World Journal of Pharmaceutical and Life Sciences

www.wjpls.org

Impact Factor: 7.409 Coden USA: WJPLA7

HERPES ZOSTER: A COMPREHENSIVE REVIEW

***MS. Shraddha Dipak Jamdade, MS. Chaitali Pundlik Gadade, MRS P. A. Mane-Patil**

Eklavya College of Pharmacy, Tasgaon Sangli Maharashtra, India 416312.

***Corresponding Author: MS. Shraddha Dipak Jamdade**

Eklavya College of Pharmacy, Tasgaon Sangli Maharashtra, India 416312.

https://doi.org/10.5281/zenodo.17480509,

**How to cite this Article: ***MS. Shraddha Dipak Jamdade, MS. Chaitali Pundlik Gadade, MRS P. A. Mane-Patil. (2025). HERPES ZOSTER: A COMPREHENSIVE REVIEW. World Journal of Pharmaceutical and Life Science, 11(11), 23–29.

This work is licensed under Creative Commons Attribution 4.0 International license.

Article Received on 26/09/2025 Article Revised on 16/10/2025 Article Published on 01/11/2025

ABSTRACT

The varicella-zoster virus, which causes chickenpox, reactivates to create herpes zoster, sometimes referred to as shingles by some people. It mostly affects people who are elderly or have compromised immune systems. The disease's hallmark is a painful, blistering skin rash that is frequently restricted to a single dermatome. The pathophysiology, clinical manifestation, diagnosis, management, and epidemiology of herpes zoster are the main topics of this review. The article also covers side effects such post-herpetic neuralgia and the role of immunisation in preventing the illness. Current approaches to controlling and preventing shingles are thoroughly reviewed, including recent developments in immune response modulation and antiviral treatments. The following aspects of HZ, including aetiology, epidemiology, pathophysiology, clinical stages, complications, diagnosis, treatment, prevention, and conclusion, are highlighted in this review. The review provides general details concerning shingles. The varicella zoster virus is the cause of the viral infection that causes the disease.

KEYWORDS - Herpes zoster, varicella- zoster virus, shingles, post herpetic neuralgia, epidemiology, treatment.

INTRODUCTION

The well-known viral illness known as herpes zoster (HZ) typically manifests as a painful unilateral vesicular rash that is limited to a sensory nerve's distribution. The reactivation of the DNA virus varicella zoster, which causes chickenpox, results in HZ, sometimes referred to as shingles, an acute viral illness. It usually shows up as a vesicular rash, which is unpleasant and takes four to five weeks to develop. After the skin sores have healed, the pain could last for months or even years. Postherpetic neuralgia (PHN) is the name given to this phenomenon. About 10–18% of zoster patients are at risk for PHN. Hospitalization rates for zoster patients are close to 3%. Patients with weakened immune systems frequently experience zoster-related morbidity. Any one of the three trigeminal nerve branches may be impacted by HZ. Just 1.7% of all HZ cases involve the mandibular and maxillary branches without ocular branch involvement, which is a rather uncommon occurrence.[1]

To find pertinent published articles on HZ, the literature was searched using PubMed and Google Scholar. Review articles, systematic reviews, meta-analyses, clinical trials, clinical investigations, case series, and case reports were all taken into consideration. The articles were searched using the following keywords: "Varicella-zoster virus", “Herpes zoster", "treatment AND Herpes zoster", and "prevention AND Herpes zoster".[2]

EPIDEMIOLOGY OF HERPES ZOSTER

There are no seasonal fluctuations in the occurrence of HZ, which occurs globally. Age-dependent, the incidence of HZ varies from 1.2 to 3.4 per 1000 persons annually in younger adults to 3.9 to 11.8 per 1000 persons annually in older patients (i.e., those over 65). A systematic evaluation of research conducted between 2002 and 2018 found that the cumulative incidence, with a female predominance, has been estimated to be between 2.9 and 19.5 instances per 1000 population. Age over 50, immune suppression, infections, and mental stress are common risk factors

