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Delayed, transient quadriplegia; the importance of spinal cord perfusion

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SUMMARY

Transient quadriplegia developed in a man, a short time after, he sustained a cervical spinal fracture in a surfing mishap. The neurological deficit appeared complete, and developed some 30 min after the initial injury in the presence of moderate hypotension. It resolved over a further period of 1–2 hours following restoration of normotension. This case highlights the importance of the maintenance of spinal perfusion pressure in the acute management of traumatic spinal injury.

BACKGROUND

This is an unusual sequence of events in a relatively common injury that may have devastating consequences. This case highlights the importance of the maintenance of spinal perfusion pressure in the acute management of traumatic spinal injury.

CASE PRESENTATION

A man in his late 60s sustained a forceful, hyperflexion, cervical injury while body surfing. His medical history included osteoarthritis leading to bilateral knee replacements and a single episode of sciatica that resolved fully following paraspinal, corticosteroid injection. There was no history of hypertension and no evidence of any specific spinal disorder apart from osteoarthritic, degenerative changes. He led a very active life with enthusiastic involvement in social road cycling, kayaking and ocean swimming up until the time of the accident. A younger sibling suffered a bifacet C4-5 fracture dislocation without any neurological deficit in a Rugby scrum incident some 40 years earlier. Again, there was no suggestion of any underlying spinal disease.

Following the acute injury in the surf, the patient was able to self-extricate but suffered severe neck pain and was observed to have marked cervical muscle spasm. He walked up the beach without any real assistance and was dried and warmed by bystanders. He was unable to lie flat because of pain but was carefully supported in a semisitting position while awaiting ambulance retrieval from the beach.

The patient was initially fully functional with intact motor and sensory function on limited, beachside assessment. He was able to move all limbs on request and noted no sensory disturbance although this was not systematically assessed. He was specifically observed to have intact intercostal muscle function with a normal breathing pattern. Over the ensuing 15–30 min,

he developed rapidly progressive quadriplegia. The patient reported a sense of ‘shutting down’ and became quite distressed with a sense of imminent death. Full neurological assessment was not undertaken but there was no voluntary movement or subjective sensation in any limb. He had some dyspnoea. Intercostal muscle function was now lost with the onset of paradoxical breathing and intercostal recession. He could eventually be laid flat with ongoing manual neck support. This improved the sensation of dyspnoea.

On the arrival of the ambulance more than 60 min after the initial injury, the blood pressure was recorded as 70 mm Hg systolic with a heart rate of 60 beats per min. The absence of neurological function below C4 was confirmed, once again without a detailed neurological examination but rather with simple questioning and observation. The relative bradycardia rather than reflex tachycardia suggests a neurogenic mechanism for the hypotension consistent with the observed tetraplegia. An intravenous line was established and fluids administered. On the later arrival of the helicopter transport team, the emergency doctor administered metaraminol with normalisation of blood pressure to 140/80. A hard, cervical collar was applied. The patient was carefully loaded onto a palisade and manually transported off the beach. Immediately prior to loading in the ambulance, first one toe and then one foot were observed to have moved. The patient was transported to the regional spinal trauma centre by helicopter. Further doses of metaraminol were administered en route. On arrival at the tertiary centre, neurological function had almost fully recovered with only some residual hand weakness and minor sensory dysfunction evident.

INVESTIGATIONS

Neurological imaging confirmed spinal disruption at the C3–4 level ([figure 1](#)).

TREATMENT

Anterior reduction and C3–5 fixation were undertaken the same evening and posterior C3–C6 operative fixation 3 days later.

OUTCOME AND FOLLOW-UP

The patient made an uneventful recovery following surgery and was discharged home on day 8 following injury. Neurological examination over the recovery period detected no objective abnormality although some subjective weakness and altered sensation was reported by the patient



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Figure 1 MRI scan of the cervical spine indicating anterior dislocation of C3 on C4 with marked canal narrowing and displacement of CSF but without any direct injury or compression to the spinal cord.

in the right hand together with some neuralgic discomfort over the crown of the head. Following surgery, blood pressure remained within normal limits with no further intervention required. Approximately 4 months after the injury, the subjective hand symptoms resolved suddenly and completely. The vague neuralgic discomfort over the crown persists but has diminished significantly. The patient has resumed road cycling, kayaking and swimming in spite of some restriction in neck movement but is yet to resume open-ocean event swimming.

DISCUSSION

This patient described an unusual clinical course, unique in my experience. It seems most likely that the mechanism behind the transient, apparently complete, loss of spinal cord function relates to impaired perfusion. Certainly, the blood pressure was low, and the spinal canal markedly narrowed as observed on the MRI scan such that spinal perfusion pressure must have been compromised. Spinal cord concussion (neuropraxia) seems very unlikely given the time course. Spinal concussion can certainly result in fully reversible spinal cord dysfunction but it, by definition,¹ occurs immediately after the trauma rather than in a delayed fashion as occurred in this case. It is difficult to imagine a mechanism that could account for such a delay with a subsequently quite rapid and complete resolution. Spinal compression due to subsequent movement is possible, but the deforming force is usually greatest at the moment of injury and the neck was carefully supported by an experienced practitioner following the injury to avoid further flexion. Also, the transient loss of function was apparently complete, affecting the entire cord rather than the anterior or posterior segments alone. Finally, cord function began to

improve soon after restoration of blood pressure with vasoconstrictor administration. Hypotension is a consequence as much as a cause of spinal cord dysfunction, but it seems likely that a cycle of hypotension related to the trauma and mild dehydration led to impaired cord perfusion that was then markedly exacerbated by the consequent spinal cord dysfunction-induced vasodilation (neurogenic shock).

This case highlights the utility of established principles of acute management of spinal injury such as field support with neck stabilisation and early surgical fixation. The unusual course, however, strongly emphasises the importance of spinal cord perfusion in the postinjury management of acute spinal cord injury. The favourable impact on spinal recovery of maintenance of cord perfusion is now well established^{2,3} although the evidence is mostly derived from human cohort and observational studies. Nevertheless, systematic reviews over the years^{4,5} have consistently reported a beneficial effect of blood pressure maintenance in spite of the absence of high-level studies. In consequence, the maintenance of perfusion pressure has become part of established clinical guidelines.^{6,7} While the precise pathogenesis of the transient quadriplegia observed in this case cannot be proven, the personal and societal impact of spinal cord injury is so great that any initiative that might ameliorate or even avert the clinical impact of spinal injury, as in the case described, warrants consideration. Initiatives with very low cost and low complication rate such as blood pressure maintenance should be ‘standard of care’.

Learning points

- ▶ Spinal cord injury is a common and potentially devastating injury.
- ▶ Spinal cord hypoperfusion may contribute to the extent of dysfunction and may limit recovery such that hypotension should be rapidly corrected and the mean arterial pressure should be maintained within the range of 85–90 mm Hg for 7 days following acute injury.
- ▶ Stabilisation of the cervical spine in the field with manual support and hard cervical collar and early cervical spinal fracture reduction with spinal decompression and internal fixation remain essential components of the management of acute spinal injury.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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