Eur Spine J
DOI 10.1007/s00586-016-4769-9

GRAND ROUNDS

Increased intrathecal pressure after traumatic spinal cord injury: an illustrative case presentation and a review of the literature

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Received: 10 June 2016 / Revised: 15 August 2016 / Accepted: 2 September 2016

Abstract

Purpose Early surgical management after traumatic spinal cord injury (SCI) is nowadays recommended. Since post-traumatic ischemia is an important sequel after SCI, maintenance of an adequate mean arterial pressure (MAP) within the first week remains crucial in order to warrant sufficient spinal cord perfusion. However, the contribution of raised intraparenchymal and consecutively increased intrathecal pressure has not been implemented in treatment strategies.

Methods Case report and review of the literature.

Results Here we report a case of a 54-year old man who experienced a thoracic spinal cord injury after a fall. CT-examination revealed complex fractures of the thoracic spine. The patient underwent prompt surgical intervention. Intraoperatively, fractured parts of the ascending Th5 facet joint were displaced into the spinal cord itself. Upon removal, excessive protruding of medullary tissue was observed over several minutes. This demonstrates the clinical relevance of increased intrathecal pressure in some patients.

Conclusion Monitoring and counteracting raised intrathecal pressure should guide clinical decision-making in the future in order to ensure optimal spinal cord perfusion pressure for every affected individual.

Keywords Spinal cord injury · Spine surgery · Intraspinal pressure · Outcome

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Case presentation

A 54-year-old man was transferred to our trauma center after falling from a tree (approximate height 3 meters). The man was paralyzed from the level Th4 downward immediately after the fall. Initial body computer tomography (CT) revealed a predominant chest trauma with complex fractures of the thoracic spine (Fig. 1a–d), a sternal fracture, multiple rib fractures and pulmonary contusions. Focused Assessment with Sonography for Trauma (FAST)

