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CASE REPORT

Cardiac tamponade in adolescence: two cases reports

Filipa Valadares [1], Maria Mendes[2], Eugénia Matos[2], Carlos Escobar[2], Marta Moniz[2], Clara Abadesso[2], Pedro Nunes[2]

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Abstract

Cardiac tamponade is a medical emergency and is the most serious complication of pericardial effusion. The authors describe two case reports with different etiologies, in adolescence. 17 years old female patient, goes to the emergency department due to polyarthralgia, chest pain, orthopnea and fatigue. At observation: fever, tachycardia, decreased cardiac sounds, decreased pulmonary sounds in bases and tibio-tarsal joint edema. Chest radiography: cardiomegaly and bilateral interstitial infiltrate; electrocardiogram: ST elevation; echocardiogram: massive pericardial effusion ("swinging heart"). Pericardiocentesis was performed, with no significant changes in the pericardial fluid. Therapy with non-steroidal anti-inflammatory drugs (NSAIDs), acetylsalicylic acid (ASA) and colchicine was started. Further investigation led to systemic lupus erythematosus (SLE) diagnosis and improvement happened after correct therapy. 16-year-old male patient with Crohns disease under azathioprine and adalimumab, goes to the emergency department due fever, cough and chest pain. Three days before he had started amoxicillin-clavulanic acid for pneumonia. At observation he was tachycardic, with decreased cardiac sounds, decreased right breath sounds and painful abdomen on palpation. Thoraco-abdominal ultrasound: pleural effusion, hepatomegaly, splenic nodules, adenopathies in the celiac trunk. 72 hours after admission, under triple antibiotical therapy, there was clinical worsening and a thoracoabdominal CT was performed: pericardial and pleural effusion, mediastinal adenomegalies, hepatosplenomegaly, splenic lesions. Echocardiogram: pericardial effusion with collapse of the right cavities. Pericardial fluid showed an adenosine deaminase (ADA) of 53U/L. Clinical improvement after therapy for tuberculosis was started. Clinical suspicion is essential for a rapid therapeutic intervention, with echocardiography being the gold standard.

[1] Torres Novas Hospital - Centro Hospitalar do Médio Tejo, Pediatrics - Torres Novas - Lisbon and Vale do Tejo - Portugal.
[2] Hospital Professor Dr. Fernando da Fonseca, Pediatrics - Lisbon - Lisbon - Portugal.

Correspondence to:
Filipa Valadares.
Hospital de Torres Novas - Centro Hospitalar do Médio Tejo. Rua Xanana Gusmão, nº 45, Torres Novas, Portugal. CEP: 2350-754. E-mail: fvaladaresb@gmail.com

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INTRODUCTION

The pericardium, consisting of a double membrane that surrounds the heart and the root of the great vessels, functions to protect the heart against infections or tumor dissemination from neighboring structures[1]. Pericarditis can occur alone or from underlying systemic diseases, consisting of inflammation of the pericardial leaflets, due to fluid buildup, and its severity depends on the volume of the pericardial effusion, its distensibility and disease duration[2]. Because it is infrequent in pediatric patients, and it often has a nonspecific presentation, it is important to have a strong clinical suspicion[1]. Etiologically, it can occur due to infectious, neoplastic, or rheumatologic disease; vasculitis, intoxication, uremia, hypothyroidism, iatrogenic, trauma or being idiopathic[3].

Cardiac tamponade is a medical emergency[2], constituting the most serious complication of pericardial effusion, compromising cardiac function, which can culminate in shock. It occurs when the accumulated volume limits ventricular filling, causing an increase in intrapericardial pressure[2], a decrease in cardiac output and an increase in central venous pressure with jugular engorgement, hepatomegaly and dyspnea[3]. Clinical suspicion is based on Beck's Triad (present in 30-40% of cases): jugular venous distention, hypotension, and hypophonic heart sounds, with echocardiography being the definitive diagnostic test[2]. We describe two cases of cardiac tamponade, with different causes, one in a patient later diagnosed with Systemic Lupus Erythematosus (SLE) and the other as a complication of tuberculosis in an immunocompromised patient.

