8 Years' Experience in Repair of LV Aneurysm with CABG

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Abstract

Background- Over 95% of true left ventricular aneurysms result from coronary artery disease and myocardial infarction. The incidence of left ventricular aneurysm in patients suffering myocardial infarction has varied between 10-35%. Large left ventricular aneurysm can cause arrhythmias, congestive heart failure, recurrent myocardial infarction, thromboembolic events and sudden death and operation is indicated for symptomatic large left ventricular aneurysms. In this study we evaluated results of surgical repair of left ventricular aneurysms in association with coronary artery bypass graft.

Methods- In this descriptive, cross-sectional study from September 1997 to March 2005, we had 1894 CABG operations. Concomitant left ventricular aneurysm repair was done in 54 cases. Surgical complications, clinical findings, left ventricular ejection fraction, NYHA classes, morbidity and mortality were evaluated.

Results- NYHA classes were reduced from 3±0.7 preoperatively to 1.23±0.4 postoperatively ($p<0.05$), and left ventricular ejection fraction changed from 23.82±5.72% to 34.12±7.25% ($p<0.05$). Surgical complications were re-operation for bleeding in 4 cases (7.4%), sternal dehiscence in 1 case (1.8%) and intra-aortic balloon pump insertion for weaning of CPB in 8 cases (14.8%). Mean ICU stay was 3±1.1 days, mean hospital stay was 13±2.3 days; hospital mortality was 1 case (1.8%). During follow up (1-5 years with a mean of 1±04), all patients are alive, free from cardiac events and have good functional classes.

Conclusion- Early and mid-term results of CABG with repair of left ventricular aneurysm are excellent with low morbidity and mortality, and we recommend CABG and repair of left ventricular aneurysm in case of large ventricular aneurysm ***(Iranian Heart Journal 2007; 8 (1): 38-42)***.

Key words: left ventricle aneurysm ■ left ventricle function■ psuedoaneurysm■ Dor operation■ coronary artery bypass graft (CABG)■ aneurysmorrhaphy

A post-infarction LV aneurysm is a well-delineated trans-mural fibrous scar, virtually devoid of muscle, in which the characteristic fine trabecular pattern of the inner surface of the wall has been replaced by smooth fibrous tissue. In such areas, the wall is usually thin, and both inner and outer surfaces bulge outward.

During systole, the involved wall segments are akinetic (without movement) or dyskinetic (characterized by paradoxical movement).

Intraoperatively, left ventricular aneurysm may also be defined as an area that collapses upon left ventricular decompression.

Johnson and colleagues defined aneurysm as a large single area of infarction (scar) that

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causes the left ventricular ejection fraction to be profoundly depressed (to approximately 0.35 or lower).[1]

True LV aneurysms involve bulging of the full thickness of the LV wall, while a false aneurysm of the LV is in fact, a rupture of the LV wall contained by surrounding pericardium. Also we must distinguish between scar tissue secondary to infarction and true LV aneurysm. These scars are usually not transmural and the LV wall is not thinned. The endocardium beneath retains its trabeculations, and the area of scarring is not clearly demarcated from the rest of the wall, mural thrombi are not commonly present, and the pericardium is not commonly adherent to the area.

Diagnosis of LV aneurysm can be done in 75% of patients by natural history, physical examination, ECG and radiological examinations.[2]

The incidence of LV aneurysm in patients suffering MI varied between 10% and 35% depending on the definition and the methods used. Of patients undergoing cardiac catheterization, 7.6% had angiographic evidence of LV aneurysms. The absolute incidence of LV aneurysms may be declining due to the increased use of thrombolytics and revascularization after myocardial infarction.

Over 95%of true LV aneurysms reported in the literature result from coronary artery disease and myocardial infarction, but true LV aneurysm also may result from trauma, Chagas' disease, or sarcoidosis.

The precise mechanism by which LV aneurysms form has not been completely elucidated; however occurrence of a large transmural infarction is a prerequisite and it has been suggested that patients who developed LV aneurysms have few intercoronary collateral arteries. Total occlusion of a coronary artery and poor development of collateral flow is a determinant of LV aneurysm formation. Apparently, normal or supernormal systolic function in adjacent ventricular segments is necessary for generating sufficiently high intraventricular pressure and wall tension in the infarcted area to result in aneurysm formation. Historically about 10% to 35% of patients who survived a major myocardial infarction developed an LV aneurysm.[5]

In patients without symptoms managed conservatively, 10-year survival was 90% but it was only 46% in symptomatic patients. In general, risk of thromboembolism is low in patients with aneurysm (0.35% per patient/ year), and long-term anticoagulation is not usually recommended. However in patients with mural thrombus visible by echocardiography after myocardial infarction, 19% develop thromboembolism over a mean follow-up period of 24 months. In these patients, anticoagulation and close echocardiographic follow-up may be indicated. AF and large aneurysmal size are additional risk factors for thromboembolism.[6]

When LV aneurysm is associated with dyskinesia, survival is reduced because LVEF is less than in akinetic LV.

