The Heart Surgery Forum #2003-13111
6 (5), 2003
Online address: www.hsforum.com/vol6/issue5/2003-13111.html

Improved Survival with Ventricular Assist Device Support in Cardiogenic Shock after Myocardial Infarction

(#2003-13111)

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ABSTRACT

Background: Cardiogenic shock after acute myocardial infarction is associated with a very high mortality rate.

Methods: A retrospective review was performed of records of all patients supported with an Abiomed device at our institution between 1994 and 2002 to identify those patients who underwent device insertion for the treatment of acute myocardial infarction complicated by cardiogenic shock (AMI-CS).

Results: Seventeen patients who were suffering from AMI-CS and for whom medical management was failing were supported using the Abiomed BVS 5000. The average age of these patients was 57.6 years. Eleven patients were suffering primarily from left ventricular dysfunction and were supported with a left ventricular assist device (LVAD). Eight of these patients were weaned from device support, and 6 survived to hospital discharge (54%). In contrast, 6 patients presented with biventricular failure and were supported with biventricular VADs (BiVADs). None of these BiVAD patients could be weaned from device support. Two BiVAD patients underwent cardiac transplantation, and only one survived.

Conclusion: In the presence of left ventricular failure producing cardiogenic shock after myocardial infarction, LVAD support can produce a 54% survival rate in those patients who are failing medical management. However, in patients in biventricular failure after myocardial infarction, BiVAD support may be used to stabilize the patient until transplantation, but the overall prognosis remains poor.

INTRODUCTION

Cardiogenic shock (CS) occurs in a minority of patients suffering acute myocardial infarction (AMI), but remains the chief cause of mortality after infarction. The management of AMI has evolved with the increased use of thrombolytic therapy and percutaneous interventions early after the diagnosis of infarction has been made. Aggressive therapy has resulted in the improved preservation of myocardium and improved survival. However, a small percentage of patients continue to exhibit signs of cardiogenic shock despite urgent revascularization and intra-aortic balloon pump (IABP) support.

Despite aggressive support, the mortality rate for CS following AMI (AMI-CS) remains high. In the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) Trial Registry, the mortality rate varied from 47% in patients treated with IABP support and thrombolytics to 77% in those patients receiving neither therapy. Revascularization is important in minimizing the mortality rate and reduced the mortality rate to 37% in those patients treated with IABP and thrombolytics [Sanborn 2000].

There is no consensus regarding the management of patients who remain in CS after the institution of appropriate measures. In many of these patients, end-organ dysfunction worsens as the duration of shock lengthens, making further therapy less likely to be effective the more it is delayed. Ventricular assist devices (VADs) are useful in treating either acute or chronic cardiac failure from a variety of causes. Short-term VADs are most commonly used in the treatment of postcardiotomy CS and can improve the survival in cases in which patients are unweanable from cardiopulmonary bypass after a cardiac operation and in numerous other conditions. These devices may also be used in the temporary support of patients who are exhibiting signs of CS following MI. This period of support allows for the reversal of systemic hypoperfusion, improvement of end-organ damage, and performance of other interventions that may result in improved cardiac function once the period of shock has resolved.

MATERIALS AND METHODS

The records of patients supported on an Abiomed 5000 BVS (Abiomed, Danvers, MA, USA) at Hahnemann University Hospital (HUH) between 1994 and December 2002 were retrospectively reviewed to identify those patients who underwent placement of an Abiomed BVS 5000 LVAD or biventricular assist devices (BiVAD) for the diagnosis of CS-AMI. CS was defined as systemic hypotension despite the use of IABP and/or inotropic support in patients who had suffered a documented AMI and were initially seen either at HUH or a

Presented at the 9th Annual CTT Meeting 2003, Miami Beach, Florida, USA, March 19-22, 2003.

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referring hospital. Patients were included in the study if the onset of CS occurred either before or after admission or transfer to HUH. They were included if they had the VAD inserted at HUH, or if they were transferred to HUH for management of a VAD inserted at an outside hospital. At the time of VAD insertion, all patients had evidence of end-organ hypoperfusion with the presence of diminished urine output, a rising serum creatinine, cardiac index <2.0 L/min per m^2, altered mental status, or cool extremities. Patients were included in the review if CS was caused by a complication of AMI such as a ventricular septal defect (VSD) or ruptured papillary muscle, but they were excluded from the study if insertion of the VAD was not the primary reason for performing cardiac surgery or if CS was not present prior to surgery. Patients who underwent concomitant surgical revascularization were included only if the operating surgeon performed the bypasses as an adjunctive therapy to VAD support.

