CASE REPORTS

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Massive Intraoperative Pulmonary Embolus Diagnosed by Transesophageal Echocardiography

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INTRAOPERATIVE pulmonary embolism is an important cause of morbidity in patients undergoing pelvic surgery.[1] However, the diagnosis of pulmonary embolism may be difficult to make because intraoperative hypotension can have many different causes. Indeed, 30% of patients who succumb to fatal pulmonary emboli have an incorrect provisional diagnosis before autopsy.[2] The transesophageal echocardiogram (TEE) may improve the anesthesiologist's ability to diagnose emboli by allowing rapid and specific visualization of the thromboembolism.

We report a case of severe hemodynamic deterioration due to a massive intraoperative pulmonary embolus diagnosed by TEE in a morbidly obese patient undergoing pelvic surgery. This case reveals a complete hemodynamic profile, from initiation through clinical resolution of the embolus, with concomitant TEE documentation of intracardiac presence and dissolution or passage of a massive pulmonary embolus.

Case Report

A 46-yr-old, 144-kg woman was scheduled for a total abdominal hysterectomy and bilateral salpingo-oophorectomy for bleeding fibroids. The patient's past medical history was significant for sarcoidosis with cor pulmonale. The latter was attributed to her morbid obesity with some contribution from parenchymal pulmonary disease secondary to her sarcoidosis. The patient was able to walk one block without shortness of breath before admission, her usual state of health. She smoked half a pack of cigarettes per day. Her medications included furosemide, methylprednisolone as well as beclomethasone, albuterol, and beclomethasone inhalers. TEE revealed normal left ventricular size and function, a slightly enlarged right atrium and a markedly enlarged right ventricle (RV) with decreased systolic function. A preoperative impedance plethysmogram was normal. Results of pulmonary function tests were forced expiratory volume in 1 s = 1.21 l (48% of predicted) and forced vital capacity = 1.93 l (64% of predicted). Analysis of an arterial blood gas sample taken during room-air breathing revealed *p*H = 7.45, oxygen tension = 61 mmHg, and carbon dioxide tension = 41 mmHg. Her preoperative hematocrit was 29%.

The patient was brought to the operating room where a lumbar epidural catheter was placed for postoperative analgesia. Pneumatic leg compression cuffs were placed. A catheter was inserted into a radial artery and a pulmonary artery (PA) catheter was placed *via* the right internal jugular vein. Before induction of anesthesia, hemodynamic data included arterial blood pressure = 120/63 mmHg (mean arterial pressure = 79), central venous pressure = 18 mmHg, PA wedge pressure = 28 mmHg, PA pressure = 97/37 mmHg, heart rate = 79 beats/min, and cardiac output = 10.3 l/min. Anesthesia was induced with fentanyl 2,000 μg, midazolam 2 mg, and vecuronium 10 mg. Tracheal intubation was performed without hemodynamic alteration and a TEE probe was passed revealing no significant differences from the preoperative study. No thrombi were noted in the cardiac chambers. Anesthesia was maintained with isoflurane from 0.2–1.0% with no alteration in vital signs. The epidural catheter was injected with preservative-free morphine 3 mg.

The patient was initially hemodynamically stable with a systemic blood pressure of 120/60 mmHg and a PA pressure of 60/30 mmHg. After 80 min of surgery, the systemic blood pressure decreased abruptly from 120/62 to 45/32 mmHg, while the PA pressure initially decreased then increased to 100/70 mmHg (fig. 1, left). The rhythm was sinus with a rate of 86. End-tidal carbon dioxide tension decreased from 32 to 14 mmHg. The hemoglobin oxygen saturation decreased from 100% immediately before the event to an unmeasurable value. ST depression was noted in leads II and V5. Isoflurane was discontinued and surgical packs were removed from the pelvic region. The surgeons denied recent manipulation of the pelvic vessels. Intravenous calcium chloride 500 mg and intravenous epinephrine 2 mg (in 500-μg increments) were administered. The PA pressure returned to the 50/30-mmHg range after 2 min with no recovery of systemic arterial blood pressure. Further doses of epinephrine were given and a dopamine infusion was started at 15 $\mu g \cdot kg^{-1} \cdot min^{-1}$ for support of systemic arterial blood pressure. The vasopressors increased the systemic blood pressure to 60/30 mmHg but also increased PA pres-

