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Complete recovery from segmental zoster paresis confirmed by magnetic resonance imaging: A case report

Recovery from segmental zoster paresis

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Abstract

BACKGROUND

Segmental zoster paresis, depending on the affected area, can present with severe clinical manifestations and render patients unable to perform activities of daily living. Therefore, it is necessary to rapidly diagnose and treat such a condition. No studies have reported the use of magnetic resonance imaging (MRI) to identify clinical abnormalities associated with this condition or its complete recovery. This rare case report evaluates the changes in MRI findings before and after the recovery of the patient's motor symptoms.

CASE SUMMARY

A 79-year-old woman with a history of rheumatoid arthritis and psoriasis visited the hospital for skin rashes and pain in the C5-T2 skin segments. The diagnosis was herpes zoster infection and treatment was initiated; however, motor weakness suddenly occurred 14 days after the initial symptom presentation. We confirmed abnormal findings in the nerves and muscles invaded by the shingles using electromyography and MRI. After 17 mo, the patient's symptoms had completely normalized, and MRI confirmed that there were no abnormalities.

CONCLUSION

MRI can be a useful diagnostic modality for segmental zoster paresis and patient evaluation during recovery from motor complications.

Key Words: Case report; Complication; Electromyography; Herpes zoster; MRI; Paresis

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Core Tip: Segmental zoster paresis is a rare motor complication in herpes zoster infection. Generalized severe pain and low incidence of motor complications make its diagnosis challenging for clinicians. Depending on the affected area, it can present with various motor symptoms. Although the prognosis of segmental zoster paresis is known to be fair, it is important to administer multiple treatments promptly considering the relatively long period required for recovery from motor symptoms. MRI can be advantageous in diagnosis of segmental zoster paresis and its evaluation after recovery.

INTRODUCTION

Herpes zoster (HZ) is a sporadic disease caused by reactivation of latent varicella zoster virus (VZV) in the dorsal root ganglion or cranial nerve sensory ganglion. The prevalence of HZ infection is approximately 10–20% in the general population[1]. HZ infection is characterized by unilateral vesicular skin rashes and is often associated with severe pain. The most common complication of HZ infection is postherpetic neuralgia, which persists for months to years after healing of the skin rash.

Motor complications occur rarely along the involved sensory segments, resulting in muscle weakness referred to as segmental zoster paresis. Motor complications are due to the spread of VZV from the dorsal root ganglion to ventral horn cells. The incidence of motor complications is reported to be 0.5–5% of patients infected with HZ[2,3].

Here, we report a case of severe pain with motor paresis in the left upper extremity of a 79-year-old woman due to HZ infection. MRI was performed to evaluate motor paresis. This is a rare case report in which we evaluated the changes in MRI findings before and after the recovery of the patient's motor symptoms.

CASE PRESENTATION

Chief complaints

A 79-year-old woman (height, 155.0 cm; weight, 50 kg) visited our hospital's pain clinic with a characteristic rash of HZ following the distribution of C5-T2 Left dermatomes with severe pain scoring 10 on the numeric rating scale. Fourteen days after the onset of

her initial symptoms, she complained of motor weakness without sensory defects in her left upper extremity.

History of present illness

Considering her severe pain, we decided to hospitalize her. Upon her first visit to our pain medicine clinic, the patient was alert, and her vital signs were stable except for body temperature (37.2 °C). The patient was clinically diagnosed with an HZ infection. Medical treatment with intravenous acyclovir (5 mg/kg) for 9 days and analgesics (celecoxib, targin CR) was initiated. In addition, neuraxial block for pain control, including cervical plexus block, axillary nerve block, and cervical epidural block with a local anesthetic (0.15% ropivacaine diluted with normal saline) injections was performed during her hospitalization. Symptoms of skin rash and pain were relieved over time.

However, 14 days after the initial symptoms, the patient complained of motor weakness without sensory defects in her left upper extremity. There was no history of recent trauma or surgery.

History of past illness

The patient had underlying diseases, including hypertension, psoriasis, and rheumatoid arthritis, for which she was taking amlodipine, ustekinumab, and methotrexate.

Personal and family history

Personal and family history was unremarkable.

Physical examination

The patient was examined neurologically to assess the motor dysfunction of the left upper extremity. She complained that she could not lift her upper extremity against gravity. (Shoulder abduction/adduction G2+/G2+, elbow flexion/extension G3+/G3+).

Laboratory examinations

Initial laboratory tests results were within normal limits; complete blood cell count and blood chemistry tests, such as renal panel, hepatic panel, and coagulation tests were performed. Electromyography (EMG) of the left arm suggested denervation potentials in the brachioradialis, supraspinatus, infraspinatus, deltoid, extensor indicis, and C6 paraspinalis muscles (Figure 1).

Imaging examinations

MRI of the brachial plexus showed hyperintense signal on T2-weighted images with low signal on T1-weighted images in the supraspinatus, infraspinatus, and subscapularis muscles of her left shoulder indicating the potential for neuropathy without any muscle injury (Figure 2).