All patients were supported on the Abiomed BVS 5000 VAD, in either the LVAD or BiVAD configuration. VAD implantation was performed under full anticoagulation with heparin and was done with or without the assistance of cardiopulmonary bypass support during device placement. For the LVADs, inflow cannulation was either through the left atrium near the right superior pulmonary vein or through the left ventricular apex. The outflow cannula was attached to the ascending aorta. The inflow cannula for the right VAD (RVAD) in the BiVAD configuration was placed in the right atrium, and the outflow cannula was either advanced into the main pulmonary artery through a pursestring in the right ventricular outflow tract or sewn directly onto the main pulmonary artery.

Postoperative management of the patients on VAD support varied significantly because of the diverse nature of the patient population and has been described previously [Samuels 2001]. In general, systemic anticoagulation with heparin was initiated as soon as the mediastinal bleeding had diminished, usually within 24 hours. IABP support was discontinued at the time of VAD placement. Inotropic support was maintained if necessary to support the right ventricle. Patients were monitored with Swan-Ganz catheters and echocardiograms as necessary to determine the timing for VAD weaning. Patients were evaluated for cardiac transplantation if it appeared that cardiac recovery was not likely and if there were no absolute contraindications to transplantation. VAD weaning was undertaken when it was determined that ventricular recovery had occurred and end-organ function had stabilized to the point that discontinuation of support appeared to be reasonable. Patients with acute renal failure were supported with continuous veno-venous hemodialysis when necessary. VAD blood pumps were changed at the bedside if organized thrombi developed in the chambers.

Patients who underwent VAD weaning were taken back to the operating room, and explantation occurred as described [Samuels 2001]. Invasive monitoring lines were placed and the patient anticoagulated with heparin to produce an activated clotting time (ACT) of greater than 300 seconds. Explantation was usually performed with the assistance of transesophageal echocardiography.

RESULTS

A total of 88 patients were supported on Abiomed BVS VADs between June 1994 and December 2002 for a variety of causes. The devices were implanted at HUH in 69 patients and at outside hospitals in 17 patients. The overall rate of survival to discharge in this group of patients was 35.2%.

Seventeen patients were supported on an Abiomed 5000 BVS LVAD or BiVADs with the primary diagnosis of AMI-CS. There were 11 male patients and 6 female patients. The average age was 57.6 years (range, 41-79). Triple-vessel disease was present in 7 patients, double-vessel disease in 7 patients, and single-vessel disease in 3 patients. The MI was anterior in 8 patients and posterior/inferior in the rest. Eleven patients were supported with an LVAD alone and 6 with BiVADs. Of the 6 female patients, 5 required BiVAD support. Eleven patients were transferred to HUH for management while in CS, 1 was transferred after LVAD placement for AMI-CS, and the remaining patients developed CS while at HUH.

The primary reason for VAD insertion was MI in 11 patients, infarction complicating primary percutaneous transluminal coronary angioplasty or stent placement in 3 patients, and complicated MI in 3 patients. Patients with complicated MI included 1 patient with CS due to acute papillary muscle rupture and 2 patients with postinfarction VSD. The average documented time between MI and the onset of CS was 2.38 days. The average duration of CS was 1 day before VAD insertion (range, 1-4 days).

Fifteen patients had an IABP placed to treat the CS before VAD insertion. Seven patients underwent pre-VAD percutaneous intervention to reopen the culprit vessel, but this procedure often resulted in "no-reflow." Six patients underwent coronary artery bypass graft (CABG) at the time of VAD insertion, but the bypasses were performed for concomitant disease, to the target vessel that had already been opened through percutaneous techniques, or to an area where the infarct was judged to be completed but residual viable myocardium appeared to remain. Two patients underwent percutaneous revascularization after several days of VAD support in anticipation of explantation. One patient underwent mitral valve replacement and single-vessel CABG at the time of explantation for papillary muscle rupture.

