Postoperative Venous Air Embolism

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Venous air embolism can precipitate cardiac arrest if it is of sufficient size to decrease cardiac output seriously. The presence of a central venous pressure (CVP) catheter and a low CVP predispose a patient to the development of a venous air embolism should the catheter somehow become open to air. The following case illustrates a situation in which this occurred.

Case Report

A 56-year-old, 60-kg woman with a history of chronic renal failure underwent a gastric antrectomy-vagotomy because of partial gastric outlet obstruction resulting from severe peptic ulcer disease. On the day before surgery dialysis was performed with a loss of 1 kg of body weight. Preoperatively her blood pressure ranged between 168/70 to 120/60 torr, serum potassium (K+) was 3.9 meq/L, and hemoglobin was 8.0 gm/100 ml. Her anesthetic course was uneventful. The anesthetic agents were thiopental, halothane, nitrous oxide-oxygen, and curare. A CVP catheter was placed through the left external jugular vein to monitor the CVP. The catheter could not be seen on the postoperative chest x-ray. During the 5-hour operation the patient received 900 ml of 5% dextrose in half-normal saline, 250 ml of normal saline, and two units of packed red blood cells. Estimated blood loss was 450 ml.

During the night following surgery the patient had several episodes of hypotension with the systolic blood pressure falling to 85 to 100 torr, associated with a pulse rate of 90 to 110 beats per minute and a CVP ranging between less than 0 to 1.0 cm H2O. Each of these episodes responded to fluid administration. Two units of packed red blood cells, 250 ml of 25% albumin, 500 ml of normal saline, and 1000 ml of 5% dextrose in half-normal saline were administered in 16 hours. Measured fluid loss was confined to 400 ml through a nasogastric tube. There was no urinary output. The patient was kept at bedrest during the night.

At 6:00 a.m. the CVP was 0.5 cm H2O and at 6:30 a.m. her blood pressure was 120/60 torr with a pulse of 85 beats per minute. At 7:00 a.m. respirations were "noisy" at 24 per minute. She was medicated for pain with 6 mg of morphine intramuscularly. A blood specimen was drawn for hemoglobin (Hb) and electrolyte determinations (Hb, 9.2 gm/100 ml; K+, 5.8 meq/L). At 7:15 a.m. the floor nurse heard the patient call for help. She found the patient supine, pale, with irregular respirations and a barely palpable carotid pulse. The CVP catheter, which had become separated from the tubing at the catheter hub, was reconnected. No blood was present on the bedsheets. The nurse did not attempt to aspirate blood through the CVP catheter or examine the catheter hub for air. She called the nearest physician, and cardiopulmonary resuscitation was instituted when he found no peripheral pulses.

Ventilation with 100% oxygen by mask failed to improve the patient’s cyanosis despite obvious expansion of the chest and breath sounds in both axillae with each ventilation. The cyanosis persisted despite endotracheal intubation and continued adequate ventilation with 100% oxygen until the patient’s hypotension began to resolve. No evidence of aspiration of gastric contents was found on laryngoscopy. A chest x-ray taken after resuscitation showed left lower lobe atelectasis which had been present on the immediate postoperative x-ray. An arterial blood sample taken during the period of cyanosis while the patient was being ventilated with 100% oxygen revealed pH 7.22, PaCO2, 101 torr; HCO3, 63 torr; Hb, 27 meq/L; and base excess of -3.

With external cardiac massage palpable pulses returned and were well maintained without further assistance, but the patient remained hypotensive for 15 minutes. Initially the blood pressure of 70 torr systolic was attributed to a bradycardia, but correction of the 40 beats per minute pulse rate with intravenous atropine failed to improve the blood pressure. No other arrhythmias occurred. The heart was not auscultated during resuscitation; the presence or absence of a murmur remains unknown. The CVP was 12 cm H2O after correction of the bradycardia; yet only 300 ml of 5% dextrose in 0.2 normal saline had been administered during the arrest whereas during the night the administration of 150 ml of 25% albumin, one unit of packed red blood cells, and 250 ml of normal saline over a 3-hour period had raised the CVP from less than 0 to 4.5 cm H2O. An hour after resuscitation the CVP was again 0.5 to 1 cm H2O.

