Post-traumatic pneumocephalus: a case report

Case Report

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ABSTRACT

Pneumocephalus is an uncommon neurosurgical pathology that usually follows cranio-facial trauma usually resulting from cranial surgery and trauma to the adjourning paranasal sinuses. Other causes are tumours of the skull base and also, rarely it could arise spontaneously.

A 39-year old male who sustained head injury from road traffic accident was managed in our center with clinical and radiological evidence of pneumocephalus. The patient was managed operatively with a very good outcome.

Keywords: Cerebrospinal fluid leak, cranio-facial, imaging

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INTRODUCTION

refers intracranial Pneumocephalus to collection of air. Synonyms include aerocoele, pneumatocoele, and pneumocranium. This is generally an abnormal or accidental finding, as the normal intracranial cavity is devoid of air. It was first described by Chiari in an autopsy report in 1884, and radiologically by Luckett in 1913.^{1,2} Pneumocephalus may be classified based on anatomical location into subdural, subarachnoid, epidural, intraventricular and intra-parenchymal. Whilst pneumocephalus presents as a rare but often benign neurological injury, tension

pneumocephalus life-threatening is а neurosurgical emergency where the intracranial air, trapped under tension, acts as a space occupying lesion causing a shift of the midline structures, and brain compression resulting in neurological deterioration. Trauma, including that following surgery, remains the leading cause of pneumocephalus.

The incidence of pneumocephalus is low as shown by several studies. Gill and van As reported an incidence rate of less than 1% in the head trauma population.³ This rises to 8% These figures may be deceptive however, for the following reasons: the diagnosis of pneumocephalus requires a high index of suspicion; the presence of a fracture and, together with this, signs of a brain contusion is clinically often regarded as sufficient to explain the condition of the patient; small amounts of air can be disregarded in the acute radiological examination; and pneumocephalus often develops after a certain period of latency, and so is missed altogether especially when it is asymptomatic.

Following diagnosis however, a patient with pneumocephalus would require some form of treatment which may include surgery. The treatment depends on the neurological state of the patient and should be individualized, accordingly.⁵

Tension pneumocephalus requires emergency decompression to alleviate pressure on the brain parenchyma, but with simple traumatic pneumocephalus, treatment depends on whether or not CSF leak is present. Small volumes of air with no CSF leak will be resorbed with time. If there is CSF leak and the patient is stable, an initial conservative approach may be adopted. This consists of bed rest with 15^o head elevation, oxygen therapy where necessary, appropriate antibiotics and anticonvulsants as required.

Operative treatment is indicated when conservative treatment fails or when there is tension pneumocephalus. The surgical options include placement of twist drill or burr holes followed by needle aspiration of air. Craniotomy is required when the fistula is at the base of the brain. The fistula is repaired either through an intradural or extradural approach using autologous, pericranium or fascia lata. In the case being reported, fat harvested from the anterior abdominal wall was tucked into the frontal sinus after exenterating the mucosa. For pneumocephalus arising from spinal dural defects due to epidural or spinal anaesthesia, blood patch has been used to seal the defect. Pneumocephalus arising from gas forming infections requires appropriate antibiotic therapy and drainage of associated abscess.

Prognosis depends on factors like type and mechanism of injury, associated complications and time of presentation. Shehu, *et al*, highlighted the challenges of management of pneumocephalus ranging from delayed presentation and diagnosis, socio-economic issues to low manpower.⁵ Follow up of the patient is important to detect any residual neurological deficits, or rarely, a recurrence of pneumocephalus.

CASE SUMMARY

A 39-year old man was referred with a 6week history of rhinorrhoea from the left nostril, with a 4-week history of headache and abnormal behaviour. He was apparently well until two months prior to presentation when he fell off his motorcycle and hit his head on the ground not using a helmet. He had a history of loss of consciousness and an episode of generalized, tonic-clonic convulsions. Two weeks later, he started draining clear fluid from his left nostril, with headache and behavioural changes of speech, blank irrational staring, aggressiveness and indiscriminate micturition. He also walked with an abnormal broad-base gait, but there was no history of fever or neck stiffness. Other systems were normal.

Clinical examination showed an unkempt middle aged man, not in obvious distress, afebrile and not pale. The Glasgow Consciousness Score (GCS) was 14/15 (EOR=4, BVR=4, BMR=6), the pupils were equal and reactive to light bilaterally, and cranial nerves were intact. Muscle power, tone and reflexes were normal, but he walked with broad-base gait. The other systems were normal.

Skull x-rays and brain computed tomographic scan showed a depressed skull fracture of both outer and inner tables of the frontal bone, a huge bi-frontal aerocoele that was increasing progressively in size on repeat xrays with enlargement of both lateral ventricles, see figures 1, 2 and 3.

Full blood count, blood urea, serum electrolytes and creatinine were normal.

Figure 1. Skull radiograph antero-posterior view showing pneumocephalus



Figure 2. Skull radiograph lateral view showing pneumocephalus aerocele and ventriculogram



A diagnosis of frontal pneumocephalus with depressed skull fracture was made and the patient was prepared for surgery. A bi-frontal craniotomy with dural repair under general endotracheal anaesthesia, were done.

Figure 3. Cranial computerized tomogram showing air in the lateral ventricles and left frontal aerocele



Phenobarbitone was administered intravenously and orally at a dose of 3mg/kg in three divided doses. Perioperative intravenous ceftriaxone was administered for at the induction of anaesthesia.

