



An Overview about Management of Pneumothorax in ICU

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Abstract

The pleural space is considered a potential space defined by visceral pleural lining of the lung and parietal pleural lining of the chest wall, diaphragm, and mediastinum. Under normal conditions, the pleural space in the human is a sealed cavity that contains a small volume of fluid estimated to be 0.26 mL/kg body mass, or 15 to 20 mL. The prevalence of pneumothorax in ICU patients requiring mechanical ventilation ranges from 4% to 15%, and incidence in the 1990s was up to 24%, but more recent data show that the incidence has come down to about 3%. This is likely from a change in medical management of ICU patients over time. Thoracic ultrasound use has been rapidly expanding and becoming an essential part of ICU care and emergency medicine. Point-of-care ultrasound is easy to use, readily available in most ICUs, efficient, reliable, and cost effective, with the added advantage of real time imaging. It also has the advantage of safety, as it does not expose the patient to radiation like a chest X-ray or CT scan. Since these exams can be done at bedside, they avoid the risks associated with transporting a critically ill patient. Thoracic ultrasound has also been noted to be helpful in detecting occult pneumothorax. It is shown that comparing ultrasonography to CT scan and chest radiography in diagnosing occult pneumothorax showed 92% diagnostic yield with thoracic ultrasound when compared to 52% diagnostic yield with chest radiograph and when compared with CT in which all the patients were noted to have pneumothorax. Tension pneumothorax is a medical emergency in which there is expanding air volume within the chest cavity with clinical signs and symptoms of progressive hypoxemia, tachycardia, respiratory distress and hypotension, and requires rapid decompression of air from the chest cavity. Presentation can be more pronounced, and progression can be rapid in mechanically ventilated patients when compared to those breathing unassisted. Hence, it is important to make a timely diagnosis of tension pneumothorax and intervene immediately. In critically ill patients, the diagnosis often needs to be made clinically, as there is not enough time to get a chest radiograph for confirmation. Chest ultrasound can be helpful if readily available, but intervention should not be delayed if there is high clinical suspicion for tension pneumothorax.

Keywords: Pneumothorax, ICU

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Introduction;

The pleural space is considered a potential space defined by visceral pleural lining of the lung and parietal pleural lining of the chest wall, diaphragm, and mediastinum. Under normal conditions, the pleural space in the human is a sealed cavity that contains a small volume of fluid estimated to be 0.26 mL/kg body mass, or 15 to 20 mL (1).

It is not clear why humans have a pleural space, because other species, such as elephants, do quite well without a pleural space, as do humans following pleurodesis. Postulated reasons for having a pleural space

containing a small amount of liquid include allowing a reduced surface tension and friction for efficient lung expansion. The elasticity of both the chest wall and the lung parenchyma in opposing directions results in a slightly negative pressure within the pleural space at functional residual capacity. Despite the negative pleural pressure, air does not normally enter the pleural space because the intact visceral pleura and chest wall prevent air access (1).

The prevalence of pneumothorax in ICU patients requiring mechanical ventilation ranges from 4% to 15%, and incidence in the 1990s was up to 24% in USA, but more recent data show that the incidence has come down to about 3%. This is likely from a change in medical management of ICU patients over time (2).

Iatrogenic pneumothorax occurs as a result of diagnostic and therapeutic intervention and is the most common cause of pneumothorax in the ICU setting (3).

Despite that it remains one of the most serious complications of positive pressure ventilation. Fortunately, if identified in a timely fashion, pneumothorax in the ICU can be treated effectively with pleural drainage, minimizing both acute and long-term adverse sequelae (4).

Pneumothorax can be misdiagnosed at initial presentation in about 32% of patients admitted to the ICU. It has a mortality rate of 68%, with the worst prognosis being in patients with septic shock and barotrauma (5).

Etiology and risk factors

Air can enter the pleural space by three routes: (I) Communication between alveoli and pleura (II) communication (direct or indirect) between atmosphere and pleural space, and (III) presence of gas producing organisms in the pleural space (6).

Risk factors for primary spontaneous pneumothorax: Smoking, Tall thin body habitus in an otherwise healthy person, Pregnancy, Marfan syndrome and Familial pneumothorax. (7)

While there are some diseases associated with secondary spontaneous pneumothorax

As: COPD, asthma, HIV with pneumocystis pneumonia, necrotizing pneumonia, tuberculosis, sarcoidosis, cystic fibrosis, bronchogenic carcinoma, idiopathic pulmonary fibrosis, severe ARDS, Langerhans cell histiocytosis, collagen vascular disease, inhalational drug use like cocaine or marijuana, thoracic endometriosis (8).

Causes of iatrogenic pneumothorax: pleural biopsy, transbronchial lung biopsy, transthoracic pulmonary nodule biopsy, central venous catheter insertion, tracheostomy, intercostal nerve block and positive pressure ventilation (9)

Causes of traumatic pneumothorax: penetrating or blunt trauma, rib fracture and diving or flying (9)

Causes of tension pneumothorax: penetrating or blunt trauma, barotrauma due to positive pressure ventilation, percutaneous tracheostomy, Conversion of spontaneous pneumothorax to tension and Open pneumothorax when occlusive dressing work as one way valve (9)

Causes of pneumomediastinum: asthma, parturition, emesis, severe cough and traumatic disruption of oropharyngeal or esophageal mucosa. (7)

Pneumothorax in the ICU occurs mostly as a result of barotrauma as well as various invasive procedures. *It is* shown that about 55% of the pneumothoraces in the ICU were from invasive procedures and the rest from barotrauma (5).

