Editorial Comment: Cardiac Arrest from Local Anesthetic Toxicity After a Field Block and Transversus Abdominis Plane Block: A Consequence of Miscommunication Between the Anesthesiologist and Surgeon *AND* Probable Local Anesthetic Systemic Toxicity in a Postpartum Patient with Acute Fatty Liver of Pregnancy After a Transversus Abdominis Plane Block

Two case reports by Naidu and Richebe,[1] and Scherrer et al.[2] highlight the risk of local anesthetic systemic toxicity (LAST) after transversus abdominus plane (TAP) block. In the Naidu and Richebe report, a parturient with acute fatty liver of pregnancy received bilateral TAP blocks 15 hours apart for postcesarean delivery analgesia.[1] Thirty minutes after the second block with 0.375% bupivacaine (40 mL [150 mg], 20 mL per side), she had a seizure. Although other etiologies of seizure were possible in this sick patient, the temporal relationship of the seizure to the TAP block suggests LAST may have been responsible for the seizure. In the report by Scherrer et al.,[2] a woman received intraperitoneal infiltration with ropivacaine 0.75%, 20 mL (150 mg) by the surgeon at the conclusion of a laparotomy. Forty-five minutes later, the anesthesiologist performed bilateral TAP blocks with 0.75% ropivacaine (40 mL [300 mg], 20 mL per side). Ten minutes later, the patient had a seizure followed by bradycardia and asystole. The TAP blocks in both patients were performed using ultrasound guidance, making an unintentional intravascular injection unlikely. Both patients were relatively small (weights 51[1] and 57[2] kg).

Recent investigations suggest that plasma levels of local anesthetic after a TAP block are within the range that might cause LAST. Griffiths et al.[3] reported plasma concentrations of ropivacaine in 30 parturients receiving bilateral TAP blocks after cesarean delivery (total ropivacaine dose 2.5 mg/kg in 40 mL, 20 mL per side). The highest individual plasma ropivacaine concentration was 3.76 μg/mL measured 10 minutes after the TAP block. Twelve patients had ropivacaine concentrations in the first hour exceeding 2.2 μg/mL, the widely quoted threshold for LAST, and 3 patients had mild symptoms of LAST. The peak plasma level occurred at 30 minutes. In contrast, in 8 men volunteers (mean weight 81 kg) receiving bilateral TAP blocks with 0.375% ropivacaine (total 60 mL, 225 mg), all plasma ropivacaine concentrations were <2.2 μg/mL.[4] Similar to the Griffiths et al.[3] study, the mean (± SD) peak plasma concentration occurred at 35 ± 7 minutes.[4] In a third study, Kato et al.[5] performed bilateral TAP blocks in 12 women (mean weight 55 kg) undergoing gynecologic procedures under general anesthesia. The blocks were performed with 1% lidocaine (40 mL [400 mg], 20 mL per side) after induction of anesthesia. Similar to the previous studies, the mean peak plasma concentration occurred at 30 minutes (range 15–60 minutes). The mean (± SD) maximum plasma concentration was 3.6 ± 0.7 μg/mL (range 2.7–5.5).[5] Similar to the findings with ropivacaine, the lidocaine concentration levels measured in some study subjects were within the concentration range in which mild LAST may be observed (5–10 μg/mL).[6]

These recent studies[3,5] suggest that local anesthetic absorption from the TAP is significant, and even the use of "usual" doses can result in plasma levels which approach or exceed levels at which mild LAST is observed in some individuals. The risk for toxicity may be greater in women or those of small stature. The case reports confirm that the safety margin is low. The patient in the Naidu and Richebe report[1] may have had altered protein binding or slowed metabolism because of her liver disease, leading to prolonged plasma levels of free bupivacaine. The patient in the Scherrer et al. report[2] inadvertently received a double dose of ropivacaine because both the surgeon and anesthesiologist injected ropivacaine within a 45-minute interval. The studies and reports suggest that patients should be observed in a monitored setting for at least 45 minutes after a TAP block is performed. Doses may need to be adjusted, particularly in smaller adults. Ropivacaine may be a safer drug than bupivacaine for this indication.

