Evaluation and management of pneumothorax

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ABSTRACT

Pneumothorax is defined as the presence of air within the pleural space and implicates that either the visceral or parietal pleura have been disrupted. In this article the classification of pneumothorax, incidence, pathogenesis, and the radiological evaluation and different quantitative measures of pneumothorax were reviewed. In this communication, we also explored the goals of therapy and the variable treatment options, and elaborated on re-expansion pulmonary edema as a potentially serious complication of pleural drainage. Finally, air travel as a predisposing factor for secondary pneumothorax, the possible pathogenesis and incidence were discussed.

Definition of pneumothorax. Pneumothorax is defined as the presence of air within the pleural space and implicates that either the visceral or parietal pleura have been disrupted.

Classification of pneumothorax. Pneumothorax can be classified as spontaneous or traumatic. Spontaneous pneumothorax can be further divided to primary or secondary. Traumatic pneumothorax can be either iatrogenic or related to chest wall trauma.

Primary spontaneous pneumothorax. Primary spontaneous pneumothorax, is a primary process that occurs in the absence of underlying clinical or radiological lung disease. However, most patients with a primary spontaneous pneumothorax do have unrecognized lung disease, as many observations suggest that the pneumothorax may result from the rupture of a subpleural bleb.

Incidence. The incidence of primary spontaneous pneumothorax has been estimated at between 2-18 cases/ 100,000 population/year. The peak incidence is between the second and the third decade of life. Smokers are more likely to develop primary spontaneous pneumothorax.

Pathogenesis. Primary spontaneous pneumothorax develops secondary to a visceral pleural tear, which results from rupture of pleural blebs and bullae. They are usually apical in location and communicate partially with the tracheobronchial tree. Several theories exist regarding the development of blebs and bullae in patients with primary spontaneous pneumothorax. In the case of blebs, the pathogenesis is attributed to the dissection of air from a ruptured alveolus into the thin fibrous layer of visceral pleura where it accumulates forming cysts. Although that the blebs may be present congenitally, acquired causes, however, are developed as a result of local inflammation (for example, caused by tobacco smoking) or secondary to disturbed collateral ventilation. Their formation is probably associated with the degradation and weakness of the elastic fibers of the lung. Such process is caused by the imbalance between protease and anti-protease and oxidants and anti-oxidants, in which inflammatory cells (mainly neutrophils and macrophages) play a major role. This is supported by the fact that smoking is associated with an increase in the relative risk of developing pneumothorax in a dose-response relationship. Compared to non-

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smokers, the relative risk of a pneumothorax in men is 7 times higher in light smokers (less than 12 cigarettes per day), 21 times higher in moderate smokers (13-22 cigarettes per day), and 102 times higher in heavy smokers (more than 22 cigarettes per day). In females, the risk is 4, 14 and 68 times. The inflammatory changes in the distal airways of smokers may lead to endobronchial obstruction in the small airways leading to "check-valve" mechanism, inducing over pressure in alveolar tissue, which results in rupture of pulmonary parenchyma. The mechanisms behind the formation of bullae are even less clear than those of blebs. The current evidence is directed towards the possibility of regional damage to the apical portion of the lung. This damage is probably related either to ischemia or to the greater distensive forces of apical alveoli caused by more negative pleural pressure at the apices. A high frequency of occurrence of primary spontaneous pneumothorax has been noted after abrupt swings in atmospheric pressure. A 46% increase in the incidence of primary spontaneous pneumothorax has been reported when the atmospheric pressure fell 10 millibars or more within the preceding 48 hours.

Secondary spontaneous pneumothorax. Secondary spontaneous pneumothorax develops in association with a wide variety of pulmonary diseases of either a focal nature or with a diffuse involvement of the lung. Among all pulmonary diseases associated with pneumothorax, chronic obstructive pulmonary disease (COPD) is the most common. Secondary to appropriate prophylaxis, the role of acquired immunodeficiency syndrome (AIDS) and pneumocystis carinii pneumonia infection is declining as a major etiology of secondary spontaneous pneumothorax. The incidence of secondary spontaneous pneumothorax has been estimated at between 2-6 cases/100,000 population/year. The peak incidence is at 60-65 years of age.

