Spontaneous Pneumomediastinum in Pneumocystis Pneumonia and Acquired Immunodeficiency Syndrome
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Citation

Abstract
Pneumomediastinum is defined as air within the mediastinal cavity, and is usually accompanied by subcutaneous emphysema. It is a relatively uncommon and infrequently reported entity, specifically in patients with AIDS. Reported is a 39-year-old male with chest pain and radiologic findings of tension pneumomediastinum, prompting emergent surgical consultation for decompression. Due to the patient's hemodynamic stability, a conservative approach of observation was employed. Over the following days the patient's pneumomediastinum decompressed naturally into the neck and eventually resolved. Radiographic studies are invaluable in diagnosing and following individuals with pneumomediastinum, but tension pneumomediastinum by radiologic evidence alone is not an indication for surgical intervention.

INTRODUCTION
The presence of air within the mediastinal cavity is an unusual finding on a routine chest X-ray. Pneumomediastinum can occur spontaneously or secondary to trauma or pathological disease states. In most instances the pneumomediastinum resolves without treatment, though in rare cases surgical intervention is needed. It is rarely described in patients with Acquired Immunodeficiency Syndrome (AIDS), although their frequent pulmonary complications make them at risk for its development.1,2,3,4 Below, we report the case of a patient with AIDS and Pneumocystis jerovici pneumonia (PJP) who developed the findings of pneumomediastinum while hospitalized for worsening respiratory function.

CASE REPORT
A 39-year-old Hispanic male presented to the Emergency Department with progressively worsening shortness of breath, pain on inspiration, productive cough, fevers, and new-onset lower extremity weakness over the past 2 weeks. His past medical history was significant for a positive Tuberculin, Purified Protein Derivative (PPD) test present since 1988, and treated with one-year isoniazid therapy. He was diagnosed in 2000 with AIDS following a hospitalization for severe community acquired pneumonia. His past medical history was significant for hepatitis A. On presentation in the ED, the patient denied night sweats, hemoptysis, nausea, or vomiting. He smokes 10 cigarettes per day, and has done so for the last few years. He is a past intravenous heroine user on a methadone maintenance program. He has no history of asthma or other pulmonary disease.

On physical exam the patient was noted to be cachectic and dyspneic. He was afebrile, with a blood pressure of 98/64, tachycardic to a heart rate of 100/minute, a respiratory rate of 24/minute, and oxygen saturation on room air of 93%. He had oral thrush. No nuchal rigidity or tenderness were noted. He had palpable cervical lymphadenopathy. His lung exam had faintly coarse breath sounds with good air movement bilaterally. The remainder of his physical exam was unremarkable. A chest X-ray showed bilateral diffuse ground glass opacity in both lung fields.

The patient was admitted with a presumed diagnosis of PJP and was started on intravenous antibiotics and steroids. Bronchoscopy for confirmation of the diagnosis was deferred due to the patient's tenuous respiratory status. Over the subsequent days the patient's respiratory status worsened, requiring a non-rebreather facemask to maintain adequate oxygen saturation.

Ten days post-admission, the patient began to complain of a sensation of panic and chest pain with worsening dyspnea. A chest X-ray from that day revealed the new finding of
pneumomediastinum in addition to his bilateral infiltrates. A subsequent computed tomography (CT) scan showed intraparenchymal cysts consistent with PJP and extensive mediastinal air with compression of the lungs and heart. (See Image 1. Chest CT showing antero-posterior compression of the heart)

**Figure 1**

On exam the patient was found to have distant heart sounds without audible mediastinal crepitus. With the radiologic diagnosis of “pneumomediastinum with a ‘tension’ component causing considerable compression of the intrathoracic viscera” reported, an emergent surgical consult was requested.

On evaluation by the thoracic surgical team, the patient demonstrated stable hemodynamic parameters and a respiratory status that required the use of the non-rebreather facemask, but was stable and unchanged over the previous few days. A small amount of subcutaneous air was palpated in the lower neck. Despite the radiologic inference, no hemodynamic or respiratory collapse was imminent. The decision was made to hold off on any type of surgical decompression, such as emergent placement of a mediastinostomy tube or a percutaneous drainage catheter. The patient was monitored closely both clinically and with daily chest X-rays.

