Case report

Segmental zoster paresis

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Abstract

Herpes zoster is often associated with neurological manifestation but less frequently associated with motoric nerve involvement. The most common motoric involvement is paresis, seen only in 1-5% of all zoster cases. This case report a 39-year-old male, with recurrent abdominal liposarcoma under chemotherapy, presented with symptoms of fever, and painful and weak right leg for 2 weeks. Vesicles eruption was seen on his right leg nine days after these symptoms occurred. Physical examination revealed groups of haemorrhagic vesicles with erythematous base on the right lower leg. Lumbosacral spine x-ray showed spondylosis with radiculopathy. Electromyography (EMG) examination revealed lower motor neuron total denervation corresponding to right L5 radix. The patient was diagnosed as herpes zoster on right L5-S1 segment, herpetic neuralgia, and segmental zoster paresis with recurrent liposarcoma under chemotherapy. He was treated with oral acyclovir 800 mg five times a day and gabapentin 300 mg twice a day. Physical therapy and rehabilitation were started concurrently. Paresis is a rare complication of herpes zoster. Radicular pain and weakness preceded the skin lesion potentially lead to misdiagnoses. The most frequent diagnosis for patient suffering pain and weakness in the extremities is spinal disorder, such as stenosis and disc herniation. EMG can be helpful to recognise motoric involvement of herpes zoster, and preclude other diagnoses.

Keywords: Herpes zoster, paresis, radicular pain

Introduction

Herpes zoster is often associated with various neurological manifestations including post-herpetic neuralgia, myelitis, encephalitis, cranial arteritis, segmental radiculitis, and polyradiculitis.1,2 Amongst those complications, motoric nerve involvement is extremely rare1-4 The most common symptom of motoric involvement in zoster radiculitis is muscle weakness due to peripheral motoric neuropathy.1 The most frequent form of motoric involvement in zoster is facial paresis, occurring nearly 50% of all cases.5 The second highest prevalence is limb paresis, which affected 1-5 % of all cases.1,5

The pathogenesis of paresis is predicted due to varicella-zoster virus (VZV) invasion to motor neurons and anterior horn motoric nerve inflammation.2,4 The most prevailing diagnosis of radicular pain associated with muscle weakness is spinal disorder, such as disc herniation and spinal stenosis.1 Electromyography (EMG) examination is usually performed to help determine the muscle and nerve involvement and to follow up the course of the disease. Several studies report abnormal EMG in herpes zoster patients with and without motor weakness that shows motoric involvement and denervation.1,4 Physical therapy and rehabilitation are widely used as adjunctive therapies to oral therapy in zoster paresis.1,4 Range of motion and muscle strength exercises are important to prevent muscle atrophy and contracture.1 The prognosis of zoster paresis is generally good, with more than 50% patients show significant improvement in 6-12 months.1,4
This case is reported because of the rare incidence of motoric involvement in herpes zoster, which potentially lead to misdiagnosis. Early detection and comprehensive treatment is necessary to prevent permanent paresis.

**Case Illustration**

A 39-year old man came to dermatology clinic with blisters on his right leg that developed since 5 days previously. Two weeks before, he experienced fever, pain, and weakness on his right leg that made him difficult to walk. He went to see an internist, had a leg x-ray. The x-ray showed straight lumbalis with lumbar spondylosis and intervertebral disc degeneration on the fifth lumbar (L5) to first sacrum (S1) without compression on radii. The patient was diagnosed as spondylosis with radiculopathy, and treated with pregabalin 75 mg twice daily and tramadol 50 mg three times per day. He was then consulted to neurologist and orthopaedist. Nine days after the weakness begun, a group of vesicles arose over his right leg. He suffered intense radicular pain from his right bottom to his right sole that made him ignored the vesicles. The neurologist suspected that patient had L4-L5 stenosis, and treated with arcoxia 90 mg daily, pregabalin 75 mg twice daily, and mecobalamin 500 mcg three times per day. Magnetic resonance imaging (MRI) examination showed lumbar spondyloarthrosis with herniated nucleus pulposus (HNP) in L2-3 to L4-5. The differential diagnosis from orthopaedist was HNP, and he was suggested to have surgical procedure accordingly.

In the intervening time, the vesicles on his right leg were getting prominent, distributed in groups. Five days after the first vesicle appeared he was consulted to dermatologist. Taken from the anamnesis, the patient denied any burn injury, insect bite, and history of applying any material to his leg. He had history of varicella when he was a child. This was the first time he experienced localized vesicles.

The patient had history of recurrent abdominal liposarcoma under chemotherapy. The lump on his stomach was first found 4 years ago, and had been surgically removed. Two years after the surgery, the lump reappeared. He then underwent 30 sessions of radiotherapy. A year after finishing radiotherapy, he had chemotherapy. Until the patient came to dermatology clinic, he had performed 10 cycles of chemotherapy.

