CHEST

Right Middle Lobe Collapse and Pleural Effusion in an 18-Year-Old Man

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An 18-year-old African American male subject presented to an acute care clinic with 3 days of productive cough, chills, pleuritic right chest pain, sore throat with hoarseness, congestion, and intermittent shortness of breath. He recently relocated to Texas from Georgia to undergo basic military training. He denied any other recent travel or contact with persons with pulmonary TB or other respiratory illnesses. His medical history was significant for glucose-6-phosphate dehydrogenase deficiency and sickle cell trait. CHEST 2017; 152(2):e33-e38

Case Report

Examination revealed a well-nourished male subject with reduced air entry into the lower right hemithorax. Initial vital signs were normal with room air oxygen saturation of 98%. A chest radiograph (Fig 1) showed a large right pleural effusion with possible consolidation.

The patient was transferred to our medical facility for further evaluation. A CT scan (Fig 2) confirmed a large right effusion with right lower lobe (RLL) and right middle lobe (RML) consolidations with patchy airspace disease of the right upper lobe. Massive right hilar, infrahilar, mediastinal calcific lymphadenopathy and splenic calcifications (Fig 3) were noted.

Thoracentesis with subsequent pigtail chest catheter placement revealed an exudative pleural effusion. Results of the bacterial, mycobacterial, and fungal cultures of the pleural fluid were negative. Adenosine deaminase and interferon gamma release assay for TB were negative. Urine and serum *Histoplasma* antigen were negative and equivocal, respectively. Serum immunoglobulin levels, including IgG4, were within normal limits.

Bronchoscopy revealed extrinsic compression of the bronchus intermedius with complete obstruction of the RML bronchus. Endobronchial stenting was considered but was not feasible. Results of the endobronchial ultrasound lymph node biopsy were negative for malignancy, sarcoidosis, and fungal infection. BAL

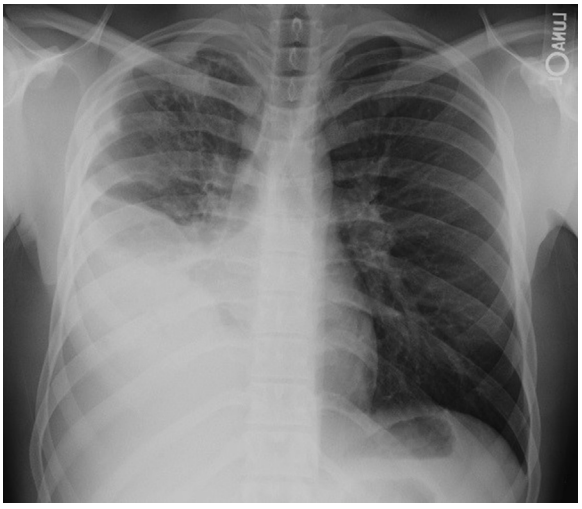

Figure 1 – *Posteroanterior radiograph shows findings show dense opacification of the right lower hemithorax associated with shift of the heart to the right side, findings consistent with likely combination of effusion and underlying basilar atelectasis.*

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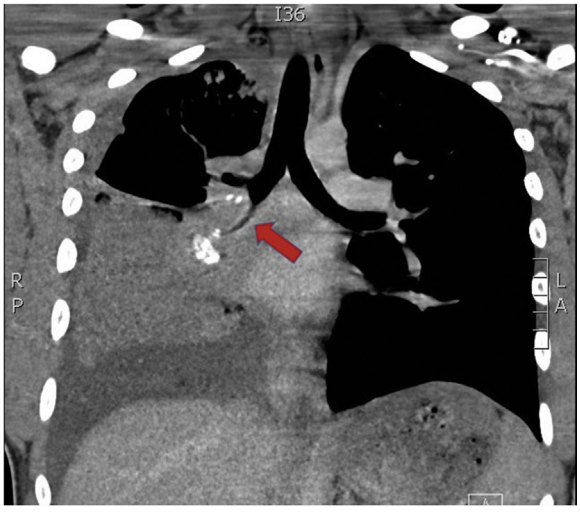

Figure 2 – *Contrast-enhanced coronal CT image at the level of the carina shows combination of large right-sided effusions and diffuse opacification/atelectasis of the corresponding lower lung field lobe confirming shift of the heart to the right (Fig 1). There are right hilar calcifications, including bulky calcified nodes causing marked narrowing/obstruction of the bronchus intermedius (arrow). Ill-defined nodular densities are also noted in the right upper lobe/apex.*

cultures were negative for fungi, bacteria, and mycobacteria.

Secondary to the complex fluid collection and RML/RLL collapse from marked lymphadenopathy of unclear etiology, surgery was suggested. Intraoperatively, dense adhesions and a thick inflammatory pleural rind covered the RLL and diaphragmatic surface. The dissection planes at the lung hilum were obliterated due to a baseball-sized conglomerate of lymph nodes extending from the subcarinal space along the bronchus intermedius into a group of lymph nodes that receive drainage from all three lobes, known as the sump of Borrie. RML/RLL bi-lobectomy was required given the extent of disease.

Gross pathologic examination of the bi-lobectomy specimen revealed a 5.5 × 4.5 cm white, firm, mass-like proliferation centered in the hilum. In addition, 1- to 3-mm nodules were noted throughout the lung and mediastinal lymph node samples.

Histologic evaluation of the large mass-like area revealed dense collagenous fibrosis with intermixed variably dense inflammatory cells, including lymphocytes, plasma cells, histiocytes (including rare multinucleated cells), and eosinophils. Areas of caseating necrosis with microcalcifications were surrounded by fibrosis (Fig 4). The fibrosis encompassed large hilar vessels and abutted the bronchus. The smaller nodules exhibited fibrosis with caseating necrosis and microcalcifications. A Grocott methenamine silver (GMS) stain highlighted scattered, thin-walled yeast-like organisms ranging 3- to 5-μm within areas of necrosis (Fig 5).

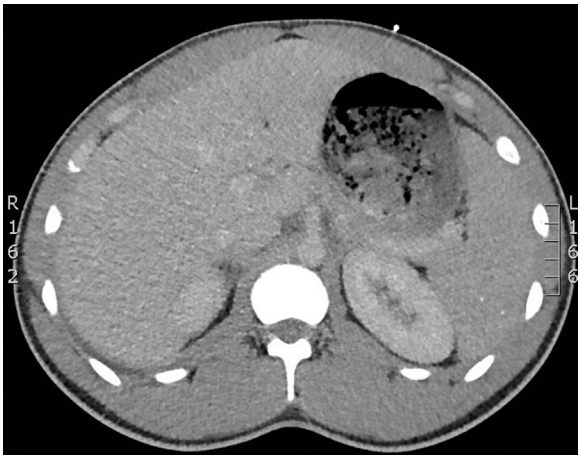

Figure 3 – *There are subtle punctate calcifications in the spleen that is otherwise normal in size. There is no evidence of ascites.*