Pathogenesis. Two mechanisms can lead to secondary spontaneous pneumothorax; 1. Hyper-expansion of the distal airspace due to the obstruction of inflammed airways, or 2. Visceral pleural tear due to the erosion of the visceral pleura by an underlying inflammatory parenchymal process such as necrotizing pneumonia.

Catamenial pneumothorax. This is a secondary spontaneous pneumothorax. It is a syndrome of recurrent pneumothorax in women occurring within 72 hours of the onset of the menses. Although uncommon, its recognition is important, since the management is distinct from other forms of spontaneous pneumothorax. Incidence: Catamenial pneumothorax is a rare disease. Approximately, 3%-5% of secondary pneumothoraces in women is catamenial. The peak incidence is 30-40 years of age.

Pathogenesis. The pathogenesis of catamenial pneumothorax remains obscure. The first hypothesis is that air enters the peritoneal cavity during menstruation, then escapes into the thoracic cavity through a diaphragmatic defect. This defect might be related to either a congenital defect or it may be caused by a breakdown of diaphragmatic endometrial implants. Diaphragmatic defects, however, were not demonstrated in the majority of cases. The second hypothesis is the presence of a focal implant of endometrial tissue either on the pleural surface or in the sub-pleural lung parenchyma, with pneumothorax resulting from the swelling of the implant in response to hormonal changes. The third hypothesis is the development of an alveolar rupture as a consequence of vascular and bronchial damage caused by an increased serum level of Prostaglandin F (PGF2), known to exist in menstruating women, or by local effects of PGF2 from the local implant.

Traumatic pneumothorax. 1. Iatrogenic Pneumothorax. The etiology and incidence of iatrogenic pneumothorax are shown in **Table 1**. 2. Traumatic Pneumothorax. The etiology of traumatic pneumothorax is shown in **Table 1**.

Tension pneumothorax. This is defined as the occurrence of a pneumothorax with the development of intrapleural pressure exceeding atmospheric pressure throughout all or most of the respiratory cycle. It is considered a medical emergency with an acute deterioration in cardio-pulmonary functions.

Incidence. Tension pneumothorax develops in association with a spontaneous pneumothorax in 1-2% of cases, mainly with traumatic penetrating lung injury and barotrauma secondary to mechanical ventilation.

Pathogenesis. The basis of tension pneumothorax depends on the development of a condition of a uni-directional flow of air from injured pulmonary parenchyma into the pleural space in such a way that a "check-valve" mechanism results. Initially, the pleural pressure is positive only during expiration, but with increasing accumulation of air, the pressure becomes positive throughout the entire respiratory cycle. Circulatory collapse may follow, mainly as a result of the reduction in oxygen delivery due to severe hypoxemia secondary to the development of a large shunt in the involved collapsed lung. Reduction in cardiac output and systemic blood pressure will follow later.

Radiological evaluation of pneumothorax. Chest x-ray is a helpful tool in making the diagnosis of pneumothorax. Demonstration of the thin (<1 mm thick) visceral pleural line displaced away from the chest wall is confirmatory. An expiratory film is more sensitive in picking up a small pneumothorax by compressing the lung and increasing its density. It also increases the relative amount of air in the pleural space. Lateral decubitus film is another sensitive way of picking up a small pneumothorax. A pneumothorax must be differentiated from a large, thin-walled, air-containing bulla. In general, the pleural line with a pneumothorax is oriented in convex fashion toward the lateral chest wall, whereas the apparent pleural line with a large bulla

Table 1 - Etiology and incidence of iatrogenic and traumatic pneumothorax.

Procedure	Incidence
Iatrogenic	
Trans-thoracic needle aspiration	32
Pleural biopsy	10
Thoracentesis	12
Transbronchial biopsy	6
Mechanical ventilation	4
Traumatic	
Penetrating chest wall trauma	
Blunt chest wall trauma with rib fracture	

is usually concave. In case of doubt, computed tomography (CT) scan of the chest can easily differentiate the 2 conditions. Radiological evaluation of the patient with pneumothorax should include an estimate of the percentage of lung volume lost due to pneumothorax. For clinical purposes, an approximation of the % collapse can be made from the following equation:

$$\% \text{ Collapse} = [(D-d)/D \times 100]$$

When D and d are the average diameter of the chest wall and lung.

