CLINICAL REPORT

Pneumorachis: A possible source of traumatic cord compression


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Summary
The presence of air within the spinal canal secondary to trauma is a rare condition. These rare cases are generally asymptomatic. We report our first case of closed thoracic trauma with pneumorachis associated with neurological disorders. According to a review of the literature and after personal record analysis, neurologic symptoms can be correlated to the occurrence of intraspinal air. Therefore pneumorachis appears as a possible cause of traumatic spinal cord compression. In this particular case, pneumorachis spontaneously resolved and early outcome was favourable.

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Introduction

Pneumorachis is a relatively rare phenomenon where air enters the spinal canal. Cases of pneumorachis associated with spontaneous pneumothorax and pneumomediastinum were reported [1–3], however its occurrence secondary to a closed thoracic trauma is rare. Only six cases of that type were reported in the literature [3–7]. This condition is typically benign and to our knowledge, no publication has reported neurological deficits associated with traumatic pneumorachis. Therefore, if spontaneous disappearance of intraspinal air was radiographically confirmed in cases with no sign of neurologic compromise [8], the evolution of some deficitary signs remains unknown since it has never been reported up to now.

We reported a case of closed thoracic trauma leading to non-displaced multilevel spinal fractures and pneumorachis associated with partial neurological deficits. The purpose of the present study is to discuss the correlation between neurological disorders and pneumorachis and share our experience regarding the evolution of our patient’s lesions.

Observation

A 23-year-old patient, with no past medical history, was admitted to the emergency trauma department of our insti-
tution after a motocross accident. On admission, his Glasgow score was 15/15 and no hemodynamic or respiratory dysfunctions were observed. The patient complained of upper thoracic dorsal pain. Clinical examination gave evidence of neurological deficits with monoplegia and hypoesthesia in the left lower limb (maximum quotation of 1/5 in all territories). Osseotendinous reflexes were intact with no pyramidal syndrome. A saddle block anesthesia was initially observed associated with sphincter disturbances of acute urinary retention type, which required insertion of a catheter. The patient was conscious but not aware of the catheterization.

A chest CT scan was performed upon the patient’s arrival according to the usual protocol. It revealed a small right pneumothorax, a pneumomediastinum and intraspinal air (pneumorachis) at T7 and T8 levels (Fig. 1). A non-displaced fracture involving the left transverse process and the right articular process of T7 was seen combined with a non-displaced fracture of the left isthmus extending to the base of the pedicle at T8. The location of these multilevel fractures suggested a rotation mechanism but without any displacement or reduction of the canal surface (Fig. 2).

A spontaneous clinical improvement was observed within 6 hours and was associated with recovery of perineal and urethral sensitivity. A slight recovery of motor function was achieved with a left distal quotation of 3/5.

Since bone lesions could not be correlated with neurological deficits, a spine MRI examination was scheduled. It was performed 12 hours after trauma and demonstrated complete disappearance of the initial intraspinal air. MRI did not reveal any medullary hypersignal on T2-weighted images suggestive of contusion or any compression factor such as hematoma, hernia or bone fragment (Fig. 3).

A spinal osteosynthesis was performed in the management of the above-mentioned rotational mechanism to prevent the risk of secondary displacement of this type C fracture according to the Magerl. A T7-T9 posterior screw plate fixation was carried out (Domino® Stryker®, France) without spinal canal opening (Fig. 4). When the patient woke up, his neurologic state remained unchanged and demonstrated recovery of motor functions within the following days. The left lower limb achieved a 4−/5 motor quotation in the proximal region and a 3/5 quotation in the distal region from the fifth postoperative day. Catheterization was removed before the patient was transferred to a functional rehabilitation centre. Normal walking could be achieved at four postoperative months with a 5/5 motor quotation, except for both the extensor digitorum longus and the extensor hallucis longus which quotation was 4+/5.
Figure 4  Postoperative aspect of construct on anteroposterior and lateral radiographs.

