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Can CBNAAT Positivity in Fluid Confirm Tuberculosis Disease?

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Case Vignette

A young man in his early twenties, a chef from Delhi, presented with a 15-day history of low-grade fever in the evenings, associated with progressive painless bilateral pedal edema. Over the past 7 days, he had developed a gradual painless abdominal distension. In the last 2 days, he developed cough and worsening dyspnoea. He denied any recent travel, past history of tuberculosis, or known exposure to tuberculosis. No history of oral ulcers, sicca symptoms, photosensitivity, alopecia, or Raynaud's phenomenon.

On examination, he was dyspneic with raised jugular venous pressure (JVP), and Ewart's sign was noted on chest auscultation along with a pericardial rub on cardiac auscultation. Electrocardiography (ECG) showed electrical alternans, and the chest roentgenogram (Image 1) demonstrated an enlarged cardiac silhouette.

An emergency pericardiocentesis was performed using a pigtail drain. The complete hemogram, kidney function tests, serology, and procalcitonin levels were non-contributory. However, liver enzymes and total bilirubin were modestly elevated. His ESR was mildly elevated, and the Mantoux test was negative. However, pericardial fluid CBNAAT (Cartridge-based nucleic acid amplification test) came positive.

Choose the most likely diagnosis:

A. Takotsubo pericarditis

B. TB hypersensitivity pericarditis

C. Viral pericarditis

D. Tubercular pericarditis

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Answer:- D. Tubercular pericarditis

Comments

Based on the patient's presentation, clinical findings, the presence of electrical alternans on ECG, and an enlarged cardiac silhouette on chest X-ray, a diagnosis of pericardial effusion with a possible infectious or inflammatory etiology can be made. In this case, CBNAAT positivity in the pericardial fluid supports an active tubercular infection rather than a hypersensitivity reaction or viral etiology. CBNAAT detects Mycobacterium tuberculosis complex DNA, which confirms the presence of TB organisms in fluid. It has a robust specificity but unsatisfactory sensitivity in diagnosing TB pericarditis[1].

In tubercular pericarditis, patients typically report constitutional symptoms along with pleuritic chest pain followed by progressively worsening dyspnea. This form of pericarditis also carries a higher risk of pericardial tamponade. The pericardial fluid in tubercular pericarditis commonly shows high lymphocyte counts, elevated ADA levels, and may contain acid-fast bacilli or positive CBNAAT. In contrast, TB hypersensitivity pericarditis often presents with acute or subacute dyspnea but lacks significant constitutional symptoms. The pericardial fluid in hypersensitivity reactions usually shows mixed cellularity, lower ADA levels, and negative results on CBNAAT. DNA detection is crucial for diagnosing active TB pericarditis, as it directly identifies mycobacterium tuberculosis. Tubercular antigen detection is more indicative of hypersensitivity TB pericarditis, suggesting an immune reaction to tubercular antigens in the absence of detectable bacteria. Tubercular pericarditis typically shows pericardial thickening on imaging and responds to excellently to anti-tubercular therapy (ATT), whereas TB hypersensitivity lacks pericardial thickening and responds better to steroids. Pericardial biopsy confirms the diagnosis.[2]

In this case, the pericardial fluid serosanguinous, total leukocyte count (TLC) was 80 cells/µL, with 60% lymphocytes and 40% neutrophils, and an adenosine deaminase (ADA) level of 36 U/L, which favours the diagnosis of hypersensitivity TB pericarditis. However, a positive CBNAAT result clinches the diagnosis of TB pericarditis.

Viral pericarditis is the second most common cause of pericarditis after idiopathic pericarditis. It is diagnosed via nucleic acid amplification testing. Typical presentation includes acute chest pain, a viral prodrome, and self-limited inflammation, unlike the chronic, progressively worsening symptoms seen here.

Takotsubo cardiomyopathy (TM) is more common in females than males and is often preceded by emotional stressors, such as a sudden family death, abuse, or a quarrel. The most common symptoms are chest pain and dyspnea, resembling those of myocardial infarction. Electrocardiography typically shows ST elevation, particularly in the precordial leads.[3] In rare cases, TM is associated with pericarditis. Although the exact mechanism is unknown, it is postulated that transmural inflammation may extend to the pericardium, resulting to pericardial inflammation.[4]

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