Accepted for publication August 6, 2013.

DOI: 10.1097/ACC.0b013e3182a88776

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Cardiac Arrest from Local Anesthetic Toxicity After a Field Block and Transversus Abdominis Plane Block: A Consequence of Miscommunication Between the Anesthesiologist and Surgeon

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We report the case of a 25-year-old female scheduled for laparoscopic gynecologic surgery under general anesthesia. At the end of laparoscopy, an intraperitoneal infiltration (ropivacaine 0.75%, 20 mL) was administered by the surgeon without informing the anesthesiologist. After tracheal extubation due to significant postoperative pain, the anesthesiologist performed a bilateral transversus abdominis plane block (ropivacaine 0.75%, 40 mL). A seizure followed by ventricular arrhythmia developed 10 minutes after local anesthetic injection. An infusion of 20% lipid emulsion was successful in converting the ventricular arrhythmia to a sinus rhythm. This overdose could have been avoided with better communication between anesthesiologist and surgeon. (A&A Case Reports 2013;1:75–6)

To enhance postoperative pain control, increased use of local anesthetics and regional analgesia is frequently advocated.[1] However, whereas local anesthetics are used by anesthesiologists, infiltration with local anesthetics may also be used by surgeons. For example, after laparoscopic procedures, infiltration of the peritoneum and trocar sites may be performed to reduce postoperative pain. Although most of the time anesthesiologists are apprised of local anesthetic administration by surgeons, a failure to communicate this may have dramatic consequences. We describe, after obtaining patient consent to publish the report, a case involving local anesthetic systemic toxicity (LAST) resulting in cardiac arrest directly related to a lack of communication between the anesthetic and surgical teams at the end of surgery.

CASE DESCRIPTION

A 25-year-old woman (57 kg, body mass index = 19.7 kg/m^2), ASA physical status I, presented for laparoscopic exploration after several episodes of salpingitis. Induction and maintenance of general anesthesia (propofol, remifentanil, and atracurium) proceeded uneventfully. Just before the end of the surgical procedure, the surgeon performed an intraperitoneal infiltration with 20 mL ropivacaine 0.75% without informing the anesthesiologist. Peritoneal infiltration by surgeons is not a current procedure for this type of surgery in our center.

The trachea was extubated after muscle relaxant reversal, and the patient was discharged to the recovery room. Acetaminophen (1 g), tramadol (100 mg), and morphine (5 mg) were administered IV for pain relief. However, 45 minutes after the ropivacaine peritoneal infiltration by the surgeon, the patient complained of severe pain (visual analog scale >60 mm) and the anesthesiologist performed an ultrasound-guided bilateral transversus abdominis plane (TAP) block (100-mm 22-G regional block needle, a Nerve S ultrasound with a 12–8 MHz linear probe) using 150 mg ropivacaine on each side (ropivacaine 0.75%, 20 mL).

Ten minutes after the TAP block, a clonic seizure occurred and after manually ventilating the lungs using 100% oxygen, thiopental 500 mg was given IV and orotracheal intubation was performed. Two minutes later, the patient developed a severe bradycardia rapidly followed by asystole seen in the electrocardiogram. Cardiopulmonary resuscitation was initiated and the patient was given a bolus of 250 mL lipid emulsion 20% followed by a continuous infusion of 10 mL/min, which was immediately followed by a restoration of cardiac activity. The patient regained consciousness 15 minutes later and exhibited no further signs of cardiac toxicity. She was discharged home on the second postoperative day without physical sequelae.

DISCUSSION

In this case, LAST was observed as a direct consequence of the peritoneal infiltration of ropivacaine (150 mg) injected by the surgeon and the addition, 45 minutes later, of ropivacaine (300 mg) administered as a TAP block postoperatively by the anesthesiologist. After intraperitoneal administration of ropivacaine at doses varying from 100 to 300 mg, her mean Cmax ranged from 0.66 to 3.76 μg/mL and mean Tmax ranged from 15 to 35 minutes.[2] Using ropivacaine, Griffiths et al.[3] reported a mean serum concentration of 2.54 mg/mL, 30 minutes after an injection of 3 mg/kg of ropivacaine during a bilateral TAP block. While our ropivacaine dose of 5.2 mg/kg used for TAP block was higher, it was still within French guidelines which recommend a maximum dose

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Accepted for publication April 5, 2013.

