Case Report

Herniation of unruptured tuberculous lung abscess into chest wall without pleural or bronchial spillage

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ABSTRACT

A 22-year-old unmarried man presented to the chest outpatient department with a history of productive cough of two-month duration. He also complained of pain and swelling on the anterior aspect of right side of chest of one-month duration. Imaging studies of the thorax, including chest roentgenography and computerized tomography, revealed an unruptured lung abscess which had herniated into the chest wall. Culture of pus aspirated from the chest wall swelling grew *Mycobacterium tuberculosis*. He was diagnosed to have a tuberculous lung abscess which had extended into the chest wall, without spillage into the pleural cavity or the bronchial tree. Antituberculosis drugs were prescribed, and he responded to the treatment with complete resolution of the lesion.

KEY WORDS: Herniation, lung abscess, tuberculosis

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INTRODUCTION

Lung hernia is protrusion of lung tissue beyond the thoracic cavity through an anomalous opening in the chest wall, diaphragm, or mediastinum lined by pleura. The lung hernia can be located in the cervical, thoracic, or diaphragmatic regions. Based on etiology, lung hernias can be classified as congenital or acquired. The majority (82%) falls into the latter category and of these, only 1% is due to tuberculosis, inflammation, or neoplastic disease.[1] Review of literature has revealed only six cases of tuberculous pulmonary lesions, which included either a cavity or a mass, herniating into the chest wall without generalized involvement of the pleural cavity.[2-7] However, an unruptured tuberculous lung abscess extending into the chest wall without spilling into the pleural cavity or the bronchial tree is uncommon. We are reporting such a case which responded to antituberculosis chemotherapy.

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CASE REPORT

We are reporting the case of a 22-year-old unmarried male waiter who presented to the chest out patient department with a history of cough with scanty whitish expectoration of two-month duration. He also complained of pain and swelling on right side of the anterior aspect of chest of one-month duration. He had loss of appetite and loss of weight of 5 kg over two months. There was no history of trauma or tuberculosis. He did not smoke or consume alcohol. On general physical examination, patient was moderately built and nourished, afebrile, pulse rate was 72/min, regular and good volume, respiratory rate was 15/min, and blood pressure was 124/74 mmHg. There was no significant lymphadenopathy. Chest examination revealed an ill-defined, smooth, nontender, soft, fluctuant swelling of 4 × 4 cm size located close to the sternum overlying the right second and third intercostal spaces. The swelling increased in size on coughing and on performing Valsalva maneuver. It could be reduced partially by manual pressure. The signs of inflammation, such as redness and increase in local temperature, were absent. Right infraclavicular and mammary areas had reduced respiratory movement, percussion note was dull, and breath sounds were diminished on auscultation. Examination of abdomen and other systems did not reveal any abnormality. Hemogram showed hemoglobin of 11.4 gm/dl; total leukocyte

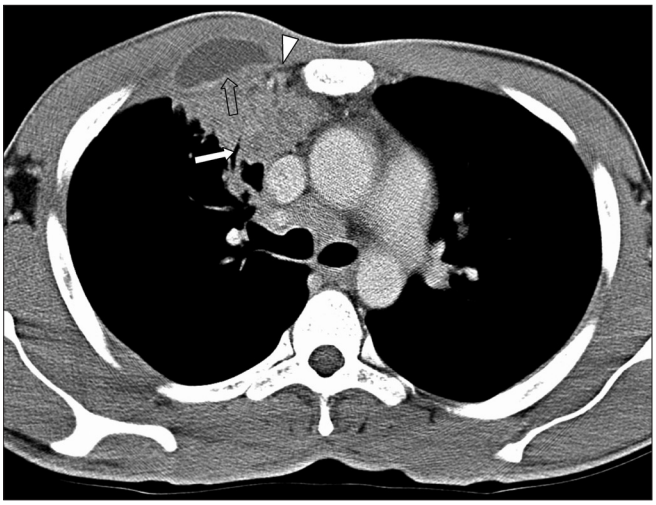

Figure 1: CECT of thorax reveals necrotic areas and small bronchi within the lesion (solid arrow), a contiguous abscess in the chest wall (hollow arrow), and internal mammary vessels which are seen separate from the lesion (arrowhead)

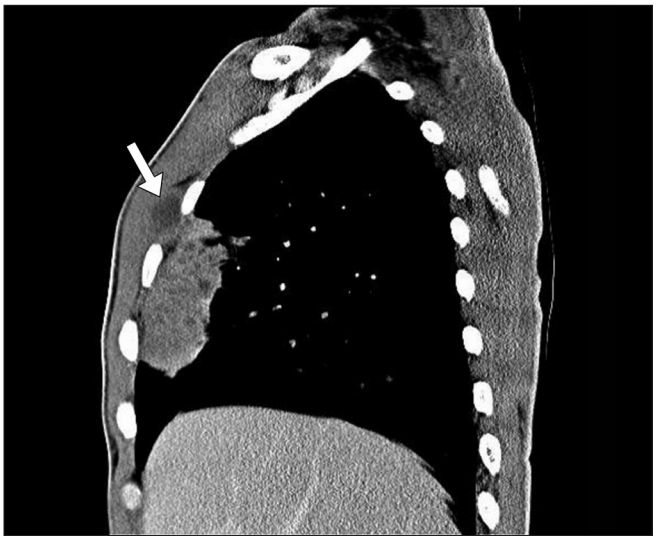

Figure 2: Sagittal reconstruction image of thorax demonstrates extension of the lesion into the chest wall (arrow)

