Pulmonary Hemorrhage from Pulmonary Artery Catheterization Treated with Endobronchial Intubation

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Since the introduction of the balloon-tipped pulmonary artery catheter in 1970, several cases of associated pulmonary artery rupture have been reported.1-5 The majority of these have been fatal. We report the management of two cases of pulmonary artery catheter related massive endobronchial hemorrhage treated non-operatively, in which early placement of a double-lumen endobronchial tube may have permitted survival.

REPORTS OF TWO CASES

Patient 1. A 90-year-old woman was admitted because of abdominal pain, weight loss, and dehydration. Her past medical history included sigmoid colectomy with colostomy three years earlier for a perforated carcinoma, myocardial infarction, congestive heart failure, and hypertension. Physical examination was consistent with intestinal obstruction and marked dehydration. She was admitted to the Respiratory-Surgical Intensive Care Unit (R-SICU), where nasogastric suction and intravenous hydration were begun. A 7-French pulmonary artery catheter was inserted via the left subclavian vein. On the second hospital day, signs and symptoms of intestinal obstruction disappeared. The pulmonary artery catheter and nasogastric tube were removed, and she was fed.

On day three of her hospitalization, pain and vomiting reappeared, and a laparotomy was scheduled. A jejunojejunostomy was performed to bypass an unresectable obstructing tumor mass. The patient was returned to the R-SICU, with the trachea intubated with a 7.5-mm National Catheter endotracheal tube and ventilation controlled. A second 7-French pulmonary artery catheter was inserted without difficulty via the left internal jugular vein. Its tip was seen by roentgenogram to be in the right main pulmonary artery. The pulmonary artery pressure was 35/16 torr. On the second postoperative day the patient was weaned from mechanical ventilation with acceptable pulmonary mechanics and arterial blood gases. Tracheal extubation was planned. The pulmonary artery catheter showed a normal pulmonary artery tracing. An attempt to obtain a pulmonary capillary wedge pressure was abandoned after inflation of the balloon with only 1/4 ml of air was met with resistance. Thirty seconds after inflation had been attempted, a large amount of bright red blood emerged from the endotracheal tube, and the arterial blood pressure fell from 140/70 to 70/

30 torr. With an FIO₂ of 1.0 and controlled ventilation, P ao₂ was 310 torr, P ACO₂ 53 torr, and pH 7.22.

Intravenous pulmonary artery rupture was diagnosed. The trachea was extubated and immediately reintubated with a National Catheter 35-French endobronchial tube, which was positioned with the endobronchial portion in the left main stem bronchus. Several hundred milliliters of blood were suctioned from the tracheal lumen. Aspiration of the bronchial lumen revealed only scant amounts of blood. The pulmonary artery catheter was withdrawn into the main pulmonary artery. The patient was given one unit of packed red blood cells, sedated with morphine, paralyzed with pancuronium, and controlled ventilation was re instituted through the double lumen tube, using both lumina. Lactated Ringer’s solution and dopamine were administered. After 30 min the bleeding remitted, and the vital signs stabilized. Her hematocrit, which had been 39 per cent, fell to 33 per cent (posttransfusion).

With V'T of 700 ml, an FIO₂ of 0.4, and a respiratory rate of 10/min, P ao₂ was 112 torr, P ACO₂ 37 torr, and pH 7.36. Chest roentgenogram showed clear lung fields and a left pleural effusion. Chest physical therapy was discontinued. Over the next 48 hours the patient improved, and there was no evidence of further hemorrhage through the tracheal lumen. The double lumen tube was removed, and an 8.0-mm Portex® endotracheal tube was substituted. Over the following 72 hours dopamine administration was gradually decreased and the trachea was extubated. She continued to have congestive heart failure which was managed with oxygen, furosemide, and morphine.

On postoperative day 14, she was transferred from R-SICU, and by postoperative day 18 she was well enough to be discharged from the hospital.

Patient 2. An 89-year-old man was admitted because of heart failure. His past medical history included renal insufficiency, permanent pacemaker implantation for sick sinus syndrome, and cystectomy with ileal loop for bladder carcinoma. Over the three days prior to admission he had been treated with furosemide, digoxin, aminophylline, and dopamine without improvement, and he was transferred to the Beth Israel Hospital.

Physical examination revealed an elderly male in respiratory distress. The blood pressure was 110/70 torr, heart rate 72/min (paced), and the respiratory rate 30/min. There were jugular venous distension and rales throughout both lung fields. Chest roentgenogram revealed pulmonary edema. With 100 per cent oxygen by mask, P ao₂ was 158 torr, P ACO₂ 31 torr, and pH 7.38. A 7-French pulmonary artery catheter was inserted percutaneously through the right internal jugular vein.

