A 58-year-old man was admitted for relapsing pericarditis. His past medical history included a transient ischemic attack when he was 47 years old, radical right nephrectomy for neoplasia 8 years earlier, and hypertension.

One year earlier, a diagnosis of pleuropericarditis was made in another hospital. The patient was febrile and complained of chest pain; an echocardiogram showed thickening of parietal pericardium, hyperechoic matter on visceral pericardium, and the presence of pericardial and pleural effusion, without hemodynamic impairment. Concomitantly, the patient had a dental abscess; cultures from this abscess were negative during antibiotic therapy. He was treated empirically with piperacillin/tazobactam for 15 days and then with amoxicillin/clavulanate for 1 month and aspirin 500 mg 3 times daily for maintenance.

The patient experienced 2 further relapses, which were treated successfully with aspirin, but soon after aspirin discontinuation, pain and fever recurred. One year after the original attack, he was admitted to our hospital for pericardial pain and fever with chills. Blood cultures were persistently negative. Laboratory tests were remarkable for neutrophilia and an increase of C-reactive protein (32 mg/dL). The echocardiogram showed a lesion in the anterior pericardial space (Figure 1; Movie I in the online-only Data Supplement). A computed tomography scan (Figure 2) revealed a polilyobated lesion in the anterior pericardial space with peripheral contrast enhancement and a necrotic core. Computed tomography–positron emission tomography showed intense uptake at the level of the anterior wall of the right ventricle (Figure 3). Magnetic resonance imaging confirmed that the mass was confined to the pericardial space (Figures 4 and 5; Movies II through V in the online-only Data Supplement).

One year after the original attack, he was admitted to our hospital for pericardial pain and fever with chills. Blood cultures were persistently negative. Laboratory tests were remarkable for neutrophilia and an increase of C-reactive protein (32 mg/dL). The echocardiogram showed a lesion in the anterior pericardial space (Figure 1; Movie I in the online-only Data Supplement). A computed tomography scan (Figure 2) revealed a polilyobated lesion in the anterior pericardial space with peripheral contrast enhancement and a necrotic core. Computed tomography–positron emission tomography showed intense uptake at the level of the anterior wall of the right ventricle (Figure 3). Magnetic resonance imaging confirmed that the mass was confined to the pericardial space (Figures 4 and 5; Movies II through V in the online-only Data Supplement).

Because of progressive hemodynamic impairment, the patient underwent total pericardiectomy; at sternotomy, a mass 4×8 cm in diameter was evident (Figure 6), which was almost totally excised. All of the surgical specimens were positive for Providencia stuartii, a Gram-negative germ; the histological examination did not reveal the presence of neoplastic cells, but only a suppurative process (Figure 7). The patient was then treated with cefepime and amikacin for 8 weeks and made a full recovery. This is the first report of bacterial pericarditis due to P stuartii.2,3 The clinical course resembled that of recurrent “idiopathic” pericarditis, with the exception that fever and pain recurred soon after aspirin discontinuation.

Disclosures

None.

References


From the Department of Internal Medicine (M.D., A.B., S.F.C., M.S., F.S.), Radiology (P.B.), Pathology (S.P.), and Cardiovascular Surgery (C.S., P.F.), Ospedali Riuniti di Bergamo, Bergamo, Italy, and CRM Unit Cardiology Department (P.P.), Ospedale Niguarda Ca’ Granda, Milano, Italy.

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/cgi/content/full/122/4/e401/DC1.

Correspondence to Antonio Brucato, Ospedali Riuniti di Bergamo, Largo Barozzi 1, 24128 Bergamo. E-mail albrucato@ospedaliriiuni.bergamo.it

(Circulation. 2010;122:e401-e403.)

© 2010 American Heart Association, Inc.

Circulation is available at http://circ.ahajournals.org DOI: 10.1161/CIRCULATIONAHA.110.943118
Figure 2. Computed tomography scan. Polylobated mass with necrotic cores and calcifications in the anterior pericardial space associated with thickening of the anterior pericardium, presenting a diffuse contrast enhancement.

Figure 3. Computed tomography–positron emission tomography shows abnormal uptake due to high glucose metabolism in the pericardial mass.

Figure 4. Magnetic resonance imaging (steady state free-precession cine image frame, 4-chamber off-axis view) shows mass expanding in the pericardial space and infiltrating the right ventricular free wall.

Figure 5. Cardiac magnetic resonance late-enhancement image shows enhancement of the pericardium; no late enhancement is visible within the pericardial mass.
Figure 6. Macroscopic view of the pericardial mass before (A), during (B), and after (C) surgery.

Figure 7. Histological picture of diffuse inflammation mainly composed of granulocytes and histiocytes associated with focal necrosis and hemorrhage. Hematoxylin and eosin stain, original magnification ×200.