

PNEUMOTHORAX — DIAGNOSIS AND TREATMENT

Milislavljec Slobodan,^{1,2} Spasic Marko,¹ Milosevic Bojan¹

¹ General and Thoracic Surgery Clinic, Clinical Centre Kragujevac, Serbia

² Faculty of Medical Sciences University of Kragujevac, Serbia

Primljen/Received 20. 09. 2015. god.

Prihvaćen/Accepted 10. 11. 2015. god.

Abstract: Introduction: Pneumothorax is defined as the presence of air in the pleural cavity, ie, the space between the chest wall and the lung itself. Pneumothorax is classified etiologically into spontaneous pneumothorax and traumatic pneumothorax. Spontaneous pneumothorax is further classified into primary and secondary. Traumatic pneumothorax may result from either blunt trauma or penetrating injury to the chest wall. It can also be caused by iatrogenic injuries. Spontaneous pneumothorax is a significant health problem because of the high recurrence rate (this is so called recurrent pneumothorax).

The aim of the study: the review of modern diagnosis and surgical management of pneumothorax.

Methodology: This is a review article. We used Medline and Pubmed databasis for retrieving the literature.

Conclusion: Pneumothorax, either spontaneous or traumatic, demands urgent intervention in order to normalize lung function and save life of the patient.

Keywords: pneumothorax, chest drainage, thoracotomy.

INTRODUCTION

Pneumothorax is defined as the presence of air in the pleural cavity, ie, the space between the chest wall and the lung itself. Itard first recognized pneumothorax in 1803, and Laennec himself described the full clinical picture of the condition. In the second part of XIX century it was believed that tuberculosis was the main cause of pneumothorax since it was present mostly in patients with tuberculosis. On the other hand, Forlanini (Europe, in 1882) and John B. Murphy (the USA, in 1898) pointed out the useful results of pneumothorax in tuberculosis treatment (collapse therapy) (1, 2).

Although pathophysiological processes of pneumothorax are not fully known, it is known that pleural pressure is negative with values –2 to –40 cm H₂O.

If a communication develops between the pleural space and an alveolus, air will flow into the pleural space until a pressure gradient no longer exists or until the communication is sealed. Without the negative intrapleural pressure holding the lungs against the chest wall, their elastic recoil properties cause them to collapse. The main physiologic consequences of pneumothorax are a decrease in the vital capacity and a decrease in the partial pressure of arterial oxygen (PaO₂). In the otherwise healthy individual, the disease and the vital capacity is well tolerated. If the patient's lung function is compromised before the pneumothorax, however, the decrease in the vital capacity may lead to respiratory insufficiency with alveolar hypoventilation and respiratory acidosis. In a tension pneumothorax, the intrapleural air pressure exceeds atmospheric pressure. The mechanism by which a tension pneumothorax develops is probably related to some type of a one-way valve process in which the valve is open during inspiration and closed during expiration. If extra thoracic air pressure remains relatively higher than the pressure in the pneumothorax over a period of time, then the air in pleural space and the ambient atmosphere will begin to approach equilibrium. This can cause mediastinal shift, compression of the superior vena cava, compression of the contralateral lung. The reduced preload (volume returning to the heart) causes a reduced stroke volume and therefore reduced cardiac output. This may result in hemodynamic collapse and obstructive shock (3).

CLASSIFICATION OF PNEUMOTHORAX

According to aetiology pneumothorax is classified into spontaneous and traumatic (Table 1). Spontaneous pneumothorax is further classified into primary and secondary. Traumatic pneumothorax may result from either blunt trauma or penetrating injury to the

Table 1. *Classification of pneumothorax*

Spontaneous
Primary (<i>a rupture of a subpleural bleb</i>)
Secondary
Chronic obstructive pulmonary disease (COPD)
Cystic fibrosis
Bronchial asthma
Connective tissue diseases (Marfan Syndrome)
Interstitial lung diseases (<i>Eosinophilic granuloma</i>)
Pneumocystis carinii pneumonia (in AIDS patients)
Pneumonia with lung abscess
Pulmonary hydatid disease
Lung cancer (metastatic sarcoma)
Esophageal perforation
Catamenial pneumothorax
Neonatal pneumothorax
Traumatic
Iatrogenic
Central venous catheter insertion
Pacemaker implantation
Transthoracic needle biopsy
Transbronchial needle aspiration
Thoracocentesis
Laparoscopic surgery
Barotrauma
Blunt trauma
Road traffic accident trauma, falls, sports injuries
Penetrating trauma
Shot wounds, stab wounds

Source: Spasić M, Milisavljević S, Gajić V. Analiza učestalosti javljanja i načina lečenja pneumotoraksa u petogodišnjem periodu u Kragujevcu. Med Pregl 2012; LXV(Vol 5–6): 238–43.

chest wall. It can also be caused by iatrogenic injuries (3). This condition occurs in 7.4 to 18 per 100 000 men each year and 1.2 to 6 per 100 000 women each year. The incidence of secondary spontaneous pneumothorax is 6.3 per 100 000 men each year and 2 per 100 000 women each year (4). Some British studies that have been done recently show the incidence of primary spontaneous pneumothorax of 24 per 100 000 in men and 9.8 per 100 000 in women (5).

