Local and Regional Anesthesia

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CASE REPORT

Foot Drop Following a Popliteal Sciatic Nerve Block with Ropivacaine, A Case Report and Literature Review

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Abstract: Although peripheral nerve blocks are deemed very safe, a significant number of patients for whom this anesthetic technique may be particularly appealing to apply may present with preexisting peripheral neuropathies, putting them at risk for further nerve damage. We present a case with a 74-year-old male with several risk factors for peripheral neuropathy who developed a foot drop following a popliteal sciatic nerve block with ropivacaine. We suggest that the vasoconstrictive properties of ropivacaine may have contributed to a preexisting neuronal ischemia, thus further damaging an already compromised nerve.

Keywords: peripheral nerve block, peripheral neuropathy, vasoconstriction, local anesthetics

Introduction

Peripheral nerve blocks (PNB) offer good operating conditions whilst having a minimal impact on the central nervous system and the patient's cardiovascular and respiratory physiology. As such, they are a favored method for patients with cardiovascular or pulmonary disease undergoing orthopedic surgical procedures. However, a significant number of patients with such comorbidities may present with neuropathies.[1,2] Neuropathic nerves respond to local anesthetics differently, potentially rendering them more vulnerable to injury.[2]

Although considerations pertaining to regional anesthesia in patients with neuropathies have been raised previously, no consensus regarding the anesthesiologic approach to this patient category has currently been established.[2–4]

We present a case where an uneventful bunion surgery under coverage of a popliteal sciatic nerve and saphenous nerve block using ropivacaine was associated with peroneal nerve neuropathy. Written and informed consent was obtained from the patient for publication for this case report. No institutional approval was required to publish the case details.

Case

A 74-year-old male, American Society of Anesthesiology physical status 3, with a long standing history of ischemic heart disease, multiple acute myocardial infarctions and stents, stent in the right thigh, arterial hypertension, hypercholesterolemia, stroke with sequelae in the form of decreased motor function in right arm and leg, gout, periodic alcohol abuse, pancreatitis, smoking, COPD, microscopic colitis treated with budesonide along with newly diagnosed diabetes type 2 likely developed due to aforementioned steroid treatment was posted for bunion surgery at our facility. The patient had no known allergies.

His routine medication included clopidogrel, atorvastatin, isosorbide mononitrate, furosemide, potassium chloride, metformin, dapagliflozin, allopurinol, promethazine, terbutaline and formoterol inhalers along with calcium and D-vitamin supplements. Budesonide treatment was initiated 11 months prior to surgery, dosage varied between 3 and 9 mg depending on stages of colitis exacerbation. PRN medication included loperamide, glycerol nitrate and paracetamol.

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Surgical history included several colonoscopies, gastroscopies, a cystoscopy and percutaneous stents.

Preoperative laboratory results were unremarkable. The patient's HbA1c having been in the eighties five months prior upon initiation of antidiabetic treatment had become stationary at 45 mmol/mol.

Upon preoperative surgical evaluation the surgeon was unable to detect a pulse in the right foot and deemed the foot arteriosclerotic. However, three months prior, blood pressure was found to be 128/86 and 92/71 mmHg in the right and left foot respectively, showing a reasonable potential for healing following surgery.

Clopidogrel was paused five days preceding surgery. No premedication was administered.

The PNB was effectuated by an experienced consultant anesthetist according to the department's standard operating procedure. With the patient lying in the supine position, the right sciatic nerve in the popliteal fossa, and the right saphenous nerve at the height of the adductor canal were identified with ultrasound. A 15 mL mixture of equal proportion ropivacaine 7.5 mg/mL and mepivacaine 20 mg/mL was injected under ultrasonographic guidance with a Temena USB 80 Evolution needle, a 21G, 80mm needle with 360 degrees ultrasound reflectors and a 30 degree bevel, around the sciatic nerve and in the adductor canal respectively. The local anesthetic was applied both superior and profound to the nerves. Upon advancement of the needle during the sciatic nerve block, the patient complained of a momentaneous pain sensation originating from the area pertaining to the innervation of the tibial nerve. That resulted in a different insertion without any pain during needle advancement or retraction, nor during administration of LA. Ultrasonically, the point of the needle was visible at all times and no visible spread of LA inside the nerve was noticed. There were no adverse events during the administration of the local anesthetic.