Four hours after the arrest, the patient’s abdomen was reexplored when blood was found leaking from the abdominal wound. A small "ooze" in the retroperitoneal space near the pancreas was found with sufficient free blood and clot in the abdomen to explain a blood loss of 2 to 3 units.

Mild pulmonary edema by both x-ray and clinical criteria developed 6 to 8 hours after the resuscitation, but only vigorous fluid therapy during a subsequent hypotensive episode and during the abdominal reexploration may have contributed to this. There was no evidence of sepsis as the basis for the patient’s requirements of large volumes of intravenous fluids. There was no evidence of a myocardial infarction by sequential electrocardiograms.
Discussion

The signs and symptoms of a venous air embolism are generally nonspecific. Only changes in precordial Doppler ultrasound or a step-decrease in end-tidal CO₂ are specific signs for a pulmonary embolism of air or particulate material. A mill-wheel murmur may be the only specific sign of an air embolism. The diagnosis of venous air embolism in this patient is based on nonspecific clinical signs which resolved over a 15-minute period after her CVP catheter was open to air for several minutes. There is no evidence to support other possible causes, including myocardial infarction, pulmonary embolism of solid matter, aspiration, hemorhagic shock, or sepsis.

The physiologic abnormalities exhibited by this patient during the cardiac arrest illustrate the physiologic derangements described by English et al in experimental venous air embolism in dogs. Specifically, the abnormalities shown were increases in P(A-a)o₂, alveolar-arterial oxygen difference (A-a)o₂, and CVP, as well as hypotension and bradycardia. The prolonged cyanosis with a PaO₂ of 101 torr in our patient presumably reflected a low cardiac output with consequent oxygen desaturation of venous hemoglobin. The pulmonary edema that developed may also have been related to air embolism though other causes may have been involved. The hypercapnia and large A-aO₂, associated with air embolism are due to a "redistribution" of a major portion of ventilation to areas of high ventilation-perfusion ratio (V̅ₐ/ Q̅) and the coincident creation of areas of low V̅ₐ/ Q̅ in the remaining portion of lung. In addition, the time course for resolution of the hypotension and cyanosis in this case approached that described by Hlastala et al for the resolution of experimental venous nitrogen embolism in dogs.

The importance of securing vascular lines well is emphasized by this case, as is the importance of closely following and appropriately treating a patient’s fluid requirements. The major precipitating factor, however, was probably the development of a negative pressure gradient between the intrathoracic end of the CVP catheter and the end of the catheter which was open to the atmosphere. The negative intrathoracic pressure generated during inspiration with a partially obstructed upper airway or when a maximal inspiratory effort was made to call for help presumably resulted in a massive venous air embolism. This patient’s continuing hypovolemia probably further increased the negative pressure gradient between the two ends of the catheter. Air probably entered the catheter when the catheter and CVP line came apart; no blood was present on the bedsheets.

Once a patient who has suffered air embolism has lost effective cardiac output, reestablishment of an adequate cardiac output is of paramount importance. If a cardiopulmonary arrest has occurred, as in this patient, implementation of basic life support is essential. Appropriate advanced life support can then be given. If an arrest has not occurred, however, what action should be taken? Alvaran et al have shown that the left lateral decubitus position, external cardiac massage, and intracardiac aspiration of air via a venous catheter are all equally effective methods of resuscitation in terms of survival following air embolism in dogs. They also described, however, a shorter resuscitation time (2.7 minutes) with intracardiac aspiration of the air as compared to external cardiac massage (18.3 minutes) and a left lateral decubitus position (19.5 minutes). All of these maneuvers displace the right ventricular airlock from the right ventricular outflow tract allowing effective right heart filling and ejection of blood. External cardiac massage presumably pushes the airlock out of the heart into the pulmonary artery.

In summary, a patient appeared to suffer a large venous air embolism while supine in bed when her CVP line became disconnected at the catheter hub. Predisposing factors were hypovolemia and an increased negative intrathoracic pressure. The air embolism was large enough to precipitate a cardiac arrest. With the displacement of the right ventricular airlock by external cardiac massage the sequelae of a massive air embolism gradually resolved with the patient’s return to her prearrest state.

REFERENCES