Published online: 21 September 2016

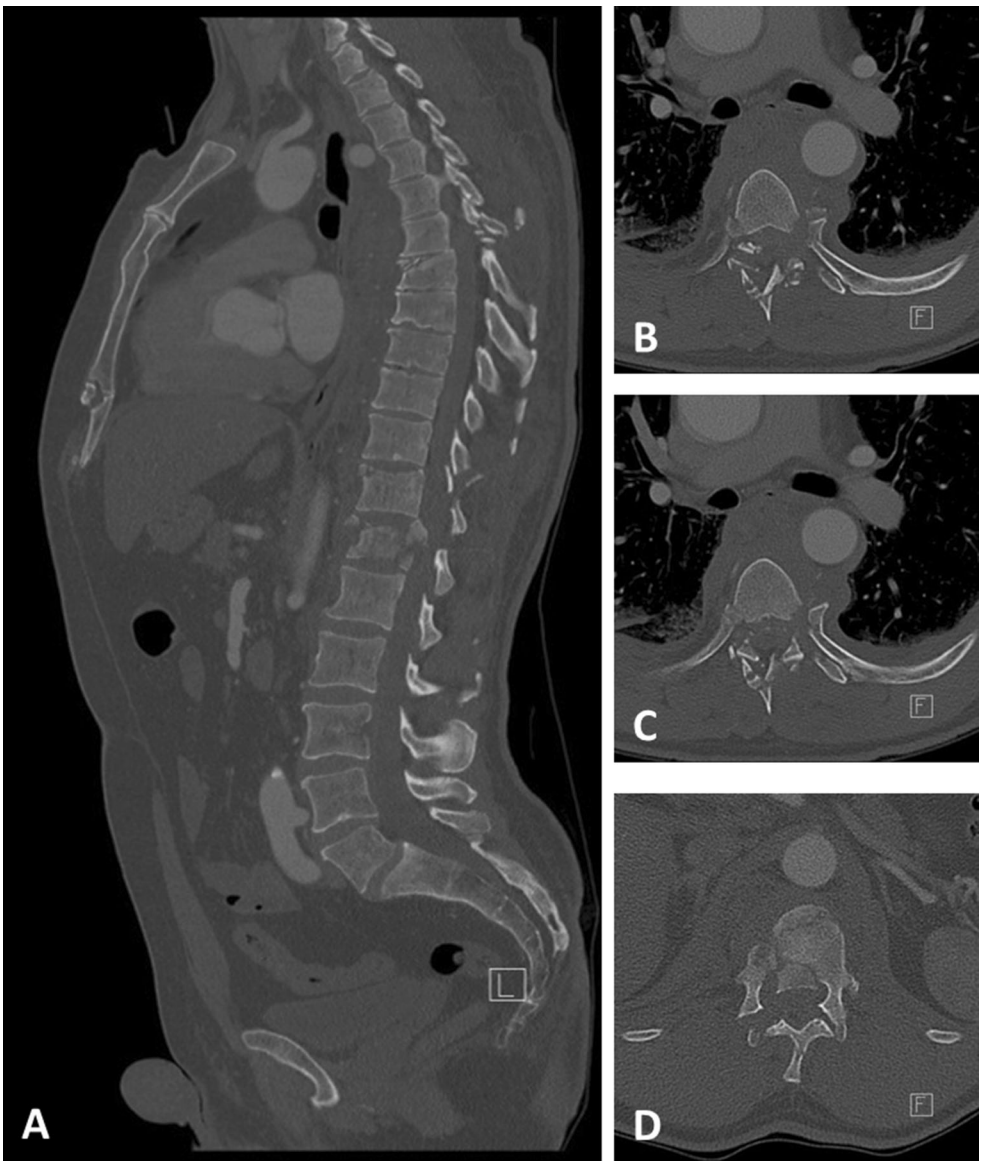

Fig. 1 Initial computer tomography showing complex fractures of the thoracic spine. **a** Sagittal CT of the vertebral column. **b**, **c** Axial CT at the level Th4/Th5. **b** shows displaced facet joints in the spinal cord. **d** Axial CT at the level Th12

was unremarkable. The patient underwent prompt surgical management via dorsal stabilization and decompression. Intraoperatively elevated intraspinal pressure (ISP) after traumatic spinal cord injury (SCI) was observed vividly at the site of dural laceration (Fig. 2).

Diagnostic imaging

The initial body CT scan revealed multiple fractures of the thoracic spine [most importantly: T5-T6: B2 (T6: A1; F4, F3; N4) and T11-T12: B2 (T11: A1; T12: A4; F4; N4) according to the AO classification system] (Fig. 1a–d). After surgery, the patient underwent routine CT examination of the thoracic spine to ensure correct positioning of the inserted internal fixation (Fig. 3) as well as magnetic resonance imaging (MRI) to verify sufficient osseous decompression of neural elements within the vertebral canal. Radiologic follow-up (MRI and X-ray of the thoracic spine) was carried out in regular time intervals according to institutional guidelines.

Historical review

The concept of increased ISP after SCI or due to other pathologic conditions is not entirely new. However, to the best of our knowledge, incision of spinal meninges was never performed routinely after traumatic SCI. Harvey Cushing incised the pia mater in a patient with

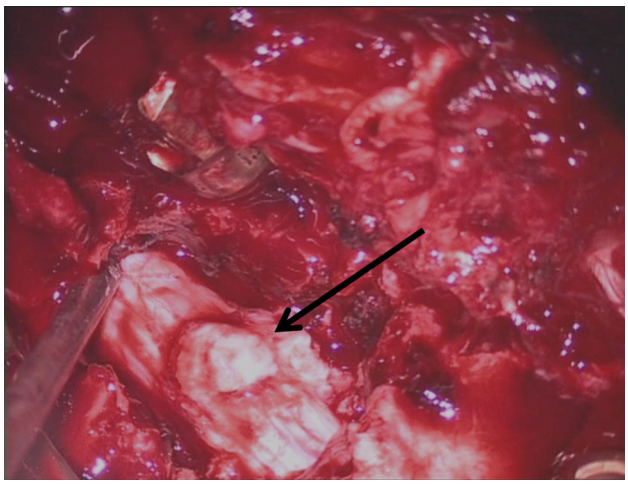

Fig. 2 Intraoperative image showing protruding spinal cord tissue (*arrow*) due to increased intraspinal pressure

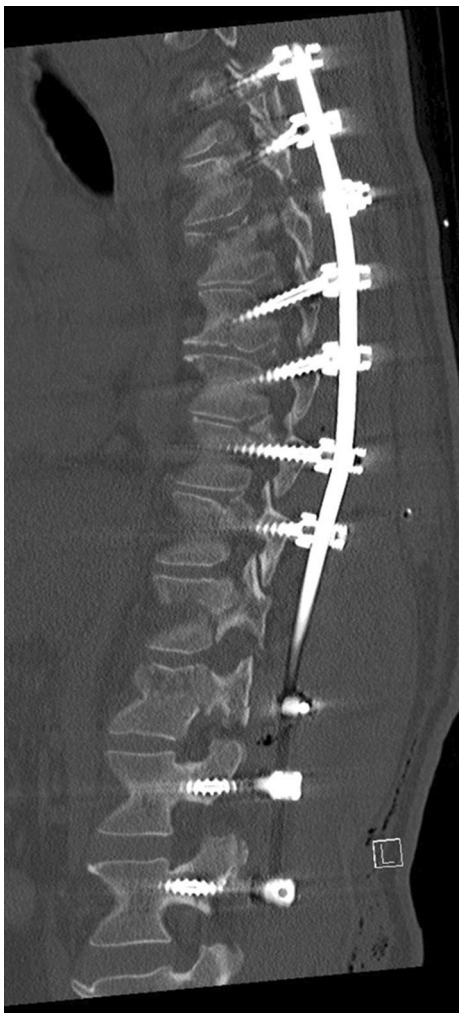

Fig. 3 Postoperative sagittal CT showing correct positioning of the internal fixation