Use of excessive airway pressure (barotrauma) and/or tidal volume (volutrauma) are important etiological factors in the development of pneumothoraces in mechanically ventilated patients, particularly in those with severe underlying lung disease such as acute respiratory distress syndrome (ARDS). Other pulmonary diseases associated with an increased risk of pneumothorax include chronic obstructive pulmonary disease, bullous emphysema, *Pneumocystis* pneumonia (PCP), cystic fibrosis and tuberculosis. In these patients multiple, complex and repeated air leaks are often seen. Weight <80 kg, acquired immunodeficiency syndrome (AIDS) and cardiogenic pulmonary oedema at the time of admission have been identified as additional risk factors in a prospective cohort study (4).

Invasive procedures that commonly cause pneumothorax in the ICU include thoracentesis, central venous line insertion, and bronchoscopy with biopsy (10).

Papazian *et al.* demonstrate significant decrease in pneumothorax occurrence in patients with severe ARDS who were started on muscle relaxants within 48h of disease onset (11)

Implementation of lung protective ventilation has resulted in a significant decrease in pneumothorax incidence when compared to the pre lung protective ventilation era (5).

Traumatic pneumothorax is also seen in ICUs with trauma patients. Pneumothorax can occur in about 35% of patients with thoracic injuries, and of these about 22% of the pneumothoraces can be occult (12).

Traumatic pneumothorax should be suspected and sought in cases of direct chest wall trauma, pulmonary contusion and diaphragmatic injury. In this situation the pneumothorax is said to be 'open' if it communicates with the exterior. There are few data regarding prognostic factors and outcomes in critically ill patients, although pneumothorax associated with barotrauma, tension pneumothorax and concurrent sepsis is reported to carry a particularly bleak outlook (12).

Common causes for pneumothorax in the ICU are listed in Table 1 (3).

Common causes for pneumothorax in the ICU

(I) Underlying lung disease:

- ARDS
- Pneumonia
- COPD/emphysema

(II) Trauma

(III) Invasive interventions

- Positive pressure ventilation
- Central venous line insertion
- Bronchoscopy
- Surgical procedures involving thorax, head and neck
- Abdominal procedures

(I) Underlying parenchymal lung disease predispose patients to develop pneumothorax. (II) Post trauma patients are at risk for developing pneumothorax. (III) Iatrogenic pneumothorax can be caused due to various invasive diagnostic and therapeutic interventions.

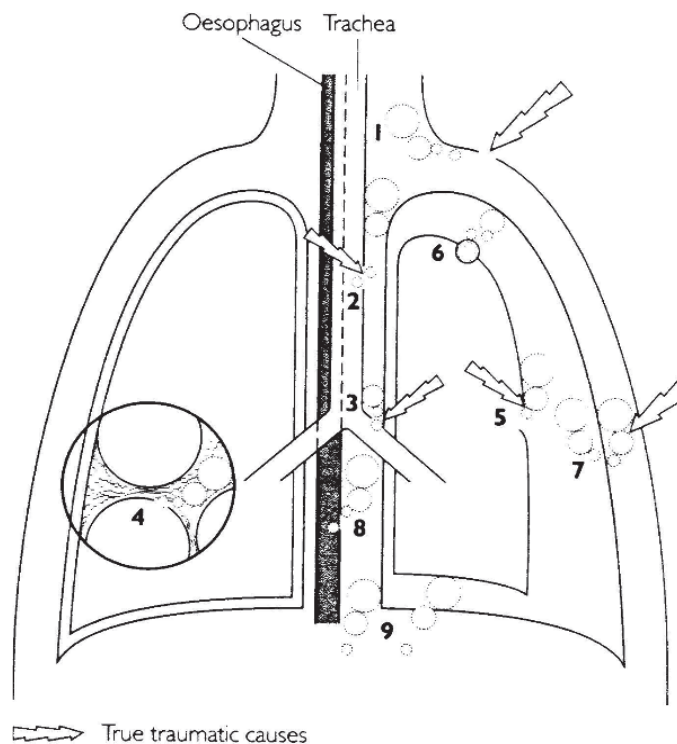


Figure 9 Causes of air penetration into the pleura: (3)

1. Soft tissue injury to the subclavian region
2. Trauma to the trachea
3. Trauma to the bronchus
4. Alveolar rupture
5. Rupture of the visceral pleura
6. Rupture of preformed bullae or blebs
7. Trauma to the external chest wall and parietal pleura
8. Rupture of the oesophagus
9. Entry of air from the abdomen

