Case Report

Fatal Retrograde Aortic Dissection During Cardiopulmonary Bypass for Reoperative Mitral Valve Replacement

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Abstract

In September 1991 a 62-year-old female presented to our institute for replacement of a prosthetic mitral valve. She was cannulated for cardiopulmonary bypass via the right femoral artery with venous drainage through the inferior and superior vena cava. During rewarming there was a sudden drop in arterial blood pressure. After the cross clamp was removed the heart remained flaccid with no apparent perfusion of the coronary arteries. As a result of this, the diagnosis of retrograde aortic dissection was made and the ascending aorta was immediately cannulated. In spite of this action, severe metabolic acidosis, hyperkalemia and oliguria had developed. After two attempts to wean the patient off bypass and when maximal pharmacological support had failed, bypass was discontinued and the patient was pronounced dead.

Introduction

Acute dissection of the aorta is a potentially catastrophic occurrence that is described by separation of the medial layer of the cannulated artery which results in the creation of a false lumen[1]. Subsequently, blood flow in the false lumen may cause distal or proximal dissection that leads to compression of the true lumen occluding major branches of the aorta. Consequently, death can result due to multi-organ ischemia and or rupture of the aorta.

Retrograde aortic dissection originating from the femoral artery cannulation site is a disastrous complication during cardiopulmonary bypass. Reported incidence during the early years of extracorporeal circulation range from 0.6 percent to as high as 5.3 percent[2-7]. A more recent report has shown the incidence to be as low as 0-0.7 percent for dissection of the ascending aorta[3-7,9-11].

Case Report

In September 1991 a 62-year-old female presented to our institution for replacement of a prosthetic mitral valve. The patient had developed mitral valvular disease as a result of rheumatic fever in her youth. She underwent closed mitral commissurotomies in 1961 and 1976, and received a Bjork-Shiley prosthetic valve in 1984. In October and December 1986 the patient sustained a Wenchenback type stroke secondary to septic embolization that left her with left hemiparesis. She had been well since that time except for some breathlessness and motor difficulty. In 1989 the patient was admitted in congestive heart failure with fever. Subsequently, a diagnosis of enterococcus bacterial endocarditis was made and treated medically. She was readmitted in the summer of 1991 again with bacterial endocarditis. As a result of this, cardiac catheterization was performed and revealed wide open mitral regurgitation due to a perivalvular leak. Consequently, it was decided that the patient needed replacement of the prosthetic valve.

Preoperative evaluation showed a cachexic patient with a weight of 45 kilograms, a height of 167 centimeters and a BSA of 1.47 meters square. Admission lab values were within normal limits except for a hematocrit of 30 percent and a PTT measuring 108 seconds. The patient was noted to be on I.V. nitroglycerin, digoxin, antibiotics and heparin.

The patient was taken to the operating room and prepared

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for surgery. With the patient under general anesthesia an endotrachael tube, pulmonary artery catheter and right radial arterial line were inserted for perioperative monitoring.

A Bio-Medicus BP-80[a] blood pump was used in conjunction with a Bard HF-5400 hollow fiber membrane oxygenator and tubing pack[b], which included a Pall auto vent arterial filter[c]. The prime consisted of Normosol-R-1500ml[d], hetastarch-500ml, packed red blood cells (PRBCs)-250 ml, mannitol-25 g, $NaHCO_3$-50 mEq and heparin-10,000 units.

13,500 units of heparin were administered via the central line with a resultant activated clotting time (ACT) of 370 seconds. An additional 7000 units were administered prior to going on bypass. The patient needed a total of 19,000 units throughout the duration of the case to maintain an ACT > 480 seconds. The surgeon decided to cannulate the femoral artery due to the numerous open heart procedures the patient had experienced. Subsequently, the right femoral artery was cannulated with a Cinco 5.4 mm stainless steel cannula[e]. Venous access was obtained via bicaval cannulation of the superior and inferior venae cavae with a 32 French TF cannula[f]. The patient was then placed on cardiopulmonary bypass and immediately cooled to 26°C. The heart was arrested with cold blood cardioplegia mixed 4:1. At 26°C the pump flow rate was maintained at 3.2 l/min (2.17 l/min/m2) and the line pressure ranged from 160 to 200 mg. Sodium nitroprusside and isoflurane were administered to maintain the blood pressure between 50 and 60 mmHg. Arterial and venous blood gases were obtained every 30 minutes and analyzed by the GEM 6 Plus on-line analyzer[g].

