Controversial issues in the management of pericardial diseases

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Supplemental data
1. Definition of acute vs. chronic pericarditis

Many terms have been introduced for classificatory purposes and are rather arbitrary. The distinction between “acute” and “chronic” pericarditis is simply based on the duration of signs and symptoms beyond an arbitrary observation time (generally 3 months).

2. Definition of incessant vs. recurrent pericarditis

“Incessant pericarditis” indicates cases with recurrent symptoms either during drug discontinuation or attempted weaning, while “recurrent pericarditis” is a term reserved for cases with remission within 6 weeks and subsequent recurrence of symptoms.

3. Diagnosis of pericarditis

The diagnosis of pericarditis is based on simple criteria, not always clearly reported. They include typical chest pain, pericardial friction rub, widespread ST-segment elevation, and pericardial effusion. At least 2 of 4 should be present for the diagnosis of acute pericarditis.\(^3\)-\(^{12}\) The inclusion of pericardial effusion, not universally accepted,\(^1\),\(^5\) is justified considering that this feature, although not necessary, is a confirmatory finding when present. At the same time, evidence of elevated inflammatory markers (e.g. C-reactive protein, CRP) is also confirmatory and should be considered for the diagnosis and follow-up of pericarditis (Table 1).\(^4\),\(^{13}\) Erythrocyte sedimentation rate (ESR or SED) is cheaper but less specific; CRP values raise and then decrease sooner than ESR, being a better marker for monitoring, moreover are less influenced by other confounding conditions (e.g. haematocrit).

The basic diagnostic evaluation should include physical auscultation, ECG, transthoracic echocardiography, routine blood tests, including markers of inflammation (i.e. CRP and/or ESR) and myocardial lesion (creatine-kinase, troponins), and chest X-ray in all cases of suspected pericarditis.\(^2\)
4. Laboratory tests.

Specific laboratory tests should be related to suspected etiology such as a systemic disease (autoimmune, metabolic, neoplasms) or infectious condition (especially tuberculosis). Routine use of laboratory tests could lead to irrelevant findings. For instance, antinuclear antibodies (ANA) are often performed in patients with acute and recurrent pericarditis and low-positive titres are commoner in patients with idiopathic recurrent pericarditis than in healthy controls (43.4 vs. 9.8%, p<0.001), suggesting a possible autoimmune pathogenesis, although they are often a clinically non-specific finding in the single patient.\(^a\) Routine serologic testing for ANA suggests a source for recurrent pericarditis in less than 10% of cases, and in these cases other evidence typically indicates the underlying disease.\(^{17,a}\) Anti-SSA antibodies may suggest a subclinical Sjogren’s syndrome, with xerostomia and xerophthalmia.\(^{13}\)

Diagnostic studies of the pericardial fluid may be useful: adenosine deaminase for tuberculosis,\(^{b-d}\) tumor markers (CEA and CYFRA)\(^{d,e}\) and cytology\(^{f,g}\) for neoplasms, culture and polymerase chain reactions\(^{h,i}\) for infections. Other commonly used data (i.e. protein, LDH, glucose, cell count) may be less useful.\(^j\)

5. Imaging

Integrated imaging including echocardiography, computed tomography (CT), and cardiovascular magnetic resonance (CMR) may provide valuable aid in the etiology search.

Echocardiography accurately detects pericardial effusion providing semiquantitative size estimation as well as assessment of its hemodynamic importance but only limited non-specific data for the characterization of the effusion and etiology search. Most trasudates are relatively anechoic, while exudates, and blood resemble spontaneous echo contrast. Chronicity is associated with prominent strands or septations between layers, that can be found also more commonly in bacterial etiologies (i.e. tuberculous and purulent pericarditis).\(^k\) Stronger indicators of a possible specific etiology
include the presence of large pericardial effusion and/or cardiac tamponade. However important limitations of echocardiography include its inability to provide accurate estimation of pericardial thickness, study of entire pericardium and surrounding chest structures, or evaluation of loculated effusion because of the limited acoustic window.

On the contrary, CT and CMR provide a larger field of view, allowing detection of loculated pericardial effusion, pericardial thickening and masses, as well as associated chest abnormalities. CT attenuation measurements and analysis of CMR signals may enable the characterization of pericardial fluid better than echocardiography. Another specific advantage of CT imaging includes the ability to detect minimal amounts of pericardial calcium, but if performed without ECG gating, CT may lead to cardiac motion artefacts, that limit the evaluation of pericardial thickness.

CMR has a superior ability to characterize pericardial effusions and masses with the use of a combination of T1-weighted, T2-weighted, and gradient-recalled echo cine sequences without the use of either iodinated contrast material or ionizing radiation. Moreover CMR is superior to CT in differentiating fluid, especially highly proteinaceous exudative effusions, from thickened pericardium; it may also provide non-invasive evidence of myocardial involvement (myopericarditis).

In conclusion, CT and CMR imaging should be considered as useful adjunct to echocardiography in cases with loculated or haemorrhagic effusions, suspected pericardial thickening and constriction, and pericardial masses, but also when findings at echocardiography are difficult to interpret or conflict with clinical data, and when examination of the entire chest is required to assess possible neoplasms or tuberculosis. Integrated cardiovascular imaging (usually data from cardiac catheterization, but also serial Doppler and CMR findings before and after pericardiocentesis) is also useful to detect effusive-constrictive forms. The diagnosis of effusive constrictive pericarditis often becomes apparent during pericardiocentesis in patients initially considered to have uncomplicated cardiac tamponade. Unexpected persistence of the v wave of right atrial pressure is a clue to the possibility of effusive constrictive pericarditis that may be present before
pericardiocentesis. After pericardiocentesis, despite lowering of the pericardial pressure to near zero, persistence of elevated right atrial pressure suggests the presence of effusive constrictive disease. The diagnosis has been defined by failure of the right atrial pressure to fall by 50% or to a level below 10 mmHg after pericardiocentesis.27 A summary of the relative contribution of different diagnostic tools for the specific etiologic search is reported in table 6.
Additional References