Angina is the most frequent symptom in most series of patients operated upon for LV aneurysm. Given that three-vessel coronary disease is present in 60% or more of these patients, the frequency of angina is not surprising.[7]

Dyspnea is the second most common symptom of ventricular aneurysm and often develops when 20% or more of the ventricular wall is infarcted. Dyspnea may occur from a combination of decreased systolic function and diastolic dysfunction. Either atrial or ventricular arrhythmias may produce palpitation, syncope or sudden death or aggravate angina and dyspnea in up to one third of patients.

Thromboembolism is unusual but may produce symptoms of stroke, MI, or limb or visceral ischemia.

The morphologic diagnosis of post-infarction LV aneurysm can be made with assurance only at operation or autopsy. This is because the akinetic or dyskinetic segmental wall motion of an LV aneurysm can be mimicked by non-transmural scar or early infarcts that

are not morphologic aneurysms. Small and moderate–sized aneurysms are often associated with no specific symptoms, although the patient may experience angina because of stenosis in other portions of the coronary arterial tree. On physical examination palpitation over the heart often demonstrates a diffuse sustained apical systolic thrust and a double impulse on auscultation; usually a third heart sound and often a fourth (atria) sound are present. Chest x-ray and fluoroscopy may show an external bulge or convexity when the aneurysm is large enough and profiled, but the chest radiograph is not usually specific. The ECG frequently demonstrates Q waves in the anterior leads, along with persistent anterior ST-segment elevation. LV ventriculography is the gold standard for diagnosis of LV aneurysm. The diagnosis is made by demonstrating a large, discrete area of dyskinesia (or akinesia) and occasionally LV ventriculography also may demonstrate mural thrombus.

Two-dimensional echocardiography is also a sensitive and specific means of diagnosing left ventricular aneurysm; mural thrombus and mitral valve regurgitation are detected most readily by echocardiography. It is also useful for distinguishing false aneurysm from true aneurysm by demonstrating a defect in the true ventricular wall.

Thallium scan and positron emission tomography can be helpful early after infarction to differentiate true aneurysm from hibernating myocardium with reversible dysfunction. MRI accurately depicts LV aneurysm and is a reliable means for detecting mural thrombosis.[8]

Because of the relatively good prognosis for asymptomatic LV aneurysm[9] no indications for repairing chronic, asymptomatic aneurysms are established. Yet, in low risk patients during operation for associated coronary disease, investigators report repairing large, minimally symptomatic aneurysms.[10] On the other hand, operation is indicated for symptoms of angina, congestive heart failure or selected ventricular arrhythmias. For these symptomatic patients, operation offers better outcome than medical therapy.

Because LV aneurysm may have a tendency to rupture when acute or large (either with or without symptoms), operation is indicated. Similarly, congenital aneurysms have a presumed risk of rupture and should undergo repair regardless of symptoms.[11]

Methods

In this descriptive, cross-sectional study from September 1998 to March 2005 in Shaheed Rajaei Cardiovascular Medical Center, we had 1894 CABG operations. 54 cases had concomitant left ventricular aneurysm repair (over 5 cm diameter) with CABG (0.35%). Mean age was 33-70 (mean 57.6±5.1 years, Table I). Presentation of symptoms in patients was dyspnea in 81%, stable angina in 62%, unstable angina in 34%, and CHF in 2.7% of patients. In 100% of patients Q wave was seen in ECG and in 30%, exact diagnosis of aneurysm was made before operation. There was a doubt about the diagnosis of LV aneurysm in 46% and in 24% of cases diagnosis was done intraoperatively (Fig. 2). LV clot was found in 35%, and in 84% of patients the aneurysm was anterior or anterolateral and in 16% it was in the posterolateral portion of the heart.

***Surgical Procedure*:** In all patients surgery was done with the use of cardiopulmonary bypass. Moderate hypothermia (28-39˚C) and cold blood cardioplegia was used, and in two patients there were septal defects after MI which were repaired concomitantly.

We found three vessel coronary artery disease in 81% and two vessel disease in 19%. Total of 3.05±0.99 grafts were done per case and in 83% of patients, LIMA was used as a conduit.

We used four methods for repair:

1/ Plication was used in 61% (Fig. 1)

2/ Endoventricular patch repair (Dor) in 18%

3/ Circular patch in 14% 4/Linear patch in 5% 5/other methods, 2% (Fig. 2).

