but close monitoring is mandatory. Bronchoscopic therapy should be considered in patients too debilitated for surgical therapy.


Further Reading


Introduction

Pneumonectomy, the surgical removal of one lung, is a procedure associated with significant morbidity. Expected complication rates can range between 40% and 60%. Many complications of pneumonectomy are not unique to this procedure, but are in fact common complications of any major surgery, for example, arrhythmias, myocardial infarction, thromboembolism, hypoxemia, and pneumonia. There are other complications that also are not unique to this procedure, but are more commonly associated with it, for example, empyema, bronchopleural fistula, esophagopleural fistula, chylothorax, intracardiac right-to-left shunt, cardiac herniation, and postoperative pulmonary edema. Finally, there is one complication truly unique to this surgical procedure, namely the postpneumonectomy syndrome. This is a condition resulting in large airway obstruction. It is a rare complication, and develops in probably less than 1% of the patients undergoing this procedure. The postpneumonectomy syndrome develops as a result of severe anatomical changes that occur in the post-pneumonectomy thorax. Initially, this condition was called the right pneumonectomy syndrome, as it was initially reported to occur only after right-sided procedures. However, a few rare subsequent reports have described this syndrome occurring after left-sided procedures.

Etiology

In order to understand how the postpneumonectomy syndrome develops, we will first review the typical changes that occur in the thorax after pneumonectomy. Within the first 24 h after surgery, the mediastinum starts to shift towards the pneumonectomy space, and the ipsilateral hemidiaphragm becomes slightly elevated. These changes are thought to occur due to the negative pressure that develops within the empty pleural space. Immediately after surgery, air within the postpneumonectomy space starts to be absorbed, and serosanguineous fluid begins to accumulate. As seen on serial chest radiographs, fluid
typically accumulates at a rate of 1–2 rib spaces per day in the immediate postoperative period (Figure 1). After 2 weeks, 80–90% of the postpneumonectomy space is typically filled with fluid. Complete opacification of the postpneumonectomy space is seen on chest radiograph anywhere between 2 and 7 months after pneumonectomy.

Over the course of several months to years, there is further shifting of the mediastinum and vital structures (heart, great vessels, and esophagus) towards the surgical side, further elevation of the hemidiaphragm, and hyperinflation of the remaining lung. With elevation of the hemidiaphragm, the liver, stomach, spleen, and large bowel can elevate into the thorax (Figure 2). Also during this time, there is narrowing of the ipsilateral intercostal spaces, and the development of mild scoliosis. The scoliosis is usually limited to the upper thoracic spine, and results in a 5–20° curvature, with the convexity of the spine towards the unoperated side. Symptoms associated with this degree of scoliosis have not been reported.

Over time there is slow reabsorption of the fluid within the postpneumonectomy space. However, complete absorption of all fluid and obliteration of the postpneumonectomy space is uncommon, even 30–40 years after surgery. Autopsy studies have revealed that the postpneumonectomy space is completely obliterated in less than one-third of patients, with the remaining space containing various amounts of fluid. It is felt that this remaining fluid can serve as a nidus for infection in bacteremic patients, resulting in the development of a late postpneumonectomy empyema.

The degree of shifting of the mediastinal structures and hyperinflation of the remaining lung depends, to
some extent, on the age of the patient at the time of pneumonectomy. The younger the patient at the time of surgery, the more shifting and hyperinflation is likely to occur. The explanation for this is that younger patients have more elasticity and compliance of the mediastinal tissue and remaining lung, allowing for more shifting and hyperinflation. Women, for reasons not entirely clear, also tend to have more shifting and hyperinflation after a pneumonectomy than men do.

It is the shifting of the vital structures and hyperinflation and herniation of the remaining lung that places the postpneumonectomy patient at risk for developing the postpneumonectomy syndrome.

**Clinical Presentation**

Risk factors for developing the postpneumonectomy syndrome are age at the time of pneumonectomy, right-sided procedures, and female sex. Most of the cases of postpneumonectomy syndrome are seen in patients who have had a right pneumonectomy in childhood or early adulthood. However, this syndrome is also well described in patients who had lung removal during their late adulthood, as well as in patients who have undergone left-sided procedures.

Onset of symptoms of postpneumonectomy syndrome can occur anywhere from a few months to over 35 years after surgery. Classic symptoms include progressive dyspnea, cough, and stridor. The narrowing of the bronchus can also result in the development of recurrent pneumonia and bronchiectasis. A history of recurrent pneumonias in a patient who has undergone a pneumonectomy, especially a right-sided procedure, should alert the physician to the possibility of this syndrome. If left untreated, postpneumonectomy syndrome can result in respiratory insufficiency, recurrent pneumonia, development of tracheobronchomalacia at the site of airway compression, and death.