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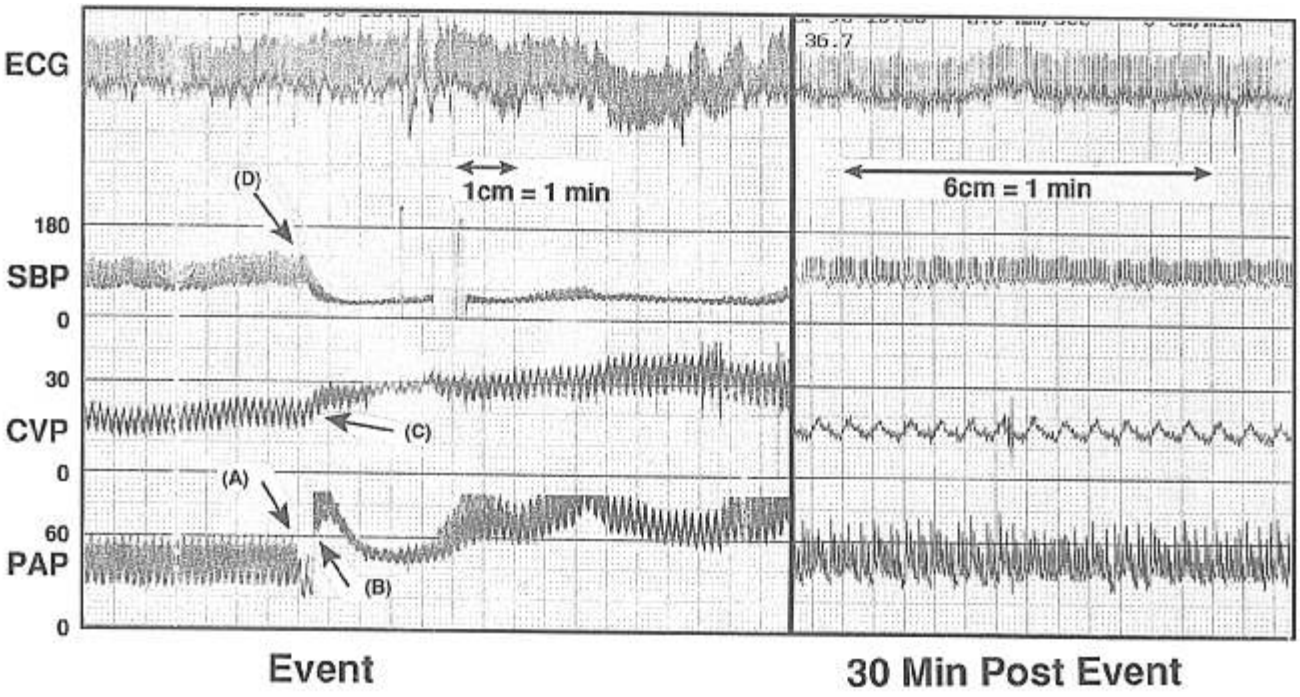

Fig. 1. Hemodynamic data during (*left*) and after recovery from (*right*) pulmonary embolus. (*A*) Initial, transient decreased pulmonary artery (PA) pressure. (*B*) Abrupt, sustained increase in PA pressure. (*C*) Progressive increase in right atrial pressure. (*D*) Acute and profound systemic arterial hypotension. Chart paper speed (*left*) 1 cm/min and (*right*) 6 cm/min.

sure into the 120/70-mmHg range approximately 5 min after the event.

At this point, TEE examination revealed a large right atrial mural thrombus with right atrial dilatation, RV dilatation with inferior wall hypokinesis. Notably, the intramural thrombus (approximately 10 cm × 2 cm × 3 cm) extended from the inferior vena cava through the right atrium and was adherent to the tricuspid valve (fig. 2). The left chambers were nearly empty of volume. No thrombus was seen in the proximal pulmonary arteries. However, by 10 min postevent the PA pressure was 180/130. Severe pulmonary vasoconstriction was considered as a possible contributing factor to the pulmonary hypertension, and prostaglandin E_1 (40 $ng \cdot kg^{-1} \cdot min^{-1}$) and nitroglycerin (5–10 μg/min) infusions were started and the anesthesia was deepened with fentanyl 80 μg/kg to treat any reversible vasoconstriction. The PA hypertension and systemic hypotension slowly resolved. Approximately 15 min postevent, arterial blood pressure was 100/80 mmHg, PA pressure was 170/100 mmHg, central venous pressure was 35 mmHg, and heart rate was 108 min^{-1}. Although the hemoglobin oxygen saturation improved to 99%, there was still a

