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EPINEPHRINE-INDUCED PULMONARY EDEMA DURING ARTHROSCOPIC KNEE SURGERY

A CASE REPORT

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In recent reports, use of dilute epinephrine irrigation fluid has been recommended as a safe and effective way to improve visualization in arthroscopic knee and shoulder surgery^{1,2}. We are not aware of any reports, in the orthopaedic literature, of adverse reactions to epinephrine utilized in this manner. The anesthesiology literature contains a single case report of nearly fatal ventricular tachycardia in a patient undergoing shoulder arthroscopy with use of dilute epinephrine irrigation fluid. We present a case of nearly fatal cardiopulmonary collapse during arthroscopy of the knee in which dilute epinephrine irrigation fluid was utilized. The patient was informed that data concerning her case would be submitted for publication.

Case Report

A nineteen-year-old female college athlete underwent arthroscopy for reconstruction of the anterior cruciate ligament and transplantation of a medial meniscal allograft to treat chronic knee pain and instability. The patient had no relevant medical history, did not take any medications, and had a normal preoperative hemoglobin level and hematocrit. She had no family or personal history of cardiac, pulmonary, or endocrine abnormalities and no history of adverse reactions to general anesthesia. She stated that she did not use any overthe-counter supplements or medications.

General endotracheal anesthesia with midazolam, sufentanil, and propofol was administered uneventfully in the operating room. Inhalation anesthetics were not utilized, and no other medications were administered prior to induction. A pneumatic tourniquet was not used. After administration of 0.25% bupivacaine with epinephrine (1:100,000) in the subcutaneous tissue, standard anteromedial and anterolateral arthroscopic portals were made.

The arthroscopy was performed with utilization of a pressure-controlled pump system set at 40 mm Hg for irrigation with a dilute epinephrine solution, with the concentration of epinephrine calculated to be 0.3 mg/L (1.5 mL of 1:1000 [1 mg/mL] epinephrine mixed into 5 L of saline solution). The actual concentration of the epinephrine in the ir-

rigation fluid was not confirmed with laboratory testing, as this preparation is routinely mixed on a daily basis in a highvolume surgical center. The arthroscopic surgery proceeded uneventfully until preparation for the medial meniscal transplant was begun approximately fifteen minutes into the procedure. Approximately 12 L of irrigation fluid had been infused and an equal amount of outflow had been collected. As the tibial tunnel was being drilled in preparation for the posterior horn of the meniscal allograft, there was a sudden onset of severe hypertension and tachycardia with a blood pressure of 240/150 mm Hg and a pulse of 150 beats per minute. During this episode of hypertension, the patient's temperature was 36.1°C and the oxygen saturation was between 98% and 100%. A standard 50-mg single dose of esmolol, a short-acting beta-blocker, was administered to correct the hypertension and tachycardia. Immediately following administration of the esmolol, 700 mL of pink frothy secretion was seen to come from within the endotracheal tube, and the patient subsequently required increased ventilation pressures to maintain oxygenation. This clinical scenario was thought to be consistent with florid pulmonary edema, which was confirmed radiographically. The arthroscopic irrigation was discontinued, the procedure was aborted, and the portal sites were closed. Furosemide and hydrocortisone were immediately administered intravenously, and there was a rapid decrease of both the hypertension and the pulmonary edema. The patient was transferred to the surgical intensive care unit while on mechanical ventilation.

Once the patient was in the surgical intensive care unit, a cardiology consultation was obtained and the patient stabilized hemodynamically without further intervention. The patient was extubated later that same day, after clinical and radiographic resolution of the pulmonary edema. An echocardiogram demonstrated a decreased left ventricular ejection fraction of 30% with global hypokinesia; the ejection fraction improved to 60% on the day after the surgery. Measurement of serum cardiac enzyme levels revealed no evidence of ischemic damage. The electrocardiogram demonstrated a prolonged QT interval on the first postoperative day, despite

normal serum electrolyte levels, and this resolved without intervention the following day. Urine and serum catecholamine levels were within normal limits. All cardiac parameters returned to normal on the second postoperative day, and the patient was discharged home on the third postoperative day. By twelve weeks after the operation, the patient resumed light aerobic exercise without sequelae.

