Paraplegia

Ischaemic Lumbosacral Plexopathy in Acute Vascular Compromise: Case Report

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Summary

Anterior spinal artery syndrome (ASAS) is a well reported cause of spinal cord injury (SCI) following thoracoabdominal aortic surgery. The resultant deficits are often incomplete, typically attributed to the variable nature of the vascular distribution. Our Physical Medicine and Rehabilitation (PM and Rehabilitation) service was consulted about a 36-year-old patient with generalised deconditioning, 3 months after a stab wound to the left ventricle. Physical examination revealed marked lower extremity weakness, hypotonia, hyporeflexia, and a functioning bowel and bladder. Further questioning disclosed lower extremity dysesthesias. Nerve conduction studies showed slowed velocities, prolonged distal latencies and decreased amplitudes of all lower extremity nerves. Electromyography revealed denervation of all proximal and distal lower extremity musculature, with normal paraspinalis. Upper extremity studies were normal. Recently, 3 cases of ischaemic lumbosacral plexopathy, mimicking an incomplete SCI, have been reported. This distinction is particularly difficult in the polytrauma patient with multiple musculoskeletal injuries or prolonged recuperation time, in addition to a vascular insult, as in this patient. The involved anatomical considerations will be discussed. A review of the electrodiagnostic data from 30 patients, with lower extremity weakness following acute ischaemia, revealed a 20% incidence of spinal cord compromise, but no evidence of a plexopathy.

Key words: Ischaemia; Lumbosacral plexopathy; Electromyography.

Recent advances in cardiovascular and trauma surgery have led to increased survival of patients following cardiac and great vessel trauma or insult. The associated generalised or localised hypotension often seen may cause varying degrees of ischaemia to vital organ systems, including the spinal cord and peripheral nervous system. The syndrome of anterior spinal artery hypoperfusion with resultant midthoracic anterior spinal cord ischaemia, most typically seen following abdominal aortic aneurysm resection, is a well documented example of this phenomenon. [Adams, 1956; Blenow, 1987; Dodson, 1973; Fosburg, 1976; Foo 1983].

The clinical picture of anterior spinal artery syndrome (ASAS) is protean in nature, often attributed to the variable nature of the vascular supply to the spinal cord or the varying degree of ischaemia suffered. Classically, an initial clinical picture of flaccid paraplegia often progressing to an upper motor neuron picture, accompanied by dissociated loss of sensation has been described. The sensory changes are characterised by a loss of pain and temperature sense but with preservation of deep sensation and proprioception. [Adams, 1985; Blenow, 1987; Fosburg, 1976; Foo, 1983] Paralysis of the bowel and bladder is also found. [Blenow, 1987; Fosburg, 1976; Foo, 1983] Electrodiagnostic testing reveals normal nerve conduction studies, variable electromyographic findings depending on the completeness of the lesion, but typically evidence of denervation of all musculature caudal to the lesion, including the paraspinals. [Foo, 1983; Kimura, 1985] Neurophysiological investigation typically shows slow or abnormal SSEP's [Kimura, 1985]. Often, these 'classic' findings are rarely all present, and the lesion is often defined as 'incomplete'.

Lumbosacral plexopathy (LSP) can mimic an 'incomplete' spinal cord infarction. While rarely reported following vascular insult, lumbosacral plexopathy has been well documented in the oncological literature [Calverly, 1975; Evans, 1981; Jaeckle, 1985]. According to one report, as many as 15% of neurological consultations at a major cancer centre for leg pain and weakness, and low back pain were secondary to LSP, arising from primary and secondary pelvic tumours (colorectal, sarcoma, breast, lymphoma) [Jaeckle, 1985]. It is interesting that there is difficulty in distinguishing between spinal cord, cauda equina and lumbosacral plexus involvement, so often noted in these patients. The typical findings in LSP secondary to tumour include, paraparesis with variable sensory loss, hyporeflexia, dysesthesias, preserved sphincter control, electromyographic evidence of denervation, polyphasic MUP's with decreased amplitudes and normal to borderline nerve conduction velocities [Jaeckle, 1985]. Similar findings have also been reported in lumbosacral plexus involvement secondary to radiation, diabetes, compression and traumatic plexopathy [Brown, 1984; Emery, 1978; Sander, 1981]. The degree of return of neurological function tends to vary with the cause, rapidity of onset and extent of involvement.

