

Post-cardiac injury syndrome. Post-traumatic pericarditis apropos of a case

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ABSTRACT

Post-traumatic pericarditis is included in post-cardiac injury syndromes. This syndrome is an inflammatory process that affects the pericardium, pleura, or both, and it is secondary to myocardial infarction, thoracic trauma or cardiac surgery. Clinical features include chest pain of pericardial origin, fever and elevated acute phase reactants. It usually responds satisfactorily to treatment with nonsteroidal antiinflammatory drugs and corticosteroids, but the lack of diagnostic tests can lead to unnecessary studies or inadequate treatment with possible side effects. The case of a young man who progressed favorably after initiating appropriate treatment is presented.

Key words: Pericarditis, Colchicine, Post-cardiac injury syndrome

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RESUMEN

La pericarditis postraumática se incluye dentro de los síndromes posesión cardíaca. Este síndrome es un proceso inflamatorio que afecta al pericardio, la pleura, o ambos, secundario a un infarto de miocardio, a un traumatismo torácico o a una cirugía cardíaca. Las características clínicas incluyen el dolor torácico de perfil pericardítico, fiebre y elevación de los reactantes de fase aguda. Generalmente responde de manera satisfactoria al tratamiento con antiinflamatorios no esteroideos y corticoides, pero la falta de pruebas diagnósticas puede llevar a estudios innecesarios o tratamientos inadecuados con posibles efectos secundarios. Se presenta el caso de un hombre joven que evolucionó favorablemente tras la instauración del tratamiento apropiado.

Palabras clave: Pericarditis, Colchicina, Síndrome posesión cardíaca

INTRODUCTION

Post-cardiac injury syndrome includes three different conditions; post-infarction pericarditis, post-pericardiotomy syndrome and post-traumatic pericarditis. The underlying cause of heart damage is highly variable; from necrosis or

heart surgery to minor injuries —often unnoticed— of the pericardium, following percutaneous coronary intervention, the insertion of stimulation electrodes or radiofrequency ablation. Although not known for certain, it is postulated that the pathogenesis of this syndrome is autoimmune, initiated by the combination of damage to the pericardial mesothelial cells and presence of blood in the pericardial sac¹. It is believed that the initial injury stimulates the release of cardiac antigens leading to antibody formation. As a result, immune complexes are detected in the pericardium, pleura and lungs. They are believed to be responsible for the inflammatory response, via the activation of the complement, with the corresponding clinical manifestation²⁻³.

CASE REPORT

A 32-year-old, male patient, who was an active smoker and had no other medical history, was admitted to the emergency room for chest a pain with characteristics of pericarditis. The pain had six hours of evolution, without infectious clinical symptoms in the previous days. An electrocardiogram was performed, which showed sinus rhythm with mild diffuse concave ST segment elevation (**Figure**) without elevated markers of myocardial necrosis.

Suspecting an acute pericarditis, a transthoracic echocardiography was performed. It showed a normal systolic function without segmental alterations in contractility, and no pericardial effusion. Therefore, a treatment with ibuprofen is started on an outpatient basis.

In the absence of clinical improvement, and with a fever up to 38 °C and malaise, the patient went to the emergency room of our hospital where an empirical antibiotic therapy is started and chest radiograph is performed, showing a cardiomegaly that was not present in previous studies.

The medical history informed of a high-energy midthoracic trauma, two months earlier, at the patient's work, which had not been previously identified. There was bruising at that level, which resolved spontaneously. The patient had malaise and a stable hemodynamic status. Initial blood tests did not detect elevated markers of cardiac necrosis and highlighted the presence of a slight leukocytosis along with elevated erythrocyte sedimentation. Microbiological studies and the determination of antibodies were negative.

A new transthoracic echocardiography was performed. It showed normal systolic function with small amount of pericardial effusion in the posterior pericardial sac. With the diagnosis of post-traumatic pericarditis the dose the nonsteroidal antiinflammatory