An attempt was made to determine the etiology of the pulmonary edema in order to ascertain the proper treatment of this patient, and the differential diagnosis of pulmonary edema in an anesthetized patient was entertained. Iatrogenic fluid overload was considered unlikely because a relatively small amount of intravenous fluid (1400 mL) had been administered to an otherwise healthy patient who had no cardiac disease. Negative pressure pulmonary edema should also be considered in such situations, but it is extremely unlikely to occur in a well-sedated patient. Congestive heart failure or cardiomyopathy should rank high on the list of possible diagnoses; however, this patient had no history of cardiac problems or symptoms and an echocardiogram made on the second postoperative day ruled out those potential causes of the pulmonary edema. Finally, an anaphylactic or Type-1 hypersensitivity reaction must be considered, but this patient did not manifest any of the other signs, such as dermatitis, angioedema, or bronchospasm, that are typically present in an anaphylactic reaction. In addition, no new medications had been administered just prior to the onset of symptoms, as the antibiotic was given prior to induction of anesthesia without complication. Therefore, excluding these possible causes of pulmonary edema and considering the immediate onset of symptoms just after the initial intraosseous drilling by the surgeons, we concluded that the cardiopulmonary compromise was attributable to the epinephrine contained in the arthroscopic irrigation fluid.

Discussion

Indirect evidence supporting use of a dilute epinephrine irrigation solution as an intra-articular hemostatic agent was found in two studies in which serum concentrations of bupivacaine were evaluated after intra-articular injection in the knee⁴⁵. Both studies demonstrated that the addition of epinephrine resulted in a reduction of bupivacaine absorption from the knee compared with that following administration of bupivacaine alone.

In a prospective, randomized, double-blind study, Olszewski et al. investigated irrigation with a dilute solution of epinephrine in saline solution (1 mg/L) delivered by gravity flow during routine knee arthroscopy. They reported that irrigation with the epinephrine solution resulted in a 50% decrease in tourniquet use compared with that in a placebo group, with no changes in heart rate or blood pressure attributable to the epinephrine. In a retrospective study of sixty-seven patients who underwent arthroscopically assisted ligament reconstruction, a satisfactory bloodless field was reported in thirty of thirty-seven patients treated with an intra-articular anesthetic and epinephrine (1:200,000). A pneumatic tourni-

quet was used for intra-articular hemostasis in the remaining thirty patients, and the authors reported no difference in the mean operative time between that group and the group treated with epinephrine. Furthermore, there were no identifiable complications in either group.

A prospective, randomized, double-blind, placebocontrolled study of the effects of irrigation with a dilute epinephrine solution (0.33 mg/L) on intra-articular hemostasis and adverse cardiovascular reactions in fifty-four patients undergoing shoulder arthroscopy demonstrated a reduction in intra-articular bleeding and improved visualization². Furthermore, the authors reported no elevation in blood pressure or heart rate and no adverse cardiovascular reactions in any of the study patients.

