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Commentary

Combined Thoracic Effusions: A Diagnostic Challenge

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COMMENTARY

Either chest computed tomography (CT), CT angiography, or echocardiography have superb sensitivity in detecting pleural and pericardial effusions, and quantifying their extent. Moreover, in the evaluation of dyspnea, point of care ultrasonography in the emergency department is being increasingly used to accurately detect effusions [1]. As a result of the expanding use of these imaging modalities, the number of patients diagnosed with pleural and pericardial effusions occurring together is increasing. Such patients are not infrequently encountered both in ambulatory settings and in hospitalized patients, sometimes in the context of a known overriding disease such as a metastatic lung or breast cancer; and sometimes as part of a new disease presentation, such as acute 'idiopathic' pericarditis or SLE.

The latter often include patients, who are acutely ill, in need of a rapid diagnosis and treatment, yet the differential diagnosis of associated pleuropericardial effusions is very wide [2], and clinicians may benefit by a systematic approach to diagnosis. A concise differential of concurrent pleural and pericardial effusions is presented in Table 1. No prospective studies exist. A single retrospective series described 92 internal medicine patients over 11 years who had involvement of ≥ 2 serous membranes, including ascites. The majority (n=79) had pleuropericardial involvement. Neoplasm was the most frequent diagnosis (24%) followed closely by infection (18%) and autoimmune disease (14%). In almost half, the etiology remained unknown but effusions resolved in 86%, and no new diagnoses were found [3]. This study underplays the importance of iatrogenic causes which are not infrequently encountered.

For example, thoracic effusions can be either drug-induced (quite rare) or procedure-related, and among the latter, post-pericardiotomy syndrome which was reported not only after cardiac surgery but also following percutaneous interventions, is especially prominent [4]. In acutely ill, highly symptomatic patients with pleuropericardial effusions, acute respiratory distress syndrome, acute pancreatitis and acute aortic dissection (type A) need to be considered, as well as pulmonary embolism.

(A) Infectious Cause
Acute 'Idiopathic'/viral pericarditis^
COVID-19, severe/critical illness
Tuberculosis^, especially in endemic areas
(B) Neoplastic Disease^
Lymphoma (Non-Hodgkin, Hodgkin, primary effusion) Cancer (lung, breast, gastrointestinal, gynecologic, metastatic unknown primary) Mesothelioma
(C) Inflammatory Disease^ (also includes A1)
Postcardiac injury (Dressler) syndrome
Systemic lupus erythematosus
Rheumatoid arthritis
(D) Iatrogenic Cause^
Drugs: e.g. Drug-induced SLE, Drug-induced pulmonary hypertension, Anti-neoplastic drugs, Gonadotropins, etc.
Procedures: e.g. Postpericardiotomy syndrome, Postradiation
(E) Varied Other Causes A. Supradiaphragmatic
Transudative^: e.g. Congestive failure, Pulmonary hypertension, Pulmonary embolism Exudative: e.g. Blunt chest trauma, Aortic dissection type A, Ac. respiratory distress syndrome, pothyroidism^
B. Subdiaphragmatic Transudative: e.g. Hypoalbuminemia (advanced) Exudative: e.g. End-stage kidney disease
(F) Idiopathic
Note: * A detailed list based on a recent review of the literature can be found in [2]. ^ Reported as a presenting manifestation

Table 1: A shortlist of the major conditions causing concurrent pleural and pericardial effusions.

Three not uncommon causes of combined thoracic effusions need to be considered in more detail.

