Sir,

Trauma and iatrogenic injury are the most common causes of subcutaneous emphysema. Subcutaneous emphysema (with or without pneumothorax or pneumomediastinum) that occurs spontaneously is less frequently seen. It occurs as a result of a breach in either the airway or the alveolar lining following which the air tracks through bronchovascular sheaths (Macklin effect) to reach the mediastinum and then into the subcutaneous spaces of the neck through the thoracic inlet. Spontaneous subcutaneous emphysema occurring as a result of a rupture in the chest wall is rare.

A 60-year-old gentleman presented with swelling of the face, neck, and chest for 3 days following a bout of cough. He also had fever, productive cough, and chest pain for 2 months. About 6 months earlier, he was advised antituberculosis treatment (ATT) for sputum-positive pulmonary tuberculosis, that he discontinued prematurely after 1 month. Examination on presentation revealed subcutaneous emphysema involving the face, neck, and chest with reduced breath sound intensity bilaterally. A part of the left anterior chest wall showed paradoxical movement, i.e., bulging on expiration and drawing in during inspiration (supplemental video). Computed tomography (CT) of the chest revealed multiple cavities with fibrosis in the entire right lung and left-sided hydropneumothorax [Figure 1a]. The left thoracic cavity was communicating with the subcutaneous space resulting in surgical emphysema [Figure 1b]. An intercostal drainage tube (ICD) was placed in the left pleural cavity. Examination of the sputum and pus (from the chest drain) revealed the presence of acid-fast bacilli. Subsequently, both the specimens showed growth of Mycobacterium tuberculosis, sensitive to all first-line antituberculosis drugs. The cultures sent for bacteria were sterile. Enzyme-linked immunosorbent assay performed for the human immunodeficiency virus was negative in the serum. The patient was advised to undergo surgical repair of the defect that he refused. He was started on four-drug ATT (rifampicin, isoniazid, pyrazinamide, and ethambutol). The patient’s fever abated in 2 weeks and the pus was completely drained. After 4 weeks, the ICD was removed once the daily drainage decreased to less than 30 mL of clear fluid.

Tuberculous empyema signifies a chronic, active infection of the pleural space that contains numerous tubercle bacilli. It is encountered much less frequently than tuberculous pleural effusions that are paucibacillary and occur due to a type IV hypersensitivity reaction to the proteins of M. tuberculosis. Uncommonly, tuberculous empyema can cause contiguous involvement of the thoracic cage resulting in destruction of the bones and soft tissue of the chest wall. This leads to its evolution into empyema necessitans that is defined as a collection of inflammatory tissue that ruptures spontaneously through a weakness in the chest wall into surrounding soft tissues. Our patient had a tuberculous empyema that ruptured through the chest wall and also led to air dissecting through the subcutaneous space.

Spontaneous subcutaneous emphysema has previously been described in association with pulmonary tuberculosis. The index patient not only had chest wall destruction and spontaneous subcutaneous emphysema due to pulmonary and pleural tuberculosis but also a chest segment with paradoxical motion akin to a flail chest. In the current era of improved diagnostic and therapeutic modalities for tuberculosis, such manifestations of the disease are still encountered due to delayed diagnosis and poor compliance with ATT.

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Figure 1: (a) Computed tomography of the thorax showing subcutaneous emphysema (asterisk) and hydropneumothorax on the left side; the right hemithorax shows pulmonary cavities and fibrosis (b) Computed tomography of the thorax shows a defect in the left anterior chest wall (white arrow) demonstrating a communication between the thoracic cavity and subcutaneous space

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pulmonary tuberculosis and was on antitubercular treatment for a month without clinical and radiological response. On examination, vital parameters were stable, and there was no peripheral lymphadenopathy. Respiratory examination revealed an impaired percussion note in right suprascapular area with crackles. Other systemic examinations were normal.

Laboratory analysis revealed white blood cell count 8400/cumm (neutrophils - 71, lymphocytes - 25, eosinophils – 01, and monocytes - 03), hemoglobin - 13.0 g%, and an elevated ESR of 23 mm/h. Other routine biochemical parameters including bleeding and clotting parameters were within normal limits. Serological markers for human immunodeficiency virus and hepatitis virus (B and C) titers were negative. Collagen vascular disease workup was negative. His sputum for acid fast bacilli (AFB) by Ziehl–Neelsen stain was negative. Ultrasonogram of the abdomen revealed a normal scan. Chest X -ray showed right-sided upper zone opacity [Figure 1a]. The computerized tomography (CT) of the chest revealed well -defined, smoothly marginated, and solid mass lesion [Figure 1b] in the right upper lobe which showed peripheral contrast enhancement with small nonenhancing areas [Figure 1c]. There were incidental emphysematous changes in the rest of the lungs [Figure 1d lung window]. There were associated right tiny paratracheal and hilar lymphadenopathies. Bronchoscopy showed mucosal irregularity in right upper lobe bronchus. Bronchial lavage and brush cytology were

Dear Sir,

Castleman's disease is a rare disorder characterized by two histopathological varied subtypes but differs in their clinical symptoms, progression, and response to therapy. Most often this disease has predilection for the lymph nodes in sites of the neck, mediastinum, and abdomen where the nodes conglomerate in the disease process. Parenchymal lung involvement of the disease is exceedingly rare. [1] Angiofollicular or giant lymph node hyperplasia was first described in 1954 by Benjamin Castleman as a cause of mediastinal lymphadenopathy. [2] The disease has been classified on clinical profile as localized or multicentric Castleman's disease (MCD) and on pathological findings as a hyaline vascular pattern (HV) 90% of cases, plasma cell predominance (PC) 8–9%, and a mixed variant 1–2% of cases. [3] The etiology is largely unknown, but it is proposed to be due to antigenic hyperstimulation of unknown origin. Definitive diagnosis is achieved mainly by histopathological analysis as clinical and radiological features overlap and are nonspecific. [4] A 51-year-old heterosexual male of Caucasian origin, a chronic smoker since 15 years presented to our hospital with a history of chronic cough, episodic chest pain, and dyspnea of 3 months duration. The patient had no other comorbid illness. The patient had a history of being treated for pneumonia 6 months back with a course of parenteral antibiotics. He was diagnosed with smear negative Multicentric Castleman's disease: “A rare entity that mimics malignancy”