PRIMARY SPONTANEOUS PNEUMOTHORAX

Primary spontaneous pneumothorax (PSP) commonly occurs in tall, thin, adolescent men (male-female

ratio 6:1). Smoking is associated with a risk of developing pneumothorax in healthy smoking men (5). Because the gradient in pleural pressure is greater from the lung base to the lung apex in taller individuals, the alveoli at the lung apex are subjected to a greater mean distending pressure in taller individuals. Over a long period, this higher distending pressure could lead to the formation of subpleural blebs (6). The occurrence of PSP seems to be related to the level of cigarette smoking. The relative risk of a pneumothorax is 100 times higher in heavy smokers (more than 20 cigarettes/day) than in nonsmokers (7).

Some studies suggest that there is a familial tendency for the development of primary spontaneous pneumothorax. In some cases of PSP the mode of inheritance for the tendency for primary spontaneous pneumothorax is either autosomal dominant with incomplete penetrance or X-linked recessive (8). Primary spontaneous pneumothoraces are believed to be the result of rupture of sub-pleural blebs (9). Sub-pleural blebs and bullae are found in up to 90% of cases at thoracoscopy or thoracotomy and in up to 80% on computerised tomography (CT) scanning of the thorax (10, 11). The pathogenesis of the blebs remains unclear. There are suggestions that they may be congenital or inflammatory in origin or the result of disturbance of collateral ventilation (12). According to some studies, precipitating factors may be atmospheric pressure changes, physical activity, and exposure to loud music (13). Sadikot et al, study showed a recurrence rate of 39% during the first year (14). It also indicated that there was 54% risk of recurrence of pneumothorax in 4 years. According to their studies, factors that have been proposed to predispose patients to primary spontaneous pneumothorax (PSP) include smoking and patient's height. The peak age for the occurrence of primary spontaneous pneumothorax is the early 20's and it rarely occurs after age 40. Primary spontaneous pneumothorax usually develops while the patient is at rest. Main symptoms are chest pain and dyspnea. This pain may be mild or severe, sharp and steady ache in character, and usually resolves within 24 h even though pneumothorax still exists (15). It is interesting that many patients with a primary pneumothorax do not seek medi-

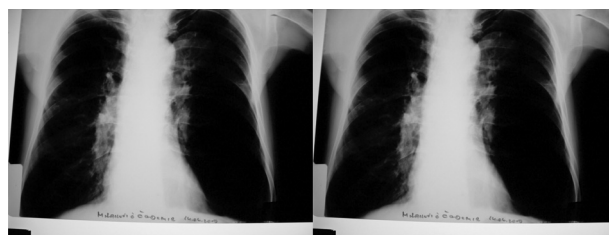


Figure 1. *1a Spontaneous pneumothorax in the left lung; 1b Bilateral pneumothorax*

cal attention for several days- more than 50% of patients waited more than 24 hours after their symptoms started to seek help, and 18% waited more than a week after the symptoms appeared (16) (Figure 1a, 1b).

SECONDARY SPONTANEOUS PNEUMOTHORAX

Secondary spontaneous pneumothorax (SSP) occurs in patients with underlying lung disease.

The incidence of secondary spontaneous pneumothorax is similar to that of primary spontaneous pneumothorax. It usually occurs in older people, after the age of 60 (13). Some research show that the peak incidence for males occurs in the seventh decade of life, 60/100.000 each year (5). Many lung diseases can cause SSP: chronic airway and alveolar diseases (COPD, bronchial asthma, cystic fibrosis); infectious lung diseases (tuberculosis, pneumocystis carinii, lung abscess leading to pneumothorax with pleural empyema); interstitial lung diseases (idiopathic fibrosing alveolitis, sarcoidosis, histiocytosis X, lymphangio leiomyomatosis); systemic connective tissue diseases (rheumatoid arthritis, ankylosing spondylitis, scleroderma, Marfan- and Ehlers Danlos-syndrome); malignant lung and chest diseases (bronchial cancer, sarcoma) (13). The most common lung disease that causes spontaneous pneumothorax is chronic obstructive pulmonary disease (COPD).