Ischaemic lumbosacral plexopathy as a result of spinal cord infarction has been mentioned in the medical literature, but poorly documented [Lerin, 1984]. Recently, the diagnosis of ischaemic lumbosacral plexopathy has been applied to three cases of presumed incomplete ASAS, based on the relative preservation of bowel and bladder function, persistence of hypotonia and hyporeflexia, progressive recovery and the absence of paraspinal involvement by EMG [Stutesman, 1987]. In these three cases of ischaemic lumbosacral plexopathy, significant recovery of isolated muscle groups was noted for up to 6 months post-insult, allowing for functional ambulation with assistive devices in all patients. Balanced bladders were also achieved. Whereas a significant proportion of patients achieve some neurological return following ischaemic spinal cord injury, it appears to be less common than following ischaemic LSP [Foo, 1983; Stutesman 1987].

Case report

A thirty-six-year-old man was seen by our PM and Rehabilitation consultation ser-

vice for generalised deconditioning. Premorbidly, he was in excellent health, until he presented to our Trauma Centre following a stab wound to the left chest and through the left ventricle. Initially, the patient was hypotensive (systolic Bp 40) and underwent emergency thoracotomy with repair of the cardiac laceration. In the post-operative period he sustained a cardiopulmonary arrest, secondary to cardiac arrhythmia, from which he was resuscitated within 5 minutes. Over the next 2 weeks he proceeded to develop renal failure, hepatitis and generalised encephalopathy. Over the next 2 months, while on ventilatory support, the patient developed several respiratory infections. He was noted to have increasing alertness approximately 13 weeks post-insult, and at that time PM and Rehabilitation was consulted.

The patient had a variable level of consciousness, but when responsive he complained of pain (dysesthetic) in both lower extremities. Physical examination revealed marked atrophy of all lower extremity musculature with fair (anti-gravity) strength, hypotonia, hyporeflexia, decreased but intact sensation (all modalities) and bowel/bladder continence. Upper extremity and cranial nerve exams were normal. Functionally, the patient required maximal assistance for mobility and lower extremity ADL's, and minimal assistance for upper extremity skills.

An EMG/NCS was obtained to more clearly elucidate the underlying pathology, which had been diagnosed as ischaemic damage to the spinal cord (anterior spinal artery syndrome). Nerve conduction studies revealed slowing (32–37 m/sec) of all lower extremity nerves with prolonged distal latencies and low amplitudes of motor action potentials. Upper extremities were within normal limits (Table I). Electromyography demonstrated marked denervation of all major muscle groups of both legs, with large, long duration, serrated to polyphasic MUP's and a decreased recruitment pattern. Paraspinal and upper extremity examination was normal (Table II). Based on this examination and the clinical picture, the diagnosis of ischaemic lumbosacral plexopathy was made.

The patient was initiated in a program of pain management, reconditioning, strengthening and functional skills training, and following a brief stay in our

Table I Nerve conduction study

Normal	Abnormal
Rt median	Bilat peroneal
Rt ulnar	Bilat tibial
Rt radial	Bilat sural

Table II Electromyography

Normal	Abnormal
Rt biceps brachii	Bilat adductor longus
Rt 1st dorsal interosseous	Bilat vastus medialis
Bilat L1 to S1 paraspinals	Bilat tibialis anterior
	Left gluteus medius
	Right gluteus maximus
	Bilat gastrocnemius

Rehabilitation Unit, he was able to be discharged home at a stand-by assist level. The patient failed to return for future scheduled appointments and therefore follow-up evaluation could not be completed.

Discussion

The lumbosacral plexus, comprised of branches from L2 to S3, innervates the entire lower limb, with the exception of some skin on the buttock. There is some disagreement as to whether the proximal musculature (glutei, psoas) is innervated from the plexus or directly from the root level, thus their involvement in plexopathy may be variable [Brown, 1987]. The plexus is not involved directly in the innervation of the bowel, bladder or paraspinal musculature. Isolated insult to the lumbosacral plexus would therefore be expected to affect all the nerves and muscles of the lower limb, without involving the bowel, bladder or paraspinal musculature. The spinal cord, however, controls all of these elements, and therefore spinal cord injury affects them all. The degree of involvement of the different structures will vary to some degree with the amount of insult sustained. In the case presented, there is concurring clinical and electrophysiological evidence supporting the diagnosis of an isolated bilateral lumbosacral plexopathy following an acute ischaemic event. While this phenomenon has been suggested in the recent literature, this report represents the most complete presentation of ischaemic LSP.

