral
	3 X500 Mg /day	In uncomplicated HZ	
	3- 5 x 10 mg/kg/day	In severe HZ, in case of immunosuppression	
		For 10 day, usually 5- 7 days	
	Children : 3x 10 mg/kg/day	Maximum daily dosage 2.5 g	
BRIVUDIN	Adults : 125 mg once a day p.o	For 5 days	Oral
VALACICLOVIR	Adults : 3 x 1000 mg / day p.o	For 7 days	Oral
FAMICLOVIR	Adults : 3 x 250 – 500mg/ day	2nd line in ACV- resistant patients	Oral

The standard therapy of HZ is acyclovir (ACV) and its prodrug valacyclovir or brivudine (Table 1). Oral valacyclovir offers the advantage of a three- to fivefold increase in acyclovir bioavailability [19]. ACV and valacyclovir are processed to nucleoside analogues, which specifically block viral DNA replication in affected cells. Mutations in the viral thymidine kinase and/or DNA polymerase are responsible for ACV resistance [18].

TABLE 2

DRUG	PURPOSE	DRUG FREQUENCY	METHOD
Anti inflammatory drugs including lbuprofen	To ease pain swelling	Every 6 to 8 hours	Oral
Narcotic medications or pain relievers	To reduce pain	Likely to be prescribed once or twice daily	Oral
Anti convulsants or tricyclic antidepressants	To treat prolonged pain	Once or twice daily	Oral
Antihistamine, such as diphenhydramine	To treat itching	Every 8 hours	Oral
Capsaicin[zostrix]	To help to reduce the risk of a nerve pain called postherpetic neuralgia, which occurs after recovery from shingles	Applied as needed	Topical

SHINGLES HOME REMEDIES

- Taking cool bath or showers to clean and soothe your skin
- Applying wet cold compresses to the rash to reduce pain and itching
- Applying calamine lotion or making a paste with water and baking soda or cornstarch to reduce itching
- Eating foods with vitamins B12, vitamin C, and vitamin E
- Taking L – lysine supplements to strengthen your immune system

CONCLUSION

Varicella zoster infection (VZV) disease can essentially affect wellbeing related QOL, particularly in older people. As well as being intensely agonizing, herpes zoster might cause serious ongoing intricacies, including PHN, cerebral arteritis, and herpes zoster ophthalmicus . Current therapy of the intense and constant side effects of herpes zoster incorporates the utilization of antiviral specialists and pain relieving drugs, however no single prescription or mix of meds can forestall or totally ease zoster side effects.

Given the restrictions of existing zoster treatments, the avoidance of VZV contamination has acquired abrogating significance. VZV immunizations can diminish both the wellbeing and monetary impacts of herpes zoster. Albeit generally expensive, immunizations lessen the gamble of disease as well as save wellbeing related QOL in the geriatric populace.

RFERENCES

[1] Gershon A.A., Breuer J., Cohen J.I., Cohrs R.J., Gershon M.D., Gilden D., Grose C., Hambleton S., Kennedy P.G.E., Oxman M.N., et al.

Varicella zoster virus infection. Nat. Rev. Dis. Prim. 2015;1:15016. doi: 10.1038/nrdp.2015.16. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[2] Li Puma D.D., Marcocci M.E., Lazzarino G., De Chiara G., Tavazzi B., Palamara A.T., Piacentini R., Grassi C. Ca2+-dependent release of ATP from astrocytes affects herpes simplex virus type 1 infection of neurons. Glia. 2021;69:201–215. doi: 10.1002/glia.23895. [PubMed] [CrossRef] [Google Scholar]

[3] Zerboni L., Sen N., Oliver S.L., Arvin A.M. Molecular mechanisms of varicella zoster virus pathogenesis. Nat. Rev. Genet. 2014;12:197–210. doi: 10.1038/nrmicro3215. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[4] Marin M., Leung J., Lopez A.S., Shepersky L., Schmid D.S., Gershon A.A. Communicability of varicella before rash onset: A literature review. Epidemiol. Infect. 2021;149:1–18. doi: 10.1017/S0950268821001102. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[5] Kett J.C. Perinatal Varicella. Pediatr. Rev. 2013;34:49–51. doi: 10.1542/pir.34.1.49. [PubMed] [CrossRef] [Google Scholar]

[6] Rafferty E., Reifferscheid L., Russell M.L., Booth S., Svenson L.W., MacDonald S.E. The impact of varicella vaccination on paediatric Herpes zoster epidemiology: A Canadian population-based retrospective cohort study. Eur. J. Clin. Microbiol. Infect. Dis. 2021;40:2363–2370. doi: 10.1007/s10096-021-04298-z. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[7] Rajbhandari L., Shukla P., Jagdish B., Mandalla A., Li Q., Ali M.A., Lee H., Lee G., Sadaoka T., Cohen J.I., et al. Nectin-1 Is an Entry Mediator for Varicella-Zoster Virus Infection of Human Neurons. J. Virol. 2021;95:e01227-21. doi: 10.1128/JVI.01227-21. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[8] Nair P.A., Patel B.C. StatPearls [Internet] StatPearls Publishing; Treasure Island, FL, USA: 2021. Herpes zoster [Google Scholar]