First, whereas in marked hypoalbuminemia of any cause transudative pleural effusion may occur, pericardial effusions are more unusual. However, heart failure and Pulmonary Arterial

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Hypertension (PAH) are other important, interrelated causes. In decompensated left heart failure, secondary PAH often leads to increased right ventricle afterload and failure with pleural transudates (in approximately 50%, mostly bilateral or right-sided), and small pericardial effusions (20%) and [5,6]. Idiopathic-PAH (group 1) and connective diseases-associated-PAH (notably scleroderma) are similar in this respect. Larger effusions correlated with higher right-atrial pressure. In acute pulmonary embolism-PAH, one third to one half of the patients had pleural effusion and 7-24% had pericardial effusion, both strongly associated with mortality [7]. Second, Combined pleuropericardial effusions associated with neoplastic diseases are common and frequently large and symptomatic. Most malignancies presenting as concurrent effusions or revealing them to be metastatic are lung cancer, breast cancer and lymphomas [8], including the rare primary effusion lymphoma, almost always associated with HHV-8 and HIV. Practically, almost any cancer can be involved, including metastatic carcinoma of an unknown primary. In addition to imaging and cytological studies, tumor markers in the effusions may support the diagnosis. Benign intrathoracic tumors, ovarian fibroma, histiocytoses, and Castleman disease are far less common but also possible. Third, acute viral/idiopathic pericarditis is the most common condition affecting the pericardium with an estimated incidence of 28 per 100,000 per year. It has a cluster of typical findings on presentation [9] and may be associated with unilateral or bilateral exudative sterile pleural effusions in 33-50% of the patients, and pericardial effusions in 66% [10]. In contrast with "secondary" causes, pericardial effusion is often small (<10 mm) or absent; The pericardium is not thickened; fever and symptoms are mild; inflammatory markers are pronounced; typical ECG changes are associated; hemodynamic compromise is not seen in most patients; no extrathoracic disease is present; and patients respond to NSAID and colchicine within ≤ 7 days [2,4].

In the diagnostic approach, echocardiography remains a crucial initial study (class I indication), evaluating. cardiac function, pulmonary artery pressure (N<25 mm), effusion size (small <10 mm vs. moderate >10 mm, large >20 mm), position (posterior vs. circumferential), and possibly pericardial thickening (>4-5 mm). Cardiac compression, an emergency, can be first suspected by bedside determination of pulsus paradoxus >10mm; its negative likelihood ratio of 0.03 is particularly all very useful [11]. Imaging, usually by CT/CT-angiography or MRI/CMR is also a frequently used early in the diagnostic workup with a high yield in selected patients with 'red flag' presentations , but little value in patients likely to have acute 'viral' pericarditis or autoimmune disease. In the diagnostic evaluation, several general principles apply. First, systematic diagnostic evaluation should as always be based on a good history and full examination complemented by patient-tailored laboratory tests [12]. The etiology of many effusions will be evident from the data already available on presentation. The unique patient's "pre-test probability" and risk factors must be carefully assessed since the diagnosis is often pathogenetically-related to the patient's susceptibilities. A complete history focusing on a cancer, autoimmunity, organ failure, or PAH, recent procedures, drug exposure, ethnicity, travel history, occupational and family history are all very useful in directing investigations. Especially, HIV-status and immunosuppressive medications need to be quickly ascertained.

Second, the patient's presenting features, although nonspecific may yield essential etiological clues. For example, a highly acute presentation with hemodynamic instability and considerable pain may suggest aortic dissection, massive pulmonary emboli and if febrile, purulent pericarditis, mediastinitis, or complicated pneumonia. In contrast, a stable patient with pleuritic and positional retrosternal pain, sometimes pericardial friction-rub and diffuse ST elevation/PR depression suggest acute pericarditis. Third, pleural fluid is easily accessible for examination by thoracentesis and should be carefully examined, more than once if necessary [13]. Transudates fulfilling all Light's criteria have very few causes, obvious on initial evaluation. Fourth, when the pericardium appears to be the major serous cavity involved (moderate to large effusions, 'red flag' features) and diagnosis remains unclear, early pericardiocentesis should be considered, especially when bacterial or neoplastic etiology is suspected and cannot be verified by less invasive means [14]. This is increasingly performed by video-assisted thoracoscopic surgery also enabling a high-yield pericardial biopsy and if necessary, drainage. Fifth, a substantial minority of patients remain 'Idiopathic' despite high-quality investigations. Moreover, the practical yield and necessity of establishing an etiology in many low-risk cases remains controversial. Thus, not identifying an etiology after covering the major categories identified is not unexpected, and in selected patients, follow-up may suffice.

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CONFLICT OF INTEREST

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