Another formula to estimate the % collapse can also be used:

$$\% \text{ Collapse} = 4.2 + [4.7 \times (A+B+C)]$$

(see A,B,C in **Figure 1**).

Recurrence rate. The risk of subsequent pneumothorax following an initial primary spontaneous pneumothorax is considerable. The recurrence rate ranges between 30%-50% after the first episode and 50%-80% after the second. The majority of the recurrences appear within 6-months to 2-years. The risk of recurrence is not affected by treatment modality (with the exception of chemical or surgical pleurodesis. Several studies have been performed concerning the possible relationship between the patient's characteristics, pulmonary diseases and the development of the recurrence. The patient's age, smoking and the presence of pulmonary fibrosis as detected by chest x-ray have been found to be independent risk factors for recurrence. No association could be demonstrated between recurrence rate and COPD. The presence of blebs and bullae has no predictive value for future recurrence. Bilateral presence of blebs and bullae in patients with unilateral primary spontaneous pneumothorax has been documented by both CT scan and surgical exploration. Blebs and bullae were found in 89% of patients on ipsilateral side and up to 80% in both sides. In a controlled study, blebs and bullae have been found in 20% of healthy volunteers as well. So investigation, in order to diagnose blebs and bullae, should not influence the choice of treatment modality to prevent recurrence.
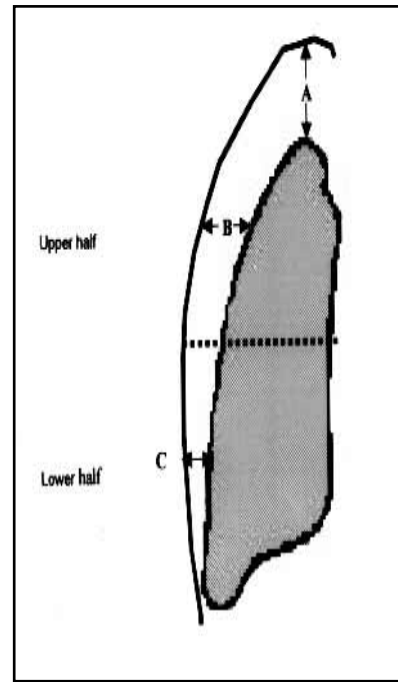

Figure 1 - Quantification of pneumothorax size on chest radiograph using intra-pleural distances at 3 locations: **(a)** the apex of collapsed lung to cupola, **(b)** the midpoint of the upper half of collapsed lung to chest wall, **(c)** the midpoint of the lower half of collapsed lung to chest wall.

Management of pneumothorax. The goals of the management of pneumothorax are simply to: 1. Remove the air from the pleural space to allow re-expansion of the collapsed lung. 2. Reduce the likelihood of recurrence.

General physiology. Air in the pleural space Nitrogen (N_2), Oxygen (O_2), Carbon Dioxide (CO_2) and water H_2O) will slowly equilibrate with tissue gases. Oxygen and CO_2 will be absorbed and the remaining N_2 will have a small pressure gradient between pleural gas and tissue gas, mainly capillary blood. Nitrogen in the pleural space will then be absorbed slowly (secondary to pressure gradient) at a rate of approximately 1.25% per 24 hours, and so lung expansion will take place gradually. This means that a 30% pneumothorax will need 24-days to resolve spontaneously.

Treatment options. A simple observation is a reasonable option when the following criteria are met: 1. Small primary spontaneous pneumothorax (<15%-20%). 2. Healthy and asymptomatic patient. 3. No evidence of progression of the pneumothorax.

Oxygen supplementation. A high concentration of inspired oxygen (FIO_2) will simply widen the nitrogen gradient between the pleural space and the capillary blood thus allowing the rapid absorption of the pleural gas. This is achieved by getting a higher O_2 tension in the blood, leading to a reduction in the blood nitrogen tension, and so widening the nitrogen gradient between the blood and pleural space. Based on this simple

physics, pleural space air absorption can be increased by 4-6 fold. Oxygen administration should be considered for any patient admitted for observation and treatment of pneumothorax.

