

SHORT REPORT

Focal weakness following herpes zoster

O C Cockerell, I E C Ormerod

Abstract

Three patients presented with focal weakness of an arm which followed segmental herpes zoster affecting the same limb. Neurophysiological investigations suggest that the site of the lesion lay at the root, plexus, or peripheral nerve level. This reflects the various ways in which the virus may affect the peripheral nervous system.

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Motor involvement following herpes zoster was first described in 1866.¹ Since then there have been numerous reports of paralysis affecting the limbs, cranial nerves, sphincters, intercostal, abdominal, and diaphragmatic muscles.²⁻¹⁰ These complications may occur in up to 31% of cases of herpes zoster,^{10,11} but in only 5% are the limbs affected.⁷⁻⁹ Despite the frequency of this condition most of the reports are now over 20 years old and the latest series 16 years old. Only one study attempted full nerve conduction studies.⁹ We report three patients who exhibited different forms of this complication and review the literature on the subject.

Case reports*Case 1*

A 58 year old woman presented with pain in her right shoulder and arm for three weeks. One week later she developed a rash on the outer border of the arm, also involving the thumb and the index finger. One week after this she noticed weakness in the right arm which progressed over several days and was associated with impaired sensation over the outer border of the arm in the distribution of the rash. She also had bronchiectasis, quiescent pulmonary sarcoidosis, and hypertension.

There was a fading rash, typical of shingles, on the right arm affecting dermatomes C5 and C6. The scabs had all separated. There was marked weakness of the right deltoid, supraspinatus, and biceps. There was slight weakness of infraspinatus and extensor carpi radialis. There was sensory loss to pinprick, light touch and temperature in dermatomes C5 and C6. The median and ulnar

nerve motor conduction studies and sensory nerve action potentials (SNAPs) were normal. The F waves proximal conduction velocity was 66 m/s and the latency of the motor response in the biceps from stimulation at Erb's point was 3.9 ms. Three months later there was minimal residual weakness of the deltoid, biceps and the spinati and some mild sensory change in the C6 dermatome.

Case 2

An 86 year old man had an attack of shingles affecting his right arm over the C5, C6, and C7 dermatomes. The rash and pain resolved after two weeks, but his right arm started to become weak and worsened over six days until he was unable to use the limb at all. There was global wasting of all the muscles of the right arm. He had marked weakness of deltoid, supraspinatus, and infraspinatus, and distally power was absent. The right biceps, triceps, and supinator tendon reflexes were all absent. Sensory testing revealed a patch of decreased sensation to light touch and pinprick over the C4-T1 dermatomes on the right. Nerve conduction studies revealed no SNAPs recordable from the right median or ulnar nerves, and the ulnar mixed nerve action potential from the right wrist was also absent. SNAPs from the left hand were normal (amplitude of the median-18.4 μ v, and ulnar-10.4 μ v). No compound motor action potentials (CMAPs) were obtained from abductor pollicis brevis (APB), or from abductor digiti minimi (ADM).

Small CMAPs were obtained from the right biceps and triceps with stimulation at Erb's point, and these were moderately delayed (Erb's point to triceps latency—12.1 ms), and dispersed. Needle EMG showed complete denervation of right APB and ADM with partial denervation of right biceps and triceps. He was treated with oral acyclovir. Six months later he had made an almost complete recovery.

Case 3

A 63 year old man had an episode of shingles over his left arm, mainly on the tips of his fingers and the thenar pad. Two weeks after the onset of the rash he experienced weakness of his left hand which progressed over a two week period. He was diabetic, well controlled on diet and metformin. He also had hyper-

Department of
Neurology, St
Thomas' Hospital,
London SE1 7AH, UK
O C Cockerell
I E C Ormerod

Correspondence to:
Dr Cockerell, Epilepsy
Research Group, NSE,
Chalfont St Peter, Bucks
SL9 7BR, UK

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tension. Two months later there was thinning of the ulnar border of the left forearm and the ulnar innervated intrinsic muscles of the left hand. There was weakness of flexor digitorum profundus to the ring and little fingers as well as ADM. APB was normal. There was a deficit of pinprick and light touch sensation over the ulnar border of the left hand. Nerve conduction studies showed slowing of the motor conduction velocity (20m/s) along the entire length of the left ulnar nerve from axilla to wrist, with a low amplitude CMAP recording from ADM at the wrist (0.341 mv), below the elbow (0.326 mv), above the elbow (0.334 mv), and axilla (0.294 mv). The right ulnar nerve had less marked slowing of the motor conduction velocity (40 m/s) and normal amplitude CMAP recording from ADM (8.1 mv). The SNAPs from the right sural and right median nerves were of low amplitude (2.8 μ v and 1.8 μ v respectively) but conduction velocity was normal. No SNAPs were recorded from either of the ulnar nerves. There were prolonged minimum F-wave latencies in the ulnar nerves recording from ADM stimulation at the wrist (39.4 ms right and 40.5 ms left). Six months later only minimal improvement had occurred.

