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Polymicrobial Infection in COPD Exacerbation

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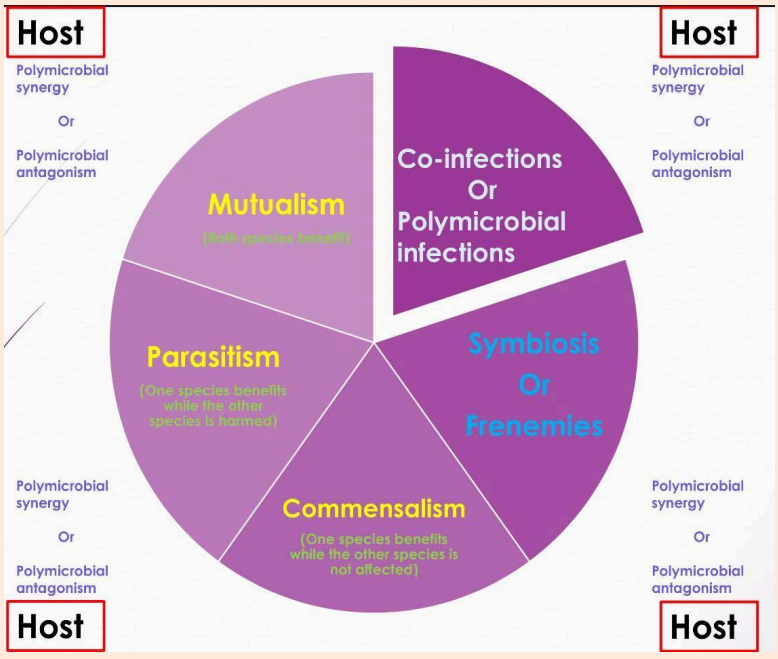
Case Vignette

A middle-aged, thinly built woman who smokes beedi and has a history of biomass exposure, has been suffering from COPD for the last 4 years. She has been taking inhaled betamethasone and tiotropium. Additionally, she had uncontrolled diabetes for a few months. She presented with fever, productive cough, shortness of breath, and chest pain for 5 days. Chest X-ray and CT confirmed pneumonia, cavities, and abscesses in both lungs. Repeated sputum and bronchoalveolar lavage confirmed *Pseudomonas aeruginosa* and *Aspergillus fumigatus*. Along with supportive therapy, she was treated with tablet levofloxacin and injection amikacin for 6 weeks based on culture sensitivity reports, and capsule itraconazole for 6 months. She recovered completely to her baseline COPD and diabetes status.



Ques. What is this polymicrobial diagnosis (figure) in this patient?

A) Polymicrobial - Synergy

B) Polymicrobial- Antagonism

C) Polymicrobial- Mutualism

D) Polymicrobial- Commensalism

Answer: B. Polymicrobial antagonism

Comments:

While Pseudomonas inhibits fungal growth and biofilm production, the interaction between Pseudomonas and Aspergillus is complex, with both organisms inhibiting each other's growth. *A. fumigatus* not only endures the antifungal effects of *P. aeruginosa* but also protects itself. Phenazines, the quorum sensing system, iron competition, bacteriophages, and small colony variants of *A. fumigatus* inhibit fungal biofilm formation. Conversely, A. fumigatus's siderophores, phenazine metabolic transformation, gliotoxin production, and reduced antibiotic sensitivity inhibit bacterial biofilm formation. Pseudomonas can inhibit the growth of Aspergillus conidia but cannot inhibit preformed hyphae, likely due to the gliotoxin production by the hyphae.

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Diagnostic Dilemma in Fever with Right Chest Pain

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Case Vignette

A young man in his late twenties, labourer, presented with a 20-day history of low-grade intermittent fever, accompanied by right lower chest pain radiating to the right shoulder and anorexia. The pain worsened with deep inspiration and coughing. Over the past 7 days, he had developed a productive cough with yellowish expectoration. He denied any recent travel or known exposure to tuberculosis but reports occasional alcohol consumption. Physical examination revealed tenderness over the right upper quadrant and reduced breath sounds at the right lung base. Initial laboratory tests showed leucocytosis. A chest X-ray (figure 1) revealed a right-sided pleural effusion.

