

Hepatic Hydrothorax and Complex Tuberculous Pleuritis-Contarini's Condition in a Patient with Hepatitis C-related Chronic Liver Disease

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ABSTRACT

Contarini's condition refers to the presence of bilateral pleural effusions with distinctly different biochemical characteristics, indicating their separate aetiologies. A 44-year male with hepatitis C-related chronic liver disease (CLD) presented with a one-month history of dyspnoea, anorexia, and fever. Imaging and biochemical studies revealed bilateral pleural effusions with contrasting features: A free-flowing right-sided transudative effusion (hepatic hydrothorax) and a large left-sided exudative effusion with loculations and pleural thickening. Pleuroscopic examination of the left pleural surface and histology confirmed tuberculous pleuritis. This case highlights the importance of diagnostic thoracentesis and imaging studies in identifying Contarini's condition, particularly in patients with underlying CLD.

Key Words: Exudate, Hepatic, Pleural effusion, Tuberculosis, Transudate.

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INTRODUCTION

Contarini's condition is a rare entity characterised by bilateral pleural effusions with distinct differences in their biochemical composition and radiological characteristics. This phenomenon is named after the 95th Doge of Venice, who died of cardiac decompensation with a unilateral transudative pleural effusion and a contralateral empyema as a complication of pneumonia.¹ Hepatic hydrothorax typically occurs with ascites, but it can also present without ascites when fluid from the peritoneal cavity migrates to the pleural space due to negative pleural pressure.² The condition has a poor prognosis, is associated with significant morbidity and mortality, and has a median survival rate of approximately 8-12 months.³ A rare case of Contarini's condition is presented, which was characterised by bilateral pleural effusions of differing aetiologies (right hepatic hydrothorax and left complex tuberculous pleuritis), in a middle-aged male with hepatitis C-related CLD, highlighting the importance of diagnostic thoracentesis and imaging studies in identifying this complex condition.

CASE REPORT

A 44-year male presented with a one-month history of progressive exertional dyspnoea, dry cough, and low-grade pyrexia. He had a background of hepatitis C-related CLD, portal hypertension, and moderate ascites. Physical examination revealed signs of CLD, including palmar erythema, bilateral gynecomastia, and atrophied testes, along with dull percussion and absent breath sounds over the left haemithorax. Laboratory investigations showed pancytopenia (haemoglobin of 10.0 g/dl, white blood cell count of 4000/cmm, and a platelet count of 68,000/cmm), coagulopathy (prothrombin time 12/14 seconds and activated partial thromboplastin time 12/14 seconds), and abnormal liver function tests (LFTs) (alanine transaminase 46 U/L, aspartate transaminase 65 U/L, bilirubin 2.1 mg/dl, and albumin 2 mg/dl). Chest radiography revealed a blunted right costophrenic angle and opacities in the left upper and lower zones with intervening lung parenchyma. Contrast-enhanced computed tomography (CT) thorax demonstrated bilateral pleural effusions with contrasting features: A small right-sided free-flowing pleural effusion and a large left-sided loculated pleural effusion with enhancing pleural surfaces (Figure 1).

Diagnostic pleural aspiration was performed bilaterally, yielding distinct fluid characteristics. On the right side, 50 ml of light-yellow fluid was aspirated, whose biochemical analysis revealed to be a transudate (protein 1.2 g/dl, lactate dehydrogenase [LDH] 120 IU/L, glucose 100 mg/dl, total leucocyte count (TLC) 1000/cmm, neutrophils 67%, and lymphocytes 33%), consistent with hepatic hydrothorax. In contrast, the left-sided yellowish pleural fluid exhibited characteristics of an exudate (protein 3.4 g/dl, LDH 420 IU/L, glucose 32 mg/dl, TLC

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32,000/cmm, neutrophils 10%, and lymphocytes 90%, suggestive of a chronic disease process confined to the pleura. Renal profile, kidney ultrasound, and echocardiography collectively ruled out renal and cardiac origins for the right-sided transudative effusion.

