Pericarditis as a window into the mind of the internist

In this issue of the *Journal* (page 285), Alraies et al comment on how extensively we should look for the cause of an initial episode of pericarditis.

The pericardium, like the pleura, peritoneum, and synovium, can be affected in a number of inflammatory and infectious disorders. The mechanisms by which these tissues are affected are not fully understood, nor is the process by which different diseases seem to selectively target the joint or pericardium. Why are the joints only minimally inflamed in systemic lupus erythematosus (SLE), while lupus pericarditis, in the uncommon occurrence of significant effusion, is often quite inflammatory, with a neutrophil predominance in the fluid? Why is pericardial involvement so often demonstrable by imaging in patients with SLE and rheumatoid arthritis, yet an acute pericarditis presentation with audible pericardial rubs is so seldom recognized?

Although nuances like these are not well understood, in medical school we all learned the association between connective tissue disease and pericarditis. The importance of recalling these associations is repeatedly reinforced during residency and in disease-focused review articles. During my training, woe to the resident who presented a patient at rounds who was admitted with unexplained pericarditis and was not evaluated for SLE with at least an antinuclear antibody (ANA) test, even if there were no other features to suggest the disease. Ordering the test reflected that we knew that, occasionally, pericardial disease is the sole presenting manifestation of lupus.

Such is the plight of the internist. Pericarditis can be the initial manifestation of an autoimmune or inflammatory disease, but this is more often relevant on certification examinations and in medical education than in everyday practice. We are now charged with ordering tests in a more cost-effective manner than in the past. This means that we should not order tests simply because of an epidemiologic association, but only when the result is likely to influence decisions about testing or treatment. But that creates the intellectual dissonance of knowing of a potential relationship (which someone, someday, may challenge us about) but not looking for it. There is an inherent conflict between satisfying intellectual curiosity and the need to be thorough while at the same time containing costs and avoiding the potential harm inherent in overtesting.

A partial solution is to try to define the immediate risk of not recognizing a life- or organ-threatening disease process that can be suggested by a positive nonspecific test (eg, ANA), and to refine the pretest likelihood of specific diagnoses by obtaining an accurate and complete history and performing a focused physical examination. For example, if we suspect that SLE may be the cause of an initial episode of symptomatic pericarditis, our initial evaluation should focus on the patient's clinical picture. Is there bitemporal hair-thinning? New-onset Raynaud symptoms? Mild generalized adenopathy or lymphopenia? A borderline-low platelet count, or any proteinuria or microhematuria (which should warrant a prompt examination of a fresh urine sediment sample by a physician at the point of care to look for cellular casts indicative of glomerulonephritis)?

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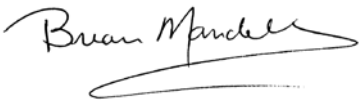
As internists, we should try to fulfill our need to be thorough and compulsive by using our honed skills as careful observers and historians—taking a careful history from the patient and family, performing a focused physical examination, and appropriately using disease-defining or staging tests before ordering less specific serologic or other tests. Practicing medicine in a conscientious and compulsive manner does not mean that every diagnostic possibility must be tested for at initial presentation.

Reading how experienced clinicians approach the problem of pericarditis in a specialized clinic provides a useful prompt to self-assess how we approach analogous clinical scenarios.



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