Catheter aspiration. Catheter aspiration should always be considered for the initial treatment if primary spontaneous pneumothorax (PSP) is exceeding 15-20% of the value of the hemi-thorax. It is successful in avoiding tube thoracostomy in 65% of such cases. If the catheter aspiration method is chosen, air should be aspirated using a 50 ml syringe attached to a stopcock, until a resistance is felt, which indicates lung expansion. If no resistance is felt after 4 L of air has been aspirated, a persistent leak is present and tube thoracostomy is indicated. Catheter aspiration does not reduce the incidence of recurrence of spontaneous pneumothorax.

Tube thoracostomy. Tube thoracostomy is indicated for patients with secondary spontaneous pneumothorax of more than 15%-20% of the hemi-thorax value. It is also indicated for patients with PSP who have severe shortness of breath or chest pain, are hypoxic or failed catheter aspiration. Finally, it is also indicated for traumatic pneumothorax including mechanical ventilation induced. Following placement, the thoracostomy tube is connected to a one-way valve or a chest drainage system, which has 2 basic components. A water seal chamber, which functions as a one-way valve, consists of a tube submersed under water to prevent air entry into the negative-pressured pleural cavity. When pleural pressure becomes positive and exceeds the height of the submersed tube (usually 2 cm), air can escape. The second component is the suction control chamber, which provides a controlled amount of suction. Generally speaking, suction should be reserved for patients in whom expansion does not take place within 24-hours. The tube is left in place until lung expansion is complete and the air leak is closed. At this point, the tube is clamped for 12-24 hours and the patient is observed for recurrence by chest x-ray. If the lung remains expanded, the tube is removed. Failure of tube thoracostomy is considered to be the inability to expand the lung fully or the presence of a persistent air leak after 7 days of tube placement (in the absence of positive pressure mechanical ventilation). The thoracostomy tube (without pleurodesis) does not reduce the incidence of recurrent spontaneous pneumothorax.

Chemical pleurodesis. Intrapleural administration of a chemical to produce adhesion of the visceral and parietal pleura is a therapeutic option in selected cases of pneumothorax. Properly applied, it can decrease the recurrence rate of spontaneous pneumothorax. Chemical pleurodesis, however, is not effective for the closure of broncho-pleural fistula. Tetracycline was considered as the sclerosing agent of choice due to its efficacy, ease of administration, low cost and lack of significant side effects. In a prospective randomized trial, tetracycline reduced the recurrence rate to 25% compared to the tube thoracostomy rate of 41%. In several other studies, tetracycline reduced the recurrence rate further to 9-16%. Different studies have documented a dose-response relationship. The proposed mechanism of action is the release of cytokines (IL 6-8 and tumor necrosis factor), which are markers of inflammatory response that attract neutrophils to the pleural space causing pleural symphysis. Tetracycline is no longer available for clinical practice and doxycycline has been used as an alternative. A number of other sclerosing agents have also been utilized including bleomycin, aerosolized talc and talc slurry. Aerosolized talc has the lowest reported recurrence rate (4-6%) and is highly efficacious in achieving pleurodesis in both persistent secondary spontaneous pneumothorax and recurrent primary spontaneous pneumothorax. Despite its efficacy, its application needs a thoracoscopic procedure under local or general anesthesia and special surgical skill. Talc slurry has recently gained much interest, and it might be proven to be the sclerosing agent of choice. Preliminary data indicates that talc slurry has a high success rate. In a randomized prospective trial of 96 patients, pleurodesis with talc slurry resulted in the lowest recurrence rate of 8%, compared to 13% with tetracycline and 31% with simple tube drainage. The major concern with talc slurry is the reported association with acute respiratory distress syndrome (ARDS). This complication is probably dose related and occurred when high doses (>10 g) of talc were used. Pain, which might be severe, is the most common side effect of chemical pleurodesis, and so liberal analgesia is usually indicated.

