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An unusual case of high cervical spinal cord injury

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Abstract

Isolated spinal cord injuries can rarely be found in patients with no traumatic radiological abnormalities of the spine. Stenoses of the medullary canal and degeneration of cervical spine are the predisposing factors. A case report of a 68-year-old patient is described, who developed quadriplegia with cardiac arrest due to isolated cervical spinal cord injury while jumping on a trampoline. Compressions of the spinal cord with intramedullary and epidural haemorrhage between vertebrae C3 and C6 were observed with no traumatic radiological abnormalities of the spine skeleton.

Key words: spinal cord, spine, radiological abnormality, quadriplegia, cardiac arrest.

Introduction

Trauma to the spinal cord is nowadays a frequent cause of pathology and spinal cord injuries bear potentially devastating consequences [8,10,11]. Some lucky patients recover completely from neurapraxia causing sensorimotoral dysfunction with no neurological sequelae. However, the most frequent outcomes are paraplegia and quadriplegia as spinal cord injuries are irreversible with no ability to restore function [3,11].

Usually, injuries to the spinal cord are associated with damage to other structures surrounding the tender spinal cord, the most common being vertebral fractures and dislocations, disc protrusions with compression of the cord and severe damage to ligamentous apparatus [2,11]. However, spinal cord lesions can be sometimes found in patients demonstrating no concomitant radiological abnormalities. Pre-existing stenoses of medullary canal and degenerative changes of the cervical spine are important predisposition factors [2,6]. Although injury to the spinal cord without osseous damage is well documented in the paediatric population, it is infrequently reported in adults. Due to the rarity and absence of demonstrable osseous damage on radiograph the diagnosis and management are aggravated [1,5,7]. To date, we found in the literature only one case of a quadriplegic patient (73-year-old) suffering from a similar injury without concomitant osseous damage, who eventually improved after conservative treatment [12]. However, this was not the case in our patient. We present a case report of a 68-year-old gentleman who suddenly collapsed during jumping on a trampoline and after successful cardiopulmonary resuscitation developed irreversible

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quadriplegia as a result of an isolated injury to high cervical spinal cord.

Case report

A 68-year-old man, suffering from arterial hypertension but otherwise in good health, was urgently admitted to the emergency department due to a sudden loss of consciousness. At home, after the first jump on the trampoline he fell onto the ground and was unconscious. There was no associated injury to the head or neck observed and only slight flexion and extension of the neck was reported by the relatives. According to the history, the gentleman simply collapsed following a trivial trampoline jump. The relatives immediately started cardiopulmonary resuscitation (CPR). Upon arrival of the emergency team, the pulse was palpable with no respiration observed. The Glasgow Coma Scale (GCS) was rated at 3. There was no clear reason for collapse and respiratory arrest. Few hours later, on regaining consciousness in the hospital, the patient was noted to be quadriplegic.

Laboratory findings were uneventful. The computed tomography scan (CT) of head and spine was normal apart from distinctive degenerative changes with vertebral osteophytes on all vertebrae (cervical osteoarthritis) and degenerative narrowing of the spinal canal at C3 and C4 level, as well as minimal anterior atlanto-axial dislocation on lateral atlanto-axial joints. Median atlanto-axial joint was intact. No lateral dislocations at atlanto-occipital and the lateral atlanto-axial joints were confirmed (Figs. 1 and 2). Magnetic resonance imaging (MRI) of cervical spine (Figs. 3 and 4) showed a rupture of the tectorial membrane and transverse ligament of the atlas with an 1.2 cm epidural haematoma between the dens axis and the spinal cord, with no compression of the cord. A minor haematoma at the apex of the dens axis and a 4 mm contusional mid-cord haematoma at C1 level with a diffuse oedema of the cord extending from medulla oblongata to the C3 level were seen. Vertebral fractures were not confirmed, nether on following images days later. Angiography, which was done in order to elucidate the epidural component of the intra-axial bleeding, was negative. Controlled functional X-ray imaging (fluoroscopy) of the cervical spine, which is very controversial in such cases, was not taken due to instability of the patient and imminent cardiac arrests.

