Is preemptive decompression of an asymptomatic spinal epidural hematoma justified?

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Spinal epidural hematoma, an extremely rare entity, has a variable presentation. Delayed diagnosis and treatment can result in permanent neurologic deficit and even death. We describe a case of a young man presenting as a polytrauma victim whose injuries included a Chance fracture of the thoracic spine with associated asymptomatic spinal epidural hematoma.

Case report

A 22-year-old man, who suffered from epilepsy and had a mental age of 8 years, was ejected during a single-vehicle rollover crash. He sustained bilateral lung contusions, fracture of the right ipsilateral hip and femoral shaft, right navicular fracture, a Chance fracture of the T7 vertebra and a compression fracture of superior end plate of the T8 vertebra. Because he was agitated he was intubated to facilitate transfer to the tertiary care facility. On arrival, he underwent CT of the thoracic spine, which, in addition to the fractures of T7 and T8 (Fig. 1, top), showed a small noncompressing epidural hematoma at the level of T7 (Fig. 1, bottom). The patient was transferred to the intensive care unit where he was later extubated. He was alert and neurologically intact at that time. He underwent antegrade reconstruction with femoral nailing the following day. His navicular fracture was treated nonoperatively. Postoperatively, he bled significantly from his wounds, and when his hemoglobin decreased from 100 to 66 g/L, he received a blood transfusion. In addition, his thigh continued to swell. Lower limb angiography, performed to rule out a missed vascular injury, showed no abnormality. We suspected compartment syndrome, so right thigh fasciotomies were performed on postoperative day 1. He did well postoperatively and the fasciotomy wounds were closed after 2 days, at which time his platelet count was quite low (61 000/mL [reference range 140 000–450 000/mL]). Oozing from the surgical incisions decreased and the hemoglobin level stabilized. Anticoagulant drugs for deep venous prophylaxis had not been prescribed at any time during his hospital stay. No neurologic deficits were noted. He was taking sodium valproate as treatment for his epilepsy. He was mobilized in a thoracolumbosacral orthotic brace.

Early on day 9 after his injury, his neurologic findings were normal. That afternoon while sitting in his brace he reported sudden-onset paraplegia. There was no evidence of a fall or further trauma. On clinical examination he had a flaccid paralysis with a T7 sensory level. MRI showed a large epidural hematoma extending from T6 to T8, centering on T7. This had caused significant compression of the spinal cord at T7 (Fig. 2, top), and on the sagittal view (Fig. 2, bottom) the cord diameter was reduced to 5 mm. There was no change in the alignment of the spine. Decompressive laminectomy from T7 to T8 was performed. Clotted epidural hematoma was found beneath the ligament flavum (Fig. 3). Some of the clot was easily removed with suction and looked fresh; the deeper portion appeared clearly organized and chronic and was tethered to the dura. Complete evacuation of the hematoma was followed by instrumentation and fusion from T5 to T10 with use of pedicle screws and local bone graft. Significant bleeding was encountered during the procedure and required treatment with desmopressin acetate and transexamin acid. The wound was closed over a Hemovac drain. No hematologic abnormality could be detected on extensive investigation.

After decompression, the patient had some recovery in sensory function but no motor recovery. He had no bladder or bowel control. He was discharged 10 days after his spine surgery. At follow-up 3 months later, there was some evidence of motor recovery, with grade 2 power in most muscle groups of the lower extremities and partial return of bladder and bowel sensation and function.

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Discussion

Symptomatic spinal epidural hematoma has been described as being associated with the following: trauma, anticoagulation (iatrogenic, acquired or congenital), vascular malformations, lumbar puncture, spinal anesthesia, tumour, hemorrhage, pregnancy, immune-mediated vasculitis, any activity that requires exertion and arterial hypertension.[1] The source of bleeding is acknowledged by most authors to be the venous epidural plexus. The hematoma is mostly located posteriorly or posterolaterally in the cervicothoracic or the thoracolumbar regions. The reason cited for this is the purported weak points in the epidural venous plexus at these locations.

The age frequency peaks are 15–20 years and 47–75 years. Males are affected more commonly than females. Commonly, the presentation is acute, severe pain at the level of the hematoma. Usually there is a rapid progression, with motor and sensory deficits, to flaccid paralysis associated with bladder and bowel dysfunction. These deficits are usually symmetric, but they can present asymmetrically as Brown-Séquard's syndrome.[2–4]

MRI is the investigation of choice.[5] The hematoma appears as isointense on T_1 sequences. In T_2 sequences, acute hematomas appear hyperdense at the periphery with a hypodense centre. The MRI appearance of the hematoma changes with its chronicity. MRI differentiates spinal epidural hematoma from herniated disc, tumour and infection and demonstrates the complete extent of the pathologic features.