The blood supply to the lumbosacral plexus is primarily derived from numerous small branches of the major pelvic arteries. The inferior gluteal, via the *arteria comitans nervi ischiadici*, and the superior gluteal artery, via the lateral sacral artery, provide the major supply. The gluteal arteries are branches of the internal iliac artery, which arises from the abdominal aorta [Anderson, 1982; Gardner, 1975; Hollingshead, 1974]

The blood supply to the spinal cord is provided by three major vertical arteries, the single anterior spinal artery and the paired posterior spinal arteries. Typically, the anterior spinal artery (ASA) provides the majority of the spinal cord circulation. The ASA narrows or becomes discontinuous as it descends the spinal cord, creating areas of watershed at the cervicodorsal, mid-dorsal and lumbosacral regions. It is connected to the systemic circulation via radicular branches of the vertebral, ascending cervical, intercostal, lumbar and iliolumbar arteries, however these connections are often variable and inconsistent, leading to a segmental nature. The posterior and anterior spinal arteries are linked by numerous communicating arteries. The major factor contributing to the so-called anterior spinal artery syndrome, is the location and patency of the *arteria radicularis magna* (artery of Adamkiewicz). If this vessel originates above the renal arteries, it usually is the only significant radicular vessel, and all branches caudally are smaller and insignificant. If its origin is below the renal arteries, the segmental blood supply above and below is relatively well preserved. [Adams, 1956; Anderson, 1982; Fosburg, 1976; Gardner, 1975; Hollingshead, 1974]

Episodes of ischaemia in those patients with suprarenal origins of the *arteria radicularis magna* may lead to ASAS. The blood supply to the nerves supplying the bowel and bladder, via the spinal cord (sympathetic trunk, pelvic splanchnic nerve, pudendal nerve and superior hypogastric plexus), is provided by the spinal arteries, radicular branches and small branches of the pelvic vessels. In a person

with an infrarenal artery of Adamkiewicz, and therefore a relatively good collateral blood supply to the lumbar spinal cord, an ischaemic event might be expected to most affect the more distal structures, such as the lumbosacral plexus. This would help to explain the case report presented.

In an attempt to explore the frequency of ischaemic LSP, the electrodiagnostic studies (EMG/NCS and SSEP) of 30 consecutive patients referred to the PM and Rehabilitation service for electrodiagnostic evaluation following an episode of ischaemia (operative abdominal aortic cross-clamping) with subsequent lower extremity weakness, were reviewed. Sixteen (53%) of the patients had normal studies; 8 (27%) had evidence of a peripheral neuropathy without other deficit, and 6 (20%) had evidence of ASAS. None of the studies reviewed were suggestive of a plexopathy. Clearly, this is a very limited group, and further investigation is needed, but it does support the notion that lumbosacral plexopathy following ischaemia is not a common event.

Conclusion

Ischaemia to the central or peripheral nervous system may result in various clinical pictures, and these resulting syndromes may be due to different pathologies. The most common syndrome seen with acute vascular compromise to the spinal cord is due to involvement of the anterior spinal artery, with impairment of function in all caudal structures. Although rare, a second syndrome seen is that of ischaemic lumbosacral plexopathy, with impairment limited to the lower extremity nerves and musculature. It is important for the clinician to be aware of the phenomenon of ischaemic lumbosacral plexopathy, and the anatomical relationships involved for several reasons. Knowledge of it better allows one the ability to; assess if specific acute interventions (i.e. maintaining the patient in the Trendelenberg position) may mitigate or prevent neurological insult, differentiate between its lower motor neuron origins and other upper motor neuron causes (spinal cord insult) for management issues (bowel and bladder care, presence of dysesthesias), explain physical and electrodiagnostic findings based on physiological reasons, and more accurately provide prognostic information based on objective data. Finally, it is important to increase one's level of awareness, so that the relative incidence of occurrence can be better assessed.

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