an intramedullary tumor in 1905 and did recognize significant improvement afterwards [1]. Elsberg further applied this concept in a proposed two-stage procedure for intramedullary tumors, where piotomy was performed in the first session. Perkins and Deane performed durotomy in six patients after traumatic SCI with "tense" dura and decreased cerebrospinal fluid pulsations. They reported remarkable neurologic recovery [2]. A more recent case series of initially clinical complete SCI patients reported neurological benefit of "intramedullary decompression" via removing arachnoid adhesions and intraparenchymal necrotic areas [3]. The additional role of durotomy or even piotomy with all potential benefits and complications still needs to be investigated in future studies.

Procedure

After completing diagnostics in the emergency room, the patient underwent prompt surgical management via dorsal open stabilization from Th4 to L2 (Longitude, Medtronic) with laminectomies at the level Th6 as well as Th11 and Th12. Extensive dural laceration was observed at the level Th6. One fragment of the dislocated facet joint was displaced into the thoracic spinal cord (as seen radiologically in Fig. 1b). Intramedullary osseous fragments within the spinal cord were cautiously removed. Afterwards progressive protruding of medullary tissue was observed for several minutes (Fig. 2). The dura was subsequently closed with single sutures.

Outcome/follow-up

There were no complications related to the surgery. Early rehabilitation was started at the intensive care unit and continued at the institutional Center for Spinal Cord Injuries. The neurologic status did not change until discharge and upon follow-up 1 year after the trauma (complete injury – AIS A – below the level of Th4 according to the ISNCSCI protocol – International Standard for Neurological Classification of Spinal Cord Injury).

Rationale for the treatment and evidence based literature

Maintaining adequate spinal cord perfusion pressure (SCPP) is crucial after traumatic spinal cord injury (SCI). Therefore, a mean arterial pressure (MAP) of 85–90 mmHg over the first week after the insult is

recommended [4, 5]. SCPP is computed as MAP minus ISP. Intramedullary edema within the spinal cord and consecutively raised intrathecal pressure at the injury level are important secondary injury mechanisms in the pathobiology after traumatic SCI [6, 7]. Increased ISP reduces spinal cord perfusion pressure, which leads to worsening of posttraumatic ischemia [8]. However, the relative contribution of medullary edema after SCI is less clear compared to the current understanding of the pathophysiology and treatment strategies of brain edema after traumatic brain injury (TBI) [9]. It has been shown that increased swelling with consecutive intracranial pressure (ICP) elevation after TBI worsens the outcome with increased morbidity and mortality [10, 11].

Further, early surgical management after SCI is nowadays recommended as early surgical intervention have been shown to improve neurologic and functional outcome [12–14]. The beneficial effect has been partially attributed to the osseous decompression of neural elements, which should theoretically improve SCPP [15]. However, a significant amount of patients does not respond to early surgical management. The meninges themselves may cause significant cord compression after traumatic SCI [16] leading to an increase of ISP. Further, the value of osseous decompression to decrease elevated ISP at injury site without durotomy followed by subsequent duroplasty to improve SCPP has been questioned recently [17, 18]. Here we describe a case where increased intrathecal pressure can be observed vividly in a patient with a severe thoracic SCI with dural (including pial) laceration. This case presentation supports recent concepts described below.

The relevance of increased intrathecal, most importantly intraparenchymal pressure after SCI has probably been underestimated. Several contributors to the raised ISP at the injury site as well as in adjacent segments have been identified in laboratory and more recently in clinical studies [6, 16, 18–22].

