Segmental Zoster Paresis: A Case Report on Zoster-Associated Plexopathy Following Varicella Zoster Reactivation

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BACKGROUND

Segmental Zoster Paresis (SZP) is a rare motor complication of shingles (0.5–5% of cases), involving VZV-induced inflammation of the spinal cord, nerve roots, plexus, or peripheral nerves, causing focal weakness matching the rash's dermatome. Onset averages 3.5 weeks post-rash (range 1–8 weeks), often after the rash resolves. Most commonly affects the facial nerve, then upper (C5-7) then lower (L1-4) extremities. About 67% recover spontaneously over 6–9 months. Management is typically conservative.

OBJECTIVE

To present a case that highlights the utility of electrodiagnostic studies combined with careful history taking in SZP

CASE DESCRIPTION

A 67-year-old woman with type 2 diabetes, hypertension, hyperlipidemia, and asthma developed progressive right upper extremity weakness, pain, and muscle atrophy four weeks after a C8-dermatome herpes zoster rash (treated with a five-day valacyclovir course). Exam showed atrophy of the right forearm and intrinsic hand muscles, healed vesicles on the medial palm, decreased sensation in the thenar eminence and digits 4–5, mild weakness (4/5) in triceps/wrist extensors, and severe weakness (1/5) in finger flexion, abduction, and adduction. Electrodiagnostic testing showed prolonged latencies and reduced amplitudes on median and ulnar sensory studies (post-ganglionic lesion), absent CMAPs in median/ulnar intrinsic hand muscles, abnormal ulnar F-waves (proximal pathology), and diffuse fibrillations on EMG across median, ulnar, and radial-innervated muscles—consistent with a plexus-level injury.

RESULTS

Cervical spine MRI showed congenital stenosis and disc bulges without nerve root or cord compression, excluding radiculopathy. Given the dermatomal rash history and electrodiagnostic findings, the patient was diagnosed with SZP due to zoster-associated plexopathy. Since antiviral treatment was already completed, she was started on gabapentin and tizanidine and referred to physical therapy. At six months, pain had improved, but motor deficits remained largely unchanged.

CONCLUSIONS

SZP is an uncommon, debilitating shingles complication requiring careful history-taking and clinical judgment for proper diagnosis and treatment. This case highlights electrodiagnostic studies as key tools for localizing the injury, distinguishing SZP from more common conditions, and gauging severity of deficits. While MRI isn't required for diagnosis, it can serve as a helpful adjunct. Prompt diagnosis and localization offers a more accurate prognoses, initiate targeted therapy, ensure adequate pain control, and maximize functional recovery

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