Massive Thrombosis Associated with Use of the Swan-Ganz Catheter*

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A patient with severe respiratory failure due to overwhelming pneumonia was monitored with a Swan-Ganz catheter. Postmortem examination revealed massive ante-mortem thrombosis surrounding the Swan-Ganz catheter tip. The presence of the thrombus about the catheter tip was not suspected before death, but retrospective analysis of a variety of parameters suggested massive pulmonary vascular obstruction.

Recently the use of a flow-directed balloon-tipped catheter has been introduced to safely measure and record right atrial, right ventricular, pulmonary arterial, and pulmonary capillary wedge pressures in patients with acute myocardial infarction. At our institution, use of this catheter has been extended to the differential diagnosis and management of other cardiopulmonary disorders, including certain acute respiratory distress syndromes in the adult, such as those recently reviewed by Petty and Ashbaugh. Infrequent complications have been reported with the use of this catheter, including rare instances of thrombosis about the catheter tip.

The purpose of this report is to describe a patient who presented with an acute respiratory distress syndrome and in whom placement of a flow-directed balloon-tipped catheter in the pulmonary artery was associated with the acute development of massive thrombosis about the catheter tip, an event that was probably contributory to the patient’s death. Certain clinical and physiologic features suggestive of massive pulmonary vascular occlusion, which may be valuable in the diagnosis of this serious complication were noted retrospectively.

CASE REPORT

A 63-year-old man was admitted to the hospital because of the onset of acute progressive dyspnea over the previous five days. Four years previously, peptic ulcer disease had been demonstrated. For several months prior to his hospitalization the patient had taken up to 18 tablets of a mixture of oxydoxane terephthalate, aspirin, phenacetin and caffeine (Percodan) a day for discogenic back pain and had used mineral oil nightly for several years prior to his hospitalization to relieve constipation. The onset of dyspnea was associated with a dry cough without fever, chills, upper respiratory symptoms, sputum production, chest pain, or gastrointestinal complaints. The patient related no history of aspiration or prior cardiac or pulmonary disease.

Physical examination revealed an acutely ill appearing man in severe respiratory distress. His temperature was 38.8°C, blood pressure 200/100 mm Hg in the supine position, pulse rate 100 per minute, and respiratory rate 30 per minute. There was dullness at the right posterior base and basal inspiratory râles bilaterally. Chest expansion was symmetrical. Supraventricular retractionas as well as tracheal descent were present with each inspiration. No neck vein distention, gallop rhythm, heart murmurs, abdominal tenderness, organomegaly, or ankle edema was present.

The hematocrit level was 33 percent and the white blood cell count was 7,500/mm³, with a normal differential. Urinalysis, serum electrolytes, and creatinine values were normal. No blood was present in the stool. Gram stain of the sputum revealed moderate numbers of polymorphonuclear cells, but only scant Gram-positive and Gram-negative diplococci. Arterial blood gas measurement done while the patient was breathing room air showed a pH of 7.52, arterial carbon dioxide pressure (PaCO₂) of 30.5 mm Hg, and an arterial oxygen pressure (PaO₂) of 42 mm Hg. The electrocardiogram was unremarkable except for a QRS axis of −30°. Studies performed later in this patient’s course included a normal prothrombin time, partial thromboplastin time, fibrinogen level and platelet count. The chest x-ray film on admission revealed extensive bilateral diffuse parenchymal infiltrates extending from the hilum to the peripheral lung fields. A chest x-ray film made on the second hospital day revealed further extension of the infiltrates, with nearly complete opacification of both lung fields.

