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MIXTURE OF LOCAL ANESTHETICS FOR BRACHIAL PLEXUS BLOCKADE - FRIEND OR FOE

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Abstract: *Traditionally surgeries on upper limb used to be performed under general anaesthesia but due to associated sequelae, increasing cost of anaesthetic agents and the problems of operation theatre pollution, focus has been shifted towards regional anaesthesia. Moreover postoperative pain relief is an added advantage of regional techniques. Here, we report a case where supraclavicular block was performed taking usual precautions however, the patient suffered local anesthetic systemic toxicity.*

Keywords: *brachial plexus blockade, lipid emulsion, local anesthetic systemic toxicity, seizures, supraclavicular block*

I. INTRODUCTION

Brachial plexus blockade is the most commonly performed of the peripheral nerve anesthesia by most anesthesiologists. Although challenging, as many vital structures are located around brachial plexus, it still remains the preferred choice of many anesthetists and surgeons due to its advantages like reduced blood loss, reduced nausea and vomiting, decreased opioid use, postoperative pain relief, reduced recovery time and less airway manipulation. Apart from knowledge of anatomy of the region and pharmacology of drugs used, it is important to choose correct doses, correct technique and use adequate and continued monitoring. We performed successful supraclavicular block in a patient posted for operative procedure of fracture distal end of humerus, however the patient had an episode of seizures after 20 minutes of drug administration. Her vitals were stable throughout the episode. We discuss probable causes and principles of management of such cases.

II. CASE REPORT

46 year old female, weighing 80 kg, with fracture of shaft of right humerus due to road traffic accident was posted for plating of humerus. There was no history suggestive of head injury. She did not have any other fracture. On examination, her vitals were within normal limits. She had no systemic co-morbidities. Airway examination graded her to be Malampati Class 1. Her routine haematological and biochemical investigations were obtained. It was within normal limits. Patient was explained about the procedure of supraclavicular block and possibility of general anesthesia in case if the block did not provide surgical anesthesia. Informed consent was taken. Her local anesthetic sensitivity test was performed as a routine and was found to be negative. Patient was kept starved for overnight.

Patient was taken to operation table. IV line was secured on nonoperative limb with 20G cannula. Ringer lactate was started. All baseline monitors were applied. Her heart rate was 74 per minute, blood pressure was 132/82 mmHg, respiratory rate was 16 per minute and SpO_2 was 100%. She was comfortable and no sedation was given. Patient was made to lie supine with her head turned to opposite side and arm pulled down gently. A small pillow was placed below the shoulder to make the field more prominent. A point 1 cm above the clavicle at a junction of inner 2/3 and outer 1/3 of the clavicle was chosen for the conduction of block. Under all aseptic precautions an intradermal wheal was raised with 1% lidocaine at the selected point. With anaesthesiologist standing at the head end, slightly towards the side, a 5 cm long 22 SWG needle was inserted through the wheal directed medially and inwards at the angle of 20 degrees to the skin, parallel to clavicle avoiding the external jugular vein. Paraesthesia was elicited in the hand and block was performed in single attempt with 280 mg of 1% Inj. Lidocaine with adrenaline 1:1,00,000 (14 ml of 2% Lidocaine with adrenaline) 100 mg of 0.25% Inj. Bupivacaine (20 ml of 0.5% Bupivacaine) and 60 mcg Clonidine (14 ml); total volume of 40 cc was given. Negative aspiration for blood was confirmed after every 5 cc aliquots.

Immediately after the injection of drug, patient felt warmth in right upper limb and pain relief at fracture site. Dense sensory and motor block was confirmed within 15 minutes. At this point in time, patient was clear headed with stable vitals. After 20 minutes she became restless, complaining heaviness in chest and something going wrong. Within seconds, she had seizures. Midazolam 2 mg IV was given, 100% oxygenation was started with mask on Bain's circuit. Due to persistent seizures after Midazolam, decision for intubation was taken. Propofol 100 mg IV and Succinylcholine 100 mg IV was given and airway was secured. Arterial blood gas was within normal limits at this point. Other causes of seizures like hypoxia, hypoglycemia and electrolyte imbalance were ruled out with the help of arterial blood gas analysis, blood sugar level and electrolyte levels simultaneously. Muscle relaxation was maintained with Vecuronium Bromide. Patient was electively ventilated for four hours and extubated uneventfully. Surgery was postponed.

She was evaluated post procedure for detection of etiology. Chest X-ray was done in recovery which showed no findings suggestive of pneumothorax. Electroencephalogram and Magnetic Resonance Imaging were done. Both studies showed no pathology. Psychiatric opinion was also sought however mental illness was ruled out. Doses, concentration and site of administration of drugs were also correct. With this workup and background, we thought of cumulative toxicity of lidocaine and bupivacaine leading to local anesthetics systemic toxicity (LAST).

