

Massive Pneumorrhachis, Pneumocephalus and Pneumoopticus Following Thoracic Trauma and Avulsion of the Brachial Plexus: Case Report and Review of the Literature

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Abstract: A 41-year-old man with injury of right half of the thorax, fractures of the left crural bones and paralysis of the right upper limb was admitted to our hospital. A CT examination at admission revealed bilateral pulmonary contusion and bilateral fluid- and pneumothorax. In addition pneumomediastinum, pneumopericardium, subcutaneous emphysema and pneumorrhachis at the cervicothoracic transition was demonstrated. Abnormal findings in the skull and brain were not revealed. The fifth day after admission repeated CT examination demonstrated extensive frontal pneumocephalus on the right, presence of air in several cisterns and in the right optic nerve sheaths (pneumoopticus). Right frontal craniotomy was performed, dura mater was incised and air was evacuated. Rapid regression of pneumocephalus was evident postoperatively. The tenth day after admission MRI of the cervical spine and brachial plexus was performed. At the level of the C7 and C8, nerve roots pneumomenigocele and a nerve retracting ball indicating the presence of a nerve root injury

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were discernible. This case demonstrated that severe thoracic blunt trauma leads to acute increase of intrathoracic pressure with concomitant fluid- and pneumothorax, pneumomediastinum and pneumopericard. From the mediastinum air propagated subcutaneously. Disrupted cervical dural sheaths resulted in leakage of cerebrospinal fluid and entry of air from mediastinum to subdural and subarachnoid spinal and cranial space and to the subarachnoid space of the optic nerve.

Introduction

Various traumatic and non-traumatic conditions may produce presence of air within the spinal canal (pneumorrhachis) and/or in cranial cavity (pneumocephalus). Air within the spinal canal may be located in the epidural or subarachnoid spaces. The occurrence of air within the epidural space is more frequent. This condition is also known as epidural emphysema or epidural pneumorrhachis. Subarachnoid pneumorrhachis is presence of air within subarachnoidal space and this condition is frequently associated with major trauma. Traumatic subarachnoid air is frequently associated with more serious injury frequently producing pneumocephalus. Pneumocephalus may be not only the consequence of subarachnoid pneumorrhachis, but also the cause (Adams et al., 2003; Goh and Yeo, 2005; Oertel et al., 2006; Coscun et al., 2009). The aetiology of pneumorrhachis can be classified as iatrogenic (surgical and anaesthesiological interventions) traumatic and nontraumatic. Traumatic pneumorrhachis has frequently been demonstrated secondary to traumatic pneumothorax or pneumomediastinum (Hwang and Kim, 2000). Less frequent causes were summarized by Goh and Yeo (2005). In addition, thoracic and cervical stab wounds have been reported as a cause of pneumorrhachis (Tejirian et al., 2009). Pneumorrhachis has also been reported after cranial polytrauma (Oertel et al., 2006; Coscun et al., 2009).

Important factor contributing to the origin of pneumorrhachis are conditions that produce high intrathoracic pressure and barotrauma (Oertel et al., 2006; Zakyntios et al., 2008). Non-traumatic causes include spontaneous pneumothorax and pneumomediastinum. Spontaneous pneumothorax occurs frequently in previously healthy young adults and is caused by a rupture of pulmonary bullae and blebs. The air may consecutively propagate from the pleural cavity to the mediastinum and via neuroforamina to the spinal epidural space (Aribas et al., 2001; Goh and Yeo, 2005).

Nontraumatic (spontaneous) pneumorrhachis originating from vacuum disc degeneration was more rarely described (Kim, 2007).

Analogously, pneumocephalus can result from many different causes. Traumatic conditions are most frequently reported, especially trauma involving the paranasal sinuses, basilar skull fractures and communication of the ethmoid sinus with intracranial space. Pneumocephalus in the absence of cranial fractures, is not encountered as frequently but has been reported after lumbar puncture,

thoracotomy, thoracoscopy and after trans-sphenoidal surgery (Kozikowski and Cohen, 2004; Sudhakar et al., 2004; Tejirian et al., 2009).

