# Lessons of the month: Herpetic viral dermatomyositis

**Authors:** Siddharth Bhattacharjee<sup>A</sup> and Boby Varkey Maramattom<sup>B</sup>

We present the case of a man who presented with severe left lower back pain radiating to the anterior aspect of left thigh. He also had fever and headache. Due to the exquisite tenderness along the inguinal region, the possibility of a psoas abscess was considered. Magnetic resonance imaging of the spine and thigh were performed. These revealed left psoas muscle abnormalities suggestive of an evolving myositis or abscess. However, the next day, he displayed florid rashes in the left L2–L3 dermatomes consistent with herpes zoster. The clinical manifestations of herpes zoster include neuralgic pain and dermatomal skin rashes. It also presents with a prodrome of fever, headache, myalgia, myositis and Guillain-Barré syndrome. In a developing embryo, somites split to form dermatomes, myotomes (skeletal muscles), syndetomes (tendons and cartilage) and sclerotomes (bones). Our case illustrates that herpes zoster can involve the so called 'dermomyotome', a combination of the dermatome and myotome and result in a localised dermatomyositis.

**KEYWORDS:** Herpes zoster, myositis, dermatomyositis, VZV, psoas

**DOI**: 10.7861/clinmed.2020-0078

# Case presentation

A 29-year-old, physically active, man developed lower back pain on the left side 4 days prior to admission. The pain was initially mild and gradually became severe over the next 3 days (visual analogue scale 8/10). The pain was pulsating and radiated to the front of the left thigh, causing severe movement limitation. The thigh pain was aggravated by hip flexion. On day 4, he developed mild fever, headache, nausea and vomiting. There was no history of recent travel, lifting heavy weights or dysuria. On clinical examination, the femoral stretch test was positive. There were no other clinical signs. Routine blood tests were normal. Magnetic resonance imaging (MRI) of the lumbar spine and pelvis with short TI inversion recovery (STIR) and contrast images revealed contrast enhancement in the left psoas muscle (Fig 1). This was suggestive of an evolving psoas abscess. X-ray of the chest was normal and tuberculosis panel was negative.

**Authors:** <sup>A</sup>postgraduate registrar, Aster Medcity, Kochi, India; <sup>B</sup>lead consultant neurologist, Aster Medcity, Kochi, India

# Diagnosis

The differential diagnosis included a bacterial psoas abscess, tuberculous (cold) abscess, septic hip arthritis, inflammatory hip arthritis, lumbar radiculopathy, lumbar plexopathy or a retroperitoneal mass. Due to the associated fever and headache, an infectious process was considered more likely.

# Initial management and prognosis

He was started on non-steroidal anti-inflammatory drugs and pregabalin for his pain. On day 5, he developed rashes on the left anterior thigh (Fig 2). The rashes were vesicular on an erythematous base. This clinched the diagnosis of herpes zoster. He was then started on valacyclovir 1 g three times a day for 7 days.

# Case progression and outcome

Over the next 2 days, he improved symptomatically. On follow-up at day 10, the pain and rashes had completely resolved.

### Discussion

Primary infection with varicella-zoster virus (VZV) results in chickenpox. During the process of viraemia, a centripetal rash (involving the trunk and face, more than the extremities) is seen. Subsequently, the VZV utilises retrograde axonal transport and ascends centripetally via the sensory neuronal axons and seeds the ganglia of cranial nerves, dorsal root ganglia of the spinal cord as well as autonomic ganglia along the entire neuraxis in more than 90% of patients. Here the VZV lies quiescent and latent lifelong until circumstances permit reactivation.

Although the virus can be present in ganglia along the entire neuraxis (cranial nerve, cervical, thoracic, lumbar and sacral root ganglia), the most common sites of reactivation are the trigeminal and dorsal root ganglia. During ganglionic latency, the VZV burden is low and only around 6–30 VZV deoxyribonucleic acid copies are found per 100,000 ganglionic cells. The VZV cannot be cultured from ganglia and it is likely that rather than the whole virion, VZV DNA is found extra-chromosomally in a circular or end-to-end configuration inside the neuronal cell body. During periods of reactivation, the VZV burden increases and the virion again spreads centrifugally via sensory nerve axons producing dermatomal herpes zoster. From the dorsal root ganglion, the virus spreads along the sensory nerve to the corresponding dermatome. Contiguous motor nerve branches are sometimes involved as

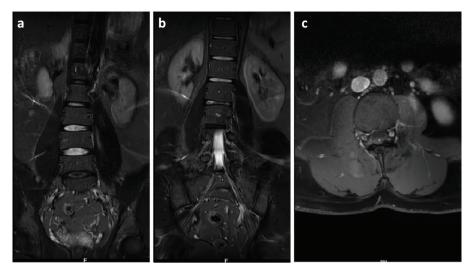


Fig 1. Magnetic resonance imaging of the lumbar spine and pelvis with short TI inversion recovery and contrast. a and b) Coronal image showing contrast enhancement in the left psoas muscle suggestive of an evolving psoas abscess. c) Axial image also showing contrast enhancement in the left psoas muscle.

in facial palsy (Ramsay—Hunt syndrome). Often, only a single dermatome is involved, although the rash can be disseminated or involve more than three dermatomes (disseminated zoster). This occurs in severely immunocompromised individuals and can be difficult to differentiate from a primary varicella infection. Clinically, herpes zoster reactivation can be divided into three phases.

