Case 2011-2 – Local Anesthetic Toxicity

A 48 year old man presented to the O.R. for a right total knee arthroplasty. In the pre-induction holding area, a right femoral nerve catheter was placed to facilitate postoperative pain management. Ultrasound was used to localize the right femoral nerve, and 20cc of 0.5 percent ropivacaine was injected into the nerve sheath under ultrasound visualization. Good spread of local anesthesia was seen. Following injection, a catheter was threaded through the needle and secured.

The patient was taken to the OR and underwent an uneventful surgical procedure under spinal anesthesia. In the PACU an infusion of 0.1 percent ropivacaine at 10 ml/hour was begun through the previously placed femoral nerve catheter. Twenty-five minutes later the patient became somnolent and had a brief episode of seizure-like activity. Blood pressure declined to 70/40, and heart rate fell from 65 to 52. Upon questioning, the patient complained of perioral numbness and a metallic taste in his mouth.

Discussion

The patient’s symptoms are consistent with local anesthetic toxicity. Given the lack of symptoms seen with the initial block, this must be the result of the 4 mg of ropivacaine the patient has received in the PACU (25 minutes at 10 ml/hr of a 0.1 percent solution). The relatively small dose and slow infusion rate suggests that the most likely explanation is direct intravascular injection. Appropriate actions at this point might include:

- Immediate cessation of the ropivacaine infusion.
- Administration of a vasopressor to support hemodynamics.
- Administration of a bolus of 1.5 mL/kg of a 20% fat-emulsion solution, followed by continuous infusion at a rate of 0.25 mL/kg/min until 500 mL has been infused. (ASRA Practice Advisory on Local Anesthesia Systemic Toxicity. (Accessed online at journals.lww.com/rapm/Fulltext/2010/03000/ASRA_Practice_Advisory_on_Local_Anesthetic.7.aspx).
- Inspection of the femoral catheter, the infusion line, the pump, and the infusion bag itself.

No prospective, randomized studies have examined the therapeutic benefit of fat emulsion therapy for local anesthetic toxicity in humans. The utility of this therapy is based on animal data and on case reports and small series. Given that cardiac dysrhythmia from local anesthetic toxicity is frequently fatal, and that fat emulsion solution is relatively benign, the risk:benefit ratio of this approach favors early administration whenever toxicity is suspected. Rapid access to a volume of this solution in any unit where regional anesthesia is provided is an emerging national standard of care, equivalent to keeping a supply of naloxone on hand when intravenous opiates are administered.

Further care of the patient is supportive, and based on response to initial therapy. Further seizures, significant dysrhythmia, or deteriorating hemodynamics might necessitate intubation and mechanical ventilation. Close monitoring will be required for 12-24 hours, which may mean an extended PACU stay or transfer to an intensive care unit. If the symptoms do not progress – likely if no further intravenous ropivacaine is administered – then an alternate approach to postoperative pain management will be required. Finally, and despite a presentation which strongly suggests local anesthetic toxicity, it is always possible that the diagnosis is wrong or incomplete. The treating anesthesiologist must remain vigilant for other causes of postoperative hemodynamic instability, including bleeding, pulmonary embolus, myocardial ischemia or allergic reaction.

“...This case report suggests that an easily visualized single-shot nerve block via needle may not mean that a catheter placed through the needle will remain in the correct space.”
Case Report From the Anesthesia Incident Reporting System

Continued from page 28

Once the patient’s clinical stability is assured, the orthopedic surgeon, patient and family should be informed of these events and the changes in care which will result. An effort should be made to discover the specific cause of the complication. Repeat ultrasound may suggest an intravascular catheter, as might aspiration of blood or a heart rate elevation following a small dose of epinephrine. If these investigations are negative then the pump should be checked for accurate performance, and the infusion bag assayed for the correct concentration of ropivacaine. Other potential sources of systemic local anesthetic should be excluded (e.g. a syringe swap with an intravenous medication).

Clinical Follow Up

The ropivacaine infusion was stopped. An intravenous fluid bolus and a single dose of phenylephrine restored the blood pressure to 120/60. 140 ml of intralipid and 1 mg of midazolam were administered. The patient’s symptoms, including his mental status, improved rapidly.

The femoral nerve catheter was aspirated, but no blood was seen. A test dose of 15 mcg of epinephrine caused an immediate increase in heart rate from 55 to 78. The catheter was removed from its presumed intravascular location. The patient was monitored overnight in the PACU, and postoperative pain was managed with intravenous patient-controlled analgesia. Subsequent recovery was uneventful.

The Last Word

This case report suggests that an easily visualized single-shot nerve block via needle may not mean that a catheter placed through the needle will remain in the correct space. Migration of the needle tip prior to catheter placement is a possibility, as is vascular penetration by the catheter itself. Aspiration of blood from the catheter suggests intravascular placement, although negative aspiration does not exclude this possibility because the force of aspiration may collapse a small vein. Vigilance is required, especially if dosing of the catheter does not occur for several hours after initial placement. In this case, it may have been prudent to administer a test dose through the catheter prior to starting the local anesthetic infusion. This lapse led to a potentially serious complication, although ongoing monitoring identified the problem in time for successful rescue. This case illustrates how rapidly serious complications can develop and the importance of appropriate monitoring in the prevention of adverse patient outcomes.

We Have Come a Long Way, Baby!

Continued from page 36

cytokines in the pathophysiology of disease, mechanisms involved in the pathophysiology of acute lung injury, the role of ion channels in acute injury, biomarkers of several perioperative or acute care outcomes, hyperbaric therapy, stem cell therapy and metabolomics.

Furthermore, in the most recent Residents’ Essay Contest submissions, only two of the articles submitted for judging could be construed as pharmacology-oriented. The non-pharmacology oriented essays also included several studies on cytokine responses, as well as the pathophysiology of sepsis, ischemia-reperfusion and obstructive sleep apnea. Interestingly, all the patient-oriented research submitted to the contest was outcome-based.

Clearly, investigators have expanded the scope of scientific inquiry in the specialty of anesthesiology. We have come a long way from the days of primarily focusing on drug action in the perioperative period and ventilator management. Whole new fields (genomics, proteomics and metabolomics) that were not even around when I was a resident are now finding their way into the research being conducted by anesthesiology departments. This is exciting, and for one who has been “around the research block” several times very satisfying. Hopefully, these areas of focus will open new innovative research than can improve and expand the practice of anesthesiology in all its subspecialties.