Myocardial Revascularization Combined with Intracoronary Infusion of Hyperosmolar Solution in the Early Management of Postinfarction Ventricular Septal Defect

Report of a Case

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SUMMARY
A patient is described with postinfarction ventricular septal defect in whom the perforation was successfully closed within 24 hours of septal rupture. This represents the second such case reported in the literature. Adjunctive measures consisting of myocardial revascularization and intracoronary infusion of mannitol were thought to be important in the successful outcome of the operative procedure. The importance of complete preoperative cardiac catheterization with coronary arteriography is stressed. The theoretical role of endothelial and myocardial cellular edema as a cause of depressed myocardial function immediately following an ischemic insult is proposed as a practical consideration in the high mortality associated with this condition. Methods used to prevent or reverse such cell swelling are described. The details of the operation in which viable ventricular myocardium was used to fill the septal defect are presented.

Additional Indexing Words:
Postinfarction ventricular septal defect
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Rupture of the ventricular septum has been found in 1 to 2% of all patients dying from acute myocardial infarction. 1-3 Although rare, this complication of arteriosclerotic coronary artery disease continues to represent a formidable therapeutic challenge. Medical management alone carries an 87% mortality in the first two months following rupture. 4 Operative mortality continues to be high also, in spite of recent advances in cardiovascular surgery, and has been 50% or greater in most series. 5-7 The two primary determinants of operative risk have been the interval between septal rupture and operation and residual myocardial function. 1-9 The patient described in this report represents the second successful case of rupture of the ventricular septum repaired less than twenty-four hours following perforation. A combined approach of myocardial revascularization and intracoronary infusion of an hyperosmolar solution may have been important in the successful outcome of this patient. Details of the technique are described.

Case Report
W.P., a 60-year-old diabetic male, was admitted to another hospital on 1-23-74 with the diagnosis of acute inferior myocardial infarction. The initial hospital course was uneventful, but seven days after admission he suddenly experienced severe chest pain and wheezing. Examination that morning revealed a holosystolic murmur at the left sternal border and pulmonary edema. The patient was transferred to Emory University Hospital shortly thereafter and was markedly lethargic with cool clammy skin. Blood pressure was 82/50 and heart rate 130 beats/min. Upon arrival, marked jugular venous distention was noted at 90° elevation, diffuse rales were present in both lungs and a grade IV/V1 pansystolic murmur was noted at the left sternal border. Chest X-ray showed bilateral pulmonary edema. The electrocardiogram revealed right bundle branch block with Q waves and ST elevation in the inferior leads. There had been no urine output during the three hours prior to admission.

Emergency right and left heart catheterization and coronary arteriography were performed and demonstrated total occlusion of the right coronary artery, high grade stenosis of the proximal left anterior descending coronary artery with diffuse disease, and a ventricular septal defect along the
diaphragmatic portion of the interventricular septum beneath the tricuspid valve (fig. 1).

The patient deteriorated rapidly after arrival and was taken to the operating room with a systolic blood pressure of 50 mm Hg maintained with a Levophed drip. Femoral vein to femoral artery partial cardiopulmonary bypass was hurriedly initiated under light general anesthesia. Median sternotomy was then performed, total cardiopulmonary bypass instituted, and the patient cooled to 22° centigrade esophageal temperature.

Examination of the diaphragmatic surface of the heart revealed a necrotic area 6 cm in diameter primarily on the posterior wall of the right ventricle, but also to the left ventricular side of the posterior descending branch of the right coronary artery. The area of infarction was incised and a 2 cm ventricular septal defect was seen at the lower portion of the diaphragmatic portion of the interventricular septum beneath the tricuspid valve (fig. 1).

Figure 1

Left) Coronary arteriogram demonstrating total occlusion of proximal right coronary artery. Middle) Left coronary injection showing high-grade stenosis of the proximal LAD with segmental narrowing of distal artery. Right) Left ventricular angiogram revealing early opacification of right ventricle through VSD.