Over the next few days the subcutaneous air within the patient's neck increased dramatically, while his pulmonary status remained unchanged. (See Image 2 – Subcutaneous emphysema of the neck).

**Figure 2**

A follow up CT scan showed marked improvement in the amount of mediastinal air. Over time the subcutaneous air resolved completely. The patient was eventually discharged to a subacute nursing facility.

**DISCUSSION**

Pneumomediastinum was first formally described by Hamman in 1939. However evidence of its recognition are found in writings as early as 1617. In the passage, midwives reported the association of pushing without proper breathing (Valsalva maneuver) during childbirth with a stabbing chest pain and the development of a swollen neck. Without X-ray as the diagnostic tool the only visible manifestation of pneumomediastinum would be the subcutaneous emphysema that developed in the neck. In 1819 René Laennec documented an emergent decompression of what is now known as tension pneumomediastinum in a 4-year-old boy in extremis following trauma. Laennec inserted sharp sticks into the anterior mediastinum and neck of the boy causing a notably large rush of air, after which the child's condition immediately improved.

The presentation of spontaneous pneumomediastinum is variable, but the predominant symptom is pain, classically described as substernal or precordial in location with radiation to the neck, back, and shoulders. Onset is acute
but subsides gradually. In most cases its subsidence is associated with the development of subcutaneous emphysema as the mediastinal compartment decompresses into the surrounding tissues. Aside from the associated subcutaneous emphysema, other clinical signs include the mediastinal crepitation heard with the systolic contraction of the heart, now known as Hamman’s crunch. The incidence of this finding has been reported as occurring in 40-64% of cases. Roentgenographic evidence of air within the mediastinum is diagnostic for the entity. Free air is readily seen as a thin line of radiolucency outlining the cardiac border on posterior-anterior projections. A lateral projection will show the posterior mediastinal structures clearly outlined.

In most cases of pneumomediastinum, air enters through alveolar or pneumatocele rupture. It then tracks along the pulmonary vessels and interstitium to the hilum, where it eventually accumulates. The high incidence of PCP and other cavitary or necrotizing pneumonias seen in the AIDS population makes them particularly predisposed to this condition. In addition to being caused by underlying alveolar pathology, pneumomediastinum can be caused by rupture of “normal” alveoli following the application of high extrinsic pressures, i.e., Valsalva maneuvers. Occurring during exertion or performed during the inhalation of illicit substances, the rapid fluctuations in alveolar pressures can result in rupture. The main iatrogenic cause of pneumomediastinum is mechanical ventilation via barotrauma, especially when elevated levels of positive pressure are used. Gross disruptions of the tracheobronchial tree as seen in traumatic intubations or deceleration injuries, as well as esophageal rupture as seen in Boerhaave’s Syndrome, will also result in the escape of air directly into the mediastinal space.

When air continues to enter the mediastinum without decompression into surrounding tissues as subcutaneous emphysema, the pressure can build to a tension pneumomediastinum. This buildup of air can impair venous return and limit cardiac filling in a tamponade-like picture. If there is any hemodynamic compromise or respiratory distress (related to tracheal compression), the tension pneumomediastinum should be rapidly decompressed. This can be accomplished by needle/catheter insertion into the mediastinal compartment, or through a suprasternal/tracheostomy-style incision.

For a patient with radiographic signs of a tension-like concavity of the cardiac border, but without signs of extremis, it is reasonable to manage conservatively in the face of hemodynamic and respiratory stability. The majority of cases radiographically-labeled as “tension pneumomediastinum” will decompress into the surrounding tissue on their own over 24-48 hours. (See Image 2)

Figure 3

Until spontaneous decompression occurs, close monitoring should be maintained, with a low threshold for surgical decompression at any signs of hemodynamic compromise.

CONCLUSION

Pneumomediastinum is a rare finding and usually a benign entity. Even in the face of radiographic evidence of “tension” pneumomediastinum one should be cautious and avoid doing an unnecessary surgical decompression. As long as the patient maintains hemodynamic stability a wait and watch approach is recommended. The vast majority will decompress and resolve without intervention. This approach proves specifically valuable in immunocompromised patients where a suprasternal incision or needle decompression would carry significant morbidity.

References
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