Multiple Calcified Thrombi (Rocks) in the Right Ventricle

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Friction rub secondary to calcified masses rubbing against each other in systole and diastole. Cardiac catheterization showed a constrictive-restrictive pattern that persisted after surgery. The role of noninvasive techniques in the diagnosis and long-term follow-up of the patient is emphasized.

Large, organized right ventricular thrombi are rare. This report describes a 51 year old man with a history of recurrent pulmonary emboli treated with inferior vena cava ligation who subsequently developed multiple mobile calcified thrombi in the right ventricle. He was treated successfully by surgical resection. Unusual clinical presentation on admission consisted of a two component inspiratory decrease in systolic pressure. On subsequent examination, the patient's blood pressure decreased from 94/62 to 70/50 mm Hg when he was turned on his right side.

The patient had an increased right ventricular impulse, along with a grade 3/6 tricuspid regurgitation murmur that increased during inspiration. The murmur was obscured by an apparent coarse two component pericardial friction rub that varied in intensity but was heard consistently at the lower and left sternal borders. Right heart failure was present with an elevated jugular venous pressure, enlarged pulsatile liver, ascites and leg edema. Marked venous collateral vessels were present on the abdominal wall.

Electrocardiogram revealed sinus tachycardia at 100 beats/min, generalized low voltage QRS complexes and right ventricular hypertrophy. Chest X-ray films showed a cardiothoracic ratio of 0.42, flattened diaphragm and slightly prominent right atrium, but no evidence of focal calcification. Cardiac fluoroscopy showed multiple mobile calcified masses in the right ventricle (Fig. 1).

Echocardiogram. M-mode echocardiography showed multiple echoes in the right ventricular cavity and outflow tract (Fig. 2). A small pericardial effusion was present. Two-dimensional echocardiography showed freely mobile masses in the right ventricular cavity and outflow tract (Fig. 3). These masses did not protrude into the right atrium or pulmonary artery.

Radionuclide studies. Technetium scan showed abnormal pyrophosphate uptake in the right ventricle. However, the uptake was faint and considerably underestimated the actual size of the masses removed at surgery. Gated blood

Case Report

A 51 year old white man was admitted in January 1979 with progressive dyspnea and anasarca. He had a history of repeated episodes of deep vein thrombophlebitis and pulmonary emboli despite adequate anticoagulation; these were treated by inferior vena cava ligation in 1971.

Physical findings. Physical examination on admission revealed a chronically ill, moribund patient with generalized anasarca and stasis changes in both legs due to severe venous engorgement. The heart rate was 100 beats/min, respiratory rate 16/min and blood pressure 95/65 mm Hg with a 5 mm

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pool imaging studies showed an enlarged right ventricle with filling defects corresponding to the known location of the calcified masses. The right ventricular ejection fraction was diminished (33%). Left ventricular size and its ejection fraction (51%) were normal.

Cardiac catheterization. Cardiac catheterization showed pulmonary hypertension and pressure tracings consistent with constrictive-restrictive cardiac disease (Table 1). Follow-through of right atrial angiogram revealed multiple, large, mobile calcified masses in the right ventricular cavity and moderate tricuspid regurgitation. The right main pulmonary artery was partially occluded by an organized thrombus.

Surgery. The pericardium was normal at surgery, showing no adhesions or inflammation. At least six distinct, multifaceted, friable calcified masses, all separate from one another and either entwined around chordae tendineae or attached to right ventricular trabeculae, were found and surgically removed (Fig. 4). The underlying tricuspid valve was normal, but chordae tendineae were elongated. An organized calcified thrombus was also removed from the right main pulmonary artery. Postoperative course was complicated on the 12th day by cardiac tamponade which was treated by subcostal drainage.

Clinical course and follow-up. The patient has been followed up for more than 4 years. He has dyspnea on walking about one block, but manages to do most of his housework and care for himself. His weight has remained stable on treatment with digoxin, furosemide and aldactone. Over the last year, pulmonary hypertension has increased with features of right heart failure and increasing tricuspid regurgitation.

Postoperative echocardiograms show a minimal residual mass in the right ventricle that has remained unchanged during 4 years of follow-up with serial echocardiograms.

Postoperative and follow-up radionuclide angiography showed continued right ventricular enlargement and reduced ejection fraction (36 to 39%). The left ventricle remained small but its ejection fraction dropped to 36% and remained low.

Discussion

Calcification of primary tumors of the heart is common, but large calcified right ventricular thrombi ("pseudotumor" or "rocks in the right ventricle") are rare. Only one other case has been reported (1). In another reported case (2), of a 15 year old patient with acute myelogenous leukemia, multiple rocks were found in the right atrium and ventricle. The cause of these rocks was not apparent.