[9] Wollina U. Variations in Herpes zoster manifestation. Indian J. Med. Res. 2017;145:294–298. doi: 10.4103/ijmr.IJMR_1622_16. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[10] Niederer R.L., Meyer J.J., Liu K., Danesh-Meyer H.V. Herpes zoster Ophthalmicus Clinical Presentation and Risk Factors for Loss of Vision. Am. J. Ophthalmol. 2021;226:83–89. doi: 10.1016/j.ajo.2021.02.002. [PubMed] [CrossRef] [Google Scholar]

[11] LoBue S.A., Palazzolo L., Antonova N., Bivona M.R., Smith E., Edelstein M. Sterile iris abscess associated with Herpes zoster ophthalmicus. Am. J. Ophthalmol. Case Rep. 2021;23:101144. doi: 10.1016/j.ajoc.2021.101144. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[12] Lee T., Carnahan M.C., Sazegar P. Herpes zoster Ophthalmicus Associated with Oculomotor Nerve Palsy. Am. J. Med. 2021 doi: 10.1016/j.amjmed.2021.08.029. epub ahead of print. [PubMed] [CrossRef] [Google Scholar]

[13] Liesegang T.J. Herpes zoster Ophthalmicus: Natural History, Risk Factors, Clinical Presentation, and Morbidity. Ophthalmology. 2008;115:S3–S12. doi: 10.1016/j.ophtha.2007.10.009. [PubMed] [CrossRef] [Google Scholar]

[14] Ruiz-Arranz C., Reche-Sainz J.A., de Uña-Iglesias M.C., Ortueta-Olartecoechea A., Muñoz-Gallego A., Ferro-Osuna M. Orbital apex syndrome secondary to Herpes zoster ophthalmicus. Am. J. Ophthalmol. 2021;96:384–387. doi: 10.1016/j.oftale.2020.06.009. [PubMed] [CrossRef] [Google Scholar]

[15] Blanco-Palmero V.A., Ortueta-Olartecoechea A., de Urabayen D.U.-P., Sánchez-Tornero M., Méndez-Guerrero A., Matarazzo M. Isolated Nonreactive Mydriasis in Herpes zoster Ophthalmicus. J. Neuro-Ophthalmol. 2021 doi: 10.1097/WNO.0000000000001289. epub ahead of print. [PubMed] [CrossRef] [Google Schola

[16] Davis A.R., Sheppard J. Herpes zoster Ophthalmicus Review and Prevention. Eye Contact Lens Sci. Clin. Pract. 2019;45:286–291. doi: 10.1097/ICL.0000000000000591. [PubMed] [CrossRef] [Google Scholar]

[17] Arnett N., Pavlou A., Burke M.P., Cucchiara B.L., Rhee R.L., Song J.W. Vessel wall MR imaging of central nervous system vasculitis: A systematic review. Neuroradiology. 2021;64:43–58. doi: 10.1007/s00234-021-02724-9. [PubMed] [CrossRef] [Google Scholar]

[18] Brunnemann A.-K., Bohn-Wippert K., Zell R., Henke A., Walther M., Braum O., Maschkowitz G., Fickenscher H., Sauerbrei A., Krumbholz A. Drug Resistance of Clinical Varicella-Zoster Virus Strains Confirmed by Recombinant Thymidine Kinase Expression and by Targeted Resistance Mutagenesis of a Cloned Wild-Type Isolate. Antimicrob. Agents Chemother. 2015;59:2726–2734. doi: 10.1128/AAC.05115-14. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[19] Beutner K.R., Friedman D.J., Forszpaniak C., Andersen P.L., Wood M.J. Valaciclovir compared with acyclovir for improved therapy for Herpes zoster in immunocompetent adults. Antimicrob. Agents Chemother. 1995;39:1546–1553. doi: 10.1128/AAC.39.7.1546. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[20] Beutner K.R., Friedman D.J., Forszpaniak C., Andersen P.L., Wood M.J. Valaciclovir compared with acyclovir for improved therapy for Herpes zoster in immunocompetent adults. Antimicrob. Agents Chemother. 1995;39:1546–1553. doi: 10.1128/AAC.39.7.1546. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[21] Gupta S., Arasaratnam R.J., Solow E.B., Bajaj P. A Medical Records Review Study Assessing Safety of Zoster Vaccine Recombinant, Adjuvanted in Patients with Rheumatic Disease. JCR J. Clin. Rheumatol. 2021 doi: 10.1097/RHU.0000000000001790. epub ahead of print. [PubMed] [CrossRef] [Google Scholar]

[22] https://www.mayoclinic.org/diseases-conditions/shingles/symptoms-causes/syc-20353054#overview