CASE REPORT



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# Recurrent herpes zoster with segmental paresis and postherpetic neuralgia

Rekurentni herpes zoster komplikovan segmentnom parezom i postherpetičnom neuralgijom

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#### **Abstract**

Introduction. Postherpetic neuralgia and segmental paresis represent rare complications of herpes zoster infection. Recurrent herpes zoster is also rare and occurs within the first 3 years of the begining of the illness in only 1.4% of cases but it is generally higher in cases of chronic lymphatic leukemia (3.5%). Case report. We presented a patient with lymphatic leukemia who during the remission had 3 episodes of herpes zoster over a year. All of them took diffrent parts of the body. One of these episodes was complicated by postherpetic neuralgia and segmental paresis. A complete recovery was seen in all the three episodes. Conclusion. As immunosuppression is one of mechanisms of virus reactivation, it is likely associated with the described rare complications of herpes zoster.

### Key words:

herpes zoster; leukemia, lymphocytic, chronic, b-cell; comorbidity; paresis; neuralgia; drug therapy.

#### Apstrakt

Uvod. Postherpetična neuralgija i segmentna zoster pareza neke su od ređih komplikacija herpes zostera. Rekurentni herpes zoster je, takođe, retkost a viđa se samo kod 1,4% bolesnika mada je nešto češći kod obolelih od hronične limfatične leukemije (3,5%). Prikaz bolesnika. Prikazali smo bolesnika sa hroničnom limfatičnom leukemijom koji je za godinu dana, u vreme remisije osnovne bolesti, imao tri epizode herpes zostera od kojih je jedna bila komplikovana segmentnom zoster parezom i postherpetičnom neuralgijom. Svako od ovih ispoljavanja zahvatalo je različite delove tela. Jedno od njih bilo je komplikovano postherpetičnom neuralgijom i segmentnom parezom. Kompletan oporavak usledio je posle svake od opisanih epizoda. Zaključak. Kako je imunosupresija mogući mehanizam reaktivacije virusa, moguće je da je upravo ona odgovorna i za pojavu navedenih retkih komplikacija herpes zostera.

# Ključne reči:

herpes zoster; leukemija, limfocitna, hronična; komorbiditet; pareza; neuralgija; lečenje lekovima.

#### Introduction

Herpes zoster (HZ) is most commonly seen in older people and in cases of immunodeficiency. Despite higher incidence in immunocompromised patients, the recurrence of HZ is rare <sup>1-5</sup>.

Postherpetic neuralgia is defined as the existence of pain at 4 to 6 weeks after vesicular skin eruptions resolve. Although postherpetic neuralgia is the most common complication of HZ, it occurs in only 5% to 10% of cases <sup>6</sup>. Typically, it resolves over time but some patients complain of persistent pain.

A 60 year-old patient with chronic lymphatic leukemia who in the remission developed 3 episodes of HZ within a year is presented.

Complications of HZ infection are rare. The most common is a postherpetic neuralgia and rarely segmental zoster paresis. Both complications occurred in the presented case.

#### Case report

A 60-year-old man with chronic lymphatic leukemia in remission developed in April 2010 an unpleasant burning pain along the inner side of the right lower leg, which progressed over time. The pain was scored 6 out of 10 on the pain scale. Herpetic rash developed within a week in the area of pain (Figure 1). Ten days after the initial pain, the patient had difficulty with moving the right foot, which prompted him to make an appointment. The neurological examination revealed a

moderate to severe weakness of the right ankle plantar flexors and diminished ankle jerk (3/5 on MRC-scale).



Fig. 1 – Herpetic rash along the inner lower leg.

Electrophysiological examination performed 4 weeks after the onset of symptoms did not reveal changes in the nerve motor conduction in the right leg. However, needle electromyography (EMG) at rest showed denervation potentials (fibrillations and positive sharp waves) in the right gastrocnemius muscle and paravertebral muscles innervated by S1 root. Voluntary activation during EMG examination revealed polyphasic motor unit potentials during activation of the right foot.

The magnetic resonance imaging (MRI) of the lumbosacral area showed only degenerative changes without other findings that could explain the neurologic deficits.

Testing serum (ELISA) on HZ virus was positive for IgM and IgG, which confirmed the presence of HZ infection.

The neurologic findings were ascribed to segmental herpes paresis.

The patient was referred to physical therapy and prescribed oral gabapentin (900 mg/day) for pain. The patient was on the same medication for 5 months because of severe pain, which was afterwards slowly decreased and eventually discontinued. Motor weakness completely resolved about 6 months after the onset of neurologic symptoms whereas the pain was sporadic but mild.

