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Post-surgical Ischaemic Myelopathy

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Summary

Ischaemic myelopathy is an infrequent but well-known cause of spinal cord injury. While the overall incidence of neurological injury following thoraco-abdominal aortic surgery is low (1–14%), procedures requiring surgical cross-clamping of the aorta have been reported as the major cause of ischaemic cord injury (31%). Little has been reported regarding the clinical and functional outcomes of these injuries. Three patients with a non-penetrating aortic injury who showed evidence of ischaemic cord injury within 72 hours of surgical cross-clamping of the aorta are presented. Data includes functional assessments, muscle strength testing and electromyographic findings. All three patients showed lower thoracic incomplete motor and sensory spinal cord injuries. These findings suggest that, after a period of neurological improvement, a plateau phase is reached at approximately 3 months post injury after which no significant gain in muscle strength is made. All patients were functionally independent and able to ambulate using a straight cane.
Key words: *Ischaemic myelopathy; Aortic injury; Lumbosacral plexopathy.*

Introduction

Spinal cord injury due to ischaemia has been well reported in medical literature. (Foo, 1983; Dodson, 1983; Foo, 1981; Szilagyi, 1978; Ferguson, 1975; Brewer, 1972; Herrick, 1971; Edmondson, 1970; Coupland, 1968; Garland, 1966; Weisman, 1944). The most frequent causes of this type of injury has recently been reported to be procedures requiring cross-clamping of the aorta. (Kim, 1985). The incidence of post-surgical spinal cord injury is reported as being between 1 to 14% (Laschinger, 1984).

Case Reports

We present 3 cases of postsurgical ischaemic myelopathy occurring after surgical repair of a traumatically disrupted thoracic aorta (Table I). All patients were neurologically intact on examination prior to surgical intervention. No evidence of bony fracture or dislocation of the vertebral column was seen in any patient. No episodes of severe intra-operative hypotension were recorded.

Patient 1 developed neurological deficits over a period of 72 hours postopera-

Table I

	Patient 1	Patient 2	Patient 3
Age	20 years	42 years	21 years
Sex	Male	Male	Male
Injury	Motor vehicle accident with aortic rupture, no neurological deficit.	Motor vehicle accident with aortic rupture, no neurological deficit.	Motor vehicle accident with aortic rupture, no neurological deficit.
Length of Surgery	5 hours	3 hours	4 hours 25 minutes
Length of Aortic Clamping	39 minutes	53 minutes	Unknown
Days Post-injury Transferred to Rehabilitation Center (Average 13)	16	10	13
Length of Stay in Rehabilitation Centre (days) (Average 39)	6	28	89
Neurological Deficits at time of Admission to Rehabilitation Centre.	Lower thoracic motor incomplete sensory intact	Lower thoracic motor incomplete sensory intact	Lower thoracic motor incomplete sensory incomplete

tively while patients 2 and 3 showed deficits immediately after surgery. No evidence of hematoma formation or other type of compression of the spinal cord could be found in any of the cases.

The patients were transferred to the rehabilitation unit when medically stable and began a comprehensive rehabilitation program. At the time of transfer the patients were found to have lower thoracic incomplete spinal cord lesions with motor deficits much greater than sensory deficits. Only patient 3 showed marked sensory and proprioceptive deficits. None of the patients have developed significant spasticity, remaining slightly hypotonic and hyporeflexic in the lower extremities. All patients initially required intermittent bladder catheterisation but were subsequently able to achieve balanced bladders. Patient 3 has reported the inability to achieve an erection while patient 2 complained of problems with premature ejaculation. Patient 1 reported no sexual dysfunction. No patient has had difficulty with bowel management.

Results

Recovery of one to two grades of muscle strength was seen in isolated muscle groups early in the rehabilitation process. While some muscle groups underwent rapid recovery others made no improvement and began to show atrophy. In those muscle groups which showed improvement, the greatest return of muscle strength was achieved within the first 3 months of injury. Some further recovery was seen up to 6 months post-injury but despite continued therapy, and highly motivated patients, little additional improvement in muscle strength was attained. At the time of discharge all patients were able to ambulate independently using one or two straight canes, though patient 3 also required bilateral knee-ankle-foot orthosis and the use of a wheelchair for long distances.

Each patient successfully returned to his premorbid educational or employment activity after discharge. Each patient was seen for follow-up at approximately 3 months, 6 months and 1 year after injury.

Table II Electrodiagnostic Studies

	Patient 1	Patient 2	Patient 3
Time Post Injury	11 months	8 months	12 months
Nerve Conduction Studies	Normal	Normal	Normal
Distal Motor Latency			
Distal Sensory Latency	Absent	Normal	Prolonged
F-waves	Absent peroneal Prolonged tibial	Normal peroneal Absent tibial	Absent peroneal Prolonged tibial
H-reflex	Absent bilaterally	Normal bilaterally	Normal bilaterally
Electromyography	Normal	Normal	Normal
Paraspinal Muscles L_1–S_1			
Lower Extremity Muscles	Abnormal *	Abnormal *	Abnormal *

* Variable findings of increased insertional activity, increased spontaneous rest activity, decreased recruitment patterns, increased polyphasic motor unit action potentials and various size amplitudes ranging up to 15 mV.

Nerve conduction studies and electromyographic examinations were performed during various follow-up examinations (Table II). While the nerve conductions studies showed some abnormalities such as the absence of the H-wave bilaterally in patient 1, and the prolongation or absence of the F-waves in the peroneal and tibial nerves of all the patients, distal motor latencies were found to be essentially normal. Decreases in the amplitudes of the evoked motor response were seen in the most severely affected muscles consistent with muscle atrophy. Electromyographic findings showed widespread polyphasic motor potentials, consistent with incomplete injury with signs of re-innervation. Recruitment patterns were highly variable with no voluntary recruited motor units seen in the biceps femoris muscles bilaterally in patients 2 and 3. Electromyographic studies of the L_2–S_1 paraspinal muscles bilaterally showed no abnormalities in any of the patients.

Discussion

Little has been reported regarding the natural history of post-surgical ischaemic cord injury. Several isolated cases similar to our patients have been reported, (Foo and Rossier, 1983; Garland, 1966) but little follow-up information is available for these patients. The clinical course followed by our patients led us to question the pathological basis of their neurological deficits. The electromyographic findings of normal paraspinal muscles suggests a more functional spinal cord than we expected with the persistent hypotonia and hyporeflexia. In light of essentially normal peripheral nerve conduction studies, recruitment patterns seen during the electromyographic exams were suggestive of bilateral lumbosacral plexus injury. These findings have led us to postulate that these patients' deficits may not be due to injury exclusively at the spinal cord level but may involve significant ischaemic injury at a more peripheral level, most likely at the lumbosacral plexus. These findings may have important prognostic significance for any further neurological recovery in this group. We plan sequential elect-

rodiagnostic studies and continued long term follow up of these patients. While no firm conclusions can be drawn from such a small group of patients, we believe further investigation of the pathology and natural history of these types of injuries is needed.

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