Peak pressures in the spinal cord were higher compared to the brain in a cadaveric study using a simulated model of edema [23]. One potential explanation might be, that the spinal cord possesses less space reserves compared to the brain. This again highlights the importance of counteracting increased intrathecal pressure. Until recently, clinicians faced the dilemma of not being able to monitor ISP. Since a considerable ISP interpatient variability is most likely present [24], statements about the actual and more importantly the optimal SCPP were not possible. This is especially delicate in the posttraumatic situation with impaired autoregulation and concomitant increased ISP. Over the last couple of years, Papadopoulos et al. significantly bridged this gap. First of all, they introduced a method, where a probe is inserted subdurally at the injury site [17]. They identified three different compartments (above, below and at the injury site). Pressure was highest directly at the injury site, indicating the need for measurement at this site [19]. Interestingly, subdural and intraparenchymal pressure values were comparable at the injury site, when the spinal cord is compressed against the dura. Hence, they concluded that subdural probe insertion seems to be sufficient in comparison to TBI, where most probes are inserted intraparenchymally for ICP monitoring [19]. Furthermore, ISP monitoring after SCI seems to be accurate and safe in this study population [24]. These studies already gave important new insights into the routine management during the acute phase. The supine position, for instance, should be avoided in laminectomized patients. Hence, reduction of surgical wound compression as for example by a ring-shaped pillow has been suggested [24].

As shown in our case, the spinal cord may be compressed against the dura due to intrinsic swelling. According to a recent study via assessment of magnet resonance images shortly after the trauma, this happens in approximate one quarter of SCI patients [16]. It seems that the dura itself causes a “compartment-like” syndrome in a significant amount of individuals after SCI. Hence, duroplasty might be indicated in some cases. The “Injured Spinal Cord Pressure Evaluation Study” indicated that patients who had additional dorsal midline durotomy followed by duroplasty after the internal fixation and laminectomy showed a lower ISP and higher SCPP as well as improved vascular reactivity in patients compared to patients where the dura was left intact [18].

However, the role of the relatively firm pia mater should not be underestimated. As mentioned above, once lesioned spinal cord gets edematous, medullary tissue gets compressed against the dura making subdural and intraparenchymal pressure comparable as intramedullary pulsations are transmitted via the subdural probe [19]. Noteworthy, the pulse pressure difference was twofold higher within the spinal cord compared to the subdural space [19]. One potential explanation might be that the pia mater itself is responsible for significant resistance. The pia possesses considerable stiffness, which potentially affects biomechanical properties [25]. As mentioned, peak pressures at the cervical and thoracic spinal cord were significantly greater than in all other measured brain locations. ISP immediately decreased after performing a dorsal midline piotomy [23].

Posttraumatic edema seems to develop via a pleiotropic route. Disrupted ion and water homeostasis are important sequels after SCI [26]. Raised intrathecal pressure seems to be mainly influenced by edema and hemorrhage in a time-dependent manner. According to an animal study, increased ISP is initially driven by hemorrhagic

components, whereas vasogenic edema becomes the primary contributor after 3 days post-injury. Intrathecal pressure began to rise after 5 h, reached a maximum after 3 days and gradually declined over the first week, where pressure ratios were still elevated compared to sham animals [6].

On a cellular level, robust experimental data indicates that Aquaporin-4 (AQP-4), a water channel protein exclusively expressed in spinal astrocytes [27], plays a significant role in the development of edema [7]. The overexpression of AQP-4 is triggered by posttraumatic ischemia [28]. Interestingly, knockout of AQP-4 lead to decreased intramedullary water influx and to a better outcome in locomotion in a mouse model of SCI [7]. Additionally, a variety of inflammatory factors after SCI contribute to vasogenic edema via increasing the permeability of the blood spinal cord barrier [29]. Hence, pharmacological agents counteracting edema formation might decrease tissue damage. Together with appropriate surgical management, increased tissue survival may be promoted.

Cerebrovascular autoregulation has been studied extensively. Direct translation to the spinal cord is probably not as straightforward as it seems. Neurologic compromise after SCI is partially related to decreased spinal cord perfusion [30]. A direct correlation between spinal cord blood flow and improvement as measured via electrophysiological outcome measurements has been postulated in an animal study [31] as well clinically in a recent human study [17]. SCI leads to a disturbed autoregulation within the spinal cord [32]. This is part of the rationale to increase MAP after SCI, which is nowadays recommended [5]. Noteworthy, one should remain cautious as extensively elevated MAP has been shown to induce marked adjacent hyperemia without improving SCPP [33]. Further, changes in pCO_2, volatile anesthetics (sevoflurane) and mannitol did not have a significant effect on SCPP or ISP in patients with acute traumatic SCI. Increase in inotrope dose, however, was capable of increasing SCPP significantly after trauma with altered autoregulation [17].

In summary, this case report challenges current treatment paradigms after acute traumatic SCI. In some patients osseous realignment and laminectomy do not seem to be sufficient. The potential beneficial effect of durotomy or even piotomy needs to be elucidated in the future in association with a potential higher surgical complication rate. As established in TBI [34], management after SCI should be guided individually according to the ISP as well as SCPP as considerable interpatient variability has been noticed [24].

Compliance with ethical standards

Conflict of interest None.

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