Hemopericardium, Anticoagulation, and an Endocardial Pacemaker

A Case Report with Description of New Auscultatory and Radiographic Signs

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SUMMARY

A case of organizing hemopericardium and cardiac tamponade in a patient with a permanent endocardial pacemaker who was maintained on anticoagulation is presented. The hemopericardium is concluded as solely due to the anticoagulation. A new auscultatory finding attributable to an endocardial pacemaker and a helpful radiographic sign of pericardial effusion in patients with these pacemakers is described.

Additional Indexing Words:
Cardiac tamponade  Warfarin  Pacemaker sound and murmur

Hemopericardium is most frequently the result of trauma, myocardial rupture following myocardial infarction, or rupture of the aortic root as a consequence of dissecting aortic aneurysm. In addition, several varieties of pericarditis may be accompanied by hemorrhagic pericardial effusion. Anticoagulation has been thought to increase the risk of intrapericardial hemorrhage and tamponade in such states. Moreover, the administration of anticoagulants has been the sole detectable reason for hemopericardium in a rare instance.

This communication will report a case of organizing hemopericardium of uncertain origin in a patient with a permanent endocardial pacemaker who was maintained on anticoagulation. A new auscultatory finding attributable to an endocardial pacemaker and a helpful radiographic sign of pericardial effusion in patients with these pacemakers will be described.

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CASE REPORT

The patient was a 59-year-old Caucasian male at the time of his death. He experienced good health until 1963 when he sustained anterolateral myocardial infarction complicated by supraventricular tachycardia. Upon recovery he was asymptomatic until January 1965, when he developed atrial fibrillation which converted to sinus rhythm following quinidine administration.

In August 1966 he developed congestive heart failure and left back pain of sudden onset. He was treated with digitalis and diuretics. Pulmonary embolism was suspected and warfarin therapy was initiated.

In July 1969 he developed ventricular tachycardia with hypotension. Cardioversion resulted in sinus bradycardia at a rate of 50 per minute and temporary transvenous pacing was started. Because sinus bradycardia at rates of 40 to 50 per minute persisted, a permanent endocardial demand ventricular pacemaker (Ectocor®) was implanted on July 29, 1969.

Subsequently he experienced dyspnea on exertion, easy fatigability, ankle edema, ascites and intermittent claudication which required a hospitalization in December 1970. A chest film taken on January 5, 1971 revealed a slightly enlarged cardiac silhouette with mild pulmonary congestion (Fig. 1a). Cine fluoroscopy disclosed a hypokinetic heart but no overt dyskinesia. On a regimen of digitalis and furosemide he gradually lost weight totally 24 pounds and was discharged in a much improved condition.

On June 19, 1971 the patient suddenly developed shortness of breath, cough, cyanosis, and general weakness but he had no chest pain. He was admitted to The George Washington University Hospital on July 1, 1971.

The patient was on digitoxin, furosemide, spironolactone, and warfarin. Temperature was 36.3°C, pulse 72 per minute regular, respiratory rate 15 per minute and blood pressure 98/60 mmHg with 10 mmHg inspiratory decline of systolic blood pressure. His extremities were thin and the abdomen distended. Skin revealed

Fig. 1.  a) Chest roentgenogram (1–5–71) shows slightly enlarged cardiac silhouette with mild pulmonary congestion. b) Chest film (7–7–71) shows marked enlargement of the cardiac silhouette and mild pulmonary congestion. The pacemaker catheter is seen to lie far inside the right border of the heart shadow.
acrocyanosis of the ear lobes and nose and stasis dermatitis in lower legs with one plus edema noted. The neck veins were distended at 45 degree with occasional cannon waves observed. Moist rales were present in both lung bases. A diffuse precordial lift was felt. Heart sounds were rather distant and multiple systolic clicks were heard as was a grade 1/6 early systolic murmur at the apex (Fig. 2). No pericardial rub was heard. The abdomen was markedly distended and ascites

Fig. 2. Phonocardiogram taken at the apex shows multiple systolic clicks and intermittent early systolic murmur.

Fig. 3. Electrocardiogram shows an appropriate pacemaker function at a fixed rate of 72 per minute with the atrial rate of 110 per minute.
and meteorism were noted. The liver was felt 4 finger-breathths below the right
costal margin. Slight atrophy of the musculature of the lower extremities, espe-
cially of the right side was present. Peripheral arterial pulsations were weak and the
right dorsalis pedis and both posterior tibial pulses were absent. Elevation-de-
pendency test revealed elevation pallor and dependency rubor in both feet par-
ticularly on the right. Venous filling time was delayed in the right foot.

The electrocardiogram revealed an appropriate pacemaker function at a fixed
rate of 72 per minute, an atrial rate of 110 per minute and no competition (Fig. 3).
The chest X-ray was interpreted as showing marked enlargement of the cardiac
silhouette and slight pulmonary congestion. The pacemaker catheter was seen to
lie far inside the right edge of the heart shadow (Fig. 1b).