At the time of admission, the diagnostic considerations included extensive aspiration pneumonia or a diffuse viral pneumonitis. The patient was initially treated with supplemental oxygen therapy by face mask and begun on intravenous penicillin. Because of continuing hypoxemia, which could not be adequately corrected with low concentrations of oxygen, ie, less than 50 percent inspired oxygen concentration (FIO₂), as well as progressive respiratory distress, nasotracheal intubation was performed and intermittent positive pressure breathing was instituted with a volume-cycled respirator (Bennett, MA-1). Hypoxemia worsened despite a FIO₂ of 100 percent oxygen and about 24 hours after the patient was admitted, continuous positive pressure breathing (CPPB) with an FIO₂ of 100 percent was instituted, with an end-expiratory pressure of 12 cm H₂O. It was necessary to sedate the patient heavily with barbiturates to prevent him from fighting against the respirator. His minute ventilation was then maintained with a fixed rate and tidal volume. He tolerated this without any systemic hypotension. The PaO₂ rose from 45 mm Hg to 65-70 mm Hg within 15 to 30 minutes after the institution of CPPB. To aid in the assessment of fluid administration as well as to determine whether

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any element of left ventricular failure was present, a flow-directed balloon-tipped catheter was inserted via the right basilic vein approximately 30 hours after admission. Right ventricular, pulmonary artery, and pulmonary capillary wedge pressures were measured (Fig 1). Pulmonary artery pressure was 36/15 mm Hg and the pulmonary capillary wedge pressure was 13 mm Hg. The catheter with the balloon deflated was left in the pulmonary artery. However, within two to three hours after placement, frequent flushing of the catheter was required to prevent damping of the pressure tracing (Fig 1). For the next four to five hours, it became progressively more difficult to obtain pulmonary artery and wedge pressure recordings. Over the next eight hours arterial blood pressure fell from 200/100 to 140/90 mm Hg, urine output decreased from 40 ml/hr to about 5 to 15 ml/hr. Despite the intravenous administration of colloids and diuretics, a moderate metabolic acidosis developed. An electrocardiogram was again performed shortly before the patient's death and demonstrated a shift in the QRS axis to +90°. However, oxygenation remained adequate, with the PaO₂ remaining in the 60 mm Hg range, but the PaCO₂ increased from approximately 30 mm Hg to 46 to 48 mm Hg over the five hours prior to death, in spite of the fact that minute ventilation was maintained at a constant level on the respirator. Changes in hemodynamics, urine output, and lung function are shown in Figure 2. Forty hours after admission and 12 hours after the placement of the catheter, cardiac arrest occurred associated with ventricular asystole. During resuscitative attempts the balloon-tipped catheter was withdrawn about 10 cm, with the idea that the catheter may have been obstructing pulmonary blood flow.

Pathology

Gross findings and subsequent microscopic examination revealed that the patient's respiratory disease was a massive lipid pneumonitis involving virtually all lobes. No pathogens were isolated from the respiratory tract. A large thrombus, measuring 5.5 by 1.4 cm was found encasing the distal end of the balloon-tipped catheter (Fig 3). The thrombus was obstructing the superior vena cava, and its tip was found in the right atrium. The thrombus was of antemortem character, being rough, stranded, and fibrinous. In addition, on cross section characteristic lines of Zahn were seen. Microscopic examination revealed alternating bands of fibrin and necrotizing red blood cell elements arranged concentrically about the catheter. The findings were compatible with recent thrombosis. Although not apparent clinically, evidence of a disseminated intravascular coagulopathy was found microscopically. Fibrin thrombi were found in capillaries and in small arterioles in the kidneys, adrenals, spleen, liver, heart, lungs, pancreas and testes. These thrombi were PAH-positive, indicating their fibrinous composition. No hemorrhages were seen grossly. However, microscopic focal hemorrhages were seen in the adrenals in association with the thrombi.