Discussion

The clinical picture of the first two cases demonstrated the previously well described pattern of focal weakness following a zoster rash, affecting dermatomes of the same segmental level within two months of the onset of the rash. In case 1 the location of the pathology in the motor supply to the arm remains unclear. The clinical picture was of a lower motor neuron lesion affecting the C5 innervated muscles. The electrophysiology was unhelpful. The normal motor conduction studies (including stimulation at Erb's point, and F-wave responses) gave no support to a diagnosis of a neuropathy, a plexopathy, or a motor radiculopathy. The cervical myelogram was normal and other causes of a radiculopathy, apart from herpes zoster, were excluded by appropriate investigations, although a lesion affecting the anterior horn cells in the cord is possible. In case 2 the motor involvement was more extensive than the preceding zoster rash and the weakness and wasting more profound. The SNAPs showed evidence of a post-ganglionic lesion and the slowing across Erb's point provided good evidence for a brachial plexopathy. Nevertheless he achieved a good eventual recovery, although he was the only patient to receive a full course of acyclovir. The third case was difficult to disentangle as the patient also had diabetes, and the nerve conduction studies showed a mild generalised axonal neuropathy, however, the close temporal and anatomical relation of the zoster rash to the hand weakness makes zoster the likely cause. The nerve conduction studies demonstrated abnormalities in the ulnar nerve along its whole length rather than at the elbow (the usual site for a diabetic ulnar nerve palsy),

and the neuropathy in this territory was significantly worse than the mild subclinical changes found elsewhere. The site of the lesion here is uncertain, but with an absent SNAP and generalised slowing of motor conduction along the ulnar nerve, the abnormality could not be explained by a radiculopathy alone.

The latency between the rash and the development of limb weakness has been reported in the literature to range from one day to four months,^{6,7,10} with the majority of cases developing weakness within a 2 week period.⁷ This is in accordance with this report. In all our cases the zoster rash affected the arms and was in the same anatomical distribution as the paralysis. This is generally observed but topographical dissociation has been described in up to 10% of cases.^{5,7} Segmental weakness of the arms and legs is more common than thoracic weakness.¹⁰ It is not clear why this is as the zoster rash alone is more common on the thorax. It may be that intercostal motor involvement is less well detected clinically.

It is difficult to localise the site of zoster motor involvement. In our first case the normality of the nerve conduction studies suggested that the level of involvement was at the root or cord level, whereas in the second case the evidence was more in favour of brachial plexus involvement. The last case proved more difficult but the brunt of the damage was to the ulnar nerve. Although previous studies have reported on cases with the clinical features of root,⁵⁻⁷ plexus,⁵⁻⁷ and one case of an ulnar neuropathy with less severe median nerve involvement,⁹ only two of these reports were accompanied by neurophysiological evidence.^{6,9} In only one study were nerve conduction studies reported,⁹ some of their patients had evidence of denervation but no localisation of the damage was achieved.

The zoster rash is produced when latent virus in the sensory ganglia becomes reactivated and spreads to the skin. Virus particles are present in the sensory root ganglia,¹⁰ and pathologically there is an intense local ganglionitis.¹² The virus may also cause Wallerian degeneration¹² and local neuronitis due to extension of the inflammation from the ganglion down the nerve itself.¹⁰ The localised damage caused by the virus does not explain the cases where there is anatomical dissociation of rash and motor involvement either at the segmental cord level, or those cases with ophthalmic zoster and ophthalmoplegia.¹¹ Central spread of the virus to the posterior horns and roots,¹² leptomeninges,¹³ and anterior horn cells,¹⁴ is well described and probably explains these apparent discrepancies.

The prognosis for zoster segmental motor weakness is generally favourable with complete recovery in 55–75% of cases.^{6,7,11} More distal paralysis is associated with a worse prognosis,^{7,11} and there appears to be no relation between the degree of paralysis and eventual recovery.¹¹ This is in accord with our first two cases. The bad prognosis in case 3 may be related to the predominant nerve, as

opposed to root or plexus, involvement. The other report of ulnar neuropathy also had a comparatively poor outcome.⁹ This may relate to a different type of damage with direct invasion by the virus down the motor root which has been suggested before,¹⁰ but never proven. It would be difficult to explain the phenomenon of a single nerve being affected if the neuropathy were secondary to Wallerian degeneration from either ganglionitis or alpha horn cell involvement.

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