

Purulent pericarditis: early ultrasound diagnosis in the emergency department

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The diagnosis of purulent pericarditis (PP) faces several challenges at the present time. The first of them is the epidemiology of the disease. PP is very uncommon and requires the physician best efforts to avoid a fatal diagnostic delay. The second challenge is the aetiology of the disease. On one hand deep changes in the bacteriological spectra has been reported since the antibiotic drug era began. An increasing trend of PP cases are now caused by anaerobic, gram- and fungus species in contrast with gram+ bacteria cultured before. There is also a predominant role of underlying conditions neither necessarily infectious nor previously diagnosed that increase the risk for PP. On the other hand a controversy related to the most efficient surgical drainage technique still remains. We report a case of a patient with PP who underwent bedside ultrasound at emergency department that reduced both diagnostic and treatment times and achieved an important aim: the patient remained in the most adequate setting for medical assistance, the Emergency Department. [Emergencias 2008;20:135-138]

Key words: Pericarditis. Cardiac tamponade. Emergency services, medical. Ultrasonography. Pericardial effusion.

Introduction

Purulent pericarditis – defined as purulent fluid in the pericardial sac – can be lethal if not tackled immediately with the current mortality ranging between 2 and 20%¹.

In recent years the percentage of infectious aetiology in up to 85% of cases (usually Gram-positive cocci) has been reduced to 22%². The types of predisposing pathology and incidence thereof have also changed (chronic renal failure, connective tissue diseases, etc.) due to the development of new medical therapies (immunosuppressants, biological, etc.), less invasive cardiovascular surgical procedures and the universal use of antimicrobial agents.

This case of a patient diagnosed with purulent pericarditis is highly illustrative for a number of reasons; firstly, with regard to the importance for the patient of a fast and structured approach to differential diagnosis of his condition and almost immediate confirmation via early ultrasound² in the hospital emergency department (HED). Bedside

ultrasound has proven to be a suitable and accessible diagnostic procedure that significantly increases the emergencists' diagnostic capacity, to the point of having become one of the main diagnostic advances in Emergency Medicine in the last few decades. Secondly, it reveals the importance of patient clinical history, both regarding the current pathology and medical and surgical history, in understanding the aetiopathogenesis of this process and immediate action.

Clinical case

A 62-year-old male presented at the HED in January 2006 with a 5-day progressive symptomatology including asthenia, precordial oppression, dyspnoea at rest and sweating. Medical history included seropositive rheumatoid arthritis (RA) diagnosed in 1981 with a poor response to treatment (methotrexate, prednisone, leflunomide and etanercept), two episodes of diverticulitis (in 1997 and 2002) and peritonitis secondary to diverticu-

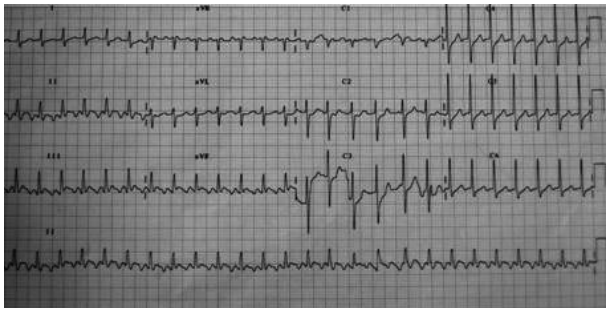


Figure 1. ECG: Atrial flutter 2:1 (no previous episodes in patient history).

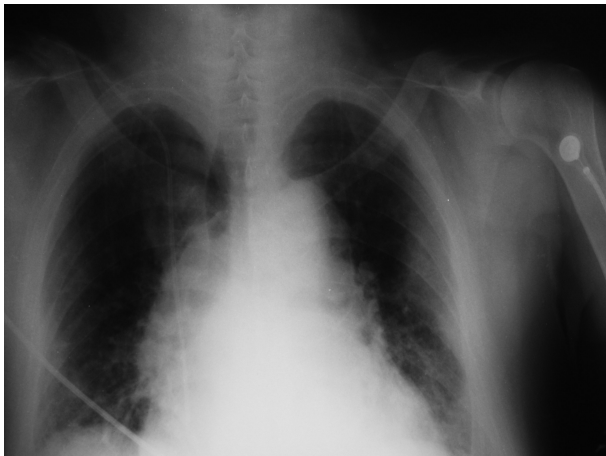


Figure 2. Chest X-ray (decubitus). Non-assessable cardiomegaly, Discrete mediastinal widening.

lar perforation. The initial physical exploration showed respiratory distress (28 bpm), 88% Sat = 2, low blood pressure in decubitus position 70/50 mmHg and signs of poor tissue perfusion (sweating, paleness) in addition to jugular ingurgitation. Cardiac auscultation was arrhythmic with no murmurs (150 bpm) with diffuse pain upon abdominal palpation. The ECG performed in the triage area (Figure 1) showed type I atrial flutter (Negative F waves in lower side) with 2:1 conduction ratio (150 bpm). The clinical situation, interpreted as cardiogenic shock due to tachyarrhythmia, led to the performance of emergency cardioversion procedures at the HED with recovery of sinus rhythm (120 bpm) but not blood pressure levels.