About 4 months after the first symptom appearance (August 2010), the patients noted new-onset burning pain in the right hand graded 8 out 10 on the pain scale. Five days later, the herpetic rash developed in the painful area. The patient did not complain of motor weakness in the right hand. The overall presentation was consistent with recurrence of HZ but without other neurological complications. Gabapentin was again prescribed for pain (1200 mg/day) but only for 6 weeks at which point the rash and pain disappeared (Figure 2).

Six months later (February 2011), the patient noted discomfort and increased skin sensation over the abdomen followed by burning pain (4/10). A few days later herpetic rash developed in the same area (Figure 3). There were no other symptoms or sign. Gabapentin was not prescribed this time considering mild pain. Pain and rash disappeared within 2 months.



Fig. 2 – Herpetic rash in the hand area.



Fig. 3 – Herpetic rash over the abdomen.

#### Discussion

After primary infection, varicella zoster virus remains latent in the dorsal root ganglia for several years. The virus can get reactivated and migrate along the sensory nerves toward the skin innervated by the involved nerve. This results in HZ rash characterized by vesicular skin eruption accompanied by pain and dysesthesia or hyperesthesia corresponding to the affected dermatomes. Pain usually develops first followed by rash within a week of pain onset. The disease typically lasts 5–8 weeks <sup>7</sup>. In the presented case, pain preceded the onset of rash.

The mechanism of virus reactivation is unknown. In immunocompromised patients, it is likely associated with a decrease in the immune system, as in cases of lymphoma and AIDS <sup>3</sup>. The described patient had a chronic lymphatic leukemia that was in remission when the 3 HZ episodes occurred.

The annual incidence of HZ is 3.6/1,000 in general population or 300,000 new cases a year. Evidently higher incidence of HZ in 6 and 7 decades may be associated with a decrease in cellular rather than humoral immunity after 60 years of age <sup>5</sup>. The presented case belongs to a typical age group.

The recurrent HZ is rare and occurs within the first 3 years of the initial insult in only 1.4% of cases. The rate of recurrent infection is about 10–20% several decades later. Since the incidence of HZ is generally higher in cases of chronic lymphatic leukemia (28.6%), the recurrence rate is

also higher  $(3.5\%)^3$ . In the presented patient, 3 recurrences occurred within a year.

There are several approaches to managing post-herpetic neuralgia. Beginning treatment with antiviral agents as soon as the rash appears is considered to be the key for preventing the development of postherpetic neuralgia. Prescribing antiviral agents for a week within 3 days of rash (famcyclovir 500 mg tid or acyclovir 1 g tid), decreases pain and the incidence of postherpetic neuralgia <sup>8–11</sup>. Oral corticosteroids for 3 weeks (prednisone 60 mg/day in week 1,30 mg/day in week 2, and 15 mg/day in week 3), along with antiviral agents, can also reduce the overall severity and duration of pain, and thereby reduce the incidence of postherpetic neuralgia 11, 12. However, it is not clear whether the mechanism of corticosteroid action is local or systemic <sup>10</sup>.

The second most common complication of HZ is segmental motor paresis, which occurs in 0.5% to 5% of patients 12, 13. Although the pathophysiology of muscle weakness remains unknown, the overlap between segmental paresis and dermatomal area affected by rash suggests that HZ virus spreads from the dorsal ganglia toward anterior spinal roots<sup>2,4</sup>. This is supported by histological findings typical for HZ in the anterior roots. Moreover, the absence of fasciculations in the affected muscles on EMG and slowing of motor conduction velocities suggest axonal lesions rather than the affection of the anterior horn of the spinal cord. Weakness typically develops within 2 weeks of rash, however, it may also precede pain <sup>7, 13, 14</sup>. The possible invasion of the anterior roots by the HZ virus was first considered by Wohlwill in 1924 <sup>15</sup>. In the described case, muscles paresis and the distribution of skin lesions were in the tibial nerve distribution.

In terms of neurophysiology, EMG shows denervation in the presence of motor weakness. Usual EMG findings include fibrillation potentials, PDP, and prolonged insertion activity whereas fasciculations are typically absent. Polyphasic motor unit potentials are present upon attempts of volun-

tary activation of weak muscles. This is typically seen in myotomes that correspond to dermatomes affected by pain and herpetic eruption. In addition, EMG may reveal a prolonged terminal latency, slowing of motor conduction velocity, occasionally absent motor response, or prolonged motor latency of the affected nerve or decrease in amplitude <sup>16</sup>. In the presented case, there was a denervation activity in the muscles innervated by the tibial nerve in which distribution was also present the skin lesions.

HZ more often affects proximal than distal portion of the arms. Although zoster paresis seems more common in the upper limbs, it was present in this case in the lower limbs but not in the upper limb that was later affected by the skin lesions.