Epidemiology

Primary spontaneous pneumothorax mainly occurs at 20-30 years of age. The incidence of PSP in the United States is 7 per 100,000 men and 1 per 100,000 women per year. Most recurrence occurs within the first year, and incidence ranges widely from 25% to 50%. The recurrence rate is highest over the first 30 days. Secondary spontaneous pneumothorax is seen more in old-age patients 60-65 years. The incidence of SSP is 6.3 and 2 cases for men and women per 100,000 patients, respectively. The male-to-female ratio is 3:1. COPD has an incidence of 26 pneumothoraces per 100,000 patients. **(13)**

The risk of spontaneous pneumothorax in heavy smokers is 102 times higher than in non-smokers. **(9)**

The leading cause of iatrogenic pneumothorax is transthoracic needle aspiration (usually for biopsies), and the second leading cause is central venous catheterization. These occur more frequently than spontaneous pneumothorax, and their number increases as intensive care modalities advance. The incidence of iatrogenic pneumothorax is 5 per 10,000 admissions in the hospital. **(13)**

The incidence of tension pneumothorax is challenging to determine as one-third of cases in trauma centers have decompressive needle thoracostomies before reaching the hospital, and not all of these had tension pneumothorax. Pneumomediastinum has an incidence of 1 case per 10,000 admissions in the hospital. **(13)**

Pathophysiology

In many severe lung diseases, such as ARDS, there is direct tissue damage due to the host inflammatory response and/or toxins produced by the causative organism, e.g. Pneumocystis. Infarction can also occur as a result of local vascular compromise. Subpleural cysts or cavities form, which may eventually leak air. If this leak is significant, air will accumulate in the pleural space, usually towards the apex in a spontaneously breathing erect patient, due to the higher negative pressure in the pleural cavity (pleural pressure gradient increases from base to apex). This may remain a small localised collection or, depending on disease progression and airway pressure, increase in size resulting in loss of lung volume and impaired ventilation/perfusion matching. As the pressure in the affected hemithorax continues to rise the mediastinal structures, including major blood vessels, become compressed and shift towards the opposite side of the chest. Venous return is reduced and cardiac afterload increases. Cardiac output is usually severely reduced under these circumstances— known as a tension pneumothorax— and if the pleural air collection is not decompressed, it may promptly culminate in a cardiac arrest. Although ventilated patients with air leaks are at constant risk of this dangerous sequence of events, in reality, tension pneumothorax is uncommon. Most patients with air leaks develop localized collections which resolve slowly in patients with underlying pathology, after drainage **(4)**.

Pneumothorax ex vacuo is a rare phenomenon, which occurs at the site of atelectatic obstructed lung or fixed trapped lung following drainage of an effusion. The rapid drainage of fluid combined with the inability of the lung parenchyma to re-expand leads to the generation of a negative intrapleural pressure which encourages the passage of nitrogen into the space from the local microcirculation leading to a collection **(14)**.

Diagnosis of pneumothorax

A. Clinical Diagnosis (respiratory disease and its management e-book, 2009)

The classical findings on clinical examination in patients with a pneumothorax are: Decreased chest movement on the affected side, Hyper resonance on percussion of the affected side, Decreased or absent breath sounds on the affected side, Hyperinflation of the affected side and Tracheal deviation away from the affected side. **(14)**.

Given the limitations of clinical examination in critically ill ventilated patients, clinicians must also be alert to other signs that may indicate the development of a pneumothorax: Worsening gas exchange or increased oxygen requirements, A need for increased ventilatory support, raised peak airway pressures or abnormal respiratory pattern, Cardiovascular changes such as tachycardia, hypotension or rising central venous pressure and Evidence of decreased end-organ perfusion, e.g. metabolic acidosis (elevated lactate), oliguria **(14)**.

Misdiagnosis of pneumothorax is particularly common in critically ill patients who have an altered mental state, are receiving sedation, present out of hours or have atypical radiological findings **(4)**.

Timely diagnosis of pneumothorax in critically ill patients is paramount given the high mortality risk **(15)**.

Diagnosis of pneumothorax starts with history and physical examination. In a critically ill patient, change in respiratory status with new or worsening hypoxia, tachycardia, hypotension, decreased breath sounds on the affected side, tracheal deviation away from the affected side, surgical emphysema, decrease in tidal volume with pressure-controlled ventilation or increase in airway pressures (peak and plateau), with volume controlled ventilation may be a sign of pneumothorax. However, these signs are nonspecific and can be related to other disease conditions. For example, airway pressures may be elevated from obstruction of the endotracheal tube and diminished breath sounds may be due to underlying lung disease such as COPD or from atelectasis (3).

As such, clinicians should maintain a high index of suspicion and given the variability of these signs and symptoms, a chest X-ray is usually needed to confirm the diagnosis (16).

However, respiratory and hemodynamic signs of tension pneumothorax should be considered a medical emergency and addressed immediately, as this requires urgent needle decompression (3).

B. Imaging

Thoracic ultrasonography

Ultrasonography in diagnosis of pneumothorax was first described in a horse by Rantanen in 1986. Thoracic ultrasound use has been rapidly expanding and becoming an essential part of ICU care and emergency medicine (17).

Point-of-care ultrasound is easy to use, readily available in most ICUs, efficient, reliable, and cost effective, with the added advantage of real time imaging. It also has the advantage of safety, as it does not expose the patient to radiation like a chest X-ray or CT scan. Since these exams can be done at bedside, they avoid the risks associated with transporting a critically ill patient (18).

