CASE REPORT

Varicella zoster lumbosacral plexopathy: a rare cause of lower limb weakness

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SUMMARY

This is a rare case of Varicella zoster virus (VZV) lumbosacral plexopathy in an 84-year-old woman presenting with lower limb weakness and rash. Contrast-enhanced MRI showed enhancement of the left L3–L5 descending nerves and left lumbosacral plexus consistent with inflammatory/infectious aetiology. Cerebrospinal fluid PCR confirmed VZV DNA and cerebrospinal fluid serological testing was positive for VZV immunoglobulin (Ig)M and IgG antibodies. The patient was treated with intravenous acyclovir but this was complicated by the development of acute renal failure attributed to acyclovir-induced nephropathy, requiring dose adjustment. After a prolonged course of oral acyclovir and inpatient rehabilitation, the patient made a partial neurological and functional recovery.

BACKGROUND

This case report describes a rare entity of radiologically and laboratory confirmed Varicella zoster virus (VZV) lumbosacral plexopathy, highlighting the importance of evaluating clinical features such as progressive proximal motor weakness in the presence of a vesicular dermatomal rash, in the formulation of VZV as a differential diagnosis.

VZV plexopathy of any aetiology is a relatively uncommon entity and appears to more commonly inflict the brachial plexus. As such, there are a limited number of reports describing the clinical, imaging^{1 2} and electromyographical^{3 4} findings in VZV brachial plexopathy. Case reports describing clinical, imaging and cerebrospinal fluid (CSF) findings in VZV lumbosacral plexopathy are rare and comparatively incomplete.⁵

CASE PRESENTATION

This is a case involving an 84-year-old woman living independently at home alone. She was previously well with her only notable medical history including hypertension, hypothyroidism, bilateral hip osteoarthritis and previous L4/5 laminectomy 46 years prior for acute radiculopathic pain and weakness. She presented to the emergency department with 3–4 weeks of progressive left lower limb weakness which had acutely worsened over the last 24 hours, culminating in two accidental falls and precipitating her presentation to hospital. She also described worsening of chronic left hip pain and a 4-week history of a rash across her left leg which was now resolving. Systems enquiry revealed recent constipation and difficulty voiding urine.

Objective examination revealed a patchy erythematous rash with a mix of crusted and healed vesicles extending across the left L2–L5 dermatomes. There was patchy loss of sharp sensation in the L2–L4 dermatomes and moderately reduced power to L4/5 (Oxford scale) across all groups at the left hip and knee with preservation of ankle plantar and dorsi flexion. The plantar reflexes were downgoing bilaterally. On digital rectal examination, the patient had significant loss of anal tone.

INVESTIGATIONS

MRI of the lumbar spine was performed early in the patient's course of care, with a view to exclude spinal cord compression given the acute deterioration of the patient's leg weakness and concerning neurological findings. Contrast-enhanced T1-weighted MR imaging demonstrated enhancement of the descending left L3–L5 nerves in their intrathecal portion (figure 1A) and asymmetrical increased signal arising from the left lumbosacral plexus relative to the right (figure 1B). The left femoral nerve along the anterolateral margin of the psoas muscle also demonstrated high signal (figure 2). These radiological findings were deemed to represent a left lumbosacral plexopathy, likely of inflammatory or infectious aetiology.

The patient subsequently went on to have fluoroscopy-guided lumbar puncture which obtained clear, colourless CSF. Initial microscopy showed a raised leucocyte count of $549 \times 10^6/L$ of 100% mononuclear cells. PCR was positive for VZV DNA and her viral enzyme immunoassay serology was positive for both immunoglobulin (Ig)M and IgG VZV antibodies.

In this case, the imaging findings obviated the need for electromyographical testing. Had MRI not been performed initially, diagnostic electromyography in combination with serology would also have enabled establishment of the patient's diagnosis.

DIFFERENTIAL DIAGNOSIS

- The presence of a healing vesicular rash favours a zoster neuropathy.
- Acute spinal cord compression secondary to degenerative spondyloarthropathy, disc osteophyte complex or metastatic disease. MRI was valuable in excluding these acute compressive lesions.
- Plexopathy due to metastatic infiltration of lumbosacral nerves.

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Figure 1 Axial T1-weighted MRI with gadolinium showing enhancement of the descending intrathecal left L3–L5 nerves (A) and asymmetrical enhancement of the left lumbosacral plexus (B).