Therefore, we would like to present a case of a cervical subarachnoid pneumorrhachis, pneumocephalus and pneumoopticus induced by a combination of pneumothorax, pneumomediastinum and cervical root avulsion. In addition, air was also distributed to other compartments of the body (pericardium, intermuscular fascia, subcutaneous emphysema).

Case report

A 41-year-old man with injuries to the right shoulder and right half of the thorax from an unblocked transported escalator was admitted to our emergency department immediately following injury.

On arrival, patient was conscious and well orientated to person, place and time. He complained of pain in the right shoulder and chest and right upper and lower extremities, as well as shortness of breath. His vital signs were not stable upon arrival and were reported as: blood pressure 83/50–120/70 mm Hg, pulse 90–85 beats per minute, respiratory rate 20 breaths per minute. Oxygen saturation was decreased. Physical examination revealed pain and excoriation of the right shoulder and increased sensitivity of the right hemithorax. Left leg exhibited deconfiguration between middle and lower third. Range of motion in right lower limb was reduced significantly in all segments.

The right upper extremity was found to be paralytic with hypaesthesia of the arm and complete anaesthesia of the forearm and hand. X-ray examination of the left leg at admission revealed comminuted fractures of left crural bones.

A CT examination was performed at admission and did not reveal any abnormal findings in the skull and brain. CT examination of the thorax revealed bilateral pulmonary contusions, bilateral fluidothorax (later haemothorax) and bilateral



Figure 1 – Axial CT scan depicts bilateral pneumothorax accompanied with bilateral contusion of lungs and with pneumomediastinum and pneumopericardium.

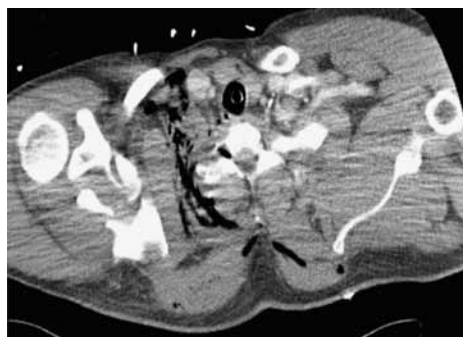


Figure 2 – Axial CT scan demonstrates pneumomediastinum and air in fascial intermuscular spaces of the neck and in right intervertebral foramen at level C7.

pneumothorax. Pneumomediastinum, pneumopericardium, subcutaneous and intermuscular emphysema in the cervicothoracic region and epidural, subdural and subarachnoidal pneumorrhachis at the cervicothoracic transition of the spine were also evident (Figures 1 and 2). Additional findings on thoracic CT were fractures of the 1st and 6th rib (right and left respectively), a comminuted fracture of the right scapula and fractures of the spinous processes of thoracic vertebrae Th7–Th12.

CT examination of the abdomen revealed a comminuted fracture of the vertebral body and arch of L1, with dorsal dislocation of the vertebral body fragment to the spinal canal. The spinal canal was narrowed to 9 mm and the vertebral body

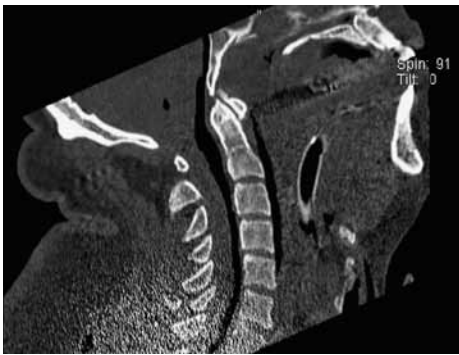


Figure 3 – A sagittal CT reconstruction of cervical spine showing extensive accumulation of air in the anterior subdural (subarachnoidal) space and in the retropharyngeal space.



