Mitral subannular left ventricular aneurysm *A case presenting with ventricular tachycardia*

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SUMMARY A young African immigrant presented with ventricular tachycardia in association with two mitral subannular left ventricular aneurysms. Although an unusual finding, the recognition of such aneurysms is important as prophylactic measures may prevent complications. Furthermore, they are a surgically treatable cause of heart failure and arrhythmias.

Aneurysms of the left ventricle in European and North American patients are usually the consequence of myocardial infarction. In patients of African descent, multiple left ventricular aneurysms of uncertain aetiology which originate below the mitral valve annulus can result in ventricular arrhythmias, heart failure, and embolic phenomenon. Although the condition is uncommon even in Africa, its recognition and diagnosis is important as a surgically treatable cause of arrhythmias and heart failure. We report the case of a young African immigrant who developed ventricular tachycardia in association with multiple calcified mitral subannular left ventricular aneurysms.

Case report

A 40 year old West African man presented with a three month history of recurrent palpitation associated with dizziness and weakness. He had come to Canada from Togo at the age of 36 and had had no previous illness.

With the paroxysms of tachycardia at 155 beats/minute blood pressure fell to 90/60 mm Hg from its normal resting value of 120/80 mm Hg. The cardiac impulse was localised to the fifth intercostal space in the mid-clavicular line and suggested left ventricular hypertrophy. There was a soft systolic ejection murmur but no additional heart sounds. The electrocardiogram showed a paroxysmal tachycardia at a rate of 155 beats/minute with a right bundle branch block configuration (Fig. 1). Independent atrial activity was confirmed with an oesophageal electrode. During sinus rhythm there was T wave inversion in leads I, aVL, V4-6.

The chest radiograph showed a slightly enlarged

cardiac silhouette with two areas of calcification. The calcification was shown by fluoroscopy to be around two spherical "cysts," which were contained within the cardiac contour and related to the mitral valve annulus. Echocardiography showed extensive calcification in the area of the mitral annulus. No aneurysmal structure could be recognised on cross sectional echocardiograms.

At cardiac catheterisation both left and right heart pressures were normal. Left ventricular angiography showed that the two calcified spherical "cysts" were filled from the left ventricle (Fig. 2). Coronary arteriography indicated that the circumflex branch of the left coronary artery was stretched around the upper of the two aneurysms (Fig. 3).

The ventricular tachycardia was controlled initially with lignocaine and later with quinidine. Cinefluoroscopy has shown no increase in the size of the calcified aneurysms and the patient remains asymptomatic 18 months later.

Discussion

Left ventricular aneurysm in North American and European populations is usually the consequence of myocardial infarction, although it can have an alternative aetiology. Trauma, Chagas's disease, syphilis, and endocarditis may result in focal myocardial damage and subsequently the development of an aneurysm. An extremely unusual form of aneurysm in Europe and North America originates in the left ventricle in the region of the mitral annulus. In this report the patient was an otherwise healthy man whose initial complaint was palpitations due to ventricular tachycardia and who was subsequently shown to have two calcified left ventricular aneurysms originating close to the mitral valve annulus.

Fig. 1(a) Electrocardiographic leads V1 and V2 showing an episode of paroxysmal ventricular tachycardia with a right bundle branch block configuration, (b) an oesophageal electrode showing the independent atrial activity during the paroxysm of tachycardia.

Fig. 2 Left ventricular angiogram in right anterior oblique projection showing that both calcified aneurysms communicate with the left ventricle.

Fig. 3 Left coronary angiogram in lateral projection showing the circumflex coronary artery stretched around the superior of the two calcified aneurysms.

Although there have been several reports of mitral subannular left ventricular aneurysms from South Africa¹⁻³ and Nigeria,^{4,5} they are not common even within these populations, and their true incidence and

natural history are not known. They seem to be confined to black Africans, yet there is one reported case in a patient of European origin.⁶ Although mitral subannular ventricular aneurysms are rare in Europe and North America, they remain an important cause of cardiac failure, arrhythmias, and systemic embolism in the black population.

Ventricular tachycardia has been noted previously in association with mitral subannular left ventricular aneurysms.^{5,7} These patients, like ours, had small aneurysms unassociated with cardiac failure. It seems plausible that the arrhythmia originates close to the aneurysms. The atrioventricular dissociation suggests a ventricular origin and the right bundle branch block configuration a left ventricular focus.

Mitral subannular left ventricular aneurysms are usually recognised with the onset of congestive heart failure.³ The aneurysms are then large (above 7-10 cm in diameter) and associated with disruption of the mitral valve annulus and mitral regurgitation.¹ Cardiac failure may be increased further by myocardial ischaemia or infarction caused by the aneurysm compressing the circumflex coronary artery.³ In the present case the circumflex branch of the left coronary artery could be seen to course around the calcified aneurysm (Fig. 2). Although the lumen of the vessel does not yet appear to have been narrowed, the stretching of the vessel might induce thrombosis.³ Furthermore, because of the proximity of the aneurysm to the artery, the vessel may be damaged during surgical closure of the aneurysm.¹ Left ventricular aneurysms originating in the mitral subvalvular region may rupture spontaneously⁸ or be the origin of systemic emboli.⁹ The border of the aneurysm became infected in one reported case,¹ resulting in a fistula between left atrium and ventricle.

Initial results of surgical resection of mitral subannular left ventricular aneurysms were poor,¹ but with improved techniques there is now a low surgical mortality and mitral valve replacement is rarely necessary.¹⁰ In the present case both the heavy calcification of the aneurysms and the lack of left ventricular failure may reduce the need for surgical intervention. Fluoroscopy at regular intervals over the past two years has shown no enlargement of the aneurysms, and the arrhythmia is readily controlled with medical treatment. Systemic emboli may also be prevented by oral anticoagulation.

The cause of mitral subannular ventricular aneurysms is unknown. If they are a congenital anomaly, as suggested by Chesler *et al.*,¹ it is surprising that they are so rarely observed in North American blacks. Yet

if they are acquired the affliction seems to spare European residents in Africa. As with endomyocardial fibrosis, the aneurysms could be an acquired abnormality which are only manifested in a susceptible population. The pathological features do not suggest an inflammatory aetiology, although focal aggregates of lymphocytes have occasionally been observed.¹¹

Although mitral subannular left ventricular aneurysms are a rare cause of arrhythmias, heart failure, and emboli, their recognition and management is important. Chest radiography and fluoroscopy are recommended as the initial investigation in the patient at risk, and the diagnosis should be confirmed by left ventricular angiography.

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