

Shortness of Breath: Pulmonary Embolism, Ischemic Heart Failure, or Both? The Role of Concomitant Surgery

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ABSTRACT

We present a case of a patient who underwent successful concomitant surgical management of his massive pulmonary embolism and severe multivessel coronary artery disease. His presentation with shortness of breath prompted a comprehensive evaluation, which revealed both problems. This experience emphasizes the importance of considering both problems, because treating one but not the other could be catastrophic.

INTRODUCTION

A cornerstone in the evaluation of patients is developing a differential diagnosis to guide therapies. Although a goal is to determine a single diagnosis, a common pitfall is failing to consider that patients can present with multiple problems, each explained by common symptoms. We discuss a patient who presented with shortness of breath and was found to have a massive pulmonary embolism (PE); however, further investigation revealed severe coronary artery disease (CAD), with both requiring surgery, simultaneously. Our experience emphasizes the importance of considering both potentially severe problems and that surgery can be successfully used to treat both.

CASE REPORT

Our patient is a 59-year-old man with a known history of hypertension, poorly controlled insulin-dependent diabetes, and peripheral vascular disease. He presented to the emergency room complaining of weakness after a near syncopal episode earlier in the day. He denied smoking, alcohol, or substance abuse. Upon arrival, the patient's pressure was 177/88 mm Hg, his heart rate was 103 beats/min, and his blood oxygen saturation was 92% on room air. Initial laboratory studies were significant for a d-dimer concentration of

2.96 $\mu\text{g/mL}$ and a troponin concentration of 0.5 ng/mL. His electrocardiogram showed only sinus tachycardia. A ventilation/perfusion scan showed multiple moderate to large mismatched perfusion defects involving the right upper lobe and bilateral lower lobes. A heparin drip was started. A transthoracic echocardiogram showed severe left and right ventricular dysfunction (left ventricular ejection fraction, approximately 20%-25%) and moderate pulmonary hypertension (right ventricular systolic pressure approximately 60 mm Hg). An initial strategy of medical management was chosen. In light of his multiple comorbidities, impaired ventricular function, and elevated cardiac biomarkers, a cardiac catheterization examination was performed. Coronary angiography revealed an 85% left main stenosis, diffuse left anterior descending coronary artery disease, an 85% obtuse marginal obstruction, and diffuse high-grade disease in his right coronary artery. A computed tomography pulmonary angiogram showed extensive PE in the distal left main pulmonary artery (PA) extending into the distal segments and showed smaller filling defects in the right main and distal right PA segments (Figure 1).

On the basis of these findings, the patient was taken to the operating room for combined coronary artery bypass grafting (CABG) and pulmonary embolectomy. Conventional

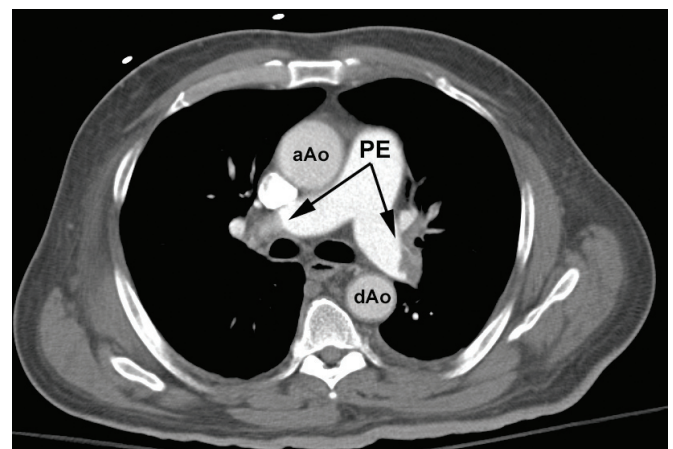


Figure 1. Computed tomography scan of the chest demonstrating complete occlusion of both the left and right main pulmonary arteries. aAo indicates ascending aorta; dAo, descending aorta; PE, pulmonary embolism.

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Figure 2. Clots removed from the left pulmonary artery. Similar clots were removed from the right side. Segments that appear to be castings of the pulmonary artery branches suggest subacute emboli.

cardiopulmonary bypass (aorta–right atrial cannulation) was used with antegrade and retrograde cardioplegia, and coronary revascularization was performed (left internal mammary artery to the left anterior descending artery, free right internal mammary artery to an obtuse marginal artery, and a vein graft to a ramus intermedius). His right coronary artery was too small and diffusely diseased to benefit from grafting. In addition, the main PA was opened longitudinally, and an extensive amount of clot was evacuated from the left PA (Figure 2). An incision was made in the right main PA between the aorta and the superior vena cava, and the clots were extracted from the right side. The subsegmental branch arteries appeared patent with no evidence of chronic thromboembolic disease. The patient was weaned from cardiopulmonary bypass with minimal inotropic support and with empiric inhaled epoprostenol (20,000 ng/min). Within 48 hours, his inotropic medications were discontinued, and he was weaned from his epoprostenol treatment and extubated. Although the patient's renal failure (creatinine at admission, 3.38 mg/dL) progressed to requiring renal replacement therapy, the remainder of his postoperative

course was unremarkable. A postoperative hypercoagulability workup was unremarkable because he had been bridged from a heparin drip to oral warfarin. The patient was discharged to a skill-rehabilitation facility on postoperative day 14.

