

RELATO DE CASO

Zoster Paresis: An Underrecognized Cause of Acute Monoparesis

Paralisia por Zoster: uma Causa de Monoparesia Aguda Mal Identificada

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Herpes zoster (HZ) is an infectious disease caused by the reactivation of the varicella zoster (VZ) virus in dorsal sensory ganglia. Its incidence and severity increase with age. After an acute phase of the illness, VZ virus lies dormant in the cranial nerve nuclei, dorsal root and autonomic ganglia of the entire neuroaxis. Viral reactivation may occur decades later. The virus travels to the skin through the sensory roots causing the characteristic vesicular rash within the corresponding dermatome. It spreads to involve the nerve roots adjacent to the dorsal root ganglia causing plexitis and neuritis¹.

Immunocompromised patients are at risk for disseminated cutaneous infection and for serious visceral complications. The incidence of HZ in patients following solid organ transplantation has been reported as 8.6%². In liver transplant recipients the frequency of VZ virus infection has been reported as 1.2-18%^{3,4}.

Acute monoparesis secondary to HZ virus infection is frequently misdiagnosed. The recognition of this disorder may avoid unnecessary laboratory investigation and inappropriate treatment.

Case report

A 64-year-old man was admitted to our ward because of vomiting, fever and abdominal pain. Medical history revealed that he was diagnosed as having cirrhosis due to hepatitis C infection and was submitted to liver transplantation six years earlier. Chronic renal insufficiency developed due to cyclosporine toxicity six months before admission. Initial laboratory work-up revealed hyperkalemia and he was treated with hemodialysis. Three days later, he developed a sudden onset of lumbar pain, left lower-limb pain and distal paresis with foot drop (Figure 1). On neurological examination, hip abduction and adduction were graded 4/5, hip flexion and extension 3/5, knee extension, dorsiflexion and great toe extension 2/5. He had a hypoactive left patellar muscle stretch reflex and an absent left



Figure 1. Left foot drop and herpetic scar lesions on dermatomes L2-L5.

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Achilles reflex. His right lower limb was entirely normal with regard to strength, sensation and muscle stretch reflexes. The remainder of his neurological examination was unremarkable. Shortly after the onset of pain, itchy erythematic and painful vesicles on left L2, L3, L4 and L5 dermatomes developed. The clinical diagnosis of VZ virus infection was made.

Cerebrospinal fluid analysis showed lymphocytic pleocytosis with high protein levels and normal glucose. Lumbar resonance imaging revealed no radicular involvement. Electromyography (EMG) showed L2-L3-L4-L5 radiculopathy and an axonal sensory-motor polyneuropathy. On laboratory work-up, the patient exhibited marked increase in liver enzymes (aspartate aminotransferase of 813 UI and alanine aminotransferase of 956 UI) possibly indicating a disseminated form of VZ virus infection. The patient was treated with intravenous Acyclovir (10 mg/kg/dose tid) for seven days followed by oral Acyclovir 2g/day for 14 days (doses were adjusted according to patient's renal function). He evolved with pain remission after gabapentin (1200 mg/day) and amitriptyline (75 mg/day) and an intensive rehabilitation program. In a nine-month follow-up visit, the patient was ambulating independently but remained with a left foot drop. Pain releasing drugs were no longer necessary.

Discussion

Segmental zoster paresis is characterized by focal motor weakness that appears in the same segment where the skin eruptions occur, and is a relatively rare complication⁵. The reported estimate of segmental limb paresis secondary to VZ virus infection is 3 to 5%^{5,6}. Kawajiri and co-workers⁵, in their review of literature, reported that the mean age of onset is approximately 70 years and weakness most commonly occurs within two weeks after the onset of pain or skin rash. In the lower limbs, proximal weakness (L1-L4 segments) is more commonly reported⁵. Electrophysiological studies are useful for the correct diagnosis and evaluation of the extent of lesions. EMG generally shows abnormal spontaneous activities, such as fibrillations and positive sharp waves, in clinically weak muscles. These activities become apparent two weeks from the onset and persist for about one to three months. A study using EMG showed that positive sharp waves and fibrillations are detected in 40 to 50% of cutaneous zoster patients, suggesting that subclinical motor involvement is not

uncommon^{1,7}. The prognosis of VZ virus paresis is generally good. More than half of the patients show complete functional recovery, and 75% of the patients show complete or partial recovery within one to two years⁸.

Our patient presented with immunosuppressive diseases which elicited an acute VZ virus infection characterized by its dermatomal vesicular rash and a left L2 to L5 radiculopathy on electromyography, which led to left foot drop as a sequela. It is an uncommon and under-recognized feature of VZ virus infection. Our patient presented also a chronic axonal sensory-motor polyneuropathy secondary to his various comorbidities and which was not responsible for his recent symptoms. In spite of the skin vesicles and all clinical setting, the diagnosis of VZ is only presumptive and one may consider differential diagnoses particularly in a patient with immunosuppression, hepatitis C and renal insufficiency.

Physicians should be aware of the possibility of monoparesis when encountering patients with lesions suggestive of VZ virus infection. Therefore, zoster paresis must be included in the differential diagnosis of acute onset paresis.

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