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CASE REPORT Open Access

Epidural catheter migration: is aspiration enough? A case report

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Abstract

Background Local anesthetic agents are widely used drugs across the globe. Within their dosage range and appropriate location of administration, they are quite safe and effective. Rarely when given via intravascular route, local anesthetics can lead to life-threatening consequences.

Case presentation Here, we present a case of a young male with an epidural catheter for femur surgery. The catheter was functioning quite well initially but later migrated in the intravascular route. When top-up was given with local anesthetics, the patient developed seizures which were managed promptly.

Conclusions Inadvertent migration of catheter may occur during patient positioning or unwanted movements during surgery, but careful aspiration before every top-up and aspiration during every aliquot of top-up must be practiced.

Keywords Local anesthetic systemic toxicity, Epidural catheter, Seizure, Hypoxia

Background

Local anesthesia systemic toxicity (LAST) is a constellation of life-threatening adverse events associated with the frequent use of local anesthetic agents during surgical procedures for pain management. It has an estimated incidence of 1 per 500 peripheral nerve blocks (Bern and Weinberg 2011). The incidence of LAST (local anesthesia systemic toxicity) during epidural anesthesia is estimated at 4 per 10,000 epidural procedures (Toledo et al. 2013; Mulroy 2002). Epidural catheter migration or displacement during patient positioning is a well-known entity with an estimated incidence of 50% (Butterworth 2010). The growing use and increased convenience of local anesthesia in multiple surgical procedures and pain management as multimodal analgesia techniques all contribute to the increased risk of LAST. Here, we present a case of LAST due to intravascular migration of epidural catheter perioperatively. All data generated or analyzed during this study are included in this published article. We adhered to the CARE Checklist 2013 for reporting this case.

Case presentation

A written consent to publish patient information was obtained from the patient prior to the submission of this case report. A 23-year-old male patient with a history of fracture of the right distal femur was operated on 2 months back and was on an external fixator. He was posted in the operating room for the removal of the external fixator and implantation with a distal femoral locking compression plate. After the pre-anesthetic checkup, proper counseling, and written informed consent, the patient was shifted to the operating room. All the monitors were attached and a wide-bore 18 G cannula was inserted in both forearms. A combined spinal epidural was planned for anesthesia. After taking all necessary aseptic precautions, an epidural catheter was inserted in the L2–L3 interspace by loss of resistance technique. The epidural catheter was fixed at 9 cm, and 3 ml of lignocaine with adrenaline was given as a test dose. No

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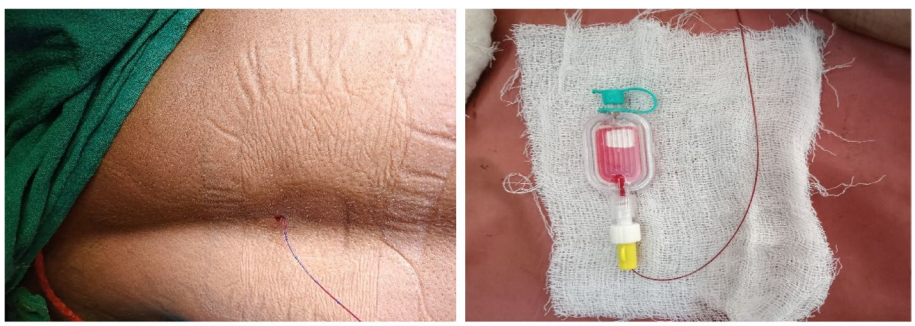
significant hemodynamic changes were observed. Soon after, spinal anesthesia was given at L3–L4 interspace with 3.6 ml of 0.5% hyperbaric bupivacaine. The patient was positioned supine, and surgery was started. Vitals were monitored throughout the procedure. After 2 h, 5 ml of 0.5% bupivacaine with 5 ml of 2% lignocaine with adrenaline was given through an epidural catheter after proper aspiration through the epidural filter. Following 1.5 h, the patient started complaining of pain and voluntary movement in the trunk and waist. After aspiration from the epidural filter, 10 ml of 2% lignocaine with adrenaline was given through the epidural catheter after which the patient was relieved of pain. After 10 min, the patient developed hypotension and tachycardia. The patient was managed with 250 ml of crystalloid bolus and 6 mg mephentermine IV immediately. The patient developed seizures soon after. The patient was immediately administered midazolam 3 mg IV and supplemental oxygen via face mask. Local anesthesia systemic toxicity was suspected, and on subsequent aspiration, blood was seen in the epidural filter (shown in Fig. 1a, b). General anesthesia was considered, and a supraglottic airway device was inserted following induction with 100 mg propofol. The patient was stabilized and meticulously monitored throughout the surgery. After the successful uneventful completion of the surgery, LMA was removed and shifted to the super-speciality anesthesia ICU for monitored care. A 250 ml of 20% intralipid was administered in ICU care as an antidote to local anesthesia toxicity. The patient was shifted to the ward after 24 h.

