ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 04 | April - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

LOCAL ANAESTHESIA SYSTEMIC TOXICITY (LAST)

Anaesthesiology

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ABSTRACT

Clinical and basic science efforts have enhanced the understanding of local anaesthesia systemic toxicity (LAST). We present a case of a 38 year old female, categorised under ASA grade I with no comorbidities, who was recently diagnosed with right renal calculus and right renal hydronephrosis and underwent right percutaneous nephrolithotomy with double J stenting under spinal anesthesia with IV sedation and local infiltration. She manifested three episodes of generalised tonic clonic seizures (GTCS) and was brought to a tertiary care hospital for further management. On admission she was found to be hypotensive, acidotic, severely hypoxic and had bradycardia; was electively intubated to secure the airway and put on mechanical ventilation. CT brain study was normal and EEG revealed subclinical seizures. The patient was sedated and paralyzed, started on ionotropes, anti-epileptic drugs and lipid emulsion resuscitation therapy to which she responded well and improved haemodynamically and was eventually weaned off mechanical ventilation and subsequently discharged with the EEG showing no further evidence of clinical seizures.

KEYWORDS

INTRODUCTION:

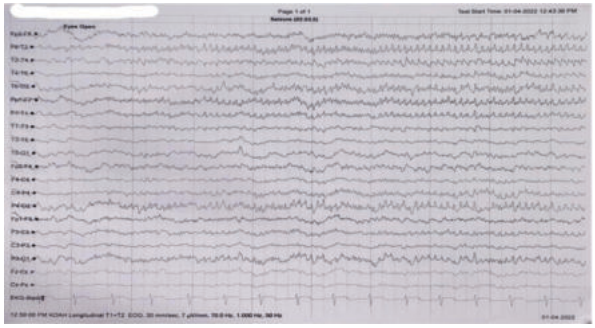
Local anesthesia systemic toxicity is a life threatening adverse event that may occur after administration of local anesthetic drugs through a variety of routes. Local anaesthetic agents are administered in practice by many clinicians, anesthesiologists, surgeons, emergency room providers, dentists and others. Despite the widespread use of local anaesthetic agents, the awareness of local anaesthesia systemic toxicity and knowledge of its management are lacking. Any unusual cardiovascular or neurological signs after administration of a local anesthetic agent should raise suspicion of LAST. Determining the optimal dose of a local anesthetic agent is complex. Elderly patients, pregnant women and paediatric patients are at a higher risk. [1,4]LAST presents with CNS changes manifested by confusion, agitation, peri-oral numbness, shivering, seizures and muscle twitching. CNS depression further causes hypoventilation, hypotension and respiratory acidosis. Cardiotoxicity presents as ST-T changes on the ECG, bradycardia and hypotension. The use of 20% lipid emulsion resuscitation therapy has helped in prompt treatment of LAST. [3]Lipid compartment scavenges local anesthetic from high blood flow and sensitive organs and redistributes the agent to the liver for detoxification. Frequent negative aspiration for blood while administration of local anesthetic agent, use of incremental doses of the drug can reduce the incidence of LAST.

CASE:

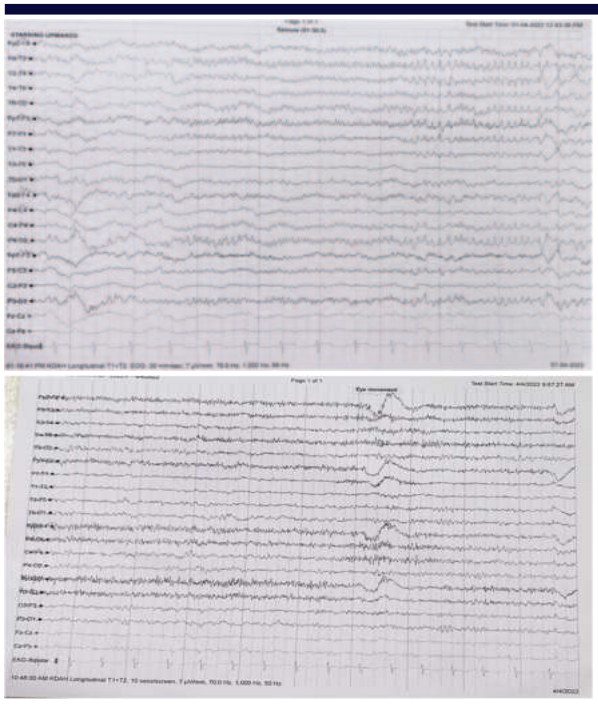
As we discuss our case, a 38 year old pre-morbidly healthy woman, categorised under ASA grade I; recently diagnosed with right renal calculus with right renal hydronephrosis was operated for the same and underwent right percutaneous nephrolithotomy under spinal anesthesia + IV sedation + local anesthesia at the site of surgery. Spinal anesthesia was administered at L3-L4 space with 3.8ml of 0.5% heavy INJECTION BUPIVACAINE and the patient was proned soon after; upon giving the surgical position she complained of pain in the right shoulder and at the surgical site and 25-30ml of 2% plain INJECTION LIGNOCAINE was administered at the site of surgery and she was sedated with INJECTION PENTAZOCINE 30MG and INJECTION KETAMINE 10MG. The haemodynamics intra-operatively remained stable and the surgery lasted for 20 minutes after which the patient was constantly complaining of pain, she was made supine and INJECTION TRAMADOL 50MG was administered with a DICLOFENAC suppository. The patient was then shifted to the recovery area for observation. 15 minutes subsequent to being shifted, she had an episode of generalised tonic clonic seizure, lasting for 1-2 minutes followed by transient recovery of consciousness and another episode of GTCS lasting for 2 minutes. She was loaded with anti-epileptic medications (IV MIDAZOLAM and IV LEVIPIL) and within 5 minutes of administration of AEDs, she had the third episode of GTCS associated with vomiting and was then shifted to a tertiary care hospital for further management. On arrival in the ICU, she was afebrile, hypoxic with SpO_2-85-88% on NRBM with 15 litres/min of O_2, maintaining suboptimal airway, haemodynamically unstable with a SBP-80mmHg and Pulse-48-50bpm with a GCS of 4/15. She was electively intubated and mechanical ventilation was initiated with PRVC mode/FiO_2-60% and was sedated and paralysed. Invasive lines were secured and she was started on ionotropes in view of persistent hypotension and bradycardia. Relevant laboratory investigations were sent and after ruling out the probability of septic shock and ongoing seizures; based on the clinical presentation and the given history, a provisional diagnosis of local anesthesia systemic toxicity was made and 1.5ml/kg IV bolus of 20% lipid emulsion was administered followed by 0.25ml/kg/minute infusion.

