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Constrictive Pericarditis Complicating Endovascular Pacemaker Implantation

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ELINAV, E., ET AL.: Constrictive Pericarditis Complicating Endovascular Pacemaker Implantation. *A patient is described who has 6 months of progressive dyspnea and peripheral edema for 4 years following implantation of an endovascular pacemaker, which was complicated by a large hemorrhagic pericardial effusion. Evaluation was consistent with constrictive pericarditis, which is an extremely unusual complication of pacemaker implantation. (PACE 2002; 25:376–377)*

constrictive pericarditis, permanent pacemaker, pericardial effusion

Case Report

An 84-year-old male tourist was admitted to the hospital with progressive dyspnea, first noted while playing golf, that progressed over a period of 5 months to marked dyspnea at rest. In addition, he developed significant peripheral edema, unresponsive to empiric diuretic treatment. Four years prior to admission the patient underwent permanent pacemaker (PPM) implantation for diagnosis of sick sinus syndrome. The patient recalled that a few hours after the procedure he felt weak, dizzy, and short of breath and was told he had "a large amount of blood around his heart." The patient was managed conservatively and was told the fluid had resolved after a few days.

On admission the patient was severely dyspneic with a normal pulse and blood pressure with no pulsus paradoxus. Jugular venous pulsation was distended to the angle of the jaw and decreased breath sounds were noted at both lung bases. Cardiac examination was normal. Abdominal examination was notable for hepatomegaly, and marked pitting edema of both lower extremities was seen.

Laboratory tests were significant for mild hyponatremia and mildly elevated alkaline phosphatase, gamma-glutamyl transpeptidase (GGTP), and lactic dehydrogenase (LDH). Erythrocyte sedimentation rate (ESR) was normal and antinuclear antibodies (ANA), C3 levels, and rheumatoid factor were negative. Chest X ray revealed bilateral pleural effusions, and electrocardiography showed (atrioventricular AV) pacing. PPD testing was unremarkable, and sputum, blood, and urine cultures for bacteria and tuberculosis (TB) were sterile. Infectious serology was significant for prior infection with Ebstein-Barr virus (EBV), cytomegalovirus (CMV), and toxoplasmosis and negative serology for human immunodeficiency virus (HIV).

Pleurocentesis revealed transudative fluid. Echocardiography demonstrated normal left and right ventricular size and function and mild mitral regurgitation. A trivial pericardial effusion was present and pulse Doppler of the mitral valve revealed an E:A ratio of 1:1.5 without inspiratory changes. Spiral computed tomography (CT) showed normal pulmonary vessels without evidence of pulmonary emboli, bilateral pleural effusions, and normal lung parenchyma. The CT was also notable for a grayish "shell" surrounding the anterior aspect of the heart, suspected to be thickened pericardium (Fig. 1). Right- and left-sided cardiac catheterization was performed. Pressure tracings revealed elevated and equalized left- and right-sided pressures and a characteristic dip and plateau or square root sign confirming the diagnosis of constrictive pericarditis.

The patient subsequently underwent pericardiectomy. At surgery a thickened, fibrotic, anterior pericardium was noted and excised. Pathological examination revealed portions of pericardial fibroadipose tissue showing fibrosis, edema, and fibrin deposition. Dyspnea and peripheral edema gradually disappeared after surgery and the patient resumed his normal daily activities. One year following surgery he was feeling well, had resumed playing golf, and was free of shortness of breath and peripheral edema.

Constrictive pericarditis (CP) is an extremely rare complication of PPM implantation.[1–5] In the present case, the post-PPM effusion, and the lack of other possible causes, by history or diagnostic evaluation, led to the conclusion that the patient's illness was secondary to the PPM implantation 4 years earlier.

Four previous cases of PPM related CP have been described in the literature, only one of which was described following endovascular placement,

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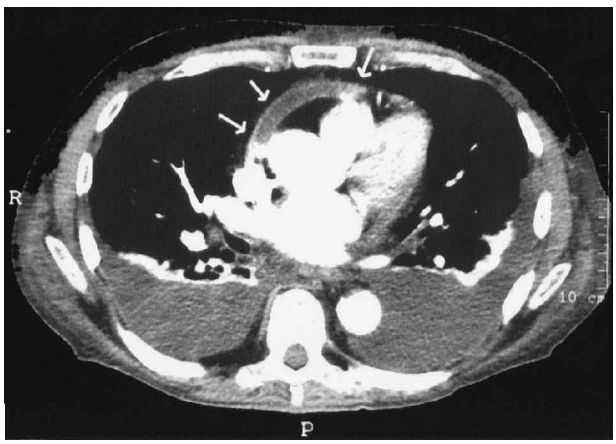

Figure 1. *Computed tomography scan showing anterior thickened pericardium (arrow) and bilateral pleural effusions.*

as in the present case.[6–9] Two of the patients suffered from immediate accumulation of blood in the pericardium presumably due to microperforation. Two others developed acute pericarditis within a month, consistent with a Dressler-like autoimmune pericarditis. Diagnosis was difficult and required multiple imaging modalities, and in all cases catheterization, to confirm the diagnosis. One of the four patients has undergone CT and magnetic resonance imaging, both revealing marked pericardial thickening.[6] Both imaging modalities enable a highly accurate measurement of pericardial thickening, and thus play an important role in the diagnosis of constrictive pericarditis.[10,11] Of interest is the limited sensitivity of echocardiography for the diagnosis of a thickened pericardium having been missed in three of the five cases. All patients had significant clinical improvement following pericardiectomy.[6–9]

Conclusion

CP is an extremely rare complication of endovascular PPM placement, which may develop following perforation by the electrode, or reactive pericarditis. The diagnosis may be elusive and a high index of suspicion and multiple imaging modalities may be necessary. As in other cases of CP the treatment of choice is pericardiectomy, which is often curative.

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