Surgery lasted 25 minutes and was uneventful. A tourniquet was applied distally on the lower leg, above the ankle. Pressure was 250 mmHg for a duration of 25 minutes. Antibiotic Cefuroxime 1500 mg was administered intraoperatively.

The patient's foot drop was acknowledged in the recovery ward. It persisted at two weeks, three months and 1 year follow-up. An electromyography (EMG) and electroneurography (ENG) were performed at 9 months showing axonal sensorimotor polyneuropathy of the peroneal nerve but without being able to confirm any mechanical damage to the nerve. Furthermore, an ultrasound examination could not identify any signs of injury to the peroneal nerve at this point. However, both the common peroneal nerve and the distal ischiatic nerve showed augmented cross-sectional area compared to the contralateral side and a few enlarged and hypoechoic fascicles. These findings were not conclusive for any specific type of nerve damage. Upon submission of this paper, two years after the procedure, the patient reported that he had recovered fully to his former motor and sensory function.

Discussion

Mechanisms leading to the development of peripheral nerve injury (PNI) in the context of a PNB has so far shown to include mechanical factors such as trauma from needle puncture, surgical injury, stretch and tourniquet compression. Other known factors include the neurotoxic effects of LA or adjuvants and ischemia.[5,6] However, the etiology of perioperative neuropathy often remains unclear.[7] Potential etiologies will be reviewed in the following.

In our case, the patient developed new-onset neuropathy of the peroneal nerve resulting in a drop foot and loss of sensitivity. Due to the discrepancy between the location of the surgical procedure and the area affected by the nerve injury, direct surgical injury to the nerve is unlikely. The PNB was performed while the patient was awake and without any sedation by an experienced consultant anesthetist. The sonographic conditions were described as good. A momentaneous sensation of pain during needle insertion pertaining to the innervation area of the tibial nerve was noted. An ENG and EMG were performed during follow-up, showing axonal sensorimotor polyneuropathy of the peroneal nerve but without being able to confirm any mechanical damage to the nerve. The findings of ultrasound were also inconclusive concerning any etiological explanation. Ultrasonically, the point of the needle was visible at all times, no pain was elicited upon injection of LA nor was there any visible spread of LA inside the nerve, rendering intraneural or intrafascicular injection very unlikely. The tourniquet was applied for a mere 25 minutes at 250 mmHg making injury due to tourniquet compression improbable.[8] The location, on the lower leg, above the ankle, renders a foot drop by means of damage to the peroneal nerve caused by the tourniquet, likewise implausible. In addition, EMG and ENG showed no sign of mechanical injury. However, the compression may have contributed to any pre-existing ischemia.

In this case, ropivacaine 0.375% was used as LA. Sung et al examined the vasoconstrictor effect of different LA on rat aortic root.[9] They demonstrated that Levobupivacaine and ropivacaine are potent vasoconstrictors at low concentrations followed by mepivacaine and lidocaine. This potency corresponds to their lipid solubility.[9] The pathway involved for ropivacaine seems to be lipoxygenase pathway mediated.[10] Ropivacaine has also shown vasoconstrictor properties in humans after intradermal injections but only at low concentrations 0.5–0.063%, being most pronounced with the lowest concentration.[11] Surprisingly, ropivacaine 1% solution showed increased blood flow.[11] Another study showed reduced acral blood flow when ropivacaine was injected at a concentration of 0.75%.[12] The same result was found in healthy volunteers after intradermal injection of ropivacaine 0.75% and 0.25% with the latter reducing blood flow the most. Following injection of lidocaine 1% and bupivacaine 0.75% blood flow was increased.[12] However, human skin and aorta from rats do not necessarily respond to ropivacaine in the same manner as human arteries.