- Pre-eruptive phase (preherpetic neuralgia): sensory phenomena or pain along one or more dermatomes, lasting from 1–10 days. This phase can mimic multiple pathologies and is a diagnostic conundrum until the rash appears.
- Acute eruptive phase: patchy erythema, occasionally accompanied by induration, in the dermatomal area.
- Chronic phase: persistent or recurring pain lasting 30 or more days after the acute infection or after all lesions have crusted (9–45% of all cases). Post herpetic neuralgia is a pain that persists for at least 3 months and sometimes years after resolution of zoster rash. It is more common in the elderly (40% of patients over the age of 60 years).

The neurological manifestations of VZV infection are myriad (Box 1). However direct muscle involvement by VZV is extremely



**Fig 2. Photographs of rashes on day of eruption.** a) Vesicular rash on an erythematous base on the left anterior thigh. b) Close up of rash.

# Box 1. Neurological manifestations of varicellazoster infection

Aseptic meningitis

Encephalitis

Brainstem encephalitis

Ventriculitis

Seizures

Cerebellitis

Myelitis

Acute haemorrhagic meningo-myeloradiculitis

Cerebral vasculitis and stroke

Granulomatous large vessel vasculitis

Vasculopathy

Cerebral venous thrombosis

Cranial nerve palsies (including Ramsay–Hunt syndrome)

Reye's syndrome (varicella and aspirin intake)

Polyneuropathy

Guillain–Barré syndrome

Radiculopathy

Zoster paresis (arm weakness with cervical zoster)

Diaphragmatic palsy

Zoster sine herpete (chronic radicular pain without rash)

Post herpetic neuralgia

Elsberg syndrome (bilateral sacral radiculopathy with conus myelitis)

Myositis

Rhabdomyolysis

Orbital myositis

Retinal necrosis (acute retinal necrosis and progressive outer retinal necrosis)

Retrobulbar optic neuritis

Central retinal artery occlusion

Proximal myopathy

Abdominal pseudo-hernia.

### Lessons of the month

rare and only a handful of cases of generalised myopathy or rhabdomyolysis have been described in literature so far.<sup>2,3</sup> VZV myositis has been demonstrated in one case along the zosteric myotome.<sup>4</sup> However further evidence of such a 'dermomyotomal' involvement has not been demonstrated.

### Conclusion

We presented a case of a man with pre-herpetic neuralgia and MRI muscle changes mimicking a psoas abscess. Subsequently, his dermatomal lesions declared the diagnosis of L2–L3 dermatomal herpes zoster. Thus, the L2–L3 dermatomal and the concurrent L2/L3 myotomal involvement was consistent with a herpetic dermatomyositis. The muscle and skin involvement provided evidence of VZV dissemination along a 'dermomyotome' (a combination of the dermatome and myotome). This results in a restricted or localised viral dermatomyositis. MRI with STIR sequences or muscle imaging can help to detect concurrent myotomal involvement, especially in the larger muscles.

### Summary

 Pre-herpetic neuralgia can precede the onset of rash by many days and cause a diagnostic conundrum.

- Herpes zoster can involve the myotome along with the dermatome (the so-called embryological 'dermomyotome')
- MRI with muscle sequences is essential to detect concurrent muscle changes.
- In the pre-eruptive stage, such a myotomal involvement can mimic a psoas abscess or other retroperitoneal pathology.

### References

- 1 Grahn A, Studahl M. Varicella-zoster virus infections of the central nervous system – Prognosis, diagnostics and treatment. J Infect 2015:7:281–93.
- 2 Pratt RD, Bradley JS, Loubert C et al. Rhabdomyolysis associated with acute varicella infection. Clin Infect Dis 1995;20:450–3.
- 3 Joseph TP, Chand RP, Tariq SM *et al.* Acute proximal myopathy due to herpes zoster. *J R Soc Med* 1993;86:360.
- 4 Schmidbauer M, Budka H, Pilz P, Kurata T, Hondo R. Presence, distribution and spread of productive varicella zoster virus infection in nervous tissues. *Brain* 1992;115:383–98.

Address for correspondence: Dr Siddharth Bhattacharjee, Dept of Internal Medicine, Aster Medcity, Kuttisahib Road, South Chittoor, Kochi, Kerala 682023, India. Email: sidbhatta@gmail.com