Degradation of elastic fibres of visceral pleura contributes the occurrence of pneumothorax in COPD (15). SSP occurred in many HIV-infected patients.

Pneumocystis carinii (PCP) infection has been considered to be the main aetiological factor for this association, because of a severe form of necrotising alveolitis that occurs in which the subpleural pulmonary parenchyma is replaced by necrotic thin-walled cysts and pneumatoceles. These patients can develop bilateral pneumothorax (15). The relative risk of recurrence of secondary spontaneous pneumothorax is 45% higher than the one of PSP (15). Risk factors for recurrence of SSP include age, pulmonary fibrosis and emphysema (17). Because lung function in these patients is already compromised, secondary spontaneous pneumothorax (SSP) often presents as a potentially life-threatening disease. The clinical signs and symptoms of secondary pneumothorax are more intense and severe. Dyspnea is the main symptom, and chest pain on the same side as the affected lung is present in most patients. Some of the most clinically significant symptoms that may develop include hypotension, tachycardia, cyanosis, hypoxemia with or without hypercapnia, and acute respiratory distress. The physical findings are often subtle and may be masked by the under-

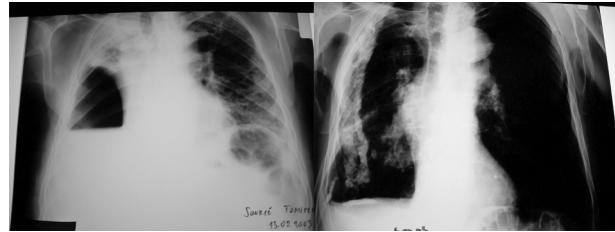


Figure 2. *2a* Hydropneumothorax in the right lung Tuberculosis “destroyed lung”; *2b* Fibrothorax in the right lung After the thoracic drainage

lying lung disease, especially in patients with COPD (13) (Figure 2a, 2b).

CATAMENIAL PNEUMOTHORAX

Catamenial pneumothorax is a spontaneous type of pneumothorax that starts at the onset of or within 24 to 72 hours after onset of menses and is usually recurrent. Catamenial pneumothorax was first described by Maurer in 1958. The initial pneumothorax usually does not occur until the woman is in her thirties. Lillington introduced in 1972 the term catamenial pneumothorax to describe the already reported phenomenon (18). This pneumothorax was considered to be a rare type with the incidence 1–5% in women in reproductive age (18). Recent studies have shown that in 25% of cases the recurrent catamenial pneumothorax was related to time of menstruation (19), so the incidence is not so low as it was believed. These pneumothoraces are usually right sided (according to some authors, in 95%) (20). The pathophysiology of catamenial pneumothorax is uncertain. Three distinct mechanisms have been proposed based on metastatic, hormonal and anatomic model (18). The metastatic model hypothesizes migration of endometrial tissue via the peritoneal cavity through transdiaphragmatic lymphatic channels, via diaphragmatic fenestrations, or hematogenously into the pleural space. Congenital fenestrations are more common in right hemidiaphragm making intrathoracic endometriosis right sided. Endometrial deposits have been identified in the pleural space in 13% to 62,5% of the cases (19, 20, 21). The hormonal hypothesis was proposed by Rossi and Goplerud in 1974. It suggests that high serum levels of prostaglandin F₂ at ovulation leads to vasospasm associated ischemia with tissue injury and alveolar rupture. However this cannot explain the preponderance of right sided involvement. Also there are no non-steroidal anti-inflammatory medications (NSAIDs) capable of preventing recurrence of catamenial pneumothorax in respective reported series. Thus, this hypothesis was rejected (18). The anatomic model for catamenial pneumothorax is based on the influx of air into the pleural space from the peritoneal

cavity via diaphragmatic fenestrations (18). Also concomitant pneumoperitoneum is found in some patients with catamenial pneumothorax (18). Diaphragmatic defects were found in 50%–62,5% of patients. To prevent recurrence, diaphragmatic defects should certainly be closed (19, 21). Patients with catamenial pneumothorax develop chest pain and dyspnea within 24 to 72 hours of the onset of the menstrual flow. It is usually recurrent and correlated with menses (18).

