

# Bacterial Meningitis After Traumatic Thoracic Fracture–Dislocation: Two Case Reports and Review of the Literature

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**Summary:** Bacterial meningitis occurs rarely in the setting of traumatic spinal injury. Bacterial meningitis after spinal surgery for traumatic thoracic fracture–dislocation injuries has not been reported. We report on two cases of bacterial meningitis in two pediatric patients undergoing posterior segmental instrumentation with pedicle screws after thoracic fracture–dislocation injuries with associated traumatic dural laceration and complete spinal cord transection. Both patients were treated and recovered. Early recognition of bacterial meningitis after traumatic spinal injury enables appropriate and timely treatment to be instituted and can yield a favorable outcome.

**Key Words:** meningitis, spinal injury, fracture dislocation, spinal surgery, traumatic injury

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## INTRODUCTION

Bacterial meningitis is a rare complication of spinal surgery with an incidence of 0.18% after elective surgery.<sup>1</sup> The only reported cases of postoperative bacterial meningitis after spinal surgery secondary to traumatic injury included one patient with thoracolumbar fracture treated with combined anteroposterior fixation and a patient who developed a pseudomeningocele complicated by meningitis after a traumatic L3 burst fracture.<sup>2,3</sup> This study reports two cases of bacterial meningitis after posterior segmental instrumentation with pedicle screws after traumatic thoracic fracture–dislocation with associated traumatic dural laceration and complete spinal cord transection.

## CASE REPORTS

### Case 1

A 17-year-old boy was the unrestrained driver ejected in a rollover motor vehicle crash. On admission, the patient had no voluntary movement of his lower extremities, a closed deformity of the thoracic spine with bony prominence and swelling, and absent

rectal tone and bulbocavernosus reflex. Radiographs were notable for a three-column burst fracture–dislocation at T9–T10 with significant fragment retropulsion into the spinal canal (Fig. 1). The patient was stabilized by the primary trauma team and treated with rigid orthosis until clearance could be obtained for spinal fixation.

Four days after admission, spinal column stabilization was performed with a posterior approach. Intraoperatively, a portion of the vertebral body of T9 was noted to have retropulsed into the spinal canal and transected the spinal cord. Absorbable gelatin sponge (Gelfoam; Pfizer, NY) was used to control bleeding and cerebrospinal fluid (CSF) runoff. Posterior segmental instrumentation with pedicle screws was performed from T5 to L1, avoiding only T9 (Fig. 2). A combination of local autogenous and allogenic bone graft was used. An absorbable gelatin patch was placed over the dural defect at T9 and sealed with a fibrin sealant (Tisseel; Baxter, IL). No CSF leak was observed with induced Valsalva maneuver. A subfacial drain was placed.

The patient developed a fever of 102°F (38.8°C) on the first postoperative day. Computed tomography failed to reveal any fluid collection. Three days later, the patient had a fever of 105°F (40.5°C) accompanied with headache, nuchal rigidity, and photophobia. Fluoroscopically guided cervical puncture between C1 and C2 was performed. The resultant CSF profile revealed a white blood cell count of 3470/μL with 95% polymorphonuclear leukocytes. Gram stain revealed Gram-negative rods, and final CSF culture yielded growth of *Enterobacter cloacae*. The patient was treated with meropenem, gentamicin, and vancomycin.

The patient subsequently underwent six additional irrigation and débridement operations secondary to persistent wound infection and required a local rotational myofascial flap. The patient continued to improve clinically and was transferred to rehabilitation care with 4 weeks of intravenous antibiotics followed by indefinite suppressive oral therapy. He was seen at 3-month follow up and at a 1-year follow up with no wound complications and no change in spinal alignment.

### Case 2

An 18-year-old man was the unrestrained passenger ejected in a motor vehicle crash. On admission, the patient had no voluntary movement of his lower extremities, no gross deformities of the spine, and absent rectal tone and bulbocavernosus reflex. Radiographic imaging revealed a T10 fracture–dislocation with significant retropulsion into the spinal canal (Fig. 3).

Two days after admission, spinal column stabilization was performed with a posterior approach. Intraoperatively, the patient was noted to have a fracture through the posterior elements of T10 and T11 along with severe disruption of the paraspinal muscles around the zone of injury. Pulsatile CSF flow was noted along with complete transection of the dura and spinal cord. The canal was packed with a microfibrillar collagen and a fibrin sealant was placed over the defect. Posterior segmental instrumentation with pedicle screws was