Biochemistry revealed hyperglycaemia at 273 mg/dl, mild renal failure (1.4 mg/dl creatinine) and 0.11 mg/dl of troponin I (normal range: 0.06-0.5) with normal creatininekinase values. The haemogram showed 28.5×10^6 leukocytes/l (85.8% polymorphonuclear), 487×10^6 platelets/l and haematocrit count of 0.43 l/l. The simple urine test showed microscopic glycosuria and hae-

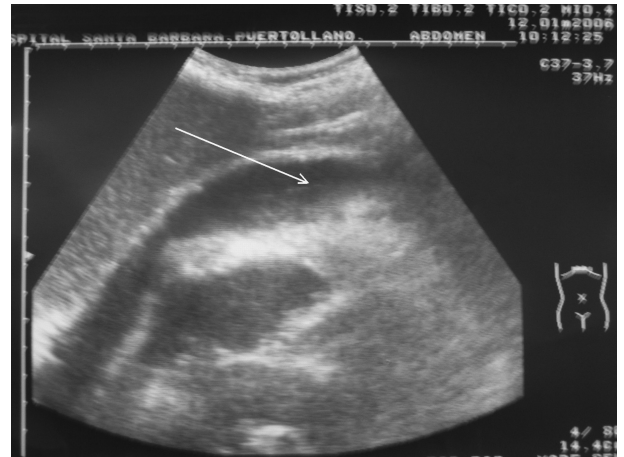


Figure 3. Bedside ultrasound performed by Emergency Department personnel: Pericardial effusion compressing right cavities to point of collapse (arrow).

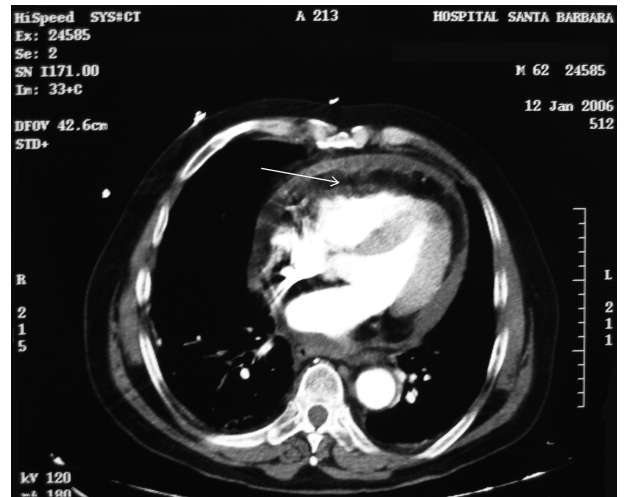


Figure 4. Thoracic CT scan: collapse of right cavities and thick, heterogeneous and septated pericardial effusion (arrow).

maturia. Chest radiography (decubitus) revealed an unassessable cardiomegaly and possible mediastinal widening (Figure 2).

Performance of an early on site ultrasonic study was decided in order to eliminate other causes of shock (aortic dissection, cardiac tamponade, etc). Ultrasound revealed the presence of pericardial effusion with compression on cavities (mainly to the right), with no sign of cardiac rupture or aneurysm. Additionally, the ultrasound showed absence of pleural effusion, moderate hepatic stenosis, distended suprahepatic veins and non-collapsible inferior vena cava of 3 cm in diameter.

A central line was inserted via the jugular vein assuming a central venous pressure of 14.5 mm

H₂O and thoracic-abdominal CT scan revealed the presence of significant pericardial effusion with a maximum of 16 mm of thickness, minor bilateral pleural effusion and chronic interstitial lung disease. The emergency department diagnosis was pericardial tamponade due to pericardial effusion with no structural lesions.

The patient was admitted to the intensive care unit where an emergency ECG-guided pericardiocentesis was performed, which failed to reveal findings due to viscosity and coagulability of the effusion. Haematic and purulent material was extracted via subxiphoid pericardiotomy. Given the cellularity of the extracted fluid (19.6×10^6 leukocytes/l with 86% neutrophils), antibiotic treatment with vancomycin, cefepim and tobramycin was begun. The patient progressed favourably. The control ECG after 24 hours revealed absence of significant pericardial effusion. Subsequent good progress on the ward resulted in patient medical discharge with 24 days of antibiotic treatment. Blood and urine cultures performed at the HED were negative, whereas the pericardial fluid culture revealed the presence of *Proteus mirabilis*.

Discussion

Pericardial involvement is the cardiac condition most frequently associated with RA, with a higher incidence over time ranging between 1.6 and 2.4%, and with a higher prevalence among males. Forms of manifestation can range from acute and exudative-constrictive pericarditis to cardiac tamponade, the least prevalent. Bearing in mind that several descriptive epidemiological studies based on ecocardiographic controls reported an incidence of 30%³ of pericardial effusion of no clinical repercussion associated with RA, it is very probable that our patient had had previous asymptomatic pericardial effusion.