for HZ. Patients with diabetes mellitus are also at higher risk, according to a meta-analysis of 16 studies published up until January 2021 (pooled relative risk: 1.38; 95% CI: 1.21–1.57). Megaloblastic anemia and pediatric HZ are significantly correlated, according to a recent Indian study published in 2021. Megaloblastic anemia is primarily caused by folic acid or vitamin B12 deficiency. For patients over 65, the mortality rate from HZ varies from 0.0022 to 82.21 per 100,000 population, according to the Global Burden of Disease database. Based on German HZ outpatient incidence data from 2007 and 2008, the yearly death rate of HZ has been calculated to be 0.29 for women and 0.10 for men per 100,000 patient years.[3] Possible heterogeneity in epidemiological data resulting from variations in reporting should be noted. Countries without effective and efficient reporting systems might not have as many people as those with such systems.[4]

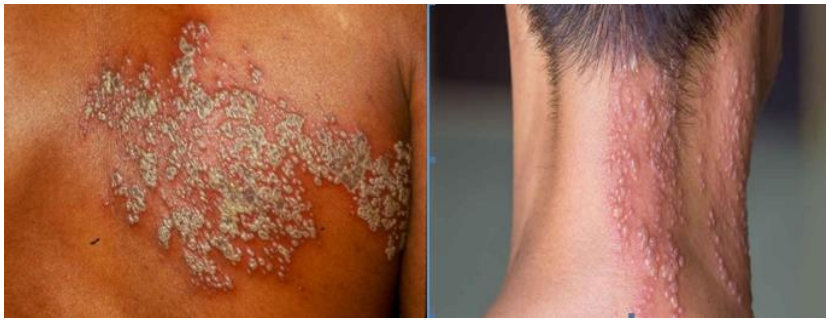


Fig. Representative image of herpes zoster.

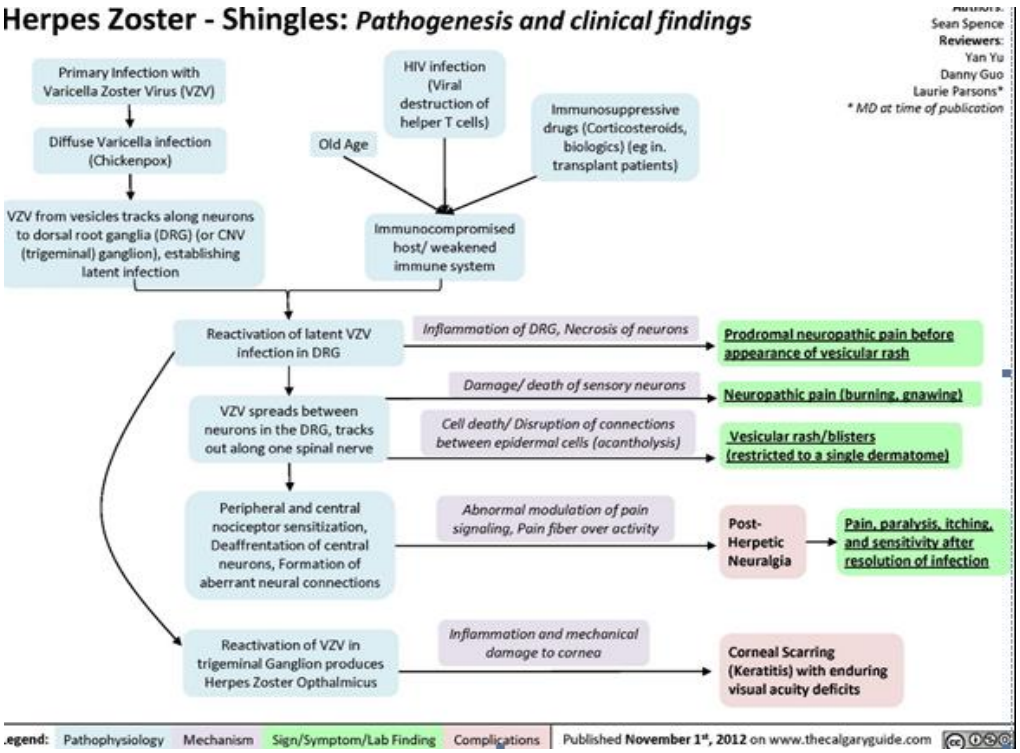
PATHOPHYSIOLOGY

Varicella-zoster virus-specific T-cell proliferation occurs in cutaneous lesions of herpes zoster, whereas interferon Alfa production results in the remission of herpes zoster. Immunocompetent patients have long-lasting, improved cell-mediated immunity to the varicella-zoster virus because certain antibodies (IgG, IgM, and IgA) develop more quickly and reach greater titters during reactivation (herpes zoster) than during the initial infection.[5]