CASE DESCRIPTIONS

CASE 1

Female, 17 years old, with chest pain, polyarthralgia, photosensitivity and alopecia for a year. No fever, anorexia, weight loss, skin changes, oral thrush, conjunctival hyperemia, Raynaud's phenomenon, respiratory or gastrointestinal symptoms. One month before hospital admission, she went to the emergency department (ER) due to dyspnea, orthopnea and chest pain, with a chest X-ray and electrocardiogram (ECG) without alterations. She was discharged, medicated with analgesia and non-steroidal anti-inflammatory drugs (NSAIDs), without improvement. Due to clinical worsening, she returned to the ER. Upon arrival she was feverish (temperature: 38.5ºC); Blood Pressure (BP): 109/72mmHg; Respiratory Rate (RR): 20 bpm; Heart Rate (HR): 140 bpm; Cardiac auscultation (CA): hypophonic heart sounds; Pulmonary auscultation (PA): decreased breath sounds at the bases. Mild swelling of the tibiotarsus. Laboratory tests: hemoglobin 9.6 g/dL; leukocytes 6500/ulu (5532/uL neutrophils; 578/uL lymphocytes); platelets 293000/uL; urea 26mg/dL; creatinine 0.7mg/dL; CRP 7.38mg/dL. Chest X-ray: cardiomegaly and bilateral interstitial infiltrate (Figure 1). ECG: sinus tachycardia with ST elevation suggestive of acute pericarditis; echocardiogram: massive pericardial effusion ("swinging heart", 33 mm), translucent content in the echocardiography; decreased systolic function; plethoric inferior vena cava (IVC) with reduced inspiratory collapse (Figure 2). Due to the imminence of tamponade, a pericardiocentesis was performed. Pericardial fluid (LP): 800 mL of serosanguineous content, characteristics of inflammatory exudate, pH 6.8, proteins 6 g/dL, glucose <2 mg/dL, lactate dehydrogenase (LDH) 1638 U/L, 31204 cells. /µL, neutrophils: 91%, lymphocytes: 1%, monocytes: 8%, ADA 35.7U/L, neoplastic cells and bacteriological examination were negative. Therapy with NSAIDs, acetylsalicylic acid (ASA) and colchicine was started and showed improvement. On suspicion of Autoimmune/Rheumatic disease, therapy with hydroxychloroquine was initiated. After ruling out infection, the autoimmunity study (Table 1) and meeting the criteria for SLE (SLICC- Systemic Lupus International collaborating Clinics), prednisolone was associated with the instituted therapy, with progressive improvement.

CASE 2

Male, 16 years old, past of Crohn's disease, under azathioprine and adalimumab. Three days before hospital admission, he came to the ER with chest pains. He was discharged on amoxicillin-clavulanic acid due to pneumonia. Due to worsening chest pain, fever and dry cough, he returned to the ER. On exam: SpO2: 97%; RR: 20 bpm; HR: 98 bpm; BP: 112/80mmHg. CA: hypophonic heart sounds, PA: diminished breath sounds on the right. Abdomen: painful on palpation in the right hypochondrium and epigastrium. Laboratory: hemoglobin 12.8 g/dL; leukocytes 3500/uL; 2100/uL neutrophils; 940/uL lymphocytes; platelets 286000/uL; CRP 11.58 mg/dL; SV 84 mm. Chest X-ray: infiltrate in the right middle third - suspected pleural effusion. Thoracoabdominal ultrasound: right pleural effusion with air bronchogram, compatible with pneumonia; hepatomegaly, splenic nodules, adenopathy in the celiac trunk, and it was not possible to rule out lymphoproliferative disease/infection by an atypical agent. He was then medicated with cefotaxime, azithromycin and vancomycin. Seventy-two hours after admission, his breathing got worse, requiring intensive care. Thoraco-Abdominal CT: pericardial effusion (35mm), right posterior pleural effusion; air bronchogram showing pneumonic process, pulmonary micronodular images; mediastinal lymphadenopathy; hepatosplenomegaly, low uptake splenic lesions (hematogenous spread of an infectious process?) (Figure 3). Echocardiogram: voluminous pericardial effusion (25-30 mm), hypoechoic; collapse of the right cavities; Plethoric IVC maintaining some inspiratory collapse (Figure 4). Pericardiocentesis was performed, PF: 400mL of citrine yellow fluid, pH 7.4, proteins 6.8 g/dL, glucose 89 mg/dL, LDH 464 U/L, 1287 cells/µL, neutrophils 2%, lymphocytes 95%, monocytes 3%, ADA 53 U/L, PCR for Mycobacterium Tuberculosis (Mt) negative. Under diagnostic hypotheses we had proliferative disease versus tuberculosis, investigation was continued (Table