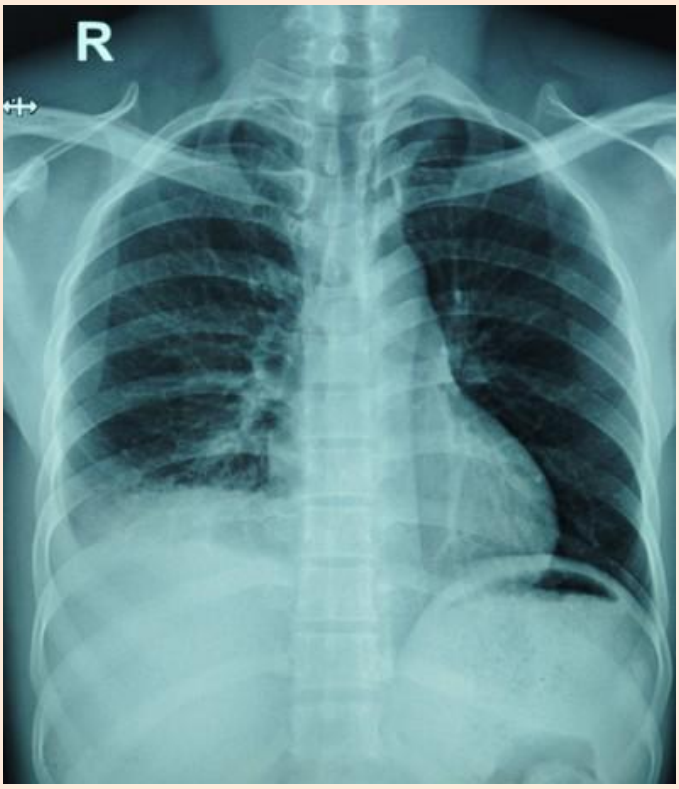

Figure 1: Chest Xray PA view

Ques. What is the next best investigation?

A) Pleural fluid aspiration

B) Sputum examination

C) USG of chest and abdomen

D) CECT chest and abdomen

Answer: C, USG of chest and abdomen

Comments:

Based on the history, initial pathology started near diaphragm and extended into further pleural involvement. Pleural fluid was analysed and suggested exudative in nature by modified Light's criteria, while Gram stain, culture, KOH mount, acid fast bacilli (AFB) staining, and cartridge-based nucleic acid amplification (CBNAAT) test were negative. Adenosine deaminase levels were low. An ultrasound and computed tomography of the abdomen revealed a single coalescing hypodense lesion in segment II of the right lobe of liver, consistent with an amoebic liver abscess (figure 2a). The serum amoebic *GalNac* antigen was positive at 36.6 (negative: < 9.0, equivocal: 9.0-11.0, positive: > 11.0), indicating *Entamoeba histolytica* infection. The patient was managed with percutaneous drainage of the abscess (figure 2b) and intravenous metronidazole 500 mg thrice a day with oral diloxanide furoate 500 mg thrice a day for 10 days.

Here, the learning point was unnecessary workup to reach final diagnosis. To be a steward, we need the right test for the right patient at the right time. Hence diagnostic pearl, subdiaphragmatic pathology including liver abscess should be considered in patients presenting with fever, anorexia, right sided lower chest pain radiating to the right shoulder, and with or without cough in the context of relevant epidemiology. [1,2] Diagnostic point of care test is USG of chest and abdomen.