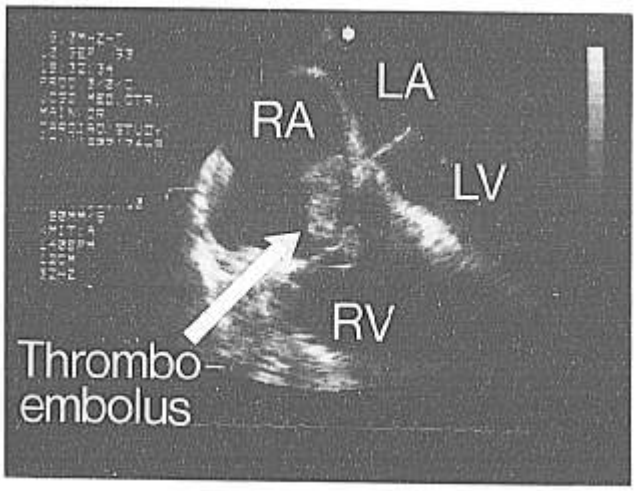

Fig. 2. Transesophageal echocardiographic image (four-chamber view of heart) demonstrating thromboembolus (arrow) extending from the right atrium (RA) through the tricuspid valve into the right ventricle (RV). LA = left atrium; LV = left ventricle. One-centimeter intervals are indicated at the right margin of the screen.

Table 1. PaO_2/FIO_2 and Calculated Percent Shunt for Patient before, during, and after Resolution of the Pulmonary Embolus

Time	PaO_2/FIO_2	Qs/Qt*
1 h before	462	9.6
During event	144	28.6
1 h after event	302	14.2
2 h after event	496	8.5

* Qs/Qt calculated using the method of Benumof JL, Rauscher A: Comparison of respiratory index {$P(AaDO_2)/PaO_2$} with transpulmonary shunt. ASA Abstracts 1977, pp 185–186.

substantial intrapulmonary shunt, demonstrated by an arterial oxygen tension of 144 mmHg with a fraction of inspired oxygen of 1.0 (table 1). Approximately 25 min postevent, the patient's hemodynamic parameters and oxygenation had nearly returned to the preevent baseline status with a PA pressure of 63/40 mmHg (fig. 1, right). One hour after the embolic event, the arterial oxygen tension had returned to 302 mmHg (fraction of inspired oxygen = 1.0) and the calculated shunt had decreased from 28.6% to 14.2% (table 1). The thrombus shown in figure 2 remained in the right atrium and RV for approximately 30 min and then suddenly disappeared (fig. 3). No new hemodynamic sequelae accompanied its disappearance. The surgery concluded without any further hemodynamic impairment.

A nuclear scan, performed immediately after the operation, showed areas of decreased perfusion in the upper two thirds of the lung fields, which were not significantly different from a previous V/Q scan. The study was interpreted as "indeterminate" for pulmonary embolus. Heparin was started approximately 3 h postevent, and the patient's trachea was extubated on the first postoperative day. At this time, impedance plethysmography found no evidence for deep venous thrombosis in either leg. The diagnosis of myocardial infarction was excluded by serial electrocardiograms and serum creatine phosphokinase measurements. On the third postoperative day, a PA angiogram showed no evidence of pulmonary thromboembolic disease, and heparin was stopped.

Discussion

A pulmonary embolus can be among the most challenging diagnoses to make because its signs and symptoms are nonspecific.[3] Indeed, Goldhaber *et al.* found that only 30% of fatal pulmonary emboli were correctly diagnosed before autopsy.[2] If we had not had specialized monitoring (TEE) for our patient, we would have considered many other causes for the acute intraoperative hypotension, including hypovolemia secondary to acute hemorrhage, inferior vena cava compression, anaphylaxis, increased vagal tone or release of vasoactive mediators secondary to visceral traction, or myocardial ischemia. In this case, we noted an initial abrupt, and transient (10–15 second) decrease in PA pressure, followed by marked increase in PA pressures, with concomitant persistent systemic hypotension (fig. 1, left). The abrupt decrease in PA pressure probably represents thrombus induced advancement of the PA catheter into a wedge position. Alternatively, it could have occurred from an initial decrease in forward flow resulting from obstruction of the RV proximal to the pulmonary arteries themselves (the result either of decreased RV filling from tricuspid valve obstruction or of RV ejection from pulmonic valve obstruction). The later explanation is less likely because systemic blood pressure was maintained for approximately 10 seconds after the PA pressures decreased, an unlikely event if a clot had acutely blocked RV outflow.