On thoracic ultrasound, pleura appears as a highly echogenic band which moves with respiration and is referred to as the gliding sign or lung sliding (10). This means that the presence of lung sliding can rule out pneumothorax at the area being examined (10).

An alternative mode named the M-Mode (motion mode) has also been used as an adjunct to detect pneumothorax. There are two different types of images obtained: the “stratosphere sign” or “barcode sign”, which represents absent pleural motion, and the “sandy beach” or “seashore sign” which represents pleural motion. Use of M mode along with conventional B mode has been shown to increase the diagnostic yield of lung sliding, especially in early users (19).

It is shown that the concept of “lung point” sign where the pneumothorax pattern noted on a sonogram is replaced by the fleeting appearance of lung pattern. This has an overall sensitivity of 66% with increased sensitivity (75%) in occult pneumothorax and has a specificity of 100% (20).

Another helpful sign on thoracic ultrasonography is the “comet-tail artifacts,” also known as B Lines, which are vertical ultrasound artifacts arising from the lung-wall interface that rule out a complete pneumothorax (20).

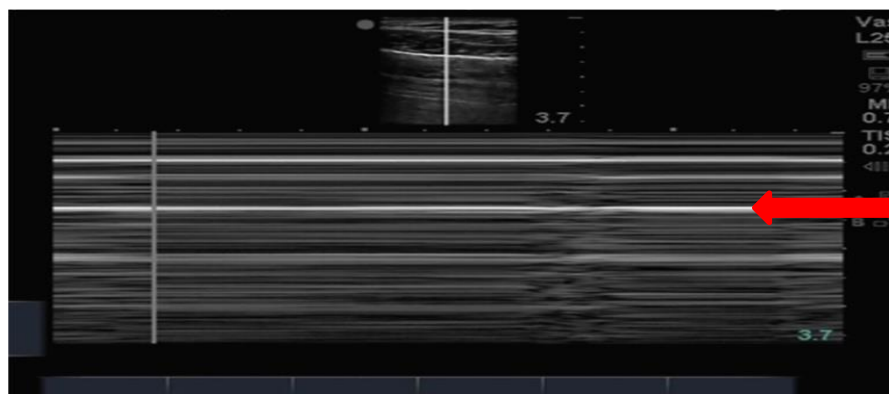


Figure 1: Barcode sign. M-Mode image demonstrates a linear and laminar pattern above the bright pleural line (arrow) corresponding to superficial tissue and an identical pattern noted below the pleural line. This sign known as “barcode sign” or “stratosphere sign” indicates absence of lung sliding and possibility of pneumothorax at this point of exam. Note that the presence of this sign is suggestive of but not confirmatory for pneumothorax (3).

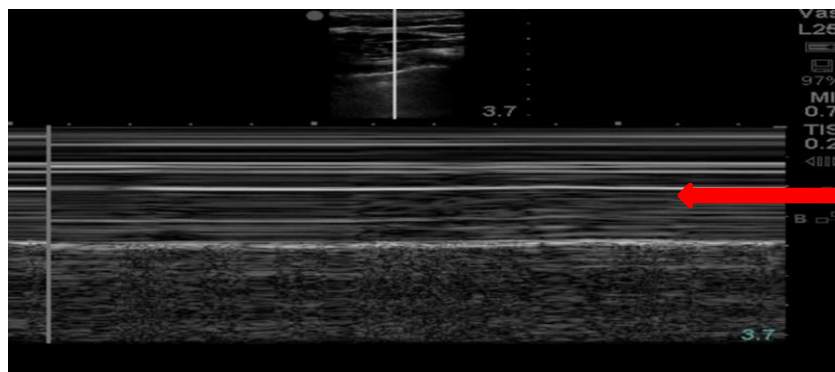


Figure 2: Sea shore Sign. M-Mode image shows a bright pleural line (arrow) with a linear and laminar pattern above the line corresponding to superficial tissue and a granular pattern seen below the pleural line. This sign known as “Sea Shore” sign indicates normal lung sliding and rules out pneumothorax (3).

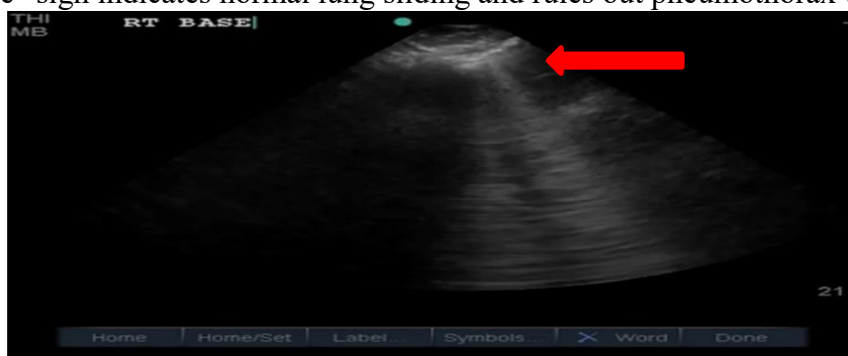


Figure 3: Comet Tail Sign Comet tail sign or artifacts also known as B-lines are hyperechoic reverberations originating from the visceral pleura and travel almost perpendicular to the pleural line. These are lost when air accumulates in the pleural space which prevents propagation of ultrasound waves. Presence of comet-tail sign rules out pneumothorax (3).

