Pneumomediastinum – What Can We Learn?

Pneumomediastinum is a common disorder which physicians, surgeons and pediatricians meet in clinical practice and on a hospital basis.

A wide variety of causes has been described to date including mediastinal, pulmonary, chest wall and even retroperitoneal pathologies resulting from defective connective tissue disorders, trauma, infection, assisted ventilation and trivial routine life activity like vigorous coughing, vomiting or deep respiratory efforts (1–4). Chest pain and dyspnea are not uncommon presenting symptoms and may aggravate preexisting diseases, however pneumomediastinum may often be asymptomatic with no grave sequela. What can we learn from this common, benign course taking pathology? Let us look at a few basic facts.

First, air allows us to demonstrate the clear anatomy of the mediastinum on the radiological images and second, through air travel routes to various locations and structures from the mediastinum or vice versa, anatomical continuity can be visually and vividly understood through the modern technology of computed tomography, which is far superior to conventional radiographic methods in terms of X-ray contrast resolution of various organs and tissues of the body.

There are several air containing structures in the body like the esophagus, bowels, trachea or lungs and interestingly enough, they could all serve as air supply sources to the mediastinum in a variety of events. The trachea and esophagus lie within the mediastinum but the stomach and bowels are located in the peritoneal cavity and retroperitoneal space (5). It is well known that pneumatosis intestinalis or perforation of colonic diverticuli could cause pneumomediastinum (4). There is another interesting report that dental extraction followed by fluorine gas insufflation triggered pneumomediastinum (6). Even paranasal sinuses could be the air supply source (1). The most common of these sources is reported to be the lung.

Overdistended alveoli may rupture easily with increased alveolar pressure and/or in the weakened, injured wall, which occurs in many pathological conditions.

Bronchial asthma is one of those typical airway obstructive diseases in which bronchial spasm and mucous plugging play roles, resulting in air trapping and the rise of alveolar pressure. Straining of weight lifting, defecation, vigorous coughing or deep respiratory action and anxiety accompanied by dyspnea also enhance the likelihood of the alveolar rupture.

Air leaked through ruptured alveolar walls enters the perivascular and peribronchial interstitial spaces with least resistance to pass centrally to the mediastinum; this has been termed interstitial pulmonary emphysema (1, 2, 7).

Interstitial pulmonary emphysema has a lucent reticular, mesh-like appearance mixed with bronchovascular markings on plain radiographs and is said to be easier to detect on neonate radiographs, but it is actually difficult to see. It is a pre-warning sign to the next consequence of pneumomediastinum or pneumothorax. It is much easier to detect air in the peribronchovascular interstitial spaces by computed tomography (CT) images as beautifully demonstrated in the article of this issue, though next to impossible to obtain in the neonates in the real clinical settings (8).

See also p 877.

Mediastinal air circulates and dissects fat tissues in every direction with no specific boundaries; much of it ascends the neck through the carotid space, retropharyngeal or prevertebral space and along the trachea or muscle planes. Some spreads through the subcutaneous fatty tissue of the neck, face, upper trunk or extremities, causing “packed snow grasp” or a “grinding sound” sensation on physical findings. Pneumothorax, a common association of pneumomediastinum, is reported at a frequency of 10–50 percent of different clinical settings, but there is a strict connective tissue boundary between the mediastinum and the pleural space.

Why then, is there the common occurrence of pneumothorax? It is universally accepted that high pressured mediastinal air often ruptures the pleura into the pleural space for decompression but no such reversed phenomenon occurs. Primary pneumothorax just compresses or displaces the mediastinum without tearing the mediastinal pleura (1, 9). This indicates the compressibility of the lungs and consequently, the potential expansiveness of the pleural space.

Mediastinal air also extends directly to the extraperitoneal and extrapleural space below the diaphragm which simulates pneumoperitoneum on the radiograph. An easy means of differentiation from pneumoperitoneum is to take another radiograph from different position because the air in the closed space tends to preserve the shape and location in different postures.

Air flow to the lung from the mediastinum has never been reported, probably reflecting a higher pressure system of the pulmonary interstitial spaces. The retroperitoneum, subserosal intestinal wall and the mesenteric fat tissues are all the continuous spaces through the aortic, esophageal and inferior vena caval hiatus to the mediastinum. Air travels easily and naturally through these structures without the need for any extraforce, simply by the rules of nature.

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References