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Delayed Onset Tension Pneumocephalus Following Lumbar Spine Surgery: A Report of Two Cases and Review of Literature

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ABSTRACT

Background: Pneumocephalus or presence of air in the intracranial cavity has been reported following head injuries and cranial surgery. However, pneumocephalus occurring after lumbar spine surgery is uncommon. In this report, we discuss the presentation and management of two patients who developed a delayed onset tension pneumocephalus following lumbar spinal surgery. **Case Presentation:** The first patient, a 54-year-old gentleman, who underwent a lumbar decompression and fusion surgery for infective spondylitis presented with tension pneumocephalus one month after the surgery. An incidental durotomy occurred during surgery. The second patient, a 74-year-old gentleman, who underwent a lumbar decompression and fusion for a post-traumatic two level lumbar fracture, was diagnosed to have a tension pneumocephalus after 10 days. The fracture fragments had caused a dural tear which was noticed during surgery. Both patients presented with headache, drowsiness and restlessness and pneumocephalus was diagnosed on brain imaging. Both of these patients were managed non-surgically with complete bedrest, supplemental oxygen, hyperhydration, analgesics, antiemetics and antibiotics and went on to make a complete recovery. **Discussion:** Pneumocephalus is a rare but potentially serious complication of spinal surgery. Fortunately, it resolves with complete bedrest, supplemental oxygen, hyperhydration, analgesics and antiemetics in a majority of patients. A high degree of suspicion is needed to diagnose this condition and provide prompt treatment.

Keywords: Cerebrospinal fluid leak; dural tear; pneumocephalus; spine surgery.

INTRODUCTION

Pneumocephalus, the presence of air in the cranial cavity,[1] was first reported by Lecat in 1866 although the term pneumocephalus was coined later by Wolff in 1914.[2,3] While pneumocephalus can occur after head and facial trauma, skull base surgeries, supratentorial craniotomy, paranasal sinus surgery and nasal surgeries, it is rare following lumbar spinal surgery where it can result following a dural tear.[4,5] Dural tears and subsequent cerebrospinal fluid (CSF) leaks are not uncommon following spinal surgery. While mostly harmless if managed appropriately, dural tears can occasionally lead to meningitis, epidural abscess, arachnoiditis, dural-cutaneous fistula, delay in wound healing and rarely pneumocephalus.[6]

In this report we discuss the presentation and management of two patients who developed a delayed onset pneumocephalus following a dural tear that occurred during lumbar spinal surgery. One patient who underwent a lumbar decompression and fusion surgery for infective spondylitis presented with tension pneumocephalus one month after the surgery while the second patient presented ten days after the index surgery. Both patients were managed non-surgically and went on to make a full recovery.

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CASE REPORT

Case Presentation 1:

A 54 years old gentleman, presented with pain in the lower back and front of the left thigh for six weeks, which was insidious in onset and gradually progressive. The pain was severe, prevented him from walking more than a few metres, aggravated at night and associated with evening rise of temperature, loss of weight and appetite. On examination, movements of the lumbar spine were severely restricted and painful with weakness in the left lower limb (Grade 4/5 power in the left hip flexors, hip adductors and left knee extensors). MRI scan of the spine revealed altered signal intensity at L1 with an epidural and left paravertebral abscess suggestive of L1 infective spondylitis [Figure 1]. In view of the severity of the pain, inability to walk and lower limb weakness, surgery in the form of T12, L1 and L2 laminectomy with T11- L3 instrumented postero-lateral fusion was planned [Figure 2]. An incidental durotomy occurred during surgery which was repaired with a 6-0 prolene suture. Tissue and purulent material obtained from the infected site were sent for Genexpert for Tuberculosis, aerobic

culture and histopathological examination. Ambulation was permitted on the day after surgery. Histopathological examination of tissue sample did not reveal any malignancy or granulomas, Genexpert for tuberculosis was negative and the aerobic culture showed no growth after 48 hours of incubation. Due to the inconclusive results, a four-drug regimen of anti-tuberculous therapy was initiated empirically, in view of the clinical features and as a part of institutional protocol for such cases.

