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REVIEW ARTICLE

LEFT VENTRICULAR ANEURYSM

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Post infarction true ventricular aneurysm may be defined as a localized area of dyskinesia with regional systolic expansion. The definition from surgical observation leads to the following criteria: the presence of a scar like deformity of the ventricular wall, the presence of akinesia or dyskinesia, myocardial thinning or scar formation and the loss of trabecular pattern of the endocardium.

It has become common clinical practice to classify ventricular aneurysms with an external bulge as true or false. The wall of the true aneurysm is derived from the myocardium itself whereas the wall of the false aneurysm (pseudoaneurysm) is derived from the peri-cardium [2].

The reported incidence of left ventricular aneurysm (LVA) ranges from 35% to 40%. This wide range is partly a reflection of a lack of generally applied definitions [2'3]. Aneurysm formation probably occurs within 3 months after the acute myocardial infarction (AMI) but has been reported to be two weeks to two years after AMI[3]. Factors such as a large transmural AMI and first AMI have been associated with LVA [4]. The greatest risk of aneurysm follows infarction in the area supplied by the left anterior descending artery [5].

SITES OF LVA

Most commonly, aneurysms involve the anterior and/or the apex of the left ventricle (LV) and such aneurysms are generally larger than those which involve other areas [5]. Visser et al [3] reported that 77% of aneurysms were located anteriorly, 17 % posteriorly and 6% anteroposteriorly.

The mechanism of LVA formation is most likely related to the influence of changes in wall stress. The interventricular tension stretches the non-contracting infarcted heart muscle, thus producing infarct expansion, a relatively thin layer of necrotic muscle and fibrous tissue that bulges with each contraction. Later, the wall of the aneurysm becomes more densely fibrotic and continues to bulge with each systole [2,3].

PATHOPHYSIOLOGY

The LV compensates after the loss of shortening from the area of the aneurysm, but for a relatively large aneurysm, compensation is impossible. The stroke volume falls or may be maintained with an increase in end diastolic volume leading to increase in wall tension and myocardial oxygen demand. Thus, heart failure may result, while angina become worse.

CLINICAL FEATURES

Patients with LVA may develop symptoms of left ventricular failure, angina or recurrent ventricular arrhythmias.

Diagnosis of LVA is classically made by the physical finding of a precordial systolic bulge (10%) and inverted T waves & significant Q waves (65-79%) [5] on ECG.

Aneurysms can be visualized by echocardiography, radionuclide ventriculography or cardiac catheterization. The signal averaged ECG may provide an additional tool to help determine which patients merit further investigation of ventricular arrhythmias. Programmed electrical stimulation during sinus rhythm can be used to guide pharmacological therapy while endocardial mapping may be helpful for surgical therapy [6].

COMPLICATIONS

Complications of LVA are more common with large aneurysms and include mural thrombus formation, calcification of thrombus or aneurysmal wall, systemic embolization, impaired ventricular function with congestive heart failure and ventricular tachycardia (VT). Although rupture of the aneurysm is rare, it can occur acutely or following reinfarction at the margin of the aneurysm [5].

Recurrent VT was observed in 16-65% of patients with LVA [7]. Miller et al [6] reported that patients who develop spontaneous uniform VT

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have larger aneurysms on ventriculography than do those without spontaneous VT and that patients with inducible VT have significantly more widespread abnormalities on their ECG with sinus rhythm than do patients without inducible VT.

MANAGEMENT

There are two options for the management of LVA, namely medical or surgical:

Medical Management: Medical management is similar to that for heart failure, & antianginal or antiarrhythmic therapy can be initiated, as indicated. The treatment of systemic embolism has been controversial. Lapeyre et al [8] suggest that the extremely low incidence of clinically evident systemic embolism associated with chronic LVA does not justify the long-term use of oral anti-coagulation.

The effect of antiarrhythmic drugs such as class I or class III agents can be evaluated by means of intra-cardiac electrography and programmed electrical stimulation, providing a basis for more rational medical management of VT and prevention of sudden death. Side effects of these antiarrhythmic agents should be strictly monitored.

Surgical Management: Surgical resection of LVA is indicated for recurrent ventricular tachyarrhythmias refractory to medical treatment, refractory congestive heart failure, or recurrent systemic embolization [5,7].

Aneurysmectomy is now a relatively safe procedure and can result in symptomatic improvement, whereas objective LV improvement has been more difficult to demonstrate. Deterioration does occur occasionally [9]. Some authors believe that coronary artery bypass grafting should be performed if there is the usual accepted indication for aneurysmectomy and/or subendocardial resection. Improved results have been obtained by combining aneurysmectomy with localized subendocardial resection at the site showing the earliest electrical activity on sequence mapping during intra- operatively induced VT. The surgical risks appear to be high with three vessel disease [6,7].

PROGNOSIS

The prognosis of a patient with previous myocardial infarction can be altered by the presence of a ventricular

aneurysm. If the aneurysm is small, involving less than 20% of the total area of LV, stroke volume is maintained and survival is similar to that of post myocardial infarction without aneurysm. However, a large aneurysm significantly reduces survival. The 5 years' survival rate of post myocardial infarction patients is 77 % in the absence of aneurysm and 27 % in those with LVA[5]. Early aneurysm formation is associated with a high 3 months (67%) and one year (80%) mortality rate [3].

There is evidence that aneurysmectomy may improve survival. The 5-year survival for patients whose post infarction aneurysms were surgically resected was 76 % compared with 20% for patients with aneurysms treated with medical management alone [5].

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