Paraplegia **32** (1994) 93–97

Spinal cord injury: patients who had an accident, walked but became spinal paralysed

M Masini MD, M R B Alencar MD, E G de Castro Neves MD, C F Alves MD

The Hospital for Locomotor System Disease – SARAH, Brasilia DF, Brazil.

Out of 1410 patients admitted to the spinal cord injury unit (SARAH) for rehabilitation during a 10-year period (1981–90) 10 had a missed spinal trauma lesion and became paralysed after having overcome the initial injuries and being allowed to walk. Patients were admitted to hospital with a history of being able to walk after an accident and subsequently developing a neurological deficit. Seven patients had radiological findings compatible with instability of the spine. Four had a head injury with coma and an undetected spinal fracture at the first evaluation in the emergency room. Other associated factors were: spinal stenosis, prolapsed disc, infection, foreign bodies, procedures for reduction and stabilization of the fractured spine. We emphasize the need for very careful clinical study and investigation for the diagnosis of multiinjured patients, especially when there is a concurrent brain injury.

Keywords: multitrauma; spinal paralysis; delayed diagnosis.

Introduction

In a small number of patients with spinal cord injury (SCI) the neurological deficit may develop later from the spinal injury. We identified a group of patients who were admitted for rehabilitation following an accident, walked after it and subsequently developed a severe spinal neurological deficit. The clinical histories of these patients indicate that after the accident there was a period when the legs had good movement and they were allowed to walk.

Population and method

A retrospective analysis of 1410 charts of patients admitted for rehabilitation between 1981 and 1990 to the spinal cord injury unit (HAL/SARAH) was carried out. Ten patients had a clinical history of an accident, walked after it and subsequently became paralysed. All had confirmation of this information made by referee doctors. The charts and radiological examinations were analysed retrospectively in an attempt to identify the cause of the late spinal cord injury and its evolution.

Results

All patients were young males aged between 6 and 30 years. In seven cases the initial injury was due to a traffic accident and in the remaining cases to a fall, a gunshot wound and a knife lesion. The initial lesion was cervical in three cases, thoracic in six cases and lumbar in one case. Patients were graded according to the Frankel scale of neurological deficit at admission for rehabilitation. Six were graded A (paraplegics) and four C (one paraparetic and three tetraparetics). The interval between the initial accident and the development of the neurological deficit ranged from minutes to 90 days, with eight cases occurring in the first 30 days, one in the second and one in the third month after the trauma to the spine. Referral to our institution for rehabilitation occurred in a mean period of 2.5 months after the appearance of the deficit. The patients were followed up by us for a mean period of 16 months.

Charts and radiological examination reviews showed that seven had spinal instability secondary to fracture or dislocation (cases 1, 2, 4, 5, 7, 9, 10); four had a head injury and coma in the initial emergency care period (cases 4, 5, 9, 10). The other

four had, respectively, associated spinal stenosis, herniated disc (case 1), and dislocation of a fixation hook at the level of T6 (case 6). The remaining two had, respectively, an epidural haematoma complicated by infection (case 8), and a surgical sequella after removal of a knife blade close to the spinal cord (case 3).

Three reports are given to provide further information on our patients. Table I details the initial neurological status immediately after the trauma, the cause of the accident, the interval between the accident and the development of neurological deficit, the neurological status at rehabilitation, the radiological findings and a commentary on the cause of late neurological deficits.

Case reports

Case 5

A 44 year old male had a car accident 3 months earlier, suffering a head trauma and being in a coma during the following 18 days. After recovering consciousness he started walking, but rapidly developed tetraparesis. Cervical X-ray demonstrated a fracture–dislocation at C6–7 (Fig 1). He was admitted for rehabilitation in a wheelchair and graded C. An anterior decompression and stabilisation procedure was performed. He was immobilised for 4 months and his neurological deficit improved to D (ambulatory condition).

