Coronary Artery Fistula Formation Secondary to Permanent Pacemaker Placement

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We present the findings in two patients who apparently developed a coronary artery fistula as a complication of an endocardial pacing electrode. This complication may actually be occurring more frequently than recognized because the patient may be asymptomatic or minimally symptomatic and therefore not undergo a coronary angiogram. Awareness of this potentially serious complication is important and stresses the need for proper electrode placement without excess pressure on the tip. (Chest 1991; 99:780-81)

Permanent cardiac pacing is a well-established therapy for a variety of rhythm disturbances, including complete heart block, symptomatic bradyarrhythmias, and some tachyarrhythmias; however, pacing is not without complications, which have been reported to include lead fracture1 or malfunction,2 thrombosis and embolism, infection,3 dislodgment,4,5 myocardial perforation or dissection,6 tricuspid regurgitation, and silicone-induced endocarditis.

We report two cases of coronary artery fistula formation apparently resulting from permanent endocardial pacemaker electrode placement. Coronary artery fistula is a recognized congenital anomaly7 and has been reported to result from penetrating and nonpenetrating chest trauma but has not been reported as a complication of a pacing electrode.

CASE REPORTS

CASE 1

A 64-year-old man had a three-year history of lightheadedness and syncope. Ambulatory monitoring demonstrated sinus pauses

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Figure 1. Coronary artery fistula originating from septal artery and communicating with right ventricle after placement of pacemaker lead (case 2).
Unusual Presentation of Recurrent Wegener’s Granulomatosis*

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A 65-year-old woman presented with recurrent Wegener’s granulomatosis following two years of immunosuppressive therapy and three years of complete remission. At her initial presentation, she had a characteristic x-ray picture showing multiple nodules with total resolution of these findings at three months. Five years later, at the time of clinical relapse, her chest x-ray film showed bilateral diffuse infiltrative disease. This change in radiologic presentation upon relapse of Wegener’s has not previously been reported. Other unusual features include diffuse infiltrates as the pulmonary presentation and the long interval between cessation of therapy and relapse. We review the radiologic manifestations of Wegener’s granulomatosis.

(Wegener’s granulomatosis is a vasculitic syndrome which always involves the lungs and upper airways and often involves the kidneys as well. It is a rare disease predominantly affecting older people.1 Before therapy with prednisone, and later, before cyclophosphamide was instituted, the disease was uniformly fatal, usually within a year. Current therapy has improved the prognosis, but resistance to initial therapy and recurrence of disease is still seen.2)

The reported pulmonary radiologic manifestations of Wegener’s are varied. However, the most characteristic x-ray presentation is multiple nodules with or without cavitation. The appearance of the chest film in recurrent disease, in the few published cases, is usually similar to the initial presentation.1–3 We report here a case of recurrent Wegener’s granulomatosis which showed a dramatic change in the radiologic presentation.

CASE REPORT

A 65-year-old woman presented in August 1988, complaining of arthralgias, right-sided jaw claudication, mild shortness of breath, nasal congestion, and fevers to 39.4°C for one week. These symptoms were identical to those she had when she was diagnosed with Wegener’s granulomatosis in August 1983.

On examination she was well nourished and in no acute distress. Temperature was 39.4°C (102°F), blood pressure was 140/65 mm Hg, pulse 100 beats per minute, respiratory rate 24 per minute. Her voice had a nasal quality. She had jaw pain induced by chewing or speaking. Her chest exam revealed bibasilar crackles to the mid-lung fields. There was no evidence of cardiac failure. The rest of her physical examination was unremarkable. The CBC showed a white blood cell count of 9.7, and hematocrit value of 35.1. Erythrocyte sedimentation rate was 91 mm/hr. Serum creatinine and urinalysis values were normal.

Chest x-ray film showed a diffuse bilateral interstitial pattern (Fig 1). There was no evidence of nodules or cavity formation. CT scan confirmed the diffuse pattern without cavities with relative sparing of the apices and bases of the lungs (Fig 2). Sputum showed polymorphonuclear leukocytes with no organisms. Room air arterial blood gas values showed pH of 7.5, Po2 of 65, and PC02 of 30.

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