Dynamic Tricuspid Valve Insufficiency Produced by a Right Ventricular Thrombus from a Pacemaker

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Acquired tricuspid valve incompetence was caused by a mobile thrombus attached to a permanent right ventricular endocardial electrode wire. The rarity of this complication is noted and the importance of its consideration in the proper setting is stressed. Diagnosis was made by angiography and confirmed at surgery.

We have recently seen a patient who had a prolapsing ball thrombus attached to a permanent right ventricular endocardial pacing wire, causing dynamic tricuspid valve insufficiency along with massive pulmonary emboli. The diagnosis was made by right atrial, ventricular and pulmonary angiography and was confirmed at surgery. This is similar to a case recently reported by Kendrick et al.

Although the condition we report is obviously rare, pacemaker thrombus with embolization should be considered in a patient with an implanted pacemaker who has episodic dyspnea, tachypnea and cyanosis. In addition, epicardial pacemaker electrodes should be considered in patients at high risk for thrombosis.

CASE REPORT

A 49-year-old white man had a mitral commissurotomy in 1966 for severe mitral stenosis with good results. He did well until November, 1976, when he developed progressive dyspnea on exertion and chest pain. The patient demonstrated physical findings consistent with moderately severe mitral stenosis and insufficiency, and mild semilunar valve incompetence. Electrocardiogram at that time showed left atrial enlargement, left ventricular hypertrophy, first-degree heart block and nonspecific ST and T-wave changes. Chest roentgenogram was consistent with classic mitral configuration with severely enlarged left atrium.

Cardiac catheterization confirmed the clinical diagnosis of severe mitral stenosis with pulmonary hypertension (110/50 mm Hg). There was minimal aortic insufficiency and no aortic stenosis. Coronary anatomy was normal. There was no suggestion of tricuspid insufficiency. The patient was taken to surgery and a No. 27 Hancock porcine valve was inserted in the mitral position. The postoperative course was complicated by left hemiparesis which subsequently resolved. There were periods of asystole requiring a permanent right ventricular endocardial pacemaker set in the demand mode at a rate of 70. The patient was discharged on Warfarin (Coumadin) and did well until October, 1977, when he reported progressive dyspnea at rest of three weeks' duration.

On physical examination the pulse rate was 110/min and irregular. The patient was dusky and cyanotic. Jugular ve
dition. The contour of this wave was variable.

The echocardiogram showed a normally functioning por-
cine valve with no evidence of clot on the prosthesis. The left
atrial cavity, though enlarged, was somewhat smaller than on
preoperative examination. Selective view of the tricuspid
valve suggested multiple diastolic echoes, preceded by an
initial clear space.

At cardiac catheterization, the mean right atrial pressure
was 16 mm Hg with a large variable C-wave of 22 mm Hg
and a V-wave of 18 mm Hg. Pulmonary artery pressure was
105/53 mm Hg with a mean of 55 mm Hg. Mixed venous
oxygen saturation was 42 percent. Pulmonary capillary
wedge pressure mean was 15 mm Hg and left ventricular
pressure was 105/12 mm Hg. No significant gradient was
noted across the aortic valve. Cardiac output by indicator
dilution technique was 5.45 L/min. Ejection fraction was 49
percent. No peri-prosthetic mitral regurgitation was noted.
Right ventricular angiography and right atrial angiography
demonstrated a mushroom-shaped filling defect which ap-
ppeared to be sliding with ventricular systole from right
ventricle to right atrium, causing moderately severe tricuspid
regurgitation (Fig 1). Occasionally, the filling defect did not
 herniate through the tricuspid valve and remained in the
right ventricle, thus giving rise to the variations noted in
jugular venous pulsations and direct right atrial pressures.

Subsequent pulmonary angiogram showed total obstruction
of three of the four main pulmonary arteries (Fig 2) with
filling of the right superior pulmonary artery. With the
patient on cardiopulmonary bypass, the right ventricular
pacing wire was removed with the movable clot at-
tached (Fig 3). The right atrial incision was closed and the
main pulmonary artery was explored. Multiple attempts at
removal of clot fragments with a Fogarty catheter were
unsuccessful. The pulmonary artery was then closed. The
patient initially came off bypass. However, his condition
deteriorated, with progressive respiratory acidosis, leading to
cardiac arrest. Resuscitative efforts were unsuccessful. At
postmortem, all four main pulmonary arteries were throm-
osed with adherent clot. There was recanalization of the
superior right main pulmonary artery. The prosthetic mitral
valve was intact.

**DISCUSSION**

We believe that this is the first case reported with a
mobile right ventricular pacemaker thrombus causing
functional tricuspid incompetence. Balau et al. reported
venograms done in 49 patients following pacemaker
insertion and found evidence of thrombi in 14. Only
three had arm edema. Bernstein and coauthors, in an
eight-year followup of permanent pacemakers, found
pulmonary emboli at autopsy in 9 of 27 patients and
suggested a role for anticoagulant therapy in "high risk"
individuals. Parsonnet et al. have discussed the problems of pacemaker thrombus and associ-
ated emboli originating from cephalic vein, great veins
or right atrium, as well as the "normal pathology" in
permanent right ventricular endocardial pacemakers.

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