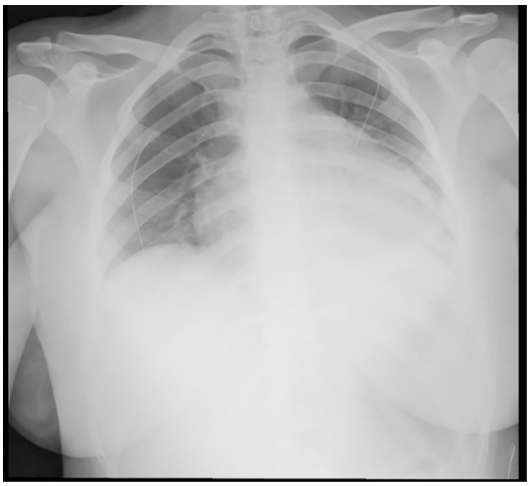


Figure 1. Chest X-ray (Case 1): cardiomegaly with "moringa heart" and effacement of the hilar girdle..

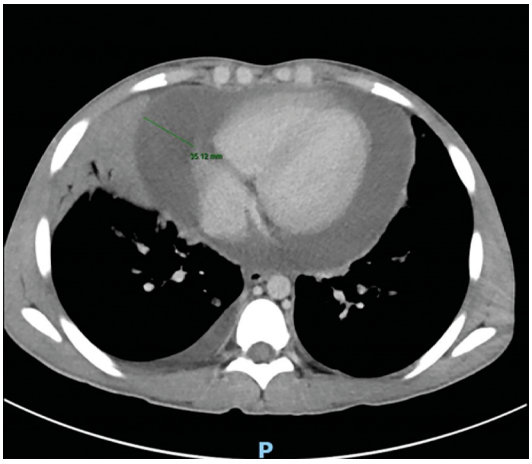


Figure 3. Chest-Abdomen CT (Case 2): large pericardium effusion (35mm).



Figure 4. Echocardiogram (Case 2): pericardial effusion (25-30 mm) with collapsed right-side cavities.

1). Tuberculosis was confirmed, and anti-tuberculosis therapy was started with improvement.

DISCUSSION

Pericarditis is infrequent in pediatric patients, requiring a high suspicion for its diagnosis[1]. With a multifactorial cause, the complications vary according to the etiology, with cardiac tamponade being the most serious complication. More than half of tamponade resulting from pericarditis is caused by malignancy, tuberculosis or other infection[2]. In pediatric patients and among infectious causes, viral infection is the most frequent cause[1].

We describe two cases of cardiac tamponade as a manifestation of SLE and tuberculosis. SLE often has cardiovascular manifestations, with pericarditis being the main one; however, cardiac tamponade is rare (1-2%)[4,5]. In the described case of tuberculosis, the particularity of being a patient on immunosuppressive therapy is highlighted.

Cardiac tamponade manifests in an acute, subacute or chronic form, depending on the time of intrapericardial fluid accumulation and the effectiveness of compensatory mechanisms. In the case of SLE, the symptoms had a year of evolution, worsening one month before hospital admission, with an insidious onset. In the case of tuberculosis, the onset was acute, with clinical worsening within 72 hours.