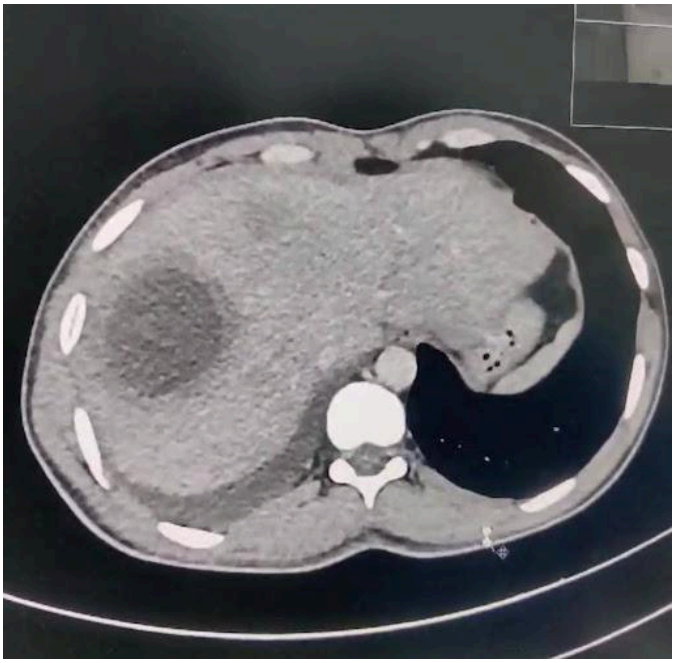

Fig-2a: Computed tomography of the abdomen

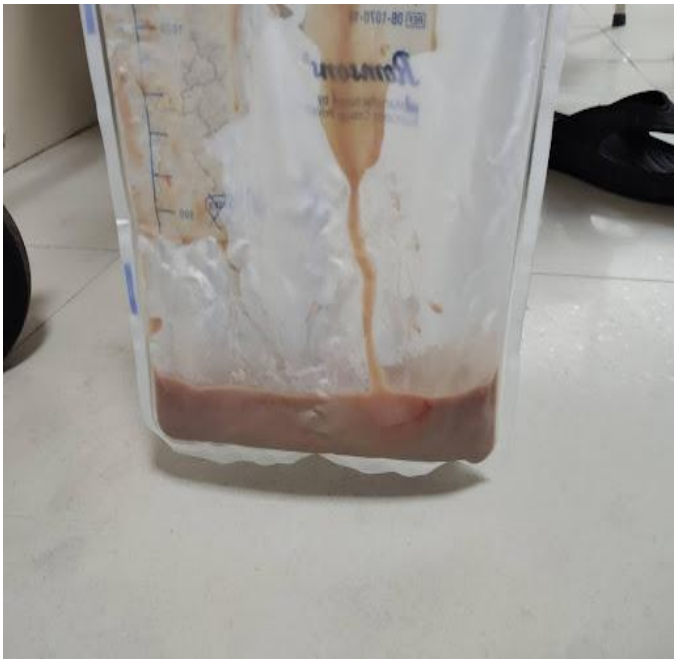

Fig-2b: Percutaneous drainage of the abscess fluid

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All Hemorrhagic Pleural Effusions are Not Infective

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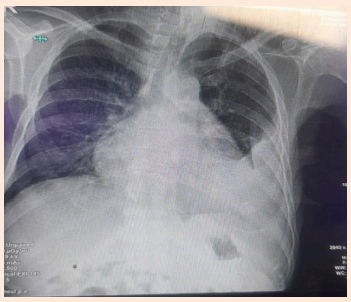
Case Vignette

A middle aged male, who has been suffering from Chronic kidney Diseases-5D, Chronic heart failure and hypertension non-compliant to medications and renal replacement therapy (RRT), presented with decreased urine output, anorexia, shortness of breath, bilateral pedal oedema, abdominal distention, productive cough and vomiting over the past 8-10 days. There was no history of chest trauma, significant weight loss, fever, or night sweats. Routine investigations revealed severe uraemia (Urea - 290 mg/dl) and a normal INR [1.07]. Chest X-ray suggested left pleural effusion and abdominal ultrasound revealed ascites, both atypical fluid collections were grossly haemorrhagic upon tapping. Empirically, the patient was treated with ceftriaxone and azithromycin for a suspected infective aetiology; however, there was no significant clinical improvement. Further evaluation, including a contrast-enhanced CT thorax, suggested malignancy as a cause of the hemorrhagic effusion. The patient received alternate day RRT and demonstrated clinical and vital improvements. Follow-up imaging, including X-ray and point-of-care ultrasound, revealed complete resolution of the pleural effusion and ascites.



Chest X-ray -> Gross left-sided pleural effusion.

Hemorrhagic pleural effusion

Ques. Which of the following best reflects a key principle of stewardship in this case?

