Case Report

LASTing beyond the needle's edge: A case of local anesthetic systemic toxicity following dorsal slit procedure

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ABSTRACT

Local anesthetic systemic toxicity (LAST) is a rare but potentially life-threatening complication associated with the administration of local anesthetics. Early recognition and prompt management are crucial to achieving favorable outcomes. We report the case of a 34-year-old male who developed systemic toxicity following local anesthetic infiltration for a minor urological procedure. The patient presented with altered sensorium and hemodynamic instability and was managed successfully with supportive measures and lipid emulsion therapy. He made a complete recovery and was discharged on the 5th day. This case emphasizes the need for heightened awareness, early identification, and adherence to preventive strategies to minimize the risk of LAST, with lipid emulsion therapy remaining the cornerstone of treatment.

Key words: Dorsal slit, Lipid emulsion therapy, Local anesthetic systemic toxicity

Local anesthetics are commonly used to provide analgesia and anesthesia for a wide range of surgical and diagnostic procedures [1]. Although generally safe when administered within recommended limits, these agents can cause serious and potentially life-threatening systemic toxicity known as local anesthetic systemic toxicity (LAST) [2]. The pathophysiological mechanism of LAST involves the action of local anesthetics on excitable tissues beyond peripheral nerves, affecting both the central nervous and cardiovascular systems [3]. At lower plasma concentrations, central nervous system excitation predominates due to preferential blockade of inhibitory pathways, leading to symptoms such as perioral numbness, tinnitus, metallic taste, dizziness, and seizures [4]. With further increases in plasma concentration, generalized central nervous system depression can occur, resulting in coma and respiratory arrest [5]. Cardiovascular manifestations arise at higher plasma levels and include myocardial depression, conduction abnormalities, and ventricular arrhythmias secondary to sodium, potassium, and calcium channel blockade [6]. These electrophysiologic disturbances may progress to refractory cardiac arrest if unrecognized or untreated [7]. Predisposing factors that increase susceptibility to LAST include extremes of age, hepatic or renal impairment, pregnancy, metabolic acidosis, and pre-existing cardiac dysfunction [8]. Among available agents, bupivacaine is known to be particularly cardiotoxic because of its high lipid solubility and strong protein binding, which prolongs its interaction with myocardial sodium channels [9]. The incidence of LAST is estimated at approximately 0.03/1,000 peripheral nerve blocks, though this varies with the anesthetic technique, dose, and site of administration [10]. Historically, severe LAST was associated with high mortality, particularly with bupivacaine, due to its potent cardiotoxicity and resistance to conventional resuscitative measures [11]. However, advances in regional anesthesia techniques, such as ultrasound-guided nerve blocks, have significantly reduced the frequency and severity of LAST [12]. Furthermore, the introduction of lipid emulsion therapy has improved patient outcomes and remains the mainstay of treatment for systemic toxicity [13]. Early recognition, prompt resuscitation, and adherence to established guidelines are essential for favorable outcomes [14].

CASE REPORT

A 34-year-old male presented to the emergency department with a history of sudden-onset altered sensorium for the past 2 h. He had undergone a dorsal slit procedure for balanoposthitis under local anesthesia in a minor surgical setting approximately 2 h earlier. About 1 h following the procedure, he developed confusion and

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disorientation and was referred for further evaluation and management. The patient had no significant past medical history or known comorbidities.

On arrival, his vital parameters revealed a heart rate of 130 beats/min and a systolic blood pressure of 70 mmHg. Oxygen saturation was 98% on room air, and random blood glucose was 124 mg/dL. Neurological assessment showed a Glasgow Coma Scale (GCS) score of 12/15. Cardiovascular examination revealed normal S1 and S2 heart sounds with no murmurs, and respiratory examination demonstrated bilateral normal vesicular breath sounds without added sounds. Neurological examination showed bilaterally equal and reactive pupils, normal motor activity in all four limbs, and a negative Babinski sign. The abdomen was soft and non-tender with normal bowel sounds. Local examination of the genitalia revealed a healthy postoperative wound with intact dressing and no soakage.