On physical examination, the patient looked moderately ill, sitting on a wheelchair. On the lower right leg to the instep there were discrete multiple haemorrhagic, miliar to lenticular, herpetiformic distributed vesicles, with erythematous base (figure 1). Based on anamnesis and physical examination the patient was diagnosed as herpes zoster right L5-S1 with acute herpetic neuralgia and recurrent liposarcoma. He was treated with oral acyclovir 800 mg five times a day and pregabalin 75 mg twice daily and suggested to do cold compress. The patient was also informed about his disease and the treatment.
Two weeks after his first visit, he came back with severe pain along his right leg. He was not able to move his leg. There were no new vesicles, while the old ones were already dried out and crusted (figure 2). The patient then diagnosed as zoster paresis and referred to a neurologist to get electromyography (EMG) examination. The patient was treated with titrating dose of gabapentin, increased every 3 days. The dosage started at 100 mg once daily, and gradually increased into 600 mg daily.

Figure 2. Herpes zoster on the 45th day. Multiple hyperpigmented macules and hypopigmented scars, milliar to lenticular in size, were found on the lower right leg to the instep.

Discussion

Zoster paresis is one of the complications of herpes zoster with motoric nerve involvement. Haematology malignancy, in particular, is one risk factor that is most commonly present in herpes zoster with motoric involvement. Our patient had a history of recurrent abdominal liposarcoma, a malignancy of fat cells, and had undergone several cycles of chemotherapy.

Zoster paresis is part of radiculopathy that shows muscle weakness due to peripheral motoric neuropathy. More than 90% of radicular pain is found on the same dermatome as the skin lesion, although can also be found on other location. As in our patient, he had muscle weakness of the same dermatome as the skin lesion. The pathogenesis of paresis following herpes zoster may involve viral invasion which spread to motor neurons and anterior horn cells, causes nerve inflammation, at the same time and same site as skin eruption. On the case of muscle weakness that is not related to the skin lesion dermatome, the inflammation first reached spinal cord then later disseminated to the nerve root, from the dorsal root ganglion to anterior spinal nerve roots. It remains unclear whether the inflammation is a result of the virus infection or an immune mediated reaction.

Muscle weakness in zoster paresis typically appears two to three weeks after the herpetic rash, but in some rare cases weakness may precede the rash. Establishing the diagnosis of zoster paresis in a patient without skin lesions may be difficult. The diagnosis was often confused by other spinal disorder including HNP and spinal stenosis. As in this case, when the patient had pain and weakness 2 weeks before the blisters appeared, he was first diagnosed as HNP. This diagnosis was built based on MRI examination, albeit there was no compression on the radix. His MRI appearance could not explain the signs of radiculopathy, such as radiating pain, numbness, and muscle weakness. When imaging examination shows no anatomical abnormality, or cannot explain the symptoms that the patient experiences, EMG may help establish the diagnosis.

EMG examination is useful to demonstrate muscle or nerve abnormality. This examination, although not specific for zoster paresis, is able to locate muscle and nerve root involvement. The EMG result of our patient showed abnormality on right tibialis muscle and right long peroneus muscle, where there were many spontaneous activities without motor unit action potential recruitment. This result showed total denervation on lower motor neuron in the radix of right L5, which was indicative for motoric involvement in herpes zoster. EMG can also be used to evaluate the effectiveness of the therapy and indicate the prognosis. Besides EMG, cerebrospinal fluid (CSF) examination is helpful for evaluating the cause of infection or inflammation. CSF examination may show a mononuclear and lymphocytic reaction with protein elevation indicative of viral meningoradiculitis. Antibody anti VZV and polymerase chain reaction (PCR) DNA can also be checked from the CSF to confirmed zoster radiculopathy.

The treatment for zoster paresis is antiviral which is best given within 72 hours after the first vesicle appears. In our case, antiviral treatment was started 5 days after initial vesicles due to delayed diagnosis confirmation. This delay was potentially increased the risk of prolonged paresis. In addition to antiviral, early administration of antidepressant or anticonvulsant is helpful to ease the pain and reduce the risk of herpetic neuralgia. The common drugs are gabapentin, pregabalin, or amitriptyline. Our patient had already under pregabalin 75 mg twice daily for 4 weeks, given by the previous treating doctor. Having minimal

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improvement, gabapentin titrating dose to reach dosage of 600 mg daily was then started. Both pregabalin and gabapentin can be used to manage neuropathic pain, including herpetic neuralgia, with similar effectivity. Drug preference depends on each individual tolerance.

The treatment of segmental zoster paresis is aimed to reduce pain, prevent muscle atrophy and contracture, as well as strengthening the weak muscles. Therefore in addition to antiviral and analgesics, Yoleri et al. recommend rehabilitation program including physical therapy such as electric stimulation, massage, warm compress, transcutaneous electrical nerve stimulation, acupuncture, and range of motion exercise, for patient with zoster paresis.

The prognosis of zoster paresis is generally good, with total recovery in 55-75% cases and partial recovery in 30% cases in six months to two years’ duration. More than 50% patients improved in six to twelve months and 75% patient healed in two years. At the time of publication, the patient had had 5 months of physical therapy. With therapy, he was able to walk with assistance of a cane.

Conclusion

We report one case of segmental zoster paresis with muscle weakness preceding the typical skin lesions. Electromyography examination was helpful in establishing the diagnosis. Early diagnosis and prompt treatment may improve the prognosis.

References