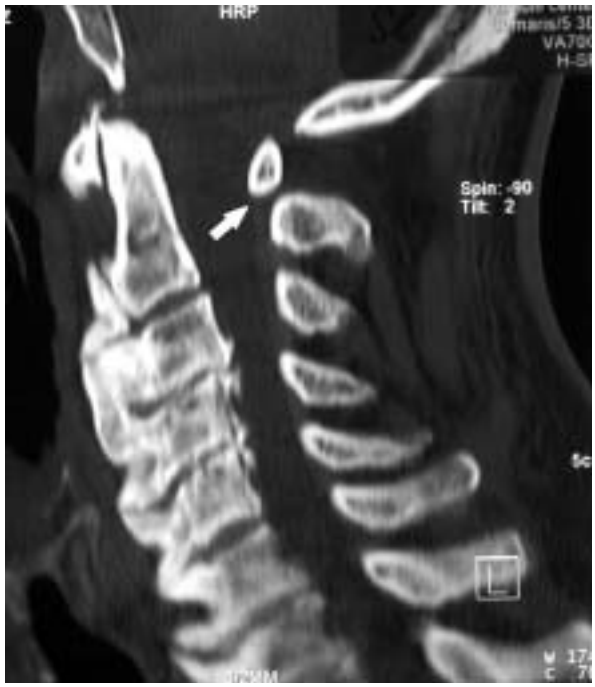

Fig. 1. A sagittal CT scan of cervical spine showing vertebral osteophytes and minimal anterior atlanto-axial dislocation (arrow).

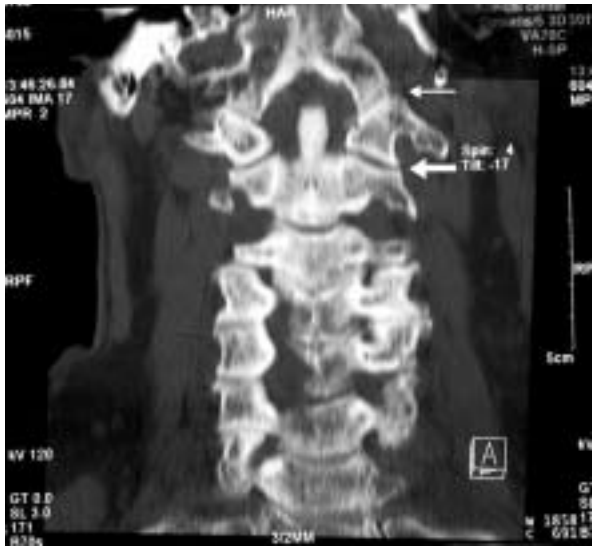

Fig. 2. A coronal CT scan of the cervical spine. There are no lateral dislocations at the atlanto-occipital and lateral atlanto-axial joints (thin and thick arrow, respectively).

Neurological examination revealed complete sensory loss below C3 and partially at C2, inability to raise the head and quadriplegia with weak myotatic reflexes. Muscle tone in flexors of the knees and feet