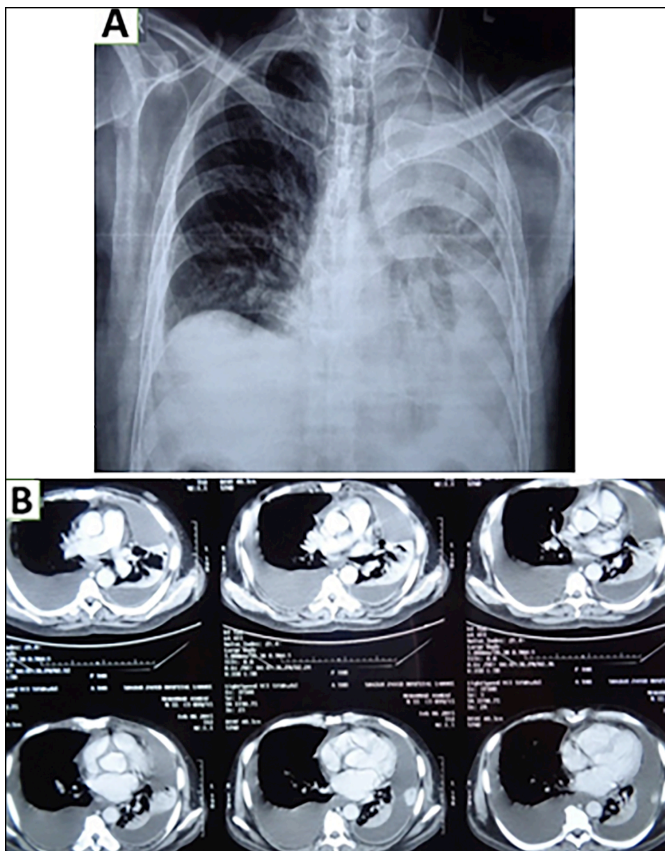


Figure 1: (A) Chest x-ray (PA view) demonstrates a slightly elevated right hemidiaphragm with associated costophrenic angle blunting, indicating a possible pleural effusion. The trachea is deviated to the left, and additionally, there is diffuse opacification in the left upper and lower zones, with intervening lung parenchyma visible. The left hemidiaphragm is obscured, consistent with a large pleural effusion. (B) Contrast-enhanced computed tomography (CECT) scan reveals a small, free-flowing pleural effusion on the right side with a curved upper margin. In contrast, the left side shows a loculated pleural effusion with a collapsed lung in the middle, and an enhanced parietal pleural surface, indicative of inflammation.

To determine the underlying cause of the left-sided pleural effusion, rigid medical thoracoscopy (Karl Storz Hopkins Telescope Optik 0°, 10 mm) was performed using a 2% lignocaine for intercostal anaesthesia and sedation (1 mg midazolam). A total of 1200 mL of dark yellow fluid was aspirated during the procedure; the visceral pleura appeared normal on thoracoscope evaluation, with good lung inflation. In contrast, the parietal pleural surface showed rough, flat, and raised yellowish-pink nodules, along with whitish-yellow patches (Figure 2). Multiple biopsies were taken using rigid biopsy forceps (5mm). A 28 French chest tube was inserted and attached to an underwater seal bottle. Post-procedure chest radiography revealed a partially collapsed lung with air above and fluid level below that inflated with application of -15 cmH₂O external suction. Over the next 24 hours, 30 mL of fluid was collected in the drainage

chamber. However, after 24 hours, 1000 mL of light fluid was collected, which surprisingly, showed biochemical characteristics consistent with a pleural transudate. The next day, an additional 900 mL of light-yellow fluid was collected, suggesting ascent of ascites through diaphragmatic pores into the pleural cavity. Concurrently, the patient's abdominal girth decreased. Hepatic hydrothorax was being drained through the left pleural space. To prevent hepatic encephalopathy due to rapid fluid output, protein loss, and the risk of acute renal injury, the pleural drain was removed. The patient was discharged after two days of observation. Histopathology reports revealed caseating granulomatous inflammation and a positive tissue GeneXpert MTB/RIF assay, leading to the prescription of anti-tuberculous medicines (isoniazid, ethambutol, rifampicin, and levofloxacin for 2 months) followed by isoniazid and rifampicin for 4 months. The patient was regularly monitored with serial LFTs and chest radiographs and successfully managed on an outpatient basis.