Very few reports describe successful conservative treatment of epidural hematomas. These are mostly reported in patients with trivial, stable or improving neurologic deficits under MRI monitoring.[6–8] Generally, the standard of treatment for symptomatic spinal epidural hematoma has been emergent surgical decompression of the hematoma through a laminectomy, hemilaminectomy or interlaminar decompression. Drainage of the hematoma by puncture is not advised since it can lead to rupture of another blood vessel and is only possible if the hematoma is still in liquid form.[9] The outcome depends on the urgency of decompression, severity of preoperative neurologic deficit, patient's age, location of the hematoma (a more cranial location is associated with a poor outcome) and extent of involvement (involvement of more spinal segments is associated with a poor prognosis).[1] Recovery was significantly improved when decompression was performed within 36 hours in patients with complete deficits and within 48 hours in patients with incomplete deficits.[1,10]

Chance fracture or flexion-distraction injuries result from an anterior force vector acting along an axis of rotation anterior to the middle column of the spine.[11] These injuries can be bony or ligamentous in nature. CT with a sagittal view is useful in delineating the anatomy of the fracture. Chance fractures with bony involvement can be reliably treated with extension casting or bracing. Ligamentous Chance injuries often result in residual instability and pain, so they are best

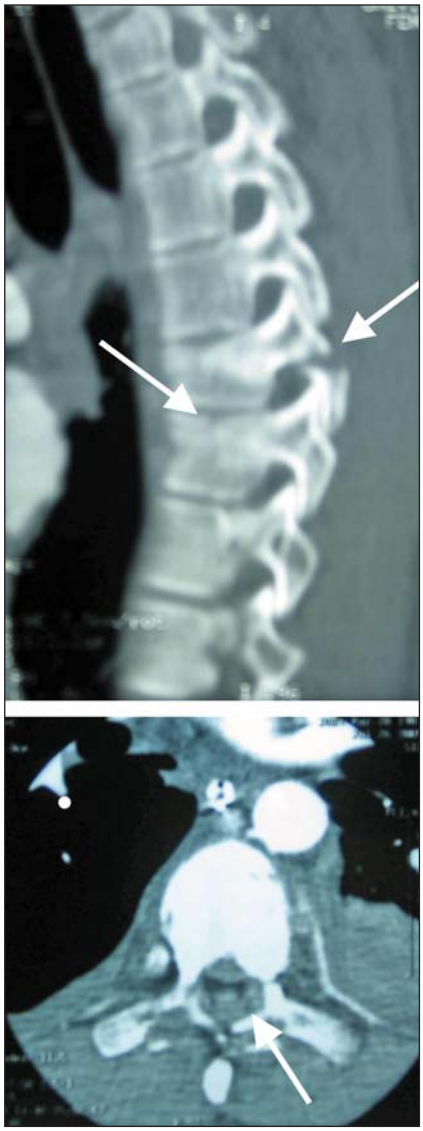

FIG. 1. Top: Preoperative CT scan showing fractures of the T7 and T8 vertebrae (arrows). Bottom: Preoperative axial section at the T7 level showing a noncompressing epidural hematoma (arrow).

