PICTORIAL REVIEW

Extracranial epidural emphysema: pathway, aetiology, diagnosis and management

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ABSTRACT. Extracranial epidural emphysema is an uncommon phenomenon that refers to the presence of gas within the epidural space. As an isolated finding, it is typically benign, but it can be a secondary sign of more ominous disease processes, such as pneumothorax, pneumoperitoneum and epidural abscess. Although the phenomenon has been cited in case reports, a comprehensive review of this topic is lacking in the radiology literature. The authors' aim is to report our experience with extracranial epidural emphysema, illustrating the spectrum of its clinical presentation. We also review the aetiology, pathophysiology, diagnosis and management of extracranial epidural emphysema.

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Pneumorrhachis (also known as aerorachia) is a term that has been used to describe air within the spinal canal. It can be separated into two distinct entities, epidural and intradural. In general, the presence of intradural intraspinal air has been associated with significant morbidity, while the presence of epidural air is typically more benign [1]. Epidural air within the spinal canal is best descriptively termed extracranial epidural emphysema and has been described sporadically in case reports. The purpose of this pictorial review is to present a diversity of clinical scenarios that can give rise to extracranial epidural emphysema and to discuss its pathophysiology, diagnosis and management.

Methods and materials

A comprehensive literature search was conducted on the MEDLINE database using PubMed. Keywords including pneumorrhachis, pneumosaccus, aerorachia, intraspinal pneumocele, pneumomyelogram, epidural emphysema and epidural pneumatosis were used in the literature search. In addition to a review of the literature, four cases of extracranial epidural emphysema collected at our institution are also reported. They illustrate the common pathways of extracranial epidural emphysema.

All the clinical records were reviewed for demographic data, symptoms and duration of symptoms. The study did not require approval from the institution's

Investigation Review Board. However, the Brooke Army Medical Center Department of Clinical Investigation reviewed and approved the manuscript for publication.

Results

Case 1

A 59-year-old male complained of left flank pain. He had a non-contrast CT of the abdomen and pelvis that showed a left ureterovesical junction stone. The examination also revealed severe degenerative disc disease at L4–5 and L5–S1 disc spaces with vacuum disc phenomenon and extracranial epidural emphysema behind the L5 vertebral body (Figure 1a,b).

Case 2

A 20-year-old male presented with diabetic ketoacidosis and crepitus in the neck and upper chest. He had palpable crepitus over his jaw and a friction rub on cardiac examination. A CT scan of the chest showed severe pneumomediastinum tracking from the mediastinum into the soft tissues of the neck, anterior and posterior chest wall with pneumopericardium and extracranial epidural emphysema (Figure 2a,b).

Case 3

A 49-year-old male with Crohn's disease presented with abdominal pain and distension post colonoscopy for polypectomy and segmental biopsies. A CT scan of the abdomen and pelvis showed pneumoperitoneum, pneumoretroperitoneum, subcutaneous emphysema and extracranial epidural emphysema (Figure 3a,b).

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Figure 1. 59-year-old male with incidental finding of extracranial epidural emphysema on non-contrast CT of the abdomen and pelvis for left flank pain. (a) Axial CT image showed extracranial epidural emphysema (arrow). (b) Sagittal CT image showed extracranial epidural emphysema owing to underlying vacuum disc phenomenon (arrow).

Figure 2. 20-year-old male presented with diabetic ketoacidosis and epidural emphysema. (a) Axial CT image of chest shows pneumomediastinum, pneumopericardium, subcutaneous emphysema and extracranial epidural emphysema (arrow). (b) Portable chest radiograph shows evident pneumomediastinum (arrow).

(a)

(b)

Figure 3. 49-year-old male with Crohn's disease complained of abdominal pain and distension post-colonoscopy. (a) Axial CT image of abdomen and pelvis shows pneumoretroperitoneum, pneumoperitoneum, subcutaneous emphysema and extracranial epidural emphysema (arrow). (b) Scout CT image shows obvious pneumoretroperitoneum, pneumoperitoneum and subcutaneous emphysema.

Case 4

An 80-year-old female presented with severe radiating lower back pain 8 h post total knee arthroplasty. She previously had epidural anaesthesia and an epidural catheter in place. MRI of the lumbar spine revealed an epidural haematoma extending from T7 to L3 with compression of the anterior spinal cord as well as punctate air in the non-dependent portion of the haematoma at the level of T12 (Figure 4a–c).

