Severe Pericardial Effusion. Percutaneous Balloon Pericardial Window

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ABSTRACT

Most patients with chronic pericardial effusion are women aged 50 years or older. This presentation describes a 63-year old female patient with severe chronic pericardial effusion; she had received radiotherapy for cancer of the left breast, had a history an unconfirmed presumptive diagnosis of pulmonary tuberculosis and was under treatment for hypothyroidism. A pericardiocentesis was performed; nevertheless the patient presented recurrence of the pericardial effusion and was treated with NSAIDs. As this therapy failed, she underwent a percutaneous pericardial window with no adverse outcomes. The patient started treatment with colchicine. The potential causes of the pericardial effusion were tuberculosis, malignant neoplasms, hypothyroidism or radiotherapy. The latter etiology was considered as the most probable once the others had been excluded. An echocardiogram performed one month after the procedure showed no signs of pericardial effusion.

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Key words > Pericardial effusion - Pericardiocentesis

Abbreviations >

ADA	Adenosine deaminase	**CRP**	C-reactive protein
NSAIDs	Non-steroid anti-inflammatory drugs	**MRI**	Magnetic resonance imaging
AFB	Acid-fast bacillus	**TBC**	Tuberculosis
FC	Functional class	**CT**	Computed tomography
LVSF	Left ventricular systolic function		

CLINICAL PICTURE

A 63 year-old female patient consulted because of dyspnea in FC II. She had a history of left-sided breast cancer in 1999 treated with surgery, tamoxifen and radiotherapy; she had meningitis when she was 18 years old, was under treatment for hypothyroidism and had an unconfirmed presumptive diagnosis of pulmonary tuberculosis (a live-in partner with pulmonary tuberculosis) but was not receiving prophylaxis. At the moment of the visit her weight was 42 kg.

She underwent assessment of thyroid function and oncological and pulmonary status with no evidence of active disease. An echocardiogram revealed moderate pericardial effusion. A computed tomography scan of the chest ruled out the diagnosis of constrictive pericarditis and revealed the presence of images localized at the posterior area of the right upper lobe consistent with residual lesions in the pleura and pulmonary parenchyma, peribronchial thickening of the middle lobe and a right sub-pleural cyst. The patient started therapy with NSAIDs which she later discontinued. Thus, expectant management was decided.

She remained symptomatic with progression of dyspnea to FC II - III. In February 2007 the patient started complaining of orthopnea; the echo-Doppler showed the presence of severe pericardial effusion with predominance in the anterior pericardium, partial collapse of the RA and normal LVSF.

A pericardiocentesis was performed with a minimal invasive pericardial approach. A drainage catheter was implanted in the pericardial sac using the Seldinger technique and remained in place for 48 hours; pericardial fluid was analyzed thereafter. Physical characteristics of the fluid and chemical tests were normal; direct Gram and AFB stains on the pericardial fluid were negative; cytological examination revealed few epithelial cells and *absence of neoplastic cells*.

The patient remained under therapy with NSAIDs. She evolved with deterioration of her functional class (III-IV); in August 2007 she presented orthopnea and severe pericardial effusion (Figure 1). The clinical examination revealed absence of pericardial rub, fever or pain; the ECG showed low voltage QRS complexes. In consequence, a percutaneous pericardial window was performed under intravenous sedation. The subxiphoid area was anesthetized and a conventional pericardiocentesis was performed; a yellowish crystalline fluid was drained and samples were obtained to

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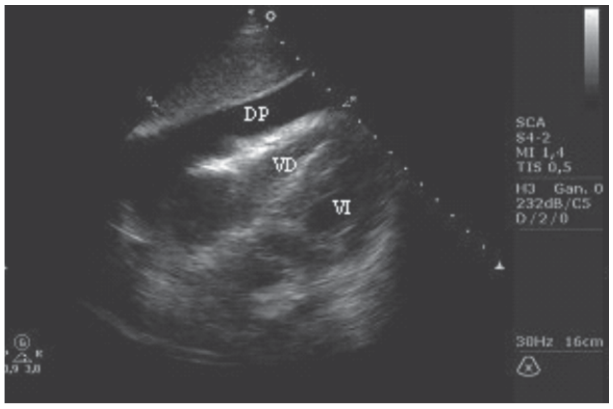

Fig. 1. Two dimensional echocardiography (subcostal view) shows severe pericardial effusion (DP). VD: Right ventricle. VI: Left ventricle.