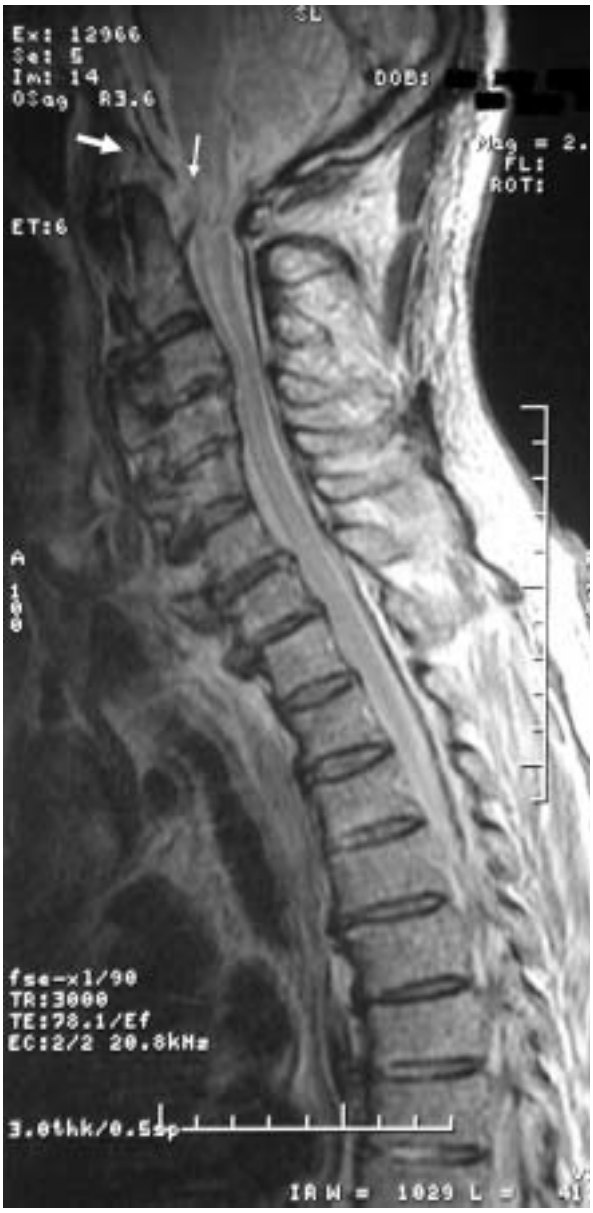

Fig. 3. A sagittal T2 weighted spin echo image, TR 3500, TE 91, showing a ruptured transverse ligament of the atlas and tectorial membrane (thin arrow) with an epidural haematoma between the dens axis and the spinal cord. A minor haematoma at the apex of the dens, probably due to the rupture of the apical dental ligament and the superior longitudinal band of the cruciate ligament (thick arrow) can also be seen. A high signal spot in the centre of the cord at the level of C1 is a contusional haematoma, surrounded by oedema form medulla to C3 level. Prominent ventral spondylosis as well as degenerative stenosis of the spinal canal at the C3/4 can be seen, with a mild compression of the cord.

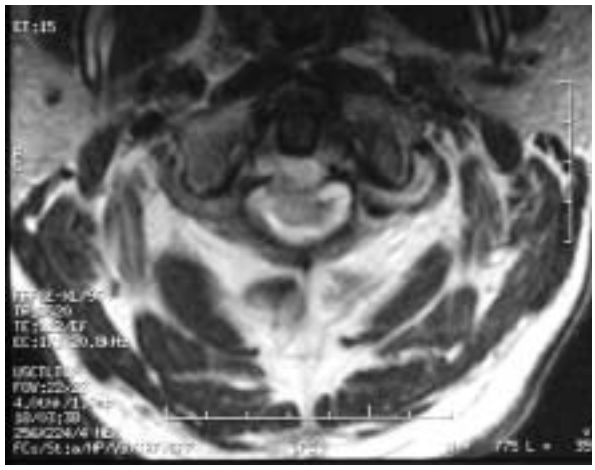

Fig. 4. An axial T2 weighted spin echo image, TR 3520, TE 112. An epidural haematoma with no compression of the cord at the level of C1 and a rupture of the transverse ligament of the atlas is evident.

was increased and plantar responses were extensor. He was conscious with intact cognitive function, mimics, swallowing and whisper but required continuous artificial ventilation. During the hospitalisation, the patient experienced recurrent cardiac arrests with successful CPRs. Unfortunately, neurological status was unchanged during the entire duration of the hospitalisation. On discharge the patient was contactable and was breathing with the help of an intermittent positive pressure respirator. He was transferred to a specialised care centre.