Funding: Not funded.

The authors declare no conflicts of interest.

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DOI: 10.1097/ACC.0b013e3182973a3f

of 300 mg ropivacaine for peripheral block.[4] However, the total ropivacaine dose of 450 mg (7.9 mg/kg) was far larger than the maximum recommended dose.

Only 2 cases of LAST due to ropivacaine overdose after TAP block have been reported. In the first case, 40 mL of 0.375% (150 mg) ropivacaine was injected during ultrasound-guided TAP block under general anesthesia at the end of a laparoscopic myomectomy of the uterus. Anesthesia ended uneventfully but 3 hours after TAP block, the patient displayed systemic clonic seizure associated with hypotension, interpreted as LAST. A rapid recovery occurred after 20% lipid emulsion administration.[5–7] In the second case, Benhamou et al.[8] reported LAST occurring a few minutes after injection of 225 mg ropivacaine for a bilateral TAP block which was reversed in 2 minutes after 100 mL of 20% lipid emulsion administration. The rapid onset of toxicity was related to technical difficulties leading to direct IM local anesthetic injections.

In our case, this ropivacaine overdose would have been avoided with better communication between surgeons and anesthesiologists. As stated by de Vries et al.,[9,10] in a systematic review including studies from several developed countries, 1 in every 150 patients admitted to a hospital dies as a consequence of an adverse event and that almost two thirds of in-hospital events are associated with surgical care. In recognition of the disproportionate number of such events that are associated with surgical care, several interventions have been proposed to increase patient safety, including improving the quality of teamwork in the operating rooms.[11] Moreover, the use of a perioperative surgical safety checklist has been associated with a reduction in major complications.[12] Before the patient leaves the operating room, this checklist includes a review by the surgeon, anesthesia professionals, and nurses on the key concerns for recovery and management of the patient. However, the standardization of medical processes should not be limited to the operating room. Several studies have shown that the majority of errors occur outside the operating room, before or after surgery.[13,14] The implementation of a multidisciplinary checklist (Surgical Patient Safety System; SURPASS) that follows the surgical pathway from admission to discharge is associated with a reduction in surgical complications and mortality in hospitals with a high standard of care.[10] Instructions concerning medication (including pain medication) are required. Thus, with the increase in local anesthesia and regional anesthetic procedures, communication between the surgical and anesthesia teams is critical to avoid life-threatening complications.

ACKNOWLEDGMENTS

We are grateful to Nikki Sabourin-Gibbs, Rouen University Hospital, for her help in editing the manuscript.

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Probable Local Anesthetic Systemic Toxicity in a Postpartum Patient with Acute Fatty Liver of Pregnancy After a Transversus Abdominis Plane Block

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We present the case of a 25-year-old woman with acute fatty liver of pregnancy, a rare mitochondrial disorder that manifests during pregnancy and has a significant mortality rate. Postoperative pain management is challenging for myriad reasons. With the increasing application of transversus abdominis plane blocks for postcesarean delivery analgesia, we describe the real and potential complications of this method of regional analgesia in patients with this disease. (A&A Case Reports 2013;1:72–4)

Acute fatty liver of pregnancy (AFLP) is a rare mitochondrial disease that manifests in the latter stages of gestation with potentially fatal consequences to both mother and child. According to a 2008 United Kingdom cohort study, the incidence of AFLP is 5.0 per 100,000 maternities.[1] Although the mortality rates have much decreased since the disease was described in 1940 by Sheehan, the estimated maternal mortality is still approximately 10%.[2] AFLP appears in the third trimester of pregnancy and appears to be a result of the build-up of long-chain 3-hydroxyacyl metabolites from inherited maternal defects in long-chain 3-hydroxyacyl coenzyme A dehydrogenase. The metabolites accumulate leaving microvesicular infiltrates or steatosis in hepatocytes that will likely result in severe hepatic dysfunction. There is no medical treatment of AFLP; delivery of the fetus abates the disease.

