Case report

Bilateral lumbosacral plexopathy after mesenteric thrombosis

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Objective: A case of lumbosacral plexopathy (LSP) following operation for mesenteric thrombosis.

Design: Case report of a 64-year-old man who developed weakness and numbness of the distal legs after an operation for mesenteric thrombosis.

Setting: Department of Physical Medicine and Rehabilitation, University Hospital La Fe, Valencia, Spain.

Subject: Single patient case report.

Main outcome measure: Clinical and electromyography follow-up of the patient between October 1996 and August 1997.

Results: Physical examination revealed marked lower extremity weakness, hypotonia, hyporeflexia and normal bowel and bladder function. Electromyography demonstrated marked denervation of all major muscle groups, and sensory nerve conduction showed absence of responses in all peripheral nerves, in both legs.

Conclusion: To our knowledge, bilateral LSP following an intervention of mesenteric thrombosis, has never been reported in the literature. Diagnosis of LSP might be based on electromyography and nerve conduction studies that demonstrate electrodiagnostic criteria for LSP, including denervation in muscles innervated by at least two lumbosacral segmental levels and involving at least two different peripheral nerves, without paraspinal involvement.

Keywords: lumbosacral plexopathy; mesenteric thrombosis; electromyography

Introduction

LSP can mimic an 'incomplete spinal cord infarction'. The typical findings in LSP include paraparesis with variable sensory loss, hyporeflexia, disesthesia and preserved sphincter control.¹ Among other causes of LSP are: Idiopathic lumbosacral plexitis,² vasculitis,³ infection,⁴ hereditary liability to pressure,⁵ hemorrhage,⁶ trauma,⁷ intra-arterial injections,⁸ surgical conditions⁹ and tumors.¹⁰

Case report

A 64-year-old man was operated on for mesenteric thrombosis in October 1996. In the early postoperative period he developed weakness and numbness of the legs. At no time did he experience pain. He denied back pain or problems with bowel or bladder function.

No cases of hereditary liability to pressure palsies have been reported in the family. Physical examination

showed an equine attitude in both feet and distal edemas in both ankles. Homans' sign was negative and distal pulses were normal. Neurological examination showed sensory disturbances below both knees (all modalities: touch, pin, proprioception and vibration). Patellar reflex was normal in both legs and tendocalcaneus reflex was diminished in both ankles. Intact perineal and rectal tone. Bulbocavernosus reflex was normal. Muscle strength was graded on the Medical Research Council (MRC) scale as follows (Right/left): Iliopsoas 5/5, Quadriceps 4/4+, Hip Adductors 4/4, Tensor fasciae latae 5/5, Gluteus medius and minimus 5/5, Gluteus maximus 4+/4+, Hamstrings 4/4+, Gastrocnemius 1/4+, Soleus 1/4+, Tibialis anterior 0/0, Tibialis posterior 0/2, Peroneus 0/1, Extensor digitorum longus 2/3, Flexor digitorum longus 1/3.

There was no abnormality in the arms. Laboratory studies included: Complete blood count (CBC), erythrocyte sedimentation rate (ESR), electrolytes, renal and liver functions. Folic acid: 3'7 (N:3.3-6). B12 vitamin: 1623 (N:200-978). No disturbances of coagulation were observed.

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MRI of the cervical, dorsal and lumbosacral spine was normal. CT of the abdomen and lumbosacral spine showed osteoarthritis of L4/5 zygapophysal joints without nerve root compression.

Electrodiagnostic studies

Electromyography The first investigation (12-11-96) demonstrated marked denervation of all major muscle groups in both legs, with large, long lasting, polyphasic MUP's and a decreased recruitment pattern. Nerve conduction studies showed absence of right and left sciatic motor responses. The second electromyography (14-2-97) Table 1. Demonstrated electrodiagnostic criteria for LSP³ including denervation in muscles innervated by at least two lumbosacral segmental levels and involving at least two different peripheral nerves, without paraspinal involvement. Sensory nerve conduction showed absence of responses in all peripheral nerves in both legs. The third electromyography (4-8-97) showed light recovery of the left internal sciatic nerve.

Somato-sensorial evoked potential Figure 1. Tibialis posterior nerve showed a P1 wave of 51 ms in the right side, and a P1 wave of 53 ms in the left side. Both were very slow. Medullar responses could not be obtained.

