

Motor radiculopathy in Herpes Zoster: a case report

A. C. Rubicondo., S.Grioli .,D.Restivo ., F. Matta ., G. Vitale., E. Saracco., L Marturano .,A.Ignoto., A. Pavone .

U.O.C. NEUROLOGIA
ARNAS "Garibaldi" CATANIA

Case Presentation

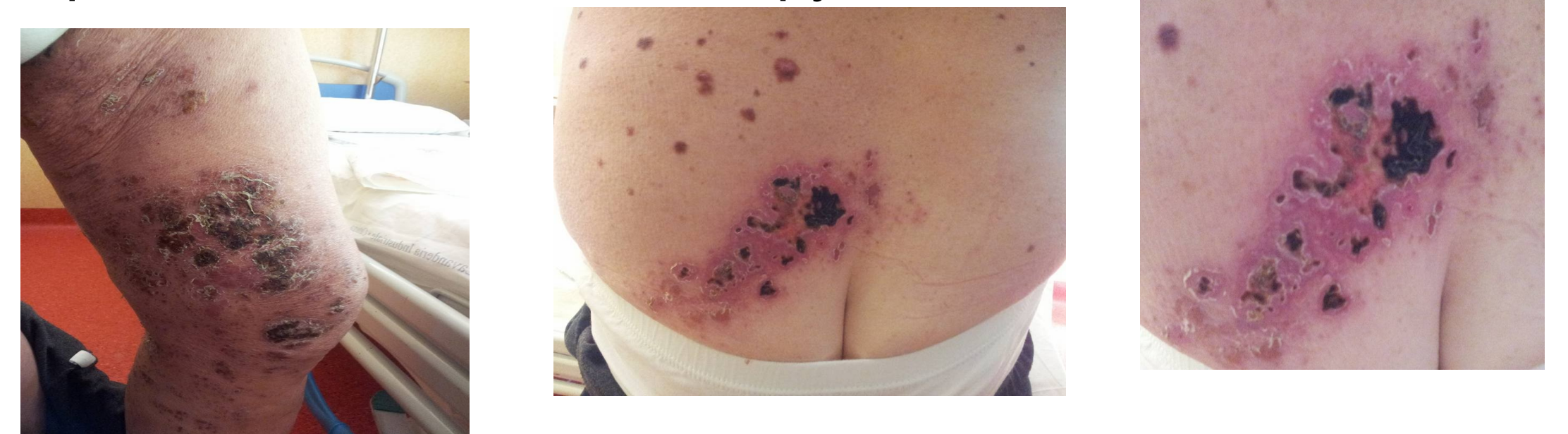
We are presenting a the case of a 67-year-old man who has been suffering for some years from hypertension and diabetes mellitus treated with metformin . One month before being admitted to the clinic, he had experienced an outbreak of a red vesicular rash on the lateral aspect of his left upper and lower leg and in the left lumbar region, involving the L3 dermatomes. He was treated for herpes zoster infection with acyclovir. Four weeks after he was seen in the neurology clinic for a weakness in the left leg as well as pain and numbness around the area of the rash. An examination revealed that he had multiple scarred lesions over the area involved. He had reduced movements in the left knee and thigh joint . Patellar reflex was reduced on the affected side. Sensation was also significantly reduced on the affected side in the dermatomal distribution of L3 on left. Nerve conduction study and electromyography findings were compatible with deficit innervations preganglionic root L3 territory as from herpes zoster motor radiculopathy. He continued the therapy with acyclovir and was referred to a physiotherapists, and this led to an improvement of symptoms. Varicella-zoster virus, a member of the herpes virus family, is a neurotropic virus that primarily affects afferent sensory neurons. Although herpes infection with motor nerve involvement may be infrequently found, it is not rare and has been reported as early as 1949. Unless the immune system is compromised, it suppresses reactivation of the virus and prevents herpes zoster. Why this suppression sometimes fails is poorly understood, but herpes zoster is more likely to occur in people whose immune system is impaired due to ageing, therapy and psychological or other factors. Reactivation of latent virus within the dorsal root ganglion and axoplasmic transport to epithelial nerve terminals causes the segmental cutaneous rash and neuralgic pain characteristic of herpes zoster. It is still unclear the reason why motor paresis may occur. However, it is postulated that the virus spreads proximally as well as distally, causing a local neuritis in the spinal nerve and subsequently gaining access to the motor axons. I think this is an important case study because radiculopathy is a rare complication of herpes zoster.

Discussion

Varicella-zoster virus, a member of the herpes virus family, is a neurotropic virus that primarily affects afferent sensory neurons. Although herpes infection with motor nerve involvement may be infrequently encountered, it is not rare and has been reported as early as 1949. Unless the immune system is compromised, it suppresses reactivation of the virus and prevents herpes zoster. Why this suppression sometimes fails is poorly understood but herpes zoster is more likely to occur in people whose immune system is impaired due to ageing, therapy, psychological or other factors. Reactivation of latent virus within the dorsal root ganglion and axoplasmic transport to epithelial nerve terminals causes the segmental cutaneous rash and neuralgic pain characteristic of herpes zoster. It is unclear as to why motor paresis may occur; however, it is postulated that the virus spreads proximally as well as distally, causing a local neuritis in the spinal nerve and subsequently gains access to the motor axons. Only 5–30% of patients with typical cutaneous lesions developed some form of motor weakness affecting the myotomal muscles corresponding to the dermatomal distribution of skin lesions. Three-quarters of those with zoster paresis are more than 50-year-old. These patients and immunocompromised individuals are at increased risks for severe complications. The weakness usually develops within 2–3 weeks of skin eruptions but varies from an hour to a month. The prognosis of herpes zoster-related paresis is good, with more than half the patients showing good recovery. The learning point from this case report would be to suspect herpes zoster radiculopathy when immunosuppressed patients present cutaneous herpes zoster infection following to a motor weakness. In patients with herpes zoster radiculopathy the incidence of zoster paresis and the progression and severity of electrophysiological changes have been found to decrease in patients with antiviral therapy.

Conclusion:

Herpes zoster radiculopathy should be suspected when immunosuppressed patients present motor weakness followed by a cutaneous herpes zoster infection. I think this case is important because radiculopathy is a rare complication among herpes zoster cases.



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