Management and outcomes of spinal epidural hematoma during vertebroplasty

Case series

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Abstract

Rationale: Spinal cord injury (SCI) is one of the common complications of spinal surgery. There is no definite treatment and time of decompression for spinal cord induced by epidural hematoma during vertebroplasty.

Patient concerns: A total of 6 patients with SCI during vertebroplasty were included in our research. All of them occurred sensory disturbance and motor dysfunction due to a lower or same level operative vertebral body lesion in vertebroplasty.

Diagnoses: Neurological manifestations during vertebroplasty, postoperative magnetic resonance imaging and computed tomography.

Interventions: Once SCI occurred in vertebroplasty, four patients were underwent spinal cord decompression immediately, and two patients were done after 14 and 22 hours, respectively.

Outcomes: Before decompression operation, one patient was Frankel A, three were Frankel B, and two were Frankel C. One day after evacuation of the SEH, three patients recovered to normal neurological function (Frankel E), one to Frankel C, and one to Frankel D, but the other one did not recover. At the last follow-up, five patients had recovered to Frankel E and one patient to Frankel D.

Lessons: According to our experience, when SCI occurs during vertebroplasty, neurological deficits are always secondary to acute SEH. Timely decompression, particularly transfer surgery, can shorten recovery time.

Abbreviations: CT = computed tomography, MRI = magnetic resonance imaging, SCI = spinal cord injury, SHE = spinal epidural hematoma.

Keywords: outcome, spinal cord injury, spinal epidural hematoma, vertebroplasty

1. Introduction

The vertebroplasty technique was first applied to the treatment of osteoporotic vertebral compression fractures by Galibert et al.[1] and has quickly gained impetus worldwide. It requires the placement of a large-bore spinal needle under fluoroscopic guidance into each affected vertebral body via a posterior, transpedicular approach.[2] Some complications, such as pedicular fracture, nerve root injury, and spinal cord lesion, may occur during this puncture process. A spinal epidural hematoma (SEH) is an emergent complication. Without appropriate management, it may result in a poor prognosis and permanent harm.

2. Materials and methods

From March 2012 to September 2016, 6 patients with a SEH were treated for spinal cord decompression. All fractured vertebral bodies were thoracic vertebra. Neurological outcomes were assessed via the Frankel system.[3] Four of them (No. 1–4) were transferred for decompression operation when they incurred neurological deficit symptoms (Frankel B to C) due to a lower or same level operative vertebral body lesion during vertebroplasty. One patient (No. 5) incurred the symptoms of SCI during vertebroplasty. Physical examination after SCI resulted in SCI of Frankel D after the vertebroplasty. These deteriorating symptoms were the result of spinal cord compression by a SEH, seen upon computed tomography (CT). Decompression surgery was then performed 14 hours after vertebroplasty. Patient No. 6 evidenced neurological deficits during the vertebroplasty; therefore, the operation was stopped. The neurological deficit was rated as Frankel B, but not managed. The patient was transferred to our hospital immediately, and an SEH was confirmed via emergency magnetic resonance imaging (MRI). Surgery was then performed 22 hours after the initial vertebroplasty and the SCI was rated as Frankel A at that time (Table 1).

2.1. Statement of ethics

This study was approved by the ethical committees of Second People's Hospital of Chengdu. All aspects of our research were in compliance with applicable ethical standards, and informed consent was also obtained from all patients enrolled in the study. We obtained written informed consent from all participants.
and edema around the axon and myelin sheaths. Pressure to spinal cord causes necrosis in the central region of the spinal cord, are easily subjected to direct compression. Compression of the canal decreases further, and the spinal cord and other structures in the thoracic spinal canal, the effective area of the thoracic spinal buffer space. When an acute epidural hematoma forms in the spaces, resulting secondary hematoma can cause further neurological injuries often result in fracture or nerve root damage, and a catastrophic complications. In one report, the incidence of SCI in vertebroplasty is often considered moderately severe. Needle traversal of the lamina instead of the pedicle can occur, especially in the thoracic vertebra, where pedicles are smaller, and can lead to catastrophic complications. In one report, the incidence of SCI in vertebroplasty was 2.21%; meanwhile, the incidence of injury caused by cement leakage was 1.02% and puncture and puncture-related injuries 0.51%. Puncture and puncture-related injuries accounted for 23.08% of all neurological injuries. Puncture injuries often result in fracture or nerve root damage, and a resulting secondary hematoma can cause further neurological deficits. According to our clinical experience, lower extremity pain, numbness, and weakness are among the most common symptoms of SCI caused by an SEH during vertebroplasty. The anatomy of the thoracic vertebral canal is specialized, meaning that the diameter of the canal is small with very limited buffer space. When an acute epidural hematoma forms in the thoracic spinal canal, the effective area of the thoracic spinal canal decreases further, and the spinal cord and other structures are easily subjected to direct compression. Compression of the spinal cord causes necrosis in the central region of the spinal cord, and edema around the axon and myelin sheaths. Pressure to the spinal canal affects local blood vessels and causes dysfunction in feeding and draining. This dysfunction can lead to the accumulation of harmful substances and a secondary cascade of deleterious biochemical and cellular processes. Previous work in animal models demonstrated that recovery of neurological function is related to the degree of spinal cord compression, duration of hemorrhage, and the rate of development of neurological deficits. Functional recovery of the spinal cord further depends on the size of the spinal cord and the duration of the compression event. Survival time of spinal cord tissues under high pressure was 1 minute, although this was extended to 2 hours under relatively small amounts of pressure. When spinal cord compression exceeds these limits, complete recovery becomes nearly impossible. However, when spinal cord compression occurs progressively rather than acutely, the degree of nerve recovery can be increased.