Three weeks later, the patient presented with headache exaggerated on sitting and standing, vomiting, generalised weakness and disorientation. The surgical drain (without vacuum suction) was in place due to purulent material and CSF discharge. An MRI Brain revealed air in the left subdural space along the left fronto-parieto-temporal convexity causing mass effect and midline shift of the brain parenchyma to the right by 1.5 cm suggestive of tension pneumocephalus [Figure 3a, 3b]. Subtle basal hyperintensity and enhancement was also noted which was suggestive of meningitis. Oxygen via a face mask and intravenous antibiotics (Meropenem 2 gm thrice a day and Vancomycin 1 gm twice a day) was administered along with the anti-tuberculous therapy. A CT scan of the brain performed on the next day showed a reduction of the pneumocephalus and the midline shift [Figure 4a, 4b]. The wound drain was removed and the drain site was sutured. Over the next 48 hours, the headache and vomiting subsided with significant improvement in sensorium and the patient was discharged from the hospital. The IV antibiotics were continued for 10 days. The patient returned to his home town and continued the TB medications for 18 months. At the 18-month follow-up he was completely asymptomatic and had returned to his normal activities.

Case Presentation 2

A 74-year-old gentleman with a background history of hypertension, presented with acute mid-back pain following a seizure episode and consequent fall at home. He was diagnosed to have L1 and L2 burst fractures with conus compression. He subsequently underwent an L1-L2 decompression and T11-L4 posterior instrumented fusion surgery [Figure 5a, 5b]. Intra-operatively, an irreparable dural tear caused by the fracture fragments was noted. A water-tight closure of the surgical wound was performed over a drain which was maintained on gravity drainage without suction. Post-surgery, he was monitored in the ICU in view of post-op confusion and involuntary oro-facial movements. A metabolic work up did not show anything abnormal and an MRI Brain showed a small left medial temporal lobe infarct which was insufficient to explain the post-operative confusion. By the evening of Day 2, his sensorium improved and was transferred to the ward the next day. Following his transfer to the ward, he was allowed to walk with a thoracolumbar brace. At the time of discharge, he was walking comfortably and the wound was healing well. However, the drain was retained as there was a minor CSF leak.

On the 10th post-op day, he presented to the ER with vomiting, confusion and drowsiness of 24 hours duration and was noted to have a GCS 13 (E3V5M5). An MRI Brain revealed bilateral fronto-parietal pneumocephalus with mass effect on the lateral ventricles and mild midline shift (Figure 6a, 6b). High flow oxygen was administered along with strict bed rest and intravenous Meropenem (2 gms i.v. thrice a day). The drain was removed and drain site sutured and tight dressing was applied. The laboratory workup did not reveal any metabolic or infective cause. Over the next 36 hours, his sensorium improved and he was mobilized with a brace. The wound gradually healed and he returned to his normal activities. A CT scan of the brain performed 3 months later showed that the pneumocephalus observed on the previous scans had subsided [Figure 7]. However, eighteen months later he died of un-related causes.