NEONATAL PNEUMOTHORAX

Spontaneous pneumothorax is present shortly after birth in 1% to 2% of all infants. It is twice as common in boys as in girls. The incidence of neonatal pneumothorax is higher in cases of preterm birth and low birth weight. (15%). Also, the cases of infants with fetal distress and respiratory distress syndrome have higher incidence (19%) (15). The pathogenesis of neonatal pneumothorax is related to the mechanical problems of first expanding the lung. Transpulmonary pressures have average values 40cm H₂O during the first few breaths of life, with occasional transpulmonary pressures as high as 100 cm H₂O. If bronchial obstruction occurs, high transpulmonary pressures may lead to rupture of the lung (15). The signs vary from none to severe acute respiratory distress. In the infant with a small pneumothorax, mild apneic spells with some irritability or restlessness may be present. Large pneumothoraces incur varying degrees of respiratory distress, and, in severe cases, marked tachypnea, grunting, retractions, and cyanosis are present (15). The most reliable clinical sign of neonatal pneumothorax is a shift of the apical heart impulse away from the side of the pneumothorax (15) (Figure 3a, 3b)

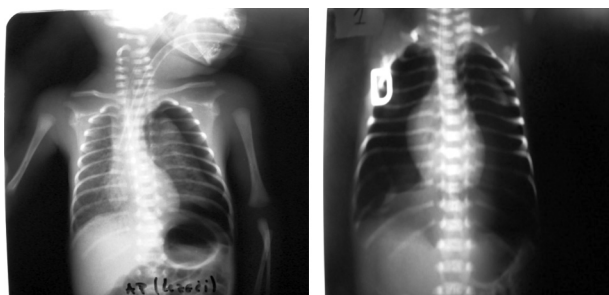


Figure 3. 3a Neonatal pneumothorax in the left lung; 3b Bilateral neonatal pneumothorax

IATROGENIC PNEUMOTHORAX

The leading cause of iatrogenic pneumothorax is transthoracic needle aspiration (24%), subclavian needle (22%), thoracentesis (20%), transbronchial biopsy (10%), pleural biopsy (8%) and positive-pressure ventilation (7%) (13). Other procedures associated with

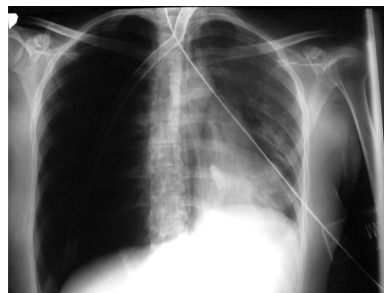


Figure 4. Iatrogenic pneumothorax in the right lung. The rupture of membranous tracheal wall caused by reinforced tubus

the iatrogenic pneumothorax development include tracheostomy, intercostal nerve block, mediastinoscopy, liver biopsy, the insertion of nasogastric tubes, cardiopulmonary resuscitation (15). Iatrogenic pneumothorax should be suspected in any patient with respiratory distress symptoms as well as in patients who underwent some procedures (15) (Figure 4).

TRAUMATIC PNEUMOTHORAX

Traumatic pneumothorax may result from either blunt trauma or penetrating injury to the chest wall. Pneumothorax can occur at the time of the injury, immediately after the injury, or later. The incidence of severe traumatic pneumothorax is higher than 20% (22), and the incidence of chest injury is 50% (13). With non-penetrating trauma, a pneumothorax may develop if the visceral pleura is lacerated secondary to a rib fracture, dislocation. Sudden chest compression abruptly increases the alveolar pressure, which may cause alveolar rupture. Blunt trauma can also cause alveolar rupture (23). With penetrating chest trauma, the wound allows air to enter the pleural space directly through the chest wall or through the visceral pleura from the tracheobronchial tree (23). Traumatic pneumothorax can also be classified as simple, open (“sucking”) and tension pneumothorax. In simple pneumothorax, the air from the injured lung enters the pleural space. There are not many symptoms of this type of pneumothorax (1). Open pneumothorax occurs when a wound on the chest is large enough to allow air to pass freely in and out of the pleural space. In this case, the atmospheric pressure is in equilibrium with intrapleural pressure, blocking the lung inflation and alveolar ventilation. The rush of air through the wound in the chest wall produces a sucking sound. In such patients the lung collapses. Traumatic open pneumothorax calls for the emergency intervention- sealing the open wound with Vaseline gauze and placing the chest tube. The wound treatment involves common surgical procedures (1, 23) (Figure 5). A tension pneumothorax is the result of the chest wall or lung injury. A one-way valve mechanism

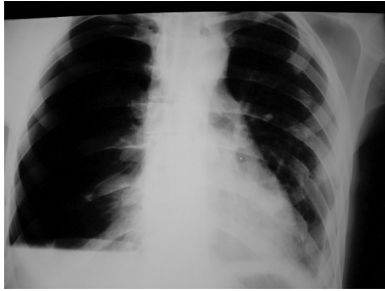


Figure 5. Traumatic pneumothorax in the right lung (traffic accident trauma). Serial rib fractures on the right side Left pulmonary contusion

