



CASE REPORT

Acquired acute resistance to local anaesthetics in a patient of total knee replacement - A case report

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ABSTRACT

A 72-year-old female patient weighing 80 kg underwent right total knee replacement (TKR) under successful subarachnoid block (SAB) and continuous femoral nerve block (cFNB). Five days later, SAB failed twice for left TKR, with no sensorimotor block observed. The patient refused general anaesthesia on the first day but underwent surgery under general anaesthesia the next day. Postoperative analgesia via ultrasound-guided cFNB was inadequate. This unusual sequence of events, involving differential success of SAB and nerve blocks during two surgeries with different drugs and techniques, suggests acquired acute resistance to local anaesthetics. While technical failure is a common cause of SAB failure, genetic variations in sodium channels, potentially acquired over time, may explain this phenomenon. Such resistance is rare and challenging to diagnose, highlighting the need for further investigation into molecular mechanisms and genetic testing in similar cases.

Keywords: Femoral nerve block, general anaesthesia, local anaesthetics, nerve block, ultrasonography

INTRODUCTION

Tachyphylaxis, or acute tolerance to local anaesthetics (LAs), manifests as a reduced duration, spread, or intensity of regional blocks following repeated administration of equal LA doses.^[1] Though the incidence of LA resistance is unclear, reported cases are rare.^[2] Scepticism often surrounds such reports, with failures frequently attributed to technical issues, drug quality, or practitioner inexperience.^[3] Accurate diagnosis of LA resistance is challenging.^[4] Since all LAs act by blocking sodium channels, any alteration in these channels may lead to resistance.

We present a case involving a patient scheduled for left total knee replacement (TKR), who experienced no sensorimotor block despite multiple correctly performed subarachnoid blocks (SABs). Notably, this same patient had undergone a successful right TKR 5 days earlier using SAB and continuous femoral nerve block (cFNB).

CASE REPORT

A 72-year-old woman, weighing 30 kg, was scheduled for a left TKR. Her history included childhood bronchial asthma, hypertension, and diabetes mellitus, managed with a salbutamol inhaler, metformin (500 mg), and cilnidipine (10 mg). She had previously undergone two caesarean sections under SAB and, 5 days prior, a right TKR under SAB and cFNB, which was

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uneventful. Postoperative analgesia was provided via ultrasound-guided cFNB using 0.125% bupivacaine with 2 µg/cc fentanyl infusion at 5 ml/hour for 72 hours. There was no relevant family or substance use history.

For the current surgery, the plan was SAB followed by cFNB. Upon arrival in the operating room, the patient was connected to standard American Society of Anesthesiologists monitors, including an electrocardiogram monitor, pulse oximeter, and non-invasive blood pressure monitor. Pre-induction vitals were noted. Under aseptic precautions and with the patient in a sitting position, after 2% lignocaine infiltration at L3-L4, a 25-gauge Quincke's spinal needle was used to confirm free cerebrospinal fluid (CSF) flow, and 15 mg (3 ml) of 0.5% hyperbaric bupivacaine was injected. Motor block was assessed using the Modified Bromage Score, which remained zero at 10 and 20 minutes.

A repeat SAB was performed at L2-L3 space with a different batch of 0.5% hyperbaric bupivacaine (15 mg, 3 ml) by a senior anaesthesiologist. Once again, no motor block developed after 20 minutes. The patient declined general anaesthesia that day.

The next day, SAB was attempted again with a different agent, 15 mg (3 ml) of 0.5% hyperbaric levobupivacaine at L3-L4, by the same senior anaesthesiologist. Despite proper CSF flow and administration, the Modified Bromage Score remained zero after 20 minutes. General anaesthesia was then administered using isoflurane for maintenance.

At the end of surgery, intravenous paracetamol (1 g) was given, and a cFNB was performed using 15 ml of 0.125% bupivacaine bolus, followed by infusion at 5 ml/hour under ultrasound guidance. In the recovery room, no sensorimotor blockade was noted after regaining consciousness, even after 30 minutes. The patient reported severe pain with a visual analogue scale (VAS) score of 9–10.

A repeat femoral nerve block with 15 ml of 2% lignocaine also failed to provide relief. Intradermal infiltration using 0.2 ml of 0.5% bupivacaine on the flexor forearm showed no hypoaesthesia for up to 30 minutes.

Intravenous tramadol (50 mg) was administered with minimal effect. Fentanyl via patient-controlled analgesia was then started for 48 hours, resulting in significant pain relief. The remainder of her hospital stay was uneventful, and she was discharged comfortably 5 days later.

All LA agents used in the case were stored under optimal conditions (22–25°C, away from sunlight) and were separated from intravenous anaesthetics to prevent contamination.

DISCUSSION

Acute resistance to LAs is rarely discussed in medical

literature. Anaesthetic failures are typically ascribed to errors in technique or drug handling. However, in our case, repeated administration of different LAs at varying intervertebral spaces and under the care of experienced anaesthesiologists eliminated these factors. Proper CSF flow was verified, and all procedures were done aseptically. Moreover, correct drug dosing further excluded underdosing as a cause.

Amino-amide LAs, such as lidocaine, bupivacaine, and levobupivacaine, block nerve conduction via sodium channel inhibition.^[5] Complete resistance to these drugs raises the possibility of altered sodium channel function, possibly due to genetic mutations.^[6] Given that the patient had previously responded well to spinal and peripheral blocks just 5 days earlier, this acute and selective unresponsiveness is highly unusual.

Interestingly, Liam *et al.* suggested a link between LA resistance and mutations in the melanocortin-1 receptor gene, commonly found in individuals with red hair, who often require higher doses of LA and exhibit increased thermal pain sensitivity.^[7] While our patient did not have red hair, this finding highlights the potential role of genetic predisposition in LA responsiveness. Patients with opioid use histories may also show altered responses to LAs, requiring dose adjustments and extended latency periods.^[8]

In a study by Trescot involving 250 patients with reported LA failures, 43 were found to be hypoaesthetic to lidocaine after subcutaneous testing, though complete failure rates and patient histories were not detailed.^[9]

In our case, intradermal testing with bupivacaine showed no sensory effect, pointing toward a localised acquired resistance. As genetic or molecular testing was not feasible in our setting, and the patient declined further investigation, the aetiology remains unconfirmed.

CONCLUSION

Acute LA resistance is rare but should be considered when conventional techniques fail despite accurate administration and proper dosing. In this case, resistance was the most plausible cause after ruling out other factors. We recommend evaluating patients for possible LA resistance during pre-anaesthetic assessments, particularly those with a history of pain management challenges or previous LA failures. Further research is needed to explore the molecular basis of such resistance to improve patient safety and outcomes.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be

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