Thoracotomy. Thoracotomy is the most effective treatment modality for preventing recurrent pneumothorax, with a recurrence rate of 0.6-2%. The advantages of surgical management are controlling the air leak, decreasing the recurrence rate, and addressing any underlying lung pathology that may have contributed to the pneumothorax. Surgical pleurodesis can also be performed if indicated. The indications for thoracotomy and surgical management of pneumothorax are as following: 1. Large (>50% minute ventilation) air leak for over 24 hours with proper drainage. 2. Continued symptoms due to persistent air leak. 3. Recurrence of pneumothorax with failure or contraindication to chemical pleurodesis. 4. Pneumothorax accompanied by hemorrhage, loculated effusion or empyema. 5. Recurrent contralateral pneumothorax. 6. Simultaneous bilateral pneumothorax (single-side procedure). 7. Pneumothorax following previous pneumonectomy. 8. Treatment of initial episode in patients with high-risk occupation (for example, pilots). Recently, video-assisted thoracic surgery (VATS) has been introduced as a possible alternate to thoracotomy. A recurrence rate of 5% has been reported for VATS compared to <1% for thoracotomy. However, treatment with VATS resulted in a significantly shorter hospital stay and drainage duration, mainly in patients with PSP or post traumatic pneumothorax. Video-assisted thoracic surgery has also proved to be more cost effective compared to

conservative therapy either by observation or tube thoracostomy. Recent data has shown that, irrespective of the presence of blebs or bullae, drainage and hospitalization time are shorter and complications and recurrence are much lower in patients treated with VATS when compared to conservative therapy with either first time or recurrent spontaneous pneumothorax.

Complications. Re-expansion pulmonary edema is a rare, but potentially serious complication of the treatment of pneumothorax by pleural drainage. The major risk factors are the duration of collapse prior to the attempts of re-expansion (>3 days) and the rapidity of the re-expansion, especially if suction is applied. The pathogenesis of re-expansion pulmonary edema is not clear, but may be related to increased pulmonary capillary permeability secondary to the vascular damage following prolonged collapse, perhaps compounded by increased capillary blood flow due to re-expansion. Re-expansion pulmonary edema is usually self-limited and management is supportive. In high risk patients, staged lung expansion and avoidance of active suction, if possible, should be considered in order to minimize the risk of this complication.

Pneumothorax and air travel. The exact incidence of pneumothorax during commercial air travel is unknown, but seems to be exceedingly rare. In one series of 704 COPD patients referred for airline medical advisory service, none of them had experienced a significant medical in-flight problem.

Pathogenesis. The main factor that might predispose patients to pneumothorax during air travel is the expansion of the trapped air due to the reduction of barometric pressure according to Boyle's law. Non-communicating pulmonary blebs and bullae will enlarge as the air expands and could rupture. The Federal Aviation Administration requires that aircraft maintain a "cabin altitude" less than 8,000 feet at the highest operating altitude (usually 22,000-44,000 feet), which is achieved by pressurizing the cabin. Such pressurization will not only maintain an acceptable partial pressure of oxygen in the cabin, but it will also reduce the risk of spontaneous pneumothorax, as trapped gas within the lungs expands by only 38% at a cabin altitude of 8000 feet compared to 400% at 35,000 feet. Based on the current available data, air travel may be contraindicated in patients with severe bullous emphysema, large congenital pulmonary cysts or a prior spontaneous pneumothorax in the preceeding 30 days.

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Abstract

We present a retrospective study of 25 patients with spontaneous pneumothorax (3 recurrent), comprising 16 saudis (9 males and 7 females) and 9 non-saudis (8 males and one female), seen at the Asir Central Hospital, Abha, Kingdom of Saudi Arabia, over a period of 45 months. Almost one-third of the patients (9/25) had no underlying cause discernible by our investigational facilities (chest x-ray, ultrasonography, computed tomographic scan, and flexible bronchofiberscopy). Underlying pneumonia (3 patients), pulmonary tuberculosis (2 patients), lung abscess (one patient), and congenital bullae (one patient) constituted the etiology in another third of the spontaneous pneumothorax patients. Other underlying pulmonary diseases precipitating spontaneous pneumothorax in the group included pulmonary fibrosis, metastatic mesothelioma, and immunosuppression in a medulloblastoma patient undergoing chemotherapy with the development of chickenpox. Closed thoracostomy tube drainage was the only method of treatment in 20 out of the 25 patients, with 3 failures of closed thoracostomy tube drainage needing thoracotomy and resection of blebs/pullae. The only complication was empyema in 2 of the patients. Two patients were successfully treated conservatively with observation alone.