The absence of lung sliding is suggestive of pneumothorax, but it is not specific for diagnosis. This is because it can be seen in other conditions such as atelectasis, especially related to selective intubation, conditions that causes adherence of pleura to the chest wall, jet ventilation, esophageal intubations and apnea. “Lung pulse” is described as the perception of heart activity at the pleural line, which, along with absent pleural sliding, represents atelectasis (20).

Thoracic ultrasound has also been noted to be helpful in detecting occult pneumothorax. It is shown that comparing ultrasonography to CT scan and chest radiography in diagnosing occult pneumothorax showed 92% diagnostic yield with thoracic ultrasound when compared to 52% diagnostic yield with chest radiograph and when compared with CT in which all the patients were noted to have pneumothorax. The learning curve for chest ultrasound is relatively short, and it can be a useful tool in the ICU to look for pneumothorax after central venous line placement (21).

Chest radiograph

Critically ill patients are often supine or semi-recumbent. This poses a challenge to interpreting chest radiography. It is found that in critically ill patients noted that 30% of the pneumothoraxes were initially missed on chest radiograph. This was mostly due to the involvement of anteromedial and subpulmonic pleural recess in about 64% of the patients (3).

On a supine radiograph, pneumothorax may be seen as air present in the deep lateral costophrenic angle known as the deep sulcus sign. Expiratory chest radiographs were assumed to enhance the diagnostic yield of small apical pneumothoraces. However, it was found to have no increase in diagnostic yield and is not recommended based on current guidelines (22).

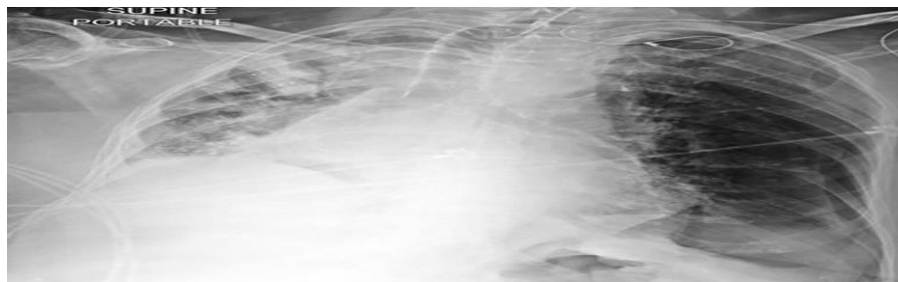


Figure 4: Deep sulcus sign on left side. Deep sulcus sign noted on this chest X-ray shows a deep radiolucent area at the left lateral costophrenic angle (arrow) (6).

Select situations, such as presence of skin folds, emphysematous bullae, and visceral gas within the chest may mimic a pneumothorax on a chest radiograph. If chest X-ray findings do not correlate clinically, then a CT may be considered prior to acting on the radiograph findings (3).

In critically ill patients with abnormal lung disease from pneumonia, ARDS or interstitial lung disease, diagnosis of pneumothorax on routine chest radiograph may be challenging. In these conditions, the lung may not collapse because of parenchymal stiffness or pleural adhesions. This leads to development of small loculated pneumothoraces, which can later develop into tension pneumothorax (3).

Computed tomography

Patients with respiratory failure in the ICU, especially in the medical ICU, often have underlying lung disease that makes it difficult for a diagnosis of pneumothorax to be made based on a chest radiograph. Chest X-rays in patients with complex cystic lung disease may be difficult to interpret and can mask pneumothorax. These conditions can also cause lung adherence to the chest wall, resulting in loculated pneumothorax, which can be difficult to detect on chest radiograph (4).

As previously mentioned, pneumothorax can be localized in the pleural recesses, causing it to be missed initially in about 30% of the patients on initial chest radiograph with a risk, as it may progress to tension pneumothorax. CT scan has been shown to be superior to chest radiograph in diagnosing and sizing of pneumothorax; hence, it is considered the gold standard test for evaluating pneumothorax (23).

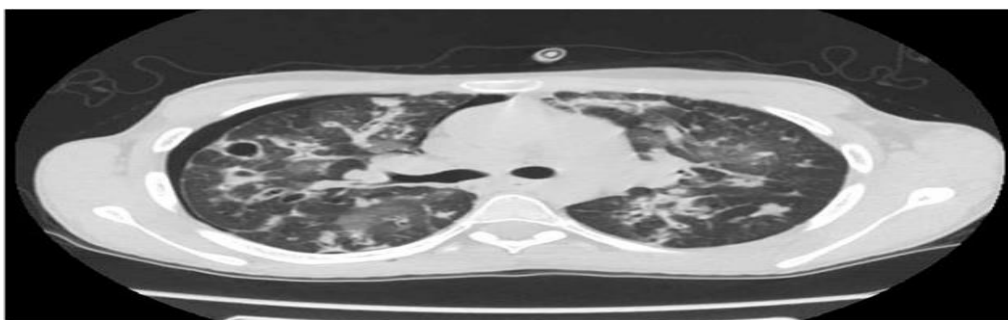


Figure 5: CT scan of a right pneumothorax in a patient with cystic fibrosis (6).