We found no reports of adverse cardiopulmonary effects of epinephrine in the English-language orthopaedic literature, even though such effects might be expected from an agent with such potent vasoconstrictive properties. However, the otolaryngology literature contains several case reports of cardiovascular compromise after local administration of dilute epinephrine solutions (1:100,000 to 1:500,000) during surgical procedures7-9. In a recent anesthesiology publication, it was reported that the New York State Department of Health had formed the Phenylephrine Advisory Committee to investigate the morbidity and mortality associated with topical vasoconstrictors frequently used in otorhinolaryngological surgery10. This committee of physicians reviewed state reports of intraoperative morbidity over a seven-year period and identified nine patients in whom severe hypertension, tachycardia, and pulmonary edema developed after administration of topical phenylephrine, a potent alpha-adrenergic vasoconstrictor. Three of the nine patients had fatal cardiac arrest. This constellation of cardiopulmonary symptoms was identified in all of the otorhinolaryngology case reports⁷⁻¹⁰, as it was in the case presented here. The pattern consists of an abrupt onset of hypertension, tachycardia, and pulmonary edema after local administration of epinephrine. The mechanism is likely activation of alpha-adrenergic receptors. Alphaadrenergic stimulation by epinephrine increases peripheral vascular resistance, which in turn increases the left ventricular filling pressure. This process shifts blood volume from the peripheral circulation into the pulmonary vasculature, which is less sensitive to the vasoconstrictive effects of epinephrine. The physiological consequences manifest as a severe and abrupt increase in blood pressure, depressed myocardial contractility, and mild pulmonary edema. The normal physiological compensatory mechanism to preserve cardiac output is to increase heart rate and/or cardiac contractility. Beta-blockers inhibit these compensatory mechanisms, exacerbating the detrimental effects of alpha-adrenergic stimulation. The resultant diminished cardiac output may be demonstrated by echocardiography, as it was in our patient, and pulmonary edema may ensue. Consequently, as recommended by the Phenylephrine Advisory Committee, beta-blockers should be avoided in this situation. The treatment of epinephrineinduced hypertension and tachycardia should consist of selecTHE JOURNAL OF BONE & JOINT SURGERY · JBJS.ORG VOLUME 85-A · NUMBER 5 · MAY 2003 EPINEPHRINE-INDUCED PULMONARY EDEMA DURING ARTHROSCOPIC KNEE SURGERY

tive alpha-adrenergic blockers and potent vasodilators such as phentolamine and nitroprusside, respectively. The pulmonary edema responds well to supportive mechanical ventilation and diuretics such as furosemide.

On the basis of the recurring pattern of symptoms reported in the literature, it appears that the inciting event for this potentially disastrous complication of arthroscopic surgery may be a combination of an increased alpha-adrenergic agonist response to epinephrine^{7,8} and rapid access to the venous circulation through an intraosseous route. The anesthesiology literature contains a single case report³ of nearly fatal ventricular tachycardia in a patient undergoing shoulder arthroscopy with use of dilute epinephrine irrigation fluid (1.0 mg/L). The ventricular tachycardia occurred immediately after the posterior humeral cortex was penetrated. The author postulated that intraosseous infusion of epinephrine resulted in the adverse cardiovascular reaction. Our patient had severe cardiopulmonary compromise immediately after the initial drilling of the intraosseous tibial tunnel, which again suggests that the epinephrine gained rapid access to the venous circulation through an intraosseous route. It does not appear that routine arthroscopy results in unsafe plasma levels of epinephrine through the absorption of arthroscopic irrigation fluid. Jensen et al.2 measured serum epinephrine levels during routine shoulder arthroscopy in a randomized, placebocontrolled study. They found statistically similar elevated levels of serum epinephrine in both the epinephrine group and the placebo group after arthroscopy, indicating that the elevated levels reflected a systemic reaction to the surgery and not absorption of irrigation fluid.

In the present report, we have described a rare but potentially devastating complication of arthroscopy performed with use of dilute epinephrine irrigation fluid. Both orthopaedic surgeons and anesthesiologists should be familiar with

this pattern of symptoms in patients undergoing epinephrineassisted arthroscopy. Once the complication has been recognized, it is essential to institute proper and timely treatment consisting of discontinuation of the irrigation and administration of a potent peripheral vasodilator with symptomatic treatment of the ensuing pulmonary edema. In addition, prevention of this disastrous complication is of paramount importance. A thorough history and physical examination should be an integral part of the preoperative evaluation of every patient, with particular attention paid to cardiovascular history or previous experiences with anesthesia. Every effort should be made to ensure proper preparation of the epinephrine irrigation fluid so that an inappropriate dose of epinephrine is not administered. Finally, there should be clear communication between the orthopaedic surgeon and the anesthesia team during the procedure, and the anesthesiologist should be notified prior to any osseous drilling.

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