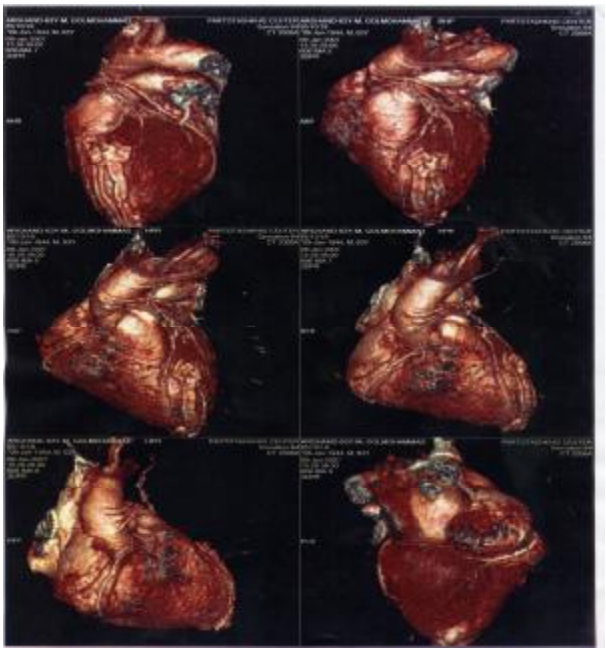

Fig. 1. CT-Angiography in one of our patients 7 years after 4 graft CABG and LV aneurysmal plication.

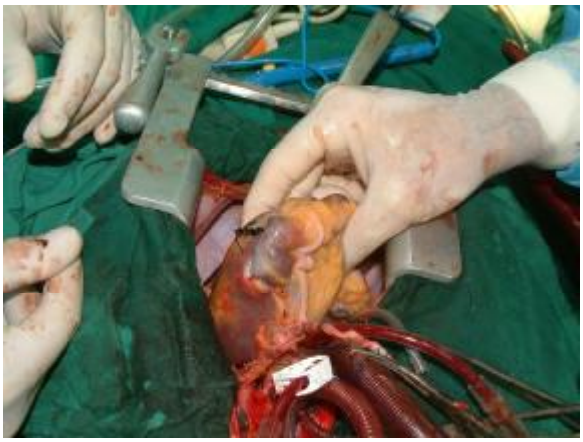

Fig. 2. Plication technique in LV apical aneurysm.

Results

73% of our patients were male and 27% were female. Preoperative diagnosis was made by ventriculography in 30%, by echocardiography in 46% and intraoperatively in 24%. Mean cross clamp time was 56.55±9 minutes and mean pump time was102.61± 21.18 minutes. We used intraaortic balloon pump for 21.7% of patients and in 81.2% of them we used it before operation. Re-operation for surgical bleeding was done in 7.4% of patients. Sternal dehiscence occurred in 1.8%, stroke in 5.4% and mortality in 2.7%. NYHA class was 3±0.7 in our patients pre-operatively, that was reduced to 1.23±0.4 after surgery.

Table I. Results of LV aneurysm operation in Shahid Rajaie Heart Center.

Mean Cross Clamp Time	56.55±90 minutes
Mean Pump Time	102.61+_21.18 minutes
Balloon Pump Insertion	21.7% (81% before operation)
Reoperation for surgical bleeding	7.4 %
Sternal dehiscence	1.8 %
Stroke	5.4 %
Mortality	2.7 %
NYHA Class change	From3 to 1
Improvement in LVEF	From 24 % to 34 %

Mean LVEF was improved from 23.82±5.3% to 34.11±6.18%. Mean hospital stay was 25±11.2days (from 15 to 55), and mean ICU stay was 3.4±2.1 (from 2 to 10 days). Mean follow-up was 13±3.2 months.

Discussion

Concomitant surgery of LV aneurysm and CABG not only does not increase the risk of surgery but also can improve the cardiac function and clinical symptoms of patients.[12] In our study mean patient age at concomitant operation was lower than other studies, (57.6±5.1 compared with 61.1±8.5 years).

In 84% of cases LV aneurysm was in the antero-lateral wall and in 16% of cases, it was in the postero-inferior wall. In other studies 71% were anterolateral and 29% postero-inferior. In our study, mean NYHA class was 3±0.7 preoperatively and post-operatively it was reduced to 1.23±0.4; in other studies it was reduced from 2.9 to 1.8±0.6 postoperatively.

Improvement in LV function in our study was 10.29±0.88% compared to 16% reported by Kawachi.[14] Sternal dehiscence in our study was 1.8% and in Stahle et al. study, it was 1.5%[15] and 1.86% in Ottiro, et al. report.[16] In Gardner's study, stroke rate was 3% in above

60 year old patients and 8% in above 75 year olds. In our study, it was 5.45% overall.
Post-operative hemorrhage that needed re-operation was 0.5 to 5% in Blackstone, Elte and Lauer's study and it was 8.1% in our study. Hospital mortality in our study was 2.7% and in other studies it was between 3 to 7%. It is obvious that ejection fraction under 20 to 30% can add to mortality rate.

Conclusion

In general, this study showed that our mortality and morbidity rate is as low as other studies and considering improvement in the life quality, postoperative EF improvement and function class improvement, we advise concomitant LV aneurysmal repair with CABG operation when indicated.

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