The dermatological involvement follows a dermatome and is centripetal. While motor involvement is uncommon, the lumbar and cervical roots are typically affected. Although the rates of transmission are modest, the virus can spread to those who have never had varicella-zoster. Either direct skin contact or breathing contaminated droplets can spread the virus.

It's crucial to remember that herpes infections can potentially happen simultaneously.

Patients with shingles have been discovered to have human herpes viruses, CMV, EBV, and herpes simplex.[6]



RANDOMIZED CLINICAL STAGES OF HERPES ZOSTER

There are three phases of clinical symptoms: pre-eruptive, acute exudative, and chronic.[7] Least two days before cutaneous eruptions, the pre-eruptive stage is characterized by burning or pain inside the afflicted dermatome. There may also be non-cutaneous symptoms like headaches, a general feeling of unwellness, and photophobia.

Many painful, umbilicated vesicles form during the acute eruptive phase. Frequently, the vesicles rupture, develop ulcers, and finally dry out. The most contagious stage is this one. Nonsteroidal painkillers often don't work for severe pain. Two to four weeks may pass during the acute eruptive phase. Longer periods of pain are possible.[8]

The hallmark of a chronic HZ infection is excruciating discomfort that lasts for more than four weeks. Dysesthesias, paraesthesia, and even shock-like sensations are experienced by patients. The incapacitating discomfort can linger for months.[8]

The majority of patients have a clinical diagnosis. In certain patients, the diagnosis of HZ may be difficult because of unusual cases and varied clinical presentation.[9]

1. Pre eruptive phase - The pre eruptive phase, often referred to as the pre herpetic neuralgia stage, typically lasts 48 hours, though it occasionally lasts up to 10 days. Sensory phenomena along one or more dermatomes—an area of skin mostly supplied by a single spinal nerve are its defining characteristics. This stage is characterized by fever, light sensitivity, headache, and general exhaustion.

2. Acute phase - The physical symptoms that started during the pre-eruptive phase continue during the acute eruptive phase, along with the development of lesions and excruciating pain. The lesions begin as macules, which are tiny, flat, confined changes in skin pigment, and swiftly develop into clusters of fluid-filled vesicles. Over the course of three to five days, new vesicles keep forming and rupturing. The virus is most easily spread to other people during this stage. It may take up to four weeks for the vesicles to recover after they finally dry out and crust over. The lesions may result in persistent skin scarring and changes in skin pigmentation.

3. Chronic phase - Up to 20% of shingles patients get postherpetic neuralgia, another name for the chronic phase. Recurrent pain that persists for more than four weeks after the vesicles have healed is what characterizes this phase. Unusual skin sensations including tingling, burning, and numbness brought on by nerve damage (dysesthesia) or pressure on a nerve (paraesthesia) are additional symptoms. Months or even years may pass before the agonizing and incapacitating pain subsides. The majority of individuals need relief from the pain and discomfort that come with having shingles, even though the rash is typically self-limited and goes away without medical help.[10]

Patients with severe symptoms, immunosuppression, disseminated herpes zoster, ocular involvement, and other severe consequences are the only ones admitted to the hospital. Managing pain with postherpetic neuralgia is infamously challenging. The topical analgesics in lidocaine and capsaicin transdermal patches are two choices.99% of people 50 years of age and older in the US have the varicella zoster virus in their body as a result of having chickenpox as children. Every year, almost 1 million of these people will get shingles.[11]

PREVENTION

Among immune competent people aged 60 and over, Zostavax, a live attenuated varicella zoster virus-based zoster vaccine, has been demonstrated to lower the prevalence of herpes zoster and post-herpetic neuralgia globally. The vaccine protects or lessens the severity of herpes zoster infection by increasing the varicella zoster virus-specific cell-mediated immunity, which in turn controls the latent varicella zoster virus's reactivation or replication. [12] The Oka varicella zoster virus strain is the source of both the varicella and herpes vaccines. But compared to varicella vaccine, the herpes vaccine has a 14-fold higher vaccine viral. Uncompromised patients, children, and pregnant women are not advised to get this live attenuated vaccination.[13]

