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 institution 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. 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. A more recent report has shown the incidence to be as low as 0-0.7 percent for dissection of the ascending aorta.

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 commissurotomy 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 PTIT 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...
for surgery. With the patient under general anesthesia a
dotracheal tube, pulmonary artery catheter and right radial
arterial line were inserted for perioperative monitoring.

A Bio-Medicus BP-80a blood pump was used in conjunc-
tion with a Bard HF-5400 hollow fiber membrane oxygenator
and tubing packb, which included a Pall auto vent arterial
filterc. The prime consisted of Normosol-R-1500ml4,
hetastarch-500ml, packed red blood cells (PRBCs)-250 ml,
mannitoll-25 g, NaHCO3-50 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 cannulae. 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 200mg. 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.

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 pres-
sure, 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 esophageal 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 re-
moved 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 Sams 24 mm
aortic cannulae. 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 equal-
ized and the patient was prepared for termination of bypass.

Arterial blood gases revealed a slight metabolic acidosis that
was treated with 50 meq of NaHCO3. 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 NaHCO3 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 ar-
teries, 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 parasagittal area of the brain.

Discussion

Early recognition and management of retrograde aortic dissec-
tion 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 aorta12. Benedict
and colleagues reported on five patients with retrograde dissec-
tion and found a sudden and unexplained fall in venous

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b Bard Cardiopulmonary Division, Tewksbury, MA 01876
c Pall Biomedical Products, Glen Cove, NY 11542
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 Sams, 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.

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, and in vessels with atherosclerosis or cystic medial necrosis. 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. 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. 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. 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 esophageal 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, arterial 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.

**References**