The diagnostic suspicion of cardiac tamponade was based on the finding of cardiac biventricular failure with insufficient filling of both ventricles. Immediate confirmation was based on the presence of fluid in the pericardial sac as shown in the portable ultrasonograph performed by the authors in the HED.

The definitive diagnosis of purulent pericarditis in this patient is defined as the presence of an infection in the pericardial sac which produces a micro- or macroscopically purulent fluid. The condition is fatal if untreated and carries a high mortality rate.

Among the pathogenic mechanisms of purulent pericarditis described in the literature⁴, it is reasonable to discard the direct spreading of an intra-thoracic focus, including myocardial infection, or trauma and/or thoracic surgical procedures. Its origin must therefore be found in haematogenous dissemination or spreading of a subdiaphragmatic infection focus. The medical history of the patient (perforated diverticulitis with associated peritonitis requiring colostomy) contained examples of subdiaphragmatic infectious foci and septic sources of possible haematogenous spreading. The second hypothesis is the most likely, given that spreading from a diverticular focus would have required greater anatomical proximity. The CT scan, moreover, did not reveal abdominal foci⁵.

The pathogenic diagnosis, but not syndromic diagnosis, was delayed by the presence of tachyarrhythmia in addition to the pericardial tamponade. Once the tamponade was identified as the main cause of the symptoms, a final diagnosis and the restoration of a haemodynamic balance could only be achieved via pericardiocentesis.

The treatment is based on haemodynamic support measures, drainage and early use of wide spectrum antibiotics, subsequently adjusted to the antibiogram if the bacterial responsible for the infection has been identified. Empirical therapy, both in immuno and non-immunosuppressed patients, must address Gram positive and Gram negative bacteria⁷. Recommended combinations include vancomycin plus ceftazidime or cefotaxime or gentamicin or alternatively, monotherapy with imipenem or meropenem, ticarcillin or piperacillin, tazobactam or cefepim. A combination with fluconazole is recommended for severely immunosuppressed patients.

With regard to pericardial drainage, the simplest, most frequent and fastest method to extract pericardial fluid is pericardiocentesis, but loculations, fluid viscosity or the septa may hinder drainage, despite inoculation of fibrinolytic agents. Subxiphoid pericardiotomy usually enables better drainage, as it allows for manual removal of adhesions and is the method recommended in the 2004 European Cardiology Society guidelines. Other techniques, such as pericardial stripping or video-assisted surgery present a higher risk for the patient⁷.

As for the causal agent, *Proteus mirabilis* is a Gram negative enteric bacillus that causes infections in extended care centres and hospitals. *Proteus* species can form part of colonic flora (50% healthy humans) although their preferred location is the urinary tract. Although most of the infections manifest

in the urinary tract, these bacteria can occasionally produce pneumonia (especially in patients admitted in chronic care centres or hospitals), nosocomial sinusitis, intra-abdominal abscesses, biliary tract infections, surgical wounds, soft tissue lesions (diabetic or decubital ulcers) and osteomyelitis.

Cardiac tamponade is an uncommon disease and is infrequently caused by purulent pericarditis, which makes this case even rarer. However, the most relevant factor is that diagnostic confirmation was made via ultrasonography performed by emergency physicians, despite not being the ideal method, not having the proper sound (5-7 MHz instead of a convex 3.5 MHz) nor the right sonography equipment⁸, and bearing in mind that substernal projection is perhaps not the best.

This technique is adapted to the premises of an "Emergency Ecography", that is, available 24/7, performed by emergency physicians at patient bedside, portable, fast and focused on a clinical question, and can be performed simultaneously with any other procedures⁹.

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Pericarditis purulenta: diagnóstico ecográfico precoz en el servicio de urgencias

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La pericarditis purulenta presenta, en la actualidad, diversos retos. Primero, en el aspecto epidemiológico, a su rareza lo que nos exige tenerla siempre *in mente* para evitar un retraso fatal en el diagnóstico. Segundo, en el aspecto etiológico debido a los cambios en el espectro bacteriológico responsable del cuadro (desde el descubrimiento y generalización de las drogas antimicrobianas han aumentado los casos asociados a gérmenes anaerobios, gram negativos y fúngicos frente a los clásicos aerobios gram positivos de otras etapas), así como por su cada vez mayor vinculación con enfermedades y condiciones predisponentes, no necesariamente infecciosas ni previamente conocidas. Tercero, en el aspecto terapéutico, alimentado por la aún viva polémica en torno a la técnica de drenaje más eficaz. Presentamos un caso de pericarditis purulenta en el que la práctica de la ecografía a la cabecera del enfermo acortó los tiempos de diagnóstico y tratamiento del proceso y permitió que el paciente no abandonase el medio y lugar más apropiado: el Servicio de Urgencias. [Emergencias 2008;20:135-138]

Palabras clave: Pericarditis purulenta. Taponamiento cardiaco. Ecografía en urgencias. Derrame pericárdico.