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Case report

Trapped lung secondary to cardiomegaly in a 78 year-old male with congestive heart failure

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ABSTRACT

Although the etiologies of both trapped lung and cardiomegaly are well-established, co-presentation of the two conditions, and possible interactions between them, are much rarer. Here we describe the case of 78 year-old male found to have both cardiomegaly and trapped lung, with a cause of death of congestive heart failure and subsequent cardiac arrest. This case prompted consideration of possible interactions between the two conditions. Issues related to decision-making for imaging and clinical interventions are also discussed.

1. Introduction

A trapped lung, or fibrothorax, is a shrunken lung surrounded by a cortex of fibrotic visceral pleura. This fibrotic peel prevents inflation of the lung, and is typically caused by a chronic inflammatory process leading to uncontrolled fibrin deposition. Although it is a known sequela of recurrent pleural effusion, other disease processes, such as malignant or metastatic visceral pleural disease, can also lead to trapped lung [1,10,14,18]. Cardiomegaly is defined as an increase in heart size caused by ventricular hypertrophy or chamber dilation such that the ratio of heart diameter to maximum thorax diameter, measured transversely, is greater than 0.5 [24]; [25].

Although neither trapped lung nor cardiomegaly is considered unusual, simultaneous presentation of both conditions, or a causal connection between them, is virtually unmentioned in the literature. Furthermore, both conditions can be difficult to diagnose with a plain chest radiograph, which is the most common imaging ordered for patients with dyspnea. Failure to recognize and identify trapped lung can lead to unnecessary interventions such as repeated thoracenteses, or can delay more appropriate interventions such as surgical decortication.

2. Case report

During routine dissection of a 78 year-old male cadaver we found a trapped left lung and enlarged heart almost completely occupying the left pleural cavity (Fig. 1). The subject's past medical history included congestive heart failure (CHF), chronic pleural effusion with prior thoracenteses, atrial fibrillation, non-sustained ventricular tachycardia, stage III kidney disease, hypertension, chronic anemia, cardiac pacemaker placement, and hydrocephalus with ventriculoperitoneal shunt placement. Cause of death was given as cardiac arrest and CHF.

One week prior to death, our subject was admitted to the hospital with complaints of shortness of breath and diffuse edema, and was given primary diagnoses of anasarca and CHF exacerbation. At that time his vital signs were normal, and he required 2 L of oxygen by nasal cannula to maintain an oxygen saturation of 95%. Multiple chest radiographs taken during that hospital stay (Fig. 2) showed “persistent subtotal opacification of the left hemithorax,” and “massive left pleural effusion with minimal aeration of the left lung [and] at least mild cardiomegaly.” The subject then underwent thoracentesis to remove the fluid in his left hemithorax, and a repeat radiograph showed “persistent opacification” of the left hemithorax, and noted that “cardiac and mediastinal contours are difficult to evaluate due to adjacent opacification.”

At the time of our dissection, the subject's right lung appeared

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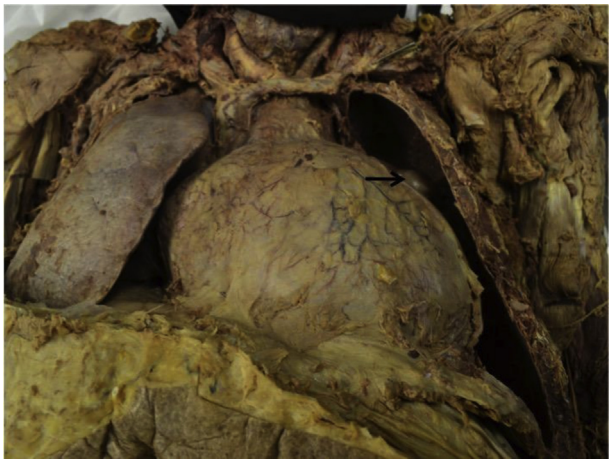

Fig. 1. Anterior view of lungs and pericardial sac after removal of the anterior chest wall. Note the tip of superior lobe of left lung protruding over the left contour of the heart (arrow).