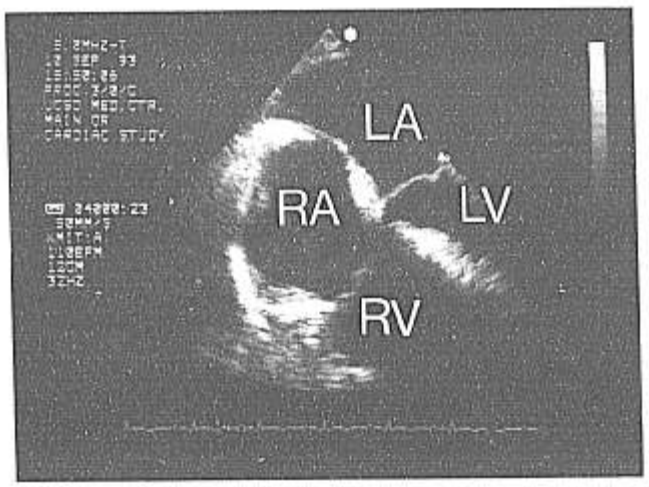

Fig. 3. Transesophageal echocardiographic image (four-chamber view of heart) taken after the passage of the thromboembolus. RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle. One-centimeter intervals are indicated at the right margin of the screen.

The subsequent increase in PA pressures accompanying the systemic hypotension eliminated causes involving reduced preload and suggested the diagnosis of pulmonary embolism. Other causes were still possible, such as myocardial ischemia, acute right heart failure, and reactive pulmonary hypertension. We were able to make the diagnosis of pulmonary embolism because the TEE allowed direct visualization of myocardial function and the contents of the intracardiac chambers.

The diagnosis of embolism allowed us to initiate aggressive therapy directed toward the treatment of a massive pulmonary embolus. The TEE demonstrated inferior and lateral hypokinesis and RA enlargement in addition to the thromboembolus. These characteristics are consistent with right-sided failure resulting either from ischemia or from mechanical obstruction of flow. Ellis *et al.* reported similar evidence for right-sided failure in orthotopic liver transplant patients who had thrombi demonstrated by TEE.[4] The worsened pulmonary hypertension can be explained by mechanical obstruction of pulmonary flow by the embolus or fragments originating from it and possibly by the release of vasoconstricting mediators acting on the pulmonary vascular bed.[5] The systemic hypotension resulted from decreased blood flow to the left heart with decreased systemic cardiac output. TEE eliminated left ventricular failure as the cause of the hemodynamic changes because the left ventricle was empty of volume, and contracting normally.

Therapy was initially supportive. Our first priority was to provide adequate coronary perfusion pressure. Right coronary artery perfusion was considered most important in this patient because of the baseline RV hypertrophy, along with the further increased RV intramural pressure required to eject blood through the acutely increased RV afterload.

Pulmonary vasodilators were added secondarily to help maintain flow through the lungs, and fill the relatively empty left ventricle. Pearl and colleagues reported that nitroglycerin was efficacious in reducing pulmonary vascular tone in patients with primary pulmonary hypertension,[6] but it has inconsistent effects in patients with pulmonary hypertension of other origins. Good results have been obtained using prostaglandin E_1 in patients undergoing pulmonary thromboendarterectomy for chronic thromboembolic disease.[7] The administration of high dose fentanyl was aimed at attenuating vasoconstriction that may have resulted from light anesthesia (with resulting pain and sympathetic stimulation). Inhaled nitric oxide, may have provided pulmonary vasodilation,[8] and was considered. However, in our patient, the pulmonary vascular resistance had returned to baseline within 30 min (less than the amount of time required for us to set up and calibrate our nitric oxide administration apparatus).

This patient's hypertrophied RV from long-standing pulmonary hypertension probably helped her to survive this event. Her RV was able to generate the high pressures (as great as 180/130 mmHg) necessary to drive blood through the compromised pulmonary vascular bed without failing. In previous case reports of intraoperative pulmonary embolus diagnosed by TEE, patients succumbed with acute right heart failure.[9,10]

Resolution of the event occurred over 30 min, probably the result of dissolution or fragmentation of the clot. We do not think the thromboembolus shown in figure 2 slipped downstream into the pulmonary bed intact because the pulmonary hypertension and systemic hypotension were improving at this time, and after it disappeared from the heart we could not find any thrombus in the proximal pulmonary vessels with TEE. Furthermore, there was no evidence of large pulmonary embolus seen postoperatively by V/Q scan or pulmonary angiogram.