Clinically, it presents with dyspnea, orthopnea, chest pain and anxiety, coughing, hoarseness, hiccups, nausea and dysphagia may ensue. The first patient had chest pain, dyspnea and orthopnea, unlike the second case with only chest pain, justified by pneumonia. The most common findings are hypotension and tachycardia, the latter constituting one of the most evident signs of impending tamponade, present in both. Classically, the CA is hypophonic, also present. Pulsus paradoxus (a reduction of more than 10 mmHg in systolic BP during inspiration) is a characteristic sign, neither always present nor specific[2].

Table 1. Etiological investigation in both cases.

ETIOLOGY INVESTIGATION	
Case 1	**Case 2**
• Microcytic, hypochromic anemia (Hb 8.4 g/dL, MCV 74.7fL, MCH 23,6pg,), reticulocytes 29000/uL • lymphopenia • Haptoglobin: 331mg/dL • Direct Coombs test: positive • Sedimentation rate: 89 mm • Proteinuria/Creatinuria ratio in a 24h collection: normal • Protein electrophoresis: hypergammaglobulinemia 22.7% • Serology tests for VIH, VHB, VEB, VVZ, CMV: all negative • Tuberculin test (Mantoux): anergic • IGRAs T-SPOT: negative <u>Autoimmunity study</u> • Ac. ANA: 1:600, positive mitoses and nucleoli • Ac. Anti-dsDNA: Error • Ac. Anti-Smith: Negative • Ac. Anti β_2Glucoprotein: IgG positive, IgM Error • Ac Lupus: High, 2.4 ratio • Ac. Anticardiolipin: IgG negative, IgM Undetermined • Ac. Anti-nucleosomes: Positive • Ac. Anti-Ku: Positive • Ac. Anti-SRP: Positive • Ac. Anti p-ribosomal: Positive • Rheumatoid factor: Negative Complement: C4 8mg/dL; C3 91mg/dL; CH50> 101,2 UI/mL	• Mantoux: positive at 48h (12 mm) • IGRAs T-SPOT: positive • BAAR study in sputum: negative • Bacteriologic culture in sputum: negative • Exam Lowenstein culture test in sputum: negative • BAAR test in the gastric acid secretion: negative <u>Vídeobronchofibroscopy</u> • Positive PCR for *Mt* in bronchial biopsy • Negative Rifampin Resistance Gene • Negative PCR for *Mt* in bronchial secretions • Negative PCR for *Mt* in the BAL • Negative Grocott & Ziehl-Neelsen dye in BAL Lymph nodes and BAL: no neoplastic cells

(Ab: antibody; ANA: antinuclear drugs; BAAR: Alcohol-Acid-Resistant Bacteria; CMV: Cytomegalovirus; IGRA T-SPOT: Interferon Gamma Release Assay by Elispot technique; Mantoux: tuberculin skin test; BAL: bronchoalveolar lavage; Mt: Mycobacterium tuberculosis; EBV : Epstein-Barr Virus; HBV: Hepatitis B Virus; HIV: Human Immunodeficiency Virus).