Discussion

The lumbar plexus is formed from the L1, 2 and 3 root ventral rami. The sacral plexus derives from the S1-S3 ventral rami, the lumbosacral cord (L4, L5), and a contribution from the S4 ventral ramus.¹ Most of the lumbosacral plexus lies encased within the pelvic bony ring, which protects it from direct trauma unless the severity is enough to fracture-dislocate the pelvic ring.¹

The lumbosacral plexus is vulnerable to externally applied indirect trauma only if the pelvic ring is disrupted by double fracture dislocations or when traction injuries result from dislocation of the hip joint.⁷ The femoral nerve is vulnerable to compression due to its location in the gutter between the psoas and the iliacus muscles just above the inguinal ligament. Such pressure may arise from surgical retractors or from lateral pressure by hematoma beneath the iliacus fascia. The femoral nerve may be compressed by extreme angulation under the inguinal ligament during prolonged flexion abduction of the thighs in the lithotomy position under a general anesthesia.

A lumbosacral plexus lesion should be suspected when neurologic abnormalities develop in the leg within the context of abdominopelvic operation.¹ In our case, the first differential diagnosis should be made with the syndrome of Anterior Spinal Artery Hypoperfusion (SASAH)—most typically seen following thoracoabdominal aortic surgery¹¹—and the ischemic LSP. The diagnosis of ischemic LSP is based on the relative preservation of bowel and bladder function, persistence of hypotonia and hyporeflexia, progressive recovery and the absence of paraespal involvement by EMG (Table 1). The plexus is not directly involved in the innervation of the bowel, bladder or paraespal musculature. Nevertheless, all of these elements are controlled by the spinal cord, and therefore affected by the spinal cord injury. Moreover, in SASAH, sensory changes are characterized by pain and temperature loss, but deep sensation and proprioception remains.

Anatomically, the LSP is supplied by five lumbar arteries, which originate from the abdominal aorta, the deep circumflex iliac artery, a branch of the external iliac artery and the iliolumbar and gluteal

Table 1 Electromyography

	<i>Innervation</i>	<i>Interpretation</i>	<i>Spon act</i>	<i>Fib</i>	<i>Amp</i>	<i>Dur</i>	<i>Poly</i>	<i>Stabil</i>	<i>IP</i>
Izqui. Tibialis anterior	Peroneus profundus 14 L5	Complete denerv	0 (ex. fib)	10	?	?	?	Jitter	0 act.
Izqui. Gastroc caput med	Tibialis S1 s2	Mod subac Neur	0 (ex. fib)	5	+	++	++	Jitter	—
Derech Tibialis anterior	Peroneus profundus 14 L5	Complete denerv	0 (ex. fib)	10	?	?	?	Norm	0 act.
Derech Gastroc caput med	Tibialis S1 s2	Pron subac Neur	0 (ex. fib)	10	Norm	+	++	Jitter	—
Derech Rectus femoris	Femoralis 12 L3 14	Sl ineat Neur	0 (ex. fib)	0	++	+	+	Norm	Norm
Derech Gluteus medius	Gluteus inferior 14 L5 s1	Sl inact Neur	0 (ex. fib)	0	+	++	+	Norm	Norm
Derech Gluteus maximus	Gluteus superior 15 S1 s2	Sl inact Neur	0 (ex. fib)	0	+	++	+	Norm	Norm
Derech Becips fem cap long	Tibialis 15 S1 s2	Mod inact Neur	0 (ex. fib)	0	+	+++	++	Jitter	—
Derech Paravert L5	L5	Norm	0 (ex. fib)	0	Norm	Norm	+	Norm	Norm

Figure 1 Somato-sensorial evoked potential

branches of the internal iliac artery.¹² In our patient, ischemia of the LSP could be caused by a decrease of the blood flow through the lumbar segmental arteries and branches of the iliac arteries during the intervention.

MRI and CT findings were normal and therefore excluded a cancer-induced LSP. The total absence of pain and the bilateral distribution excluded idiopathic lumbosacral plexitis² and a hereditary liability to pressure palsies.⁵ These entities are also discarded because they recover spontaneously, whereas in our patient weakness was permanent. Differential diagnosis also includes retroperitoneal hemorrhage, but this etiology is observed during anticoagulation therapy,¹³ while our patient developed weakness before the treatment with heparin. Coagulation studies were normal.

Conclusion

In the case presented, there is clinical and electrophysiological evidence to support the diagnosis of an isolated bilateral lumbosacral plexopathy, probably due to ischemia in the lumbosacral plexus or to a prolonged position of the thighs during intervention. Clinicians must be aware of the LSP phenomenon. A better knowledge of this allows to assess whether specific acute interventions (i.e. maintaining the patient in the Trendelenburg position) may mitigate or prevent neurological insult, and more acutely provide prognostic information based on objective data.

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