

Acute Pulmonary Hypertension After Wedging of a Pulmonary Artery Catheter as Clues to Pulmonary Artery Perforation

To the Editor:

Although the incidence of pulmonary artery (PA) perforation with pulmonary artery catheter (PAC) is low, 0.06 to 2%,^{1,2} the complication is fatal in 45% to 53% of cases.^{3,4} The severity of PA rupture ranges from minor endobronchial hemorrhage to massive hemoptysis, from contained parenchymal hemorrhage to free bleeding into the pleural space.³ In 90% of cases, hemoptysis is a major presentation of PA perforation.⁵ We would like to share our experience of a case of PA perforation with a PAC in which the acute increase of PA pressures after wedging might be a clue to the diagnosis of perforation.

A 76-year-old, 60-kg man underwent an urgent triple coronary artery bypass grafting and placement of a Tenckhoff peritoneal dialysis catheter. Prior to induction of anesthesia, a right internal jugular VIP PAC (Edwards Swan-Ganz 93A-831-7.5 F, Baxter Health Corporation, Irvine, CA) was placed atraumatically with a normal appearing PA tracing. Pulmonary artery systolic/diastolic pressure (PAs/d) was 29/19 mmHg, central venous pressure (CVP) 13 mmHg, cardiac output (CO) 3.3 L/min, and systolic blood pressure (BP) 150/70 mmHg. The PAC tip was never in the wedge position and was never wedged throughout the entire operative period.

The operation proceeded uneventfully and the patient was subsequently transferred to the surgical intensive care unit (SICU) in stable condition. BP was 121/64 mmHg, CVP 6 mmHg, PAs/d 21/10 mmHg, and CO 3.3 and 3.6 L/min. His immediate postoperative AP portable chest x-ray (CXR) showed postoperative changes consistent with coronary artery surgery; mediastinal and left chest tubes were in proper position; the PAC tip appeared to be in good position in the proximal right pulmonary artery. Shortly after admission to the SICU, the PAC was inflated for wedging and an occlusion pressure of 7 mmHg was obtained. Minutes later, the patient became hypotensive and hemodynamically unstable. The PAP and CVP were recorded as acutely increased (Table 1). The patient's hematocrit dropped from 33% to 16%, with arterial blood gases showing a persistent metabolic acidosis: F_iO_2 40%, pH 7.24 to 7.13, PCO_2 43 to 45 mmHg, HCO_3^- 18 to 16 mEq/L, PO_2 86 to 95 mmHg, SaO_2 99%. PT/PTT at this time were within normal limits (14.5/29). Drainage from all chest tubes was minimal. Although a PA perforation was initially considered, it was dismissed because the first SICU portable CXR obtained only 20 minutes earlier was normal, and the absence of mediastinal chest tube drainage or hemoptysis. Upon aspiration, bloody drainage was noted from his Tenckoff catheter. It was believed that the source of bleeding might be intraabdominal and the patient was brought back emergently to the operating room for an exploratory laparotomy. Intraabdominal exploration was negative, except for a caudad bulging of the right hemidiaphragm. A chest tube was placed in the right thorax and about 2 L of gross blood was obtained. Intraoperative CXR confirmed a right hemothorax. Reexploration of the mediastinum revealed no bleeding. Upon opening the right pleura, a free linear perforation of an anomalous superficial major branch of the right PA was found. Following surgical correction and evacuation of the right hemothorax, PAP decreased to the preruptured values. The patient was once again transferred back to the SICU in stable condition. Subsequent blood gases showed correction of the acidosis, with pH 7.37, PCO_2 38.7, PO_2 141, HCO_3^- 22, SaO_2 99%, and hematocrit stabilizing at 29% to 30%. The patient recuperated uneventfully and was discharged 10 days later.

