Delayed neurological deterioration due to progressive pneumocephalus

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Abstract: Pneumocephalus can develop immediately following head trauma or clinical presentation may be delayed for days. We report a case of 35 year male whose initial CT scan brain plain small specks of pneumocephalus in left para-sellar region. However the next day he was complaining of severe headache and had multiple episodes of vomiting. Repeat CT scan showed increase in the size of pneumocephalus including appearance of intraventricular air with mild cerebral edema. The patient recovered well with conservative management. The present case is a gentle reminder that in a subgroup of head injury patients, intracranial air can produce significant mass effect leading to tension pneumocephalus which can can behave like other intracranial mass lesions and causes worsening of the neurological status of these patients.

Key words: Pneumocephalus, traumatic pneumocephalus, traumatic brain injury, basal skull fracture.

Introduction

Pneumocephalus or intracranial aerocele is collection of air in the intracranial cavity and is an uncommon complication of head injury. 1 Pneumocephalus can develop immediately following head trauma or may be delayed for days before patients become clinically symptomatic. (2) In majority of the patients the size of pneumocephalus is small, behave benign and can be managed conservatively. (3) However in some cases there can be a significant amount of air collection which can lead to tension pneumocephalus behaving like an intracranial space occupying lesion and causing increase in intracranial pressure with subsequent neurological deterioration. (1, 3, 4)

Case report

A 35 year male presented with the history of fall from motor cycle. He had transient loss
of consciousness for 15 minutes and multiple episodes of vomiting. There was history of nasal bleeding. There was no history of seizures. His general and systemic examination was unremarkable. At the time of examination in emergency room he was opening eyes to all, obeying commands and well oriented. Motor and sensory examination was normal. There was evidence of nasal bleed. The patient underwent CT scan brain plain and it showed fracture of left temporal bone with small specks of pneumocephalus in left para-sellar region (Figure 1). The patient was admitted for observation. Next day morning he was complaining of severe headache and had multiple episodes of vomiting. The CT scan was repeated and it showed increase in the size of pneumocephalus including appearance of intraventricular air with mild cerebral edema (Figures 2 and 3). There was no watery discharge through nose and there was no fever and meningeal signs. The patient was started on 100% oxygen and anticonvulsants. With conservative treatment he recovered completely and is doing well at follow up.

**Discussion**

Pneumocephalus can be acute (<72 hours) or delayed (>72 hours) and occur in 3.9-9.7% of the cases of head injury. (4-6) Two theories have been postulated to explain the underlying mechanism by which pneumocephalus develop after injury i.e. ball valve mechanism by Dandy (a uni-directional air movement from the outside into the cranial cavity which then gets trapped) (7) and the soda bottle theory (drainage of CSF leading to a negative intracranial pressure gradient which is relieved by the influx of air). (8) The air may be located in the extradural, subdural, subarachnoid, intraventricular, and intracerebral spaces (intracerebral and intraventricular pneumocephalus suggest breach in the dural and arachnoid layers). (2) The amount of pneumocephalus is usually independent of the
size of the defect and smaller defects are sealed easily by blood clots or granulation. (9) However a large collection of intracranial air can produce the mass-effect intracranial hypertension (tension pneumocephalus) and neurological deterioration. (3, 6) In presence of head injury, the diagnosis of tension pneumocephalus can be difficult as the symptoms and mechanism of injury may mimic other intracranial lesion (i.e. hemorrhage). (2) Usual clinical features of tension pneumocephalus include headache, nausea and vomiting, seizures, dizziness, altered sensorium (2, 3, 10-12) and CSF rhinorrhea or otorrhoea. (2) Plain radiographs can show the presence of pneumocephalus. CT scan of the brain is a golden standard to diagnose pneumocephalus. (2, 3, 13, 14) CT scan will allow determining the precise location of the air collection, amount of mass effect on the brain and its relationship to the basal skull fracture site or air sinuses. (2, 13, 15) Depending on the site and extent of air distribution various signs have been described to identify the tension pneumocephalus (“Mount Fuji sign”- significant quantities of air over the frontal convexities and “air bubble sign” producing characteristic multiple small air bubbles scattered through several cisterns). (2, 13, 16, 17) Majority of the cases of traumatic pneumocephalus respond well to conservative management (bed rest, 100% supplemental O2, head end elevation, avoidance of positive pressure, and pain control). (2, 3, 9, 11, 18, 19) Surgical intervention can be needed in patients with recurrent pneumocephalus & in patients with significant tension pneumocephalus with signs of increasing intracranial pressure. (2, 3, 6, 9, 20) Surgery is aimed to remove mass effect as in cases of tension pneumocephalus and for adequate skull base defects closure (particularly large defects). (2, 6)

**Conclusion**

In summary - in a subgroup of head injury patients, intracranial air can produce significant mass effect leading to tension pneumocephalus. This can behave like other intracranial mass lesions and causes worsening of the neurological status of these patients. It is important to have a high index of suspicion to make the correct diagnosis as appropriate intervention will prevent morbidity and mortality in these patients.

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