Segmental motor paralysis of the right upper limb in herpes zoster

Sir,

Herpes zoster is due to reactivation of varicella-zoster virus lying dormant in the dorsal root ganglion. It is characterized by pain or burning sensation in the involved dermatome with a grouped vesicular rash. In herpes zoster, acute inflammatory changes occur in the posterior nerve root and ganglia. Among the complications of herpes zoster, involvement of motor neurons is rare and may lead to paresis or complete paralysis of the muscles supplied by the affected segments. We are reporting a case of unilateral right C4, C5 and C6 segmental motor paralysis attributable to herpes zoster in an otherwise healthy individual for its rarity.

A 70-year-old man had severe pain and multiple grouped vesiculobullous eruption over erythematous background along $C_{4,5,6}$ dermatomes since 5 days. Seven days after the onset of rash, he noticed inability to move his right upper limb. The patient was not a known diabetic or hypertensive. On examination of the right upper limb, there were absolutely no movements of the right shoulder joint. In the right elbow joint, the power of the muscles supplied by $C_{4,5,6}$ segments was grade zero. Other systemic examination was normal.

Routine hematologic, biochemical, urine and stool examinations were within normal limits. Blood VDRL, HIV were negative. Radiographs of the cervical spine (anteroposterior, lateral and oblique views) were normal. We could not perform electromyogram and nerve conduction studies due to lack of facilities. The patient was treated with oral acyclovir 800 mg, five times per day, for 10 days for herpes zoster along with symptomatic care. The skin lesions healed slowly in about 3 weeks. Patient was seen by a physician and was advised physiotherapy. However, muscle power recovered gradually at the end of 5 months. He was able to flex and extend his elbow, with recovery of shoulder movements.

Broadbent first described herpes zoster related paralysis in 1866. About 5-30% patients^[1] with typical cutaneous lesions develop some form of motor weakness affecting the myotomal muscles corresponding to the dermatomal distribution of skin lesions. The weakness usually develops within 2 to 3 weeks after the onset of the skin eruption. Its onset is abrupt, occurring over hours or 1 to 2 days, with little or no subsequent deterioration. Zoster paresis occurs more often in the upper than in the lower extremity, preferentially segments C5 and C6.^[1] Segmental motor paralysis of the limbs (SMP) complicates 2-3% of the cases of cutaneous herpes zoster. Viral invasion and inflammation of the motor neurons of the anterior horn cells by the Varicellazoster virus (VZV) causes clinical weakness at the same time and site as the cutaneous eruption. VZV should be considered amongst the etiologies of SMP, even in the absence of cutaneous lesions (zoster sine herpete).^[2] During reactivation of virus, direct invasion of adjacent structures may occur. This may lead to involvement of the anterior horn neurons, resulting in muscular weakness, cranial nerve palsies, diaphragmatic paralysis, neurogenic bladder and colon pseudo-obstruction. Rarely, VZV infection may result in myotomal motor weakness or paralysis in addition to a painful dermatomal rash.^[3] But limb muscles paralysis is rare.^[4]

The cause for motor paresis is unclear. It is postulated

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Herpes zoster affects 10-20% of the general population.^[5] While the majority of complications manifest as sensory disturbances, 5% are motor neuropathies, with half involving the cranial nerves.^[5] The motor system may be affected in the form of paresis or paralysis of the muscles. A patient with herpes zoster of the C4, C5 and C6 dermatomes developed left upper arm monoplegia. Brachial plexus neuritis may be a direct cause of reversible upper limb paresis in herpes zoster.^[6] Our patient had complete paralysis of the muscles supplied by the C4, C5 and C6 segments. The cause of muscle paresis in herpes zoster is unclear. It may probably be due to myositis or ganglio-muscular disease following infection of proprioceptive ganglion cells.^[7]

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Karjigi Siddalingappa, K. Lokanatha Department of Skin and STD, VIMS, Bellary, India.

Address for Correspondence: Dr. Karjigi Siddalingappa, Department of Skin and STD, VIMS, Bellary, Karnataka 583 104, India. E-mail: karjigi267450@yahoo.com