Intra-operative finds were those of a depressed frontal bone fracture affecting the outer and inner tables, dural laceration, frontal aerocoele with markedly thinned out cerebral cortex. The frontal paranasal sinus was denuded of mucosa and filled up with fat harvested from the anterior abdominal wall. Durotomy was done to decompress the aerocele and to evacuate the air in the ventricle using a brain cannula. Duroplasty was subsequently done with pericranium, and the wound was closed in layers over a tube drain.

The tube drain was removed on the second post-operative day. Post-operative recovery was uneventful with complete resolution of symptoms including cerebrospinal fluid rhinorrhoea, headache, behavioural changes and abnormal gait Repeat skull x-rays did not reveal any aerocoeles, see figure 4. The patient was discharged home on the fifth post-operative day on anti-convulsants and head injury advice. Follow-up visits revealed complete recovery and full return to normal activities with no recurrence of symptoms.

Figure 4. Skull radiographlateral view showing complete resolution of the pneumocephalus (note the radio-lucent burr hole points)



DISCUSSION

Pneumocephalus is even more likely with the presence of a fracture, especially those involving the paranasal sinuses or temporal air cells. The index case reported above followed head trauma with frontal bone fracture, involving the frontal air sinus, from a motorcycle accident. Shehu, et al, in a study of 20 cases of pneumocephalus, found that trauma accounted for 90%.5 Another form of trauma is the *iatrogenic* type, following brain surgery (craniotomy, burr hole, etc.). All patients who had craniotomy or craniectomy will have detectable air in the cranium within the first week of surgery; thereafter, the incidence decreases.⁶ Bhawani, et al, reported 5 cases of tension pneumocephalus following evacuation of chronic subdural haematoma.7 Ventriculo-peritoneal shunting has also been implicated causation in the pneumocephalus. Here, significant lowering of intracranial pressure following shunting causes unplugging of defects in the skull and dura resulting in a fistula.8

Cardiothoracic surgeries have also been reported to cause pneumocephalus.^{9,10} Non-traumatic causes of pneumocephalus include

infections such as chronic otitis media and sinusitis. Patients have been known to have spontaneous pneumocephalus from these. Microorganisms implicated in these infections include escherichia coli, peptostreptococcus species, streptococcus pyogenes, fusobacterium species, and gas producing organisms like klebsiella and bacteroides species.

Other causes of pneumocephalus include oxygen therapy following traumatic head injury. Nicholson and Dhindsa reported a case of traumatic tension pneumocephalus after blunt head trauma and positive pressure ventilation.¹¹ Pneumocephalus may also complicate spinal or epidural anaesthesia, or lumbar spinal drainage.¹²

The pathophysiology of pneumocephalus has been explained by two mechanisms: the "ballvalve mechanism" and, the "Coke-bottle mechanism".12 The ball-valve mechanism postulates that a cranial defect adjacent to an air containing space such as a sinus allows air into the cranial vault. This fistula allows ambient air at a pressure above intracranial pressure (ICP) to force itself into the intracranial space. For example: coughing, sneezing, nose blowing or straining at micturition. This process continues until ICP overcomes the ambient pressure and the brain and dura mater are forced over the fistula. The process may repeat itself multiple times until the higher-pressure ambient air no longer overcomes ICP. This process explains the mechanism post traumatic of pneumocephalus, as seen in the case we have reported.

The "Coke-bottle mechanism" on the other hand, postulates that a continuous cerebrospinal fluid (CSF) leak from the enclosed cranial vault is present. The loss of CSF creates a void space and relative negative pressure allowing air to bubble in and fill the void. This fluid-gas exchange may continue until no additional loss of CSF is possible. Pneumocephalus may form either as a large isolated pocket or as a series of smaller bubbles.

The clinical diagnosis of pneumocephalus requires a high index of suspicion. The symptoms and signs may occur immediately after trauma or may be delayed for several days or weeks. The clinical features may mimic those of any intracranial space occupying lesion. The most common symptoms reported are headache and CSF rhinorrhoea.5,12 Other features include CSF otorrhoea, seizures, dizziness, deteriorating level of consciousness, anosmia, focal neurological signs, and frontal lobe features. The underlying pathology of brain injury and presence of complications like meningitis may further confuse the picture. The mainstay of diagnosis is radiological evaluation.

Skull radiographs and brain computed tomography (CT) scans confirm the diagnosis. Brain CT scan is also better for assessment of other associated pathological conditions. Tension pneumocephalus can be recognized by the Mount Fuji sign on brain CT scan.¹³ The due bilateral sign is to subdural hypoattenuating collections causing compression and separation of the frontal lobes. The volume of air does not seem to be important in differentiating tension pneumocephalus from non-tension pneumocephalus.⁵ No definite radiological diagnostic sign, or a particular volume of air sufficient to cause tension pneumocephalus can be spelt out, since the vulnerability of the brain varies from patient to patient depending on the status of intracranial contents and pressure. Other specialized investigations may be necessary when the site of fistula is not obvious.

CONCLUSION

Pneumocephalus is a rare but challenging problem which confronts not only the neurosurgeon, but also other specialists like the trauma surgeon, neuroradiologist, anaesthetist, and ultimately the entire healthcare system. The incidence in this environment may be on the increase as accident rates are still unacceptably high. Prompt diagnosis and treatment will often result in full recovery with very low morbidity and mortality from this condition.

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