Postoperative pain control is an important aspect of patient care that is particularly challenging in these patients. Because of the pathology, clinicians are advised not to give opioids which can worsen mental status when metabolism and excretion is impaired. Acetaminophen is relatively contraindicated given the acute and severe hepatic dysfunction. Nonsteroidal anti-inflammatory drugs are also relatively contraindicated because of platelet aggregation inhibition in the postsurgical setting, as well as the likelihood of acute kidney disease often seen with AFLP. In 1 case, inherent coagulopathy was a major risk factor for the development of epidural hematoma with neuraxial anesthesia.[3]

Transversus abdominis plane (TAP) block is a suitable method of postoperative analgesia after cesarean delivery to avoid the toxicities and side effects related to acetaminophen, nonsteroidal anti-inflammatory drugs, and opioids. McDonnell et al.[4] reported a morphine-sparing effect in women who benefited from a bilateral TAP block after cesarean delivery. In this case report, we present complications associated with TAP blocks in a woman with severe AFLP.

Consent was obtained from the patient for publication of this case.

CASE REPORT

A 25-year-old G2P0 woman at 37 0/7 weeks of gestation presented in labor with cervical dilation of 4 cm. Her symptoms included a history of 3 weeks of nausea/vomiting, somnolence, mild hypertension (140/85 mm Hg), and clinical jaundice. She was diagnosed with hyperemesis gravidarum at an outside hospital 2 weeks before her admission to our unit. Although she comprehended questions and was oriented to situation, time, and place, she was lethargic and was a grade 2 per the West Haven Criteria for hepatic encephalopathy.[5]

The patient weighed 51 kg, and her height was 150 cm (body mass index = 22.7 kg/m^2). Initial laboratory tests showed white blood count 20,000/μL, hematocrit 41%, platelets 141,000/μL, international normalized ratio 4.1, fibrinogen 50 mg/dL, a 2+ proteinuria, elevated creatinine 2.1 mg/dL, hypoglycemia 48 mg/dL, direct bilirubinemia 9.0 mg/dL, aspartate aminotransferase/alanine aminotransferase 59/63 U/L, alkaline phosphatase 1000 IU/L, and ammonia 101 mg/dL.

The initial differential diagnosis for this patient included preeclampsia, HELLP syndrome, and AFLP. Given the clinical presentation and laboratory values, AFLP was the lead diagnosis.

After stabilizing the coagulopathy with cryoprecipitate and fresh frozen plasma, an urgent cesarean delivery was necessary because of late decelerations on fetal heart monitoring and the mother's tenuous status. General anesthesia was provided with a rapid sequence induction with fentanyl (150 mcg), propofol (100 mg), lidocaine 2% (60 mg), and succinylcholine (70 mg). Intubation with a 6.5 oral endotracheal tube and a Macintosh #3 laryngoscope blade while holding cricoid pressure was uneventful. Maintenance of anesthesia involved isoflurane and then a transient administration of nitrous oxide. At the end of the cesarean delivery, the trachea was extubated and she was taken to postanesthesia care unit. The child was a healthy male, with an APGAR (5 minutes) of 8, weighing 2879 g.

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Accepted for publication April 4, 2013.

Funding: The authors received no funding for this submission.

The authors declare no conflicts of interest.

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DOI: 10.1097/ACC.0b013e3182973a2f

Due to her fluctuating mental status, opioids for postoperative analgesia were withheld. She was able to follow commands and communicate, but was intermittently lethargic, similar to her predelivery status. After numerous options were considered, it was decided that a TAP block would be optimal in this particular situation. Using ultrasound guidance (Sonosite, Bothell, WA), 20 mL of bupivacaine 0.375% was injected on each side 3 cm anteromedial to the point midway between the iliac crest and costal margin along the midaxillary line using a 5-cm needle (B-Braun Medical USA, Bethlehem, PA). The international normalized ratio was 2.1, and fresh frozen plasma was given during the procedure. Pain scores decreased from 9 of 10 down to 3 of 10 twenty minutes after the procedure and remained less than 5 of 10 for 12 hours. The patient was transferred to the intensive care unit (ICU) for closer postoperative monitoring and frequent neurological assessment.

