Cardiac Aneurysm with Ventricular Tachycardia and Subsequent Excision of Aneurysm

Case Report

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With the introduction of ventriculoplasty the importance of diagnosis of ventricular aneurysm has increased.

Prior to operation the subject of the following case report had episodes of ventricular tachycardia controlled with difficulty by continuous quinidine administration. Following excision of the ventricular aneurysm there were no further episodes despite the omission of quinidine.

REPORT OF CASE

The patient was first seen in September 1953 at the age of 54, when she described short abrupt spells of fluttering of the heart of 3 years' duration. She had known high blood pressure for 10 years.

Physical examination at this time revealed moderate obesity and a blood pressure of 190/110 mm. Hg. The heart and lungs were normal on examination, an electrocardiogram showed minimal ST and T-wave changes, and the heart was of normal size on chest x-ray. The patient was given rauwolfia, and her blood pressure dropped gradually to 150/80.

In April 1955, after occasional anginal pains, she suffered more prolonged pain and she was hospitalized for over 1 month with an extensive acute anterior wall myocardial infarction. The heart sounds were distant and somewhat muffled, and a protodiastolic gallop was heard. The blood pressure was 120/80. X-ray showed definite left ventricular enlargement with a convexity bulging outwardly along the upper portion of the left ventricle suggesting a ventricular aneurysm (fig. 1). The pulsations were of poor quality. Salt was restricted, the patient was digitalized, and she was also given reserpine.

Following discharge she had premature beats which persisted despite quinidine and proesine amide. On September 30, 1955, she was still wek, described one episode of nocturnal dyspnea, and showed a trace of pedal edema. An electrocardiogram in November (fig. 2) showed signs of previous infarction and ST-segment deviations consistent with aneurysm. In December she had several episodes of paroxysmal tachycardia, one of which lasted 12 hours; an electrocardiogram showed it to be ventricular in origin (fig. 2).

Physical examination showed the heart rate to be 140 and regular. The liver was felt 2 fingerbreadths below the costal margin but was smooth and nontender and there was no other sign of failure. Quinidine sulfate was started at 0.4 Gm. every 4 hours and subsequently increased to 0.6 Gm. every 4 hours. The patient reverted to a regular rhythm on the second hospital day. The dosage of quinidine was then lowered to 0.4 Gm. every 4 hours but she again developed ventricular tachycardia, this time associated with acute pulmonary edema. She was given 0.6 Gm. of quinidine every 4 hours and the rhythm again reverted to normal. The QRS interval became prolonged and the quinidine dosage was again reduced to 0.4 Gm. every 4 hours. On the ninth hospital day, however, she again developed ventricular tachycardia. She was given 0.5 Gm. of quinidine every 4 hours with a rapid return to normal sinus rhythm. She was discharged on this dosage and on 0.375 mg. of digoxin daily.

Following discharge the patient got along well except for one episode of rapid heart action that had a sudden onset but gradual cessation. She seemed to reach a plateau with very little improvement. Because of this lack of improvement and because of the difficult prospect of continuing quinidine dosage around the clock indefinitely, surgical therapy was considered. She was referred to Dr. Charles P. Bailey, of Philadelphia. The presence of the aneurysm was confirmed by roentgenography and left cardiac atriography.

Ventriculoplasty was carried out on April 3, 1956. During induction of anesthesia cardiac arrest occurred and was successfully treated with cardiac massage. Otherwise the procedure was uneventful. Postoperatively a gallop rhythm was heard for a few days and heart failure developed, but it was successfully treated. Heparin and Dicumarol were also used. Two weeks after surgery...
Fig. 1. Left. Chest film showing a bulge in the region of the apex of the left ventricle.
Right. Postoperative film.

the patient was permitted out of bed in a chair and was gradually ambulated during succeeding days.

On May 26, 1956, physical examination showed an increase in the pulmonic second sound and a loud systolic pulmonic murmur. The left border of heart dullness was still at the anterior axillary line. The lungs were clear. Fluoroscopy showed poor cardiac pulsation. An x-ray of the chest (fig. 1) showed moderate pleural reaction at the left base and disappearance of the previously described hump in the left heart border. The heart was approximately the same size but was a little more globular and not quite so prominent in the apical region.

On August 27, 1956, she was hospitalized because of mild constricting chest pain. Examination of the heart was unchanged. The white blood cell count and sedimentation rates showed moderate elevations. There were minor electrocardiographic changes but none was diagnostic of infarction. However, it was thought that the patient had probably sustained a myocardial infarction or coronary insufficiency. She was therefore started on long-term Dicumarol therapy with satisfactory maintenance of the prothrombin time within therapeutic levels. Because of a blood pressure elevation of 160/100 mm. Hg rauwolfia was given and the pressure promptly returned to normal; her heart rate also slowed considerably. She has shown no evidence of congestive failure and has been able to increase her activity over that which was possible preoperatively.

Discussion

Only a few cases of ventricular tachycardia in association with ventricular aneurysm have been reported. In 1953 Wasserman and Yules\(^5\) reported a case of a 67-year-old man
in whom an effort to control the tachycardia with intravenous and oral procaine amide was not successful. Conversion to a normal sinus rhythm was accomplished by the administration of quinidine but death occurred 3 hours later. In 1954 Walker and Richmond described a case with a posterior ventricular aneurysm in a patient with dextrocardia and situs inversus. While in the hospital the patient developed paroxysmal ventricular tachycardia that was treated successfully with quinidine. No statement was made whether continuation of the quinidine was necessary to maintain the normal sinus rhythm. Dick in 1955 mentioned a 59-year-old man with a posterior ventricular wall aneurysm who was seen with an episode of ventricular tachycardia. No statement was made regarding treatment. In the present case the ventricular tachycardia was controlled only with rather exactly regulated doses of quinidine sulfate. This drug was continued until the time of operation. Following the operation the quinidine was discontinued without further recurrence of the ventricular tachycardia. This sequence suggests that an irritable focus causing the tachycardia had been removed with the removal of the ventricular aneurysm. Despite the cardiac arrest at the time of operation the patient’s postoperative recovery has been excellent. She has gained strength progressively and has reached a considerably higher level of activity than was possible prior to operation. Because of an episode of chest pain that probably represented an infarct or an episode of coronary insufficiency permanent anticoagulant therapy was started. Although she has shown no evidence of congestive failure, the preoperative dosage of digoxin has been continued.

**SUMMARY**

A case of ventricular tachycardia in a patient with an anterolateral ventricular aneurysm is reported. The aneurysm was excised and the ventricular tachycardia did not recur, despite discontinuation of quinidine.

**SUMMARIO IN INTERLINGUA**

Es reportate un caso de tachycardia ventricular in un patiente con aneurysma ventricular anterolateral. Le aneurysma eseva excidite. Le tachycardia ventricular non reccurreva in especto del discontinuation de quinidina.

**REFERENCES**

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