The use of CT scan has led to a better understanding of thoracic anatomy. This has also led to an increase in diagnosis of pneumothoraxes, which were initially not recognized clinically or on chest radiographs. These are called occult pneumothoraxes. The incidence of occult pneumothorax ranges between 1.8–4% (24).

In patients with occult pneumothorax, especially in patients receiving mechanical ventilation, there is a concern for worsening of pneumothorax and potential development of tension pneumothorax. In the past it is shown that conservative management to be a safer option in patients with occult pneumothoraxes (25).

In a prospective multicenter randomized controlled trial, Kirkpatrick *et al.* demonstrated that occult pneumothorax can be safely observed in patients receiving mechanical ventilation. However, those requiring mechanical ventilation for more than a week had an increased requirement of pleural drainage tube. In the

pleural drainage group, 15% experienced complications related to the procedure. The possibility of occult pneumothorax should be kept in mind for patients with a history of blunt trauma (26).

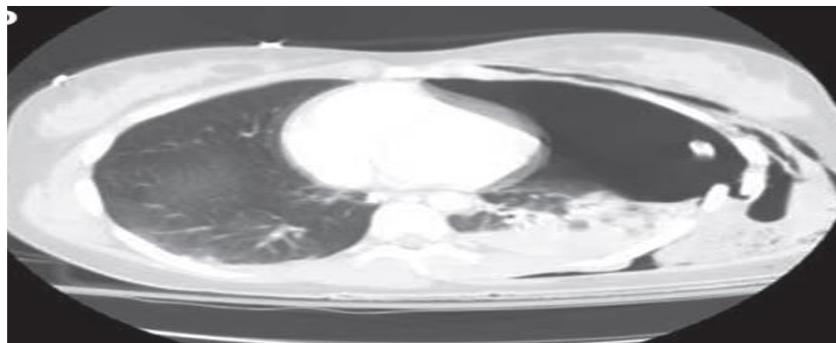


Figure 6: CT scan reveals a large occult left-sided pneumothorax (4).

Differential Diagnosis

Differential diagnoses of pneumothorax include: (16) Aspiration, bacterial or viral pneumonia, Acute aortic dissection, myocardial infarction, pulmonary embolism, acute pericarditis, esophageal spasm, esophageal rupture, rib fracture and diaphragmatic injuries.

Prognosis

Primary spontaneous pneumothorax (PSP) is usually benign and mostly resolves independently without any significant intervention. Recurrence can occur for up to three years period. The recurrence rate in the following five years is 30% for primary spontaneous pneumothorax and 43% for secondary spontaneous pneumothorax. The risk of recurrence increases with each subsequent pneumothorax; it is 30% with the first, 40% after a second, and more than 50% after the third recurrence. PSP is not considered a significant health threat, but deaths have been reported. SSPs are more lethal depending on underlying lung disease and the size of the pneumothorax. Patients with COPD and HIV have high mortality after pneumothorax. The mortality of SSP is 10%. Mortality of tension pneumothorax is high if appropriate measures are not taken. (27).

- **Complications:** Respiratory failure or arrest, Cardiac arrest, pyopneumothorax, empyema, rexpansion pulmonary edema, pneumopericardium, pneumoperitoneum, pneumohemothorax, bronchopulmonary fistula, damage to the neurovascular bundle during tube thoracostomy, pain and skin infection at the site of tube thoracostomy. (27)

Management of pneumothorax

Management of pneumothorax in critically ill patients depends on the cause as well as the underlying clinical condition. The rate of resolution of spontaneous pneumothorax is around 1.25–2.2% per 24 hours if it is managed conservatively without pleural drainage. However, ICU patients often have poor pulmonary reserve, and even a small pneumothorax can cause a significant cardiopulmonary effect (27).

Pneumothorax secondary to barotrauma, tension pneumothorax and concurrent septic shock are all significant independent risk factors for mortality in critically ill patients (3).

British Thoracic Society (BTS) guidelines recommend chest drain insertion for any patients on mechanical ventilation, with tension pneumothorax after initial needle decompression, traumatic hemopneumothorax and post-surgical pneumothorax. Hence, many of the critically ill patients will end up needing chest drainage tube either because of the underlying cause or because of their poor reserve requiring the pneumothorax to be drained even if it is small. There are various management options available based on the severity of symptoms, size of pneumothorax and persistence of bronchopleural fistula (28).