Of the 11 patients supported on an LVAD alone, 8 were successfully weaned from support for a period of 24 hours or greater. One of these patients underwent insertion of a HeartMate LVAD (Thoratec, Pleasanton, CA, USA) the day after Abiomed removal, because of recurrent heart failure. Seven of the LVAD patients survived >30 days after device placement. One of these patients was supported on the LVAD for 9 days but was ventilator dependent and died with multiple medical problems 171 days after device removal. Six LVAD patients were discharged, including 1 patient who was discharged to home on HeartMate support while awaiting transplantation.

The overall length of support was 13 days for the entire group of patients but was only 6 days for the patients who survived AMI-CS with LVAD support. The shortest duration of support was 6 hours in a patient who had acute thrombosis of her LAD during manipulation of a proximal circumflex

coronary lesion and who could not be stabilized after perfusion to the LAD was restored in the catheterization lab. She began to exhibit cardiac improvement shortly after arrival to the intensive care unit, but then her condition deteriorated when deoxygenated blood began to shunt across an undiagnosed patent foramen ovale. Device removal was tolerated and led to immediate resolution of the profound arterial oxygen desaturations. The longest period of support was in a patient who had a postinfarct VSD and was in profound CS on arrival to the hospital. BiVADs were placed immediately. Once the patient was stabilized, he declined conversion to a Thoratec (Thoratec, Pleasanton, CA, USA) BiVAD system and awaited successful orthotopic heart transplantation (OHT) combined with renal transplantation that occurred after 58 days of support.

The average length of hospitalization was 45 days after VAD implantation for all patients and 55 days for those patients who survived after LVAD support. The shortest duration for hospitalization in a survivor was 33 days after the initiation of support. Three deaths occurred within 5 days of initiation of VAD support, but many of the nonsurvivors required prolonged hospital care before succumbing. The causes of death were varied, but were usually related to end-organ damage suffered during the period of CS or to complications from VAD support. Significant complications included sepsis (n = 3), renal failure (n = 3), multisystem organ failure (n = 5), stroke or other serious neurological injury (n = 6), and a fatal ventricular arrhythmia that occurred upon device removal in 1 patient. Most of these complications resulted in or contributed to mortality of the patient.

DISCUSSION

Despite advances in medical care, a small percentage of patients continue to suffer from CS following AMI. Although survival rates have been improved with the combined use of thrombolytics and IABP support, some patients remain in shock. As the cardiac injury becomes irreversible, surgical or percutaneous revascularization become less likely to reverse the hemodynamic instability. As the duration of end-organ malperfusion lengthens, chance of survival diminishes. In these patients, VAD therapy may be useful to provide hemodynamic support, allowing cardiac function to recover while peripheral perfusion is maintained at adequate levels.

VAD support for AMI-CS is not a new concept, but its adoption has been met with resistance from cardiologists and cardiac surgeons alike. This reaction may be related in part to the intensive use of resources required in the VAD population and the assumption by many that the complications of short-term VADs are excessive. However, patients with AMI-CS have a high mortality rate that can be decreased only with the widespread use of available technologies [Sanborn 2000]. When the conventional management strategies fail, consideration must be given to more aggressive methods of treatment. Support for this strategy is uncommon in the literature. Thiele et al [2001] used a percutaneous left atria-to–femoral artery device to support 18 patients who were in AMI-CS for a mean of 4 days. The overall mortality rate was 44%, but these patients were not treated with an IABP prior to VAD implantation. Thus it is difficult to say whether VAD insertion was necessary, because conventional treatment methods had not been exhausted. Indeed, the overall mortality of the patients in this study was not much different from that of patients who were treated by thromolysis and IABP support, with or without revascularization, in the SHOCK Trial Registry [Hochman 2000, Sanborn 2000]. LVAD use was also mentioned in the reports of the SHOCK Trial Registry, but was used in only 0.8% of the patients, and no data are given to evaluate the outcomes of this subgroup of patients [Hochman 2000].