Discussion

Pneumorachis is a rare phenomenon. In the present case, it is associated with neurological deficits after closed thoracic trauma which does not correspond to any current data from our review of the literature.

Nosology and pathophysiology

Pneumorachis, defined as the presence of air within the spinal canal, is also described under various terms such as aerorachia or epidural emphysema. It most commonly occurs in the extradural space but may also extend within the subarachnoid space with disruption of the surrounding dura mater spinalis [4].

Pneumorachis associated with pneumomediastinum or pneumothorax has been commonly reported. The most frequent initial mechanism is a marked increase in intra-alveolar pressure (acute asthma, recurrent vomiting or closed thoracic trauma). Alveolar rupture allows air migration along the bronchovascular axis up to the mediastinum. The collected air then separates the mediastinal pleura from the aorta and the parietal pleura from the spine therefore entering the epidural space via the intervertebral foramina [7].

In our observation, the small pneumothorax does not easily give evidence of air distribution between the pleural cavity and the peridural space. However, it is the only pathophysiological explanation described in the literature. Pneumorachis is not only induced by the amount of air collection but also by the suddenness of its occurrence.

As usually described [1] and observed in our study, pneumorachis typically resolves spontaneously within a few hours on MRI imaging.

Aetiologies

The most frequent causes of pneumorachis are iatrogenic aetiologies (peridural anaesthesia, lumbar puncture or spinal surgery). Pneumothorax and pneumomediastinum are commonly associated and should be systematically investigated in case of intraspinal air [7]. However, epidural abscesses due to anaerobic germs may also appear, particularly in patients suffering from diabetes or Crohn’s disease. Pneumocephalus following skull base fracture leading to the presence of air in the cervical medullary canal [8] was reported. It is rarely associated with closed thoracic trauma [6].

Diagnosis and therapeutic management

Diagnosis includes CT scanning of the spine to detect the presence of low density intraspinal air [9]. The clinical aspects are unspecific except for patients with pneumothorax and pneumomediastinum. Pneumorachis usually reabsorbs spontaneously [10] This correlates our findings since complete resorption had been observed on radiographic examination prior to the surgical procedure. Taking into account the associated bone lesions, the indication of surgical stabilization without canal opening was independent from pneumorachis and did not modify the treatment modalities. A single case of surgical treatment of postoperative pneumorachis has been reported. It involved an atypical case of cauda equina syndrome following lumbar spinal stenosis surgery. Inopportune wakening of the patient during repair of a dural defect led to a massive in-draught secondarily entrapped by airtight suture. The patient was reoperated for air removal and demonstrated complete recovery [11]. However, we believe this very specific case cannot be compared with traumatic pneumorachis.

Causative relationship between pneumorachis and medullary signs

In the present case, no bone or tissue compression could be detected. No hypersignal on T2 weighted MRI images could attest to a medullary contusion. However, this negative sign is not helpful for diagnosis of coexisting neurological deficits. Intraspinal air was the only abnormal element suggesting the hypothesis of a transitory compression. Both neurological improvement and spontaneous resorption of the pneumorachis contribute to reinforce these arguments. However, our interpretation of this clinical case should be considered cautiously due to the absence of similar cases in the literature. Moreover, intraspinal air might also be interpreted as the result of a major local instability. Neurological deficits could therefore be attributed to a larger initial displacement of the spontaneously reduced lesion. In the present case, the whole chronological order of events (intrinsic imputability), the absence of other causes and the lack of information collected from the literature on that specific subject (extrinsic imputability) lead us to conclude that neurological disorders were induced by pneumorachis.
Conclusion

Pneumorachis after closed thoracic trauma has rarely been described. Our case suggest that pneumorachis could be one of the various causes for sublesional post-traumatic neurological deficits. Even if not always directly responsible for medullary compression, it promotes instability of a spinal fracture. In our observation, the overall management was not modified despite spontaneous resorption of intraspinal air and satisfactory clinical evolution.

Conflicts of interest statement

None.

References