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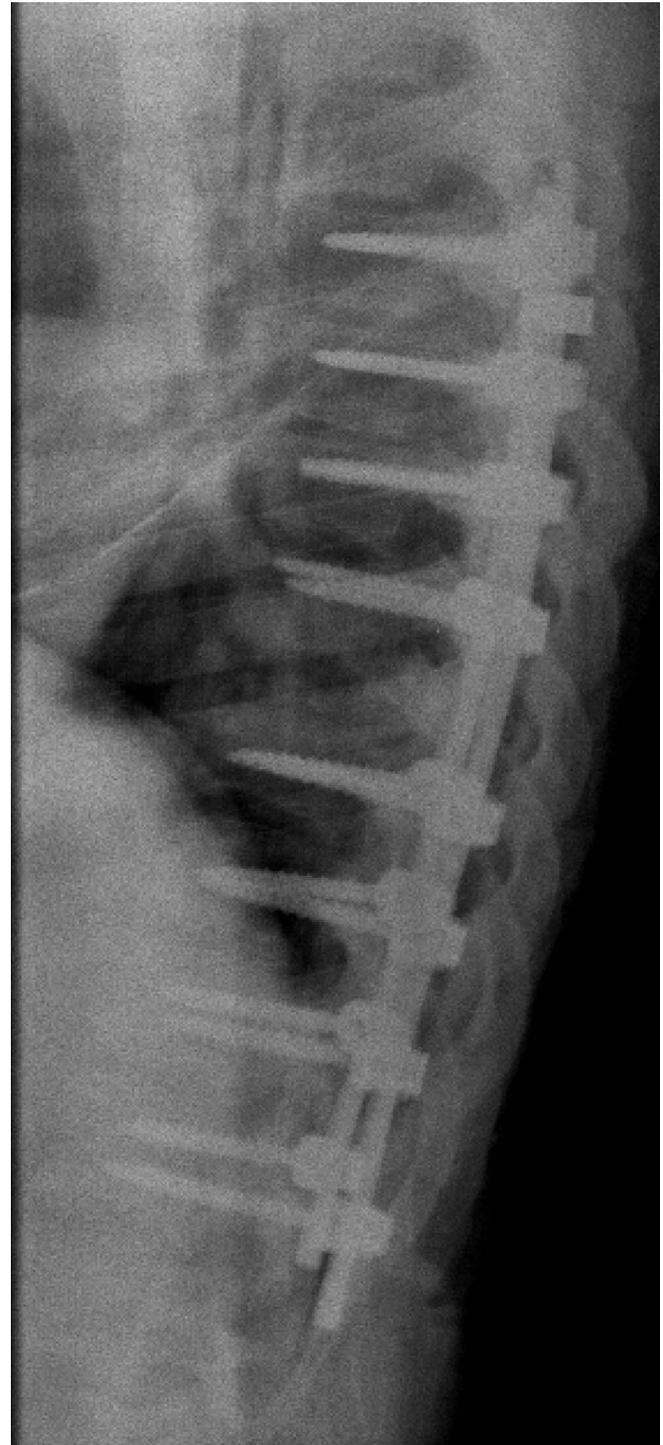


**FIGURE 1.** Sagittal T2-weighted image of Case 1 demonstrating fracture dislocation at T9–T10 with a severe three-column burst fracture of T9 and retropulsed fracture fragment into the spinal canal.

performed from T8 to T12 avoiding only T10 (Fig. 4). A combination of local autogenous and allogenic bone graft was used. A subfacial drain was also placed.

The patient developed drainage from his wound and underwent irrigation and débridement on postoperative Day 8. The fracture site and site of cord transection were explored and CSF was noted to be draining from the spinal canal. Fibrin sealant and Gelfoam were used to seal off the canal and a deep drain was placed. Broad-spectrum antibiotics were administered postoperatively until culture results were finalized as negative.

The patient convalesced until postoperative Day 19 when he became increasingly somnolent and developed nuchal rigidity and photophobia with a fever of 101.3°F (38.5°C). Repeat irrigation and débridement was performed and a CSF leak was again noted. CSF samples were taken intraoperatively and final cultures grew an extended-spectrum  $\beta$ -lactamase producing *Enterobacter aerogenes*.



**FIGURE 2.** Postoperative lateral radiograph of Case 1 demonstrating proper spinal alignment after T5–L1 posterolateral arthrodesis with instrumentation.

Amikacin was administered for 14 days and then an additional 6 weeks of vancomycin and meropenem were given.

The remainder of the patient's postoperative course was complicated by a generalized tonic-clonic seizure and a questionable hypotensive event involving bilateral basal ganglia, which left him



**FIGURE 3.** Sagittal T2-weighted image of Case 2 demonstrating T10 fracture–dislocation with significant retropulsion into the spinal canal.

unresponsive. He was eventually discharged to a rehabilitation hospital. His wound was healed at 6-week follow up and his sensorium continued to improve. He was also seen again at 1-year follow up. He was doing well and has not shown any evidence of spinal instability.

