

Case Report**Post-herpetic Brachial Plexopathy : Diagnosis and MRI Findings**Anuja Sagdeo¹**ABSTRACT**

Herpes zoster or shingles is the reactivation of a dormant varicella zoster virus (VZV), typically in the dorsal root ganglia. It usually manifests with sensory features of pain, paraesthesia and vesicular rash in the affected dermatome. Less commonly, other neural structures are affected. This is a report of a case of brachial plexopathy highlighting the differential diagnosis, investigation, treatment and prognosis of this uncommon but important condition.

Key Words : Herpes zoster, shingles, brachial plexopathy

Case Description :

A sixty three year old female presented with progressive weakness in the left arm and hand, worsening over 10 days. A diagnosis of shingles had been in Primary Care and weakness appeared a week after the rash. Subsequently, the rash settled though pain persisted and low dose Amitriptyline was started.

Past medical history included hypertension, knee osteoarthritis, dyspepsia, erythema nodosum and tonsillectomy.

Three weeks later patient was seen in ambulatory care clinic with weakness in left arm. Examination revealed a flaccid, diffuse, areflexic weakness, without wasting, affecting elbow flexion and extension, wrist & finger extensors, long finger flexors, and all intrinsic hand muscles in the left upper limb. Power at the shoulder and periscapular muscles was preserved. Sensory impairment to light touch and pin prick was present. There was no clinical evidence of Horner's syndrome and rest of the neurological examination was unremarkable. Initial management included IV Acyclovir. Routine investigations such as full blood count, renal and liver function, inflammatory markers, blood cultures, HIV test were negative including serum viral screen. Cerebrospinal fluid (CSF) analysis was normal. CSF culture and oligoclonal bands were

negative. CSF PCR was negative for Herpes simplex 1 and 2, VZV.

Magnetic resonance imaging of the neck revealed increased signal in the left brachial plexus extending from the level of the exiting nerve roots. Cranial MRI scan was normal. The appearances were consistent with an inflammatory brachial plexopathy.

Once CSF Herpes PCR was known, acyclovir was discontinued and IV Methylprednisolone 500 mg OD for 5 days was commenced.

Her condition remained unchanged over the next 3 weeks. Oral prednisolone was commenced at an initial dose of 60 mg a day for 8 weeks then tapered over next 2 months. Seven months later, there was substantial motor improvement in all affected muscle groups and the sensory deficit had resolved with complete resolution of her pain.

Discussion :

Herpes zoster infection is common with a lifetime risk of 25-30%¹. Incidence increases with age and can necessitate admission to hospital². Occurrence may herald a drop in the virus specific cell mediated immunity³ and presentation with neuropathic pain and localised vesicular rash in a dermatomal distribution is typical.

Neurological complications may follow reactivation of latent varicella-zoster virus infection.

Recognised complications include myelitis, meningoencephalitis, Brown-sequard syndrome, plexitis, poly or mononeuritis, polyradiculitis and segmental zoster paresis⁴.

¹Acute Medicine Consultant
Royal Shrewsbury Hospital, United Kingdom

Address for Correspondence -

Dr. Anuja Sagdeo
E-mail : anujasagdeo@yahoo.co.in

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The incidence of involvement of motor function is reported to be around 0.5% to 3%⁵.

Risk factors for these uncommon manifestations include advancing age, diabetes and smoking.

This appears to occur from a direct invasion of the virus from the dorsal root ganglions to the anterior horn cells, which causes inflammation of the motor nerves resulting in motor deficits⁶.

Cases of brachial plexus involvement related to shingles are generally based on weakness following painful skin lesions. However the interval between the skin lesions and weakness ranges from days to months⁷. Chang et al describes an evolving motor paresis 3 days prior to the rash⁸, which may reflect a different process albeit linked to shingles than paresis weeks after skin lesions have resolved.

Pathological findings are described with histological evidence of extensive lymphocytic infiltration and myelin breakdown in the brachial plexus; and perivascular lymphocytic cuffing in the cervical spinal cord in a man with a left arm monoplegia developing 3 weeks after shingles in C4,5,6 dermatomes. This suggested that the motor neuropathy was due to an inflammatory demyelination process that recovers in most patients; and that the prolonged post herpetic neuralgia is due to an ongoing inflammation¹⁰.

The Magnetic Resonance imaging appearances (*Figure 1*) are consistent with plexitis / plexoradiculitis.

