Disseminated Zoster with Polyneuritis Cranialis and Motor Radiculopathy: Letter to Editor

Disseminated zoster is defined as zoster involving one dermatome and at least 20 vesicles outside the primary and immediate adjacent dermatomes. In 10-15% cases, cutaneous dissemination of lesions is seen after onset of dermatomal lesions. In 1/3rd of these patients, nervous system involvement is seen in the form of cranial nerve palsies or encephalitis. Motor neuropathy, myelitis, myositis and delayed cerebral angiitis, is rare.

A 48-year-old male presented to the skin department with vesiculobullous lesions on the left side of forehead with orbital edema and was diagnosed as herpes zoster ophthalmicus and treated with oral acyclovir. Two days later, cutaneous lesions of varicella appeared all over and the patient developed increased bilateral orbital edema. On third day, he developed difficulty in swallowing, hoarseness of voice and throat pain. This increased to total inability to swallow both solids and liquids from day 6. The patient was first seen in the neurology department a week after onset of illness. On examination, he had small follicles on right tonsil and slough on left tonsil. His higher mental functions were normal except for dysarthria. Cranial nerve examination revealed left herpes zoster ophthalmicus, left infranuclear facial palsy, left palatal palsy and left sternocleidomastoid weakness. Motor, sensory examination was within normal limits. All deep tendon reflexes were present, and plantars were flexor. Two days later, he developed bilateral ptosis, left internal and external ophthalmoplegia in addition to the previous deficit. Patient was advised injectable acyclovir (after the onset of neurological symptoms) but could not afford the same. On day 14, he developed radicular pains in both arms, along with weakness of arms abduction (grade III) and left triceps (grade IV). The left biceps, triceps and supinator reflexes were absent. There was no sensory deficit. He could afford injectable acyclovir only after 14 days, which was given for 10 days. There was no further progression of neurological symptoms. On follow-up, 3 months later all neurological complications had improved. Only residual left corneal opacity with congestion remained.

The trigeminal nerve is the most common extraspinal nerve affected by Herpes zoster. Involvement of the ophthalmic branch of trigeminal nerve is seen in 7% of all cases of zoster. Involvement of nasociliary division in ophthalmic zoster is associated with high complication rate. Our patient developed conjunctivitis, scleritis, keratitis, bilateral ocular motor palsy even though Hutchinson’s sign was negative. It is interesting to note that ocular motor palsies developed after lower cranial nerve involvement. The mechanism of involvement of cranial nerves in Herpes zoster is possibly the spread of virus along trigeminal and other ganglionic afferent fibres to small vessels that supply cranial nerves. The cranial nerves also receive afferent fibres from trigeminal or other ganglia. Motor weakness follows dermatomal zoster in 1-5% of zoster patients. Paralysis usually occurs within first 2-3 weeks after onset of rash, peaks in severity within days and can persist for several weeks. Paralysis frequently involves muscle groups innervated by nerves, contiguous with those primary dermatomes. Although peripheral motor weakness commonly leads to muscle atrophy, about 75% of patients with motor involvement achieve complete or functional recovery as in our patient.

Extensive cranial neuropathy involving 3rd, 4th, 5th (ophthalmic division) 6th, 7th, 9th, 10th, 11th cranial nerves and cervical radiculopathy (C5-7) in a case of disseminated zoster has not been reported.

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References