

Fatal Massive Air Embolism during Transurethral Resection of the Prostate

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It is important for the anesthesiologist to be familiar with various complications associated with transurethral resection of the prostate in order to make a correct diagnosis and institute early treatment. To our knowledge, a fatal case of massive air embolism occurring under spinal anesthesia during a transurethral resection of a prostate has never been reported in the anesthesia literature. We report such a case occurring in a 55-yr-old previously healthy male.

CASE REPORT

The patient, a 55-yr-old, 85-kg male, previously in good health, was scheduled to undergo elective transurethral resection of a benign hypertrophic prostate. He had no history of drug allergies and was taking no medications. Admission laboratory values included a hematocrit of 42%, glucose 97 mg/dl, creatinine 1.1 mg/dl, and BUN 16 mg/dl. Preoperative ECG and chest x-ray were normal. One hour prior to the procedure, the patient was given atropine 0.4 mg im, meperidine 75 mg im, and hydroxyzine pamoate 100 mg im for sedation.

Upon arrival in the operating room, he was somewhat anxious. A 16-G intravenous catheter was placed, and administration of 1 l 5% dextrose in 0.25 N saline was begun *via* collapsible bag. Diazepam 5 mg iv was given for sedation. Blood pressure cuff and electrocardiogram were applied and vital signs recorded. With the patient in the left lateral decubitus position, a spinal anesthetic of 12 mg hyperbaric tetracaine *via* 22-G spinal needle was administered without difficulty. The patient was then turned and kept supine for 5 min before being placed in the lithotomy position without complication. Ten minutes after administration of the anesthetic, the sensory level stabilized at T6; blood pressure stabilized to 115/60 mmHg, and the pulse was 70 beats per min. An additional 5 mg diazepam iv was administered during the procedure.

With the patient well anesthetized, transurethral resection of the prostate was carried out through a constant flow resectoscope and with 5% glycine as an irrigating solution. Approximately 6 l irrigating solution had been used by the end of the procedure, which appeared to proceed uneventfully and which lasted approximately 30 min. The patient was comfortably sedated but easily arousable. After bleeding was controlled, the bladder was evacuated by irrigation with an Ellik

evacuator. At this time, a 3-way Foley catheter was placed, but no fluid was returned. The surgeon then irrigated the bladder through the Foley catheter, again without being able to recover the fluid instilled. Suspecting that the catheter had been obstructed by a blood clot, the surgeon, using a 60-cc syringe, attempted to "blow" the clot through the catheter situated in the urethra. He flushed multiple syringes of irrigating solution and air through the catheter but without fluid return. He then removed the Foley catheter, and pouring irrigating solution through it, discovered that it had not been plugged. The Foley catheter was reinserted and now functioned well: urine and irrigating solution flowed out of the bladder.

Vital signs then were taken and were found to be unchanged. The patient was easily arousable and comfortable. The monitors were removed and his legs were lowered as we prepared to lift him from the cystoscopy table. At this time, the patient looked up, took a gasping breath, and then ceased to breathe. He rapidly became cyanotic and was found to be pulseless. The patient's lungs were ventilated manually with 100% O₂ *via* face mask, and the trachea was intubated with an 8.0 mm-endotracheal tube as cardiopulmonary resuscitation was instituted. The monitors were reattached. A blood sample was taken from a vessel in the groin in which a palpable pulse was felt during external cardiac compressions and was sent to the laboratory. Twenty-five grams of glucose then was administered intravenously. The cardiac rhythm appeared to be sinus bradycardia, but no spontaneous pulses were detected, and no heart sounds were heard. The patient did not respond to pharmacologic therapy. Serum electrolytes and glucose were normal. Initial blood gases revealed a pH 7.28, PaCO₂ 84 mmHg, PaO₂ 42 mmHg, and O₂ saturation 70%. His pupils became fixed and dilated, and his cardiac status deteriorated to ventricular fibrillation.

Approximately 45 min after resuscitation was instituted and only a few minutes prior to its termination, one observer detected air in a vessel in the patient's groin by palpation during external cardiac massage. An x-ray of the abdomen at this time revealed the presence of massive amounts of air in femoral veins. Chest x-ray revealed the heart to be decreased in density, a condition believed to represent air in the heart.