The diagnosis of postpneumonectomy syndrome should be entertained in any patient who develops the above symptoms and evidence of airflow obstruction on pulmonary function testing months to years after pneumonectomy. Spirometry may reveal a forced vital capacity (FVC) that is reduced, but higher than expected for a patient with one lung. This supports the finding of hyperinflation of the remaining lung. Forced expiratory volume in 1 s (FEV₁)/FVC is usually reduced. Flow-volume loops may reveal a pattern of variable intrathoracic upper airway obstruction, suggesting the diagnosis. Compression of the airway is best confirmed with a chest computed tomography (CT) scan or with bronchoscopy. Bronchoscopy is particularly helpful in evaluating for the development of tracheobronchomalacia at the site of compression, as well as evidence of purulent secretions trapped distal to the obstruction.

**Pathophysiology**

This syndrome develops after a right-sided pneumonectomy when the mediastinal structures shift posteriorly and to the right, into the vacant pleural space. The left lung becomes severely hyperinflated and also herniates into the vacant pleural space. The rotation and shifting of the mediastinum coupled with hyperinflation of the remaining lung results in stretching and narrowing of the left mainstem bronchus (Figure 3). In addition, the stretched and narrowed bronchus can become compressed by the left pulmonary artery anteriorly, and the aorta or thoracic vertebrae posteriorly.

In the rare patient who develops this syndrome after a left-sided pneumonectomy, severe shifting of the mediastinum and excess hyperinflation of the right lung are required. This results in stretching and narrowing of the distal trachea and right mainstem bronchus. Further compression of the distal trachea and right mainstem bronchus occurs between the mediastinum and thoracic vertebrae. It has also been reported that patients with right-sided aortic arches can develop postpneumonectomy syndrome after a left pneumonectomy when the stretched right mainstem bronchus is compressed posteriorly by the right-sided aorta.

**Treatment**

Treatment of postpneumonectomy syndrome is surgical and is aimed at relieving the obstruction of the
airway. Earlier surgical interventions to relieve the symptoms of postpneumonectomy syndrome were directed at the great vessels that were compressing the airway. Specifically, dividing the aortic arch, bypass grafts, and suspending the aorta from the posterior surface of the sternum were all attempted in an effort to relieve the compression of the airway. These procedures, however, had limited success. In patients who had airway compression from a vertebral body, attempts to remove part of the bone by surgery were also unsuccessful in the long-term management of this syndrome. In the early 1990s, Grillo and colleagues reported good success by repositioning of the mediastinum in patients with this condition. During this procedure, the mediastinum was repositioned in the center of the chest after being freed up from adhesions. To prevent re-shifting of the mediastinum, the postpneumonectomy space was filled with non-absorbable materials. Material such as silicone or saline breast implants, testicular implants, and sulfur hexafluoride have been used. Today, saline-filled expandable breast implants are most frequently employed as they are felt to be the safest, and offer the advantage of easily being made larger if needed. This is of particular importance in the management of growing children with postpneumonectomy syndrome. Patients undergoing surgical re-positioning of the mediastinum with placement of an implant into the pleural space have had resolution of symptoms, and good long-term survival.

Patients with underlying tracheobronchomalacia require an additional procedure after repositioning of the mediastinum, that is, treatment of the airway malacia. Placement of self-expanding metallic stents appears to be the most effective treatment for the tracheobronchomalacia. Resection of the damaged airway or placement of Silastic tubes has been associated with high mortality rates and poor outcomes.

See also: Bronchomalacia and Tracheomalacia. Symptoms of Respiratory Disease: Cough and Other Symptoms.

Further Reading


Subcutaneous Emphysema

R C Hyzy and M R McClelland, University of Michigan, Ann Arbor, MI, USA

© 2006 Elsevier Ltd. All rights reserved.

Abstract

Subcutaneous emphysema describes the presence of gas in subcutaneous tissue. It has several known causes, among them anaerobic infections, traumatic disruption of mucosal surfaces, and alveolar rupture. Diagnosis is characterized by the presence of crepitation on palpation over the affected area and radiographic evidence of gas in the subcutaneous tissues. The pathogenesis relates to the tracking of gas along fascial planes into the subcutaneous space of the neck, chest, or extremities. Therapy of subcutaneous emphysema itself is generally supportive and involves the treatment of the underlying condition, as subcutaneous emphysema is usually self-limited and benign. However, the appearance of subcutaneous emphysema often warrants a search for more serious underlying pathology such as pneumothorax, pneumomediastinum, or tracheobronchial or esophageal disruption.

Introduction

Subcutaneous emphysema is a term used to describe the finding of gas within subcutaneous soft tissues, usually in the thorax or neck. The first known description dates back to the seventeenth century, when Louise Bourgeois, midwife to the Queen of France, described crepitations in patients associated with the extreme effort of childbirth. In the nineteenth century, Laennec provided a description in his Treatise on the Diseases of the Chest and Mediate Auscultation. Subcutaneous emphysema is a relatively common clinical finding associated with a broad range of disorders of varying degrees of severity, including trauma, surgery, tube thoracostomy placement, infection, and positive pressure ventilation. One study of 134 consecutive patients with chest tubes found 25 (18.6%) had subcutaneous emphysema. The incidence has not been described in any systematic way, presumably because subcutaneous emphysema is associated with such a diverse range of conditions and is typically a self-limited process of little direct clinical consequence. In general, knowledge of subcutaneous emphysema stems from individual case reports, small series, and clinician experience.