- Diabetic amyotrophy/neuropathy cannot be excluded by MRI alone.

TREATMENT

The patient was treated with high-dose intravenous acyclovir at 500 mg eight hourly.

Unfortunately, after 12 doses, the patient developed acute renal failure (estimated glomerular filtration rate <15 mL/min) and this was attributed to acyclovir-induced nephropathy. Acyclovir was subsequently withheld for 16 hours, and the patient treated with intravenous hydration before stepping down to oral acyclovir therapy at 500 mg eight

Figure 2 Axial MRI with gadolinium showing prominent enhancement of the left femoral nerve along the anterolateral margin of the psoas muscle.

hourly. Renal function improved rapidly and the dose was then increased to 500 mg five daily. The patient completed a total of 14 days' acyclovir therapy and her renal function returned to baseline within 7 days.

OUTCOME AND FOLLOW-UP

After the prolonged course of acyclovir and an inpatient rehabilitation admission, the patient made a partial neurological recovery and was discharged home with a walking aid. Prior to this admission she mobilised independently and was driving a vehicle. At an 11-month follow-up, her mobility continues to be dependent on a walking aid secondary to residual weakness of the left leg, and she can no longer drive a vehicle. The patient continues to experience significant postherpetic neuralgia in the dermatomes affected and remains on oral analgesia for these symptoms.

DISCUSSION

Lumbosacral radiculopathy and polyradiculopathy due to degenerative spondyloarthropathy is an extremely common presentation and cause for medical referral. In contrast, lumbosacral plexopathies remain a rare cause for asymmetrical limb weakness and may mimic polyradiculopathy from degenerative disease.⁶

Idiopathic and diabetic neuropathy appear to be the predominant causes of lumbosacral plexopathy.⁷ Other leading causes for plexopathy include central nervous system (CNS) malignancy and locally invasive malignancy (eg, retroperitoneal tumours),⁸ radiation therapy and macrovascular causes such as retroperitoneal haemorrhage, trauma and post-partum haemorrhage.^{9 10} Inflammatory, autoimmune and infectious causes including VZV plexopathy occur far less frequently.¹¹

The characteristic vesicular rash in dermatomal distribution arises due to VZV reactivation. The virus usually lies dormant within the neurons of the dorsal root ganglion. Acute rash is often preceded by pain in the corresponding dermatome. Diffuse lumbosacral plexus involvement may occur in approximately 20% of lumbosacral region reactivations.⁵ Motor neuropathy and paresis secondary to either plexopathy or single neuropathy occurs in 1%–20% of confirmed VZV reactivations.¹

While cerebrospinal fluid PCR for VZV DNA can be rapidly acquired, it has been shown to be of limited sensitivity in determining the presence of central nervous system pathology.¹² CSF serological testing demonstrates greater sensitivity in confirming the presence of CNS infection.^{12 13} MR imaging can be used to identify regions of abnormal plexus signal, however its utility lies mainly in ruling out other anatomical causes for neurological symptoms. MR sensitivity for detecting plexopathy is in the order of 70%.¹⁴

Antiviral therapy remains the mainstay of treatment of acute VZV neuropathy and plexopathy. More research regarding the early use of corticosteroids is required but limited studies combining corticosteroids with antiviral therapy have suggested faster healing of acute cutaneous herpes zoster lesions (<72 hours), however, the addition of corticosteroids appears to have no effect on the risk of developing postherpetic neuralgia.¹⁵

There are few published case reports and series describing zoster associated lower limb plexopathy,⁵ likely due to the rarity of the condition. As such, there is no consensus guideline for the long-term management of the condition, especially with respect to reducing the risk of long-term neuralgia.

Learning points

- Varicella zoster virus (VZV) lumbosacral plexopathy is a rare and probably under-recognised disease which can mimic common spondyloarthopathy presentations.
- In the context of the history, examination and imaging findings, positive cerebrospinal fluid serological testing for immunoglobulin (Ig)M and IgG antibodies confirms the diagnosis of VZV lumbosacral plexus infection.
- It should be noted that electrodiagnostic testing is widely considered the preferred diagnostic modality to confirm and localise suspected zoster plexopathy. Early MRI can play a role in the exclusion of acute spinal cord compression.

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