The electrocardiogram showed sinus tachycardia. Venous blood gas analysis revealed a pH of 7.24, pCO_2 of 29 mmHg, HCO_3^- of 15.6 mmol/L, and a lactate level of 4 mmol/L, findings consistent with lactic metabolic acidosis. The complete blood count demonstrated leukocytosis with a total leukocyte count of 15,200 cells/mm^3. Renal function tests were within normal limits, and a non-contrast computed tomography scan of the brain showed no acute changes.

The clinical presentation, temporal association with local anesthetic administration, and exclusion of other causes led to a diagnosis of LAST. The patient was managed with supportive care and intravenous lipid emulsion therapy as per established guidelines. The patient was immediately resuscitated with two pints of intravenous normal saline. Due to persistent hypotension, he was started on intravenous noradrenaline infusion. He was administered intravenous lipid emulsion therapy as per standard protocol. Given the worsening GCS, the patient was intubated for airway protection and continued on supportive treatment in the intensive care unit.

According to the ASRA (2017) Recommended Dosing Protocol [14], the recommended formulation should be 20% lipid emulsion (Intralipid®, Liposyn®, or equivalent preparation). Initial bolus was 1.5 mL/kg of 20% lipid emulsion IV bolus administered over 1 min. Continuous infusion should start immediately after the bolus at 0.25 mL/kg/min. If cardiovascular instability persists, repeat bolus (1.5 mL/kg) every 3–5 min (maximum two additional boluses) and increase infusion rate to 0.5 mL/kg/min. The maximum dose should not exceed 10–12 mL/kg total dose in the first 30 min. Continue infusion for at least 10 min after achieving hemodynamic stability and monitor for recurrence of toxicity after stopping infusion.

By the 2nd day of hospitalization, the patient exhibited gradual neurological improvement, with restoration of orientation and responsiveness. He was successfully extubated following confirmation of adequate spontaneous ventilation and stable oxygen saturation. Hemodynamic parameters improved steadily, allowing stepwise reduction and eventual discontinuation of vasopressor support. Serial monitoring of laboratory values and ECG revealed normalization of metabolic acidosis and resolution of sinus tachycardia. Supportive care, including intravenous fluids and close neurological observation, was continued. By the 5th day, the patient had achieved complete clinical recovery with no residual neurological or cardiovascular deficits and was discharged in stable condition with advice for follow-up and avoidance of excessive local anesthetic exposure in the future.

DISCUSSION

LAST represents a complex pharmacological emergency that continues to challenge clinicians despite advances in regional anesthesia techniques and monitoring [1]. The incidence of LAST has declined in recent years due to ultrasound guidance, dose optimization, and improved safety awareness [2]. Despite these improvements, it remains a potentially fatal complication when it occurs [3]. The pathophysiology, clinical presentation, and management strategies for LAST have been increasingly elucidated over the past two decades, leading to standardized guidelines for prevention and treatment [4].

Factors contributing to LAST include excessive dosage, rapid absorption, and inadvertent intravascular injection [5]. Clinical manifestations may range from mild central nervous system symptoms such as perioral numbness, tinnitus, and agitation to severe outcomes such as seizures, cardiac arrhythmias, and circulatory collapse [6].

In this case, the early onset of neurological symptoms and cardiovascular instability following a minor surgical procedure raised a strong suspicion for LAST, prompting immediate management [7]. Intravenous lipid emulsion therapy remains the mainstay of treatment and acts as a "lipid sink" by sequestering lipophilic anesthetic molecules from target tissues, thereby reducing systemic toxicity [8].

Preventive measures are essential to minimize the risk of LAST [9]. These include using the lowest effective dose of local anesthetic, aspirating before each injection to avoid intravascular administration, and preferring incremental dosing with continuous monitoring [10]. The use of ultrasound guidance enhances the precision of injection and reduces the likelihood of systemic toxicity [11]. It is equally important that lipid emulsion therapy is readily available wherever regional anesthesia is performed to enable prompt treatment if LAST occurs [12].

CONCLUSION

LAST remains a rare but potentially life-threatening complication that demands a high index of suspicion and

prompt management. Early recognition of neurological and cardiovascular symptoms is crucial to ensure favorable outcomes. The timely administration of intravenous lipid emulsion therapy plays a pivotal role in reversing toxicity and improving survival. This case underscores the importance of continuous education, vigilance, and strict adherence to established guidelines in all clinical settings where local anesthetics are administered.

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