The neurotoxicity effect of LA should also be considered as a possible risk factor for nerve damage. Among the different LA drugs, lidocaine is the one that has been most extensively studied. Radwan et al examined the neurotoxic effect (growth cone collapse) of LA on cultured chick peripheral neurons.[13] They found that lidocaine was the most toxic (IC_{50}) at 15, 30, 60 minutes and mepivacaine the least toxic with bupivacaine and ropivacaine values lying in between. In this in vitro study, the neurotoxic post exposure effect of ropivacaine and bupivacaine was more reversible 20 hours after stopping the exposure (washout) compared to both lidocaine and mepivacaine. Koo et al compared the neurotoxic effect of lidocaine, bupivacaine and ropivacaine on developing motor neurons in a rat model.[14] Neurotoxicity was measured based on cell viability, cytotoxicity (LDH), ROS production and apoptosis. Overall, lidocaine was the most neurotoxic agent after both 1 hour and 24 hour exposure at all investigated concentrations followed by bupivacaine. Ropivacaine only demonstrated neurotoxicity at the highest concentration (1000 microM = 1×10-3 M) after 24 hours but not after 1 hour. For comparison, ropivacaine concentrations used in clinical practice range between 9.1 x 10–3 M – 2.73 x 10–2 M.[9] Malet et al also found ropivacaine to be significantly less toxic (LD50) on human neuroblastoma cells compared to lidocaine, bupivacaine and mepivacaine.[15] Perez-Castro also examined human neuroblastoma cells but found the potency (LD50) after 10 minutes to be procaine < mepivacaine < lidocaine < chloroprocaine < ropivacaine < bupivacaine.[16]

Several risk factors predisposing patients to the development of PNI subsequently to a PNB have been published, including the type of nerve block performed and the presence of pre-existing neuropathy and neuronal ischemia.[17] It has been advanced that chronically compromised nerves may have their damage further exacerbated by subsequent insults, also if these occur at different locations along the axons.[17,18]

In our case, the patient had a long standing history of peripheral vascular disease, episodic alcohol abuse and recent diabetes diagnosis and thus may have had, if not a clinical neuropathy, a subclinical neuropathy upon presenting for surgery.[1,19,20] Furthermore, his lower extremities nerves likely have been subject to chronic ischemia caused by his history of PVD.[21] Similarly, diabetes, in the presence of diabetic neuropathy shows impaired neural blood vessel function.[22] As such, the patient presented with several risk factor predisposing to PNI following a PNB.

While in this case we cannot rule out the potential neurotoxic effects of ropivacaine, albeit being less in comparison to other LA, we hypothesize that the vasoconstrictive properties of ropivacaine may, in theory, in conjunction with a preexisting chronic neuronal ischemia and neuropathy have damaged an already compromised nerve.

Diabetic neuropathic nerves show increased sensitivity to LA as well as prolonged duration of block.[23,24] Albeit no evidence-based recommendation of dosage in this patient population is available, exercising caution and administering the smallest effective doses would be prudent.[25] Likewise, the use of adjuvants potentially contributing to neuronal ischaemia should be avoided.

Conclusion

The use of ropivacaine used in clinically relevant doses for PNB may cause nerve injury in patients with peripheral neuropathies. A potential mechanism could be due to neuropathic nerves, often already subject to a compromised vascular supply, in conjunction with the vasoconstrictive properties of ropivacaine would be subject to critical ischemia. Further investigations regarding the use of ropivacaine in this population are warranted.

As a footnote, the authors would like to add that upon the follow-up of the patient in this case we came to realize that our healthcare system disposed of no guideline for the follow-up of PNI in the setting of a surgical/anesthetic procedure,

nor did there exist any database for the registrations of those. We propose the development of national guidelines as well as a national database for iatrogenic PNI. Meanwhile, we recommend the use of the guidelines developed by the Regional Anaesthesia - UK society, also used in this case.[26]

Disclosure

The authors report no conflicts of interest in this work.

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