Answer

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The correct answer is A: pericarditis. The troponin was measured again 6 hours later, yielding a value of 2.07 µg/L. An echocardiogram demonstrated trace pericardial effusion with normal cardiac anatomy and function. The patient was discharged home on ASA and had complete resolution of his symptoms and of his pericardial effusion when seen in follow-up 8 days later.

Background

The pericardium consists of an outer (parietal) and inner (visceral) layers. Between these layers is a potential space normally containing approximately 20 mL of an ultrafiltrate of plasma.[1,2] Numerous villi and cilia enhance resorption of this fluid and facilitate movement of the pericardial surfaces over one another. Inflammatory disruption of this system by various local or systemic disease processes leads to pericarditis.[1–3]

Pericarditis may be classified both clinically and etiologically. Clinically, it is divided thus: 1) acute (<6 weeks); 2) subacute (6 weeks to 6 months); or 3) chronic (>6 months).[4] Of these, acute pericarditis is, by far, the most common and is most commonly idiopathic in nature.[5] When a cause is found, common underlying precipitants include infection, malignant disease, medication and radiation-induced, autoimmune and connective tissue disease, post-myocardial infarction and uremia.[5]

Clinical presentation

Acute pericarditis typically presents as sharp central chest pain that worsens with recumbency and is relieved by leaning forward.[6] The most common physical finding is the pericardial friction rub, which is usually auscultated along the lower left sternal border and is described as a scratching sound. The severity of symptoms relates the level of pericardial injury and the resultant effect on cardiac function.[5] In its most severe form, pericarditis may present as cardiogenic shock secondary to cardiac tamponade or myocarditis.

Investigations

Electrocardiography

Electrocardiographic findings reflect re-polarization abnormalities involving both the atria and ventricles as a result of epicardial and superficial myocardial inflammation. There are no changes related to depolarization, and consequently the P wave and QRS complexes are normal. Typically, the most sensitive finding is diffuse concave-upward ST-segment elevation that, unlike acute myocardial infarction (AMI), encompasses multiple vascular territories (consistent with the diffuse inflammatory nature of the disease process).[6] Also unlike myocardial infarction, there are no reciprocal changes. Other features that aid in differentiating acute pericarditis from AMI are the absence of Q waves and the absence of T-wave inversion at the time of ST-segment elevation.[6] Occasionally, PR-segment depression, which is attributed to subepicardial atrial injury, may be the earliest sign and is very specific for pericarditis when present.[6]

Changes on the ECG classically occur in 4 stages; however, all 4 stages are present in less than 50% of patients. Stage I occurs during the first few days and is characterized by diffuse ST-segment elevation. This stage may last up to 2 weeks. Stage II is characterized by a return of the ST segments to baseline and flattening of the T waves, and may last from days to several weeks. Stage III usually begins at the end of the 2nd or 3rd week and is characterized by inversion of the T waves in the opposite direction to the

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https://doi.org/10.1017/S1481803500008381 Published online by Cambridge University Press

ST segment; this stage may last several weeks. Stage IV represents the gradual resolution of the above.[5,6]

Cardiac troponins

Cardiac troponins have been shown to be highly specific markers for myocardial injury in the context of acute ischemic cardiac disease. However, a number of other processes can also lead to an elevation of serum levels, including pericarditis.

Bonnefoy and colleagues,[7] in a retrospective study involving a cohort of 69 patients, investigated the sensitivity of using cardiac troponin I to assess acute idiopathic pericarditis and its relationship to ST-segment elevation. In 22% of the patients with acute pericarditis, there was a detectable increase of cardiac troponin to beyond the myocardial infarction threshold. In the subset of patients with ST-segment elevation, the proportion rose to 93%. Coronary angiography was performed in approximately half of the patients with abnormal troponin levels and revealed normal coronary arteries.

In a follow-up prospective study by Brandt and coworkers[8] involving 14 patients, it was demonstrated that troponin I was elevated in 10 of 14 patients (71%). Moreover, the temporal release pattern of cardiac troponin resembled that of an acute coronary syndrome, and thus, it was concluded, troponin testing was not useful in distinguishing pericarditis from ischemia as the cause of acute myocardial injury.

The reason for troponin release in pericarditis is unknown, but is thought to be related to a more extensive inflammatory process involving the superficial myocardium (myopericarditis).[9] Also unknown are the prognostic and treatment implications of a positive finding of troponin in the context of pericarditis.

Echocardiography

Echocardiography may be warranted to detect and quantify an effusion, to assess cardiac function and to identify underlying cardiac pathology.[5]

Treatment

Therapy is directed at the underlying disease if a specific cause is identified. In viral or idiopathic pericarditis, the mainstay of treatment is with NSAIDs for a period of 1 to 3 weeks.[4]

Competing interests: None declared.

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