The catheter appeared to function well, and revealed a central venous pressure of 13 torr, pulmonary artery pressures of 45/15 torr, and a pulmonary artery wedge pressure of 20 torr. Immediately after passage of the catheter, as it was being secured to the neck, the patient began to have hemoptysis of approximately 250 ml of bright red blood. P ao₂ was 48 torr, P ACO₂ 52 torr, pH 7.20. The systolic blood pressure fell to 80 torr and the patient became agitated and then stuporous. The patient was given succinylcholine 20 mg, iv, cricoid pressure was applied, and an 8.0-mm National Catheter endotracheal tube was passed under direct vision. Another 100 ml of blood was suctioned through the tube. Ventilation was controlled and paralysis induced with pancuronium. Intra venous saline was given.

Under direct laryngoscopy the endotracheal tube was removed and...
a National Catheter 37-French endobronchial tube was passed into
the right main stem bronchus. Good differential ventilation was ob-
tained to auscultation, and proper position of the tube was confirmed
with a chest roentgenogram. Blood was aspirated from the endobron-
chial but not the tracheal lumen. At one point a large blood clot
completely obstructed the endobronchial lumen, and this was success-
fully dislodged with a suction catheter. Mechanical ventilation of both lungs
was continued with the double lumen tube, and with an FiO2 of 1.0,
Pao2 was 385 torr, Paco2 42 torr, and pH 7.22. The hematocrit, which
had been 33 per cent on admission, fell to 30 per cent. The pulmonary
artery catheter was withdrawn into the main pulmonary artery. Chest
roentgenogram showed pulmonary edema and a pulmonary artery cath-
ter coiled in the right main pulmonary artery. The patient had been
receiving chest physical therapy and this was discontinued. The hem-
optysis slowed significantly after 30 min and the vital signs stabilized.
The FiO2 was decreased to 0.4 within 12 hours after intubation of the
trachea. Pancuronium and morphine were given. Within 36 hours,
only small amounts of blood were suctioned through the bronchial
lumen. The endobronchial tube was replaced with an 8.5-mm National
Catheter single-lumen tube, and muscle relaxants were discontinued.
On day 6 the trachea was extubated with acceptable arterial blood
gases and an improved roentgenogram. He felt well and was not dys-
peptic. Oral feeding was begun, and the patient became ambulatory.
He was transferred from the intensive care unit much improved. He
expired suddenly from ventricular fibrillation on the fourteenth hos-
pital day.

DISCUSSION

The use of pulmonary artery catheters has become widespread and routine in the management of critically ill patients. The most serious complication associated with its use, usually fatal in outcome, is rupture of a pulmonary artery with consequent endobronchial hem-
orrhage. Although no definite information exists as to its incidence, in our own institution we know of two cases in the last 2,000 pulmonary artery catheter insertions.

Other reports of this complication have suggested that associated factors include: 1) pulmonary hypertension, 2) advanced age, 3) high pulmonary artery to wedge pressure gradient, 4) peripheral placement of the catheter (facilitated by a redundant loop of catheter in the heart), 5) prolonged balloon inflation, and 6) multiple catheter manipulations.

These patients usually present with hemoptysis, dyspnea, and rapid deterioration and will become hypoxemic secondary to hemorrhage into the ipsilateral alveolar spaces and overflow into the contralateral lung. Treat-
ment has consisted of turning the patient with the affected side down, fluid support, correction of any bleeding di-
athesis and rapid thoracotomy and pulmonary resection if conservative methods fail. It has been suggested that bronchoscopy may allow localization and tamponade of the bleeding site.

In our two cases an initial concern for adequate oxygenation and isolation of the affected lung suggested immediate placement of a double-lumen endobronchial tube. This allowed both a route for evacuation of blood and protection and ventilation for the uninvolved lung. While placement of an endobronchial tube is not without difficulty and complications, it is the only means to pro-
vide lung isolation, bilateral suction capability and bi-
lateral lung ventilation in the setting of acute endobronchial hemorrhage. Other methods of lung isolation such as the use of a single-lumen endobronchial tube or a bronchial blocker do not allow these capabilities. Other measures in the treatment of pulmonary hemorrhage caused by a pulmonary artery catheter should include: 1) withdrawal of the pulmonary arterial catheter to a proximal site, 2) sedation and paralysis to avoid increases in pulmonary arterial pressures associated with coughing and straining and to prevent dislodgement of any clot sealing the perforation, 3) discontinuation of deep endo-
bronchial suctioning and omission of chest percussion, and 4) mechanical ventilation.

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