Discussion

Observers have used many terms to describe the presence of intraspinal air, including pneumorrhachis, pneumosaccus, aerorachia, intraspinal pneumocele, pneumomyelogram, epidural emphysema, and epidural pneumatosis [1]. For the purposes of discussing air within the epidural space of the spinal canal, the most anatomically descriptive way to describe this phenomenon is extracranial epidural emphysema.

Knowledge of the anatomy of the intracranial and extracranial epidural space helps to understand the mechanism by which air accumulates in the extracranial epidural space. Whereas the cranial dura mater comprises two membranes (an endosteal (outer) and a meningeal (inner) membrane), the spinal dura mater represents the continuation of only the meningeal layer

of the dura mater from the level of the foramen magnum. The spinal dura mater continues as an enveloping cylinder to the second sacral vertebral level and then envelopes the filum terminale as the filum terminale externum that attaches caudally to the dorsum of the first coccygeal vertebrae. The arachnoid mater closely abuts the dura throughout its course, while the pia mater continues as the filum terminale from the tip of the conus medullaris [2].

The epidural space of the spinal canal is therefore a separate entity from the intracranial epidural space as the intracranial epidural space ends with the termination of the endosteal layer at the foramen magnum. This distinction is critical for two reasons. First, the development of epidural emphysema intracranially will not necessarily lead to extracranial epidural emphysema and *vice versa*. Secondly, introduction of intracranial epidural emphysema requires either penetration of the calvarium or skull base or presence of a gas-forming organism along the intracranial dura mater. The epidural space of the spinal canal can be accessed in a variety of ways via the vacuum disc phenomenon of the intervertebral discs [3], the neuroforamina [4,5], the direct introduction of gas via spinal procedure (open surgical procedure or lumbar puncture) or open fracture of the axial skeleton [6], the venous plexus of the spine via communication with the pelvic venous system [7], gas-producing organisms from an epidural abscess [8], or the disruption

Figure 4. 80-year-old female complained of severe radiating low back pain 8 h post-total knee arthroplasty. (a, b) Sagittal T_1 and T_2 MRI show epidural haematoma (thick arrow) and focus of low-signal intensity (thin arrow). (c) Axial T_2 MRI show epidural haematoma (thick white arrow) and focus of low signal intensity (thin black arrow).

of the endosteal termination of the cranial dura mater via skull fracture and dissection of intracranial epidural air into the extracranial epidural space. Figure 5 depicts these pathways.

Multiple case reports have described clinical scenarios that lead to the development of extracranial epidural emphysema. Of the clinical scenarios, the most common is probably due to rupture of the vacuum disc. The presence of gas within an intervertebral disc is common and occurs in 2.0–3.2% of the standard population but may be seen in up to 20.8% in elderly populations [9]. Presence of gas within an intervertebral disc can expand or contract based on position of the patient and, rarely, can lead to pressure on neural elements [3, 10, 11]. Although not yet reported, aerorachia may potentially be seen in association with facet joint vacuum phenomenon. In Case 1, the extracranial epidural emphysema was discovered incidentally and seen in the context of the vacuum disc phenomenon. Because of the common occurrence of vacuum disc phenomenon, it is probably the most common aetiology of extracranial epidural emphysema.

Another common scenario is barotrauma in the setting of pneumomediastinum or pneumothorax [6], which tracks along the fascial planes through the neuroforamina and into the epidural space. This is possible because of the lack of true fascial envelopes protecting the spinal epidural space [5]. The lack of fascial planes overlying the epidural space would permit air under significant pressure to dissect along pre-vertebral fascial planes and into the epidural space [6, 12]. In Case 2, the aetiology of the patient's extracranial epidural emphysema is owing to pneumomediastinum from barotraumas induced by hyperpnoea or severe vomiting from diabetic ketoacidosis. Diabetic ketoacidosis is a rare cause of pneumomediastinum and therefore a rarer

cause of extracranial epidural emphysema; however, the actual incidence of pneumomediastinum complicating diabetic ketoacidosis is unknown [13, 14].

