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Life-threatening Pulmonary Embolism at Induction of Anesthesia: Utility of Transesophageal Echocardiography

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THE origin of sudden shock during general anesthesia without obvious cause (allergic reaction, hemorrhage, hypovolemia, or pneumothorax) may be difficult to identify because specialized diagnostic facilities often are not readily available in the operating room. When more complex cardiovascular events have to be considered, such as myocardial infarction, cardiac tamponade, aortic dissection, or massive pulmonary embolism, transesophageal echocardiography (TEE) allows rapid and precise assessment of the heart and its surrounding structures and may thus be of great value in the differential diagnosis of these life-threatening conditions.

We report a case in which TEE helped in the rapid diagnosis and management of massive pulmonary thromboembolism during the induction of general anesthesia.

Case Report

A 62-yr-old woman, weighing 70 kg, with tonic-clonic epilepsy was found to have a frontoparietal cerebral mass of unknown cause. She was scheduled for an exploratory craniotomy. Her medical history included arterial hypertension and chronic pericardial effusion of unknown origin. The results of preoperative examination were normal except for cardiac enlargement noted on the chest x-ray. Echocardiographic examination revealed a moderate pericardial effusion. Eight hundred fifty milliliters nonhemorrhagic pericardial effusion was drained 2 days before surgery. The electrocardiogram showed rapid atrial fibrillation for which treatment with amiodarone was

started. Ranitidine 150 mg orally, hydrocortisone 100 mg, and promethazine 25 mg intravenously were given for premedication.

On arrival in the operating room, monitoring with electrocardiography, noninvasive blood pressure recording, and pulse oximetry was instituted. The Glasgow Coma Score was 13 (4/6/3); the blood pressure was 125/65 mmHg; the electrocardiogram showed sinus rhythm at 92 beats/min; and the hemoglobin oxygen saturation was 95% while she breathed room air. Catheters were inserted into the radial artery and the internal jugular vein. After the infusion of 500 ml 0.9% saline and oxygen breathing for 5 min, anesthesia was induced by the intravenous injection of propofol 1.5 mg/kg, alfentanil 15 µg/kg, and vecuronium 0.1 mg/kg.

During the next 1–2 min the blood pressure decreased to 40/20 mmHg and tachycardia (140 beats/min) with marked jugular venous distension was noted. The hypotension neither responded to the rapid infusion of 500 ml of 0.9% saline nor to repeated injections of phenylephrine up to a total dose of 500 µg. After tracheal intubation, the lungs were ventilated with 100% oxygen. Capnography revealed an end-expiratory carbon dioxide concentration of 2.3%. The hemoglobin oxygen saturation was 98%. An arterial blood gas sample showed severe acidosis (pH 7.23, carbon dioxide tension 6.41 kPa [48 mmHg], oxygen tension 15.9 kPa [119 mmHg], bicarbonate 19.6 mM, and base excess -7.9 mM).

Because of the absence of a response to the initial treatment within a few minutes, 100 mmol sodium bicarbonate and repeated intravenous bolus injections of 50 µg epinephrine were given, followed by a continuous infusion of 0.06 µg · kg⁻¹ · min⁻¹. The blood pressure increased to 90/50 mmHg. Digoxin 0.25 mg was given intravenously to treat atrial fibrillation. A chest x-ray was taken. It showed an enlarged heart with decreased vascular markings over the right lung field. Biplane TEE showed dilated right heart chambers and a small, hyperkinetic left ventricle. Shifting of the interventricular septum to the left suggested pulmonary hypertension. Significant pericardial effusion was excluded. Examination of the main pulmonary artery demonstrated a partially obstructive mass extending into the right pulmonary artery (fig. 1), consistent with massive pulmonary embolism.