Figure 2

Perforation in posterior-inferior ventricular septum, beneath septal leaflet of tricuspid valve.
the septum (figs. 2, 3). The posterior right ventriculotomy and VSD were closed as a unit by invaginating the edges of the right ventricular myocardium into the septal defect, using interrupted mattress sutures of O-Tevdek tied over teflon felt (figs. 4, 5). In addition, an aortocoronary saphenous vein bypass graft was placed to the left anterior descending coronary artery. During performance of the coronary-vein anastomosis, while the aorta was cross-clamped, 15% mannitol in distilled water was infused into the root of the aorta at a rate of 4 ml/min (total = 82 ml). Rewarming to 37°C was accomplished and the patient was slowly weaned off cardiopulmonary bypass with the aid of epinephrine.

The postoperative course was complicated by a hyperosmolar hyperglycemic nonketotic diabetic state requiring 875 units of regular insulin and a large parenteral free water load over the next three days. On the ninth postoperative day, gram negative bacteremia caused by a urinary tract infection occurred. This was successfully treated with gentamycin and the subsequent postoperative course was uneventful. The patient was discharged on 2-21-74, three weeks after operation. Repeat cardiac catheterization six months following surgery revealed no left-to-right shunt by oximetry or angiography, normal right-sided pressures (PA mean = 17 mm Hg), and an LV pressure (rest) of 150/17. Left ventricular end-diastolic pressure two minutes after angiography was 28 mm Hg. Left ventricular angiography demonstrated a high inferior ventricular aneurysm and mild impairment of apical and antero-lateral wall contractility. The LAD saphenous vein graft was patent (fig. 6). The patient is presently working full time with no angina and minimal exertional dyspnea.

**Discussion**

Rupture of the ventricular septum may occur anytime from four to 12 days following acute myocardial infarction and is heralded by the sudden appearance of a loud systolic murmur and thrill along the lower sternal borders with radiation of the murmur to the right of the sternum or toward the apex. It is frequently confused with papillary muscle rupture, but the two conditions can be differentiated by cardiac catheterization.

The high medical mortality, 70% within two weeks

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Figure 4

Initial layer of closure, using adjacent right ventricular myocardium to obliterate hole in septum.

Figure 5

Completed double layer closure of septal perforation and posterior right ventriculotomy.

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and 80 to 90% within two months of septal rupture, is a consequence of the great hemodynamic burden (acute left-to-right shunt) imposed on a previously injured left ventricle with poor cardiac reserve. 1-4 It has been adequately demonstrated that if operation and closure of the ventricular septal defect can be delayed for six to eight weeks, at a time when hemodynamic stability and collagenation of myocardial fibers can occur, the incidence of residual left-to-right shunt is less, and patient survival rate is much higher. 5, 10-12 In a review of four successful cases of postinfarction VSD and ventricular aneurysm reported by Freeny et al., 13 the interval between infarction and operation ranged from ten weeks to eight years. Although the operative results are clearly better with such delayed treatment, this group comprises only 10 to 20% of the entire patient population, with the overwhelming majority dying sometime during the observation period.

In 1957, Cooley first reported successful closure of a postinfarction ventricular septal defect. 16 Since that time, approximately 60 cases of postinfarction VSD have undergone surgical closure with a hospital mortality varying between 33 and 80%. 5, 8, 14 The two primary determinants of operative success have been the time between perforation and operation and the amount of residual myocardial function following infarction. Probably of equal importance to survival is the ability to revascularize an ischemic myocardium in order to improve contraction of remaining viable tissue. Thus, coronary arteriography and left ventricular angiography are now mandatory prior to surgical intervention. Most patients operated upon in the past have had neither coronary arteriography nor myocardial revascularization. 5, 8, 13, 14

Approximately one third of all previously operated patients have had an associated ventricular aneurysm. In these, the necessity to combine excision of the aneurysm with closure of the septal defect has been stressed. 5, 15, 16 The technique of VSD repair has varied depending upon anatomic findings. Basically, the best repair has consisted of resection of the infarcted segment, repair of the defect with a single or double teflon patch in sandwich fashion on both sides of the septum, and buttressing all sutures with teflon pledgets. 5, 10, 15 With defects located in the posterior portion of the septum beneath the tricuspid valve, as in our patient, exposure is best accomplished through a posterior right ventriculotomy incision and the margins of the ventricular wall invaginated with butressed sutures to fill the ventricular septal defect with viable tissue.

