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Spinal segmental myoclonus improved with epidural blockade in a patient with herpes zoster radiculitis

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Received: October 6, 2016 Revised: November 21, 2016 Accepted: November 30, 2016 The spinal segmental myoclonus by viral radiculitis has been rarely reported and the pathophysiology remains to be elucidated. However, the hyperactivity of contiguous anterior horn neurons induced by viral irritation has been suggested to be a possible patho-mechanism. In general, spinal segmental myoclonus is not well-controlled by medication and the patient suffers from continuous involuntary movement. We recently experienced a case of spinal segmental myoclonus induced by herpes zoster radiculitis, and which was successfully relieved by epidural injections.

Key words: Spinal myoclonus; Myoclonus; Herpes zoster; Radiculitis

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Fax: +82-2-6299-1504 E-mail: sukwonahn@gmail.com Spinal segmental myoclonus is 'involuntary rhythmic contractions of muscle groups innervated by limited spinal cord region'. Spinal segmental myoclonus is due to pathology at the involved level of the spinal cord and the lesion have included tumor, infection, trauma, peripheral nerve disease, radiculopathy and degenerative process. Spinal myoclonus associated with herpes zoster radiculitis is not common, and a few clinical cases have been reported. The mechanism has been suggested to be the hyperexcitability of spinal segmental systems by viral irritation, however the distinct patho-physiology remains to be elucidated.

In the patients with herpes zoster infection, epidural injections of corticosteroids with or without local anesthetics have an effect on the uncontrolled neuropathic pain.⁸ However, the resolution of spinal segmental myoclonus by epidural block has not been reported to our knowledge.

We present a case of spinal segmental myoclonus induced by herpes zoster, and which was completely resolved after epidural spinal block of injection with steroid and local anesthesics.

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CASE

A 77-year-old man admitted to our hospital presenting spontaneous jerky movement in his right lower limb 2-3 weeks after right lower limb herpes zoster (at L3-S1). And also, he complained of uncontrolled burning pain, numbness, and paresthesias in the anterior aspect of the right leg distal to the knee. The patient showed the abrupt and continuous muscle contraction of the hip and thigh (quadriceps, iliopsoas) with a rate of 10/minute corresponding to the root levels affected by zoster infection, and which was not improved by distraction (Supplementary Video 1).

Physical examination revealed paresthesia in the right L3-S1 dermatomes, and the stretch reflex of right patellar and achilles tendon were decreased. Motor strength of the right lower limb was normal and the other neurological examination was normal.

The electroencephalogram and brain magnetic resonance image (MRI) were normal, and lumbosacral spinal MRI did not showed distinct cord lesion involving tumor, inflammation and cord compression. Needle electromyography revealed the acute denervation-potentials in the right paraspinal muscles, and vastus lateralis muscle and peroneus longus muscle, which was indicative of lumbosaral radiculopathy (L3, 4, 5, and S1).

The patient was diagnosed as herpes zoster radiculitis and induced spinal segmental myoclonus. For 2 weeks, he was treated for relief pain and myoclonus with various medications— acyclovir 800 mg for 10 days and oxcarbazepine 1,200 mg, gabapentin 1,200 mg, pregabalin 300 mg, amitriptyline 20 mg and clonazepam 0.5 mg a day, and which medicines had some effect on neuropathic pain. However, the spinal segmental myoclonus of right lower limb persisted without improvement. Because the neuropathic pain of right leg was intolerable with oral medications, we performed a right L4, S1 epidural block, epidural injection of triamcinolone acetate and ropivacaine just for neuropathic pain. Unexpectedly, after epidural block, the myoclonus of the patient completely resolved immediately, but pain relief was not significant (Supplementary Video 1).

DISCUSSION

Herpes zoster, also known as shingles, is a latent viral infection of the sensory ganglia with spread along the sensory nerves, resulting in a dermatomal vesicular rash. Motor neuron involvement can occur in 0.5-31% of cases of herpes zoster and is caused by the extension of inflammation first from the dorsal root ganglion proximally into the dorsal root and posterior horn of the spinal cord, and then into the adjacent anterior horn and ventral root.⁹

The jerky movement of our patient showed the characteristics of the myoclonus originating in the spinal root. Semirhythmic feature, mono-limb involvement, and muscle jerking limited to muscle innervated by one to three adjacent spinal levels were indicated spinal segmental myoclonus in our patient. Psychogenic jerk or pain-related movement could be excluded by consistent character, continuous jerks with distraction, stationary myoclonus after some effect of medication and disappeared myoclonus after epidural block in spite of remained neuropathic pain. The pathophysiology of spinal segmental myoclonus includes abnormal loss of inhibition from suprasegmental descending pathways, loss of inhibition from local dorsal horn interneurons, hyperactivity of contiguous anterior horn neurons, and aberrant local axon re-excitations.⁶ Spinal segmental systems may become hyperexcitable and trigger abnormal activity of alpha motor neurons often by viral irritation.^{6,7} In our patient, we suggested that the myoclonus was caused by zoster infection and induced inflammation of anterior horn neurons.

The underlying mechanism of epidural steroid and local anesthetic injections is still not well understood. ¹⁰ But, epidural blockade for patients with acute zoster can shorten the duration of the treatment. ¹¹ Also, Conliffe et al. ⁹ reported in 2009 successful treatment of herpes zoster-induced radiculopathy with L5 distribution with a lumbosacral transforaminal epidural steroid and lidocaine injection. Thus we tried to the use of epidural block just for the uncontrolled neuralgic pain in our patient. The belief is that use of corticosteroid in acute or chronic pain syndrome helps decrease inflammation as well as causing reversible local anaesthetic effect. ^{10,12}

Interestingly, in our patient, the spinal segmental myoclonus disappeared after performing of epidural block and which has not been reported in the document. There is temporal correlation between improvement of myoclonus and

epidural steroid injection. To our knowledge, the improvement of spinal myoclonus by epidural injection has not been reported in the previous document. We can propose that the steroid and analgesics during the epidural blockade might help to decrease inflammation of anterior horn cells or suppress abnormal activity of anterior neurons. Even if it is difficult to determine the definite patho-mechanism of epidural block for clinical improvement of the myoclonus, in the case of uncontrolled spinal segmental myoclonus, epidural block can be considered as an alternative treatment.

In conclusion, this is the rare case of spinal segmental myoclonus associated with herpes zoster radiculitis and the first case of improving myoclonus after epidural block. Although, we could not explain the exact mechanism, this case report can serve as the starting point for further studies to evaluate the effect of epidural blockade for spinal segmental myoclonus and to identify the underlying patho-mechanism of epidural blockade and spinal segmental myoclonus.

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