Pulmonary Hemorrhage from Pulmonary Artery Catheterization Treated with Endobronchial Intubation

JONATHAN M. STEIN, M.D.,* AND ALAN LISBON, M.D.†

Since the introduction of the balloon-tipped pulmon- ary 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 nonoperatively, 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 coloectomy 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 jejunostomy was performed to bypass an unresolubling 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 mm Hg. 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 mm Hg. With an FIO2 of 1.0 and controlled ventilation, Pao2 was 310 mm Hg, Paco2, 53 mm Hg, 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 hematuria, which had been 39 per cent, fell to 33 per cent (postransfusion). With Vt of 700 ml, an FIO2 of 0.40, and a respiratory rate of 10/min, Pao2 was 112 mm Hg, Paco2, 37 mm Hg, 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, amrinonil, 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 mm Hg, heart rate 72/min (paced), and the respiratory rate 30/min. There were jugular venous distention and rales throughout both lung fields. Chest roentgenogram revealed pulmonary edema. With 100 per cent oxygen by mask, Paco2 was 55 mm Hg, Paco2, 31 mm Hg, and pH 7.13. 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 mm Hg, pulmonary artery pressures of 45/15 mm Hg, and a pulmonary arterial wedge pressure of 20 mm Hg. 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. Pao2 was 48 mm Hg, Paco2, 52 mm Hg, pH 7.20. The systolic blood pressure fell to 80 mm Hg 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. Intravenous 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 obtained to auscultation, and proper position of the tube was confirmed with a chest roentgenogram. Blood was aspirated from the endobronchial but not the tracheal lumen. At one point a large blood clot completely obstructed the endobronchial lumen, and this was successfully 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, Pao₂ 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 catheter coiled in the right main pulmonary artery. The patient had been receiving chest physical therapy and this was discontinued. The hemoptysis 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 dyspneic. Oral feeding was begun, and he became ambulatory. He was transferred from the intensive care unit much improved. He expired suddenly from ventricular fibrillation on the fourteenth hospital 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 hemorrhage. 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. Treatment has consisted of turning the patient with the affected side down, fluid support, correction of any bleeding diathesis 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 provide lung isolation, bilateral suction capability and bilateral 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 endobronchial suctioning and omission of chest percussion, and 4) mechanical ventilation.

REFERENCES