The third episode of HZ affected the abdominal skin but it was not accompanied by weakness of the abdominal muscles or post-herpetic neuralgia in this region. The outcome of zoster weakness is generally good. Complete or almost complete recovery within one year occurs in 2/3 of patients <sup>1, 5</sup>, whereas additional 9% has a partial recovery.

Recovery takes place between 1 and 2 years <sup>17, 10</sup>. Segmental zoster paresis treatment includes analgesics for pain control, physical therapy, and prevention of contractures <sup>10, 18</sup>. The presented case was treated in such a way and the degree and timing of recovery is in agreement with the literature.

#### Conclusion

The presented case with chronic lymphatic leukemia is unique because of 3 recurrent episodes of HZ skin eruptions in different body regions over the course of several months. Since only the first episode was accompanied by motor weakness, it appears that an earlier affliction of the motor nerve is not a risk factor for developing nerve lesions in the other area in case of HZ recurrence.

#### REFERENCES

- Akiyama N. Herpes zoster infection complicated by motor paralysis. J Dermatol 2000; 27(4): 252-7.
- Fabian VA, Wood B, Crowley P, Kakulas BA. Herpes zoster brachial plexus neuritis. Clin Neuropathol. 1997; 16(2): 61-4.
- Hamblin AD, Hamblin TJ. The immunodeficiency of chronic lymphocytic leukaemia. Br Med Bull 2008; 87: 49-62.
- Hanakawa T, Hashimoto S, Kawamura J, Nakamura M, Suenaga T, Matsuo M. Magnetic resonance imaging in a patient with segmental zoster paresis. Neurology 1997; 49(2): 631-2.
- Hoque R, Schwendimann RN, Liendo C, Chesson AL Jr. Brachial neuritis with bilateral diaphragmatic paralysis following herpes zoster: a case report. J Clin Neuromuscul Dis 2008; 9(4): 402-4.
- Kranse PR, Strans SE. Zoster and its complications. Hosp Pract (Off Ed) 1990; 25(10A): 61–71, 75–6.
- 7. Wendling D, Langlois S, Lohse A, Toussirot E, Michel F. Herpes zoster sciatica with paresis preceding the skin lesions. Three case-reports. Joint Bone Spine 2004; 71(6): 588-91.

- Jackson JL, Gibbons R, Meyer G, Inonye L. The effect of treating herpes zoster with oral acyclovir in preventing postherpetic neuralgia. A meta-analysis. Arch Intern Med 1997; 157(8): 909–12.
- Smith KJ, Roberts MS. Antiviral therapies for herpes zoster infections. Are they economically justifiable? Pharmacoeconomics 2000; 18(2): 95–104.
- Schuchmann JA, McAllister RK, Armstrong CS, Puana R. Zoster sine herpete with thoracic motor paralysis temporally associated with thoracic epidural steroid injection. Am J Phys Med Rehabil 2008; 87(10): 853–8.
- Whitley RJ, Weiss H, Gnann JW Jr, Tyring S, Mertz GJ, Pappas PG, et al. Acyclovir with and without prednisone for the treatment of herpes zoster. A randomized, placebo-controlled trial. The National Institute of Allergy and Infectious Diseases Collaborative Antiviral Study Group. Ann Intern Med 1996; 125(5): 376–83.
- Yann BP, Saddier P, Wollan PC, St Saurer JL, Kurland MJ, Sy LS.
   A population-based study of the incidence and complication rates of herpes zoster before zoster vaccine introduction.
   Mayo Clin Proc 2007; 82(11): 1341–9.

- 13. Ter Meulen BC, Rath JJ. Motor radiculopathy caused by varicella zoster virus without skin lesions ('zoster sine herpete'). Clin Neurol Neurosurg 2010; 112(10): 933.
- 14. Umehara T, Sengoku R, Mitsumura H, Mochio S. Neurological picture. Findings of segmental zoster paresis on MRI. J Neurol Neurosurg Psychiatry 2011; 82(6): 694.
- Wohlwill F. Zur pathologischen Anatomie des Nervensystems beim Herpes zoster. (Auf Grund von zehn Sektionsfällen.) Z Ges Neurol Psychiat 1924; 89(1): 171–212.
- McAllister RK, Borum SE, Mitchell DT, Bittenbinder TM. Thoracic motor paralysis secondary to zoster sine herpete. Anesthesiology 2002; 97(4): 1009–11.
- 17. Kawajiri S, Tani M, Noda K, Fujishima K, Hattori N, Okuma Y. Segmental zoster paresis of limbs- report of three cases and review of literature. Neurologist 2007; 3(5): 313–7.
- 18. Sachs GM. Segmental zoster paresis: an electrophysiological study. Muscle Nerve 1996; 19(6): 784-6.

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