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Author

Kieu, Anh

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CLINICAL VIGNETTE

Primary Spontaneous Pneumothorax

Anh Kieu, M.D.

The patient is a 24-year-old male nonsmoker with a history of asthma. He works as an auditor at a hotel. He has a male partner and his HIV status is negative. He was at work when he felt a pulling sensation in his lower left back and lower chest, without antecedent trauma. He developed shortness of breath and chest pain after the pulling sensation. Over the next day, his pain worsened with exacerbation with coughing and sneezing and dyspnea on exertion. There was no fever, chill, URI symptoms, or leg edema. He has a cousin with history of pneumothorax.

On exam, he was a tall thin man with normal temperature, blood pressure of 116/71, heart rate was 74, 22 respirations and 100% saturation on room air. He was sitting comfortably but had chest pain with movement. He had decreased breath sounds at the left base; his cardiac exam and abdominal exam were normal. He had no peripheral edema.

Laboratories revealed with normal white cell count, his hemoglobin and hematocrit were 9.7 and 29 respectively, his platelets were 126. His coagulations tests were normal. His d-dimer was negative. His troponins were negative.

CXR showed a large left hydropneumothorax with near complete collapse of left lung, and minimal rightward shift of mediastinum. The patient underwent chest tube insertion and xray showed partial reexpansion of the left lung, with residual moderate to large pneumothorax. His left hemidiaphragm was elevated. The chest tube drained 2300 ml of thick bloody fluid. The patient subsequently had placement of a thoracostomy tube for his hemothorax.

CT scan showed incomplete reexpansion of the left lung and left apex loculated hemothorax. He had retained clot in the thoracostomy tube.

The patient was seen by thoracic surgery and taken to the operating room. Bronchoscopy showed no endobronchial lesions or masses. A left sided VATS

port incision was done at the 8th intercostal space in the midaxillary line. The thoracoscope showed hemothorax in left apex with resultant atelectasis of left upper lobe. Blood clots were present overlying the pericardium and left paravertebral area extending down to the diaphragm. Two additional VATS port incisions were used to drain the hemothorax. The left upper lobe apical segment had a raw surface with dense blood clots attached. This was thought to be the site of the air leak and was resected as a wedge. The left lower lobe superior segment had overlying blood clots and after the clots were removed revealed an inflamed area with a raw surface of the superior segment which was resected with stapler.

Doxycycline was instilled in the left pleural cavity. The patient also had mechanical pleurodesis by mechanically abrading the parietal pleura with multiple scratch pads from apex to diaphragm.

After surgery, the patient's drainage diminished. Follow-up CXR revealed reexpansion of left lung with trace apical pneumothorax. The chest tube was placed to water seal and then removed and the patient was discharged home. On postoperative visits, his CXR showed no pleural effusion or pneumothorax. Pathology showed reactive only mesothelial hyperplasia and the patient had negative serologic testing for occult collagen vascular disease. Follow up chest CT showed left upper lobe wedge resection with no pneumothorax, nodules, masses, or pulmonary embolism. He has had no recurrences 1 year out from his pneumothorax.

Introduction

Pneumothorax is defined as the presence of air in the pleural cavity. Pneumothoraces are divided into spontaneous (occurs without preceding trauma in someone without history of lung disease) and traumatic pneumothorax (occurs after direct or indirect trauma). Spontaneous pneumothoraces are further divided into primary and secondary spontaneous pneumothoraces¹.

Some causes of secondary pneumothorax include COPD with emphysema, cystic fibrosis, tuberculosis,

lung cancer, pneumonia and HIV associated pneumocystis carinii pneumonia, lung cancer. COPD is the most common cause^{1,2}.

Due to already reduced lung function in patients with secondary pneumothorax, the occurrence of pneumothorax can be potentially life-threatening. Signs and symptoms include dyspnea, chest pain and hypoxemia. The recurrence rate is higher than in primary spontaneous pneumothorax, up to 80% in cystic fibrosis patients².

Primary spontaneous pneumothorax has an incidence of 7.4 cases per 100,000 in males in the U.S. and 1.2 cases per 100,000 in females in the U.S. Risk factors include male gender, cigarette smoking, and a tall thin body build³. Spontaneous pneumothorax can also be precipitated by change in atmospheric pressure and exposure to loud music⁴.

Pathogenesis

Blebs and bullae are found in 48-79% of patient with unilateral primary spontaneous pneumothorax and appear to have a role in the pathogenesis. Bilateral blebs and bullae are seen on CT of the chest in patients with unilateral primary spontaneous pneumothorax. Blebs and bullae can be seen in 89% of patients on the ipsilateral side and up to 80% on both sides¹.

The formation of blebs and bullae are associated with degradation of elastic fibers caused by an imbalance between proteases and antiproteases and between oxidants and antioxidants. Neutrophils and macrophages also play a role. The site of rupture of the pleura appears to be outside the blebs/bullae because no communication is seen between the pleural cavity and the site of blebs/bullae¹. On thoracoscopy, disrupted mesothelial cells at the visceral pleura are replaced by an inflammatory elastofibrotic layer with increased porosity, allowing air leakage into the pleural space⁴.

