

An Easy and Safe Way of Left Ventriculotomy Closure in Patients with Left Ventricle Thrombi during the Subacute Phase of Anterior Myocardial Infarction

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ABSTRACT

Left ventricle thrombi occurring following myocardial infarction are usually left to spontaneous resolution to avoid a left ventriculotomy in the early phase of myocardial healing. We describe a simple and safe method of ventricular closure in patients with left ventricular thrombi embolizing to the lower extremities following acute anterior myocardial infarction. Ventricles were closed by epicardially running 5/0 polypropylene sutures in continuous fashion to avoid the myocardial tearing of heavier suture materials and the late adverse effects of Teflon use. In follow-up of the patients, no early and late complications were found with this method.

INTRODUCTION

Left ventricular thrombus (LVT) is a frequent complication of patients having transmural acute anterior myocardial infarction (AMI). The incidence of LVT after acute AMI can be more than 90% at the first week of infarction, and this high incidence is related to higher initial end-systolic and end-diastolic volume indices of the left ventricle, to a larger infarct perimeter, and to a lower initial ejection fraction [Neskovic 1998]. In prospective studies, the incidence of peripheral embolism due to LVT was 12% in the acute phase of anterior MI and 23% during the 36 months of the follow-up period [Domenicucci 1999, Jugdutt 1989]. After the beginning of the thrombolytic era, a decrease in the incidence of ischemic stroke due to LVT from 5% to 1% to 2% has still been controversial [Tanne 1997]. Patients having an embolic event due to LVT after AMI were reported to have significantly higher rates of mortality than patients who did not (38.9% versus 10.3%) [Sharma 2000].

Thrombolytic treatment of acute AMI caused faster and more frequent resolution of LVT, although the treatment did not affect the rate of thrombi development [Kontny 1993, Domenicucci 1999]. Adjunctive initial high-dose heparin therapy and long-term oral warfarin treatment have led to

decreases in thrombus size or to the disappearance of thrombi, but embolic events have been seen in patients taking full-dose anticoagulant therapy [Visser 1985].

Interrupted heavy sutures with polytetrafluoroethylene (Teflon) felt reinforcement have often been the technique of choice for the closure of a left ventriculotomy. But heavy suture materials passing through a full layer of an incompletely healed left ventricle infarct area can easily cause myocardial tears and severe bleeding from the suture line during the operation and in the postoperative period. For this reason, opening the left ventricle has been avoided in the subacute phase of myocardial infarction, and myocardial healing is expected unless the thrombus in the left ventricle is embolizing. Transatrial methods and endoscopic techniques have recently been developed and have been applied to some patients for the removal of left ventricular thrombi without a ventricular myotomy [Early 2001, Vigneswaran 1998].

In this article, we describe a new approach, first carried out by Dr. Munevver Yuksel, for closing the left ventricle in the subacute phase of AMI after the removal of an intracavitary thrombus.

PATIENTS AND OPERATIONS

Five patients underwent urgent operations for coronary artery disease and a left ventricular thrombus. All patients had an acute AMI 3 to 7 days before surgery. All had successful lower extremity embolectomies before their admission to our clinic, and 4 had unstable angina pectoris as well. Echocardiographic examination of patients showed large nonorganized and nonpedunculated left ventricular thrombi without ventricular aneurysm. In coronary angiography, 3 patients demonstrated 1-vessel disease, and the others had 2- and 3-vessel disease. Left ventriculography was not carried out so as to avoid any further embolic event. Subcutaneous heparin was administered before surgery at doses to hold the partial thromboplastin time to 1.5 to 2 times that of the control time. Patients were operated on under standard conditions of cardiopulmonary bypass and cold blood cardioplegic cardiac arrest. The apex of the left ventricle was opened, and an incision was extended through the anterior free wall parallel to the left anterior descending coronary artery to allow a complete view of the ventricular cavity. The thrombus was totally evacuated, and the ventricle cavity was washed out with saline several times to detach any coagulum particles left on the

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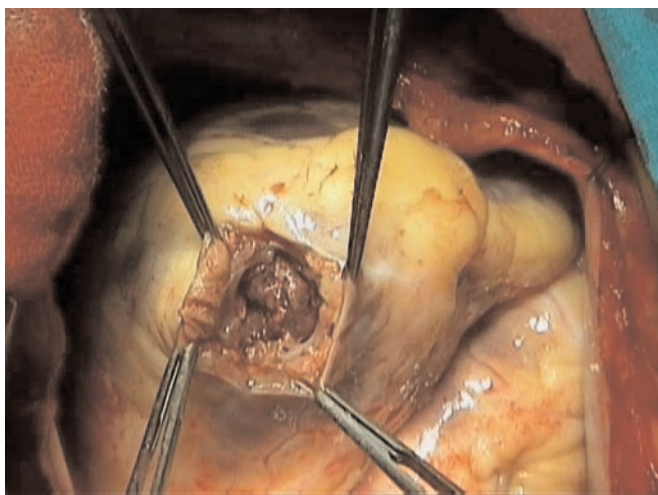


Figure 1. Fresh thrombus is seen in the left ventricle after apical incision. Color of the myocardium indicates nonvascularized ischemic ventricular area.

endocardium (Figure 1). No ventricular tissue resection was made. The ventriculotomy was closed with polypropylene 5/0 sutures biting 3 to 4 mm from the incisional rims and passing in continuous fashion through only the epicardial layer of the left ventricle. A second row of continuous suture running closer to the rims was created to fill the suture spaces of the first row (Figure 2). After the ventricle was closed, coronary artery bypass grafting was applied to all patients.