The herpes vaccination can be administered to people who have already had herpes zoster in order to stop new outbreaks. It is not recommended to provide the herpes vaccine to individuals who are using biologic medications like etanercept, infliximab, or adalimumab. However, the CDC ACIP permits the herpes vaccine to be administered to these patients either one month following the cessation of immunosuppressive therapy or fourteen days before to the start of immunosuppressive therapy.[14]

If the patient is receiving antiviral therapy, the vaccine should not be given since the antiviral medication may stop the vaccine virus from replicating, which could result in vaccine failure. As a result, individuals receiving long-term antiviral treatment must cease taking their medications at least 24 hours before becoming vaccinated and refrain from doing so for 14 days following the immunization.

DIAGNOSIS

As soon as the rash emerges, a clinical diagnosis of herpes zoster can be made. An electron microscope (EM) and Tzanck smear can identify whether a herpes virus is present in the vesicle. It is unable to differentiate between varicella zoster virus and herpes simplex virus, albeit.[15] Laboratory diagnostic methods for atypical herpes zoster include viral culture, skin biopsy,

polymerase chain reaction (PCR), and direct immunofluorescence assay (DFA).[16]

The most sensitive and specific diagnostic method for herpes zoster is PCR, which can identify varicella zoster virus DNA in vesicular fluid. Cerebrospinal fluid (CSF), blood, plasma, bronco alveolar lavage, and lesion fluid can all be subjected to PCR.[17] It is possible to utilise DFA instead of PCR. It is favoured over viral culture because of its quick turnaround, low cost, and great sensitivity.[18]

It is not possible to isolate viruses from blood or CSF fluid in patients with herpes zoster myelitis. Therefore, the only way to diagnose herpes zoster myelitis is to use magnetic resonance imaging (MRI) of the spine and the clinical emergence of a rash on the specific dermatome with clinical symptoms of transverse myelitis. [19] When segmental zoster paresis manifests as a painful dermatomal rash accompanied by muscle weakness, the diagnosis can be verified. Acute denervation of the compromised area can be seen on an electromyography.[20]

TREATMENT

Reducing pain, promoting rapid healing, and preventing complications are the primary goals of herpes zoster treatment. Antiviral medication lowers the likelihood of post-herpetic neuralgia and is used to treat herpes zoster as soon as a diagnosis is made. Corticosteroids can aid in the management of eruptions and discomfort. Isolating the patient and treating skin lesions locally are additional therapeutic components. To avoid nosocomial infection, the patient must be isolated.[21]

1. Antiviral agent – Acyclovir, famciclovir, and valacyclovir are antiviral medications used to treat acute herpes zoster. These substances aid in pain management, encourage rapid recovery, and guard against post-herpetic neuralgia. Antiviral therapy should begin within 72 hours of the rash appearing. [22] According to a Japanese study by Ono et al., famciclovir is more effective than Val acyclovir for reducing acute herpes zoster pain. During a seven-day course of famcyclovir medication, they saw an earlier decrease in discomfort within three to four days.[23]

Compared to famciclovir, oral acyclovir and Val acyclovir do not increase the incidence of acute renal injury, according to a retrospective study by Lam et al. Acute kidney damage is linked to intravenous (IV) acyclovir usage. The medication causes blockage and cellular.

Necrosis in the tubules by precipitating and crystallising.[24] Therefore, patients with renal illness should not get IV acyclovir. A brief course of acyclovir treatment (800 mg five times a day for four days) shown comparable effectiveness for individuals whose rash lasted more than 72 hours and for those whose rash lasted less than 72 hours.