Discussion

This patient presented with the acute onset of respiratory failure, which according to pathologic examination, was due to massive exogenous aspiration of lipid material. Because of the initial presentation and the possibility of left ventricular failure, a flow-directed balloon-
tipped catheter was placed in the pulmonary artery for pressure monitoring as a guide to fluid therapy. Post-mortem examination revealed a large antemortem thrombus surrounding the catheter tip (Fig 3). The narrowed portion in the middle of the thrombus suggested that this area was compressed during life, perhaps by the pulmonary conus. If this were true and considering that the catheter was withdrawn 8-10 cm just prior to death, the distal tip of the thrombus would have protruded through the pulmonic valve into the main pulmonary artery during life. Since the main pulmonary artery normally has a diameter in vivo of about 20-30 mm, and since the thrombus diameter at its distal end was about 14 mm, the thrombus must have obstructed at least 50 percent of the main pulmonary artery.

Despite the failure to recognize thrombus formation prior to death, in retrospect there were several observations that should have suggested obstruction of the pulmonary vasculature superimposed on extensive pulmonary parenchymal disease. (1) The pressure tracings early after placement indicated that only intermittent patency of the catheter was present after flushing with saline solution (Fig 1). (2) The falling systemic blood pressure and urine output, as well as the development of a metabolic acidosis (probably secondary to lactic acidosis, although not confirmed) were probably related to a decrease in cardiac output. (3) The development of hypoxemia while minute ventilation was maintained constant on the respirator, was an indication either of an increase in deadspace ventilation, which is characteristic of pulmonary artery occlusion, or an increase in CO₂ production. Since the metabolic state of the patient was stable, the latter explanation is unlikely. Although hyperventilation frequently occurs following pulmonary vascular occlusion, it could not in this patient because minute ventilation was fixed by the respirator. (4) The widening of the inspired-arterial oxygen difference is also consistent with pulmonary vascular obstruction. (5) The shift in the electrical axis of the ECG from −30 to +90° was further evidence consistent with massive pulmonary vascular obstruction with right ventricular strain.

Although results of premortem coagulation screening tests were normal, disseminated intravascular coagulation was present on pathologic examination. This may have been an important factor in the genesis of the rapid thrombus formation occurring about the catheter tip, although it is not clear whether the disseminated intravascular coagulation state initiated the formation of the thrombus, or whether the thrombus, by decreasing cardiac output with resultant tissue hypoxia, was responsible for the disseminated intravascular coagulation state.

The occurrence of massive and rapid thrombus formation about this type of catheter is most unusual and has not previously been reported. Of 70 patients with acute myocardial infarction in whom the catheter was used by Swan et al., only two had significant thrombus formation about the catheter tip. Both were associated with catheter placement for longer than 48 hours. The thrombus formation in our case with resultant pulmonary vascular obstruction was an important, if not the primary, cause of the patient's death. This complication is rare, but because of its potential life threatening effects, the catheter should probably be removed when consistently damped pressure tracings are observed in association with hemodynamic or pulmonary function abnormalities that would be consistent with pulmonary vascular occlusion, i.e, acute cor pulmonale, shock, increased deadspace ventilation (causing hypoxemia using volume ventilators), and increasing hypoxemia with a fixed inspired oxygen concentration.

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Sarcoidosis: The Significance of an Acinar Pattern on Chest Roentgenogram

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A young black woman presented with dyspnea on exertion and a productive cough. The chest roentgenogram showed predominance of an acinar rosette pattern without hilar adenopathy. Lung biopsy showed multiple non-caseating granulomata in the interstitium, compatible with the diagnosis of sarcoidosis, with alveoli filled with mononuclear cells. The term alveolar sarcoidosis is a radiologic diagnosis based on certain proposed criteria for acinar filling. Supported by pathologic correlation, it appears that the acinar radiographic pattern may represent a secondary nonspecific response of the lung to the primary interstitial injury.

The common roentgenographic features of pulmonary sarcoidosis have been well described. Lymph node enlargement, disseminated miliary densities, localized infiltrates and fibrotic changes, alone or in combination, are the more typical findings. Although it is a granulomatous disease of the interstitium, there is a form of sarcoidosis that produces an acinar roentgenographic pattern. However, histopathologic correlation of this form of sarcoidosis with roentgenography has not been reported. We have recently observed a young black

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