Approximately 70 minutes into the case the order was given to begin rewarming. Subsequently, the flow rate was increased to 4.2 l/min, this increased the line pressure to 250 mmHg. Seven minutes into rewarming the line pressure had gradually increased to 303 mmHg. Upon reaching this pressure, there was a sudden drop in the right radial pressure from 65 to 20 mmHg. The surgeon was notified of the situation and since no clear-cut sign of dissection was apparent it was decided to continue with placement of the valve. Acid base status, electrolytes and hematocrit were monitored closely and initially remained within normal limits. Venous return and urine output remained unchanged compared with that prior to the drop in arterial pressure. Arterial and venous temperatures steadily rose to 37°C although esophogeal temperature was observed to lag behind by 8°C. Forty minutes after the drop in arterial pressure the anesthesiologist noticed that the right side of the patient's face was cold while the left was warm. Ten minutes after this time and before the cross clamp was removed a left femoral artery cut down was done with no discernible pressure measured. Next, the cross clamp was removed and the heart remained flaccid with no apparent perfusion of the coronary arteries. As a result of this finding the diagnosis of retrograde aortic dissection was made. The ascending aorta was then cannulated with a Sarns 24 mm aortic cannula[h]. Immediately thereafter, the arterial pressure returned to 50 mmHg, and the heart began to fill and beat spontaneously. By this time all three temperatures had equalized and the patient was prepared for termination of bypass. Arterial blood gases revealed a slight metabolic acidosis that was treated with 50 mEq of $NaHCO_3$. Hematocrit was found to be 21 percent and two units of PRBCs were given. The initial attempt to wean the patient off bypass was unsuccessful. In spite of inotropic support the blood pressure could not be maintained greater than 60 mmHg. Blood gases showed a worsening metabolic acidosis with an acute elevation of the potassium from 4.5 to 6.5 mmol/l. After administration of 100 mEq of $NaHCO_3$ a second attempt was made to wean the patient off bypass. Initially, the patient sustained systolic pressures of 90 mmHg with a cardiac output of 3 l/min. Unfortunately, the systolic pressure progressively drifted down to 50 mmHg. As a result of this the patient was placed back on bypass. It was felt that a third attempt at coming off bypass would be futile due to the development of a severe metabolic acidosis and acute hyperkalemia. In addition to this there was an acute cessation of urine output which had been brisk throughout the case. This was attributed to redissection with occlusion of the renal arteries. Consequently, bypass was discontinued and the patient was soon thereafter pronounced dead.

A consent for autopsy was obtained and it revealed an extensive dissection of the right femoral, common iliac arteries, and aorta extending from the upper portion of the femoral artery to the root of the aorta. There was also an aneurysm of the intracranial portion of the left internal carotid artery. Finally, there was an old cerebral infarct of the right parietal and parasagital area of the brain.

Discussion

Early recognition and management of retrograde aortic dissection by the surgical team is important for a favorable outcome. The signs of impending aortic dissection may be obscure or absent until the event has already occurred. In a series of seven patients reported by Carey et al, all seven patients exhibited a sudden drop in arterial perfusion pressures along with a bluish coloration of the ascending aorta[12]. Benedict and colleagues reported on five patients with retrograde dissection and found a sudden and unexplained fall in venous

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d Abbott Laboratories, North Chicago, IL 60064
e Cinco, Wakefield, MA 01880
f Research Medical Inc., Midvale, UT 84947
g Mallinckrodt, Ann Arbor, MI 48108
h Sarns, 3M Health Care, Ann Arbor, MI 48106

return, increased perfusion line pressure, fall in arterial blood pressure, or fall in urine output as acute signs[7].

There are several theories about the cause of retrograde aortic dissection. It can occur as a result of trauma during insertion of the cannula into the artery[13], and in vessels with atherosclerosis or cystic medial necrosis[14]. The increased velocity and turbulence of blood as it exits the cannula creates jet streaming and eddy currents that may separate intimal and adventitial layers[13]. Koyama et al found much higher shear stress values in femoral arterial walls during femoral artery perfusion as compared to that of ascending aortic perfusion[8]. As a result of this finding they concluded that the ascending aorta was the preferred site for arterial cannulation.

In our experience with this case the signs of impending dissection were subtle. The gradual increase in the line pressure was not thought to be abnormal and was related to increasing the flow rate. We also did not see a sudden rise in the line pressure as has been described in the literature. This could have been related to the type of blood pump used in this case. As is commonly known, when using the Bio-Medicus pump, as resistance to flow increases, pump flow rate will decrease. Therefore there would not be the rapid build-up in pressure as would be seen when using a positive displacement pump. Another explanation is that there was reentry of blood flow back into the true lumen of the aorta. This might explain why venous return and urine output initially remained normal. The uneven rewarming observed was probably a result of either malposition of the esophogeal temperature probe or regional hypoperfusion of the vasculature supplying the esophagus. The asymmetrical facial temperatures observed by the anesthesiologist could be a direct result of obstruction of those vessels supplying the face as the dissection progressed toward the aortic root.

Conclusion

Acute retrograde aortic dissection remains a potentially disastrous complication of femoral artery cannulation. It is vital to closely monitor line pressure, blood pressure, venous return, urine output and acid base status in all patients while on cardiopulmonary bypass. It becomes even more important when the patient is being perfused via the femoral artery. In spite of close monitoring, the signs of dissection can often be obscure. Immediate recognition of retrograde aortic dissection and establishment of antegrade flow via the ascending aorta is imperative to reduce the mortality of this rare complication of cardiopulmonary bypass.

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