The parameters determining the severity of SCI are multifactorial and include rate, depth, and duration of compression. An SEH causes clinical symptoms, such as lower limb paralysis and cauda equina symptoms. An SEH is a neurosurgical emergency, and often leads to devastating sequelae unless it is managed effectively. Controversy exists between those who advocate emergency surgery and those who operate on an urgent rather than emergent basis. Some scholars argue that the recovery of spinal cord function is not dependent on the timing of surgical intervention but that a patient’s preoperative neurological status is the most important factor in predicting their outcome. Some report that outcomes are better in patients with an incomplete neurological deficit and timely surgical decompression. Dolan et al. argue that the time of spinal cord compression and the magnitude of pressure are negatively correlated with the recovery of neurological function. McQuarrie confirmed the possibility that delayed surgery reduces nerve recovery. Collectively, these studies indicate that both the extent of neurological deficits at the time of surgery and a minimal interval from symptom onset to surgical decompression are key factors in predicting a patient’s functional outcomes.

In the cases presented here, the 4 patients who were transferred to open surgery recovered better and more quickly than the 2 who were not. Neurological function deteriorated after the SCI that occurred in patient No. 5 and 6. Definite diagnoses were made on the basis of CT/MRI. Patient No. 5 exhibited poor neurological function (Frankel C) at the time of decompression. While SCI recovered to normal, the recovery time was significantly prolonged (6 weeks) when compared with the other 4 cases (No. 1–4). The SCI of patient No. 6 was Frankel C during vertebroplasty, Frankel A before decompression, and Frankel D at follow-up (40 weeks). On the basis of these cases, we speculate that aggravation of neurological deficits is related to acute SEH. The time from SCI to decompression operation was critical to the patient’s degree and speed of recovery.

### Table 1

| No. | Gender | Age, y | Fracture level | Interval time, h | During vertebroplasty | Decompression surgery | 24H | Followed-up, w |
|-----|--------|--------|---------------|-----------------|-----------------------|-----------------------|-----|---------------|
| 1   | Female | 77     | T8            | 0               | B                     | B                     | E   | E (8)         |
| 2   | Female | 85     | T10           | 0               | C                     | C                     | E   | E (8)         |
| 3   | Female | 69     | T11           | 0               | C                     | C                     | E   | E (8)         |
| 4   | Male   | 71     | T9            | 0               | B                     | B                     | D   | E (48)        |
| 5   | Female | 74     | T10           | 14              | D                     | B                     | C   | E (48)        |
| 6   | Female | 84     | T12           | 22              | C                     | A                     | A   | D (48)        |

24H = 24 hours after evacuation of the spinal epidural hematoma, Frankel = Frankel system for spinal function evaluation system, Interval time = the operation interval time was defined as time from the onset of spinal cord lesion symptoms to decompression operation, T = thoracic.
Our study has numerous limitations. First, it contained a small number of cases. Second, patients who were transferred for their operations incurred SCI during vertebroplasty, which was diagnosed on the basis of symptoms and physical examination rather than objective evidence (e.g., via CT or MRI).

5. Conclusion
Neurological deficits always occur secondary to acute SEH if SCI occurs during vertebroplasty. Neurological recovery is multifactorial, and the timing of surgery is a key factor in determining postsurgical success. Timely decompression and transfer surgery can shorten the time of recovery. Therefore, exploratory surgery is worth performing in patients who incur SCI during vertebroplasty.

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