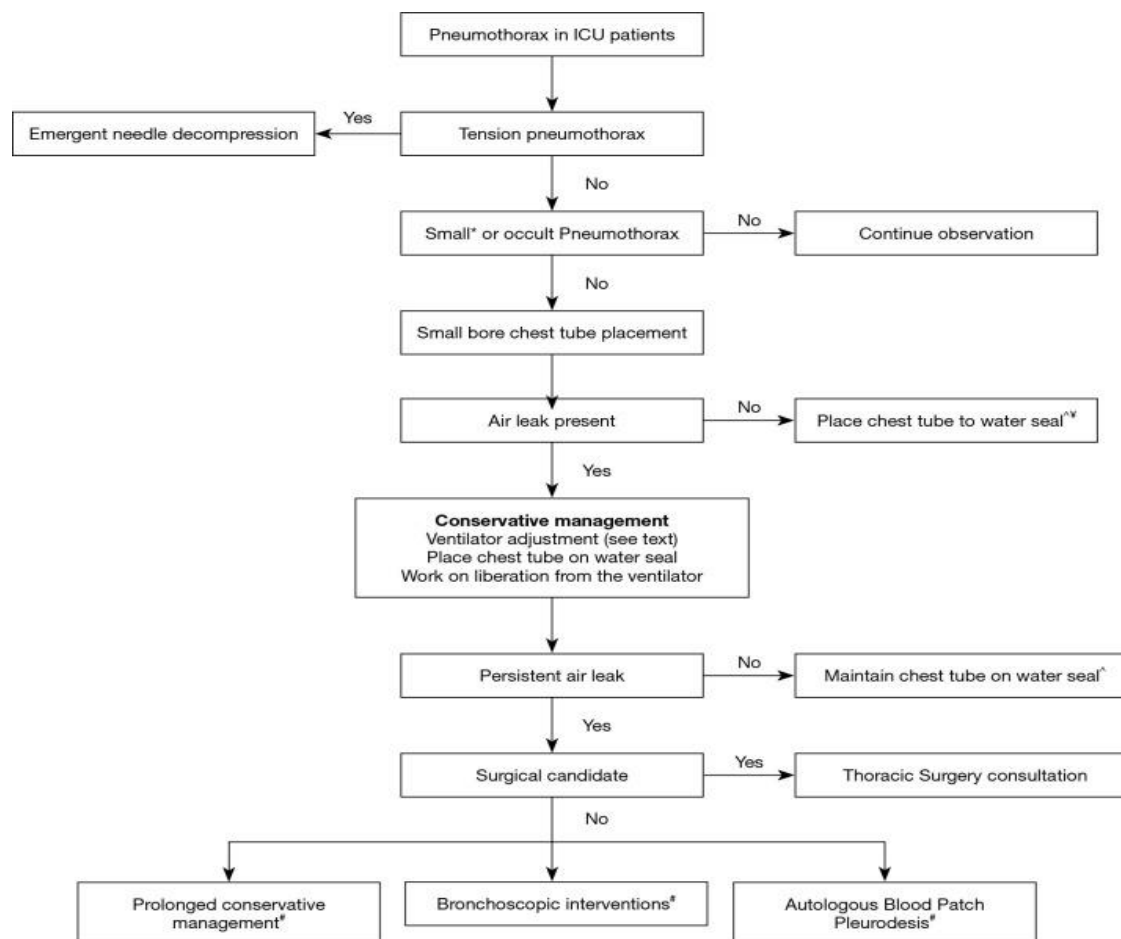


Figure 7: Algorithm for management of pneumothorax in ICU. *, based on the guidelines followed; ^, decision on when to remove the chest tube should be made by the treating physician; ¥, if lung has re-expanded; #, any of the three non-surgical interventions can be performed based on the clinical condition (3).

Emergent needle decompression

Tension pneumothorax is a medical emergency in which there is expanding air volume within the chest cavity with clinical signs and symptoms of progressive hypoxemia, tachycardia, respiratory distress and hypotension, and requires rapid decompression of air from the chest cavity. Presentation can be more pronounced, and progression can be rapid in mechanically ventilated patients when compared to those breathing unassisted (29).

Hence, it is important to make a timely diagnosis of tension pneumothorax and intervene immediately. In critically ill patients, the diagnosis often needs to be made clinically, as there is not enough time to get a chest radiograph for confirmation. Chest ultrasound can be helpful if readily available, but intervention should not be delayed if there is high clinical suspicion for tension pneumothorax (6).

Tension pneumothorax should be suspected when respiratory or cardiovascular decompensation is noted in patients who received cardiopulmonary resuscitation (CPR) or have a chest tube that is blocked (6).

Needle decompression is done in the second or third intercostal space (ICS) in the mid-clavicular line using a standard 14 gauge (4.5 cm) cannula until an audible rush of air is appreciated. However, in up to 35% of patients, a standard 4.5 cm canula may not be long enough to penetrate the chest wall, leading to treatment failure. This can be avoided by using a trocar (7 cm) or use of 4th or 5th ICS in the mid-axillary line, as it contains less fat and muscle mass. However, this area does carry the risk of lung injury in supine patients (30).

A chest drain placement should be performed after canula decompression, and the canula should be left in place until air bubbles are noted in the water seal to confirm correct placement of the chest drain (22).

Chest drain placement

There are a wide variety of chest tubes available, both small-bore (10–14 Fr) and large bore (>20 Fr). In the past, large bore chest tubes were placed for treatment of pneumothorax. However, in the last decade or so, small bore chest tubes, which are placed using the modified Seldinger technique, have become more common in managing pneumothorax. These small-bore chest tubes are easier to place and cause less pain at the insertion site. Small-bore chest tubes are associated with lower complication rates, with injury occurring in 0.2% and malposition occurring in 0.6%, as compared to large-bore chest tubes, where the injury occurs in about 1.4% and malposition occurs in about 6.5%. The current BTS guidelines recommend the use of small-bore chest tube as first line therapy for management of pneumothorax (28).