Figure 4 – Non-contrast axial CT scan demonstrates severe pneumocephalus with significant compression of the right frontal lobe.

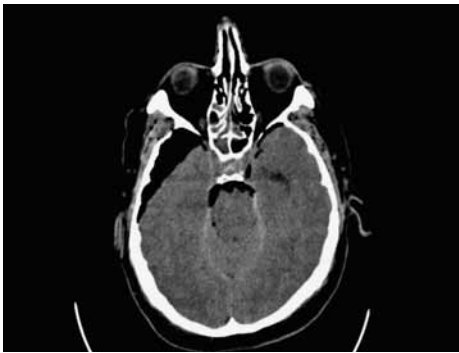


Figure 5 – Non-contrast axial CT scan demonstrating pneumocephalus causing compression of right temporal lobe and presence of air in pontine cistern.



Figure 6 – Axial CT scan demonstrates air in suprasellar cisternae and the unusual finding of air within the intraorbital portion of the right optic nerve sheaths (an arrow). In addition is evident bilateral pneumocephalus with compression of right temporal lobe.

fragment compressed the dural sac and the spinal cord. In addition fractures of the right transverse processes of vertebrae L1–L4 were demonstrated.

Neurologic examination confirmed paralysis of the right upper extremity, suspected to be due to a lesion of the right brachial plexus, and paresis of the right lower extremity.

Two hours after admission was patient underwent surgery. Decompression consisted of repositioning the L1 vertebral body and stabilization of Th12 and L1 using metallic plates. Next, metallic osteosynthesis of left crural bone was performed.

The second day after admission, chest drainage was introduced for pneumo- and fluidothorax.

High levels of myoglobin, hyperkalemia and worsening of renal parameters were repeatedly observed.

The fourth day after admission the patient exhibited abrupt anisocoria. The same day CT of the head and neck was repeated and revealed extensive frontal pneumocephalus on the right, and a lesser amount of pneumocephalus on the left.

Extensive compression of the right frontal lobe and significant displacement of the ventricular system was apparent. The suprasellar and quadrigeminal cisterns also contained air. CT of the orbits revealed the unusual finding of air within the intraorbital right optic nerve sheath. Air was also demonstrated within the anterior subdural (subarachnoid) space from C7 up to the pontine cistern intracranially (Figures 3–6).

A burr hole trephination and subdural drainage was performed on the right side on the fourth day after injury. The dura mater was cauterized and incised in a cross fashion. Immediately after, air bubbled through the incision. A subdural drain

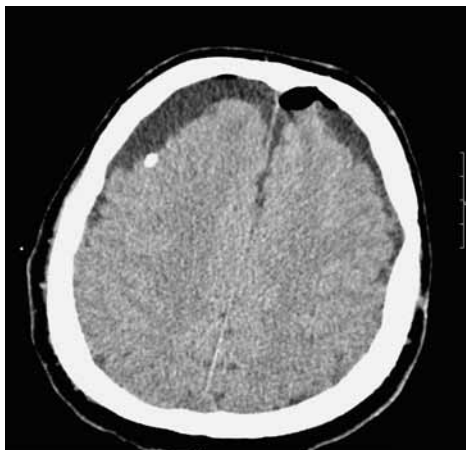


Figure 7 – Non contrast axial CT scan performed sixth day after injury shows drainage catheter placed in the subdural space.

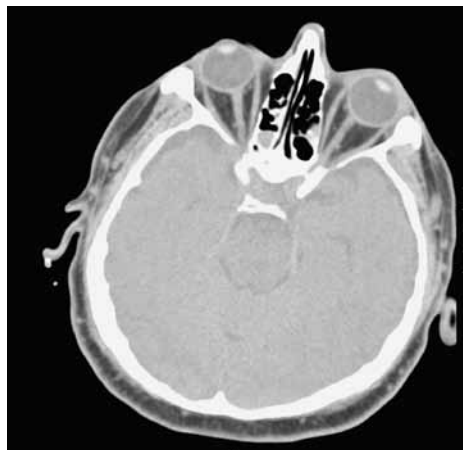


Figure 8 – Non contrast axial CT scan taken 9 days after injury showing no evidence of intracranial air.

was introduced via incision. Rapid regression of the pneumocephalus was evident postoperatively on the control CT examination performed sixth and ninth day after admission (Figures 7 and 8).

