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Monoparesis by Herpes Zooster in an Immunocompetent Patient - Case Report

Keywords: *Varicella Zoster*; Immunocompetent; Foot weakness

Abstract

Herpes Zoster is caused by the *Varicella Zoster* virus, characterized by a unilateral vesicular rash, usually restricted to a specific dermatome.

It occurs by reactivation of the latent virus in the dorsal spinal cord ganglion, normally associated with isolated sensory changes. Motor deficits and muscle atrophy of the corresponding myotome are rare manifestations of this pathology.

We present a case of Herpes Zoster neuropathy in the L5-S1 territory, with early presenting motor dysfunction.

Open Access Case Report

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Introduction

Varicella Zoster virus (VZV) can have two clinical presentations, varicella (chickenpox) or herpes zoster (shingles) [1].

Herpes zoster is characterized by a painful and unilateral rash, usually restricted to one dermatome, although it can affect adjacent ones, usually in immune-compromised patients. It has a reported incidence of 10-20%, with higher rates in the elderly and immuno suppressed population [2].

Despite its rarity, the virus can cause motor neuropathy in 0.5-5% of patients [3]. This atypical presentation can occur by several mechanisms: 1- Infection progression to the anterior horn through the motor nerve root; 2 - segmental myelitis; 3- secondary degeneration of the motor nerve root; 4 - Simultaneous inflammation of the motor and sensory system [4].

The diagnosis is based primarily on the history and clinical findings such as pain, skin rash and development of neurological deficits in the corresponding territory [5].

Electromyography is an important accessory method to confirm the diagnosis, revealing alterations in the motor evoked potentials in muscle groups [6].

The treatment described for these cases involves pain control, anti-viral medication and physical therapy, with a good prognosis in the majority of the patients [3,6].

Clinical Case

A 65-year-old man visited the Orthopedics Department with right foot weakness, without lumbar or sciatic pain. A week later he developed right-sided petechiae in the L5-S1 dermatome (Figure 1). The patient had no relevant pathology or medical history of surgeries/trauma.

He was unable to dorsiflex the right hallux and ankle (2/5), with abolition of the Achilles tendon reflex (S1) and dysesthesia in the lateral side of the right leg and foot. The remaining neurological examination was normal.

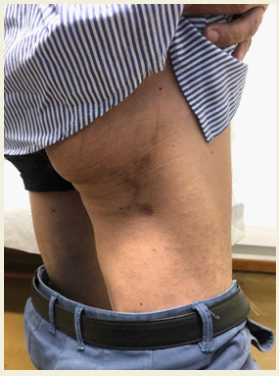

Figure 1: Skin lesions on L5-S1 dermatome.

We performed a lumbar MRI that excluded the differential diagnosis of radicular compression that could be responsible for the neurological deficits.

A histopathological analysis of the dermal alterations was performed, with positive *varicella-zoster* staining. Serological tests for HIV, Lyme disease, Hepatitis B and C, CMV, Epstein Barr and Syphilis were negative on serum samples.

The electromyogram revealed severe acute muscular denervation on the L5/S1 myotomes, compatible with severe radicular ganglionic neuritis and/or neuropathy of the common sciatic trunk.

Based on these results, a multidisciplinary group meeting was created with the departments of Orthopedics, Neurology and Physical Medicine and Rehabilitation to define the treatment of the patient. We decided for pharmacological treatment with 10 days of Valaciclovir and 3 months of Pregabalin, associated with an intensive physiotherapy regimen for 4 months. During the follow-up consultations, there was a complete recovery of muscle strength (5/5), despite maintained dysesthesia in the foot. A new electromyography demonstrated a mild to moderate proximal axonal sequel lesion of the right sciatic nerve.

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Discussion

Focal segmental paralyzes are described in up to 5% of Herpes Zoster cases, occurring in myotomes corresponding to dermatomes with skin rashes [5], and usually occur 2 to 3 weeks after the beginning of skin manifestations [8], which statistically highlights the rarity of our case.

Yoleny, et al. emphasize the underestimation of these values since the majority of patients develop herpes zoster in regions of the facial nerves, high cervical (C2-C3) nerves or dorsal roots, regions in which motor involvement is more difficult to detect.

In the described cases of non-segmental paralysis, diabetic neuropathy has always been implicated [7], a comorbidity that is duly excluded in the case presented here.

Aging appears to be the most common cause of viral reactivation, via weakening of the immune system. It is important to search for other causes of immunodepression, like HIV or immunosupressive drug therapy. Our patient was screened for these main comorbidities, but only tabbaco use was found.

The exact pathophysiology of peripheral neuropathy caused by herpes zoster is uncertain, but migration of the dorsal spinal ganglion infection to the nerve root and peripheral nervous system has been documented [1,6,7].

Saxena, et al. reported a case in which motor neuropathy precedes skin changes, underlining the uncommonness of such occurrence.

Wedling et al. described cases of foot drop that preceded the rash, and their results [9], together with those of Saxena, et al. seem to point to a worse prognosis with this early presentation, results which, fortunately, are not in agreement with our case [3].

This pathology is usually cured without complications, with a very favorable prognosis in approximately 75% of cases [2,4].

Conclusion

Motor neuropathy following Herpes Zoster reactivation is a rare entity, especially when the neurological deficits precede the skin presentation. It is paramount to include this pathology in the differential diagnosis of the patients who attend our consultations with *ad initium* neurological deficits.

Strong clinical suspicion and a multidisciplinary performance are important to minimize complications and optimize results.

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