At autopsy, the pathologist opened the heart and blood vessels under water in order to detect the presence of air. The right atrium and ventricle were grossly distended by air. An amount estimated by the pathologist to be greater than 1,000 ml air was found in the right atrium, right ventricle, and pulmonary artery, as well as in the inferior vena cava, its major branches, and the portal vein and its main branches. The site of entry of the air embolization could not be specifically identified. Multiple venous sinuses in the prostatic capsule were noted to have been transected, as would be expected during this type of procedure.

DISCUSSION

Venous air embolism is most commonly associated with neurosurgical procedures,¹ especially with patients in the sitting position. It can occur whenever the site of entry is higher than the heart and air can thus be drawn in by negative pressure. It is also possible theoretically for air to be pushed into an open channel with positive pressure.

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Recent attention has focused on procedures less commonly associated with air embolism, such as liver transplantation² and even surgical manipulation of a bone cyst. Two cases of air embolism occurring during transurethral resection of the prostate have been reported in the urologic^{3,4} literature, but none has previously been reported in the anesthesia literature. Although the cause and time of the entry of air into the circulation in this patient is unclear, we believe that the air entered at the end of the procedure when the Foley catheter was inserted. At this time, fluid could easily be instilled but not withdrawn. Multiple attempts at flushing what was believed to be a plugged catheter most likely introduced large amounts of air and fluid. The patient exhibited no manifestations of air embolism until the procedure had been completed and the operating room team prepared to move him from the cystoscopy table. This delay in signs of embolism occurred probably because the patient was in lithotomy position, such that the air most likely accumulated in his femoral vessels until his legs were lowered.

This report represents an extreme case of air embolism. Less spectacular air emboli may occur more commonly. Smaller emboli would result in increased pulmonary vascular resistance and heart failure with associated hypoxemia, hypotension, and mental confusion. Under such circumstances, air may be shunted to the systemic circu-

lation *via* a probe-patent atrial septal defect and result in a postoperative myocardial infarction or cerebral vascular accident. These symptoms can be attributed to the well-reported complications of prostatectomy, such as hyponatremia, volume overload, and glycine absorption. In the age group and population that usually undergoes prostatectomy, these complications are commonly attributable to underlying disease in combination with minor hypotensive episodes or volume abnormalities under anesthesia.

For this reason, we believe that the possibility of air embolism may be more common than anesthesiologists realize. We suggest that it be considered when cardiac problems arise during or after completion of this type of surgery.

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Intravenous Calcitonin Alleviates Spinal Anesthesia-induced Phantom Limb Pain

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CASE REPORT

Spinal or epidural anesthesia can temporarily exacerbate phantom limb pain (PLP) in patients with a prior lower extremity amputation.¹⁻⁷ Induction of PLP with regional anesthesia has been reported in patients who had been symptom-free as long as 40 yr⁴ and may occur during either onset¹⁻⁶ or regression⁷ of the block. Benzodiazepines,² barbiturates,⁶ and intravenous opioids^{1,2,4} have been tried with variable success in the treatment of acute anesthesia-induced PLP. This report describes a patient in whom PLP, induced by spinal anesthesia, ceased abruptly with a single dose of intravenous calcitonin.

A 79-yr-old, 47-kg man was scheduled for percutaneous laser angioplasty of his left superficial femoral artery. Despite treatment with aspirin and pentoxifylline the patient had developed an ischemic left foot. Two years previously, with spinal anesthesia, the patient had undergone right above-knee amputation for chronic osteomyelitis of the distal right femur. He had experienced PLP for approximately 2 weeks postoperatively but only occasional nonpainful phantom limb sensations thereafter. The patient's other medical problems included: 1) chronic obstructive pulmonary disease, treated with theophylline and aerosolized metaproterenol; 2) hypertension, controlled with diltiazem and topical nitroglycerin; 3) peptic ulcer disease, treated with Ranitidine; and 4) clinically stable Mobitz I atrioventricular (AV) block and probable old septal myocardial infarction. His physical examination and laboratory data were unremarkable except as consistent with his history.

Except for aspirin and pentoxifylline, on the morning of surgery the patient received his regular medications. In the operating room, routine monitors were applied, and a 20-G catheter was inserted into the radial artery. After 500 ml of lactated Ringer's solution was infused, with the patient in the sitting position, a 25-G spinal needle was introduced *via* the L3-4 interspace, resulting in transient paresthesias to both hips. The paresthesias, which were not referred to either the lower extremity or the phantom region, resolved immediately upon

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