There have been two previous case reports of intraoperative pulmonary emboli diagnosed by direct visualization of clot using a TEE placed after the event[9,10] and one surveillance study aimed at detecting right heart dysfunction encountered during liver transplantation.[4] Our case was unique in that we observed the clot, completely recorded the hemodynamic changes that occurred during the near fatal event, and demonstrated spontaneous resolution of the clot (fig. 2 compared with fig. 3).

We believe our patient's embolus originated from a thrombosed pelvic vein because she had no evidence of leg deep venous thrombosis (DVT) either preoperatively or postoperatively. Pneumatic compression stockings were used in this patient as prophylaxis against thromboembolic disease. These means of DVT prophylaxis may not protect against DVTs of pelvic origin.

The limitations of TEE include the possibility of missing small emboli or those that have already passed into the pulmonary vascular bed. The TEE is only a useful monitor during maintenance of anesthesia as it is not typically placed in the patient during induction or emergence from anesthesia. Furthermore the TEE requires skill in interpretation, and can divert the clinicians attention away from other aspects of patient care.

A preoperatively placed TEE may be valuable in patients at high risk for thromboembolism because it provides a baseline examination and is then in place to assist with diagnosis should hemodynamically significant events occur. In our case, the TEE helped us distinguish pulmonary thromboembolism from reactive pulmonary vasoconstriction or myocardial ischemia and guided our therapy.

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Perioperative Recognition, Management, and Pathologic Diagnosis of Transfusion-related Acute Lung Injury

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THERE are many well-described complications of hemotherapy, including acute and delayed hemolytic reactions, transmission of infectious agents, and febrile and hypersensitivity reactions. Noncardiogenic pulmonary edema resulting from blood-product transfusion has been characterized as an infrequent, though serious, complication of hemotherapy resulting from immunoreactivity of certain leukocyte antibodies. Most reports have appeared in pulmonary and hematology journals; only three case reports have been published in the anesthesia literature in the last 10 yr.[1–3] Nomenclature has been descriptive, using terms such as transfusion-associated noncardiogenic pulmonary edema and transfusion-related adult respiratory distress syndrome (ARDS).[1,4]

Other investigations, including some more recent, have been more definitive, attributing this clinical syndrome to a specific antigen–antibody mechanism involving human leukocyte antigen (HLA) and granulocyte antigens, called transfusion-related acute lung injury (TRALI).[5–10] In this report, we describe a patient who developed fulminant noncardiogenic pulmonary edema after the administration of packed red blood cells in the operating room whose hematologic and clinical evaluation fulfills criteria to substantiate the diagnosis of TRALI.

Case Report

A 51-yr-old morbidly obese gravida 5, para 5 woman with atypical adenomatous endometrial hyperplasia was admitted to our hospital for total abdominal hysterectomy and bilateral salpingo-oophorectomy. Preoperative laboratory studies were normal with the exception of mild hyperglycemia. In the operating room, she lost 2,200 ml blood acutely by hemorrhage from extensive retrouterine varices; the blood loss was difficult to control. She received four units of type-specific, crossmatched packed red blood cells, as well as crystalloid and hetastarch solution (Hespan, DuPont, Wilmington, DE).

One hour after administration of the fourth unit of blood, her hemoglobin oxygen saturation measured by pulse oximetry abruptly decreased from 95% to 85% and frothy serous fluid appeared in the tracheal tube. A Foley catheter and radial artery catheter were in place. She was given intravenous furosemide, then transported to the surgical intensive care unit with her trachea intubated and fraction of inspired oxygen 1.0, manually applied continuous positive airway pressure *via* a modified Jackson-Rees circuit, and a hemoglobin oxygen saturation of 100%. A pulmonary artery catheter was inserted. Arterial blood gas evaluation revealed *p*H 7.22, carbon dioxide tension 39 mmHg, and oxygen tension 168 mmHg. A portable chest roentgenogram showed diffuse opacity of both lung fields and indistinct vasculature consistent with pulmonary edema.

A transesophageal echocardiogram demonstrated normal mitral valve function, good left ventricular function, and no evidence of left ventricular wall motion abnormality. The electrocardiogram was unchanged from a preoperative study except for increased heart rate and a slightly prolonged QT interval. Her cardiac output was 5.1 l/min, pulmonary artery occlusion pressure 16 mmHg, heart rate 130 beats/min, and systolic blood pressure 66 mmHg with infusion of dopamine and dobutamine.

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