Discussion

The sudden and dramatic development of respiratory arrest and quadriplegia while the gentleman was jumping, strongly points to mechanical injury to the spinal cord. The cervical spinal cord can rarely be injured without concomitant fractures to vertebrae or damage to discs [2,6,9]. These injuries are more frequent and better explained in the paediatric population. They result from spine flexibility and ligamentous laxity with resultant spontaneous reduction after slight displacement [1]. With age the spine flexibility is reduced and injuries are more likely to occur [2,5]. The presence of degenerative changes such as spondylosis, posterior ligament ossification or narrowed spinal canal, are the pre-disposing factors [6]. Our patient had radiographically demonstrable nar-

rowing of the spinal canal at C3 and C4 level and clear osteophytic changes on all vertebrae, typical for osteoarthritis. We believe these were the main contributory reasons for the injury. However, the exact cause of the injury remains unclear. We are proposing three possible mechanisms. As already proposed by Bhatoe [2], we suggest the following: the spinal canal and its constituents are deformable to some extent within normal range and mechanics of movement. During jumping the spinal cord is subject to compression and stretch. In our case, the short-time non-physiological movements with excessive amplitudes of spine hyperextension and hyperflexion exceeded the elastic capacity of the transverse ligament and the tectorial membarne. This resulted in rupture of the ligaments and in the compression of the spinal cord with damage to the blood vessels that were possibly sclerotic and thus less elastic in the patient of this age, culminating in contusional intramedullary cord haematoma at C1 with surrounding oedema and an epidural haemorrhage between the dens axis and the spinal cord. Although the latter is not typically present in the central cord syndrome, it is characteristic for patients with degenerative changes of cervical spine [6,9,11], as was observed in our patient. There was no subluxation at the median atlanto-axial joint, probably due to a tamponade exerting effect of the epidural haematoma. Prominent degenerative changes of the vertebrae may have also caused damage to the spinal cord during sliding of the cord along the canal when jumping, being sufficient for such a grave injury. According to the fact that there were no vigorous but only slight neck movements reported in the history, another possible explanation of the injury is that the patient had stiffened his neck muscles as a natural reaction to the anticipation of the jump. In the lowest aspect of the jump, the muscular contraction along with the inert weight of the head may have induced the rupture of the transverse ligament and the tectorial membrane and a transient disc compression and herniation that severed the spinal cord. Together, this may have evoked vessel damage with resultant intramedullary and epidural bleeding.

As the cervical part of the spinal canal is narrow, the cord can be pinched between posterior longitudinal ligament and ligamentum flavum between vertebrae C3 to C6, causing oedema and haemorrhage [2,5]. This partially concurs with the location of damage described in our patient.

Patients with high cervical spine injury require intermittent positive pressure ventilation due to exclusion of diaphragmal muscle. They also experience hypotension and bradycardia which can cause cardiac arrest [4,11]. The mechanism for reflex cardiac arrest in quadriplegia patients has been described by Frankel *et al*. [4] and during hypoxia the prevention of compensatory sympathetic activity due to cervical cord lesion is suggested to be the cause.

Isolated spinal cord injuries with no dislocations or fractures are suggested to be managed conservatively [2], as was done in the described case. Koyanagi *et al*. [6] however, suggested an operative approach instead supportive treatment. Although Bhatoe [2] reports good outcome over some weeks, for most of the patients with isolated spinal cord lesions, recovery after all but mild injuries is unlikely [8,5,9,11].

Conclusions

Isolated spinal cord injuries without evident radiological abnormalities of vertebral column are very rarely encountered in adults. The most important are stabilisation of vital functions, appropriate transport, immediate radiological studies with CT, MRI as well as x-rays, high dose corticosteroid anti-oedema therapy and supportive treatment. In case of a clear spinal cord compression through a bone fragment, haematoma or prolapsed disc, a surgical intervention is necessary. No relevant compression was found in our patient and under conservative treatment, some recovery may be expected.

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