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SUCCESSFUL MANAGEMENT OF ROPIVACAINE INDUCED CENTRAL NERVOUS SYSTEM TOXICITY AFTER SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK- A CASE REPORT

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ABSTRACT Local anaesthetics are widely used in everyday clinical practice. Local anaesthetic systemic toxicity (LAST) is a rare but life-threatening complication that may occur after administration of local anaesthetic (LA) drugs via various routes. Ropivacaine, a relatively newer amino amide LA, has better neurologic and cardiac toxicity profile compared to bupivacaine. Despite its relatively safer profile, there are reports of ropivacaine induced systemic toxicity. We report a case in which neurologic toxicity occurred during supraclavicular brachial plexus block using dose of ropivacaine well within recommended limit and was successfully managed with intralipid emulsion. Our aim is to reinforce the importance of early detection and prompt management of a serious LAST event with lipid emulsion for reducing mortality and morbidity. We also wish to emphasise upon the fact that such an event may occur even after careful adaptation of all precautionary measures for risk reduction.

KEYWORDS : Local Anaesthetics, Ropivacaine, Toxicity, Convulsion

INTRODUCTION

Local anaesthetic systemic toxicity (LAST) is a rare but potentially lethal complication of LA that comes with their widespread use for regional anaesthesia. Ropivacaine has a better neurologic and cardiac toxicity profile than bupivacaine and hence is considered safer.**(1)** Although incidences of ropivacaine induced systemic toxicity have been reported, most of them occurred following use of a higher dose or inadvertent intravascular injection. We report a case of CNS toxicity with ropivacaine used in recommended safe dose for supraclavicular approach to brachial plexus block which was successfully managed with the use of lipid emulsion.

CASE REPORT

A 21 year old male, ASA I, weighing 50kg, with fracture of distal end of radius was scheduled for elective open reduction and internal fixation with plating under brachial plexus block. The patient had no significant past medical or surgical history. He had no known allergies.On pre-procedure examination, his blood pressure (BP) was 110/80 mm Hg, heart rate (HR) was 78 beats per min (bpm) and repiratory rate was 16 per minute. His cardiovascular, respiratory, abdomen and central nervous system (CNS) examination were unremarkable.

After establishing routine monitoring and peripheral intravenous access with 18-G cannula, brachial plexus block with supraclavicular approach was performed using 22-G insulated needle and a peripheral nerve stimulator. After frequent negative aspiration, 20ml of 0.4% (80mg) ropivacaine was administered in 3-4 ml aliquots while continuously monitoring the patient. Thereafter, while injecting 1% lignocaine with epinephrine (20 mg had been injected) the patient developed generalized tonic clonic seizure. His HR increased to 128 bpm with BP 110/70 mm Hg and oxygen saturation (SpO2) 98%. Injection of LA was immediately stopped and intravenous midazolam 2mg was injected. Patient was mask ventilated with 100% oxygen. As his seizure activity continued, intravenous propofol 50 mg was given along with simultaneous administration of 75 ml intralipid 20% as bolus over 3 minutes followed by continuous infusion 0.25ml/kg/min. His seizure lasted for 2-3 minutes after which he remained unconscious for around 8 minutes. Thereafter he regained full consciousness while responding to commands. Intralipid infusion was continued for 10 minutes and then stopped as the patient had fully recovered. He developed hypotension with BP 90/50 mm Hg, HR 110 bpm and SpO2 98%, for which intravenous phenylephrine 100 µg was given. Thereafter BP recorded was 108/60 mm Hg with HR 100 bpm. Oxygen administration at 6 L/min was continued with facemask.

No signs of sensory or motor block could be elicited. The patient had no recall of the incident, had no sequelae and was informed of the event. After consultation with the surgeon it was decided to not continue with the surgery. Patient was continued to be monitored for 6 hours and was shifted to ward.

DISCUSSION

LAST most commonly presents as CNS toxicity in the form of seizure.**(2)(3)** CNS is more susceptible to LA effects and hence CNS toxicity appears in dose or blood level lesser than that required for cardiovascular events. CNS toxicity has an initial excitatory phase followed by depressive phase. Early neurological signs include circumoral numbness, tinnitus, metallic taste, dizziness. These can eventually lead to generalized seizure followed by coma and respiratory depression which is the depressive stage. CNS toxicity can occur even without the presence of the early warning signs. Cardiovascular systemic toxicity initially leads to hypertension, tachycardia followed by myocardial depression and hypotension and finally a variety of arrhythmias including asystole.

