

Amniotic Fluid Embolism: Early Findings of Transesophageal Echocardiography

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Amniotic fluid embolism (AFE) is a rare event, but one that is responsible for approximately 10% of all maternal deaths (1). Regardless of treatment, it is associated with a very high mortality rate (2). While approximately 70% of maternal deaths caused by AFE are a result of cardiopulmonary collapse (3), the pathophysiology of the cardiopulmonary disturbance is not clearly understood. Almost all human hemodynamic data post-AFE were collected 1 h or more after the event (4), yet 25%–50% of the parturients die within the first hour of clinical presentation (5,6). We present a case in which transesophageal echocardiography (TEE) was performed during the early course of AFE. The TEE findings provide additional information regarding the cardiopulmonary pathophysiology occurring during the very early phase of AFE.

Case report

A 26-yr-old healthy woman, gravida 3, para 1, in Week 40 of gestation, was admitted to the delivery room as a result of complaints of vaginal bleeding. She had a normal delivery in Week 40 of her first pregnancy and a missed abortion in the 10th week of her second.

On admission, the patient was awake, alert, and in no distress. Blood pressure was 100/70 mm Hg, and pulse was 80 bpm. There was no vaginal bleeding. The cervix was closed, 60% effaced, and the fetal head was at minus 2 station. Ultrasound showed a normal volume of amniotic fluid and low insertion of the placenta. The fetus was in a vertex presentation. Uterine activity was irregular, and the fetal heart rate was appropriately reactive. Because of ineffective uterine contractions, IV oxytocin was administered. The oxytocin dose was gradually increased, according to the delivery room protocol, from 0.001 to 0.0075 U/min. Eighty minutes after the initiation of oxytocin, the patient complained of the sudden onset of dyspnea, blindness, and palpitations. At that time she was conscious, oriented, and cooperative, but extremely anxious and cyanotic. Her lungs were clear, and her blood pressure was 170/110 mm Hg,

although peripheral pulses were barely palpable. No arrhythmia was seen on the maternal electrocardiogram (ECG) monitor. Fetal monitoring showed sustained bradycardia, and the patient was immediately transferred to the operating room (OR) for an emergency cesarean delivery. The transport took 3–4 min.

On arrival in the OR, the patient was unconscious. Marked central cyanosis was noted, and respiration was agonal. Heart rate, as seen on the ECG cardiac monitor, was 90 bpm, but the carotid and femoral pulses were not palpable. Marked congestion of the neck veins was noted.

Endotracheal intubation was immediately performed, and the patient was mechanically hyperventilated with a fraction of inspired oxygen (F_{iO_2}) of 1.0. Closed cardiac massage was started, and an emergency cesarean delivery was performed. The baby was delivered within 2 min with an Apgar score of 1 at 1 min. Maternal cardiopulmonary resuscitation was continued. Epinephrine and sodium bicarbonate were administered in repeated doses, and a central venous catheter was inserted. Blood gases drawn from the right atrium demonstrated severe metabolic acidosis and tissue hypoxia (P_{O_2} = 17 mm Hg, P_{CO_2} = 47 mm Hg, pH = 7.11, bicarbonate = 15 Meq/L, base excess = -13, lactate = 12 mmol/L). Because no active bleeding was noted, the uterus was sutured and the abdomen closed. At that point, the pulse became palpable, with a blood pressure of approximately 90/60 mm Hg. A TEE probe was inserted, and a long axis four-chamber view was obtained. It showed acute right ventricular failure, suprasystemic right-sided pressures, bulging of the interatrial and interventricular septum from right to the left, severe tricuspid regurgitation, and a small pericardial effusion (Figures 1 and 2). Pulmonary artery systolic pressure (from tricuspid regurgitation jet) was 45 mm Hg, while at the same time no arterial pulse was palpable. The left ventricle was small and compressed. Its lateral wall had normal contraction and thickening. The bulging interventricular septum was thickening, but had abnormal contraction. Neither patent foramen ovale, nor interventricular communication could be demonstrated. Ten minutes later, profuse vaginal bleeding became evident. A subtotal hysterectomy was performed, followed by manual aortic compression. Red blood cells, platelets, and clotting factor concentrates were administered. After there was no response to the resuscitative efforts, open cardiac massage was performed, but despite all the vigorous attempts, the patient was pronounced dead 115 min after entering the OR.

