



Case Report

Potential Risk of Paradoxical Embolization during Use of a Left Ventricular Assist Device: A Case Report

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ABSTRACT

A patient undergoing left ventricular assist had a complicated post-operative course due to a patent foramen ovale. This presented as a right to left shunt with desaturation of the arterial blood, and possibly contributed to paradoxical embolization resulting in neurological complications and death. A method is presented to determine the presence of a patent foramen ovale during left ventricular assist. Possible sources of embolization are discussed.

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INTRODUCTION

A patient undergoing left ventricular assist exhibited reduced arterial blood gas saturation early in the procedure. Transesophageal echocardiography showed the presence of a patent foramen ovale (PFO). Decreasing the flow of the assist device allowed reduction of the right to left shunt, and resulted in increased arterial oxygen saturation. The patient was successfully weaned from the assist device after 65 hours, but had suffered a large left cerebral hemispheric infarct, and died from neurological complications 10 days postoperatively.

The presence of a patent foramen ovale during left ventricular assist has previously been reported. (1,2) However, possible embolic events associated with a PFO during left ventricular assist have not been observed. Air that was introduced from intravenous lines into the right heart may have been drawn into the ventricular assist device via the PFO, and pumped into the arterial vasculature, resulting in the embolic event.

CASE REPORT

A 62-year-old male was admitted to the hospital with unstable angina. He had previously undergone double bypass surgery in 1982 and 1986, and was scheduled for repeat double bypass surgery. Both previous bypass operations were complicated by cerebral vascular accidents, which resulted in transient right-sided weakness. For the present procedure, the pump circuit consisted of a roller pump^a, a Bard^b 5700 membrane oxygenator, and a Sorin^c BCD 4:1 blood cardioplegia set. The pump prime consisted of 2000 ml lactated Ringers, 50 mEq sodium bicarbonate, 5000 units heparin, 50 g of albumin, and 2 million units of aprotinin.^d Anesthesia, surgery, and cardiopulmonary bypass were initiated without difficulty. An anastomosis was made from the left internal mammary artery to a diagonal branch, and the right internal mammary was attached to the posterior descending branch of the right coronary artery. The surgery was uneventful, requiring a cross-clamp time of 52 minutes and a cardiopulmonary bypass time of 72 minutes. Weaning was attempted, but bypass was resumed after five minutes due to EKG changes indicative of global ischemia, low systemic pressures, and high pulmonary artery pressures. An additional 43 minutes of bypass were required to optimize pharmacological support, which consisted of infusions of amrinone, epinephrine, dobutamine, and phenylephrine. In addition, intra-aortic balloon pump support was initiated. The patient was again weaned from bypass, but the EKG changes recurred and the patient again developed cardiogenic shock. Bypass was resumed and a saphenous vein graft was added to the patient's diagonal artery to augment the mammary artery flow. The cross-clamp time for this procedure was 22 minutes.

Following a further unsuccessful attempt to wean from bypass, a left ventricular assist device (LVAD) was inserted using a Medtronic^e CB2978 LVAD tubing pack. This is a Carmeda Bioactive Surface^e heparin coated circuit, consisting of

a Bio-Medicus^e BP 80 biohead, 3/8 inch tubing and connectors. A Duraflo II heparin coated OxySat^f cell was added to the LVAD circuit. A Duraflo II coated RMI VR-032-60-90 32 Fr. Left Atrial Venous Return Cannula^g and RMI ARXL-024-S 24 Fr. Aortic Arch Cannula^g were utilized. Thus the entire circuit was heparin coated for thromboresistance. A pressure transducer was added using a 3/8 X 3/8 inch luer connector placed distal to the biohead. A left atrial pressure line^h was inserted to monitor the left atrial filling pressure. The LVAD was initiated at 4 liters per minute and the patient was successfully weaned from bypass. The total cardiopulmonary bypass time was 252 minutes, with a total of 74 minutes cross-clamp time. Protamine was given, and the patient was transferred to the cardiac intensive care unit with LVAD support. The patient did not receive any supplemental heparin while the LVAD was at full flow. Activated clotting times (ACT) ranged from 140 to 180 seconds (measured by a Hemochronⁱ system).

The arterial oxygen saturation measured by the OxySat began to fall approximately 8 hours into the LVAD procedure. These measurements were confirmed by arterial blood gas determinations. This fall in saturation appeared to be correlated with lower left atrial pressures and was thought to be a shunting effect in the lungs. The patient was given additional intravascular volume to increase filling pressures, but oxygen saturations remained unchanged. Approximately 18 hours later, transesophageal echocardiography revealed a patent foramen ovale (PFO), with right to left shunting augmented by the LVAD. When the LVAD flows were decreased from 4 to 3 L/min, the shunting decreased and arterial saturations increased. The patient was supported for an additional 30 hours at 3 L/min flow. Prior to weaning from the LVAD, heparin was given in order to maintain ACT at 200 seconds. The patient was successfully weaned from the LVAD after a total of 65 hours, 40 minutes of support, and the cannulae removed. Two days later the intra-aortic balloon catheter was removed. At this time, hemodynamics were maintained with infusions of amrinone, low dose epinephrine and phenylephrine with a cardiac output over 5.5 L/min. When sedation was decreased, the patient's pupils were equal and reactive, but his level of consciousness was severely decreased. Nine days postoperatively, a computed tomography (CT) scan was performed revealing a large left cerebral hemispheric infarct, estimated to be 1 - 2 weeks old, as well as two other focal infarcts on the right side. The patient died 10 days postoperatively due to neurological complications.