The patient presented a picture of hemodynamic instability of unclear etiology. The small amount of mediastinal and left chest tube drainage and bloody drainage from the Tenckoff catheter lead to the initial misdiagnosis of the source of

Table 1. Hemodynamic Changes After Inflation of the Pulmonary Artery Catheter Balloon for Wedging

| SICU | Time/Hour | | | | | To OR/ Back to SICU | | | |
|--------|------------|------------------|------------|----------------|--------------------------|---------------------------|------------|------------|------------|
| | 0 | 1 | 2 | 3 | 4 | | 0 | 1 | 2 |
| BP | 121/64 | 170/60 116/76 | 78/50 | 139/66 | 155/92 105/62 85/— | | 130/80 | 109/71 | 120/73 |
| MAP | 81 | 81 | 60 | 89 | 78 | | 92 | 84 | 90 |
| CVP | 6 | 6 | 11 | 16 | 14 | | 12 | 16 | 20 |
| PA S/D | 21/10 | 26/14 | 29/18 | 46/24 58/28 | 40/23 | | 24/13 | 21/13 | 24/15 |
| PAm | 16 | 16 | 23 | 31 37 | 29 | | 17 | 15 | 18 |
| PAWP | | 7 | | | | | | | |
| CO | 3.3 3.6 | 3.3 3.1 | 3.4 3.2 | 3.4 3.1 | 3.5 3.4 | | 3.6 2.9 | 2.9 3.0 | 2.6 3.6 |

Abbreviations: BP, blood pressure (mmHg); MAP, mean arterial pressure (mmHg); CVP, central venous pressure (mmHg); PA S/D, pulmonary artery pressure systolic/diastolic (mmHg); PAm, pulmonary artery mean pressure (mmHg); PAWP, pulmonary artery wedge pressure (mmHg); CO, cardiac output (L/min).

bleeding. The acute PAP increases might represent the "lung tamponade syndrome" secondary to compressive bleeding from the PA perforation.

The diagnosis of PA perforation can be made only by a high degree of suspicion whenever the patient with a PAC develops hemoptysis or unexplained cardiac or respiratory changes. The list of suspicions should include any acute PAP elevations after wedging of the PAC.

Thieu T. Duong, MD, MPH

*Gabriel S. Aldea, MD**

Gilbert P. Connelly, MD

Benjamin S. Suaco, MD

Lawrence C. Weinfeld, MD

Anna L. Kurian, MD

Department of Anesthesiology

*Department of Cardiothoracic Surgery

Boston University School of Medicine

Boston, MA

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Autologous Blood Deposit and Aprotinin in Elective Cardiac Surgery

To the Editor:

Patients who undergo heart surgery and cardiopulmonary bypass are at risk of serious postoperative bleeding.¹ They often require blood therapy though reoperation for bleeding is necessary for only 7% of patients. In spite of the increasing application of intraoperative hemodilution² and transfusion of mediastinal blood,³ blood from the blood bank is still widely used.

Since 1986, we have been using preoperative autologous blood donation,⁴ and since 1989, we have also been using high-dose aprotinin in an attempt to reduce homologous blood requirements.^{5,6} To evaluate whether the association of aprotinin and autologous blood donation could reduce blood loss and blood bank requirements more than autologous blood donation or aprotinin alone, we performed the following study during CABG.

Patients were divided into three Groups of 50 patients each, using a randomized assignment in a blinded way. Group A was composed of autologous blood donors: every 3 days, 350 mL of blood was withdrawn from each patient for a total blood withdrawal of 1,050 mL. The blood was centrifuged immediately in order to divide red cells from plasma. Prior to each donation the hematocrit (HCT) level was measured and blood was not drawn if it was less than 35%. The patients underwent operation approximately 11 days after the last withdrawal. Patients in Group B also had autologous donation, but they also received aprotinin during the operation. Patients in Group C were treated with the same doses of aprotinin as Group B, but without autologous donation. The HCT value was evaluated until the patient was discharged. Homologous banked blood was used only when the HCT was lower than 28%.

Table 1. Mediastinal Drainage and Homologous Transfusion Requirements

| | Group A (N = 50) | Group B (N = 50) | Group C (N = 50) |
|---------------------------|---------------------|---------------------|---------------------|
| Mediastinal drainage (mL) | 909 ± 252 | 476 ± 244* | 527 ± 238* |
| Homologous units | 1.81 ± 2.65 | 0.26 ± 0.92* | 1.22 ± 1.66† |

Note. Data are given as mean ± SD.

* $P < 0.0001$.

† $P < 0.02$.