Discussion

Local anesthesia systemic toxicity affects the proper functioning of sodium channels and is better manifested in central nervous and cardiovascular systems, the former being more sensitive than the latter. The initial CNS symptoms are dizziness, tinnitus, blurred vision, and perioral numbness. The excitatory signs such as restlessness, agitation, and muscle twitching are a result of the blockade of the inhibitory pathways (Fettiplace et al. 2015). Muscle twitching initiates generalized tonic–clonic seizures. These initial signs and symptoms progress to profound CNS depression characterized by slurred speech, drowsiness, unconsciousness, and respiratory arrest. Local anesthetics have direct toxicity on the myocardium. Though the exact mechanism is elusive, the local anesthetics cause conduction blockade by its effects on sodium, potassium, and calcium channels (Weinberg 2012; Day and Graham 2002). Normal conduction is disrupted at bundle of His as a result of the direct blockade of sodium channels. It also drives the resting membrane potential to a more negative value, causing prolongation of PR, QRS, and ST intervals. Potassium channel blockade further worsens the situation causing re-entrant tachyarrhythmia and brady arrhythmias. Calcium channel blockade diminishes intracellular stores resulting in diminished contractility. The net effect results in the interruption of AKT and AMPK pathways causing interference in intracellular glucose metabolism and reduction in intracellular adenosine triphosphate levels and adenosine monophosphate concentrations (Neal et al. 2018). Local anesthetics also have a pH-related suppressive effect on the systemic vascular tone.

Immediate treatment of local anesthesia toxicity involves the general measures of resuscitation. The first step is to stop the local anesthetic injection and call for help. Immediately secure the airway and breathing by administering 100% oxygen and stabilizing circulation (Association of Anaesthetists of Great Britain and Ireland AAGBI Safety Guideline n.d.). Treatment depends upon the systemic manifestations of toxicity. Definite treatment includes the administration of intravenous lipid emulsion. It is observed that lipid emulsion shuttles local anesthetics agents from high-perfused organs like the brain and heart to less perfused and detoxification organs such as the muscles and liver (Fettiplace et al.



a b

Fig. 1 **a** Epidural catheter in situ with blood during aspiration. **b** Aspirated blood in bacterial filter

2015). The recommended dose includes an initial bolus of 100 ml of 20% intralipid over 2–3 min (1.5 ml/kg if lean body weight is less than 70 kg). This is followed by 200–250 ml over the next 15–20 min (0.25 ml/kg/min if lean body weight is less than 70 kg) (Neal et al. 2018; Association of Anaesthetists of Great Britain and Ireland AAGBI Safety Guideline n.d.). If the circulation is not stabilized, re-bolusing can be done up to two more times or the infusion rate can be increased to 0.5 ml/kg/min. The maximum recommended dose of 20% intralipid is 12 ml/kg. Seizure activity, if present, should be managed by benzodiazepines due to their cardio-stable properties (Miler 2010). The patient should be carefully monitored after an event of local anesthesia toxicity.

In our case, the epidural catheter was placed at L2–L3 interspace and fixed at 9 cm. On initial aspiration, no blood was aspirated and no hemodynamic changes were observed on administration of the test dose (3 ml of 2% lignocaine with adrenaline) as well as supplemental dose (5 ml of 2% lignocaine and 5 ml of 0.5% bupivacaine) after 2 h. However, during patient positioning and application of traction during surgery, the epidural catheter was misplaced and migrated into a blood vessel leading to a life-threatening event of LAST. The length of the catheter inside the epidural space also plays an important role in misplacement.

The recommendations in different textbooks regarding the length of the epidural catheter inside the epidural space range from 2–3 cm to 2–6 cm (Miler 2010; Morgan et al. 2006). The shorter the length of the epidural catheter inside the epidural space, the more its chances of dislodgment. Alternatively, an increased length of the catheter causes greater chances of malpositioning, either a transforaminal escape or into anterolateral epidural space giving rise to unilateral block. In a study done by Uchino T et al., among epidurography done in 268 patients, 46 cases showed agent leakage into the psoas compartment (Uchino et al. 2010). Sanchez et al. in their study of 90 patients demonstrated a higher incidence of intervertebral foramina escapes among longer epidural catheters in epidural space (Sanchez et al. 1967).

Conclusions

Local anesthesia systemic toxicity is a life-threatening unforeseen complication that needs prompt intervention and meticulous treatment. The anesthesiologist needs to be well prepared in advance instead of any event of LAST.

Abbreviations

LAST	Local anesthetic systemic toxicity
ICU	Intensive care unit
CNS	Central nervous system
LMA	Laryngeal mask airway
AKT	Protein kinase B
AMPK	AMP-activated protein kinase

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RM: Writing the manuscript. AA: Data collection. PKT: Supervision. LL: Reviewing. TK: Review and editing.

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Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
A written consent to publish patient information was obtained from the patient prior to the submission of this case report.

Competing interests
The authors declare no competing interests.

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