Systemic thrombolysis with recombinant tissue plasminogen activator (15-mg bolus intravenous injection followed by 45 mg/h for 2 h) and intravenous heparin (5,000-IU bolus injection followed by 30,000 IU/24 h) through a peripheral vein was started. This resulted in a rapid increase in blood pressure, allowing the epinephrine infusion to be discontinued after 2 h. Subsequent TEE monitoring documented progressive and complete disappearance of the pulmonary artery mass 6 h after the beginning of thrombolysis (fig. 2). No hemorrhagic complication occurred. Later, Doppler-ultrasound examination revealed a deep venous thrombosis of the right femoral vein.

The patient left the intensive care unit after 2 days, receiving intravenous heparin at 30,000 IU/24 h. She refused further surgical treatment.

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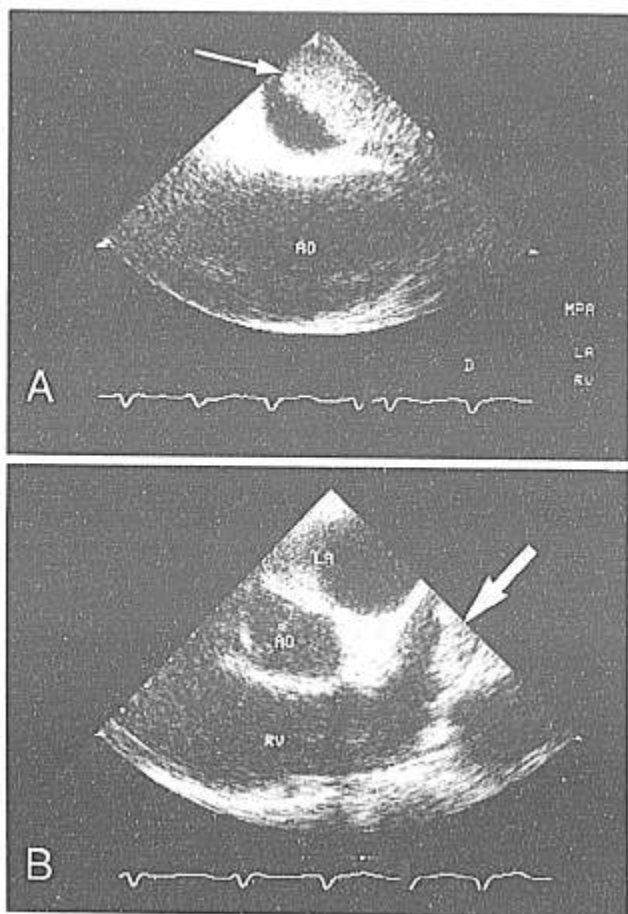


Fig. 1. Partially obstructive mass located in the right pulmonary artery (RPA) (A, small arrow), which was an extension of the mass located in the main pulmonary artery (B, large arrow). AO = aorta; LA = left atrium; MPA = main pulmonary artery; RV = right ventricle.

Discussion

Severe hypotension and tachycardia during the induction of anesthesia may be due to allergic reaction, hypovolemia, or low cardiac output. In our patient, allergic reaction and hypovolemia were clinically excluded. Jugular venous distension in addition to hypotension and tachycardia may evoke low cardiac output, as encountered in myocardial infarction, cardiac tamponade, and pulmonary embolism. This differential diagnosis is especially difficult during general anesthesia because of the lack of history or specific clinical signs. The changes in blood gas values may be useful but are not specific. In this context, echocardiographic examination is a rapid and accurate diagnostic tool that

allows assessment of the size and function of cardiac chambers. Pericardial effusion also may be easily ruled out. Thus, TEE can extend diagnostic capability, especially in the operating room, where transthoracic echocardiography often is not feasible. TEE, compared with the transthoracic approach, provides better visualization of the right ventricular outflow tract and main pulmonary artery.¹ Few cases of pulmonary thromboembolism confirmed by TEE have been reported so far.²⁻⁵

Sermeus *et al.*⁶ reported the case of an intraoperative pulmonary embolism after the application of an Es-march bandage and pneumatic tourniquet during arthroscopy of the knee. TEE performed in the operating

