CLINICAL VIGNETTE

Iatrogenic Pulmonary Artery Rupture due to Pulmonary Artery Catheter Inflation

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Case Report

An 81-year-old woman with type 2 diabetes, hypertension, and hyperlipidemia presented with one day of progressive shortness of breath. She denied any chest pain, fevers, cough, or orthopnea. She reported chronic stable bilateral lower extremity edema but denied any history of heart failure or prior myocardial infarction.

Initial vital signs demonstrated no fever but were remarkable for a heart rate of 169 beats per minute, blood pressure of 203/126 mmHg, respiratory rate of 38, and SpO2 of 82% on room air. She was placed on continuous positive pressure ventilation and given sublingual nitroglycerin and brought to the emergency room (ER).

In the ER, the patient was in moderate respiratory distress with improved oxygen saturation on positive pressure ventilation, improved blood pressure control, persistent tachycardia, elevated jugular venous pressure, bibasilar rales, and bilateral lower extremity edema.

EKG showed sinus tachycardia without acute ST-T changes. Chest x-ray demonstrated multifocal patchy bilateral hazy opacities consistent with vascular congestion versus multifocal pneumonia. Bedside echocardiogram revealed depressed ejection fraction of 20%, severe mitral regurgitation, and moderately elevated pulmonary pressures. The patient's labs showed leukocytosis with WBC of 16.7, elevated lactate of 74 mg/dL, acute kidney injury with Cr 1.6 mg/dL, elevated troponin of 2.2 ng/mL, and elevated BNP of 705.

The patient was started on antibiotics for possible community acquired pneumonia, heparin drip for NSTEMI, and intravenous furosemide. Her symptoms improved in the ER, and she was transferred to the intensive care unit (ICU).

Soon after, the patient developed respiratory distress and was intubated. This was complicated by bradycardic PEA arrest. The patient was resuscitated and required maximal ventilator support and vasopressors. Repeat EKG demonstrated new inferior ST depressions and troponin increased to 4.5ng/mL. However given the patient's unclear mental status after recent cardiac arrest, coronary angiography was declined by the family.

Over the next 24-48 hours, the patient's mental status and respiratory failure improved enough to permit extubation. However, the patient continued to have hypotension and require vasopressor support, supplemental oxygen, and developed oliguric renal failure. Diuresis of the patient was limited by hypotension. As such, right heart catheterization was performed to evaluate hemodynamics and to help guide weaning of vasopressors and optimization of volume status.

The patient underwent pulmonary artery catheter (PAC) placement through the right internal jugular vein under ultrasound and fluoroscopic guidance in the cardiac catheterization lab. Her right atrial pressure was 7 mmHg, main pulmonary artery pressure was 32/18 mmHg, and pulmonary artery wedge pressure was 18mmHg, with a cardiac index of 1.7 L/min/m^2 – these values were consistent with moderately elevated right- and left-sided filling pressures and severely depressed cardiac output. The patient was started on inotropic support with milrinone and transferred to the ICU with the PAC in place.

Chest x-ray demonstrated that the tip of the catheter was located in the right lower lobe pulmonary artery, and it was then subsequently retracted (Figure 1). The patient's cardiac output and wedge pressures were then recorded. A few minutes later, the patient developed massive hemoptysis. She rapidly became hemodynamically unstable and died from presumptive pulmonary artery rupture. The family declined autopsy.

Discussion

Since the first right heart catheterization performed in 1929 by Dr. Warner Forssmann who introduced a catheter into his own heart, right heart catheterization catheters and techniques have advanced significantly allowing catheters to be placed at the bedside within a few minutes, even in the most critically ill patients. Given the ease of placement, the use of PACs increased rapidly; in the 1980s, up to 40% of critically ill patients had received a PAC.[1]

Several studies have looked at the utility of PACs in the management of patients. The first significant trial was a nonrandomized propensity match cohort trial in 1996 by Connors et al,[2] which demonstrated a 24% increased risk of death in ICU patients who received a PAC within 24 hours of admission to an ICU. This was followed by a large prospective randomized controlled trial by Sandham et al,[3] which looked at 1,994 high-risk patients over the age of 60 who were scheduled to undergo high-risk surgery. Individuals were randomized to standard care or use of PAC preoperatively for goal directed therapy. Hospital and 6-month mortality were

similar in the two groups; however, significantly more pulmonary emboli occurred in the PAC group.[3] The role of the PAC in heart failure was studied in the ESCAPE trial. 433 patients were randomly assigned to receive therapy guided by clinical assessment, and a PAC versus clinical assessment alone. The trial did not show any difference in overall mortality or need for recurrent hospitalization between the two groups. However, hospital adverse events increased in the PAC group. These specific events were PAC-related infection, bleeding, catheter knotting, pulmonary infarction/hemorrhage, and ventricular tachycardia. [4] In the early 2000s, multiple other trials corroborated results previously discussed, which led to a meta-analysis by the Cochrane group that demonstrated no benefit for the use of PAC in high-risk surgery patients or ICU patients.[5]

As a result, most guidelines no longer supported the routine use of PACs; there has been a significant decline in PAC use in the United States.[6,7] Despite the lack of mortality benefit and potentials harms, the PAC does provide information that is not always readily available by clinical examination or by non-invasive testing. Based on expert consensus, PACs are indicated for patients with shock with unknown volume status, suspected or known pulmonary hypertension, severe underlying cardiopulmonary disease (i.e., severe valvular disease, pulmonary hypertension, complex congenital heart disease, significant intra-cardiac left to right shunting) undergoing high-risk surgery, and lastly as in our case, severe cardiogenic shock to help guide therapy.[8]