In their retrospective review, Liu *et al.* demonstrated no significant difference between the evacuation rates of large bore and small-bore chest tubes (31).

Persistent air leak

If the air leak lasts more than 5–7 days, then it is called persistent air leak. In the era prior to lung protective ventilation, there was significantly high incidence of persistent air leak in patients who developed pneumothorax while on a mechanical ventilator (3).

The overall incidence of pneumothorax has decreased with implementation of a lung protective ventilation strategy. However, patients with underlying lung disease are at risk for developing persistent air leak (32).

Initial management is often conservative, and it can be effective if enough time is given for the alveolar-pleural or broncho-pleural fistula to close. The 2001 American College of Chest Physicians consensus statement on management of spontaneous pneumothorax recommends thoracic surgery consultation if the air leak persists after four days (33).

The BTS guidelines recommend thoracic surgery consultation if the air leak persists for 3–5 days. In patients who are not a surgical candidate, non-surgical methods such as bronchoscopic interventions as well as blood patch may be considered. However, guidelines are lacking for non-surgical management of persistent air leak (32).

Conservative management

Persistent air leak can be challenging to manage when the patient is receiving mechanical ventilation. In such situations, the strategy should focus on lowering the peak airway pressure by reducing the tidal volume and end-expiratory pressure as well as use of pressure-cycled ventilation. Placing the chest tube on water seal from suction has also shown to help stop the air leak (34).

Thoracic surgery consult

Surgical pleurodesis remains the most effective method for treating recurrent or persistent pneumothorax. Open thoracotomy with partial pleurectomy remains the best modality, and pleurodesis with Video Assisted Thoracoscopic Surgery (VATS) also has comparable results. These results are based on patients undergoing surgical pleurodesis for spontaneous or secondary spontaneous pneumothorax (35).

The American College of Chest Physicians consensus statement recommends pleurodesis with VATS. Critically ill patients may not be a surgical candidate, and there is little data or guidelines to support further clinical decision making in this patient population. If the patient is not a surgical candidate, then prolonged conservative management can be tried, such as placing the chest tube to water seal as well as adjusting the ventilator settings as mentioned earlier (35).

If conservative management fails in these patients, then other available options are either bronchoscopic management or autologous blood patch pleurodesis and are discussed below (3).

Bronchoscopic management of persistent air leak

Various bronchoscopic methods have been tried in patients with persistent air leak with variable success, and all of them have been anecdotal. These include fibrin sealants, metal coils, Watanabe spigots, and alcohol sclerosis, all reported as case reports and series (32).

At present, Spiration intra bronchial valve (IBV) system is the only device approved by the FDA for bronchoscopic treatment of persistent air leak. These are one-way valves placed with a flexible bronchoscope into the segmental or subsegmental airways. This limits airflow into the distal airways while allowing

secretions and air to move proximally. There are two endobronchial valves (EBV) available in the market: Zephyr valve by PulmonX Inc. and the Spiration Valve System by Olympus (32).

In 2009, Travaline *et al.* reported the use of EBV in 40 patients with persistent air leak, with complete resolution in 47.5% and reduction in air leak in 45%. The mean time for chest tube removal was 21 days. Of the forty patients, only six patients experienced adverse events such as valve expectoration, moderate oxygen desaturation, valve mispositioning requiring redeployment and methicillin-resistant staphylococcus aureus colonization (36).

Gillespie *et al.* published their experience with valves in eight patients, where 57% of patients were discharged within 2–3 days (37).

In their case series, Mahajan *et al.* reported the safety and effectiveness of IBV in ICU patients with prolonged air leak requiring high levels of supplemental oxygen or mechanical ventilation (38).

Reed *et al.* reported the used of EBV in post-surgical patients as well as in patients with cavitory lung disease. Their series noted the successful removal of chest tube in all patients who survived the clinical course (38).



Figure 8: Chest radiograph showing placement of an endobronchial valve in the right upper lobe with resolution of pneumothorax prior to pigtail catheter removal (6).

Autologous blood patch pleurodesis

Autologous Blood Patch Pleurodesis (ABPP) was first reported by Robinson in 1987, when 25 patients with fully expanded lungs but prolonged air leak underwent instillation of 50 mL of autologous blood anywhere between one to three times. The procedure was successful in 85% of the patients (39).

ABPP is performed by injecting 50–100 mL autologous blood through the chest tube followed by flushing the chest tube with 10 mL of saline and clamping for about 30 min, followed by placing it back on the water seal. If the chest tube cannot be clamped, it can be hooked over a drip stand for the air to escape in order to prevent tension physiology (39).

A review of ten published studies showed blood pleurodesis to be superior to conservative management with optimal volume of 100 mL. Patients undergoing blood pleurodesis were noted to have an overall success rate of 92.7%, with a shorter time to achieve pleurodesis. The reported complications are rare but may include fever, empyema, pleuritis and blood clot blocking the chest tube causing tension physiology (40).

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