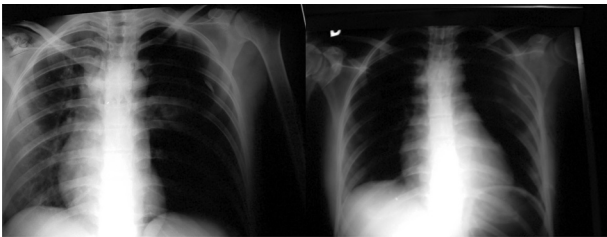


Figure 6. 6a. Tension pneumothorax in the left lung
6b. Condition after chest tube drainage in the left lung. Complete re-expansion of the left lung

occurs, where the air that enters the pleural space with each inspiration is trapped and cannot be expelled during expiration. Interthoracic pressure increases causing the lung to collapse. The collapse in the lung causes a shift in the mediastinum away from the injured side, resulting in hypoventilation, decreased venous return to the heart and potentially in development of obstructive shock. The signs and symptoms associated with tension pneumothorax include cyanosis, dyspnea, tachypnea, tachycardia, hypotension, distended neck veins, profuse diaphoresis. A tension pneumothorax is a life-threatening injury that should be diagnosed and managed urgently. Management is performed by immediate needle decompression. A large bore needle is inserted in the II intercostal space, at the midclavicular line (1, 15) (Figure 6a, 6b).

DIAGNOSIS OF PNEUMOTHORAX

The diagnosis of pneumothorax is established from the patients' history and physical examination findings that reveal decreased movement of the hemithorax, decreased or absent fremitus, hyper sonority on percussion and decreased or absent breath sounds on the affected side. Radiography of the chest in the upright position and PA projection of the chest are the most common methods of diagnosing pneumothorax. The main feature of a pneumothorax on a chest radiograph is a white visceral pleural line, which is separated from the parietal pleura by a collection of gas (15). Radiographs that are obtained in the lateral decubitus po-

sition can be useful in cases of clinically suspected pneumothorax, while PA radiograph is normal. CT scan of the chest is used to differentiate large bulla from pneumothorax (24). When PA radiograph reveals abnormalities, it is possible to calculate the actual pneumothorax size by using the Light index: $PTX\% = 100 [1 - \text{diameter lung} / \text{diameter hemithorax}]^3$, and it may be useful for research purposes (15). To calculate the size of a pneumothorax: is to measure the distance between the pleural surface and the lung edge (at the level of the hilum). If this is 2 cm or more, it represents a large pneumothorax and if it is < 2 cm it is considered to be a small pneumothorax (24).

COMPLICATIONS OF PNEUMOTHORAX

These complications include tension pneumothorax, hemopneumothorax, bronchopleural fistula, pneumomediastinum, chronic pneumothorax (failure of the lung to re-expand).

Spontaneous hemopneumothorax

The incidence of pleural effusion is 15 to 20% in patients with hydropneumothorax.

Approximately 5% of patients with pneumothorax will have concomitant haemothorax with an amount of blood in the pleural space. The mechanisms of bleeding described in SHP are bleeding either of a torn apical vascular adhesion between the parietal and visceral pleura or of torn congenital aberrant vessels between the parietal pleura and the bulla as the lung collapses or due to rupture of vascularized bullae. Manifestations depend on the amount of blood lost during this disorder. Treatment of SHP includes tube thoracostomy for drainage of the haemothorax and re-expansion of the lung. If the re-expansion of the lung does not stop the bleeding, thoracotomy is needed to stop the bleeding (15) (Figure 7).

Bronchopleural fistula

A bronchopleural fistula may occur in patients with primary spontaneous pneumothorax (3% to 4%),

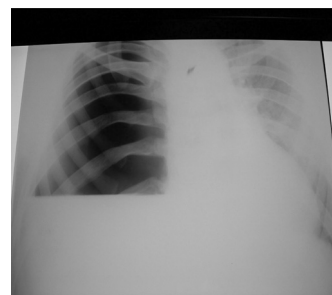


Figure 7. Spontaneous hemopneumothorax in the right lung. Upright radiography

though it is more common in patients with secondary spontaneous pneumothorax or traumatic pneumothorax. Persistent air leakage occurring after thoracic drainage for pneumothorax is the early clinical sign of this complication. It can be managed by thoracotomy, closing the fistula and pleurodesis (15).

Pneumomediastinum

Pneumomediastinum is a rare complication of pneumothorax (< 1%). It is the presence of free air within the mediastinum. Subcutaneous emphysema is often associated with pneumomediastinum. This entity is without significant clinical importance. Pneumomediastinum has rarely been reported to cause some serious complications (esophageal injuries and injuries in the large airways) (1).

Chronic pneumothorax (failure of the lung to re-expand)

Chest tubes are used for pneumothorax to promote lung re-expansion. But in some cases, this procedure fails. The thickened cortex on the visceral pleura prevents the re-expansion of the lung. Medical procedures for this condition is thoracotomy and decortication (1).

