UCLA David Geffen School of Medicine

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Abstract Form

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Project Title:	Tapping into the Heart of a Diagnosis: A Case of Pleural Effusion with a Larger Story to Tell

Research Category (please check one):							
☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: Pleural effusions are commonly associated with heart failure and other disease processes, and they can be further classified as transudative or exudative. Acute decompensated heart failure presents with unilateral or bilateral pleural effusions in over 50% of cases (1). Rarely do effusions warrant diagnostic thoracentesis in the setting of obvious heart failure exacerbation. However, it can be useful in cases where other pathology is present. We present the case of a 68-year-old male who was admitted for 4 days of worsening dyspnea and was found to have new diagnoses of HFrEF and SLE.

Case Report: The patient is a 68-year-old male with past medical history of Charcot-Marie-Tooth disease, GERD, hiatal hernia, HTN, and HLD. He presented to the emergency department for four days of acute-on-chronic worsening dyspnea that developed over the course of three months, significant bilateral lower extremity edema, and orthopnea. He denied any sick contacts. Of note, he had presented to our hospital 3 months prior with similar symptoms, polyarthralgia, one week of recurrent fevers, 20lbs of weight loss, and reduced appetite. No infectious etiology was found. Initial rheumatology work-up while inpatient eventually revealed positive ANA and dsDNA; however, the patient did not initially meet EULAR diagnostic criteria for SLE. He was also found to have a new right-sided pleural effusion. However, no safe pocket was available for thoracentesis, and he was discharged with strict return precautions.

In the ED, the patient was afebrile, tachypneic to 29, SpO2 97% on room air with BP of 201/81. Exam displayed increased work of breathing, decreased right basilar lung sounds, left-sided inspiratory crackles, and 2+ bilateral lower extremity edema extending to mid-thigh with associated venous stasis changes. No rashes or skin lesions were appreciated. CBC was significant for Hgb 9.3, normocytic, without leukocytosis, platelets 406. CMP significant for Na 132, Cr 1.36, and albumin of 1.9 with no other LFT abnormalities. He was treated with 40mg IV Lasix and hydralazine for hypertension and admitted for anasarca in the setting of hypoalbuminemia. CT Thorax revealed interval enlargement of right pleural effusion. IR CT guided thoracentesis was performed, but the effusion was unable to be fully evacuated because of significant loculations. Pleural fluid analysis of clear yellow colored fluid demonstrated pH 7.72, RBC 218/cumm, nucleated cell count 460/cumm, segmented neutrophils 1%, lymphocytes 90%. Total serum protein 5.5, pleural fluid protein 3.1, serum LDH 211, and pleural fluid LDH 95 indicating 98% positivity for exudative lymphocytic effusion per Light's criteria. There was lower suspicion for TB pleuritis given only mildly elevated adenosine deaminase. With the anti-dsDNA antibodies (1:160) and ANA (1:640), low C3 (70), and lymphocytic pleural effusion consistent with SLE serositis, our patient now met EULAR criteria for SLE, with score >10. He was started on methylprednisolone, hydroxychloroquine, methotrexate, folic acid, and Bactrim for PJP prophylaxis while pending TTE to evaluate for pericarditis. TTE revealed HFrEF (35-40%) with grade I diastolic dysfunction with no noted pericardial effusion. He was more aggressively treated with IV Lasix and initiated on full GDMT. Repeat CT Thorax revealed near resolution of residual right-sided pleural effusion just 24h later. At time of discharge, he reported improvement in his dyspnea, appetite, and lower extremity edema.

Discussion: Pleural effusions in the setting of acute decompensated heart failure often do not warrant thoracentesis and pleural fluid analysis. However, pursuit of a diagnostic thoracentesis should be considered in patients with symptoms of pleurisy in the absence of infection as pleurisy should raise the suspicion for a possible inflammatory etiology to the pleural effusion. In patients with volume overload and disease characteristics suspicious for lupus, new onset heart failure should also raise concern for possible cardiovascular manifestations of SLE such as pericarditis and myocarditis(2). This is an interesting case of heart failure exacerbation contributing to an exacerbation of SLE serositis such that pleural fluid analysis of a difficult and loculated effusion became possible. It is important to note that not all pleural effusions in the setting of acute decompensated heart failure are transudative and that keeping a broad differential and considering further investigation with thoracentesis when there is evidence of another underlying cause of effusion can be a key element of diagnostic workup.

References:

(1) Signe Glargaard, Jakob Hartvig Thomsen, Brian Bridal Løgstrup, et al. Thoracentesis to alleviate pleural effusion in acute heart failure: study protocol for the multicentre, open-label, randomised controlled TAP-IT trial. *BMJ Open*. 2024;14(1):e078155. doi:10.1136/bmjopen-2023-078155

(2) Yafasova A, Fosbøl EL, Schou M, et al. Long-Term Cardiovascular Outcomes in Systemic Lupus Erythematosus. *Journal of the American College of Cardiology*. 2021;77(14):1717-1727. doi:10.1016/j.jacc.2021.02.029