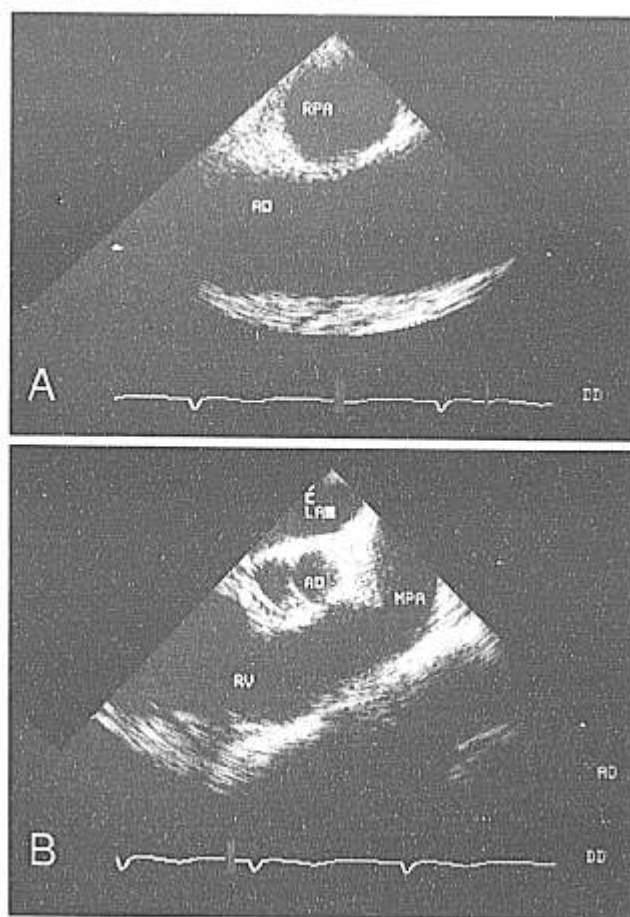


Fig. 2. Disappearance of the mass located in the main pulmonary artery (B) and extending in the right pulmonary artery (A) (RPA) 6 h after the start of thrombolysis. AO = aorta; LA = left atrium; MPA = main pulmonary artery; RV = right ventricle.

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room allowed rapid confirmation of the diagnosis and subsequent successful surgical embolectomy.

Our case is the first description of the identification by TEE of a massive pulmonary embolism occurring during induction of anesthesia. This procedure allowed a quick diagnosis and on-line assessment of successful thrombolysis. Systemic thrombolysis is now available for treatment of massive pulmonary thromboembolism. However, no studies to date have been able to demonstrate that thrombolysis results in better survival when compared with surgical therapy.^{7,8} The drugs and doses recommended for the treatment of pulmonary thromboembolism are the same as those generally used for myocardial infarction.⁹ In two studies, recombinant tissue plasminogen activator appears to possess some advantages over urokinase.^{10,11} Intrapulmonary infusion of the drug does not seem better than systemic application.¹² Surgical removal of the thrombus is another treatment option. In a recent review of published studies, it was, however associated with a perioperative mortality of 42%.¹³

In the only series comparing embolectomy to streptokinase thrombolysis or treatment with heparin alone, mortality was similar (25%) for embolectomy and thrombolysis in patients with systemic blood pressure of less than 100 mmHg.¹⁴ In this limited series, these approaches provided a significant advantage compared with heparin therapy (mortality 50%). The choice between embolectomy or streptokinase thrombolysis was mainly determined by local facilities. In our case, the main concern was the risk of cerebral bleeding associated with thrombolysis, which had to be weighed against the delay linked to surgery. Thrombolysis was finally chosen because of the reluctance of cardiac surgeons to operate on a patient with a suspected cerebral tumor. The improvement in blood pressure associated with progressive disappearance of the pulmonary artery mass on TEE examination confirmed the efficacy of thrombolysis.

To summarize, this case illustrates the versatility of TEE in the management of certain life-threatening events occurring in the operating room.

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