International Journal of Pharmaceutical Research and Applications

Volume 6, Issue 4 July-Aug 2021, pp: 1329-1331 www.ijprajournal.com ISSN: 2249-7781

A Case Report On Herpes Zoster Infection

Vaishnavi L R¹, DivyaS², SupriyaS³

¹Pharm D Intern, PRS Hospital, killipalam, Trivandrum.

²Neurologist, Department of Neuromedicine, PRS Hospital, Trivandrum.

³SupriyaS, Clinical Pharmacologist, PRS Hospitalkillipalam, Trivandrum.

Date of Submission: 01-08-2021 Date of Acceptance: 18-08-2021

ABSTRACT

Herpes zoster (HZ) is a well-known viral disease that usually presents as a painful unilateral vesicular rash restricted to the distribution of a sensory nerve. HZ which is also known as Shingles, is an acute infection of viral origin resulting from the reactivation of the DNA virus varicella zoster, which causes chickenpox. It commonly manifests as vesicular rash, which are painful and runs its progression in a matter of 4-5 weeks. The pain may persist for months or even years after healing of the skin lesions. This phenomenon is called as postherpetic neuralgia (PHN). The risk of PHN in patients with zoster is approximately 10-18%. Nearly, 3% of patients with zooster are hospitalized. Morbidity due to zoster is common among immunocompromised patients.

I. INTRODUCTION

Herpes zoster is viral infection that occurs with reactivation of the varicella-zooster virus. It is usually a painful but self-limited dermatomal rash. Symptoms typically start with pain along the affected dermatome, which is followed in 2-3 days by a vesicular eruption. Classic physical findings include painful grouped herpetiform vesicles on an erythematous base. Treatment includes antiviral medications such as acyclovir, famciclovir, and valacyclovir given within 72 hours of symptom onset. Reactivation of varicella-zoster virus (VZV) that has remained dormant within dorsal root ganglia, often for decades after the patient's initial exposure to the virus in the form of varicella (chickenpox), results in herpes zooster (shingles). The clinical manifestations can be divided into the following three phases:Preeruptive phase (preherpetic neuralgia), Acute eruptive phase&Chronic phase (PHN).

The preeruptive phase is characterized by the following:

- Sensory phenomena along 1 or more skin dermatomes, lasting 1-10 days (average, 48 hours)
- Phenomena usually are noted as pain or, less commonly, itching or paresthesias [3]
- Pain may simulate headache, iritis, pleurisy, brachial neuritis, cardiac pain, appendicitis or other intra-abdominal disease, or sciatica
- Other symptoms, such as malaise, myalgia, headache, photophobia, and, uncommonly, fever.

The acute eruptive phase is marked by the following:

- Patchy erythema, occasionally accompanied by induration, in the dermatomal area of involvement
- Regional lymphadenopathy, either at this stage or subsequently
- Grouped herpetiform vesicles developing on the erythematous base (the classic finding)
- Cutaneous findings that typically appear unilaterally, stopping abruptly at the midline of the limit of sensory coverage of the involved dermatome
- Vesicular involution: Vesicles initially are clear but eventually cloud, rupture, crust, and involute
- After vesicular involution, slow resolution of the remaining erythematous plaques, typically without visible sequelae
- Scarring can occur if deeper epidermal and dermal layers have been compromised by excoriation, secondary infection, or other complications
- Almost all adults experience pain, typically severe
- A few experience severe pain without a vesicular eruption (ie, zoster sine herpete)
- Symptoms tend to resolve over 10-15 days
- Complete healing of lesions may require up to a month

PHN is characterized by the following:



International Journal of Pharmaceutical Research and Applications

Volume 6, Issue 4 July-Aug 2021, pp: 1329-1331 www.ijprajournal.com ISSN: 2249-7781

- Persistent or recurring pain lasting 30 or more days after the acute infection or after all lesions have crusted (9-45% of all cases)
- Pain usually is confined to the area of original dermatomal involvement
- The pain can be severe and incapacitating
- Pain can persist for weeks, months, or years
- Slow resolution of pain is especially common in the elderly
- PHN is observed more frequently after cases of herpes zoster ophthalmicus (HZO) and in instances of upper-body dermatomal involvement
- Less common postherpeticsequelae include hyperesthesia or, more rarely, hypoesthesia or anesthesia in the area of involvement.

Diagnosis is based primarily on the history and physical findings. Laboratory studies for VZV include the following:

- Direct fluorescent antibody (DFA) testing of vesicular fluid or a corneal lesion.
- Polymerase chain reaction (PCR) testing of vesicular fluid, a corneal lesion, or blood.
- Tzanck smear of vesicular fluid (lower sensitivity and specificity than DFA or PCR).