TREATMENT

The objective in treating a pneumothorax is to eliminate the air from the pleural space, to allow lung to re-expand, and to prevent recurrences. The best method for achieving this depends on the severity of the lung collapse, the type of pneumothorax, patient's overall health and on the risk of complications. There are many therapeutic possibilities in clinical practice.

Observation

Observation is recommended for patients with PSP occupying less than 15% of the hemithorax. As with these patients, observation remains the first-line treatment in patients with pneumothoraces of less than 1 cm depth or isolated apical pneumothoraces (24). The rate of air absorption is 1, 25% every 24 hours. Supplemental oxygen can be administered to increase the rate of pleural air absorption. A small number of patients is treated this way (15).

Aspiration- exsufflation

Aspiration may be the initial treatment for the patients with primary pneumothorax. It may also be considered for patients younger than 50 with secondary pneumothorax of moderate size (air rim 1–2 cm). Percutaneous needle aspiration results in complete lung re-expansion in 59 to 83% patients with PSP and in 33 to 67% patients with SSP. Recurrence rate of pneumo-

thorax after the exsufflation is almost the same as the one after the chest tube drainage (24).

Tube thoracostomy

Tube thoracostomy is the most commonly performed surgical procedure in thoracic surgery.

Thoracostomy tube placement is indicated for the PSP and symptomatic patients, as well as for the symptomatic SSP, iatrogenic and traumatic pneumothorax (24).

The overall objective of chest-tube therapy is to promote lung reexpansion. The chest tube is inserted via an incision at the 4th or 5th intercostals space in the anterior axillary or mid-axillary line. It can also be inserted via 2nd midclavicular intercostal space (Figure 8). It is inserted near the upper border of the rib. There are three techniques most commonly used to place a chest tube: using the trocar, associated with a higher rate of intrathoracic organ injury, blunt dissection after skin incision (less comfortable but with lower risk of complications) (Figure 9), or Seldinger technique in which a guide wire is inserted through the introducer needle and a chest tube is inserted into the pleural space. Once the chest tube has been inserted, it must be



Figure 8. Thoracic trocar drainage in the right lung

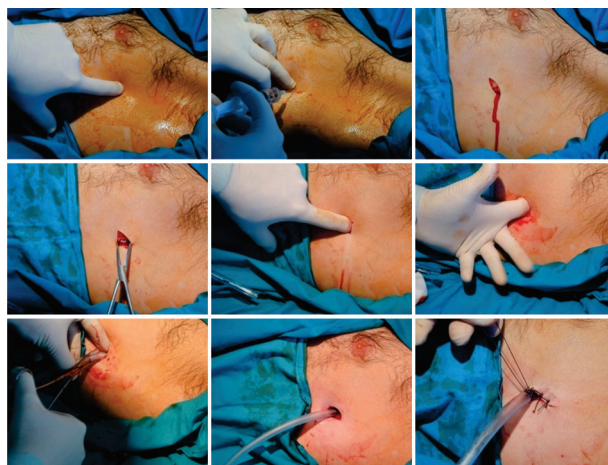


Figure 9. Tube thoracostomy drainage

connected to either suction or an apparatus to allow unidirectional drainage (water seal without suction or a Heimlich valve). If the adequate expansion is achieved, the catheter can be removed (after 5 to 7 days). The instillation of sclerosing agents (talc) through chest tubes can help prevent recurrences of pneumothorax (1).

SURGICAL MANAGEMENT AND PREVENTION OF RECURRENT PNEUMOTHORACES

Chemical pleurodesis

Chemical pleurodesis is a procedure to achieve symphysis between the two layers of pleura by sclerosing agents. These agents can be introduced into the pleural space. The therapeutic action of the agent (tetracycline or talc) instilled into the pleural cavity through a chest drain is thought to result from induction of an inflammatory reaction (24).

