Right Atrial Myxoma
Unusual Post-operative Complications


SUMMARY
This case report concerns a patient with right atrial myxoma who had few features to suggest this diagnosis before cardiac catheterization. Fifteen days after successful removal of the tumor she developed an acute anterior myocardial infarction. Her convalescence from this was complicated by various rhythm disturbances including left atrial rhythm and ventricular tachyarrhythmias. Possible underlying causes which may have been responsible for the development of left atrial rhythm are discussed. The ventricular tachyarrhythmias did not respond to drug therapy but were immediately suppressed by rapid transvenous ventricular pacing.

Additional Indexing Words:
Cardiac tumor Cardiac arrhythmias in acute myocardial infarction Left atrial rhythm Drug-resistant ventricular tachyarrhythmias

Atrial myxoma is the most common primary cardiac tumor, and most are situated in the left atrium. About one-fourth occur in the right atrium,1) and these can present with a wide variety of symptoms and signs. The clinical, radiological and surgical aspects of right atrial myxoma have been reviewed recently.2)-4)

The purpose of this paper is to describe a patient with a right atrial myxoma which was not suspected clinically. Her post-operative convalescence was complicated by several unusual and interesting problems. These included acute myocardial infarction, left atrial rhythm, and drug resistant ventricular tachycardia and fibrillation which required the use of temporary transvenous pacing.

CASE REPORT
A 53-year-old white housewife was referred on 8–18–69 for evaluation of...
dyspnea. Shortness of breath on exertion was first noticed 2 years previously, and had progressively increased in severity. She also complained of easy fatigability, and occasional episodes of lightheadness. On physical examination, blood pressure was 130/100 and the precordial activity was normal. Jugular venous pressure was not elevated. Auscultation revealed regular rhythm (rate: 72/min.), a systolic click and a Grade II/VI ejection systolic murmur best heard along the left sternal border. No diastolic murmur was noted.

The hemoglobin content was 17.7 Gm.% with a hematocrit of 55% compared with values of 14.6 Gm.% and 43% respectively in 1963. Other laboratory tests included ESR, BUN, blood glucose, and liver function tests and were within normal limits. The electrocardiogram showed normal sinus rhythm with tall P waves but was otherwise unremarkable (not shown here). Chest X-rays showed increased prominence of the right atrial border when compared with films taken in 1965 (Fig. 1).

Fig. 1. Comparison of P.A. chest X-rays taken in September, 1965 and August, 1969 showing increased prominence of the right atrial border.

No definite clinical diagnosis was made and she was admitted for diagnostic cardiac catheterization in October, 1969. During the isolation of an antecubital vein she suddenly became unconscious with a bradycardia of 40 beats per min. and an unrecordable blood pressure. Following brief external cardiac massage and intravenous administration of atropine 0.7 mg., her heart rate and blood pressure returned to normal. This was interpreted as a vaso-vagal reaction and the procedure was continued. On introduction of the catheter into the right atrium it was persistently deviated to the left and would not pass down through the right atrium in normal fashion. Hand injection of 75% Hypaque into the right atrium outlined a large spherical filling defect, and this was confirmed by cineangiography (Fig. 2). Pressure in the right atrium was 14/8 mm.Hg with a mean of 12 mm.Hg and the A wave was prominent. Pressure in the right ventricle was 20/0–5 mm.Hg. Thus, there was a persistent gradient across the tricuspid valve during diastole. Arterial oxygen saturation was 92% and indicator dilution curves showed no evi-
Fig. 2. Angiogram following injection of contrast medium outlining an irregular spherical filling defect in the right atrium.

Fig. 3. The surgical specimen. The tumor contained areas of hemorrhage in the gelatinous material, and was attached by a pedicle to the region of the fossa ovalis. A circular portion of atrial septum including the pedicle was removed.

dence of right to left shunting. Cardiac index was low at 1.75 L./min./M.² In retrospect, it was felt that her initial syncopal episode could have been due to transient obstruction of the tricuspid valve rather than a vaso-vagal reaction or significant cardiac arrhythmia.