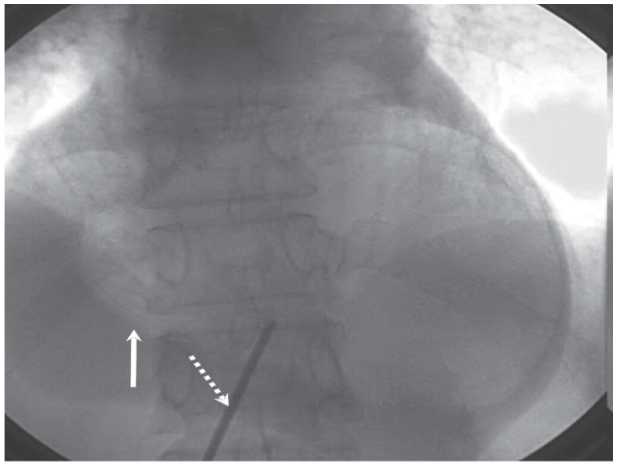

Fig. 2. Image obtained after the injection of 10 cm^3 of iodinated contrast agent into the pericardial sac. The edge of the cardiac silhouette can be distinguished as a radiolucent image which outlines the parietal pericardium. *Solid arrow:* cardiac silhouette and visceral pericardium. *Dotted arrow:* the catheter is advanced through the parietal pericardium.

analyze physical, chemical and cytological characteristics, for bacterial cultures, ADA, and CPR for AFB. Then, 10 $cm^3$of radio contrast were injected into the pericardial space to aid in visualization of the parietal pericardium (Figure 2). A 0.035-inch guide-wire was placed in the pericardial sac and a 12-French introducer sheath was advanced over the guide-wire. Two dilatations with 16-mm and 20-mm balloons were performed until the waist of the pericardium over the balloon was lost (Figure 3) producing a tear in the pericardium that created a pericardial and pleural window. After dilatation, the remaining 300 cm^3 of pericardial fluid was removed and a pig tail catheter was placed for continuous drainage. The patient remained in sinus rhythm during the procedure and did not present arrhythmias.

Immediately after the end of the procedure, she complained of intense chest pain that relieved with NSAIDs. The draining catheter was removed 24 hours after and the volume drained was 50 ml; the chest x-ray showed a left-sided pleural effusion with no evidence of pericardial effusion in the echocardiogram.

The physical and chemical analysis of the pericardial fluid revealed an exudate with absence of neoplastic cells, an ADA value of 6 UI/L, negative CPR for AFB and negative cultures.

DISCUSSION

Management of chronic pericardial effusion with a volume > 20 mm at the echocardiogram and with no signs of cardiac tamponade is controversial; (1) however, as we had previously decided not to drain the pericardium, we were able to assess its probable causes under a strict clinical surveillance. Some authors recommend performing pericardiocentesis when the size of pericardial effusion is > 20 mm at the echocardiogram. (2)

Firstly, we performed a pericardiocentesis to drain the fluid out in order to resolve the effusion, study the etiology (7-26% of diagnostic efficacy) (1) and prevent its progression towards cardiac tamponade. (2)

As the effusion recurred and therapy with NSAIDs failed, a percutaneous pericardial window (3) was performed and the pericardial fluid obtained was analyzed to rule out tuberculosis, hypothyroidism, neoplasms or radiation as the most probable etiologies of the condition.

The diagnosis of pericardial effusion secondary to hypothyroidism was ruled out as the patient remained with normal thyroid function during follow-up and there was no evidence of hypothyroidism in the results of the physical and clinical characteristics of the pericardial fluid. Antithyroid antibodies were negative, and hypothyroidism was attributed to radiotherapy.

The pericardium is a frequent site of metastases of neoplasms of the lung, breast and blood. In this particular case, the oncological assessment determined that the patient was free of disease and the citological study of the fluid (obtained from the pericardial drainage in March an from the pericardial window) had no evidence of atypical cells; thus, the presence of metastases in the pericardium was excluded, (4) even in the absence of a pericardial biopsy. According to the literature, the diagnosis of neoplastic pericarditis is based on identifying malignant cells in the pericardial fluid or on the presence of neoplastic invasion in the pericardial tissue (4); and in two-thirds of patients the malignant pericardial effusion is caused by non malignant diseases (post-radiotherapy pericarditis, opportunistic infections). Chest x-ray, CT and MRI might show mediastinal thickening, hilar masses and pleural effusion. The analysis of the pericardial fluid and pericardial or epicardial biopsies are necessary to confirm a malignant disease of the pericardium (level of evidence B, class I indication). (5)

ADA levels and CPR for AFB were negative and *Mycobacterium tuberculosis* was ruled out as the cause