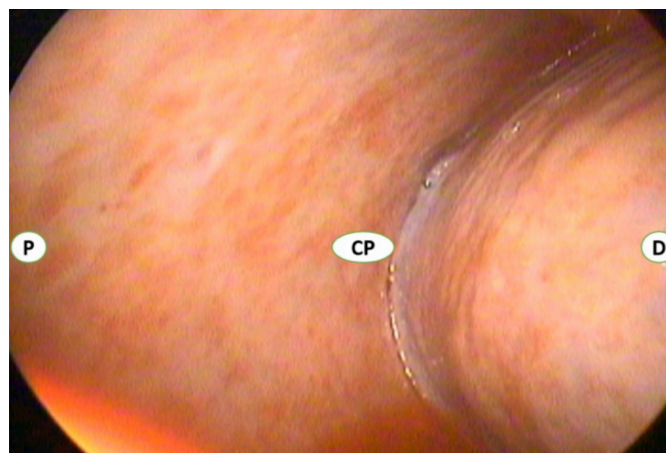


Figure 2: Medical thoracoscopic view of left pleural cavity showing slightly raised and flat nodules and patches over the parietal pleura (P), costophrenic angle (CP) and diaphragm (D).

DISCUSSION

Hepatic hydrothorax refers to a transudative pleural effusion seen in 5-15% of liver cirrhosis with or without ascites.² Effusions related to hepatic hydrothorax are typically right-sided; however, they may be bilateral or left-sided. Diagnosis is usually confirmed by pleural aspiration, demonstrating typical features of a transudative effusion in the absence of co-existent cardio-pulmonary or renal pathology.³ This patient's CLD and transudative ascites in the absence of renal or cardiac comorbidities suggested that the transudative right-sided pleural effusion was due to hepatic hydrothorax. On the other side, diagnosing pleural tuberculosis is challenging due to its paucibacillary nature, but advancements in Polymerase Chain Reaction (PCR)-based techniques such as GeneXpert MTB/RIF assay have enhanced the sensitivity of mycobacteriologic tests on typically lymphocytic exudative pleural fluid or tissue.⁴ However, a definitive diagnosis often requires histopathological confirmation through pleural biopsy, mycobacterium tuberculosis culture, and PCR, which was performed through GeneXpert MTB/RIF assay in this case.⁵⁻⁸

Pre-procedural thoracic ultrasound examination of this patient revealed right-sided anechoic and free-flowing effusion suggestive of a transudate, whereas the left-sided effusion exhibited an echogenic, septated appearance, indicative of an exudate, demonstrating the high specificity of ultrasound in differentiating transudative from exudative pleural effusions.^{6,8} The CT scan of the patient (Figure 1) revealed distinct findings for each side: A small free-flowing right-sided effusion, and a large, loculated left-sided effusion with enhancement of the visceral and parietal pleural surfaces, demonstrating the split pleura sign. This sign, characterised by thickening and enhancement of both pleural layers after contrast injection (classically seen in pleural empyema), is indicative of inflammation and suggests an exudative effusion.^{7,8}

Consistent with the findings of the above-mentioned studies in the index case, the GeneXpert MTB/RIF assay on pleural tissue was positive.^{4,5} Furthermore, the histological examination of the pleural biopsy revealed caseating necrotising granulomas, confirming the diagnosis of pleural tuberculosis involving the left pleura. Similar to this case, patients may have bilateral pleural effusions with markedly different biochemical characteristics; the situation is called Contarini's condition.¹ CT scanning in such situations can identify small collections of gas/air, loculations, or pleural thickening and pleural enhancement, characteristics that are not found in transudates.^{1,7,8} The presence of bilateral pleural effusions with markedly different characteristics (Contarini's condition) presents a diagnostic puzzle that should prompt further investigation. In the present case, the patient's underlying liver disease contributed to the development of hepatic hydrothorax, while the left-sided pleural effusion was due to tuberculous pleuritis.

In conclusion, this case highlights the importance of diagnostic thoracentesis and imaging studies in identifying Contarini's condition, particularly in patients with underlying CLD.

PATIENT'S CONSENT:

Informed consent was obtained from the patient to publish the data concerning this case.

COMPETING INTEREST:

The author declared no conflict of interest.

AUTHOR'S CONTRIBUTION:

TM: Consent from patient, inception, detailed write-up, collection, revision, and approval of the final version of the manuscript to be published.

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