Another potential pathway of extracranial epidural emphysema can arise from peritoneal sources. Multiple case reports have described extracranial epidural emphysema occurring in the context of intestinal necrosis [7], surgical resection of bowel [15] and in the presence of inflammatory bowel disease [16]. Aetiologies for introduction of extracranial epidural emphysema from intra-abdominal source have included air introduction into pelvic veins, which communicates with the epidural space via the lumbar venous plexus; however, air could also dissect along a tissue plane through the retroperitoneum and via neuroforamina into the epidural space. In Case 3 the patient underwent colonoscopy, and extracranial epidural emphysema was found in addition to pneumoretroperitoneum, pneumoperitoneum, pneumomediastinum and subcutaneous emphysema. Given that the reported risk of perforation following colonoscopy is 0.2% or less in skilled operators [17], the incidence of extracranial epidural emphysema from intra-abdominal sources is likely to be rare.

Another cause of extracranial epidural emphysema is via surgical, diagnostic or anaesthesiological interventions that violate the epidural space [1, 6]. In Case 4, a tiny amount of extracranial epidural emphysema was seen in association with a symptomatic epidural haematoma, a known complication with a reported incidence of less than 1 per 150 000 cases of epidural anaesthesia [18]. The likely subclinical incidence of epidural haematoma may be higher and may be accompanied by extracranial epidural emphysema via iatrogenic introduction.

Another potential mechanism for development of extracranial epidural emphysema is via open fracture

Figure 5. Composite diagram of skull, lower cervical and upper thoracic cross-sections illustrating different pathways of gas entry into extracranial epidural space. (1) Rupture of intervertebral vacuum disc introduces gas into the epidural space. (2) Air due to barotrauma (pneumothorax, pneumomediastinum, pneumoperitoneum, pneumoretroperitoneum) can gain access to the epidural space via the neuroforamina. (3) Air can be introduced into the epidural space by lumbar puncture, epidural anaesthesia and spinal fracture or surgery. (4) Intraabdominal infection may release gas into pelvic veins which communicate with the epidural space via the lumbar venous plexus. (5) Intracranial epidural emphysema resulting from fracture of the skull or air containing cavity can dissect into the extracranial epidural space under high intracranial pressure.

of the skull or spine and/or fracture of an air-containing cavity within the skull. While spinal fractures are a noted cause of traumatic extracranial epidural emphysema [6], open fractures of the skull and fractures of bony air cells (such as mastoid air cells and paranasal sinuses) are not. Presumably, direct introduction of extracranial epidural emphysema could occur from an open skull fracture or sinus fracture if there is disruption of the transitional zone between the cranial and spinal dura matter and high intracranial pressure, thus providing a communication between the extracranial and intracranial epidural spaces.

Extracranial epidural emphysema is diagnosed most easily by CT, which reveals a hypoattenuation isodense with air, best seen on either lung window setting. On MRI, T_1 and T_2 weighted images reveal a hypointense lesion which shows bloom artefact on T_2 or gradient echo (GRE)-weighted sequences consistent with gas. Radiography may also suggest the diagnosis of extracranial epidural emphysema if the air collection is large and a lateral view of the chest or spine is obtained. However, most cases of extracranial epidural emphysema are too small to be detected by radiography.

Extracranial epidural emphysema rarely comes to clinical attention as an isolated phenomenon and is usually identified in the context of other organ injury

with migration of gas into the epidural space. Hence, extracranial epidural emphysema is usually asymptomatic; however, sensorimotor defects have been observed in case reports of patients with extracranial epidural emphysema [1, 6]. Management of extracranial epidural emphysema is accomplished through treatment of the underlying aetiologies. Clinically, increasing inspired concentrations of oxygen may improve nitrogen washout and reduce the relative volume of the gas collection. If general anaesthesia is required in a patient with epidural pneumatosis, the anaesthetist should avoid using inhalational nitrous oxide because it may cause expansion of intraspinal air and result in an increase in cerebrospinal fluid pressure. Additionally, the anaesthetist should use alternative anaesthetic techniques such as intermittent positive pressure ventilation and avoid pressurisation of the oropharynx and nasopharynx in order to prevent an increase in the volume of any intraspinal air and promote faster reabsorption of air [1].

Conclusion

Extracranial epidural emphysema is rare. There are several pathways for introduction of gas into the epidural space. The majority of patients with extracranial epidural emphysema are asymptomatic with treatment of extracranial epidural emphysema based on the underlying aetiology. Vacuum disc phenomenon is probably the most common cause of extracranial epidural emphysema; however, the most common cause of clinically significant extracranial epidural emphysema is barotrauma. Therefore, when extracranial epidural emphysema is identified, the radiologist must look carefully for pneumomediastinum, pneumothorax, pneumoperitoneum and pneumoretroperitoneum as these aetiologies may be life threatening.

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