Operation was performed on 10–15–69 through a right antero-lateral thoracotomy and the right atrium was observed to be markedly distended by a solid tumor.
Using cardiopulmonary bypass, the right atrium was opened. There was a rounded, gelationous mass, $10 \times 7 \times 5$ cm., which virtually filled the entire right atrial cavity. It was attached to the atrial septum by a pedicle and the tricuspid valve was intact. No intra-atrial communication could be identified. The myxoma was removed by excising a circular section of atrial septum including the base of the pedicle (Fig. 3).

The immediate post-operative convalescence was uneventful apart from a minor problem with retained bronchial secretions. She was discharged 11 days after operation but was readmitted 4 days later because of the sudden development of severe, retrosternal chest pain. This was associated with nausea, sweating and palpitations. An electrocardiogram on this admission showed recent anterior myocardial infarction (not shown here) and she was admitted to the Coronary Care Unit. Her rhythm soon changed to atrial fibrillation with a ventricular rate of 110 min. and she developed some basal râles. Left ventricular failure was diagnosed and she was given digoxin 0.75 mg, intravenously and hydrochlorothiazide 50 mg.

![Fig. 4. Electrocardiogram showing changes of anterior myocardial infarction. P waves are inverted in leads II, are diphasic in III and aVF, and inverted in V₄-V₆ indicating left atrial rhythm.](image)

![Fig. 5. Leads II-a, b, c are continuous. Typical P waves of left atrial rhythm are seen in leads II, V₁ and V₆ and there is intermittent 2:1 exit block with A-V nodal escape beats.](image)
orally. Sinus rhythm resumed within a few hours and frequent ventricular premature contractions were readily controlled with oral quinidine. On subsequent days, her cardiac rhythm became unstable and varied between sinus rhythm, sinus tachycardia, A-V nodal tachycardia with incomplete A-V dissociation, sinus bradycardia with A-V nodal escape rhythm, atrial premature beats, and left atrial rhythm with intermittent 2:1 exit block (Fig. 4 and 5). Digitalis toxicity was considered as a cause and digoxin was stopped after a total dose of 2 mg. in 72 hours.

On the 6th day after the myocardial infarction she again developed frequent ventricular premature contractions. She became confused following intravenous administration of Xylocaine 50 mg. and no further Xylocaine was given. The ventricular premature contractions continued and were rapidly followed by the first episode of ventricular fibrillation. This was successfully defibrillated by the first D.C. shock of 300 watt sec. (Fig. 6), and she was then given procainamide 500 mg. intravenously. Eighty min. later the 2nd episode of ventricular fibrillation spontaneously ceased after about 20 sec., and she was then given an intravenous infusion of procainamide at a rate of 2 mg. per min. During the next 24 hours she had several transient episodes of ventricular tachycardia and 3 further episodes of ventricular fibrillation requiring D.C. countershock. The ventricular tachycardia and fibrillation recurred in spite of combined drug therapy which included intravenous administration of Dilantin and procainamide in addition to oral administration of quinidine and procainamide. Consequently a transvenous pacemaker was inserted into the apex of the right ventricle and pacing was instituted at a rate of 110/min.

Fig. 6. Ventricular fibrillation converted to sinus rhythm by D.C. countershock.

Fig. 7. Transvenous pacing at a rate of 100 beats per minute.
No further ventricular arrhythmia was observed and oral procainamide 500 mg, q.i.d. and quinidine 300 mg, q.i.d. were continued. The pacing rate was reduced to 100 per min. (Fig. 7). After 8 days of pacing, her underlying rhythm changed to atrial flutter and pacing was continued for a total of 11 days. On stopping the pacemaker, the rhythm was atrial flutter with 2 : 1 A-V conduction and digoxin was then given orally to control the ventricular rate. Cardioversion was successfully carried out on 12-1-69, 32 days after her myocardial infarction, with one D.C. shock of 50 watt sec. Following this she resumed a slow nodal rhythm which did not respond to intravenous injection of atropine. As she was otherwise progressing satisfactorily, she was discharged on 12-5-69 and when next seen on 12-11-69 she was in sinus rhythm. Further convalescence has been uneventful and her dosage of procainamide and quinidine are being slowly reduced.