Case 9

A 17 year old male had a car accident and remained unconscious for 24 hours. On recovering consciousness, he complained of dysaesthesiae in the lower limbs. The cervical X-ray was considered to be normal. After 6 days in bed he was allowed to walk to the bathroom, but on walking a few steps he became paraplegic. He was admitted 2 months later, paraplegic, with a sensory level corresponding to T6 (A). Radiological examination demonstrated a fracture dislocation at T3–4 (Fig 2). The degree of neurological deficit was unchanged during the follow up period.

Case 10

A 14 year old male had a car accident, suffering a head injury with loss of consciousness for a few minutes. On recovering, he walked a few steps and developed lower limbs paralysis. He was admitted 1 month later, with a partial neurological deficit in the lower extremities (C). Lumbar X-rays demonstrated a severe L2 and L3 luxation anteriorly to L4 and L5 (Fig 3). After rehabilitation, his condition improved to D.

Discussion

Special care to avoid secondary trauma and a neurological deficit in trauma patients is frequenlty stressed. Surprisingly, the possible consequence of this situation has not been well studied in the literature. Some contributions in the literature expressed the opinion that deterioration of the initial degree of cord injury occurs because of expansion of haemorrhage and/or oedema but none has taken into consideration patients who have suffered trauma with no initial neurological deficit, who were able to walk and who developed a serious neurological deficit some time afterwards. The overall frequency of these cases in our population is 0.7% (10 cases in 1410 total admissions). But even with a low frequency, the severity (60% being paralysed) indicates the need for better diagnosis in multiinjured and head injured patients.

Haiden *et al* (1975) and Wagner & Chehrazi (1982) consider that some patients develop their neurological deficit some time after the trauma. In their opinion this period of time, between the initial trauma and the development of neurological deficit, corresponds to the interval between a second trauma to an already unstable spine. Instability of the spine was identified in seven of our cases, and in four there was the association of a head injury and a period of unconsciousness. Our study showed that some other factors such as spinal stenosis, prolapsed disc, infection, foreign body in the canal and hook dislocation may be responsible for some late spinal cord lesions.

The definition of a second injury was clear in our patients. We found that our seven patients with spinal instability had no such diagnosis established; four had an associated head injury with a period of unconsciousness which might have failed to reveal a

Table I Patient details

Initial neurological status immediately after trauma	Cause of spinal cord injury	Interval to neurological deficit	Neurological deficit status	Radiological findings	Presumed cause of second trauma
Case 1 E Recumbent before laminectomy	Fall	15 days	Paraplegia A	Fracture/dislocation spinal stenosis T10–11	Instability after laminectomy
Case 2 E Walked to hospital for assistance	Traffic accident	30 min	Paraplegia A	Fracture/dislocation T10–11	Instability and prolapsed disc
Case 3 E Elected surgery, removal of knife	Knife	90 days	Paraplegia A	Blade of knife T8	Surgical trauma where knife removed
Case 4 E Head injury, coma without deficit, walked in hospital	Traffic accident	30 days	Tetraparesis C	Fracture/dislocation C7–T1	Instability/head injury coma/no x-ray of the spine
Case 5 E Head injury, coma without deficit, walked in hospital	Traffic accident	18 days	Tetraparesis C	Fracture/dislocation C6–7	Instability/head injury coma no x-ray of the spine
Case 6 E Surgery at 17th day, walked out of hospital	Traffic accident	60 days	Paraplegia A	Fracture/dislocation T12–L1	Hook dislocation at T8
Case 7 E Walking/progressive weakness in legs	Traffic accident	7 days	Tetraparesis C	Fracture/dislocation C6–7	Instability/fracture not visualised
Case 8 E Fever and deficit	Gunshot wound	23 days	Paraplegia A	Gunshot wound T4	Epidural abscess
Case 9 E Minor head injury, walked to bathroom	Traffic accident	6 days	Paraplegia A	Fracture/dislocation T3–4, no x-ray of the dorsal spine	Instability, head injury coma
Case 10 E Head injury, momentary loss of consciousness, walked after the accident	Traffic accident	A few steps	Paraparesis C	Fracture/dislocation L3–4, rescue inadequate	Instability, head injury

E = Frankel grade E, no neurological deficit.

