Anesthesiology

Massive Pulmonary Embolism Following Tourniquet Deflation

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Intraoperative pulmonary embolism is an unusual complication of lower extremity surgery. We report two cases of cardiac arrest due to pulmonary embolism associated with pneumatic tourniquet deflation during knee surgery. The diagnosis in the second case was facilitated by use of tranesophageal echocardiography (TEE).

CASE REPORTS

Case 1. A 55-yr-old man underwent an outpatient arthroscopy after injuring the right knee 16 days prior to surgery. Past medical history was significant for ethanol abuse, heavy smoking, and degenerative joint disease. He was taking no medications and denied any allergies. Physical examination was unremarkable. Preoperative laboratory evaluation included normal electrocardiogram, chest x-ray, serum electrolytes, and platelet count. The hematocrit was 55%.

Intraoperative monitoring consisted of continuous electrocardiography, noninvasive blood pressure cuff, esophageal stethoscope, and temperature probe. Anesthesia was induced with intravenous thiopental and fentanyl. Tracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with N2O in O2 and isoflurane. Paralysis was maintained with atracurium. A pneumatic tourniquet was inflated around the right thigh, and surgery proceeded without incident. The tourniquet was deflated after 45 min of use. The anesthetic was discontinued, and muscle relaxant was reversed. The patient awakened, and his trachea was extubated.

Approximately 5 min after tourniquet deflation, the patient developed progressively cyanosis, tachycardia, and then bradycardia. His trachea was reintubated; his lungs were ventilated with 100% O2; and chest compressions were begun. Intravenous epinephrine, atropine, calcium, lidocaine and direct current (DC) cardioversion were given for asystole and subsequent ventricular tachycardia. An episode of atrial fibrillation was treated with cardioversion and intravenous verapamil. A sinus rhythm with a systolic blood pressure of 90 mm Hg was obtained using high-dose dopamine and phenylephrine infusions. The repeated arterial blood gases during the arrest were: pH 7.21, arterial CO2 tension (PaCO2) 96 mm Hg, and arterial O2 tension (PaO2) 76 mm Hg (FiO2 1.0).

The electrocardiogram showed a new incomplete right bundle branch block with significant ST segment depressions in the lateral leads. After transfer to the intensive care unit, pulmonary angiography revealed multiple large emboli in the right middle and lower lobe arteries.

Anticoagulation with heparin was accomplished by steady progressive improvement over the next several days. The patient was discharged from the hospital 17 days later receiving coumadin therapy with no apparent sequelae.

Case 2. A 63-yr-old woman was admitted for elective right total knee replacement. Past medical history included long standing hypertension, one episode of congestive heart failure secondary to uncontrolled hypertension, hypothyroidism, and rheumatoid arthritis, which had severely limited her activity. Preoperative medications were enalapril, nifedipine, prednisone, and thyroid replacement. Past surgical history included an uncomplicated left total knee replacement under general anesthesia 5 months prior to this admission. Venography was performed after this operation as part of a research study and revealed two small clots in the superficial veins of the left calf, for which treatment was not believed to be indicated. The patient denied any allergies. Physical examination was unremarkable. Blood pressure was 150/80 mm Hg. Preoperative laboratory evaluation included normal chest x-ray, electrocardiogram, serum electrolytes, prothrombin time, partial tissue thromboplastin time, and platelet count. An echocardiogram showed normal left ventricular function. The hematocrit was 31.5%. Preoperative noninvasive vascular studies were obtained as a result of the previous venogram findings and showed no evidence of deep venous thrombosis.

Intraoperative monitoring consisted of a radial arterial catheter, continuous electrocardiography, capnography, pulse oximetry, temperature probe, and esophageal stethoscope. Anesthesia was induced with intravenous etomidate and fentanyl followed by tracheal intubation facilitated with succinylcholine. Anesthesia was maintained with N2O in O2 and isoflurane. Paralysis was maintained with vecuronium. After inflation of a pneumatic tourniquet around the right thigh, surgery proceeded without incident.

Upon completion of the procedure, the tourniquet was deflated, after 90 min of use. Anesthesia was discontinued, and neuromuscular blockade was reversed. The patient regained consciousness with a blood pressure of 170/80 mm Hg, pulse 92 beats per min, oxyhemoglobin saturation 100%, and end-tidal CO2 tension (PETCO2) 35 mm Hg.

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Approximately 6 min after tourniquet deflation, the systolic blood pressure decreased to 70 mmHg and was followed by sinus bradycardia of 40 beats per min. P\textsubscript{ET}CO\textsubscript{2} to <5 mmHg. Intravenous atropine, ephedrine, and volume infusion were given. Controlled ventilation with 100% O\textsubscript{2} was resumed. Manual chest compressions were initiated upon loss of palpable pulses. After resuscitation with intravenous epi-
ephrine, lidocaine, bretylium, bicarbonate, and DC cardioversion, sinus tachycardia with a systolic blood pressure of 70 mmHg was ob-
tained with high-dose ephedrine and norepinephrine infusions. Partial cardiac pulmonary bypass via the femoral vessels was instituted to sustain a mean arterial pressure of 60–90 mmHg. Initial arterial blood gases obtained during the arrest were: \textit{pH} 7.016, P\textsubscript{aCO\textsubscript{2}} 73.8 mmHg, P\textsubscript{aO\textsubscript{2}} 181.7 mmHg (F\textsubscript{IO\textsubscript{2}} 1.0).

