

Acquired lung herniation

A case series and review of the literature

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Abstract

Pulmonary herniation is a protrusion of the lung beyond the usual boundaries of the thoracic cavity, which is caused by increased intrathoracic pressures coupled with defects in the thoracic wall. Most lung herniations due to surgical intervention described in the literature occurred weeks to months in the post-operative period. We describe 3 cases of lung herniation occurring years after surgery, all apparently caused by acute increase in intrathoracic pressure.

Introduction

Pulmonary herniation is a protrusion of the lung beyond the usual boundaries of the thoracic cavity, which is caused by increased intrathoracic pressures coupled with defects in the thoracic wall. Herniation is usually diagnosed after trauma or noticed immediately post surgery, although spontaneous pulmonary herniation has been known to occur.¹ The increased intrathoracic pressure can be due to a variety of causes, with chronic obstructive pulmonary disease and the associated chronic cough being frequently implicated as the most common etiology. The incomplete distribution of the three muscles that cover the intercostal space (the external, medial, and internal intercostal muscles) in the intercostal space and their manipulation during surgery leads to areas of potential weakness and defects.¹ Most lung hernias are asymptomatic, but when symptomatic they present as a bulging, crepitant mass protruding through the chest wall. A computed tomographic scan is usually diagnostic, and a small subset of patients require surgery to correct the defect.²

We present three cases we recently saw at our hospital of very delayed post surgical lung herniation, which were all caused by an acute increase in intrathoracic pressure and acquired post-surgical weakness of the intrathoracic muscles.

Case 1

A 57 year-old woman with a past medical history of coronary artery disease status post coronary artery bypass six years prior, morbid obesity, and mild intermittent asthma presented with progressively worsening dyspnea, increased lower extremity edema, paroxysmal nocturnal dyspnea, 3-pillow orthopnea, and a productive cough. The patient reported shortness of breath at rest despite treating herself for a presumed asthma exacerbation with nebulizers four times a day and concomitant medication compliance with her cardiac medications. On exam, she was in respiratory distress and was only able to maintain 90% oxygen saturation on 100% non-rebreather. She

had jugular venous distention, bilateral apical wheezing, decreased breath sounds at the bases, and bilateral lower extremity pitting edema. She was treated for an acute systolic heart failure exacerbation with diuretics and placed on bi-level ventilation in the interim. The patient did not have any symptomatic improvement with diuresis.

The patient continued to have vigorous coughing spells, and on hospital day five she reported having sharp, pleuritic, left-sided chest pain radiating to her back. Her exam revealed a new focal bulge around the left parasternal area that was not tender to palpation and had paradoxical movement with respirations. A computed tomography image of the chest was obtained to evaluate both the continued dyspnea and the physical exam abnormality. It unexpectedly showed a diastasis between the 4th and 5th ribs where a 5.2 x 2.6 cm section of left upper lobe lung parenchyma had herniated (Figure 1). Adjacent to the defect was a metal clip from her cardiac surgery six years prior. Thoracic surgery was consulted, and a multidisciplinary decision was made to pursue medical management. She was discharged home on oral furosemide after further diuresis, with the plan to follow the lung herniation as an outpatient both with physical exam and computed tomography.

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Fig. 1: Computed tomography, axial view (Patient 1).

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Case 2

A 90 year old gentleman with a history of ischemia cardiomyopathy s/p coronary artery bypass grafting and biventricular ICD eight years prior presented to the emergency room with progressive dyspnea on exertion, cough, and occasional wheezing. Patient reported that his cough was bloody at times. He denied fevers, chills, or recent sick contacts. Chest x-ray was done and demonstrated pulmonary edema. Patient was started on diuretics, and his ICD battery was updated. Despite adequate diuresis, the patient continued to complain of shortness of breath and hemoptysis. Exam revealed an obese elderly man with a prior sternotomy scar and bilateral lower lobe crackles with occasional wheezing. There was mild lower extremity edema. A computed tomography scan was requested to evaluate his dyspnea and hemoptysis, and a defect in his left chest wall with a moderate sized lung herniation was detected along with evidence of a left sided pneumonia (Figure 2). A review of prior imaging, including computed tomography scans post coronary artery bypass, revealed that the current findings were new. A repeat physical exam of the chest wall again did not reveal a clear bulge or other abnormality. The patient reported that his coughing had been severe at the onset of symptoms, and that the coughing had preceded the shortness of breath and hemoptysis. A thoracic surgery consult was requested, and it was determined that surgery was not indicated at this time. The patient was treated for pneumonia with antibiotics, given cough suppressants, and felt improved enough to be discharged to rehab about seven days later. A repeat computed tomography of the chest approximately 6 weeks later showed almost complete resolution of the pneumonia and a significant improvement in the size of the lung herniation.

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Fig. 2: Computed tomography, axial view (Patient 2).

A left sided pneumonia with associated lung herniation.

Case 3

A 53 year old gentleman with a past medical history of ulcerative colitis and mitral valve repair with anterior right mini thoracotomy 3.5 years prior to presentation was hospitalized for a 2 week history of cough, shortness of breath, and hemoptysis. He required intubation and subsequent embolization of the right intercostal artery and endobronchial artery at an outside facility. He continued to have hemoptysis, and was transferred to our hospital for possible further surgical management since the persistent hemoptysis was thought to be related to lung incarceration. On physical examination by the surgeons the lung hernia was reducible, which precluded the need for emergent surgery. He had a bronchoscopy which localized bleeding to the right lower lobe, and patient was sent for a computed tomography of the chest with contrast to evaluate for a vascular source for the hemoptysis. Imaging revealed a significant necrotizing pneumonia in the right lower lobe, as well as an anterior lung herniation (Figure 3). Blood cultures subsequently grew *H. influenza*, and the patient was treated with antibiotics with subsequent resolution of the hemoptysis. He was successfully extubated, and recovered fully from his necrotizing pneumonia. A review of repeat imaging demonstrated significant reduction for the lung herniation after the treatment of the pneumonia.