Eighth day after injury patient was disconnected from ventilator and the chest drainage tube was removed. Patient was fully conscious, spontaneously breathing, and hemodynamically stable.

The tenth day after admission MRI of the cervical spine and brachial plexus was performed (T2W-TSE; T2W-STIR sagittal, transverse, coronal). No traumatic changes of bones, intervertebral discs and ligaments of the cervical spine were revealed. Extensive subcutaneous and intermuscular emphysema of the neck was evident. MR imaging of the brachial plexus demonstrated paravertebral signal intensity changes on the right side. At the level of the C7 and C8 nerve roots accumulation of fluid (pseudomeningocele) was evident, containing an oval body suggestive of a nerve retracting ball. On some axial slices continuity between subarachnoidal space and the pseudomeningocele was discernible. This finding indicated the presence of a nerve root avulsion injury (Figures 9 and 10). Eleven days after the injury the patient was transferred to Department of Orthopaedics and Traumatology.

Discussion

The patient discussed herein had no head trauma that could have lead to the direct entry of air to the intracranial space. Severe blunt thoracic trauma leads to acute increase of intrathoracic pressure and resulted in pneumothorax and pneumomediastinum. Pneumorrhachis and pneumocephalus was most likely caused by the propagation of air from the mediastinum via the neuroforamina. An



Figure 9 – T2W-STIR coronal image shows on the right side paravertebrally an accumulation of fluid (pseudomeningocele) containing low signal oval body suggesting a nerve retracting ball of C7 nerve (an arrow).

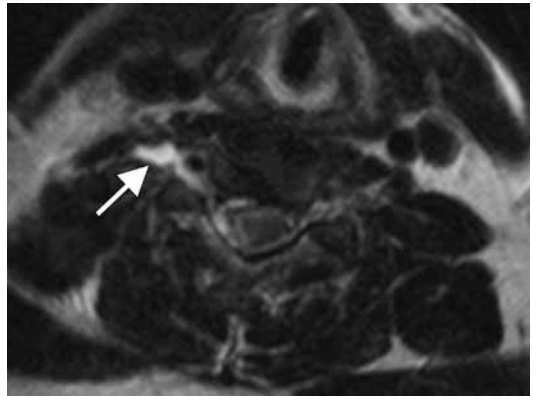


Figure 10 – T2W-TSE transversal image shows small collection of the fluid at the right side paravertebrally containing low signal oval body suspected of nerve retracting ball (an arrow).

important factor contributing to the development of subdural pneumorrhachis and pneumocephalon was avulsion of brachial plexus. In the present patient, brachial plexus root avulsion with disrupted dural sheaths at the level of C7 and C8 resulted in leakage of cerebrospinal fluid and entry of air from the mediastinum to epidural, subdural as well as subarachnoid spinal space. Consequently air migrated to the intracranial subdural and subarachnoid space. Increased pressure of air in the intracranial space resulted to its propagation to the subarachnoid space of the optic nerve.

Epidural pneumorrhachis, defined as the presence of air within the epidural space, is an uncommon clinical entity first described in 1977 that has been only rarely reported (Gordon and Hardman, 1997; Oertel et al., 2006). Epidural pneumorrhachis is relatively frequently found in combination with incidences of air distribution in other body cavities and compartments. These include pneumothorax, pneumomediastinum, pneumopericardium and subcutaneous emphysema (Zakynthinos et al., 2008; Chun and Moon, 2009; Song and Lee, 2009).