Postmortem examination revealed moderately congested and edematous lungs. Histology demonstrated small blood vessels containing fibrin thrombi, mucoid substance, and

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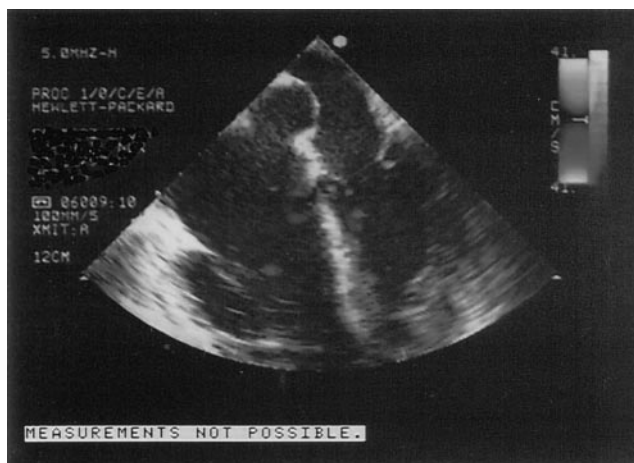


Figure 1. Transesophageal four-chamber image at diastole (indicated by mitral valve opening) demonstrating very large failing right ventricle with bulging of the interatrial septum from right to left. The interventricular septum is also shifted to the left.

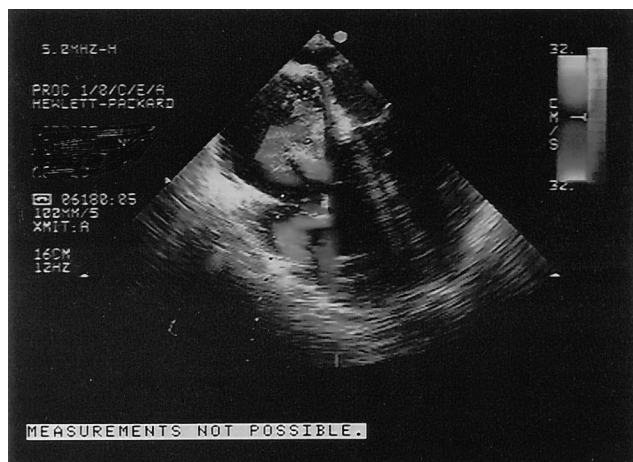


Figure 2. Transesophageal four-chamber image at systole (indicated by mitral valve closure) demonstrating severe tricuspid regurgitation.

keratin squames. Focal inflammatory perivascular infiltration by neutrophils and lymphocytes was also present. These findings are typical of AFE and disseminated intravascular coagulation.

Discussion

Animal studies have shown that the first response after AFE is an intense pulmonary vasospasm, which results in acute pulmonary hypertension and right-sided failure (4). Although data regarding human response in the first hour after AFE are very limited, there are a number of reports suggesting that the primary mechanism is left ventricular failure, with increased pulmonary capillary wedge pressure (7,8). Clark et al. (9,10) evaluated hemodynamic variables in patients with AFE and suggested that left ventricular

dysfunction is the primary cause of pulmonary edema. These authors reported an increase in pulmonary capillary wedge pressure and, in most cases, a decrease in left ventricular stroke-work index. Similar findings were also demonstrated in a case report in which echocardiography was performed 3.5 hours after the acute event (11).

Our patient presented with the typical manifestations of AFE, including the sudden onset of dyspnea and cardiovascular collapse. This is the first report of TEE findings during the hyper-acute stage of AFE, and it revealed severe pulmonary hypertension and acute right ventricular failure with a leftward deviation of the interatrial and interventricular septum. Elevated central venous pressure was diagnosed immediately on the patient's arrival in the OR; however, there were no signs of left ventricular dysfunction or of pulmonary edema. The lungs were clear on auscultation and the $\text{PaO}_2/\text{FiO}_2$ ratio was 430. These findings suggest that pulmonary vasoconstriction and increased pulmonary vascular resistance were the primary mechanisms responsible for the cardiovascular collapse. It has recently been suggested that the pathophysiology of AFE is similar to anaphylactic or septic shock (1) and is associated with the release of inflammatory mediators. Arachidonic acid metabolites may induce hemodynamic changes, which are very similar to AFE (12). Because most studies demonstrating left ventricular failure as the main pathology have been performed more than one hour after the appearance of clinical signs, it is possible that increased pulmonary vascular resistance and right-sided failure occur in the early stage of AFE. Left ventricular failure may ensue later (13) and will be detected only in a patient who survives the initial insult. The difference between our findings and those of others might be explained by the fact that previous human studies have not provided information on cardiopulmonary function during the early phase of AFE. It is also possible that the mechanism of cardiovascular collapse may vary from one patient to another.

As our experience increases, TEE may help in establishing the diagnosis and in guiding therapy for this catastrophic condition. For now, TEE examination during the acute phase will improve our understanding of the pathophysiologic changes that occur.

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