Laboratory data were red blood cell 4.15 x 10^6/mm³, white blood cell 11,200/
mm³, hemoglobin 13.7 Gm/100 ml, hematocrit 43.2%. Urinalysis was normal.
Serum electrolytes were Na 120 mEq/L, K 7.0 mEq/L, and Cl 82 mEq/L. The
serum urea nitrogen was 60 mg/100 ml, the alkaline phosphatase 280 mIU/ml, the
SGOT 70 mIU/ml, the LDH 250 mIU/ml, and the prothrombin time 40.5 sec
(control 13.0 sec).

Cardiac catheterization and angiocardiology were scheduled for the evaluation of the long standing congestive heart failure and possible pericardial effusion, but were delayed awaiting normalization of the prolonged prothrombin time and
electrolyte imbalance. The fluid intake was restricted and warfarin and spiro-
nolactone were discontinued. A cation exchange resin was administered. The
patient lost 11 pounds in 7 days. Extreme weakness, apathy, and shortness of
breath developed. On July 8, he suddenly became comatous and expired.

Pathology

The heart with pericardium weighed 1,150 Gm. Encasing the entire heart
was an organizing hematoma measuring up to 3.5 cm in greatest thickness (Fig. 4).
Epicardium and pericardium were thickened with fibrous connective tissue meas-
uring 2 to 3 mm in thickness. This grayish pink septae extended into the dark red
hematoma from both surfaces producing multiloculation. The cardiac chambers
were slightly dilated. The left and right ventricles measured 1.6 cm and 0.6 cm
in thickness respectively. Extensive fibrosis was present in the interventricular
septum and the anterior and posterior portions of the left ventricle toward the apex.

The pacemaker lead entered the right heart through the superior vena cava.
It was attached by the endothelium to the intimal surface of the right subclavian
and innominate veins and by a smooth band of connective tissue to the free edge
of the tricuspid valve leafllet (Fig. 5). With this exception the cardiac valves were
normal. The lead terminal was imbedded within the superficial myocardium of the
right ventricular apex. No perforation was present. The endocardium was nor-
mal.

The left coronary artery was generally atherosclerotic and with a 75% occlusion
present in its proximal portion. The right coronary artery exhibited a few large
plaques narrowing the lumen by 70%.

Other findings included hyperemia of the lungs, cardiac cirrhosis of the liver,
ascites (2,000 ml) and a recent thrombosis of the right iliac artery.
Fig. 4. Transverse section of the heart revealing an organizing multiloculated hemopericardium. Note the pacemaker lead in the right ventricle.

Fig. 5. The pacemaker catheter enters the right heart through the superior vena cava and the catheter is attached by a smooth band of connective tissue to the free edge of the tricuspid valve leaflet.

Microscopic examination of the heart revealed extensive fibrosis which, near the posterior septum, extended into the epicardial fat. Smaller foci of fibrosis were also present elsewhere in the left and right ventricles. The myocardium was hypertrophic. Slight subendocardial fibrosis and aggregates of lymphocytes were
present in both ventricles. In most sites the epicardial surface was continuous with dense granulation tissue that extended into the central hematoma composed of thick strands of fibrin clot and large spaces filled with intact erythrocytes and serum. Similar granulation tissue was present underlying the parietal pericardium (Fig. 6).

**DISCUSSION**

There is no clinical or pathological evidence of recent myocardial infarction, cardiac perforation of pacemaker catheter nor an evidence suggesting a preceding viral infection was noted in this case. The prothrombin time was around 40 sec for over a month. Accordingly the hemopericardium of this case will be concluded as solely due to the anticoagulant therapy.

Mid-systolic clicks and systolic murmur: These will be explained by the pacemaker catheter entrapped by a smooth band of connective tissue to the free edge of the tricuspid valve leaflet. The usual pacemaker sounds occur in pre-systole and the cause is explained as intercostal muscle twitching stimulated by the leaked current from the pacemaker. Systolic murmur has been explained to be due to impingement by the catheter on the valve leaflets.

New radiographic sign: The limitation of plain chest film roentgenography in the diagnosis of pericardial effusion is well known. However in cases of endocardial pacing the pacemaker catheter positioned at the right ventricle through the right atrium provides an index of the approximate
thickness of cardiac structure without aid of opaque contrast angiography or carbon dioxide angiography. This seems as a valuable sign of significant pericardial effusion though dilated right atrium may produce similar picture. In this case the pacemaker catheter was seen to lie far inside the right border of the cardiac silhouette comparing with a previous chest film which is highly suggestive of pericardial effusion.

A review of the reported cases of anticoagulant induced hemopericardium with cardiac tamponade indicates that the diagnosis is frequently missed.\textsuperscript{2)–8)} The sudden development of circulatory insufficiency in a formerly stable patient on anticoagulants should suggest this diagnosis.

In this case the sick sinus node which required a permanent pacemaker implantation and the later development of complete A-V block failed to increase the heart rate to compensate the cardiac output and marked diuresis resulted in low circulating volume and sudden cardiac collapse.

\textbf{References}