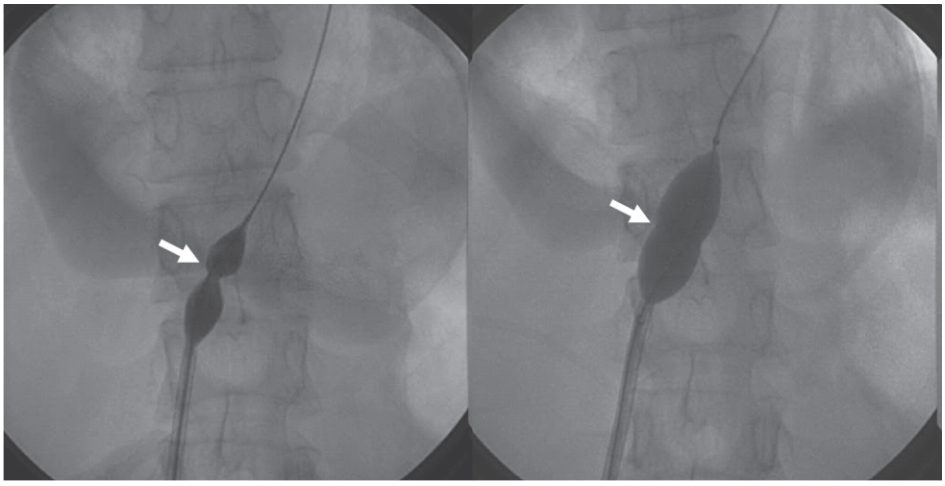

Fig. 3. Balloon inflation in two stages, with the notch (*arrow*) in the parietal pericardium.

of the pericardial effusion. High levels of ADA activity (> 45 U/ml) suggest tuberculosis, while PCR is as sensitive as ADA (75% versus 83%) but more specific (100% versus 78%) for the diagnosis of tuberculous pericarditis. (5)

As fluid cultures were negative, unspecific infections were also excluded; the presence of an exudate in the pericardial fluid could only be explained by the previous pericardiocentesis.

Finally, once excluding the aforementioned etiologies, and with the history of left-sided breast cancer treated with radiotherapy, the most probable diagnosis is post-radiotherapy pericarditis. Post-radiotherapy pericarditis may occur early or late during follow-up, as an acute pericarditis, pericardial effusion or constrictive pericarditis. It is important to distinguish it from a relapse of the cancer in the pericardium or from a pericardial effusion secondary to radiotherapy-induced hypothyroidism. (4) Chronic pericardial effusions are more frequent in women > 50 years old. (6)

Percutaneous balloon pericardiotomy should be considered for frequent and symptomatic recurrences. (5) The balloon produces a localized tear in the parietal pericardium that allows drainage of the pericardial fluid into the pleural sac. The pericardial window is seen with a flexible fiberscope as a communication with the left-sided pleural sac. According to previous experience with subxiphoid pericardial window by surgical approach, the communication between pericardial and pleural cavities does not last for ever. The success of the pericardial window is dependent on the inflammatory fusion of the epicardium to pericardium and not on the maintenance of the window. Drainage of pericardial fluid through a percutaneous window is more effective as the absence of fluid in the pericardial cavity facilitates the fusion. (7) Thus, the presence of pleural effusion in chest X-Rays reveals that the pericardial fluid is draining into the pleural cavity. (3, 5)

Symptomatic management includes exercise restriction and use of drug therapy as in acute pericarditis. As our patient had not responded to NSAIDs, therapy with colchicine was started after the procedure, in a dose of 2 mg/day for 48 hours, followed by 1 mg/day thereafter. The medication was well tolerated. Colchicine has proved to be effective when NSAIDs have failed in preventing relapses. The use of prednisone is reserved for patients with frequent crisis or with poor clinical outcomes. Azathioprine and cyclophosphamide may be used when the other options have failed. Pericardiectomy is indicated in cases of refractoriness to medical treatment. (5)

A month after performing the pericardial window, the patients remained asymptomatic, under treatment with colchicine and with no signs of pericardial effusion in the echocardiogram.

RESUMEN

Derrame pericárdico grave. Ventana pericárdica percutánea con balón

La mayoría de los pacientes con derrame pericárdico crónico son mujeres y mayores de 50 años. En esta presentación se describe el caso de una paciente de 63 años con derrame pericárdico crónico grave, con antecedente de carcinoma de mama izquierda irradiado, diagnóstico presuntivo no confirmado de tuberculosis pulmonar e hipotiroidea sustituida. Ante la recurrencia del derrame luego de pericardiocentesis y el fracaso del tratamiento antiinflamatorio con AINE se decidió realizar una ventana pericárdica percutánea, sin que se presentaran complicaciones técnicas. Se inició tratamiento con colchicina y se evaluaron las posibles causas: tuberculosa, oncológica, secundaria a hipotiroidismo o por radiación. Por exclusión se llegó a la etiología radiante. Al mes de la realización de la ventana pericárdica no se observaba derrame pericárdico en el ecocardiograma.

Palabras clave > Derrame pericárdico - Pericardiocentesis

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