The absolute contraindications to placement of a PAC include: infection at insertion site, presence of right ventricular assist device, mechanical pulmonic or tricuspid valves, right-sided endocarditis, right ventricular thrombus, and lack of consent. Relative contraindications include coagulopathy (INR>1.5), thrombocytopenia (platelet count <50K), significant electrolyte or acid/base disturbances, and ventricular arrhythmias. These should ideally be corrected before placement of the catheter. Other high-risk patients for PAC placement include individuals with preexisting left bundle branch block (risk of complete heart block), individuals with pacemakers/defibrillators (risk of dislodgment of leads), bio-prosthetic valves, those with severe pulmonary hypertension, or Eisenmenger's syndrome (risk of pulmonary artery rupture). Individuals with these high-risk features would benefit from placement of pulmonary artery catheters under fluoroscopic guidance.

There are numerous potential complications associated with PAC placement and can be divided into three categories: 1) complications at the time of insertion; 2) complications related to interpretation of hemodynamics; and 3) complications related to maintenance and use of the catheters (Table I). [9,10,11]

The most feared complication of PAC placement is pulmonary artery perforation. Incidence of rupture is rare with an observed rupture rate of 0.03% of catheter insertions. Risk factors for pulmonary artery perforation include: older individuals, severe pulmonary hypertension, mitral valve disease, hypothermia, and anticoagulation.[12] Consequences, as in our case, are devastating with estimated mortality that ranges from 30-70 percent.[13]

Episodes can occur at time of insertion, but typically occur when the PAC has inadvertently migrated distally into the pulmonary artery due to either direct perforation of the catheter tip or to rupture from over-inflation of the balloon. It typically presents with brisk hemoptysis. Recognition is vital. Treatment may require immediate catheter removal, emergent dual bronchus intubation or selective intubation of the unaffected lung, patient positioning such that affected side is down, aggressive resuscitation, and/or emergent thoracotomy.

In cases where the pulmonary artery diastolic pressure approximates the pulmonary capillary wedge pressure, it may be advisable to use this as a surrogate for the left ventricular filling pressure, rather than repeatedly inflate the balloon.

Conclusions

Routine use of pulmonary artery catheters in the management of heart failure has been shown to not improve mortality or repeat hospitalizations. Furthermore, pulmonary artery catheter use is associated with rare but potentially fatal complications. In select patients, the pulmonary artery catheter may provide useful information. Physicians need to be aware of the potential complications of PACs when assessing the risks and benefits of their use.

Tables and Figures

Table I: Complications associated with PACs

Time of Insertion
Arterial Puncture
Sustained ventricular arrhythmia in up to 3% of patients
Right bundle branch block in up to 5%
Complete heart block (in patients with pre-existing left bundle branch block)
Catheter misplacement or knotting
Valve damage
Myocardial rupture
Pulmonary artery rupture
Interpretation of hemodynamics
Improper calibration
Respiratory Variability
Dampening of tracings
Maintenance/Use of Catheters
Infections (endocarditis)
Pulmonary emboli
Pulmonary infarction
Pulmonary artery perforation

Figure 1: Panel A demonstrates incorrect positioning of PAC tip in right segmental artery. Panel B demonstrates appropriate positioning of PAC in right pulmonary artery.
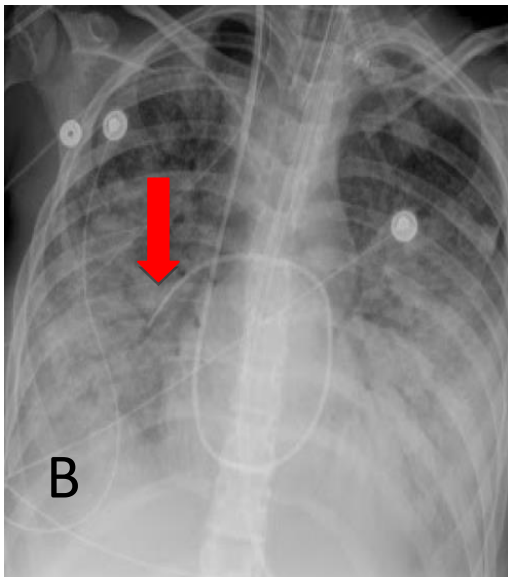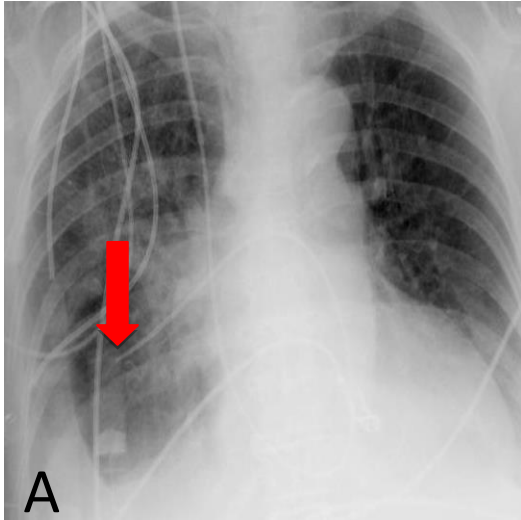
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