Acute flaccid paraparesis secondary to bilateral ischaemic lumbosacral plexopathy

S.K. CHHETRI1, G. LEKWUWA2, D. SERIKI3 and T. MAJEED1

From the 1Department of Neurology, 2Department of Neurophysiology and 3Department of Interventional Radiology, Royal Preston Hospital, Lancashire Teaching Hospitals NHS Foundation Trust, Sharoe Green Lane, Preston PR2 9HT, UK

Address correspondence to Dr S.K. Chhetri, Department of Neurology, Royal Preston Hospital, Preston PR2 9HT, UK. email: chhetri@doctors.org.uk

Learning point for clinicians

Lower limb weakness can be a presenting feature of various vascular syndromes affecting the spinal cord, lumbosacral roots or plexus. Acute bilateral lumbosacral plexopathy (LSP) secondary to aortoiliac occlusive disease is a rare presentation and can mimic an acute spinal cord or cauda equina syndrome.

Case history

A 47-year-old gentleman with a history of hypertension, type 2 diabetes mellitus, hypercholesterolemia, coronary artery disease and left iliofemoral bypass woke up with discomfort, numbness and severe weakness of both legs. He was a non-smoker and had no history of alcohol or drug abuse. There was an approximate 6 month history of intermittent claudication affecting his lower limbs. He denied recent weight loss and had a good glycaemic control. His regular medications included aspirin, atorvastatin, amlodipine, bisoprolol, gabapentin, metformin and lisinopril. Neurological examination demonstrated asymmetric flaccid paraparesis, worse on the left. He was not able to weight bear. Lower limb reflexes were absent. There was impairment of pinprick and touch sensation below the mid-thigh bilaterally but no definite sensory level. Vibration and proprioception were impaired distally in both legs. Sphincter function was preserved. Both his lower limbs appeared dusky with absent pulses.

Laboratory findings were unremarkable. Electrocardiogram showed sinus tachycardia but no arrhythmia. Magnetic resonance imaging of the spinal cord was unremarkable except for degenerative changes. Electromyogram and nerve conduction studies performed 2 weeks later showed extensive abnormalities consistent with asymmetric LSP. Sensory nerve action potentials were absent in both right and left sural and superficial peroneal nerves. Profuse denervation changes with reduced recruitment were noted in the musculature of lower limbs. The lumbar paraspinous muscles did not show any denervation changes thereby excluding radicular involvement and localizing the pathology to the lumbosacral plexus.

Peripheral angiogram of the abdominal aorta demonstrated occlusion of the infra renal aorta, the right renal artery and both common iliac arteries. No infrainguinal vessels were seen (Figure 1A). Fresh thrombus was present in the aortoiliac system and this was treated with mechanical thrombectomy. Aortic and bilateral iliac angioplasty and stenting was successfully performed using endovascular approach (Figure 1B). Both the lower limbs were salvaged after successful revascularization. Two years later, he remains wheelchair bound with very little improvement in lower limb function.

Discussion

Lower limb weakness can be a presenting feature of various vascular syndromes affecting the spinal
cord, lumbosacral roots or plexus. The clinical manifestations of these syndromes depend on the anatomical site and extent of vascular injury.1 Type I injury involves infarction of the distal thoracolumbar cord and manifests with flaccid paraplegia, sensory loss and sphincter dysfunction. Type II or anterior spinal artery syndrome results in infarction of the anterior two-thirds of spinal cord. There is flaccid paraplegia with loss of pain and temperature sensations but intact proprioception and vibration senses. Type III injury involves lumbosacral roots with or without patchy distal infarcts and manifests with bilateral asymmetric motor and sensory dysfunction. Type IV injury involves the lumbosacral plexus and presents with spastic paraplegia. Type V injury results in segmental spinal cord infarction and manifest with spastic paraplegia. Type VI or posterior spinal artery syndrome presents with loss of vibration and proprioception senses without any motor deficit.

The spinal cord has a complex vasculature consisting of the extramedullary and intramedullary systems.1,2 The intramedullary system consists of one anterior and two posterior spinal arteries, supplying the anterior two-thirds and posterior one-third of the spinal cord, respectively. This system is reinforced by the extramedullary system consisting of segmental arteries arising from the subclavian, intercostal, lumbar and lateral sacral arteries. The distal spinal cord, cauda equina and lumbosacral plexus are also fed by the pelvic circulation arising from the lumbar, internal iliac and deep circumflex iliac arteries. The clinical picture in our case was consistent with type IV injury, probably due to interruption of the pelvic circulation.

The lumbosacral plexus innervates the lower extremities and is anatomically divided into lumbar and sacral plexus.3 LSP is a clinical syndrome characterized by acute/sub-acute asymmetric weakness, reduced or absent deep tendon reflexes, dysesthesias and/or paraesthesias of the lower limb in multiple lumbosacral nerve root distribution.4–6 A number of structural and non-structural pathologies can present with LSP; common ones include diabetic and idiopathic lumbosacral radiculoplexus neuropathy which usually manifests with a sub-acute, asymmetric, progressive lower limb syndrome associated with weight loss.5,6 Other causes of LSP include neoplasia, infection, trauma, radiation, pregnancy, surgery, retroperitoneal haematoma, aneurysms of the abdominal aorta and its distal branches, vasculitis and connective tissue disorders.2–6 The acute onset of bilateral lower limb weakness in our case with multiple vascular risk factors would argue in favour of a vascular aetiology. This case highlights the need to consider aortoiliac occlusive disease as a potential cause of acute flaccid paraparesis, in the appropriate clinical context.

Conflict of interest: None declared.
References