After restoration of a stable rhythm, the electrocardiogram showed new incomplete right bundle branch block and nonspecific ST segment changes. A TEE probe was passed and demonstrated normal left ven-
tricular contractility, right ventricular dilatation, and an intraluminal obstruction of the right pulmonary artery consistent with a large pul-
monary embolism (fig. 1). Subsequent portable techniun scanning demonstrated multiple perfusion defects in both lung fields. The patient
was given a total dose of 100 mg tissue plasminogen activator (TPA) via a pulmonary artery catheter. After further hemodynamic deteri-
oration, a median sternotomy was performed in preparation for open pulmonary embolectomy. However, the patient began to improve and no further procedures were undertaken.

After a prolonged period of stabilization and removal from bypass, repair of the femoral vessels, and closure of the chest, the patient was transferred to the intensive care unit while receiving infusions of nor-
ephrine, dobutamine, and amrinone. Hemodynamics improved markedly over the next 48 h, but the patient never regained con-
sciousness. She died 3 days later with severe anoxic encephalopathy. An autopsy demonstrated multiple pulmonary emboli and thrombosis of the right popliteal and tibial veins.

**DISCUSSION**

Deep venous thrombosis (DVT) and the attendant risk of pulmonary embolism is a well-known postoperative complication of lower extremity surgery: DVT can occur in up to 80% of patients undergoing total knee replace-
ment.\(^1\) Intraoperative pulmonary embolism associated with use of an occlusive tourniquet is an unusual event. The few previously reported cases share common features. Most patients were hospitalized and immobilized with a cast or traction.\(^2\)–\(^4\) Surgery followed an initial injury by several days to weeks. Embolism occurred upon limb exsanguination prior to tourniquet inflation in all cases except two, in which cardiac arrest occurred after tourniquet inflation without prior exsanguination.\(^5\)\(^,\)^\(^7\) All pa-
tients except one 14-yr-old died.

Our cases are unique in that embolization followed tourniquet deflation at the conclusion of surgery. Pulmonary embolism occurring at this time has not been reported, although one case of postoperative stroke due to paradoxical embolism suggested this circumstance.\(^8\) Our cases also are unique and disturbing given the relatively benign preoperative history in each. In the first case, the patient was living at home, was ambulatory, and was undergoing an outpatient procedure. In the second case, although there was a significant limitation in mobility due to the arthritic knee, the patient was living at home and had sustained no discrete injury. Indeed, preoperative noninvasive studies showed no evidence of DVT. Although routine postoperative anticoagulation therapy was to be instituted in case 2, neither patient had clear indications for preoperative DVT prophylaxis. In addition to preoperative trauma and immobilization, surgery itself and the effects of the tourniquet may contribute to the development of DVT.\(^9\) Thus, in case 2, at least, it appears that deep venous thrombosis was not present prior to sur-
gery and developed probably during the period of tourniquet ischemia.

The timing of cardiac arrest in our cases was significant because it made diagnosis difficult. Hypotension and metabolic acidosis are well-known complications of tourniquet

![Fig. 1. Images obtained using a 5.0-MHz single-plane transesophageal transducer (Hewlett Packard) demonstrate a large thrombus in the right pulmonary artery. One-centimeter intervals are indicated at the right screen margin.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931845/ on 05/29/2017)
ischemia and are attributed to washout of tissue metabolites, reactive hyperemia, decreased vascular resistance, hypovolemia, bleeding, and decreased level of stimulation. Therefore, arrhythmia, left ventricular dysfunction, and cardiac arrest may be secondary to coronary hypoperfusion. It can be very difficult clinically to distinguish hypovolemia, vasodilatation, and myocardial ischemia from the pulmonary hypertension and right ventricular failure associated with pulmonary embolism. Similarly, hypercarbia following tourniquet deflation can result from CO₂ washout from the reperfused extremity or can result from an increase in deadspace ventilation. A decrease in P₅₆CO₂ reflecting increased dead space occurs in pulmonary embolism but also can accompany a low cardiac output state from any cause. Pulse oximetry and capnography were not used routinely in our operating rooms at the time case 1 occurred. The presence of pulmonary embolism could be inferred only from the non-specific findings of a new right ventricular conduction delay on electrocardiogram and a widened alveolar–arterial oxygen tension gradient. Rapid and accurate diagnosis of pulmonary embolism is important given the high mortality and unique therapy required. TEE allowed a rapid diagnosis in case 2. Direct visualization of the embolism was obtained, as was evidence of right ventricular failure with preservation of left ventricular function. The sensitivity and specificity of TEE in the diagnosis of central pulmonary emboli has recently been demonstrated in a study in which 21 of 24 cases of TEE-identified central thrombi were confirmed by angiography or direct vision with no false negatives.* TEE has identified pulmonary embolization during hip replacement and liver transplantation when used as a continuous monitor. We have highlighted another application of this technology—acute intraoperative placement for diagnosis of hemodynamic instability produced by pulmonary embolism. The safety, availability and portability of TEE in the operating room offers distinct advantages over the standard diagnostic techniques of radionuclide perfusion scanning and pulmonary angiography.

In summary, we have described two cases of massive pulmonary embolism and cardiac arrest that occurred after deflation of a pneumatic tourniquet. In the second case, the diagnosis was facilitated by the use of TEE. When posttourniquet cardiovascular collapse occurs, serious consideration must be given to rapid, aggressive pulmonary embolism therapy, such as pulmonary embolectomy. These cases suggest that pulmonary embolism may play a role in producing less severe cases of posttourniquet hypotension. Further investigation is warranted to determine the true incidence of pulmonary embolism after tourniquet deflation and to evaluate the hemodynamic consequences of any embolic events.

REFERENCES