2. Systemic corticosteroids - In certain cases, such as acute zoster pain, Ramsay Hunt syndrome, and ocular sequel, corticosteroid therapy is advised. Combining corticosteroid therapy with an antiviral medication increases its effectiveness. In the treatment of herpes zoster oticus/Ramsay Hunt syndrome, early acyclovir + steroid administration has demonstrated good improvement in both adults and children.[25] Early therapy also improves hearing recovery.[26] Patients with an early age beginning of Ramsay Hunt syndrome and those who receive combined antiviral and steroid treatment within 72 hours of the rash development have a fair prognosis.[27]

Numerous studies have used varying dosages, administration methods, and treatment durations.[28] Patients over 50 who receive acyclovir and prednisolone together for herpes zoster have demonstrated an improvement in quality of life.[29] It is unknown if acyclovir + prednisolone will have a long-term impact on preventing post-herpetic neuralgia, but it can clear up rash and lessen acute herpes zoster sickness.[30] Prednisone and ACTH were found to be ineffective in preventing post-herpetic neuralgia in a research comparing the two medications.[31]

3. Treatment of Herpes Zoster in Pregnancy – Pregnancy-related herpes zoster can be treated with acyclovir or Val acyclovir.[32] Since there is no elevated risk of birth defects or premature delivery, acyclovir is regarded as the drug of choice (DOC) in the early stages of pregnancy.[33] After receiving treatment for herpes zoster neuralgia with acyclovir and paracetamol for 28 weeks, the mother responded favourably to the medication and gave birth to a healthy baby two months later.[34] Val acyclovir showed good results in treating herpes zoster in a 17-week pregnant woman.[35]

According to the Centres for Disease Control and Prevention Advisory Committee on Immunisation Practices' (CDC ACIP) recommendations for preventing varicella, varicella zoster immune globulin is strongly recommended for immunocompromised children for passive immunisation following significant exposure to the varicella zoster virus or herpes zoster virus, as well as for susceptible pregnant women exposed to varicella to prevent varicella-related complications during pregnancy.Regardless of the mother's history of administering varicella zoster immune globulin, it is recommended that new-borns whose mothers have varicella infection or herpes zoster within five days prior to and two days following birth receive the vaccine. Since the kid is already protected from varicella by Trans placental acquired maternal antibodies, healthy neonates whose mothers had varicella for more than five days before delivery do not require varicella zoster immune globulin.[36]

4. Treatment of Herpes Zoster in Children –In children, herpes zoster is uncommon and mostly benign. Even after three weeks of illness, if the youngster still gets new lesions, there may be an underlying immunodeficiency[37] Acyclovir can be administered orally to children. It is not licensed for usage in preadolescent children, hence it is not frequently utilised for their treatment. Acyclovir treatment is advised for preadolescent children who have cancer, immunological compromise, or ocular involvement.[38]

Otherwise, this age group does not receive any special care.[39] Acyclovir was administered orally (in three cases) and intravenously (in one case) to four infants with infantile herpes zoster, ages 4 to 11 months, who recovered fully and without any aftereffects. Each of the four babies had previously been exposed to the varicella zoster virus.[40]

COMPLIATIONS

1. Post herpetic Neuralgia – The most prevalent complication of HZ is PHN. Published estimates suggest that a significant minority of patients—ranging from 5% to 30%—may also develop this condition. PHN is characterized by severe, stabbing, and burning pain along the nerve pathway, which can last for months after the first episode of rash. Other possible complications include stroke, meningitis, myelitis, secondary bacterial infections, and scarring. Over 30% of individuals suffering from PHN endure persistent pain for more than one year.[41]

2. Secondary Infection - Patients with immunodeficiency and those with normal immunological function can potentially develop secondary infections by streptococcal or staphylococcal bacteria, which can slow healing and leave scars. In patients with compromised immune systems, superficial gangrene may also develop.[42]

3. Vaculities and Stroke –Within a few weeks to several months (on average 7-8 weeks) from the initial lesion, a VZV infection in the cranial nerve can spread to the brain's blood vessels, causing vasculitis, stroke, and contralateral hemiplegia. This usually happens after HZO, but it can also happen when herpes zoster infects other cranial nerves. In addition to Myocardial infarction, aortic aneurysm, and temporal arteritis, vasculitis /angiitis can also indirectly result in subarachnoid and intra-parenchymal haemorrhage.[43]