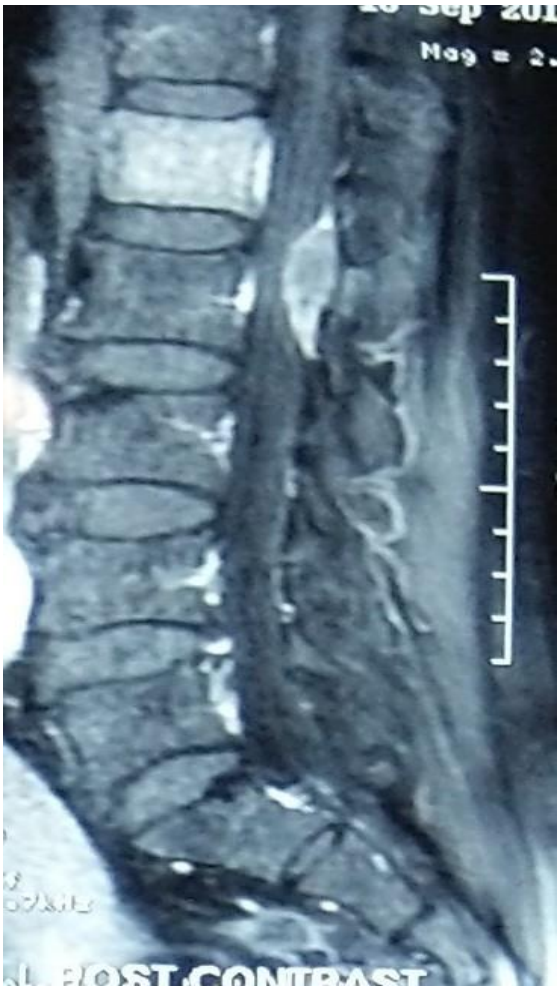

Figure 1: MRI Spine showed altered signal intensity at L1 with an epidural abscess and was suggestive of L1 infective spondylitis.

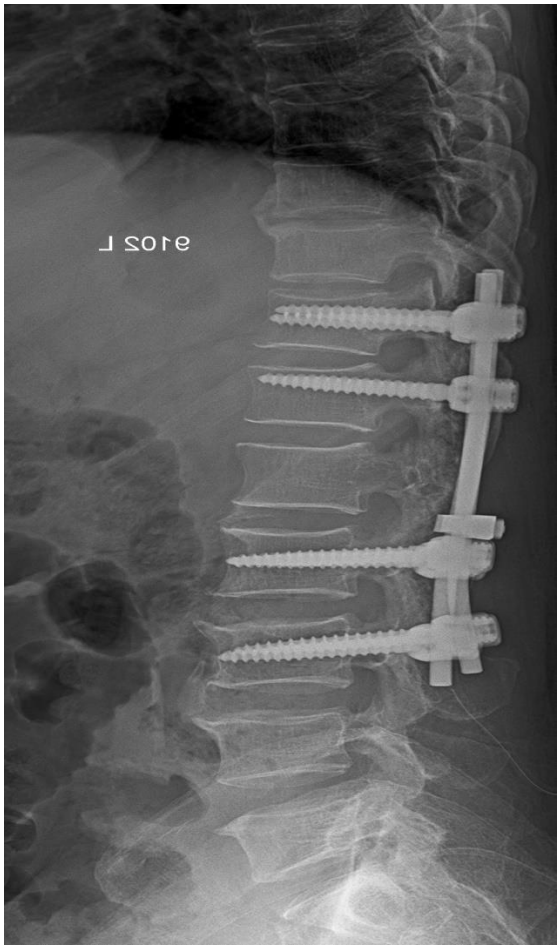

Figure 2: Post-op radiograph showing T11- L3 instrumented postero-lateral fusion.

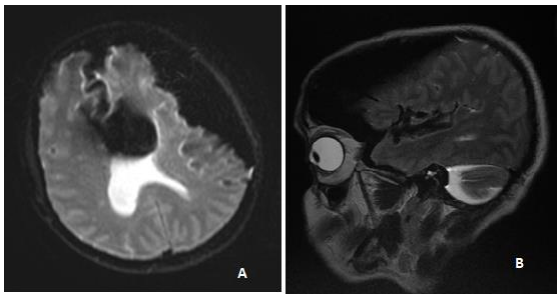

Figure 3a&b: MRI Brain revealed air in the left subdural space along left fronto-parieto-temporal convexity with mass effect and midline shift suggestive of tension pneumocephalus.