A. Antibiotic therapy should be started empirically in patients with suspected infection, and stopped only after cultures confirm the absence of infection

B. Empirical use of antibiotics was appropriate due to the presence of respiratory symptoms and radiological findings, even without microbial confirmation

C. Before initiating antibiotic therapy, a thorough diagnostic workup, including pleural fluid analysis and uremic workup, should have been prioritized to avoid unnecessary antimicrobial use

D. Given the patient's non-compliance and systemic symptoms, broad-spectrum antibiotics were necessary to cover potential multidrug-resistant organisms

Answer:- **C.** Before initiating antibiotic therapy, a thorough diagnostic workup, including pleural fluid analysis and uremic workup, should have been prioritized to avoid unnecessary antimicrobial use

Comments

Hemorrhagic pleural effusions can arise from various non-infective causes, including malignancy, trauma, pulmonary embolism, uraemia and certain inflammatory conditions. If uraemia is a potential cause, then effective RRT helps manage fluid overload and corrects coagulopathy associated with advanced kidney disease.[1] When managing a patient with a hemorrhagic pleural effusion, it's essential to carefully evaluate the underlying cause before initiating antibiotics. Overuse of antibiotics in such cases, where the effusion is not due to an infectious process, can contribute to antimicrobial resistance, unnecessary side effects, and increased healthcare costs.[2]

Surviving Sepsis Campaign Guideline (2021) recommends daily assessment for possible antimicrobial de-escalation over using fixed durations of therapy and early discontinuation of all antimicrobial therapy if the infection is ruled out.[3] So in our case, when non-infective diagnosis was confirmed, all ongoing antimicrobials were stopped at the right time which is another stewardship principle under De-escalation to curtail this rising AMR.

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Can Skin Findings Suggest Disseminated Opportunistic Infection, Malignancy in HIV/ AIDS?

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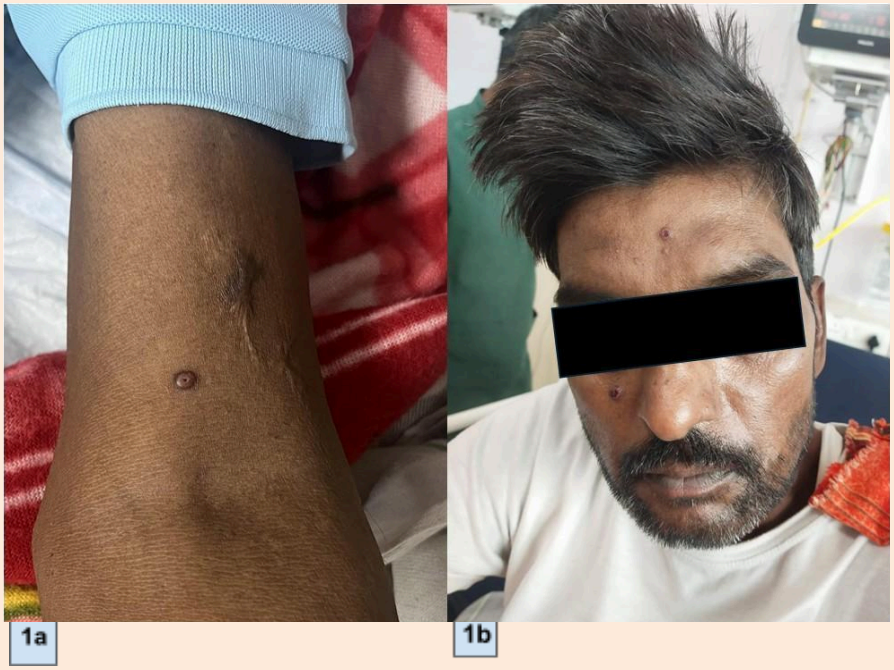
Case Vignette

A person living with human immunodeficiency virus (PLHIV) in his early thirties, an antiretroviral therapy (ART) defaulter, presented with a gradual onset holocranial, pulsatile headache for 10 days, which worsened on forward bending. The headache was followed by a few episodes of projectile vomiting. He also gave the history of anorexia, weight loss, and low-grade fever.

On examination, he was oriented and obeyed commands. He had pallor and multiple raised, skin-colored, umbilicated lesions over his face, scalp, and forearm, some with central ulceration (Images 1a and 1b). He was febrile, and neck stiffness was present. Examination of other systems was non-contributory.



What is your clinical diagnosis?

A. Kaposi's Sarcoma

B. Molluscum contagiosum

C. Histoplasmosis

D. Cryptococcosis

E. MAC infection

Image-1

Answer:- **D.** Cryptococcosis

Comments

Cerebrospinal fluid (CSF) India ink preparation showed yeast cells surrounded by clear halo and CSF biofire was positive for *Cryptococcus neoformans*. Biopsy samples from skin lesions also showed encapsulated yeast of cryptococcus on India ink preparation (image 2).