4. Encephalitis – An infection in the central nervous system can result from an infection of the proximal region of the ganglion, as was previously discussed in the pathophysiology section of herpes zoster. A rare but potential side effect of a herpes virus is encephalitis, which can particularly affect the cranial nerves. Depending on the area of the brain impacted, symptoms can vary, but generally speaking, people who are affected will have headaches, nausea, fever, sensory abnormalities, and seizures. Cerebellar ataxia is another frequently observed symptom.[44]

5. Ramsay Hunt Syndrome –Ramsay Hunt Syndrome (RHS), a consequence of VZV infection in the ENT area, is typified by peripheral facial palsy and the development of herpes rashes on the scalp, outer ear canal, and oropharyngeal mucosa. The vestibulocochlear nerve, which produces tinnitus, vertigo, and unilateral hearing loss, is one of the cranial nerves that is frequently affected by RHS, along with the geniculate ganglion. Additionally, 50% of patients lose their ability to taste in the front two thirds of their tongue.[45]

6. Myelitis - The spread of a herpes zoster infection to the proximal portion of the spinal ganglia, particularly in the thoracic section, might result in myelitis. The symptoms, which often manifest two to three weeks following the onset of herpes lesions, include par paresis, diminished sensory function, sphincter problems, and Brown-Sequard syndrome.[46]

CONCLUSION

VZV has reactivated in the host, as indicated by HZ. Patients with HZ infections may experience a variety of symptoms and repercussions. Examples of distinct clinical manifestations include Ramsay Hunt syndrome, deep HZ, purpuric HZ, disseminated HZ, HZ opthalmicus, and central nervous system HZ. It can lead to complications including postherpetic neuralgia. Recurrent HZ may occur in elderly patients and those with compromised immune systems.

Patients with haematological and solid malignancies are more likely to get HZ. Patients with solid cancer who have received chemotherapy are more vulnerable to HZ than those who have not. The main treatments for HZ are acyclovir (ACV) and its prodrug valacyclovir or brivudine. HZ immunizations aim to prevent PHN production and HZ activation. There are now two HZ vaccinations available for healthy older adults.

REFERENCE

1. Chhimwal P, Shrivastava H, Bhavsar R. Herpes zoster: A case report. J Dent Orofac Res., 2015; 11(1): 31-34.
2. Patil A, Goldust M, Wollina U. Herpes zoster: A Review of Clinical Manifestations and Management. Viruses. 2022 Jan 19; 14(2): 192. doi: 10.3390/v14020192. PMID: 35215786; PMCID: PMC8876683.
3. Koshy E, Mengting L, Kumar H, Jianbo W. Epidemiology, treatment and prevention of herpes zoster: a comprehensive review. Indian Journal of Dermatology, Venereology and Leprology, 2018 May 1; 84: 251.
4. Van Oorschot D., Vroling H., Bunge E., Diaz-Decaro J., Curran D., Yawn B. A systematiciterature review of Herpes zoster incidence worldwide. Hum. Vaccines Immunother. 2021; 17: 1714–1732. doi:

10.1080/21645515.2020.1847582. [DOI] [PMC free article] [PubMed] [Google Scholar]
5. Baumrin E, Van Voorhees A, Garg A, Feldman SR, Merola JF. A systematic review of herpes zoster incidence and consensus recommendations on vaccination in adult patients on systemic therapy for psoriasis or psoriatic arthritis: From the Medical Board of the National Psoriasis Foundation. J Am Acad Dermatol, 2019 Jul; 81(1): 102-110. [PubMed]
6. Miles LW, Williams N, Luthy KE, Eden L. Adult Vaccination Rates in the Mentally Ill Population: An Outpatient Improvement Project. J Am Psychiatr Nurses Assoc, 2020 Mar/Apr; 26(2): 172-180. [PubMed]
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] [Cross Ref] [Google Scholar]
8. Nair P.A., Patel B.C. StatPearls [Internet] Nair P.A., Patel B.C. Stat Pearls [Internet] Stat Pearls 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. [DOI] [PMC free article] [PubMed] [Google Scholar].
10. Khalil N, Walton J, Roberts N, Hussein K. Viral infections in atopic dermatitis. Clinical and Experimental Dermatology, 2025 Jan; 50(1): 46-55.
11. Hung CT, Wang LM, Hung YC. Trends and characteristics of herpes zoster vaccination among older adults with asthma in the United States, 2008–2023: Findings from self-reported national surveys. Vaccine, 2025 Jan 1; 43: 126523.
12. Diez-Domingo J, Wienke T, de Lomas JG, Meyer CU, Bertrand I, Eymin C, Thomas S, Sadorge C. Comparison of intramuscular and subcutaneous administration of a herpes zoster live-attenuated vaccine in adults aged≥ 50 years: a randomised non-inferiority clinical trial. Vaccine, 2015 Feb 4; 33(6): 789-95.
13. Boivin G, Juvey R, Elliott CT, Patrick DM. Management and prevention of herpes zoster: A Canadian perspective. Canadian Journal of Infectious Diseases and MedicalMicrobiology, 2010; 21(1): 45-52.
14. Herpes R, Ortega-Sanchez IR, Seward JF. Prevention of herpes zoster: recommendations of the Advisory Committee on Immunization Practices (ACIP). Morbidity and Mortality Weekly Report: Recommendations and Reports, 2008 Jun 6; 57(5): 1-30.
15. Schmidt DS, Jumaan AO. Impact of varicella vaccine on varicella-zoster virus dynamics. Clinical microbiology reviews, 2010 Jan; 23(1): 202-17.
16. Jeon YH. Herpes zoster and post herpetic neuralgia: practical consideration for prevention and treatment. The Korean journal of pain, 2015 Jul 1; 28(3): 177.
17. Wang X, Huang S, Shao G, Zhang J, Wang S, Lv Y, Dong F, Han J, Yang D. Efficacy and safety of bloodletting for herpes zoster: A protocol for systematic review and meta-analysis. Medicine, 2021 Jun 4; 100(22): e26169.
18. Rexes M. Herpes zoster and postherpetic neuralgia: diagnosis and therapeutic considerations. Alternative medicine review, 2006 Jun 1; 11(2).
19. Ong OL, Churchyard AC, New PW. The importance of early diagnosis of herpes zoster myelitis. Medical journal of Australia, 2010 Nov; 193(9): 546-7.
20. Ruiz Junior FB, Sinoski JS, Marques Junior W, Ferreira MS. Abdominal wall protrusion following herpes zoster. Revitas da Sociedad Brasilia de Medicine Tropical, 2007; 40: 234-5.
21. Koshy E, Mengting L, Kumar H, Jianbo W. Epidemiology, treatment and prevention of herpes zoster: a comprehensive review. Indian Journal of Dermatology, Venereology and Leprology, 2018 May 1; 84: 251.
22. Jeon YH. Herpes zoster and postherpetic neuralgia: practical consideration for prevention and treatment. The Korean journal of pain, 2015 Jul 1; 28(3): 177.
23. Ono F, Yasumasa S, Furumura M, Hamada T, Ishii N, Gyotaku T, Higuchi M, InTouch K, Jyo K, Koga H, Komai A. Comparison between famciclovir and valacyclovir for acute pain in adult Japanese immunocompetent patients with herpes zoster. The Journal of dermatology, 2012 Nov; 39(11): 902-8.
24. Lam NN, Weir MA, Yao Z, Blake PG, Beyda MM, Gomes T, Gandhi S, Mamdani M, Wald R, Parikh CR, Hackam DG. Risk of acute kidney injury from oral acyclovir: a population-based study. American journal of kidney diseases, 2013 May 1; 61(5): 723-9.
25. Aydoğdu İ, Ataç E, Saltürk Z, Atar Y, Özdemir E, Uyar Y, Arslanoğlu A, Berkiten G. Pediatric Ramsay hunt syndrome: Analysis of three cases. Case Reports in Otolaryngology, 2015; 2015(1): 971249.
26. Murakami S, Hato N, Horiuchi J, Honda N, Gyo K, Yanagihara N. Treatment of Ramsay Hunt syndrome with acyclovir- prednisone: significance of early diagnosis and treatment. Annals of neurology, 1997 Mar; 41(3): 353-7.
27. Shin DW, Kim JM, Ahn SW, Youn YC, Kwon OS. Ramsay Hunt syndrome after cervical zoster in an immunocompetent patient. Neurological Sciences, 2014 Oct; 35(10): 1635-6.
28. Monsanto RD, Bitten court AG, Bobato NJ, Beilke SC, Lorenzetti FT, Salomone R. Treatment and prognosis of facial palsy on Ramsay Hunt syndrome: results based on a review of the literature. International archives of otorhinolaryngology, 2016; 20(04): 394-400.