Ropivacaine, a pure S-enantiomer, is less lipophilic and has been shown to be lesser cardiotoxic and neurotoxic than bupivacaine.**(4)** The incidence of ropivacaine induced neurological events has been estimated to be 8 cases per 1,00,000 patients compared to 6.1 cases of cardiovascular events per 1,000,000 patients.**(5)** The majority of adverse events occur immediately following LA injection but this may not be the pattern in every case.**(6)**

Table 1 summarizes cases of ropivacaine toxicity in adults during peripheral nerve blocks reported since 2007. Dhir et al in their report summarized cases of ropivacaine toxicity in adults during peripheral nerve blocks reported since 2002.**(7)**

Table 1.Reported cases of ropivacaine induced systemic toxicity after peripheral nerve blocks in adults (after 2006)

Year	Block	Dose of ropivacaine (mg)	Effects	Total(free) plasma concentration	Time	Proposed mechanism
2014[8]	Bilateral transversus abdominis plane block	300	Seizure	3.99µg/ml	25 minutes	overdose
2012[9]	Infraclavicular	300	Tongue numbness, nausea, dizziness	Not measured	24 minutes	Rapid absorption of large dose
2012[9]	Infraclavicular	300	Seizure	Not measured	28 minutes	Rapid absorption of large dose

2011[10]	Interscalene	200	Restlessness, limb twitching, slurred speech	1.99µg/ml	1.5 hour	Slow absorption from brachial plexus
2008[11]	Axillary + interscalene	300	Seizure	2.13mg/L	25 mintutes after axillary, 10 minutes after interscalene	over dose
2007[12]	Axillary	300	Seizure	3.65µg/ml	13 minutes	overdose

Chazalon et al cited a case of severe ropivacaine toxicity leading to cardiac arrest following neurologic complications.(**13)** The total dose administered was 6.4mg/kg which far exceeds the recommended dose. Intralipid had not yet been used in humans for LAST management at that time. The patient was successfully managed by applying regular safety and resuscitation measures.

Litz et al reported a case of ropivacaine induced generalized seizure followed by cardiac arrest in a 84 year old woman.(**14)** In this case, 40ml 1% ropivacaine was mistakenly injected for axillary plexus block. Intralipid emulsion in bolus and infusion was used along with chest compression for successful resuscitation.

Dhir et al reported a case of CNS toxicity in 76 year old woman with multiple myeloma.(**7)** Ultrasound guided interscalene block was given with ropivacaine through a catheter. Age may have influenced the absorption kinetics of ropivacaine along with reduced α1-acid glycoprotein (AAG) as seen in multiple myeloma cases.

The risk factors for toxicity are usually drug related: type and dose of drug; patient related: age, comorbidities; site of block with higher risk of intravascular injection eg. interscalene; continuous infusion etc.(**15)** All the cases cited above along with others that have been reported, were a result of presence of the usual risk factors for LAST.

In our case, CNS toxicity in the form of generalized seizure occurred. The time profile, the symptoms observed and an absence of a neurologic history suggest ropivacaine induced toxicity. This was despite administering recommended dose of ropivacaine and adhering to precautionary measures to avoid intravascualar injection. Lignocaine sensitivity in the patient was ruled out following a negative skin test. Although total lignocaine dose given was 20mg (<0.5mg/kg), an additive effect of LA can not be ruled out. Also plasma ropivacaine level was not measured in our case. The threshold plasma concentration at which CNS toxicity occurs may be related more to the rate of increase of the serum concentration of LA rather than to the total dose of drug injected.(**16)** Careful monitoring, prompt recognition and treatment of toxicity with benzodiazepine and intralipid helped us in successfully managing this case.

CONCLUSION

From our case we can infer that negative aspiration does not preclude possible inadvertent intravenous injection of LA. LAST can occur even after using safe dose of LA, frequent negative aspiration and avoiding intravascular injection in a young healthy patient without any comorbidities. Hence careful monitoring, adequate and proper preparation for managing complications and being vigilant are important for safer administration of regional anaesthesia.

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