By 2 months after discharge, the patient was home and symptom free. A repeat echocardiography evaluation showed an improved left ventricular function (ejection fraction, approximately 40%–45%) and improved estimated pulmonary pressures (right ventricular systolic pressure, 30–35 mm Hg). Within 4 months, the patient no longer required dialysis, but he will remain on warfarin indefinitely.

DISCUSSION

Patients often present with signs and symptoms suggestive of multiple medical problems. Known chronic medical problems may assist in directing the initial evaluation and management, but previously undiagnosed problems must be considered. Our patient's comorbidities predisposed him to the development of severe CAD. Considering the extent of his poorly controlled diabetes (hemoglobin A_{1c}, 12.4%), his lack of chest pain was not surprising. The patient's primary problem was most likely his acute PE; however, just as plausible was that his shortness of breath was a manifestation of an ischemic cardiomyopathy and his PE was an incidental confounding problem. Nevertheless, the patient had indications for urgent surgical management of both his PE and CAD. Our patient's presentation also supports the argument that patients with PE, ventricular dysfunction, and risk factors for CAD—particularly with elevated biomarker values—should undergo cardiac catheterization.

The management of acute PE is variable; however, surgical management can be performed with minimal morbidity and mortality [Kadner 2008], even in high-risk or unusual cases [Firstenberg 2010]. Indications for surgery typically include hemodynamic instability with right ventricular dysfunction and a large thrombus burden [Moseley 2010]. Surgery is also advocated in those with large saddle emboli who are failing medical therapy or have contraindications to thrombolytic therapy. Catheter-directed therapy has been advocated [Kuo 2009] and can be successful, but there are currently no devices or drugs approved by US Food and Drug Administration for the treatment of PE—nor are there standardized protocols [Torbicki 2008]. Furthermore, elevated cardiac biomarker results are strongly associated with poor outcomes in patients with PE and suggest a significant role of CAD and myocardial ischemia [Becattini 2007].

Management decisions for CAD are complex. Nevertheless, surgery is indicated in patients with symptomatic left main disease [Eagle 2004]. Risk factor assessment can guide decision making [Biancari 2006]. However, with unusual situations, management needs to be individualized. For our patient, the decision to pursue a combined procedure was made because he had independent indications for each problem, and, in our opinion, a combined approach was deemed appropriate, safe, and the best therapeutic approach.

Although PE following cardiac surgery has been well described, rarely has it been reported as part of the initial

preoperative evaluation [Josa 1993; Nyawo 2007], although an acute massive PE during an elective CABG procedure has been reported [Simek 2005]. Because the coronary anatomy is not known at the time of the initial presentation of a PE, concomitant CABG is rarely considered or performed in patients who undergo an emergent surgical pulmonary embolectomy. Although, 10% of patients undergoing surgery for chronic pulmonary thromboembolic disease require concomitant coronary revascularization, these patients have typically undergone an extensive preoperative evaluation [Thistlethwaite 2008].

An important point in the management of our patient was the diagnostic workup. Many clinical problems present with shortness of breath. Considering his initial presentation, the logical diagnostic approach demonstrated his PE. Further testing considering his presentation and comorbidities revealed his critical coronary disease. This case emphasizes that some symptoms—such as shortness of breath—can be associated with not only an acute PE but also severe ischemic CAD. This case also emphasizes the importance of a comprehensive evaluation prior to embarking on major surgery. It would not have been difficult to picture a difficult hospital course had the PE been addressed without treating his ischemic cardiomyopathy and severe CAD—and, similarly, the challenges in treating his CAD without simultaneously addressing his PE. Patients who present with hemodynamic instability due to both severe CAD and PE might have historically been considered inappropriate surgical candidates; we hope our case demonstrates otherwise.

CONCLUSIONS

Shortness of breath, although often associated with PEs and severe CAD with heart failure, can sometimes be a manifestation of a patient presenting with both. In our patient, surgical management of both problems was successful and potentially avoided a disaster that might have occurred had we treated one problem but not the other. Failure to appropriately diagnose and manage underlying CAD may account for an increased mortality risk in patients presenting with PE. Likewise, the incomplete management of undiagnosed confounding and preoperative problems might explain adverse outcomes after cardiac surgery.

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