An unusual case of pleural effusion: Hard to diagnose and harder to manage

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Abstract

Pleural disease is one of the common respiratory condition affecting ~3000 people per million population yearly. Pleural effusion has several fundamental aetiological conditions and therefore needs a systematic assessment to reach a concluding diagnosis. In spite of accurate evaluation, there may be situations, where the aetiology of a pleural effusion remains unknown. Various professionals have suggested a step-wise path in the management of these undiagnosed pleural effusions. The role of detailed history, suitable clinical examination and appropriate investigations, including Computed Tomography (CT) of chest and pleural biopsy, in an experiment to approve the correct cause of pleural effusion cannot be over emphasized. We present an engaging case of pleural effusion that was managed at our institute.

Keywords: Pleural effusion; Investigation; Experiment; Management

Introduction

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Lung carcinoma is one the major cause of extinction worldwide. Small-Cell Lung Cancer (SCLC) includes about 10%-15% of cases of all lung cancer. It commonly affects males more commonly than females and has smoking as a high-risk factor. It generally presents with mass effects such as cough, wheezing, superior vena cava syndrome and hemoptysis and also leads to paraneoplastic syndrome [1].

It is uncommon for SCLC to exhibit as isolated massive pleural effusion. Here in, we characterize a case of massive pleural effusion which was diagnosed as small cell carcinoma with thoracoscopic pleural biopsy, and was managed with pleurodesis and cisplatin-based chemotherapy.

The main signs and symptoms of thoracic actinomycosis were cough expectoration, blood-stained sputum, hemoptysis, fever, and chest pain. Radiographically, thoracic actinomycosis may need either lung and show several cavitary lesions.

A mass lesion or pneumonia with or without pleural development is familiar and pleural thickening, effusion, or empyema is found in more than 50% of the cases. If not, an isolated pleural effusion is an especially rare clinical presentation of thoracic actinomycosis. The previously reported cases represent the distinct clinical features of an abonded pleural effusion in thoracic actinomycosis, but the cases were normally associated with structural injury or thoracic surgery [2].

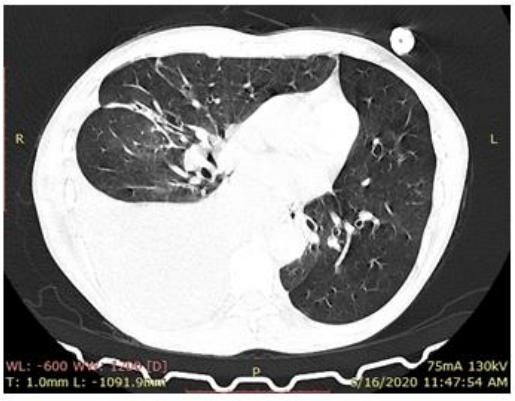
Case Report

Pleural effusion secondary to liver cirrhosis and ascites is well known, but hepatic hydrothorax without ascites is rarely reported. Here we report such an unusual case of hepatic-hydrothorax without ascites with concurrent diagnosis of liver cirrhosis on fibro scan [3-5]. A 42 year old male presented in emergency department with chief complaint of progressively increasing difficulty in breathing (mMRC grade 4) for last 3 weeks associated with easy fatigability and right sided dull chest pain. Past history was significant for Chronic Obstructive Pulmonary Disease (COPD), diabetes and hypertension. Patient was chronic smoker (smoking index 400) and had history of regular alcohol consumption for last 20 years.

Results and Discussion

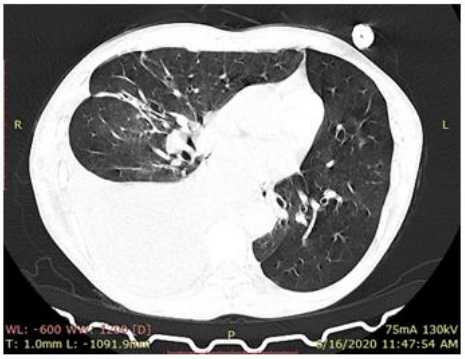
On systemic examination, patient was conscious, oriented but anxious, tachypneic and using accessory muscles of respiration; vitals were HR-130, Sp02-96 (on oxygen support at 6 lit/min), RR-28, B.P-140/90 mmHg and temperature 98.4 F. On chest examination, decreased movements, stony dull percussion note and absent breath sounds were evident on lower one-third of right hemithorax (Figure 1).