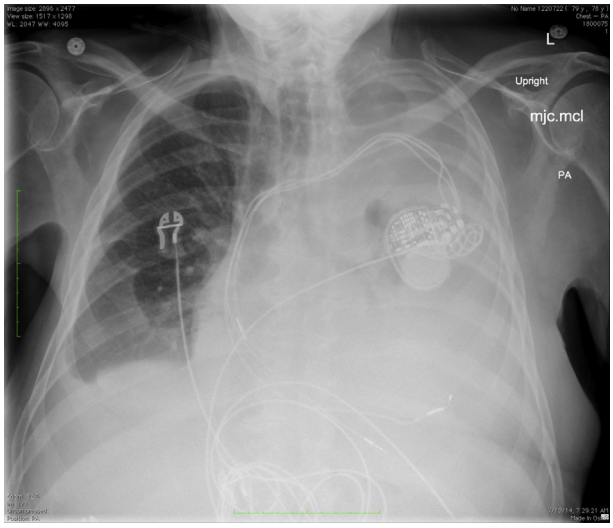

Fig. 2. Chest radiograph taken during the week before death. Note opacification in left pleural cavity which limits visualization of the heart contours and the trapped lung.

normal in dimensions and gross appearance. The left lung was deformed, and markedly diminished compared with the right lung, particularly in the lower lobe (Fig. 3). The thickness of the cortex around the lung varied from 0.5 to 1.5 mm. Subject presented with cardiomegaly, with a cardiothoracic ratio of 0.54. The right ventricular wall, interventricular septum and left ventricular wall measured 240%, 255%, and 326% of values for healthy adults, respectively (Fig. 4), indicating substantial biventricular hypertrophy [11,15]. Comparing ventricular wall thickness values can be challenging because norms are based on end-diastolic measurements, and significant regional variations exist in the thickness of any individual ventricle [5]. Wall thickness has been shown to be greatest during systole; however, the maximum differences in ventricular thickness measured at diastole and systole have been reported to average less than 62% [23]. Given this fact, the measurements in the present subject would appear to represent substantial hypertrophy regardless of the cardiac phase at the time of death.

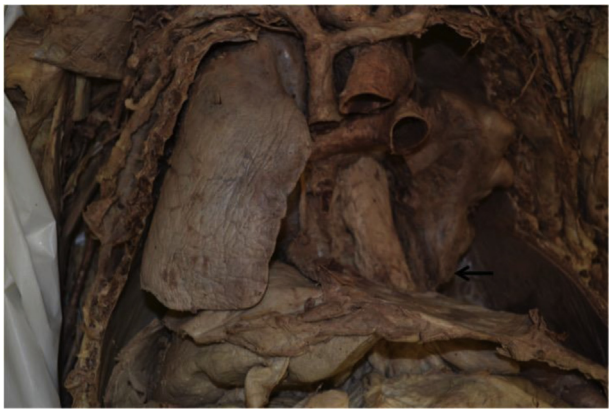

Fig. 3. Anterior view of the chest after removal of the heart, showing deformed and fibrotic left lung. Note diminutive left lower lobe (arrow).

Fig. 4. Heart opened to show hypertrophy of ventricular walls and interventricular septum.

3. Discussion

3.1. Etiology and clinical course of trapped lung and cardiomegaly

Trapped lung causes a hydropneumothorax, with both fluid and air present in the pleural cavity. Signs and symptoms of trapped lung include shortness of breath, an absence of breath sounds on the affected side and, in some cases, hypoxemia [1,8]. Diagnosis is made by clinical exam, chest xray and computed tomography (CT). Analysis of the pleural fluid is done to differentiate malignant from inflammatory or infectious processes. Although ultrasound guidance can greatly enhance the accuracy of thoracentesis for pleural effusion, it is not considered a reliable imaging modality to identify a trapped lung [7]; [18].

Chronic pleural effusion is a known cause of trapped lung. In the early stages of pleural effusion, before the development of the fibrotic cortex, lung expansion is limited only by the pleural fluid build-up separating visceral from parietal pleura. Thus, when the fluid is removed by thoracentesis, the lung is able to fully re-expand. Over time, however, the thickening fibrotic coat on the visceral pleura prevents the re-expansion of the lung, even after