After 12 hours, the patient began to complain of increasing pain in the vicinity of her abdominal incision. We agreed to perform a second TAP block given the time interval since the first block, and because of the relief the first block achieved. Fifteen hours after the first TAP block, a second TAP block with the same doses was uneventfully provided. Her visual analog scale score decreased from 10 of 10 to 6 of 10 five minutes after the block.

Thirty minutes after the TAP block a seizure was observed; lorazepam 1.5 mg IV was administered. The trachea was intubated after administration of propofol and rocuronium. Intralipid 20% (1.5 mL/kg) was given IV to prevent bupivacaine cardiotoxicity. No electrocardiogram interval changes were noted, and she remained hemodynamically stable through her entire period in the ICU. While intubated, she was initially receiving propofol infusion without any opioids. Twelve hours after her seizure, it was agreed that a remifentanil infusion at 0.1 mcg/kg/min would be the safest analgesic option in this complex patient. Her trachea was extubated within 48 hours of intubation; on her first sedation holiday, her mental status was deemed to be unsuitable to protect her airway. On hospital day #4, she was transitioned to a remifentanil patient-controlled analgesia: she was pleasant, conversing, and had no recall of the events after her delivery. Her pain score was 1 to 2 of 10. She was discharged from the ICU on hospital day #6. She continued to progress back to her functional baseline at the follow-up at 3 months after her cesarean delivery.

DISCUSSION

The differential diagnosis for the seizure included eclampsia, posterior reversible encephalopathy syndrome, hypomagnesaemia, hypoglycemia, hepatic encephalopathy, intracranial bleeding, and, due to the temporal relationship of events with the seizure occurring within 30 minutes of a second TAP block, local anesthetic systemic toxicity (LAST). Tsen et al.[6] reported that parturients, in general, may be at higher risk for LAST because of an increased free fraction of plasma bupivacaine. In AFLP, third trimester liver dysfunction leads to a decreased production of serum proteins that bind to local anesthetics such as bupivacaine. Bupivacaine, in particular, is approximately 95% protein-bound. Therefore, in patients with decreased serum albumin and α-1-glycoprotein, the free fraction of bupivacaine is augmented making an individual more susceptible to LAST. Our patient's impaired renal function also contributed to a decreased threshold for LAST.

Pharmacokinetics of other local anesthetics have been studied in patients receiving TAP blocks. Kato et al.[7] described peak plasma concentrations 15 minutes after 400 mg of lidocaine via bilateral TAP blocks; Griffiths et al.[8] noted peak plasma concentrations of ropivacaine at 30 minutes after injection for bilateral TAP blocks. Although no formal studies have been performed with bupivacaine, the onset to LAST in this patient is within the range of onset for other amide local anesthetics.[9]

The interval between the first and second blocks was 15 hours. The estimated terminal half-life of bupivacaine is 2.7 ± 2 hours per AstraZeneca, the pharmaceutical company that makes bupivacaine in the United States (Astra insert). In a study of bupivacaine pharmacokinetics after axillary blocks, the terminal half-life for bupivacaine was 11.5 hours.[10] Although we do not have data regarding the terminal half-life of bupivacaine in TAP blocks, we would usually wait 5 half-lives for elimination of the drug; given this patient's condition, 15 hours might have been not enough time to expect >96% metabolism and excretion. Further studies involving the pharmacokinetics of sequential TAP blocks would help elucidate the appropriate interval.

The TAP block has been recently described as a safe and useful postcesarean analgesic modality. In this patient, however, the threshold for local anesthetic toxicity was likely reduced and while providing a second TAP block to provide analgesia seemed appropriate, this resulted in LAST. To our knowledge, this is the first report of the use of Intralipid 20% (1.5 mL/kg) to treat bupivacaine toxicity after a repeat TAP block. Future studies should be conducted on the pharmacokinetics of local anesthetics in TAP blocks in parturients. Providing good analgesia after cesarean delivery in patients with AFLP remains a challenge; decreased liver and renal function, encephalopathy, coagulopathy, and increased local anesthetic toxicity are complicating factors.

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