Investigations:

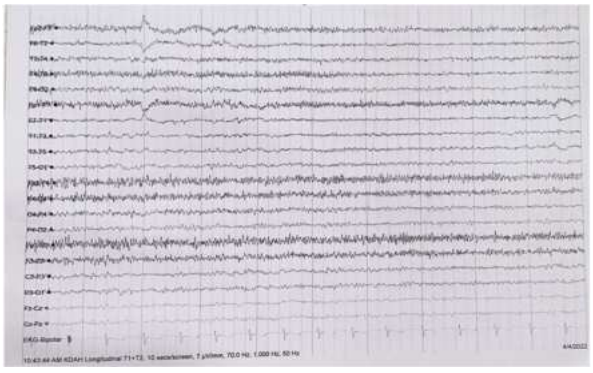
[1]Blood gas revealed significant acidosis (Ph-7.085/PCO_2-53/Bicarb-13.10/Lac-6.8). Relevant labs were sent. Kidney and liver functions were found to be normal, infective and inflammatory parameters were within normal limits and cultures were sterile. [5]CT brain plain was done and was a normal study without any evidence of intracranial hematoma or infarct. HRCT chest revealed findings consistent with aspiration and a small perinephric hematoma around the cortex of the right kidney. Her ECG revealed ST depression in the lateral leads, however the cardiac markers were normal and 2D Echo revealed a normal study. [1,4,5]EEG revealed two focal electrical seizures over the right hemisphere lasting for 1.5-2 minutes each and the anti-epileptic drugs were continued. Based on the clinical presentation, a provisional diagnosis of local anesthesia systemic toxicity was made and 1.5ml/kg IV bolus of 20% lipid emulsion was administered followed by 0.25ml/kg/minute infusion. Subsequent EEG was repeated the following day and it showed no evidence of interictal epileptiform discharges (IED) or subclinical seizures. USG KUB was done which revealed a regression of the previously noted hematoma. MRI brain with contrast was done to rule out any other causes of seizures and it revealed a normal study.



Seizure activity demonstrated on EEG at the time of admission.



Cessation of seizure activity after administration of lipid emulsion.



OUTCOME:

In the consecutive days of being treated with lipid emulsion, she showed improved haemodynamics and the ionotrope requirement decreased and were eventually discontinued, her metabolic parameters improved, sedation and paralytics were discontinued and on re-assessment GCS improved to 13/15. Weaning trials were attempted and were successful and she was successfully extubated and was oxygenating well. She improved neurologically with complete cessation of seizures as demonstrated on the EEG. She was oxygenating well on room air and maintaining stable haemodynamics; invasive lines were removed and on further improvement of her clinical condition, she was eventually discharged. She followed up in the OPD after 15 days with stable haemodynamics and denied any fresh complaints.

DISCUSSION:

CNS toxicity (peri-oral numbness, tinnitus, confusion) were difficult to elicit as seen in our case due to the use of sedation intra-operatively and seizures were the only CNS symptoms that could be appreciated. LAST usually manifests as GTCS associated with muscle twitching, rolling of eyeballs, tongue bite and urinary and stool incontinence; most of the above mentioned symptoms are masked under sedation and general anesthesia. The manifestation of LAST symptoms may commence as early as 5 minutes after administration of the drug and may last up to several hours due to probable delayed absorption of the drug. CNS toxicity is usually first to manifest followed by cardiovascular system involvement manifested by ST-T changes on the ECG, hypotension and bradycardia. The use of 20% lipid emulsion in the treatment of LAST has gained importance; it contains soyabean oil, egg phospholipids and anhydrous glycerol. There are several mechanisms that are associated with lipid emulsion-based recovery, the most common being the lipid sink. Maintaining airway, oxygenation and ventilation remains the mainstay of treatment of LAST and forms a crucial step in the management of hypoxia and acidosis. Seizure activity is best managed with benzodiazepines; propofol is preferably avoided when there are signs of cardiovascular instability. [5]The ACLS algorithm for CPR must be followed if cardiac arrest is to occur. Vasopressin is best avoided as it can worsen acidosis.

CONCLUSION:

Understanding the pathophysiological basis of LAST will improve patient safety. It is imperative that practitioners who use LA in their clinical practice are cognizant of the mechanisms, risk factors, prevention and therapeutic modalities. Meticulous and constant monitoring of patients after administration of local anaesthetic agents will aid early detection of the symptoms and their prompt management.

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