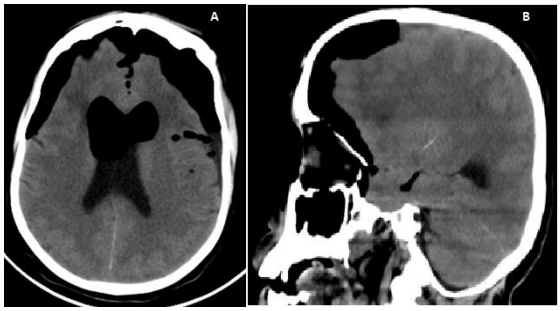

Figure 4a&b: CT Brain performed 24 hours after admission showing reduction in the volume of pneuocephalus

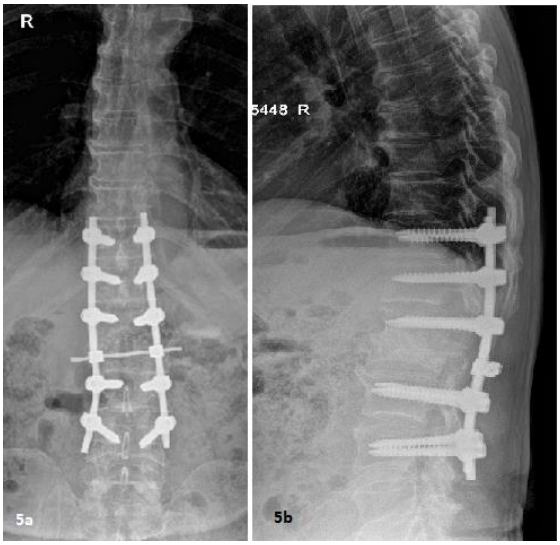

Figure 5a&b: Post-op radiographs showing T11-L4 posterior instrumented fusion.

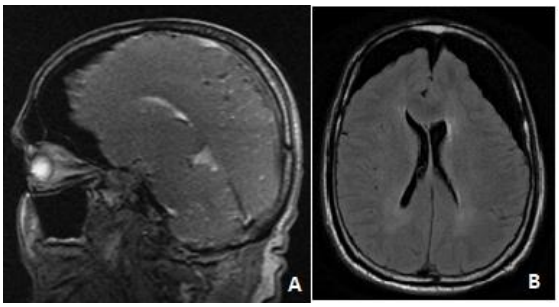

Figure 6a&b: MRI Brain showing bilateral fronto-parietal pneumocephalus with mass effect on the lateral ventricles and midline shift.

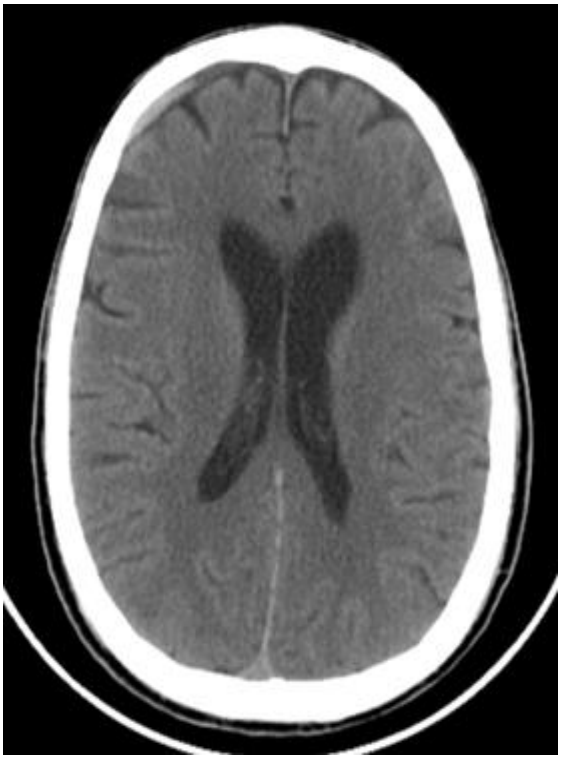

Figure 7: CT Brain performed 3 months later showed resolution of pneumocephalus.