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Fig. 3: Computed tomography, coronal view (Patient 3).

Right lower lobe pneumonia with parapneumonic effusion with associated lung herniation.

Discussion

To date, approximately 400 cases of lung herniation have been reported.³ The presentation of chest wall hernia can be variable from asymptomatic reducible bulge to severe pain associated with incarcerated ischemic tissue.⁴ In this case series, we demonstrate that remote surgical disruption of the intercostal muscles can result in continued chest wall weakness which when combined with elevated intrathoracic pressures can lead to delayed acquired lung herniation.

Herniation of the lung is usually classified as either congenital or acquired. The Morel-Lavelle classification system for lung hernias was developed in 1845, and this system divides lung hernias into anatomical and etiologic classifications.⁵⁶ Anatomical classification includes the division of hernias into cervical (15-35%), thoracic (60-80%), and diaphragmatic (2-5%). Etiologically, lung hernias are congenital in 20% of cases and acquired in 80%. The acquired cases are further split into pathological (18%), spontaneous (30%), and traumatic (52%). Spontaneous herniations are a consequence of body habitus, COPD and hyperinflation. However, the majority of causes are a result of trauma or are seen post surgery when the rib cage has been violated.

In order for an acquired lung hernia to develop there needs to be weakness in the thoracic wall and increased thoracic pressure. The weakness in the wall can be secondary to trauma or surgery, causing rib fractures and intercostal muscle tears. Increased intrathoracic pressure has been seen in patients with increased intraabdominal pressure, weight lifters, musicians, and patients with chronic cough.⁷ Overall risk factors for lung hernia also include body habitus, chronic obstructive lung disease and steroid use.⁸

Lung hernias usually present as soft, tender, subcutaneous masses that enlarge with coughing, straining, and Valsalva maneuvers.⁹ Apical lung hernias, which occurs as defect in Sibson's fascia, are the most rare, but need to be considered in cases of neck swelling, dysphagia, or cervical neuralgia from T1 compression.^{9,10}

Lung hernias are not thought to spontaneously resolve. Management is usually expectant, and increased size, pain, paradoxical respiration or signs of impending incarceration prompt surgical evaluation.⁹ Computed tomography of the chest is the most common and reliable method of diagnosis, and when surgery is considered allows the extent of the defect to be delineated.¹¹ Radiographically, intercostal lung hernias appear as hyperlucency beyond the rib structures that corresponds to air and parenchymal lung contents.¹³ This abnormality can be accentuated by having the patient perform the Valsalva maneuver during the imaging study.

Surgical repair of a hernia is termed herniorrhaphy, which can be performed either as an open procedure or video assisted minimally invasive surgery. Surgery is generally recommended to avoid the risk of strangulation of the hernia contents.¹⁴ Closure of the defect should generally be done with autologous tissue, and sometimes combined with biological glue.⁹ A variety of synthetic materials are acceptable substitutes when autologous tissue is not available.⁹ In addition, large defects may require biologic or prosthetic mesh coverage to avoid tension on the repair and provide structural integrity.¹⁵ If there is inadequate muscle coverage of the defect, soft tissue coverage with rotational muscle flaps and omentum may be used.¹⁵ In chronic herniation, the adjacent thoracic muscles are often fixed and provides little mobility. This is especially true in the elderly and patients with healed displaced rib fractures. In these cases, prosthetic mesh is used to provide scaffolding for a developing fibrous capsule.⁴ Wien *et al* have described a surgical technique to repair lung hernias using spinal instruments in which they use laminar hook fixation to utilize the strength of the bony chest wall to prevent recurrence.¹⁶

It is expected that as minimally invasive cardiac surgery becomes more common there will be an increase in lung herniations, as in our third case. Minimal-access thoracotomy frequently involves a longer intercostal incision than skin incision.¹⁷ This forces surgeons to place pericostal closure sutures under intact skin, which can be difficult when the skin incision is small.¹⁷ In robotic assisted valve repairs, lung herniation has been reported at the minithoracotomy incision to accommodate the robotic arm.¹⁸ If costal cartilage needs to be removed for better visualization during surgery, it has been suggested that it is prudent to perform primary reconstruction in patients with COPD as they are at high risk for lung herniation.¹⁹

Most lung herniations due to surgical intervention described in the literature occurred weeks to months in the post-operative period, and our cases occurred many years post surgery. Interestingly, review of imaging from our second case reveals that lung herniation can present many years post surgery in situations where imaging post surgery had been normal, indicating that the concomitant effect of increased intrathoracic pressures is

needed for herniation to develop (perhaps even at a prior chest tube site) . In addition, we demonstrate that although lung herniation may not resolve it can dramatically improve when the patient is not in distress (as evidenced in the second and third cases).

In conclusion, lung herniation is an entity to be aware of in our chronic lung disease patients, especially with the known increased risk of cardiac comorbidities which may require these patients to undergo cardiothoracic surgery. Although management is expectant for the majority of patients, there is a small subset that will require surgery should complications arise.

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