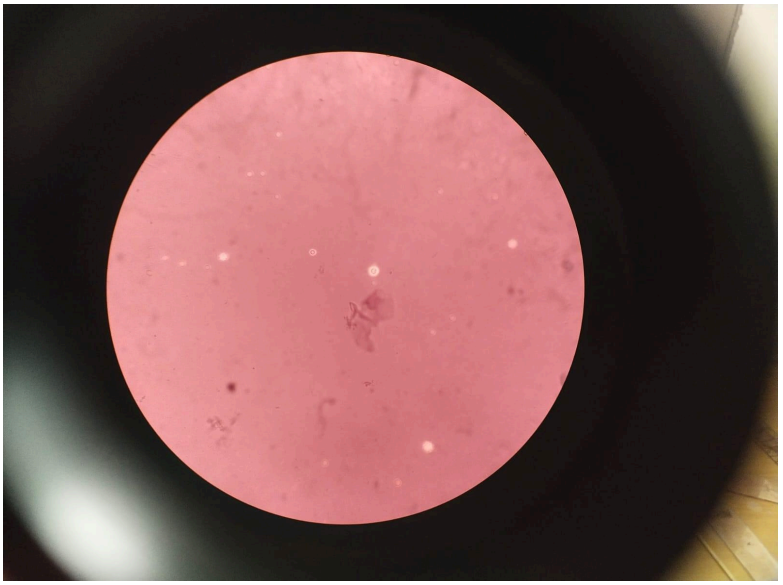


Image-2

Cryptococcus neoformans can cause disseminated infection, particularly in immunocompromised individuals, with the greatest risk when CD4 counts fall below 200 cells/mm^3. It commonly involves the central nervous system, lungs, and skin; and can produce systemic symptoms such as fever, malaise, anorexia, and weight loss. Cutaneous cryptococcosis often presents as skin-coloured or slightly erythematous papules and nodules with central umbilication, frequently accompanied by central necrosis. In profound immunodeficiency, it can present as cellulitis or abscess which mimics bacterial infection in both rapidity of onset and appearance. However, the closest differential for these lesions is Molluscum contagiosum, which manifests as dome-shaped, umbilicated papules. Other differentials include disseminated *Mycobacterium avium complex* (MAC) infection, which may present as painless or occasionally tender clusters; disseminated histoplasmosis, which can appear as papular or nodular lesions with potential ulceration; and Kaposi's sarcoma, characterized by violaceous lesions that may coalesce into plaques, though lacking central necrosis. However, cutaneous histoplasmosis presents as variable morphologies and papules may ulcerate or become crusted. Basal cell carcinoma presents as a rodent ulcer with a rolled border, usually seen in sun-exposed areas and having visible telangiectasias.

Cutaneous manifestations are the third most common presentation of cryptococcosis. Approximately 15% of patients with disseminated cryptococcosis, primarily those with advanced HIV, exhibit cutaneous manifestations[1,2]. Additionally, about 5% of cryptococcal meningitis patients report skin involvement [3]. Hence, the presence of this type of umbilicated cutaneous lesions in HIV-positive individuals warrants consideration of disseminated cryptococcosis, and differentials should be promptly evaluated.

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Can CBNAAT Positivity in Fluid Confirm Tuberculosis Disease?

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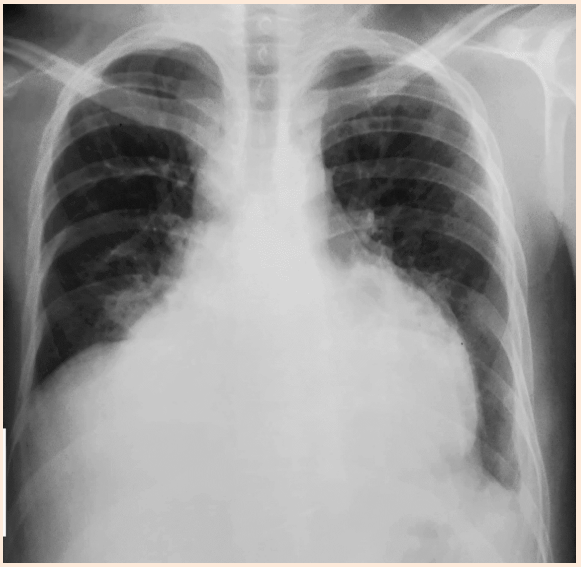
Case Vignette

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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