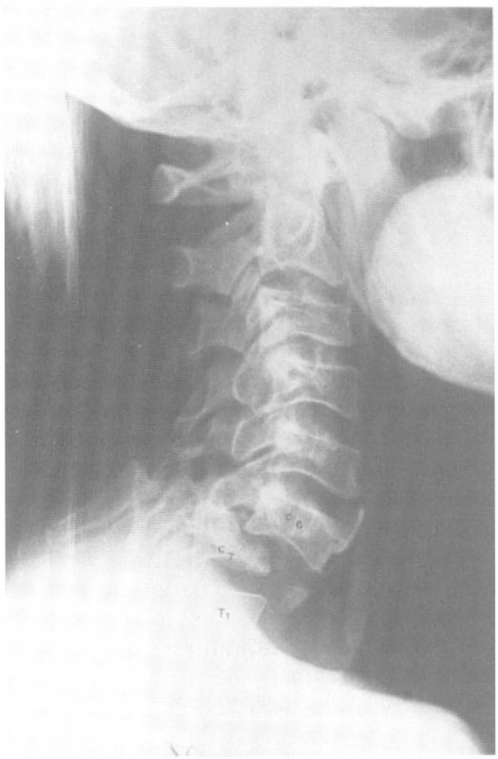

Figure 1 Case 5. Lateral cervical spine X-ray showing C6–7 fracture–dislocations.

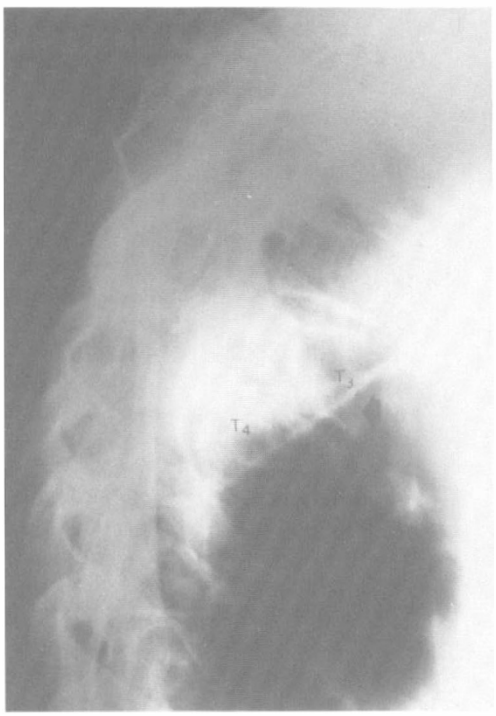

Figure 2 Case 9. Lateral thoracic spine X-ray showing T3–4 fracture–dislocation.

diagnosis of the spinal fracture/dislocation. As they were free to walk, they developed the neurological deficit. One patient was allowed to stand up immediately after the accident. He has a high level unstable fracture at the lumbar spine (case 10) and developed paraparesis after taking a few steps. Case 4 had an x-ray of the cervical spine in the emergency room but the examination did not show the C7/T1 transition and 3 weeks later, when he started walking, he became paraparetic. Concerning the other two cases (cases 5, 9), one cervical and one thoracic spine, the x-rays had not been done initially. In a review of the literature, Steudel *et al* (1988) reported the incidence of head and cervical spine injury as being between 6 and 63%; the severity of brain lesions had a marked influence on the prognosis of the spinal cord injury and the correct identification of cervical lesions was delayed in about 50% of their patients. In the group with altered consciousness, early spinal stabilization was indicated to prevent further neurological deterioration.