Figure 2. M-mode echocardiogram from left ventricle (LV), mitral valve (MV), aortic valve (AV) and tricuspid valve (TV). Arrows point to echoes from calcified masses in the right ventricle and outflow tract. IVS = interventricular septum; LA = left atrium; PLVW = posterior left ventricular wall.
**Pathogenesis.** Clinically significant right ventricular thrombi are rare in the absence of congestive (3) or obliterative (4) cardiomyopathy (Loeffler’s disease or endomyocardial fibrosis). Occasionally, a large right ventricular thrombus may develop in patients with a permanent right ventricular endocardial pacemaker (5). In an autopsy study, Baum and Fischer (6) noted association of right heart thrombi in 13 of 33 patients with pulmonary emphysema and cor pulmonale. Multiple pulmonary emboli, most of which were small and microscopic, were noted in nine patients. Precise location, size and pathogenesis of these thrombi in the right heart chambers were not documented. Pulmonary hypertension with right heart failure leading to sluggish flow and stasis probably contributed to thrombi formation in these patients. Association of thrombophlebitis, pulmonary embolization and large right ventricular thrombi has been documented in only one other patient (7). Embolization from peripheral veins with trapping of the thrombus in the right ventricle and gradual enlargement appears to be the sequence in these patients. This process may be further potentiated by cor pulmonale and right heart failure. Goodman et al. (8) reported on a 59 year old man who had inferior vena cava ligation for recurrent pulmonary emboli and had multiple polypoid thrombi in the right atrium at autopsy. This case further supports the likelihood that embolization causes these right heart thrombi. Embolization eventually may lead to calcification, as in our patient.

**Surgical resection and postoperative ventricular function.** Kawamura et al. (7) described right ventricular obliteration and tricuspid obstruction by an organized thrombus in a 26 year old Japanese patient who had had venous grafting for thrombosis of the right femoral vein 2 years before admission. This patient was treated successfully by surgical resection. Postoperatively, however, right ventricular function remained poor, and right atrial contraction played the main role in ejecting blood into the pulmonary artery. Our patient presented with severe right heart failure.
with tricuspid regurgitation, pulmonary hypertension and a restrictive right ventricular hemodynamic pattern. Postoperative improvement was significant, but restrictive right ventricular function persisted. Although there are many reports of successful resection of primary (9) and secondary (10) intracardiac tumors, these two patients are the only patients reported to have successful resection of large right ventricular thrombi.

**Clinical presentation and pathogenesis of friction rub.** A right ventricular mass typically produces symptoms by progressive obstruction of the right ventricular inflow and outflow tracts. Variable degrees of systolic and diastolic murmurs along with right heart failure are present. Our patient had a tricuspid regurgitation murmur that increased with inspiration. We also noted a drop in blood pressure when the patient changed position, as is sometimes found in patients with atrial and ventricular space-occupying lesions. An unusual auscultatory finding in our patient was an apparent pericardial friction rub. Hubbard and Neil (11) described a patient with calcified right ventricular myxoma in whom an apparent endocardial friction rub was heard. They postulated that this friction was produced by actual rubbing of the heavy calcific mass against the irregular, thickened outflow tract of the right ventricle during systole and diastole. Our patient’s pericardium was normal at surgery. His friction rub was most probably caused by irregular calcified masses rubbing against each other in systole and diastole. The intensity of this friction rub varied, as a result of probable differences in contact between the calcified masses with changing cardiac output. Recently, Waller and Roberts (2) described a patient who had several large calcified masses in the right ventricle and systolic clicks that were caused by contact of one calcified mass with another.

**Role of noninvasive studies in diagnosis and follow-up.** Several noninvasive techniques can provide definitive diagnosis of intracavitary cardiac masses. If calcification is present within the mass, it may be detected on routine chest X-ray examination. However, serial X-ray films over a 20 year period showed no evidence of calcified right ventricular masses in our patient. Bedside fluoroscopy dramatically visualized the location, extent and mobility of the tumor mass. Diagnosis was further confirmed by two-dimensional echocardiography, which also provides additional information about the size of cardiac chambers, associated valvular lesions and the presence or absence of pericardial effusion. Serial echocardiography was also useful in long-term follow-up in detecting tumor recurrence. In a patient with rhabdomyosarcoma of the left atrium, we detected tumor recurrence by two-dimensional echocardiography as early as 6 weeks after resection (12). When echocardiographic examination is suboptimal, additional confirmation may be obtained by radionuclide gated cardiac blood pool scans (13). This technique was useful in long-term sequential follow-up of ventricular function in our patient.

**References**


