Traumatic pneumorrhachis with tension pneumocephalus

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ABSTRACT

Air contained within the spinal canal is termed as pneumorrhachis. There are only few previously reported cases in the literature about pneumorrhachis. We herein report a case of pneumorrhachis associated with tension pneumocephalus in a patient with severe head injury.

Key words: Air in spinal canal, pneumorrhachis, tension pneumocephalus, trauma

INTRODUCTION

Pneumorrhachis (PR) is an imaging finding caused by various traumatic and iatrogenic etiologies. Although PR has been previously described, mainly in the radiological literature, spine and trauma specialists are less acquainted with this unusual pathological entity. Air within the spinal canal may arise due to myriad traumatic, non-traumatic, and iatrogenic causes. The intraspinal air can be epidural or subarachnoid. PR due to trauma is relatively rarely reported. We report here a young boy with skull base fracture and tension pneumocephalus, following road traffic accident with PR in the cervical spine.

CASE REPORT

A 20-year-old boy with a history of automobile accident and polytrauma came to the emergency 4 hours after the injury. He was brought to the casualty in shock with deformity of both lower limbs, suggestive of severe RTA. His initial Glasgow coma scale (GCS) was 14 (E4V4M6) on arrival in casualty. He also had profuse blood-mixed CSF otorrhoea from right ear.

As soon as patient hemodynamics became stable, he was shifted for non-contrast computed tomography (NCCT) scan of head and cervical spine and relevant X-rays of chest and extremities. NCCT head [Figure 1] revealed pneumocephalus with fracture of sphenoid sinus, right petrous temporal bone, and occipital bone extending up to foramen magnum. Pneumocephalus was present in both the supratentorial and infratentorial compartment of head with air around brainstem. Internal pneumocephalus with diffuse air distribution in the basal, prepontine, and perimesencephalic cisterns and fourth ventricle was seen. NCCT cervical spine [Figure 2] and screening whole spine showed air lucency in spinal canal up to cervico–thoracic junction, indicating subarachnoid PR with no canal compromise or misalignment of spine.

Within 2 hours, the patient became quadriparetic with power both upper limbs 3/5 and lower limbs 2/5, and, within the next 1 hour, his GCS started deteriorating and became E2V2M4. He was immediately intubated in casualty and put on ventilator support. The pneumocephalus was immediately drained through right frontal twist drill, and a gush of air came out. The patient was immediately shifted to neurosurgery intensive care unit (ICU). In the next 3 hours, the patient started obeying commands. His power in all four limbs also improved in nearly 12 hours time to previous 4+/5. He was successfully weaned off from the ventilator and extubated after 24 hours. The CSF otorrhoea also gradually reduced and stopped after 3 days. Serial NCCT scans revealed decrease in pneumocephalus over time.

On day 5, open reduction and internal fixation was done for his multiple long bone fractures. The postoperative course was uneventful, and he was discharged on day 10.

DISCUSSION

Free air in the spinal canal is an uncommon phenomenon, which was primarily reported by Gordon et al.,[1] in
Dawar, et al.: Traumatic pneumorrhachis with tension pneumocephalus

1977, and has been described under various terms such as intraspinal pneumocele or spinal epidural and subarachnoid pneumatoses, spinal and epidural emphysema, aerorachia, pneumosaccus, air myelogram, and pneumomyelogram or pneumomyelography. PR per se usually represents an asymptomatic, probably underdiagnosed epiphenomenon of coincident underlying injuries and diseases.

PR can be classified into internal (intraspinal air within the subdural or subarachnoid space) and external (intraspinal, epidural air) PR. External PR by itself is usually innocuous, whereas internal traumatic PR is frequently associated with major trauma and is believed to be a marker of severe injury.[2] Many causes of PR have been reported in the literature, and these can be broadly classified into iatrogenic, non-traumatic, and traumatic. Iatrogenic causes are the most common, and epidural emphysema is a well-known occurrence after administration of epidural analgesia.[3] Subarachnoid space air has also been reported after lumbar puncture[4] and as a result of iatrogenic subarachnoid pleural fistula after thoracic spine surgery[4] or thoracotomy.[5] Non-traumatic causes of epidural air that have been described in the literature include spontaneous pneumomediastinum or pneumothorax,[6] epidural abscess,[7] and vertebral metastases from esophageal carcinoma.[8] The occurrence of PR secondary to trauma is extremely rare.

The diagnosis of PR is difficult or even impossible to make clinically. It is usually diagnosed incidentally by plain radiography, CT, or magnetic resonance imaging. The plain radiograph is the least sensitive modality and can only detect PR when there are large volumes of air in the spinal canal.

The occurrence of air in the epidural space and subarachnoid space should be distinguished as they are associated with different pathophysiological mechanisms and, thus, causes. Nonetheless, it may be difficult to differentiate between the two, even on CT scan. Traumatic subarachnoid PR is almost always secondary to major trauma and is a marker of severe injury. Motor vehicle crashes and falls from height are the most commonly reported causes. The presence of epidural air has a different clinical implication from air in the subarachnoid space. Epidural emphysema by itself is usually innocuous and causes no clinical symptoms, whereas subarachnoid air is commonly associated with pneumocephalus and may give rise to raised intracranial pressure, resulting in headache and focal neurological signs. The presence of a dural tear may also serve as a portal of entry of bacteria into the subarachnoid space, which may result in meningitis.[9]

In our case, presenting with CSF otorrhea, intraspinal air was probably caused by the sphenoid sinus and petrosal bone fractures, thus allowing direct communication of pneumatized air containing cavities with the intracranial space. The penetrated air was then forced caudally due to the elevated intracranial pressure, resulting from severe brain injury with diminution of capacity of the intracranial space. Horizontal and head-down position of the patient finally allowed the entrapped air to pass through the foramen magnum into the spinal canal.

Because of the rarity and the different pathogenesis and etiologies, no empiric guidelines for the treatment of PR and standards of care exist. Most often, PR is an innocuous incidental finding. However, entrapped intraspinal air under pressure entering the cranio-spinal compartment usually in combination with a one-way air valve mechanism might cause tension PR and pneumocephalus with nervous tissue compression requiring intervention.

REFERENCES


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