In a recent series of 12 patients undergoing operation for this condition reported by Graham et al., 1 hospital mortality was 50% in the entire series and 100% in the five patients operated upon within twenty-four hours of septal perforation. Review of the literature reveals that only one other patient operated upon within twenty-four hours of septal rupture has survived the operation. 17 Oldham et al. 6 reported five patients operated upon nine days to two and one-half months following rupture of the septum with a 60% mortality. None of the patients operated upon less than a month following rupture survived. Selzer et al. 14 described their experience with six patients undergoing closure of a VSD within six weeks of the time of rupture, and the mortality was 83%.

During the past year we have attempted repair of a ventricular septal defect following inferior myocardial

**Figure 6**

*Left panel* Left ventriculogram demonstrating postoperative posteroinferior ventricular aneurysm. *Right panel* Postoperative patent saphenous vein graft to mid-left anterior descending coronary artery.
infarction in two previous patients shortly after appearance of a holosystolic murmur. Both attempts were unsuccessful. In neither was intracoronary hyperosmolar solution used. Although our present patient may represent a fortuitous occurrence, certain theoretical and practical considerations in the management may have been important.

Leaf18 has recently postulated a possible role of cell swelling in ischemic damage to tissues which we feel is important in patients with acute coronary insufficiency, myocardial ischemia, and cardiac decompensation such as our patient presented. As described by Leaf and coworkers,18,19 all cell membranes are permeable to sodium ions, but under usual circumstances, the inward diffusion of Na+ is balanced by its outward extrusion so that the amount of sodium within the cell is kept low and constant. The outward extrusion of Na+ is an active process deriving its energy from the metabolism of the cell. When the metabolism of a tissue is inhibited by anoxia such that the extrusion process can no longer keep pace with the rate of entry of Na+ into cells, then Na+ accumulates. This net gain of intracellular solute draws H2O into the cells by osmosis.20 If normal metabolic processes are restored before there is irreversible damage, then Na+ will be extruded from the cell, and its volume will return toward normal. Two types of cells seem to be involved in this swelling process: endothelial cells of small vessels and the cells of the organ itself. Cell swelling following a transient ischemic episode may obstruct small vessels and prolong the actual ischemia regardless of the eventual return of organ blood flow to normal or greater than normal levels.

Evidence that cell swelling is important in ischemic tissue injury in experimental myocardial infarction has been presented by Willerson et al.19 Temporary occlusion of the left anterior descending coronary artery produced elevation of the ST segments in electrocardiograms obtained with electrodes over the ischemic myocardium. If mannitol was infused prior to or during the coronary occlusion, not only was the ST segment elevation lowered, but there was an increase in force of contraction and enhanced collateral blood flow to the ischemic area. Thus, there is theoretical and experimental evidence that pretreatment with mannitol will shrink swollen cells, improve blood flow, and increase myocardial work capacity in situations of ischemic insult.

In the patient described in this report, the epicardial surface of the heart at the time of operation was noted to be pale and markedly edematous with exudation of edema fluid from the cut epicardium. It seemed logical that there might also be an element of myocardial edema which, following successful closure of the ventricular septal defect and myocardial revascularization, might inhibit adequate tissue blood flow and cardiac contractility, thereby leading to further ischemic tissue injury, as postulated by Leaf and associates. Preliminary observations in approximately ten other severely ill patients undergoing cardiac surgical procedures for acute deterioration in myocardial function, in which intracoronary infusion of mannitol has been used, have revealed no mortality and no evidence of deleterious effect of mannitol on myocardial function. Thus, in the clinical setting of severe prolonged myocardial ischemia associated with left ventricular dysfunction, as in patients with recent myocardial infarction and subsequent ventricular septal rupture, intracoronary administration of a hyperosmolar solution such as mannitol may reverse myocardial and capillary endothelial edema, thereby enhancing total cellular perfusion and improve cardiac function. It is hoped that further experimental and clinical studies will substantiate these findings.

Addendum

Since the original preparation of this manuscript, another patient with perforation of the ventricular septum has been operated upon successfully, 3 days following an inferior myocardial infarction, using identical methods as described in the above case report.

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