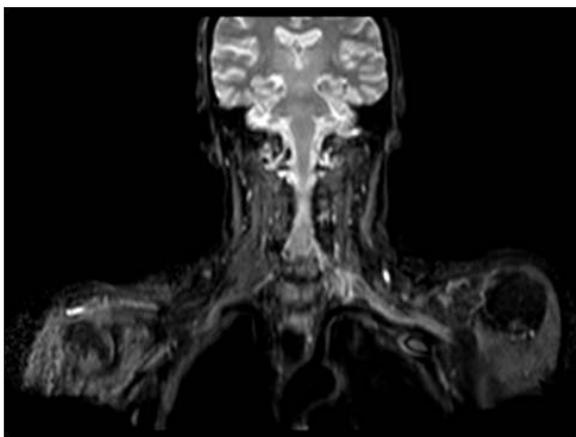


Figure 1 : MRI Image of Inflammatory Brachial Plexopathy (Arrow)

Other case reports described also the use of the nerve conduction studies to consolidate the findings of the MRI scan. Eyigor et al. published in 2006 a case report where there was a doubt about the diagnosis and the nerve conduction studies were used to confirm the MRI findings. The studies showed partial degeneration of the superior, middle and inferior trunks of the brachial plexus consisting with their MRI findings⁹.

In addition to the suggestive MRI and neurophysiological findings, an evidence of herpes zoster infection must exist to deem it as the cause of the plexopathy. This can be either the presence of the classic herpes zoster skin lesions or by confirming the infection by other means such as viral PCR, viral culture, or immunohistological analysis of skin scrapings. It is also important to exclude other potential causes of motor paresis, especially when there is a doubt about the diagnosis, such as brain lesions, MS or GBS. In this case, we performed Cranial MRI scan to exclude central structural causes, lumbar puncture to exclude central infections and Multiple Sclerosis / GBS and serum test to exclude HIV that can present in any form or shape.

Conclusion : Herpes zoster can involve the brachial plexus and affect motor and sensory function. Neurological manifestations after herpes zoster typically appear 2-3 weeks after development of rash. Magnetic Resonance Imaging (MRI) can support topographical diagnosis. In the absence of an agreed evidence base for optimal treatment, a multidisciplinary management approach, combining physiotherapy and occupational therapy with systemic Aciclovir, empirical steroids and analgesia appears appropriate.

References :

1. Johnson, R., Alvaerz-Pasquin, M., Bijl, M. and et al. (2015) Herpes zoster epidemiology, management, and disease and economic burden in Europe: a multidisciplinary perspective. *Therapeutic advances in vaccines*. 3(4), 109-120.
2. Peter H. F. Hobbelen, Julia Stowe, and et al. (2016) The burden of hospitalisation for Varicella and herpes zoster in England from 2004 to 2013. *Journal of Infection*. 73, 241-253.
3. BMJ Best Practice (2016) *Herpes zoster infection*. *British Medical Journal*. bestpractice.bmj.com/.
4. Brawerman DL, Ku A, Nagler W. (1997) Herpes Zoster Polyradiculopathy. *Arch Phys Med Rehabil*. 78, 880-882.

5. Lyneberg KK, Swensson BH. (1990) Herpes Zoster Paresis : A review of the literature and case reports. *Ugeski Laeger*. 152:1214-1217.
6. Mondelli M, Romano C, Della Porta P, Rossi A. (1996) Electrophysiological findings in peripheral fibres of subject with and without postherpetic neuralgia. *Electroencephaol ClinNeurophysiol*. 101:185-191.
7. Stowasser M, Cameron J, Oliver WA. (1990) Diaphragmatic Paralysis following cervical herpes zoster. *Med J Aust*. 153:555-556.
8. Chang CM, Woo E, Yu YL, Huang CY, Chin D. (1987) Herpes zoster and it's neurological complications. *Postgrad Med J*. 63: 85-89.
9. Eyigor S, Durmaz B, Karapolat H. (2006) Monoparesis with complex regional pain syndrome like-symptoms due to brachial plexopathy caused by the varicella zoster virus : a case report. *Arch Phys Med Rehabil*. 87: 1653-5.
10. Fabian VA, Wood B, Crowley P, Kakulas BA. (1997) Herpes Zoster Brachial Plexus Neuritis. *Clin. Neuropathol*. 16(2):61-64.
11. Choi JY, Kang CH, Kim BJ, Park KW, Yu SW. (2009) Brachial plexopathy following herpes zoster infection: two cases with MRI findings. *JNeurol Sci*. 285(1-2): 224-226.