II. CASE REPORT

A 69 year old female patient was admitted in the neurology department with complaints of acute onset altered sensorium since 1 day followed by one episode of seizure(GTCs) lasted for five minutes followed by prolonged irritability and confusion. Also she had history of herpes zoster left thoracic dermatome one week prior to the same.On general physical examination, the patient wasirritable, restless, not obeying commands and was moving all four limbs equally to pain.On evaluation of vital signs, temperature was normal pulse rate was 80 beats/minute and BP was 140/70 mm Hg.Laboratoryreports,CT and MRI scan was found to be normal.CSF varicella zoster PCR tes was done, which was negative ,there was severe electrophysiological dysfunction.Correlating the case history and clinical findings,a final diagnosis of acute varicella zoster encephalitis was given. She was treated with Acyclovir for 14 days,antioedema and antiepilepticsmeasures.Patient improved well in next 48 hours, no further headache or seizure was reported .She was better, asymptomatic and stable at discharge.

III. DISCUSSION

Varicella-zoster virus (VZV) like other herpes viruses causes both primary and recurrent infections and remains latent neurons present in the sensory ganglia. VZV is associated with two major clinical infections of humans: Chickenpox (varicella) and shingles (HZ). Chicken pox is a primary infection that occurs the first time an individual is affected by the virus with generalized manifestations. After the primary disease heals, VZV remains latent in the dorsal root ganglia of spinal nerves or extramedullary ganglia of cranial nerves. A person without any prior contact with VZV can develop chickenpox after coming in contact with an individual with HZ.patient complained of mild pain. Hence, HZ disease patients can have mild to severe pain during active stage of the disease. Patients with HZ may progress through three stages; prodromal, active &chronic

The prodromal stage is characterized by sensations such as burning, tingling, itching, pricking, occurring along the cutaneous distribution of dermatome. Odontalgia and pulpal necrosis may result if branches of the trigeminal nerve are involved, during this phase.4 These symptoms may be present up to 1-month in advance of the acute mucocutaneous lesions, and hence, this stage is difficult to diagnose.

The active stage is described by the appearance of the rash with along with the systemic upset. The skin rash is very characteristic and progresses from erythematous papules, edema to vesicles, and finally to pustules within 1-7 days. Later, these pustules dry, crust, and are exfoliated over the next 2-3 weeks leaving erythematous macular lesions that may scar. The active or "eruptive" phase of HZ is most contagious and can pose a significant risk of cross infection

Approximately 10% of all patients advances to the chronic stage of HZ, and is known as PHN which is defined as a short-lived, deep, shooting, and recurrent pain remaining for over a month or 3 months after the healing of the mucocutaneous lesions. Risk of occurrence of PHN increases significantly after sixth decade, which may be because of decline of cell-mediated immunity.Root resorption, tooth exfoliation, periapical lesions, and osteonecrosis of the alveolar bone have also been reported in association with HZ infection.



International Journal of Pharmaceutical Research and Applications

Volume 6, Issue 4 July-Aug 2021, pp: 1329-1331 www.ijprajournal.com ISSN: 2249-7781

In majority of the HZ patients, the condition is selflimiting, and healing is usually complete. However, the management is indicated as follows:

- To alleviate the symptoms of pain and malaise
- To restrict the spread as well as duration of the skin lesions and
- To prevent the development of PHN and ophthalmological complications.

Diagnosis at an early stage of the disease and prompt treatment in the prodromal phase by the antiviral drugs usage should be the mainstay of its management.

In this case, we advised the patient to be in isolation, so as to prevent the viral transmission to the healthy individuals. Drug therapy was started with analgesics and antiviral agents viz. acyclovir 14 days, post 2 weeks of which the patient showed significant improvement.

IV. CONCLUSION

Herpes zoster is viral infection that occurs with reactivation of the varicella-zoostervirus.In this case patient was admitted with complaints of acute onset altered sensorium since 1 day followed by one episode of seizure(GTCs) lasted for five minutes followed by prolonged irritability and confusion . Also she had history of herpes zoster left thoracic dermatome one week prior to the admission.She was treated with Acyclovir for 14 days,antioedema and antiepilepticsmeasures.Early diagnosis and prompt treatment by antiviral agents aid in reducing the duration and severity of zoster infections and prevent further complications .

REFERENCES

- [1]. Bandral MR, Chidambar YS, Telkar S, et al. Oral complications of herpes zoster infection—report of 3 cases. Int J Dent Clin 2010;2013:70–3
- [2]. Roxas ND Mario. Herpes zoster and postherpetic neuralgia: diagnosis and therapeutic considerations. Altern Med Rev 2006;2013:102–13
- [3]. Dworkin RH, Schmader KE. Treatment and prevention of postherpetic neuralgia. Clin Infect Dis 2003;2013:877–82
- [4]. Sook BW, Martin SG. Ulcerative, vesicular and bullous lesions.Burket's oral medicine. 11th edn BC Decker, 2008:46–9
- [5]. Sampathkumar P, Drage LA, Martin DP. Herpes zoster (shingles) and post herpetic neuralgia. Mayo ClinProc 2009;2013:274–80

- [6]. Whitley RJ. Varicella-zoster virus infections. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL,Randolph T, Harrison, eds Harrison's principles of internal medicine. 14th edn New York, NY: McGraw Hill, 1998:1086–9
- [7]. Nagel AM, Gliden HD. The protean neurologic manifestations of varicella-zoster virus infection. J NeuroVirol 2002;2013(Suppl 2):75–9.