1 week after the episode we administered general anesthesia for the same operative procedure and the case went uneventfully.

III. DISCUSSION

General anaesthesia (GA) and regional anaesthesia (RA) have been used successfully for upper extremity orthopaedic procedures. Nerve block anesthesia apart from being cheaper than GA, has many advantages such as anaesthesia targeted at the operative site, reduced blood loss, less airway manipulation, excellent postoperative pain relief, reduced nausea and vomiting, decreased opioid use and reduced recovery time .

The incidence of toxicity is greater with brachial plexus techniques than most others, because larger than usual doses of local anesthetics are used and the injections are made in and around large vascular channels in the head, neck, and axillary regions. The potential for serious complications following brachial plexus anesthesia seems to be greater with supraclavicular techniques.[1] The incidence of various complications ranges from pneumothorax, phrenic nerve block, Horner's syndrome, neuropathy, central nervous system toxicity to cardiovascular system toxicity.

We routinely perform supraclavicular block by a modified approach described by Dr. Dilip Kothari as it is associated with acceptable results and safety. [2] Needle localization by either paresthesia or peripheral nerve stimulator seems to be equally efficacious. Studies that directly compare these two modalities note similar success rates (70%–90%).[3] A combination of lidocaine and bupivacaine was used for the advantages of rapid onset and prolonged effect of block respectively.[4] Maximum acceptable dose of lidocaine came to 560mg and for bupivacaine 160mg for our patient. Doses we used were 40% less than that of maximum doses we could use. [4] We preferred adrenaline added to lidocaine as it would serve as a marker for inadvertent intravascular injection, reduce the absorption of local anesthetics into the systemic circulation and prolong duration and intensity of the block.[4, 5] Clonidine was added as adjuvant for postoperative analgesia.[5] Mixtures of local anesthetics for regional anesthesia are sometimes used in an effort to compensate for the short duration of action of certain rapidly acting agents such as chlorprocaine and lidocaine and the long latency of longer acting agents such as tetracaine and bupivacaine.

LAST occurs when threshold blood levels of local anesthetics are exceeded, which can occur with unintentional direct arterial or venous injection or slow systemic absorption of a large volume of extravascular local anesthetic or injecting large dose. Toxicity is manifest as central nervous system symptoms with increasing levels, progressing to cardiac signs with higher levels.[6] In the medical literature, the mean time to onset of LAST symptom is less than 1 min and 75% of cases present within the first 5 min after injection. The greatest time interval between local anesthetic injection and signs of systemic toxicity was 60 min.[7] Classically described prodromal signs are perioral numbness, dysarthria, and dizziness.[7] Most important; usually it is associated with cardiac toxicity.

In our case onset of seizures was at 20 minutes, with absence of classic symptoms. There were no signs suggestive of cardiovascular disturbance.

Lipid emulsion therapy can be instrumental in facilitating resuscitation, most probably by acting as a 'lipid sink' that draws down the content of lipid-soluble local anesthetics from within cardiac tissue, thereby improving cardiac conduction, contractility, and coronary perfusion. No lipid emulsion was given as in our patient as seizures were controlled after intubation and cardiovascular component was not involved.[8,9] Airway was secured to avoid hypoxemia and acidosis. Securing the airway is the key to successful care of LAST

patients.[8] Prevention of hypoxia and acidosis by immediate restoration of oxygenation and ventilation can either halt progression to cardiovascular collapse and seizure or facilitate resuscitation.[8]

Our technique was reliable, safe and conducted by expert. Our dose calculation was apparently correct. Hence our reason for toxicity was combination of drugs resulting in cumulative toxicity. Possible etiology behind this event of LAST might be when combination of two local anesthetics are used, there toxicity remains unpredictable and it is dependent of each other.[4] It is difficult to predict time to peak blood levels of both the drugs which lead to cumulative toxicity, because LAST is related to peak blood levels of local anesthetics agents used.

We have come across one similar case report where patient had cardiac arrhythmias 20 minutes after seizures. Patient died 1.5 hours after the onset of neurologic symptoms despite initiation of advanced cardiac life support and lipid emulsion therapy.[7] In our case the patient was managed successfully and the surgery was completed uneventfully in the next seating.

IV. CONCLUSION

Mixing of local anesthetics is often done in clinical practice with the intent of obtaining the faster onset and the longer duration of longer acting local anesthetic. Unfortunately, when local anesthetics are mixed, their onset, duration and potency become much less predictable. Therefore, their end result becomes much less predictable. Vigilance during the performance of regional anaesthetics and prompt intervention at the earliest signs of toxicity are most important in successful treatment.

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