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Case Report

Anaesthesia Section

Local Anaesthetic Systemic Toxicity in a Patient under General Anaesthesia (GA): A Diagnostic Challenge

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ABSTRACT

Local anaesthetic systemic toxicity (LAST) is one of the most dreadful complications after local anaesthetic (LA) use and it is very difficult to manage. The diagnosis of LAST is purely clinical and its presentation is usually obscured in a sedated or anaesthetized patient. A 25-year-old male patient undergoing laparotomy for acute duodenal perforation under general anaesthesia developed seizures after epidural administration of 0.5% bupivacaine. All other possible causes of seizures were ruled out. Seizures were controlled with antiepileptic drugs and patient recovered fully after withholding LA administration. Although, lipid rescue is recommended for LAST, our patient was managed without its use. We, therefore, recommend that utmost care and vigilance should be exercised while using local anesthetics as there is no perfectly accurate method to rule out intravascular administration of LA. Even, if LA is correctly used, LA is detected in plasma due to its systemic absorption from injection site.

Keywords: Epidural analgesia, LAST, Lipid rescue, Seizure

CASE REPORT

A 25-year-old male patient was admitted in surgical emergency with complaints of acute abdominal pain. He was diagnosed to be a case of acute duodenal perforation and was scheduled for emergency laparotomy under GA. Patient had no history of any previous medical or surgical illness including epilepsy. Patient's vitals were stable with a heart rate (HR) of 90/min, blood pressure (BP) of 130/96 mmHg and respiratory rate of 16/min. No history of any previous or concomitant medical or surgical illness was present. His hematological, coagulation and routine biochemical investigations were within normal limits. In the operating room, an 18 Gz multihole epidural catheter was placed in L1-L2 intervertebral space using loss of resistance technique with 4cm of catheter left in epidural space in cephaloid direction. A test dose of 3 ml 2% xylocaine with adrenaline 1:200000 was given to rule out intravascular or intrathecal catheter placement. Patient was induced with propofol and endotracheal intubation was facilitated with vecuronium bromide. Intraoperative anaesthesia was maintained with N_2O:O_2 in 6:4 ratio and isoflurane. Muscle relaxation was maintained with vecuronium bromide. Before surgical incision, 12 ml of 0.5% bupivacaine with 50 µg fentanyl was given through epidural catheter after negative aspiration in divided boluses of 3 ml over 20 min. for intraoperative analgesia. After one hour of initiation of surgery, when patient was in light plane of neuromuscular blockade, we observed tonic-clonic movements in all limbs. Suspecting it to be a generalized tonic clonic seizure (GTCS), inj. Thiopentone 100 mg was given, resulting in complete resolution of limb movements. We withheld further top up of LA in epidural catheter. We kept the patient in light neuromuscular blockade and similar movements again started after 15 min Inj. Midazolam 2 mg followed by loading dose of inj. Phenytoin was given leading in complete resolution of seizure. Surgery was completed within 2 h and patient was extubated after complete reversal of neuromuscular blockade. After 15 min of extubation, patient again had an episode of GTCS which was treated with inj. Lorazepam 2 mg. Patient was shifted to ICU for close monitoring as the patient was drowsy. Serum electrolytes, blood sugar and arterial blood gas analysis was done immediately. No abnormality was detected in these investigations. After 2 h of close observation, patient became conscious and oriented. His vitals were normal. No further episode of GTCS was seen and the patient was shifted to postoperative ward next day.

His postoperative investigations were: Hb-11.9 gm/dl, TLC- 11500/mm^3, DLC- $P_{70}L_{29}M_1E_0$, Platelet count- 2.5 lac/mm^3, Sr. sodium- 138 meq/l, Sr. potassium-4.0 meq/l, Sr. calcium(total)- 10 mg/dl, RBS-150 mg/dl, PT-12 sec, INR-1.0, Sr.urea-27 mg/dl, Sr.creatinine-0.7 mg/dl, ABG: PO_2-300 mmHg, PCO_2-35 mmHg, HCO_3-26 meq/l, pH-7.38 and Sr. lactate 2.0 meq/l. Patient recovered fully within one week of surgery and his postoperative period was uneventful and he was discharged after one week.

DISCUSSION

LA are widely used in anaesthetic practice for nerve blocks to provide anaesthesia and analgesia. Though they are very effective and safe in expert hands, yet they can lead to life threatening adverse effects if they are absorbed systemically or given in large doses. LAST has been a nightmare for anesthetists as it is very difficult to treat and can lead to devastating outcome. Higher concentrations of LA affect all neurons, leading to global CNS depression, clinically seen as coma. The cardiovascular toxicity includes myocardial depression, conduction block and decreased autonomic flow [1]. Central nervous system toxicity usually appears before and at lower blood concentration of LA than required to produce cardiac toxicity. Seizure is one of the commonest presentations of LAST. Acidosis and hyperkaliemia are likely to increase the risk of bupivacaine toxicity [2]. There are many case reports and studies in literature investigating the LAST [3,4]. A spectrum of toxicity from asymptomatic patient to cardiac arrest have been noted. However, the lipid rescue has changed the scenario.

Various measures have been used to prevent intravascular injections of LA including test dosing with lignocaine and adrenaline, small and divided dosing and negative aspiration before LA administration. However, these measurers do not completely rule out the possibility of intravascular administration of LA. β-Blocker therapy can prevent the sympathetic response of epinephrine test dose [5].

LA administration in regional or neuraxial blocks results in detectable plasma levels of LA due to slow systemic absorption from injection site even when they are not administered intravascular. Topical application of large doses of EMLA cream has been associated with

seizure and methemoglobinemia in infants [6]. Seizures have been reported with lidocaine at its lowest effective dose (1.5 mg/kg) [7].

Lipid emulsion as an antidote to the LAST has been recommended by American Society of Regional Anaesthesia. Intravenous lipid emulsion can be used to treat cardiovascular and central nervous system failure [8]. Our patient does not require lipid therapy and his seizures were controlled effectively with antiepileptic.

Peripheral nerve blocks in patients during GA or sedation adds an additional risk factor for neuronal damage [9]. Therefore, it is a matter of debate that whether regional anaesthesia is safe in sedated adults or not. The risk of LAST has not been found higher in sedated patient by some authors [10]. However, practically it is difficult to suspect and diagnose LAST in patient under GA as CNS manifestation are obscured as in our patient until he recovered from muscle relaxation.

Other cause of seizure includes disturbed levels of body water/ electrolytes, hypoglycaemia, head injury, cerebrovascular accident, hypoxia, previous neurological disease, hyperthermia, thyroid disorder or intracranial space occupying lesions. We ruled out these causes in our patient as shown by postoperative investigations given in case report above [11].

In spite of negative test dose and negative aspiration, our patient showed signs of LAST which may be due to inadvertent intravascular injection of LA or systemic absorption of LA from epidural space. Therefore, we recommend utmost vigilance and high index of suspicion when LA is used especially in a sedated or anaesthetized patient.

CONCLUSION

Even though, there are methods to prevent inadvertent intravascular administration of LA, yet these methods are not 100% effective and foolproof. Catastrophic mishaps can occur any time even with utmost care and these can be prevented only with most effective armor of anesthesiologist i.e. *eternal vigilance.*

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