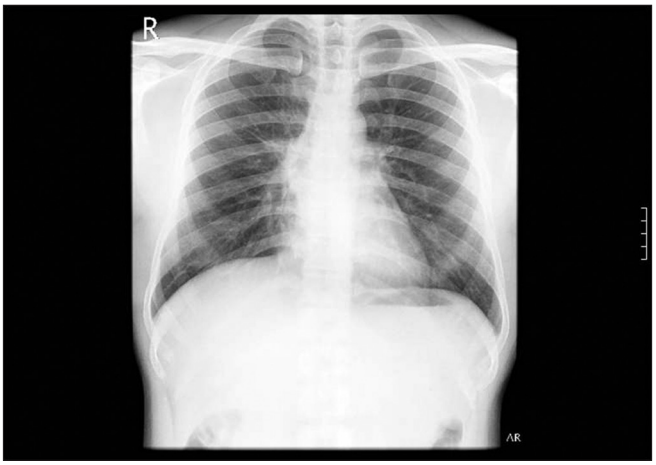

Figure 3: Chest radiograph PA view shows almost complete resolution of the lesion six months after chemotherapy

count, 6200/mm^3; neutrophils, 76%; lymphocytes, 22%; and eosinophils, 2%. Platelet count was 433000/mm^3. Erythrocyte sedimentation rate was 103 mm in the first hour. Routine urine analysis was normal. Renal function tests, liver function tests, fasting blood sugar, and serum electrolytes were within normal limits. Serological test for human immunodeficiency virus was negative. Three sputum smears were negative for acid fast bacilli. Chest roentgenogram revealed an ill-defined homogenous opacity in the right paracardiac region. Computerized tomography (CT) of thorax revealed a consolidation with necrotic areas suggesting lung abscess, located in the right upper and middle lobes, which was extending into the chest wall but with no evidence of pyothorax [Figures 1 and 2]. Fiberoptic bronchoscopy was normal. Smear of the pus aspirated from the chest wall swelling was positive for acid fast bacilli. Culture of the pus grew *Mycobacterium tuberculosis*. He was prescribed an antitubercular regimen containing isoniazid, rifampicin, pyrazinamide, and ethambutol for first two months, followed by isoniazid and rifampicin for next four months in standard doses. After starting treatment, the swelling reduced in size, and the symptoms completely resolved by the end of six months of antitubercular chemotherapy. Repeat chest roentgenogram taken at that time showed almost complete resolution of the lesion [Figure 3].

DISCUSSION

Chest wall tuberculosis can occur as a result of bloodstream spread, or a contiguous spread from adjacent pleura or lung.[7] In the present case, the patient at first developed productive cough, and only later the chest wall symptoms. CT thorax shows small bronchi entering lesion and the bulk of the lesion (epicenter) is within the lung, with only a small component being in the chest wall. Internal mammary vessel fat planes are preserved and there is no displacement of vessels, excluding the possibility of anterior mediastinal or internal mammary nodes [Figure 1]. All these findings suggest that the lesion had originated from the lung rather than extraparenchymal structures.

The tuberculous lung abscess must have evoked an inflammatory response in the adjacent pleura leading to adhesion between visceral and parietal pleura in that localized area. Hence, this area would have been sealed off from the rest of the pleural cavity, thus allowing the abscess to extend into the chest wall without spilling into the pleural cavity. As has been hypothesized in an earlier case report, the intercostal muscles would have been weakened by the advancing inflammation and the vigorous coughing, acting as the driving force, would have pushed the lung abscess to herniate into the chest wall after traversing the pleura.[8] On the anterior aspect of the chest wall, weakness usually occurs from costochondral junction to sternum due to absence of external intercostal muscle.[1]

In the present case, the lesion had no communication with the bronchus and this is evident from the fact that there was no air fluid level seen on imaging studies and also that the sputum smears were negative for acid fast bacilli. In the earlier case reports, the tuberculous pulmonary lesions, which herniated into the chest wall, had at some point of time communicated with the bronchial tree and this resulted in air being present in the lesions.[2-6] Case report of chest wall herniation of a nontuberculous lung abscess also had bronchial communication.[8] Such a communication would act as an escape route for the pus and relieve the pressure within the lesion. Hence, this would further reduce the chance of pleural spillage of the contents present inside the herniating lesion. However, in the present case, an intact tuberculous lung abscess, with no such bronchial communication, crossed the pleural cavity without spilling its contents. The tuberculous mass lesion of the lung, as reported by Ahmed *et al*., also did not have such a bronchial communication, but contrary to our case report, there was no liquefied material present within the lesion.[7]

We are reporting this case of chest wall extension of an unruptured tuberculous lung abscess, because such a complication without pleural and bronchial spillage is an uncommon occurrence. By using imaging modalities, such as chest roentgenography and CT of the thorax, the lesion was easily characterized and its anatomical location verified. Though the lesion had originated in the lungs, the sputum and bronchial aspirate smears were negative for acid fast bacilli. The etiological diagnosis was confirmed only by smear and culture of the pus aspirated from the chest wall lesion. The medical management with antituberculosis drugs alone seems to be sufficient for the resolution of the lesions.

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