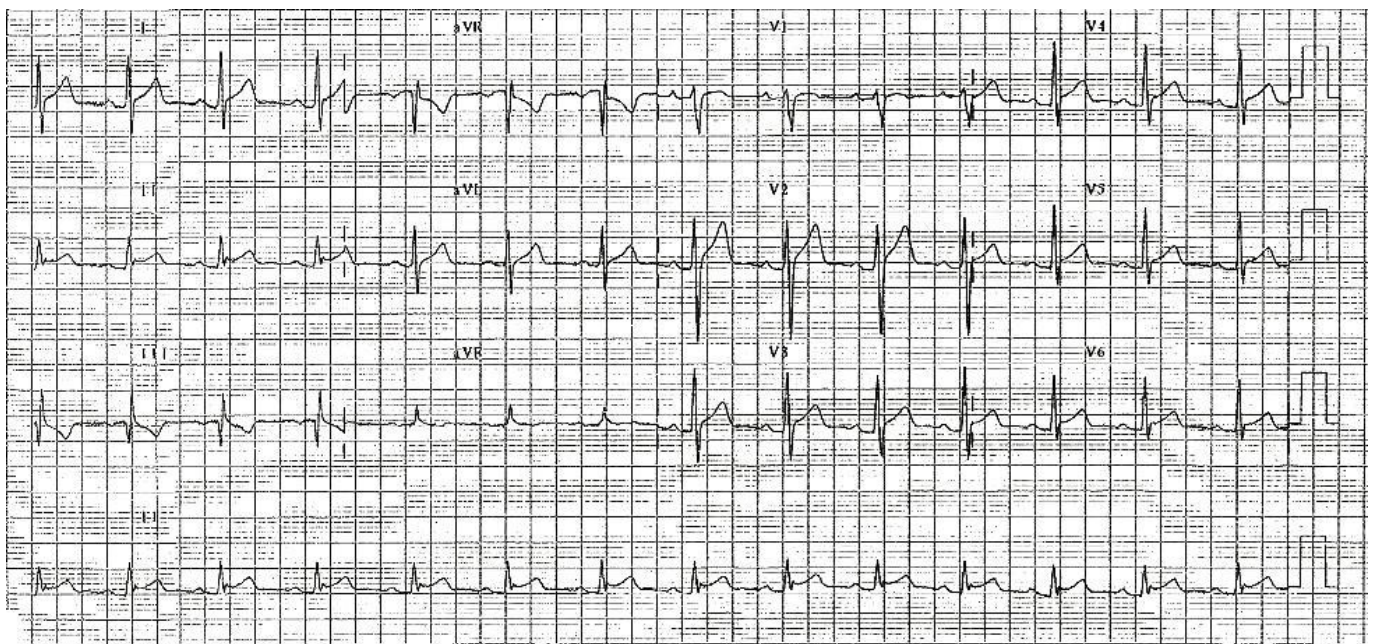


Figure. A 12-lead electrocardiogram. Diffuse concave ST segment elevation.

drug was increased, associated with colchicine. The presence of intrathoracic collections was discounted by a thoracic CT scan. The patient had a favorable outcome and was discharged with colchicine and ibuprofen treatment.

COMMENT

The clinical manifestations of patients with post-cardiac injury syndrome are similar to those of patients with acute pericarditis. Most patients have chest pain (> 80%), usually with the characteristic of pericarditis, fever (> 50-60%), elevated inflammatory markers (erythrocyte sedimentation rate, C-reactive protein) and pericardial effusion (> 80%), which is usually mild. Dyspnea is present in 50-60% of patients, pericardial friction rub in 30-60%, and changes in the electrocardiogram in 20% of them⁴. This complementary is rarely normal (especially after myocardial infarction or heart surgery) and often reflects the underlying clinical condition. ST segment elevation and the depression of the PR interval are distinctive signs of acute pericarditis, but the classic electrocardiogram changes are not usually present in these patients.

It is essentially a diagnosis of exclusion. However, post-cardiac injury syndrome includes a number of distinguishing characteristics that lead to the diagnosis; the presence of a previous injury of the pericardium or myocardium, the latency period between pericardial injury and the onset of symptoms (days to months), the tendency to recur, and sometimes the presence of pleural effusion and pulmonary infiltrates¹.

Treatment is based on the empirical use of non-steroidal antiinflammatory drugs, while the concomitant use of colchicine may be useful in preventing recurrences⁵. The use of corticosteroids in low doses is useful when nonsteroidal antiinflammatory drugs are not effective, are contraindicated or poorly tolerated^{6,7}.

Colchicine significantly reduces the incidence of post-pericardiotomy syndrome in patients undergoing cardiac surgery⁵. In the COPPS-2⁸ study, the treatment with colchicine during the month after surgery significantly reduced the incidence of this syndrome at 12 months, at the expense of a significant rate of adverse effects, mainly gastrointestinal ones; a fact which was not evident in the COPPS⁹ study. Colchicine treatment in post-cardiac injury syndrome is assumed to be ef-

fective, although there is no corroborating information.

There are few published data on the prognosis of post-cardiac injury syndrome, which is generally considered a benign condition with a recurrence rate of 10-15%. Possible complications include pericardial constriction, which is estimated at 2-5% at 72 months, a slightly higher incidence compared with idiopathic pericarditis (1%) and lower compared with purulent pericarditis (20- 30%)¹⁰.

Our patient was treated with ibuprofen (starting with 2400 mg/d, and gradual decrease of the dose) and colchicine (0.5 mg/12h), for a month. He remains asymptomatic after 12 months without recurrence of clinical symptoms or indication of pericardial constriction on the echocardiography. This is the usual course of traumatic pericarditis, an uncommon cause of post-cardiac injury syndrome.

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