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- b Bard Cardiopulmonary, Tewksbury, MA, 01876
- c Sorin Biomedica, Irvine, CA, 92713-9503
- d Bayer AG, Leverkusen, Germany
- e Medtronic Inc., Anaheim, CA, 92807
- f Baxter Bentley Laboratories, Irvine, CA, 92714
- g Research Medical Inc., Salt Lake City, UT, 84119
- h DLP Inc., Grand Rapids, MI, 49501-0409
- i International Technidyne, Edison, NJ, 08820

DISCUSSION

Although the LVAD resulted in hemodynamic improvement, the patient did not survive due to neurological deterioration. The cause of the cerebral infarction is not specifically known, but a number of contributing factors are possible. The patient may have been at risk for a stroke because of his previous history of cerebral vascular disease. Systemic heparinization for cardiopulmonary bypass might have been a contributing factor, as might have been paradoxical shunting of air or thrombus through the PFO. Previous reports noted shunting caused by a PFO during LVAD support, resulting in hypoxia, but paradoxical embolic complications have not been described. (1,2) Embolization of small amounts of air to the right side of the heart from intravenous lines is a common occurrence. In this case, the LVAD may have facilitated passage of air from peripheral intravenous lines through the PFO, and subsequently returned them to the aorta. The resulting emboli may have entered the patient's cerebral vasculature. Another possibility is that thromboemboli may have arisen from the heparin coated circuit. However, the circuit was thoroughly flushed after the procedure and no thrombus was observed in the tubing or the pump head. Previous LVAD's performed with circuits without heparin coating have often shown fibrin deposits or clot formation around connectors or at the back of the Bio-Medicus pump cone. (3,4) These were not observed in the circuit in this case. The use of aprotinin, which inhibits fibrinolysis, may enhance micro-clot formation and secondary emboli. A recent report indicates that after administration of protamine to a patients who had received aprotinin, extreme heparin resistance occurred. [Fisher T. *The Effect of Aprotinin on Reheparinization of Patients undergoing Open Heart Surgery. Presented at The Society of Perfusionists of Great Britain and Ireland, 1992 Congress on Perfusion, October 9-10, 1992.*] The Bentley OxySat meter facilitated detection of the PFO. This device is routinely used in our institution on all left or right ventricular assist cases, since it shows decreased oxygenation quickly and accurately.

The CT scan showed lesions compatible with cerebral air embolus or thrombus. The absence of other evidence of thrombosis suggests that paradoxical air embolism was the cause of the stroke. If a PFO is suspected in a patient during an LVAD, the easiest test to confirm this is to decrease the LVAD pump blood flow and note any corresponding increase in the OxySat reading and the arterial blood gas pO_2 . This decreased pump flow may diminish or stop the shunt of desaturated blood into the LVAD circuit. Furthermore, the arterial oxygen saturation should rise and the risk of paradoxical emboli would decrease. In the unlikely event the left atrial cannula was accidentally passed through the PFO into the right atrium, this could be determined by the OxySat readings which would reflect mixed venous oxygen saturations.

The use of aprotinin during surgery before initiating a ventricular assist device (VAD) may require changes in protocols for heparinization. For example, the protamine dose given

after bypass may be decreased, or heparin given to maintain an ACT of greater than 200 seconds for the first 12 hours, followed by gradual drop in ACT. The use of heparin-coated circuits may prove advantageous if systemic heparinization can be avoided, with less bleeding from cannulation sites and in the chest cavity. As our experience showed, the heparin-coated circuit seems to decrease the incidence of clot formation in the LVAD circuit. This contrasts markedly with the results obtained from previous experience with the other types of heparin coating. (5)

CONCLUSION

An estimated 35% of the adult population have a PFO. (6) If a PFO is suspected during use of a LVAD, manipulation of pump flows with simultaneous arterial oxygen saturation monitoring may support this diagnosis. Transesophageal echocardiography is useful to verify the existence of PFO. The use of aprotinin during the initial operation may require changes to the heparin protocol for all ventricular assists, even if the circuit is heparin coated. While spontaneous coagulation deficiencies may be sufficient to prevent clotting in the VAD circuit, the decrease in fibrinolysis caused by aprotinin may be sufficient to present increased risk of thrombosis to the circuit and embolization to the patient.

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