The aetiology of epidural pneumorrhachis has been classified as iatrogenic, spontaneous, and traumatic. Iatrogenic causes are the most common and follow the administration of epidural analgesia or to thoracic, abdominal and spinal surgery (Overdiek et al., 2001; Holton et al., 2002). Spontaneous causes have been described following nontraumatic pneumothorax, pneumomediastinum, or degenerative disc disease (Aribas et al., 2001; Chun and Moon, 2009). Traumatic epidural pneumorrhachis are secondary to traumatic pneumothorax or pneumomediastinum and could be associated with fractures of vertebrae and basilar skull fracture (Goh and Yeo, 2005; Oertel et al., 2006; Chun and Moon, 2009). Epidural pneumorrhachis and pneumomediastinum has only been reported four times in the English literature in association with diabetic ketoacidosis. The pneumorrhachis in diabetic ketoacidosis is related to the high intrathoracic pressures during prolonged emesis (Ripley et al., 2009). Epidural pneumorrhachis accompanied by pneumomediastinum, pneumopericardium and subcutaneous emphysema was described in young man presented with an attack of bronchial asthma. Similarly as in diabetic ketoacidosis penetration of air into several body compartments was caused by acute increase of intra-thoracic pressure. In bronchial asthma, probably, rupture of pulmonary alveolus could be an initial event (Manden and Siddiqui, 2009). An air in the epidural space was described also in gangrenous abdominal pathologies (Rehman et al., 2009).

Epidural pneumorrhachis usually does not migrate due to complicated arrangement of the vertebral epidural space where venous plexuses and epidural fat represent an obstacle, and is often asymptomatic and reabsorbs spontaneously (Goh and Yeo, 2005). Nevertheless, epidural pneumorrhachis has rarely been described as symptomatic and associated with neurological deficits (Chun and Moon, 2009; Song and Lee, 2009).

Subarachnoid (subdural) pneumorrhachis is a rare finding primarily associated with more serious injury. It is seen in cases of skull fracture, dural puncture (spine stab wounds), spinal fracture with injury to the dura, and in defects of dura and dural sheaths following brachial plexus root avulsion. Subarachnoid pneumorrhachis is frequently associated with pneumocephalus. The subarachnoid space in the brain and spinal cord is continuous and air thus may migrate from the spine into the cranial subarachnoid space or reversely (Eltorai et al., 2003; Goh and Yeo, 2005; Rahamimov et al., 2009; Rehman et al., 2009; Tejirian et al., 2009). In our case defects of dural sheaths led to leakage of cerebrospinal fluid (CSF) and to decrease of CSF pressure within subarachnoid space. Decreased CSF pressure and increased air pressure in the pleural cavities and mediastinum associated with blunt thoracic injury may lead to the entry of air into subarachnoid space at the cervical level and to consequent transport of air into cranial cavity resulting in pneumocephalus.

Nontraumatic causes of subarachnoid pneumorrhachis are rare and to date, only 2 cases of subarachnoid pneumorrhachis and pneumocephalus have been reported in literature as a lumbal and sacral pressure ulcer complication. Both cases were accompanied by the cerebrospinal fluid leak and infection (Eltorai et al., 2003; Jomir et al., 2009).

Oertel et al. (2006) stressed that iatrogenic causes are as a rule associated with surgical, anesthesiological and diagnostic interventions.

The blunt chest trauma is frequently reported as a cause of pneumomediastinum. The mechanism by which air enters the mediastinum in blunt trauma was first suggested by Macklin (1939). Air escaping from ruptured alveoli can travel along the peribronchial and perivascular connective tissue to the hilum, where it dissects into mediastinum. The Macklin effect is involved in blunt traumatic pneumomediastinum but also in pneumomediastinum arising in various conditions (bronchial asthma crises, subcutaneous emphysema). From the mediastinum, air can enter the intermuscular fascia of the neck and migrate subcutaneously (subcutaneous emphysema). There are no fascial barriers to prevent communications between the posterior mediastinum, the retropharyngeal space and the epidural space. Air can thus freely communicate via the neural (intervertebral) foramina and preferably collects in the posterior epidural space (Wintermark and Schnyder, 2001; Chun and Moon, 2009).