**DISCUSSION**

Right atrial myxomata are notoriously variable with regard to their presenting symptoms and signs, and may be unsuspected prior to cardiac catheterization and angiography. Most of the features are produced by gradual obliteration of the right atrial cavity and progressive obstruction of the tricuspid valve orifice. At times the tricuspid valve may be damaged by repetitive trauma from the myxoma and produce tricuspid regurgitation. These hemodynamic abnormalities lead to right heart failure and its associated symptoms and signs. Dyspnea is common, and is probably due to the low and relatively fixed cardiac output. Cardiac murmurs may be typical of tricuspid valve disease although other murmurs, gallop rhythms or pericardial friction rubs may be present. Occasionally there is no murmur. Other features such as pulmonary hypertension from recurrent tumor emboli, superior vena cava obstruction, right to left shunting through a patent foramen ovale, and a vague systemic illness suggesting bacterial endocarditis can also occur. Cyanosis and polycythemia is usually secondary to a right to left shunt although there have been reports of patients with polycythemia without evidence of shunting.5), 6)

Our patient had few features to suggest the correct diagnosis and presented only with dyspnea. There was no evidence of right heart failure, and only a nonspecific heart murmur. In the absence of a detectable right to left shunt the cyanosis and polycythemia could have been due to the low cardiac output with relative tissue hypoxia.

Following the successful removal of the myxoma her progress was good until the myocardial infarction which occurred 15 days after operation. It is difficult to think that the infarction was the direct result of the surgical procedure as the coronary arteries were not disturbed, and any embolus through the atrial septostomy would have produced an immediate effect. It is felt
that the myocardial infarction is most likely due to the nonspecific effect (stress) of major surgery or even may have been coincidental.

The myocardial infarction was succeeded by various rhythm disturbances. One of the more interesting was that of left atrial rhythm. This is a rare electrocardiographic entity which has been the subject of recent electrophysiological and clinical investigation.\(^7\),\(^8\) It is a slow rhythm originating in an ectopic focus in the left atrium and can be diagnosed by analysis of the P waves in the various electrocardiographic leads. With left atrial rhythm the P waves are inverted in leads II and aV\(_F\), and usually also inverted in leads I and III. Right precordial leads frequently show an upright "dome and dart" configuration of the P wave and left precordial leads demonstrate P wave inversion.\(^7\) However, the specificity of these diagnostic criteria is not universally accepted.\(^8\) It has been observed in apparently healthy individuals as well as in patients with congenital and rheumatic heart disease. It does not appear to be related to atrial myxoma but has been reported after acute myocardial infarction.\(^9\) It is interesting to speculate whether its occurrence could be due to depression of the sinus node either from surgical trauma or from ischemia. The prolonged A-V nodal rhythm following cardioversion of the atrial flutter tends to support the concept of a diseased sinus node. An alternative explanation is of an ectopic focus in the left atrium, possibly from infarction, which gave rise to both the left atrial rhythm and the atrial flutter on different occasions.

The most serious and almost fatal complication was the ventricular tachyarrhythmia which developed 6 days after the infarction. This is more commonly seen earlier after myocardial infarction and raised the possibility of a digitalis induced arrhythmia. This is unlikely as she only received 2 mg. of digoxin in 72 hours, and this was discontinued when the A-V nodal escape rhythm was noted 3 days before development of the ventricular tachyarrhythmia. Recent reports indicate the effectiveness of rapid ventricular pacing in suppressing ventricular tachyarrhythmias which are unresponsive to drug therapy.\(^10\),\(^11\) The method by which ventricular pacing can prevent ventricular tachyarrhythmias is uncertain. Possibilities include (1) reducing the length of diastole to diminish the opportunity for discharge of latent ectopic pacemakers; (2) suppression of intrinsic pacemakers by "overdriving" the heart; (3) prevention of excitation in re-entrant pathways by altering the relationship between conduction velocity and refractory period.\(^12\) In this patient, pacing at a rate of 100 per min. completely suppressed all ventricular ectopic activity, and was felt to have been a lifesaving procedure.
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REFERENCES