The current study demonstrates that VAD support can provide reasonable prospects for survival in patients who experience CS as a result of MI and who do not improve with standard therapies. Patients supported with an LVAD after AMI had improved outcomes compared to those who required BiVAD support. In this study, 8 of 11 LVAD patients could be successfully weaned from device support, and 54% of the total were discharged to home. In contrast, none of the BiVAD patients could be completely weaned off of support, although 1 patient underwent RVAD removal. The only BiVAD survivor underwent OHT 58 days after device placement for a VSD associated with severe biventricular dysfunction precluding operative VSD repair. The mechanisms behind the improved results with LVAD support are unknown, but probably reflect a lesser degree of underlying cardiac damage and less end-organ damage from malperfusion before support could be initiated. However, it is interesting that 5 of the 6 BiVAD patients were female, and the only female to survive VAD support for AMI-CS was supported on an LVAD. At our hospital, the survival rate for female patients supported with a VAD is 25%, which is slightly lower than that for males.

Unfortunately, the survival rate of patients who persist in CS despite the use of standard therapies is unknown. In the SHOCK Trial Registry, the mortality rate of patients in AMI-CS was 83% in those who received neither thrombolytic therapy nor IABP support, and this rate was decreased to 47% if they received both. The addition of revascularization further decreased the mortality rate to 37% [Sanborn 2000]. However, those patients who underwent revascularization were also younger, had CS diagnosed more frequently within 6 hours of the AMI, and had improved cardiac function at the time of presentation [Hochman 2000]. Overall, the patients represented in the SHOCK Trial Registry were not as critically ill as the patients in this current report for whom the standard treatment algorithm had already failed.

Early revascularization is an important adjunct in treating AMI-CS [Dauerman 2002]. Revascularization was attempted in all patients for whom it was felt that the procedure would be beneficial. Eight patients underwent percutaneous procedures but remained in CS. There was a high incidence of "no-reflow," suggesting that the period between MI and reperfusion was too long to acutely restore function to the damaged area of myocardium. In addition, 6 patients underwent CABG at the time to implant, including 2 who had already undergone percutaneous intervention. However, in all of these patients the operating surgeon felt that he was

performing the revascularization in order to improve the chances of successful explantation rather than to avoid VAD implantation. Those patients who did not get any type of revascularization were felt to have completed infarcts and it was believed that any further delay would jeopardize their survival. Three of the 6 LVAD survivors received either no revascularization, delayed percutaneous intervention before explantation, or CABG/ mitral valve replacement at the time of explantation, suggesting that stabilization alone may be adequate in patients presenting in profound shock, especially if it is felt that the infarct has completed.

Prompt recognition of CS that is unresponsive to conventional therapies is critical to successfully supporting AMI-CS patients with a VAD. In this study, most of the patients died either directly or indirectly from persistent end-organ dysfunction that did not improve with the institution of cardiac support. In addition, these mortalities were often delayed; only 3 patients died within the first 5 days of support. The patients who suffered delayed mortality required extensive stays in the intensive care unit and hospital, and consumed large amounts of resources in the process while the patient was supported in anticipation of renal, hepatic, or neurological improvement. In an attempt to minimize the potential to support patients who have already suffered irreversible end-organ injury, we have a policy to decline VAD insertion in any patient who is not neurologically intact or in whom a significant event has occurred since they were last evaluated if they are sedated at the time of referral. We do not insert these devices in any patient who either is not a transplantation candidate or does not have a reasonable chance for myocardial recovery. We have found it difficult to predict which patients with declining urine output and rising liver function tests are salvageable, and therefore we are somewhat reluctant to use these results alone to deny a patient potentially life-saving therapy. However, these criteria may change as we gain experience in treating AMI-CS patients.

A critical component of success with a program that uses VADs to support those patients in AMI-CS who have failed standard therapies will be prompt referral from the cardiologists, quick assessment of the recoverability of ventricular function after a period of support, and immediate institution of support. If a BiVAD is necessary, then it may be reasonable to deny the patient VAD support unless transplantation is a viable alternative, because these patients are otherwise unlikely to survive. However, as long-term VADs gain more acceptance as destination devices, this recommendation may change in the future. Regardless, there must be a mechanism to offer these patients transplantation or long-term device insertion should it become necessary. This process may occur at the implanting hospital, or the patient could be transferred to a transplantation/ VAD center once the patient has stabilized after implantation.

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