Surgical management and mechanical pleurodesis

Surgical management is the common method for pneumothoraces with persistent air leak (5 to 7 days of thoracic drainage), the failure of the lung to expand, recurrence of pneumothorax (ipsilateral or contralateral), bilateral spontaneous pneumothorax, hemothorax, high risk professions (air craft personnel, scuba divers). The objective of surgical management of pneumothorax is to remove air from the pleural cavity (resection of blebs) and to prevent recurrence (obliteration of pleural space). Small posterolateral thoracotomy, transaxillary mini thoracotomy, minimally invasive endoscopic surgery (VATS- Video-assisted thoracoscopic surgery) are the most common surgical procedures (24) (Figure 10). Bullae can be treated with different surgical procedures- lung resection, stapled excision, electrocoagulation, suture ligation. To prevent the recurrence of pneumothorax, resection is combined with some of the procedures for obliteration of pleural space. This procedure may be parietal pleurectomy (partial-apical or total), parietal pleural abrasion (mechanical pleurodesis), chemical pleurodesis (application of sclerosing agents). Parietal pleurectomy produces adhesion between visceral pleura and endothoracic fascia; pleural abrasion produces adhesions between visceral and parietal pleura while anatomic layers are preserved, reducing the risk of thoracoscopy (24). Open thoracotomy with bullectomy plus pleural abrasion or pleurectomy is effective in diminishing the rate of recurrence (1%). The rate of mortality after the procedure is low (3,7%). Compared to VATS, after this treatment the lung function is compromised and the

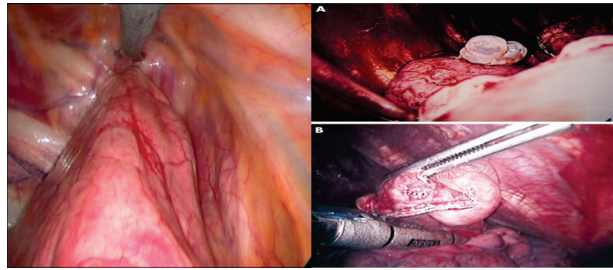
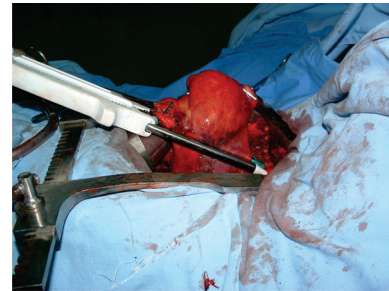


Figure 10. VATS resection of right-sided bullae



*Figure 11. Primary spontaneous pneumothorax.
Excision of the bulla using stapler*

hospitalization period is longer. In minimally invasive surgery not all blebs may be detected, and the recurrence rate is higher (5-10%), while hospitalization period is shorter, post-surgical pulmonary gas exchange is better and post-surgical pain is not so severe (24) (Figure 11).

CONCLUSION

Pneumothorax is defined as the presence of air in the pleural space. It is caused by a rupture in visceral or the parietal pleura. Pneumothoraces can be divided into spontaneous pneumothoraces and traumatic pneumothoraces. Spontaneous pneumothoraces are further divided into primary and secondary spontaneous pneumothoraces. Traumatic pneumothorax may result from either blunt trauma or penetrating injury to the chest wall. It may also be caused by iatrogenic injuries resulting from diagnostic or therapeutic procedures.

The diagnosis of pneumothorax can be established from the patients' history, physical examination findings and the chest X-ray. Pneumothorax can be managed conservatively (rest and observation), exsufflation, and chest tube thoracotomy. Recurrent pneumothorax and complications are managed through surgical procedures (thoracotomy or VATS).

Abbreviations

PaO₂ — partial pressure of arterial oxygen
PSP — Primary spontaneous pneumothorax
SSP — Secondary spontaneous pneumothorax
COPD — Chronic obstructive pulmonary disease
PCP — Pneumocystis carinii
SHP — Spontaneous hemopneumothorax
VATS — Video-assisted thoracoscopic surgery

Sažetak

PNEUMOTORAKS — DIJAGNOSTIKA I LEČENJE

Milisavljević Slobodan,^{1,2} Spasić Marko,² Milošević Bojan²¹ Klinika za opštu i grudnu hirurgiju, Klinički centar Kragujevac² Medicinski fakultet Univerziteta u Kragujevcu, Kragujevac, Srbija

Uvod: Pneumotoraks predstavlja prisustvo vazduha u pleuralnom prostoru, odnosno prisustvo vazduha između pluća i zida grudnog koša. U zavisnosti od etiologije pneumotoraks se klasifikuje na spontani i traumatski. Spontani pneumotoraks se dalje deli na primarni i sekundarni. Traumatski pneumotoraks nastaje kao posledica tupih ili penetrantnih povreda grudnog koša, ili nakon jatrogenih povreda. Recidivantni pneumotoraks se javlja kao ponovljeni spontani pneumotoraks.

Cilj rada: Prikaz savremene dijagnostike i načina hirurškog lečenja kod pacijenata sa pneumotoraksom.

Metodologija: Ovo je pregledni članak. Korišćena je literatura uvidom u bazu medicinskih podataka Medline i Pubmed.

Zaključak: Pneumotoraks, bilo spontani bilo traumatski predstavlja hitno stanje u medicini i zahteva brzu i neodložnu intervenciju lekara, kako bi se funkcija pluća što pre normalizovala i očuvao život vitalno ugroženom pacijentu.