Our study supports the view that all patients with a serious head injury should be treated as if there is a concomitant cervical spine injury until proven otherwise. There are some important associated predisposing factors which include surgery: in case 6 the neurological deficit was related to a surgical procedure and we are not implying that poor management was involved. Rods and hooks can become loose and, in few cases, cause neurological deficit as was referred to by Harrington and Dockson (1973); these risks are associated with all spinal stabilization procedures. The procedure to remove part of a knife is very dangerous and can

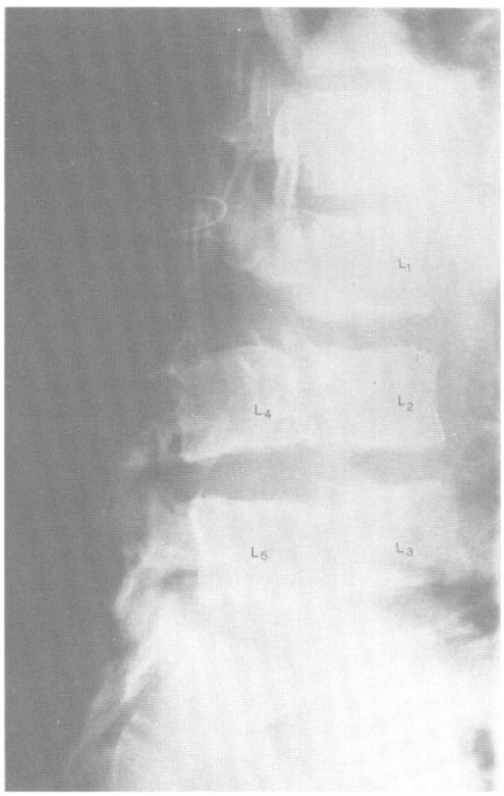

Figure 3 Case 10. Lateral lumbar spine X-ray showing severe L2 and L3 luxation anteriorly to L4 and L5.

cause an inadvertent neurological deficit as in case 3.

Laminectomy is the correct procedure for spinal stenosis as occurred in case 1, but must be associated with some stabilizing procedure when associated with instability. Infection is a possible evolution of a gunshot wound of the spine (case 8). Attention must be paid to osteomyelitis and discitis. There is often contamination of a haematoma in the wound of the spine in these cases.

Case 2 demonstrates the inadequate rescue of a patient with severe trauma to the spine, which also happened to case 10. Inadequate examination in the emergency was the most frequent cause of later neurological deficits in patients with spinal trauma in our review, calling attention to the necessity to carry out rehabilitation procedures only after recent and thorough radiological evaluation of the spine.

With this routine, doctors involved in the treatment of the mulitiinjured should appreciate the experiences of doctors in spinal cord injury units, and should protect their patients from having a second injury with a severe neurological deficit and serious consequences for the individual, family and society. In this way they will also protect themselves against being sued for malpractice.

Bibliography

Frankel HL, Hancock DO, Hyslop G, Melzak U, Michaelis LS, Ungar GH *et al* (1969) The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. *Paraplegia* **7**: 179–192.

Fried IC (1974) Cervical spinal injury during skeltal traction. *JAMA* **229**: 181–183.

Harrington PR, Dockson JH (1973) Development and further prospects of internal fixation of the spine. *Ir J Med Sci* **9**: 773–778.

Haiden JS, Weiss MH, Rosemberg AW (1975) Management of cervical spinal cord trauma in Southern California. *J Neurosurg* **43**: 732–736.

Masini M, Neto NHF, Neves EGC (1990) Experience with a spinal cord unit in Brasilia, Brazil. *Paraplegia* **28**: 17–24.

Marshall LF, Knowlion S, Garfin SR, Klauber MR, Eisemberghm, Kopaniky D *et al* (1987) Deterioration following spinal cord injury. *J Neurosurg* **66**: 400–404.

Monro E, Laborde J, Bahniuk E, Bohlman HH, Samson B (1980) Comparison of fixation of spinal fractures. *Clin Orthop* **152**: 303–310.

Soderstrom CA, Brumback RI (1986) Early care of the patient with cervical spine injury. *Orthop Clin North AM* **17**: 3–13.

Steudel WI, Rosenthal, Lorenz R, Merdes W (1988) Prognosis and treatment of cervical spinal injuries with associated headtrauma. *Acta Neurochir* (Wien) **43**: S85–90.

Wagner JC, Chehrazi B (1982) The decompression and neurological outcome in acute cervical spinal cord injuries. *J Neurosurg* **56**: 699–705.