DISCUSSION

The term "pneumocephalus" or "intracerebral aerocele" denotes the presence of air in the cranial cavity and can be localized in the epidural, subdural, subarachnoid, or intraventricular spaces.[7-9] Tension pneumocephalus refers to intra-cranial air causing mass effect and abnormal neurological signs and is classified as acute (less than 72 hours) or delayed (72 hours or more). Fractures of the skull-base, nasal sinuses and penetrating head injuries with dural breach/ laceration are the most common cause of pneumocephalus followed by infections (meningitis caused by gas forming organisms and chronic otitis media), tumours eroding the skull base and post-surgery (trans-cranial and trans-sphenoidal surgeries and ventriculostomies).[1,10]

Pneumocephalus has also been reported following lumbar punctures and lumbar epidural injections and rarely after lumbar spinal surgeries typically as a result of a dural tear.[1,5,11-13] Ayberk reported an interesting case of post-lumbar surgery pneumocephalus in the absence of an obvious dural tear which was hypothesised to result from raised intra-abdominal pressure in prone position leading to opening up of an occult defect around the cribriform plate.[2] Karavelioglu,[14] on the other hand, reported a case of pneumocephalus following excision of a calcified sequestered lumbar disc fragment without a noticeable dural tear and was eventually attributed to a unrecognised dural tear.

Numerous hypotheses have been proposed to explain the pathogenesis of pneumocephalus. According to the "inverted bottle mechanism" hypothesis, the negative intracranial pressure caused by continuous leakage of CSF is balanced and substituted by air. The "ball valve mechanism" theory proposes that air enters the intracranial cavity through a defect whenever the intracranial pressure is exceeded by the extracranial pressure (e.g., from the paranasal sinuses during sneezing, coughing, swallowing). Thirdly, nitrous oxide administered during anesthesia can diffuse from blood into the closed cranial cavity significantly faster rate than the nitrogen/air would exit from the closed space into the blood resulting in increased volume of gas in the closed space. Gas-forming bacteria can also lead to the development of pneumocephalus.[8,10,15,16] A vacuum suction device could predispose to the development of pneumocephalus after a spinal dural tear. In our cases, we believe that the dural tear and prolonged wound drain was responsible for the pneumocephalus.[3,5,14]

A strong index of suspicion needs to be maintained to diagnose pneumocephalus as the initial symptoms are often nonspecific and include headache, lethargy, drowsiness, nausea, vomiting and restlessness.[7] Pneumocephalus can be diagnosed using radiographs but CT scan is the imaging modality of choice in view of its ability to detect even small amounts (less than 0.5 cc) of air.[10] The "peaking sign," on the CT scan results from a bilateral compression without separation of the frontal lobes and is seen in the initial stages. Further accumulation of air separates and compresses both frontal lobes creating a profile similar to Mount Fuji's silhouette and is characteristic of tension pneumocephalus ("Mount Fuji sign").[2] The presence of multiple small air bubbles scattered through several cisterns and the arachnoid space is referred to as the "air bubble sign" and may indicate the development of tension pneumocephalus. On MRI scans, air appears completely black on all sequences and may be mistaken for blood product or flow voids.[3] Both our patients presented with headache, drowsiness, restlessness and nausea, and our clinical diagnosis was meningitis. The diagnosis of pneumocephalus was made only after the imaging studies.

Although the risk of post-spinal-dural-tear tension pneumocephalus can be reduced by performing a watertight dural closure supplemented by dural sealants and glues,[3,17] it cannot be completely eliminated.[4] Maintaining a modified Trendelenberg position and covering the dura with saline have also been described.[18,19] Fortunately, this condition usually responds to non-surgical treatment including supplemental oxygen, bedrest, analgesics, hyperhydration, antiemetics and symptoms subside in 1–3 weeks.[20,21] Administration of 100% oxygen via a non-breather mask leads to a faster resolution of pneumocephalus than breathing room air.[22] In both our cases, management with complete bedrest, supplemental oxygen, IV. fluids, analgesics, antibiotics and anti-emetics led to complete resolution of pneumocephalus.

CONCLUSION

Tension pneumocephalus is an uncommon but serious complication of inadvertent spinal dural tears. Postoperative drain placement has been hypothetised to be one of the primary causative factors for the same. A high degree of suspicion is needed to diagnose this condition. A majority of pneumocephalus settle down with non-surgical management, as has been shown in our case series of two patients where it was resolved by drain removal and supportive treatment.

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