Figure 1: *Axial CECT chest images in mediastinal and pulmonary windows show right sided pleural effusion. Underlying bilateral lung parenchyma is normal.*



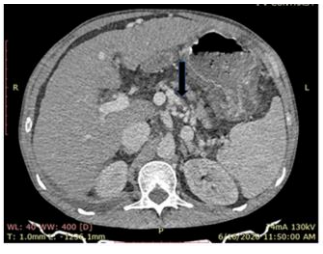
Rest of chest had bilateral diffuse polyphonic rhonchi. ABG showed mild respiratory acidosis with hypoxemia. Patient was started on empirical antibiotics, systemic steroids, inhaled bronchodilators, PPI's, O_2 inhalation with intermittent NIV on lines of provisional diagnosis of acute exacerbation of COPD. Initial laboratory investigations revealed total leukocyte count 9800 with neutrophil predominance 80%, Hemoglobin-10.1 gm/dl, Platelet 80,000/ml, SGOT/PT 72/73U/L, total Bilirubin 3.5/dl, Alkaline phosphatase 142U/L, total protein 6.2 gm/dl, albumin 3.2 gm/dl, globulin 3.0 gm/dl, blood urea 28 mg/dl and Creatinine 0.9 mg/dl. Routine viral markers showed hepatitis C positivity. Chest radiograph showed moderate right sided pleural effusion [6-8]. Patient underwent CT chest that showed patchy areas of GGO's in right lung, moderate right pleural effusion and mild hyperinflated lungs. Serum amylase and lipase were obtained within normal range. ECHO showed no evidence of cardiac disease with LVEF=56%. Ultrasound guided thoracocentesis was performed. Pleural fluid obtained was straw colored, protein=0.5 gm/dl, sugar-192 gm/dl, ADA-14, total cells-2500/dl, lymphocytes=60% and negative gram stain, culture sensitivity, acid fast bacilli stain and CBNAAT (Figure 2).

Figure 2: *Underlying bilateral lung parenchyma is normal.*



On day 3 of admission, Inter Costal Drain (ICD) had to be placed in view of rapidly progressing pleural effusion and respiratory distress. Repeat pleural fluid examination again revealed transudative effusion only. Ultrasound abdomen showed coarsened liver echo texture and portal hypertension (portal vein diameter 13 mm), splenomegaly, right sided pleural effusion and no free fluid in abdominal cavity. Fibroscan was done to measure liver stiffness, which showed a value of 63.9 kPa suggestive of liver cirrhosis. The diagnosis was confirmed as hepatic hydrothorax without ascites. Patient was started on sodium restricted diet and diuretics (furosemide and spironolactone). Upper GI endoscopy revealed esophageal varices. Diuretics dose was upstaged gradually to 160 mg furosemide and 400 mg spironolactone, but pleural fluid drainage continued although reduced in amount from about 1500 ml to 500 ml per day. Betadine chemical pleurodesis and octreotide infusion was tried but failed. In last patient was listed for liver transplantation and referred for Transjuglar Intrahepatic Porto Shunt (TIPS) as bridge therapy to liver transplantation. Further follow up of patient is awaited (Figure 3).

Figure 3: *Axial CECT upper abdomen section shows chronic liver parenchyma with multiple porto systemic collaterals (arrow).*



Pleural effusions secondary to liver cirrhosis are usually notorious to manage. Most of these patients need liver transplantation but many are not candidates due to poor health or long waiting periods for liver transplantation. While waiting for definite treatment, salt restriction and diuretics remains the mainstay of therapy. Diuretics doses needed for hepatic hydrothorax is usually higher than needed for ascites only. Chest tubes in hepatic-hydrothorax carry significant morbidity and mortality with doubtful benefit; hence not preferred. Repeated thoracocentesis can be performed for symptomatic relief.

In our case intercostal tube had to be placed for rapid increasing pleural effusion causing further deterioration of obstructive lung disease. Pleurodesis usually fails due to rapid rate of re-accumulation of fluid although have been tried with some success. Octreotide infusion and indwelling pleural catheters have also been have been tried in some cases. Video Assisted Thoracoscopic Surgery (VATS) or thoracotomy guided decortication and chemical pleurodesis may yield positive results but are associated with significant morbidity and mortality. Transjugular Intrahepatic Portosystemic Shunt (TIPS) is an effective therapy acting as bridge to liver transplantation.

Conclusion

If a patient without history of liver disease has unclear cause of transudative pleural effusion, we might consider adding hepatic hydrothorax without ascites, as differential diagnosis. The management of hepatic hydrothorax remains challenging and most of patients need liver transplantation.

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