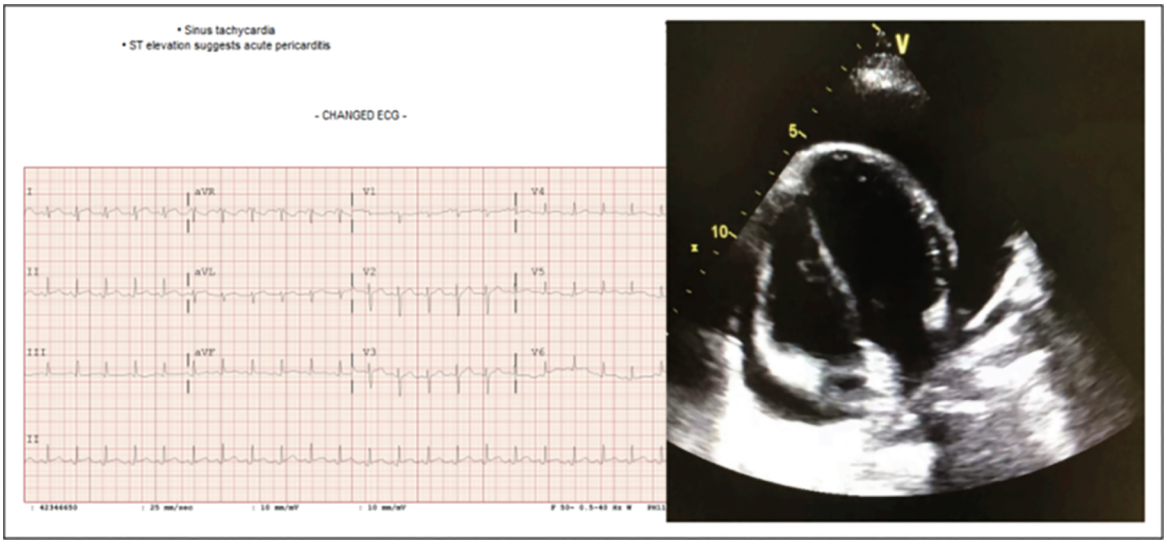


Figure 2. EKG (Case 1): sinus tachycardia with ST horizontal elevation, suggestive of acute pericarditis and Echocardiogram showing a large pericardium effusion, "swinging heart".

Upon clinical suspicion, additional diagnostic tests (spect) are required. The chest X-ray may present with no changes or with small effusions, observed in the second case. When the effusion is greater than 200mL, it is possible to identify an increase in the cardiac silhouette, “moringa heart” and superior vena cava engorgement[2,6], seen in SLE. On lateral chest radiography, a distance greater than 2 mm between the epicardial fat and the posterior aspect of the sternum, called the “epicardial fat sign” may be present[2]. If pericarditis or tamponade is suspected, an EKG should be performed. In pediatric age, the main cause of ST elevation is pericarditis (case 1). Low voltage or electrical alternation (manifested by changes in the amplitude and polarization of the

QRS complexes and/or T wave) are signals to be valued[2]. Pneumonia was confirmed by chest ultrasound, this examination was not performed *ab initio* in the second case. The echocardiogram is the test of choice and should be performed in a timely manner, enabling the characterization and quantification of the effusion, predicting its hemodynamic repercussion. Compression of the right cavities, dilation of the IVC without inspiratory collapse and "swaying heart", suggest tamponade[2,6], findings in both patients. Due to the lower muscle mass and pressure, the right heart chambers are the ones initially affected. Right ventricular collapse is the first sign of hemodynamic compromise, followed by the collapse of the ipsilateral atrium, constituting the most sensitive echocardiographic sign of tamponade[2,6], verified in the second case.

In uncomplicated pericarditis, therapy is based on treatment of the underlying cause, at rest, NSAIDs, ASA and fluid therapy in order to maintain intravascular volume and avoid hemodynamic involvement. There seems to be evidence that colchicine reduces the persistence of symptoms and the risk of relapses[2,6], the therapy used in the first case. Due to the decrease in preload, the use of diuretics is contraindicated. In cardiac tamponade, pericardiocentesis is emergent. In the event of cardiac arrest, it is essential, as the effectiveness of cardiac compressions is compromised by the stroke[2]. This procedure concurrently assists in the investigation of the underlying cause. Pericardiocentesis was performed without complications in both patients, proceeding to the study of the PF. In the patient with SLE, there was fluid with serosanguineous content (suggestive of tuberculosis, neoplasia, connective tissue disease, uremia or idiopathic), with inflammatory characteristics, excluding infection and neoplastic pathology. Considering the clinical and other findings indicative of autoimmune disease and the investigation performed, the diagnosis of SLE was made. In the second case, although the PF showed a negative PCR for Mt, it showed an ADA value of 53 U/L, suggestive of tuberculosis (ADA > 50 U/L), which was confirmed, combining the clinic, history and investigation.

CONCLUSION

Due to the medical emergency that cardiac tamponade constitutes, and due to its low frequency in pediatric age, high clinical suspicion and rapid intervention are essential, with echocardiography and pericardiocentesis being the exam and therapy of choice[1].

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