Intraoperative bleeding from the ventriculotomy suture line was not observed. Median mediastinal drainage in the postoperative period was not greater than that of patients who had not undergone ventriculotomy. The early postoperative period for all the patients was uneventful, and all were discharged on the fifth day after the operation. After the operation, low molecular weight heparin was administered to all patients until warfarin therapy had elevated the international normalized ratio to 2. All patients used therapeutic doses of warfarin after surgery for 6 months. Transthoracic echocardiography at the first, sixth, and 24th months after the operations revealed normal geometry for the apex of the left ventricle for all patients.

DISCUSSION

The incidence of left ventricular mural thrombus occurring following acute AMI as identified echocardiographically can be as high as 46%, even in patients who have had thrombolytic therapy [Moore 1995]. A left ventricular mural thrombus is usually located in the apex and is more common in cases of aneurysm and pseudoaneurysm. Autopsies of patients with mural thrombi have shown that half also exhibited evidence of systemic emboli. Five percent of patients with echocardiographically identified mural thrombi in AMI experience arterial systemic emboli. This ratio is variable and may be as high as 25%. An embolism from a mural thrombus sometimes may be the only presenting symptom of either a silent or an overlooked myocardial infarction. A diagnosed

left ventricular thrombus has often been managed by medical treatment. Thrombolytic therapy in the acute phase of myocardial infarction has been shown to be noneffective in preventing the development of a ventricular thrombus [Moore 1995], but it was found that patients receiving thrombolytic treatment after an acute AMI had a lower incidence of embolic events than did untreated patients [Domenicucci 1999]. Although anticoagulation therapy with heparin and warfarin has been known to be effective in resolving LVT, anticoagulant therapy was shown not to prevent embolization from a chronic LVT [Vidhya 1998].

None of the patients had received thrombolytic treatment before admission to our clinic. Embolized LVT and unstable angina pectoris were indications of urgent surgery. The epicardium and the visceral pericardium were experimentally found to be the layers of the heart that had the maximum tensile strength and stiffness, and these findings were correlated with collagen content. The tensile strengths of the epicardium and the visceral pericardium were 21.3 ± 3.2 g/mm² and 100 g/mm², respectively, whereas it was 4.0 ± 0.3 g/mm² in the midmyocardium. Tests after myocyte necrosis showed that the tensile strength of the epicardium and visceral pericardium remained unchanged and that the midmyocardium represented the weakest element in the ventricular wall in both the normal and the necrotic myocardium [Przyklenk 1987]. The epicardium and visceral pericardium were also speculated to be the collagenous framework that prevented the rupture of the myocardium after transmural infarctions.

Teflon reinforcement redistributes the pressure on the suture line and thereby prevents sutures from cutting through the vulnerable ventricular wall. Reinforcement has been preferred to direct unsupported suturing for avoiding the perioperative or immediately postoperative bleeding that can be fatal after ventricle closure [Vincent 1987]. A Teflon felt buttress has commonly been used for closing the ventriculotomy made

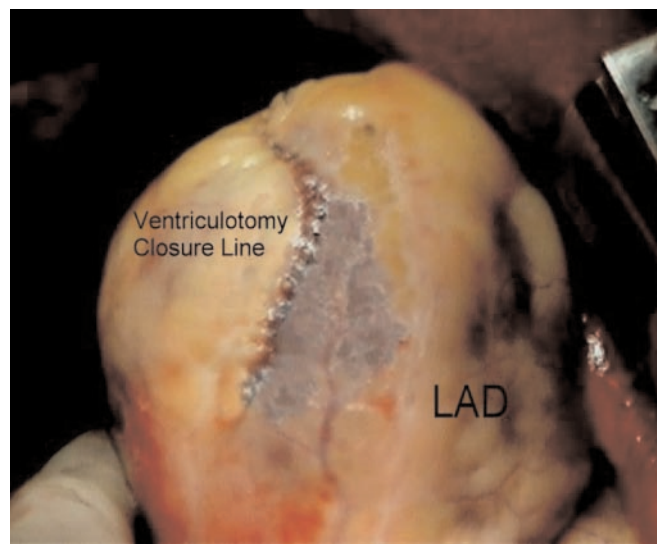


Figure 2. Left ventricular apical incision after closure with 5/0 polypropylene suture. LAD indicates left anterior descending coronary artery.

in ventricular scar tissue in the chronic phase of myocardial infarction, but it is still not reliable for use in the nonhealed myocardial wall during the subacute period. Teflon use also has rare but serious adverse effects, such as immobilization of the sutured area, extensive adhesions to adjacent tissues, calcifications, and foreign body infections of the suture line causing mediastinitis and chronic cardiocutaneous and cardioenteric fistulae [McHenry 1988, Iguidbashian 1993, Danias 1999]. We also thought that suturing the myocardium with Teflon-reinforced heavy suture materials could cause venous congestion and interruption of the lymphatic drainage and the arterial collateral circulation of the region.

New methods for the removal of LVT that avoid left ventriculotomy have been developed. Successful video-assisted cardioscopic and thorascopic left ventricular thrombectomies or transatrial thrombus removal has been reported in recent years [Mazza 1998, Vigneswaran 1998, Early 2001]. The practicability of epicardial closure in any cardiac surgery department and allowing bypasses to the circumflex and right coronary artery are considered ventriculotomy's advantages compared with endoscopic techniques. An anterior extended apical ventriculotomy can supply a better view of left ventricular cavity than the transatrial method and provides the ability to see all of the coagulum.

CONCLUSION

The incidence of left ventricular thrombi after an acute transmural AMI causes systemic embolization and stroke cannot be undervalued. Epicardial ventriculotomy closure can be safely and simply used in patients with left ventricular thrombi and needing urgent surgery because of systemic embolization and unstable angina pectoris in the subacute phase of AMI. The effects of early coronary reperfusion with thrombi removal on the development of apical dyskinesia and aneurysm formation will be investigated with a larger number of patients.

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