Ključne reči: pneumotoraks, grudna drenaža, torakotomija.

REFERENCES

- Shields TW, Thomas W Shields (Editor), Cicero JL, Ponn RB, Rusch VW, editors. General Thoracic Surgery. 6th ed. Philadelphia: Lippincott Williams&Wilkins; 2005.
- Sellke FW, del Nido PJ, Swanson SJ, editors. Sabiston and Spencer's Surgery of the Chest. 7th ed. Philadelphia: Elsevier Saunders; 2005.
- Sugarbaker DJ, Bueno R, Krasna MJ, Mentzer SJ, Zellos L, editors. Adult Chest Surgery. 1st ed. New York: McGraw-Hill; 2009.
- Melton LJ 3rd, Hepper NCG, Offord KP. Incidence of spontaneous pneumothorax in Olmsted County, Minnesota: 1950–1974. Am Rev Respir Dis. 1979; 120(6): 1379–82.
- Gupta D, Hansell A, Nichols T, Duong T, Ayres JG, Starchan D. Epidemiology of pneumothorax in England. Thorax. 2000; 55(8): 666–71.
- West JB. Distribution of mechanical stress in the lung, a possible factor in localisation of pulmonary disease. Lancet. 1971; 1(7704): 839–41.
- Bense L, Eklund G, Wiman LG. Smoking and the increased risk of contracting spontaneous pneumothorax. Chest. 1987; 92(6): 1009–12.
- Abolnik IZ, Lossos IS, Zlotogora J, Brauner R. On the inheritance of primary spontaneous pneumothorax. Am J Med Genet. 1991; 40(2): 155–8.
- Schramel FM, Postmus PE, Vanderschueren RG. Current aspects of spontaneous pneumothorax. Eur Respir J. 1997; 10(6): 1372–9.
- Donahue DM, Wright CD, Viale G, Mathisen DJ. Resection of pulmonary blebs and pleurodesis for spontaneous pneumothorax. Chest. 1993; 104(6): 1767–9.
- Lesur O, Delorme N, Frogamet JM, Bernadac P, Polu JM. Computed tomography in the aetiological assessment of idiopathic spontaneous pneumothorax. Chest. 1990; 98(2): 341–7.
- Noppen M: Con: blebs are not the cause of primary spontaneous pneumothorax. J Bronchol and Interv. Pulmology. 2002; 9(4): 319–25.
- Noppen M, De Keukeleire T. Pneumothorax. Respiration. 2008; 76(2): 121–7.
- Sadikot RT, Greene T, Meadows K, Arnold AG. Recurrence of primary pneumothorax. Thorax. 1997; 52(9): 805–9.
- Mason RJ, Broaddus VC, Murray JF, Nadel JA, editors. Murray and Nadel's Textbook of Respiratory medicine. 4th ed. Philadelphia: Elsevier Saunders; 2005.
- Seremetis MG: The management of spontaneous pneumothorax. Chest. 1970; 57(1): 65–8.
- Lippert HL, Lund O, Blegvad S, Larsen HV. Independent risk factors for cumulative recurrence rate after first spontaneous pneumothorax. Eur Respir J. 1991; 4(3): 324–31.
- Peikert T, Gillespie DJ, Cassivi SD. Catamenial pneumothorax. Mayo Clin. Proc. 2005; 80(5): 677–80.
- Alifano M, Roth T, Broet SC, Schussler O, Magdeleinat P, Regnard JF. Catamenial pneumothorax: a prospective study. Chest. 2003; 124(3): 1004–8.
- Joseph J, Sahn SA. Thoracic endometriosis syndrome: new observations from an analysis of 110 cases. Am J Med. 1996; 100(2): 164–70.
- Bagan P, Le Pimpec Barthes F, Assouad J, Souilamas R, Riquet M. Catamenial pneumothorax: retrospective study of surgical treatment. Ann Thorac Surg. 2003; 75(2): 378–81.
- Di Bartolomeo S, Sanson G, Nardi G, Scian F, Michelutto V, Lattuada L. A population-based study on pneumothorax in severely traumatized patients. J Trauma. 2001; 51(4): 677–82.
- Feliciano DV, Mattox KL, Moore EE, editors. Trauma. 6th ed. New York: McGraw-Hill; 2008.
- M. Henry, T. Arnold, J. Harvey. BTS guidelines for the management of spontaneous pneumothorax Thorax. 2003; 58(suppl 2): 1139–52.

Correspondence to/Autor za korespondenciju

Prof dr Milisavljević Slobodan

General and Thoracic Surgery Clinic, Clinical Centre Kragujevac

Phone: 034/505315

e-mail: s.milisavljevic65@gmail.com