Many cases of pneumomediastinum are caused by iatrogenic injury during dental treatment or surgery on the cervical region and chest (Heyman and Babayof, 1995; Arai et al., 2009).

From the mediastinum air may spread to pericard. Pneumopericardium is an uncommon disorder in adults defined as the presence of air in the pericardium, typically occurring after penetrating thoracic injury, and only rarely after blunt trauma (Zakynthinos et al., 2008). Mediastinal air can enter the pericardial space in the vicinity of the pulmonary veins because pericardial connective tissue is discontinuous at the reflection of parietal onto visceral pericardium at these

points (Macklin, 1939; Zakyntinos et al., 2008). Our data are consistent with these pathophysiological concepts and confirm the importance of severe blunt thoracic trauma for development of pneumothorax and pneumomediastinum and for the consecutive propagation of air to other body compartments.

Regarding the fact that patient's right upper limb was paralytic at admission, there was a pronounced suspicion of brachial plexus nerve root avulsion. MR examination confirmed traumatic lesions of the C7 and C8 nerve roots. The roots were disrupted and initially compressed by hematoma. The distal nerve roots were retracted and retraction ball surrounded by pseudomeningocele was evident. Most of the adult brachial plexus lesions are posttraumatic injuries, secondary to high-energy forces. MRI is a useful method in the evaluation of pathology of the brachial plexus and helps in the identification and accurate localization of its discontinuity (Moran et al., 2005; Gerevini et al., 2008).

Pneumocephalus is defined as the accumulation of intracranial air in the various intracranial compartments, including the epidural, subdural, subarachnoid and intraparenchymal compartments and can result from many different causes. Traumatic pneumocephalus is commonly seen after basilar skull fractures, injury of paranasal sinuses, secondary to stab wounds, thoracic and vertebral surgery and dural puncture or tears (Markham, 1967; Tejirian et al., 2009).

In the present case air accumulated in subarachnoid as well as in subdural space, filled basal cisternae and caused severe compression of frontal lobes. Immediately after opening of the dura air bubbled through dural incision. From the intracranial subarachnoidal space, air eventually entered the subarachnoid space of the optic nerve, dissecting between the enveloping meninges of the nerve. Air within the optic nerve sheaths is a unique radiological finding. To the best of our knowledge, pneumatisation of the optic nerve sheaths in association with cranial injury has only been described in two cases (Agarwal et al., 2002; Kavanagh et al., 2007). The entry of air into optic nerve sheaths has been also described as a rare complication of retrobulbar injection (Morgan et al., 1988). Another possibility is the extension of air from the intracranial cavity or following the fracture of ethmoid, sphenoid and frontal sinuses and skull base fractures via subarachnoid space. Pneumatisation of the optic nerve sheaths is considered as an indicator of poor prognosis in cases of severe head trauma (Agarwal et al., 2002; Kavanagh et al., 2007).

In the present case pneumatisation of the optic nerve sheaths was caused by the direct entry of air from the intracranial subarachnoid space as a complication of the subarachnoidal pneumocephalus. Pneumatisation of the optic nerve did not produce visual deficits in our patient.

Conclusion

This case illustrates an unusual combination of presence of air in several body compartments in consequence of severe blunt thoracic trauma and avulsion of the brachial plexus roots.

The authors report a unique case of a 41-year-old man with polytrauma including the blunt thoracic trauma and paralysis of the right upper limb associated with the pneumothorax, pneumomediastinum, subcutaneous emphysema and pneumopericard. Due to avulsion of brachial plexus roots and cerebrospinal fluid leakage air was detected also in the spinal and intracranial subarachnoidal and subdural space. Massive pneumocephalus caused propagating of air into subarachnoid space of the optic nerve.

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