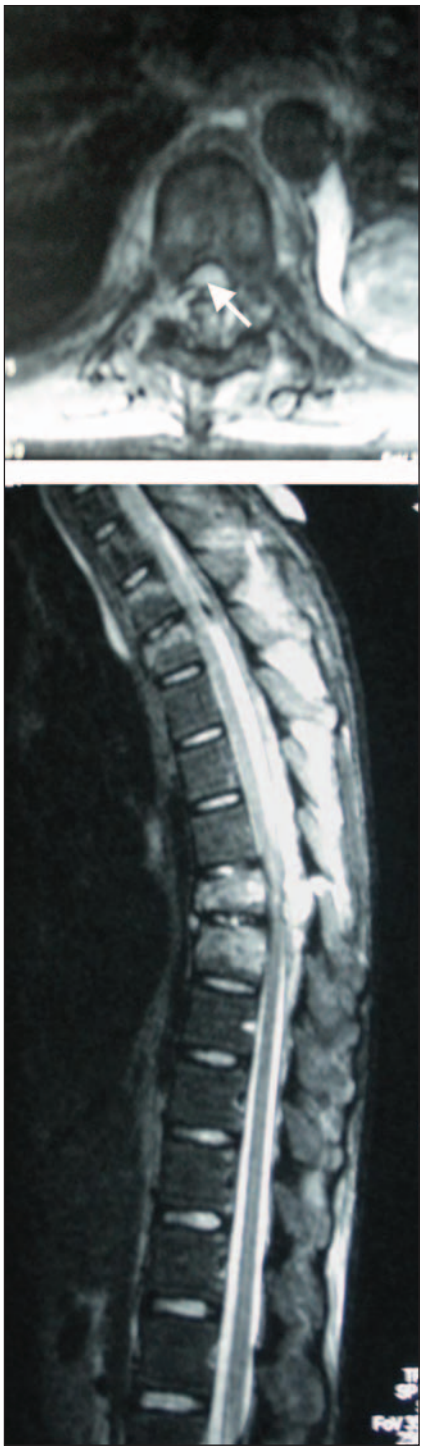

FIG. 2. Top: MRI scan after the onset of paraplegia showing cord compression in the axial section at T7 (arrow). Bottom: Sagittal section shows a posteriorly located compressing epidural hematoma at T7–8.

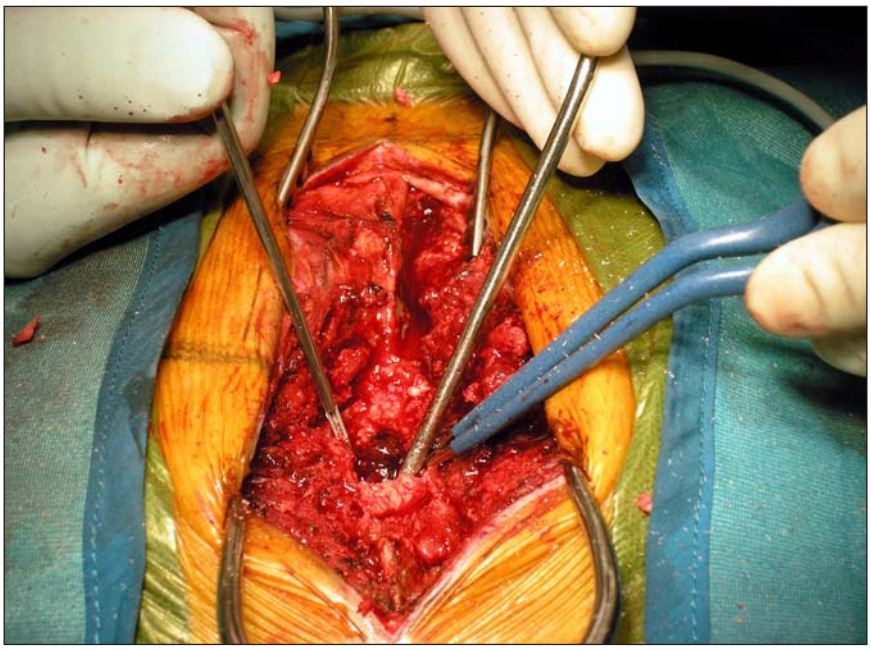

FIG. 3. Intraoperative view of the hematoma after laminectomy and removal of the ligamentum flavum.

treated by a short compression construct and fusion.[12]

The spinal injury in our patient was a bony Chance fracture with asymptomtic epidural hematoma and was therefore amenable to treatment with a thoracolumosacral orthosis. Even after extensive investigations, no hematologic abnormality could be identified to explain his bleeding tendency. Given that his coagulation profile improved by the administration of desmopressin acetate and transexamic acid, we believed that he was suffering from some form of subclinical von Willebrand disease. Sodium valproate is also linked to coagulation disturbances, but Factor XII deficiency or thrombocytopenia was not noted at the time of the paraplegia. Considering the mental age of the patient (8 yr), one possibility is that he might not have reported his neurologic deficits as soon as we would have expected. Second, since the paraplegia did not occur until 9 days after injury, the neurologic examination was reduced to routine monitoring. In view of the patient's mental age, his increased bleeding tendency and the presence of an epidural hematoma (though noncompressing), consideration could be given to preemptive decompression of the spinal hematoma and stabilization. Since our patient had multiple injuries, it could be argued that his spinal injury should be have been stabilized when the femoral fracture was fixed. However, spinal fixation alone without decompression would likely not have prevented progression of the hematoma. Again, it should be noted that no displacement of fracture was seen radiographically. We would therefore consider surgical decompression of even noncompressing spinal epidural hematomas an option when neurologic monitoring is not reliable (patient factors), an abnormal bleeding tendency is demonstrated and, in the presence of above-mentioned factors, if the hematoma is located in the cervical or thoracic regions (spinal cord).

Competing interests: None declared.

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