Smoking increased risk of primary spontaneous pneumothorax and is associated with extensive respiratory bronchiolitis which can impact the recurrence rates of primary spontaneous pneumothorax^{3, 5}. The relative risk of primary spontaneous pneumothorax is seven times higher in light male smokers, (1-12 cigs/day), 21 times higher in moderate smokers (13-22 cigs/day) and 102 times higher in heavy smokers (>22 cigs/day). For women: the relative risks are 4, 14, and 68 times higher than in nonsmokers⁶. On lavage, the BAL fluid is noted

to contain increased numbers of macrophages in the small airways of smokers. Microscopic analysis shows obstruction and stenosis of distal airways due to bronchial wall inflammation and peribronchial fibrosis¹.

Clinical Presentation

In primary spontaneous pneumothorax, patients experience spontaneous ipsilateral chest pain, usually at rest with mild dyspnea. Tension pneumothorax develops in 1-2% of cases^{4, 7}.

Treatment

If the pneumothorax is small (less than 2-3 cm between lung and chest wall), is a 1st episode, and the patient is stable, treat by administering oxygen and observe for 6 hours. If there is no progression of the pneumothorax, the patient can be discharged home. The normal rate of resorption is 1.25% of the volume of the pneumothorax per 24 hours. Supplemental oxygen increases the rate of resorption. The resorption rate is 6 times faster if humidified 100% oxygen is used^{7, 8}.

If this is a 1st occurrence and the patient is stable and the pneumothorax is greater than 3 cm rim of air or if symptomatic with chest pain or dyspnea, needle aspiration with indwelling catheter should be performed. The air is manually aspirated from an indwelling catheter until no more can be withdrawn. If after 4 liters of air aspirated and there is no resistance on aspiration, a chest tube is inserted and thoracoscopy can be considered during the same hospitalization. If there is resistance and no more air is aspirated, the patient can either go home with the catheter and Heimlich valve or be observed and followed up with chest xray 4 and 6 hours after the aspiration was performed. If the lung remains expanded on follow up chest xrays, the catheter can be removed and patient discharged home⁷.

For unstable patients, a chest tube should be placed. Chest tube should be clamped for 12 hours after the last air leak was noted and the lung has re-expanded. A follow up chest x-ray is done 24 hours after the last air leak, and the chest tube can be removed if no air has reaccumulated⁷.

For patients with persistent air leak for greater than 3 days, options of treatment include 1) video-assisted thoracoscopy (VATS) to oversee the area of the leak and perform mechanical pleurodesis, 2) attach flutter valve to the chest tube which can allow the patient to

be discharged with outpatient follow-up, 3) autologous blood patch. For patients with less than 90% lung expansion with persistent air leak, the preferred procedure is VATS⁷.

Recurrences

The recurrence rate of primary spontaneous pneumothorax treated with observation alone was 32% . Needle aspiration or chest tube evacuation was associated with recurrence rate of 30%. The majority of recurrences occurred within 6 months to 2 years¹.

Patients with recurrent primary spontaneous pneumothorax should receive treatment to prevent recurrence. Options include pleurodesis via VATS, chemical pleurodesis, or thoracotomy⁷.

The rate of recurrence is less than 5% after VATS and pleurodesis. The methods of pleurodesis include using talc or tetracycline derivative instillation into the pleural cavity, mechanical abrasion of the pleural surfaces and laser abrasion of the parietal pleura⁷.

Chemical pleurodesis can be used for patients unable or unwilling to undergo VATS. The recurrence rate is decreased to 20-25% after using doxycycline or tetracycline. Intrapleural doxycycline can be painful, limiting its use. Talc also can be used with recurrence rate of 5-8%, but there is concern for complications of malignant pleural effusions and ARDS⁷.

Thoracotomy is reserved for patient if thoracoscopy is unavailable or has failed. Apical pleural blebs are oversewn and the pleura is scarified⁷.

Smoking cessation can help prevent recurrence.

REFERENCES

1. **Schramel FM, Postmus PE, Vanderschueren RG.** Current aspects of spontaneous pneumothorax. *Eur Respir J.* 1997 Jun;10(6):1372-9. Review. PubMed PMID: 9192946.
2. **Light RW.** Secondary spontaneous pneumothorax in adults. *UpToDate.*
3. **Light RW.** *Pleural Diseases, 6th ed.* Lippincott, Williams and Wilkins, Philadelphia 2013.
4. **Noppen M.** Spontaneous pneumothorax: epidemiology, pathophysiology and cause. *Eur Respir Rev.* 2010 Sep;19(117):217-9. doi: 10.1183/09059180.00005310. Review. PubMed PMID: 20956196.
5. **Cheng YL, Huang TW, Lin CK, Lee SC, Tzao C, Chen JC, Chang H.** The impact of smoking in primary

- spontaneous pneumothorax. *J Thorac Cardiovasc Surg.* 2009 Jul;138(1):192-5. doi: 10.1016/j.jtcvs.2008.12.019. Epub 2009 Feb 23. PubMed PMID: 19577078.
6. **Bense L, Eklund G, Wiman LG.** Smoking and the increased risk of contracting spontaneous pneumothorax. *Chest.* 1987 Dec;92(6):1009-12. PubMed PMID: 3677805.
7. **Light RW.** Primary spontaneous pneumothorax in adults. *UpToDate.*
8. **Haid MM, Paladini P, Maccherini M, Di Bisceglie M, Biagi G, Gotti G.** Air transport and the fate of pneumothorax in pleural adhesions. *Thorax.* 1992 Oct;47(10):833-4. PubMed PMID: 1481187; PubMed Central PMCID: PMC464071.

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