29. Rj W. Acyclovir with and without prednisone for the treatment of herpes zoster. Ann Intern Med., 1996; 125: 376-83.
30. Wood MJ, Johnson RW, McKendrick MW, Taylor J, Mandal BK, Crooks J. A randomized trial of acyclovir for 7 days or 21 days with and without prednisolone for treatment of acute herpes zoster. New England Journal of Medicine, 1994 Mar 31; 330(13): 896-900.
31. CLEMMENSEN OJ, ANDERSEN KE. ACTH versus prednisone and placebo in herpes zoster treatment. Clinical and experimental dermatology, 1984 Nov; 9(6): 557-63.
32. Hviid A. Use of acyclovir, valacyclovir, and famciclovir in the first trimester of pregnancy and the risk of birth defects. Jama, 2010 Aug 25; 304(8): 859-66.
33. Ratanajamit C, Skriver MV, Jepsen P, Chongsuvivatwong V, Olsen J, Sørensen HT. Adverse pregnancy outcome in women exposed to acyclovir during pregnancy: a population-based observational study. Scandinavian journal of infectious diseases, 2003 Jan 1; 35(4): 255-9.
34. Sadati MS, Ahrari I. Severe herpes zoster neuralgia in a pregnant woman treated with acetaminophen. Acta Medica Iranica, 2014; 238-9.
35. Daïen CI, Cohen JD, Jorgensen C. Zoster cruralgia in a pregnant woman. Joint bone spine, 2009 Dec 1; 76(6): 724-5.
36. Marin M, Güris D, Chaves SS, Schmidt S, Seward JF. Prevention of Varicella. MMWR: Morbidity & Mortality Weekly Report, 2007 Jun 23; 56.
37. Rothe MJ, Feder Jr HM, Grant- Kels JM. Oral acyclovir therapy for varicella and zoster infections in pediatric and pregnant patients: a brief review. Pediatric dermatology, 1991 Sep; 8(3): 236-42.
38. Kakourou T, Theodoridou M, Mostrou G, Syriopoulou V, Papadogeorgaki H, Constant Poulos A. Herpes zoster in children. Journal of the American Academy of Dermatology, 1998 Aug 1; 39(2): 207-10.
39. Brar BK, Pall A, Gupta RR. Herpes zoster neonatorum. The Journal of dermatology, 2003 Apr; 30(4): 346-7.
40. JG K. Herpes zoster in the first year of life following postnatal exposure to varicella-zoster virus. Arch Dermatol, 2004; 140: 1268-72.
41. Oleszko M, Zapolnik P, Kmiecik W, Czajka H. Herpes Zoster: Risk Factors for Occurrence, Complications, and Recurrence with a Focus on Immuno compromised Patients. Diseases, 2025 Feb 26; 13(3): 71.
42. Pergam SA, Limaye AP, AST Infectious Diseases Community of Practice. Varicella zoster virus in solid organ transplantation. American journal of transplantation, 2013 Mar 1; 13: 138-46.
43. Gan EY, Tian EA, Tey HL. Management of herpes zoster and post-herpetic neuralgia. American journal of clinical dermatology, 2013 Apr; 14(2): 77-85.
44. Gilden D, Cohrs RJ, Mahalingam R, Nagel MA. Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, pathogenesis, and treatment. The Lancet Neurology, 2009 Aug 1; 8(8): 731-40.
45. Lin YY, Kao CH, Wang CH. Varicella zoster virus infection of the pharynx and larynx with multiple cranial neuropathies. The Laryngoscope, 2011 Aug; 121(8): 1627-30.
46. Staikov I, Neykov N, Marinovic B, Lipozenčić J, Tsankov N. Herpes zoster as a systemic disease. Clinics in dermatology, 2014 May 1; 32(3): 424-9.