FINAL DIAGNOSIS

The patient was diagnosed with segmental zoster paresis as a complication of HZ infection.

TREATMENT

After consultation with a neurologist, we initiated treatment with prednisolone 40 mg for 5 days, which was then reduced by 10 mg every 5 days. In addition, she repeatedly underwent nerve block procedures (cervical plexus, axillary nerve, and cervical epidural blocks) with local anesthetics (0.15% ropivacaine diluted with normal saline) injections.

OUTCOME AND FOLLOW-UP

A month later, she showed some improvement in her motor symptoms (motor grade 3) and was discharged from the hospital. In outpatient follow-up after 10 mo, she was able to move her left upper extremity against gravity with some resistance (motor grade 4). Six months later, she fully recovered to motor grade 5. MRI of the brachial plexus was

performed again to study the changes compared to the previous imaging findings. All previous abnormal findings disappeared, and there was no difference compared with the right upper extremity (Figure 3). EMG was not performed because the patient refused it due to the discomfort of the examination and the high cost.

DISCUSSION

HZ infection is associated with host immune status. The risk factors include age and immunocompromised status. The major risk factor is old age; after the age of 50 years, the incidence of HZ infection increases dramatically[4]. Because immunocompromised patients have poor T cell-mediated immunity, the risk of VZV reactivation increases in transplant recipients, patients with autoimmune diseases, and those undergoing immunosuppressive therapies. This patient was in the high-risk group based on her age (79 years), rheumatoid arthritis history, and medication history of ustekinumab and methotrexate. The common characteristic manifestation of HZ infection is dermatomal skin lesions with pain. However, as seen in this case, motor paresis can occur in 0.5–5% of patients infected with HZ, and is called segmental zoster paresis. Segmental zoster paresis has been reported to occur in the same dermatome 2–3 wk after the skin manifestations occur [5]. Depending on the site of infection of the dermatome, various symptoms can occur. Motor complications can appear as cranial (Ramsay Hunt syndrome, involving the facial nerve) and peripheral (motor paresis of extremities, diaphragm, or abdominal muscles) neuropathies. Moreover, visceral involvement of the urinary (bladder dysfunction) and gastrointestinal tracts (colon pseudo-obstruction) can occur[6]. For example, limb segmental zoster paresis can occur if the C5-7 or L2-4 nerve roots are affected. Abdominal wall muscles, receiving nerve supply from the T6-L1, can present as a pseudohernia[7]. Physicians may misjudge whether surgical treatment is necessary. Bladder and bowel dysfunction complications can occur when HZ infection involves the sacral sensory ganglia.

MRI could play a diagnostic role, demonstrating hyperintense signal on T2-weighted images in muscular lesions[8]. In water-sensitive MRI sequences, denervated muscles

show higher signal intensity than normal muscles, secondary to increased extracellular water[9]. This abnormal finding of MRI is nonspecific and can appear in other conditions, for example, infections, inflammation, ischemia, infarction, metabolic disorders, contusion, tumors and rhabdomyolysis[10]. However, when we consider the MRI findings of signal abnormalities on T2-weighted images without abnormal signals on T1-weighted images of denervated muscles and patient's clinical features, MRI can help clinicians establish differential diagnoses from other conditions because denervation injury only affects the muscles supplied by those nerves. Signal abnormalities on T2-weighted images can last for approximately 3 mo[11]. Although the pathophysiology of segmental zoster paresis is unclear, MRI findings in this case suggest that the most probable cause might be the direct spread of the VZV from the sensory ganglion to the ventral horn cells or ventral spinal nerve roots, which some studies have previously reported[12]. Another study demonstrated that motor neuropathy caused by VZV is an inflammatory demyelinating process[13].

Treatment of segmental zoster paresis, as in general HZ infection, is helpful when administered as a combination of antiviral drugs, glucocorticoids, neuraxial block, and physical therapy. Early administration of antiviral drugs is important to decrease the incidence of motor complications and degree of pain[14,15]. The early administration of glucocorticoids could decrease demyelination of the involved nerve segments and prevent degeneration of axons[16]. Kinishi *et al*[17] reported that therapy with acyclovir with a high dose of steroids was proven to maintain nerve function in good condition, as evidenced by the nerve excitability test. Moreover, in the Ramsay Hunt syndrome, the therapy resulted in an improved recovery rate of the facial nerve. Other researches have reported that, within two weeks after initial symptoms, nerve block procedures can decrease severity of pain and the incidence of complications[18].

Regarding prognosis, the ultimate recovery of motor strength is good in 70–80% of the cases, although this may take a few months to few years[19]. Although the patient in this case belonged to the high-risk group, it is important to note that she fully recovered

after approximately 17 mo of early treatment with antiviral agents, glucocorticoids, and neuraxial blocks.

CONCLUSION

Segmental zoster paresis, depending on the affected area, can present with severe clinical manifestations and render patients unable to carry out the activities of daily living. However, through prompt administration of optimal treatments, such as in this case, a good prognosis can be expected. MRI may play a useful diagnostic role in segmental zoster paresis as well as patient evaluation after recovery from motor complications.

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