### DISCUSSION

The annual incidence of spinal fracture is 64 in 100,000 patients with 30% of these fractures involving the thoracic spine and 42% involving the lumbosacral spine.<sup>4</sup> Infection



**FIGURE 4.** Postoperative lateral radiograph of Case 2 demonstrating proper spinal alignment after T8–T12 posterolateral arthrodesis with instrumentation.

after operative stabilization of thoracolumbar fractures is relatively uncommon. The incidence of deep infection in thoracolumbar fractures is roughly 5%.<sup>5,6</sup> Infection rates between 0.7% and 10% have been found after elective spinal surgery with instrumentation.<sup>7–12</sup> A higher infection rate is found after surgery for traumatic spinal injuries compared with those undergoing elective spinal surgery.<sup>11,12</sup>

Very little has been reported regarding bacterial meningitis after spinal surgery, and there are no published occurrences after instrumentation for traumatic thoracic spinal

injury. One case of bacterial meningitis was noted after 3216 elective laminectomies (0.03%) in a series by Buckwold et al,<sup>13</sup> whereas Twyman and colleagues<sup>1</sup> reported four cases of 2180 elective spinal operations. Bacterial meningitis has been described as a complication of incidental durotomy during elective spinal surgery and as a complication of scoliosis surgery with instrumentation.<sup>14–17</sup>

Bacterial meningitis can be characterized by the clinical triad of fever, neck stiffness, and altered mental status, which may be present in less than half of adult patients.<sup>18</sup> Presence of two of the four symptoms of headache, fever, neck stiffness, and altered mental status is seen in up to 95% of patients with acute bacterial meningitis.<sup>18</sup> Other associated signs and symptoms include headache, nausea, vomiting, photophobia, seizures, and malaise among others.

Clinical examination can direct the diagnosis of acute bacterial meningitis; however, the final diagnosis is made with CSF analysis. CSF samples and blood cultures should be obtained immediately in the setting of a clinical history consistent with meningitis. CSF analysis should at least include Gram stain, culture, cell count/differential, protein, and glucose to identify the offending organism and confirm the diagnosis.

Immediately after obtaining adequate CSF samples, empiric therapy with broad-spectrum antibiotics should be initiated. Empiric antibiotic therapy often includes a third- or fourth-generation cephalosporin plus vancomycin; in addition, ampicillin should be administered when *Listeria* is a suspected pathogen. Meropenem is sometimes used as an alternative to cephalosporins.<sup>19</sup> Antibiotic selection and clinical management of meningitis can also be aided by consultation with infectious disease and neurology colleagues.

The patients described here were likely at an increased risk of developing meningitis as a result of the presence of a significant and irreparable dural laceration and the higher incidence of postoperative infection in traumatic thoracolumbar fractures. Dural tears are found in up to 10% to 64% of thoracolumbar burst and chance fractures.<sup>20–24</sup> There is no literature to support any one method for managing complete dural transection as was evident in both of these cases. Dural graft material can potentially be used to manage dural tears; however, adequate dural tissue needs to be present to anchor the graft. The dural tissue in these cases would not adequately support dural graft materials.

Potential sources of infection leading to meningitis could include direct inoculation intraoperatively, deep infection from persistent dural leak, or hematogenous spread. The infections involved in these cases were likely secondary to a seeding process initiated after the injury and associated with interventions in the trauma intensive care unit (central venous catheters and chest tubes) to manage associated problems. The timing of the infections after the index procedures makes intraoperative infection less likely and each case involved a highly virulent *Enterobacter* species. Clearly, each of these cases involved more than deep wound infections secondary to persistent dural leaks because each patient displayed classic signs and symptoms of acute bacterial meningitis along with diagnostic microbiologic data.

da Costa et al<sup>16</sup> described late wound infection after spinal instrumentation to be related to one of the following

factors: 1) low virulence bacteria seed the implant hematogenously; 2) instability and/or movement of the implant induces aseptic inflammation; and 3) the implant becomes inoculated intraoperatively. Bacterial invasion of the epidural space from parameningeal suppurative foci is clearly more likely after dural trauma.<sup>25,26</sup> The presence of *Enterobacter* species in both cases is relatively unusual.<sup>12,27</sup> *Staphylococcus aureus* is involved in 50% to 93% of cases.<sup>1,12,27–29</sup>

## CONCLUSION

It is difficult to assess the relative morbidity of these patients after traumatic injury compared with those individuals reported in other series based on elective cases. Both patients reported here sustained traumatic injuries involving extensive local soft tissue and bony destruction. Although both have recovered from their respective infectious processes, this did not occur without the comorbidities involved with serial débridement, loss of paraspinal musculature, and local and allogenic bone graft inserted at the index procedure.

Acute bacterial meningitis after traumatic dural laceration and complete spinal cord transection as seen here in the setting of traumatic spinal fracture–dislocations may become more prevalent as survivorship increases, especially after high-speed motor vehicle accidents. It is important for the treating physician to recognize this complication quickly so that the appropriate treatment may be instituted.

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