CASE REPORT

Local Anesthetic Systemic Toxicity following Peribulbar Block: A Case Report

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Abstract

Cataract surgery is performed routinely under regional orbital blocks including retrobulbar and peribulbar blocks. Several complications have been reported while performing these blocks, the most significant of which is the local anesthetic systemic toxicity (LAST). The symptoms and signs present in a varied spectrum, but every such case requires early recognition and immediate resuscitation to avoid long-term morbidity and even death. Lipid emulsion therapy forms the mainstay of treatment. We present a case of a 49-year-old man who planned to undergo cataract surgery under the peribulbar block, who developed LAST and was successfully treated with 20% lipid emulsion without any adverse sequelae.

Keywords: Lipid emulsion therapy, Local anesthetic, Local anesthetic systemic toxicity, Peribulbar block.

Journal of Medical Academics (2020): 10.5005/jp-journals-10070-0059

Introduction

Cataract surgery can be performed under local anesthesia, either by topical LA application or orbital regional anesthesia, which includes techniques, such as, the retrobulbar block, the peribulbar block, or the sub-Tenon's block.[1] Complications arising from orbital regional anesthesia are well-documented and may manifest locally or systemically.[2–6] Systemic manifestations depend on the local anesthetic (LA) used and route of spread of the drug. Local anesthetic systemic toxicity (LAST) is a life-threatening adverse event requiring rapid assessment and resuscitation.[7] The anesthesia providers should be ready to deal with diverse effects on the central nervous system (CNS) or the cardiovascular system. We present a patient who developed LA toxicity following peribulbar block and recovered with lipid emulsion administration.[8,9]

Case Description

A 49-year-old, 84 kg man, was planned to undergo phacoemulsification of the right eye for a posterior subcapsular cataract and placement of posterior chamber intraocular lens (PCIOL) under a peribulbar block. He was diagnosed with primary hypertension 4 years ago for which he was receiving medication in the form of tablets of amlodipine 5 mg once daily and his preoperative blood pressure was 132/80 mm Hg.

His general physical examination during the preoperative anesthetic evaluation revealed no significant abnormality and his vital parameters were within normal limits. His Metabolic Equivalents (METs) were above 5. Airway examination was normal with an adequate inter-incisor gap, good dentition, and Mallampati score was class 3. His systemic examination revealed no abnormality as well. He had normal blood chemistry and complete blood count. The electrocardiogram showed a normal sinus rhythm and his chest X-ray was also normal. He was advised to take the anti-hypertensive medications on the morning of surgery with breakfast. Written informed consent was taken for the surgery and the peribulbar block. He was accepted in the American Society of Anesthesiologists (ASA) grade II.

On the day of surgery, a 20-gauge intravenous cannula was secured with 0.9% normal saline, and an electrocardiogram, noninvasive blood pressure, and pulse oximetry monitoring were established. The peribulbar block was given with a mixture containing 6 mL lignocaine (2%) and 4 mL bupivacaine (0.5%) with a 25-gauge, 25-mm needle. Five milliliters were given inferiorly at the junction of the outer one-third and inner two-thirds of the lower orbital rim and after 2 minutes, another 3 mL was given superiorly at the supraorbital notch. The plunger of the syringe was withdrawn before injecting the LA and no blood or cerebrospinal fluid (CSF) was observed. Three to four minutes after the block, the patient complained of palpitations, anxiety, mild suffocation with sweating, and dizziness. On checking, the patient was slightly drowsy, but was responding to verbal commands and had a slurred speech. He was sweating and had cold extremities.

The patient developed bradycardia (from 76 to 52/minute) and hypotension (from 140/88 to 96/60 mm Hg). SpO_2 was 96% on room air. The II-lead electrocardiogram reading on the monitor showed a normal sinus rhythm. The patient complained of blurring of vision with yellowish colored halos. Oxygen was supplemented with a face mask and a normal saline bolus of 250 mL was given. Atropine 0.3 mg was given intravenously followed by phenylephrine 40 µg. There was no significant improvement in the hemodynamic parameters after these injections. The intravenous spread of LA mixture was suspected at this point. Twenty percent lipid emulsion at a rate of 1.5 mL/kg was given over 3 minutes. The patient's heart rate improved to 70/minute and blood pressure recorded 110/72

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How to cite this article: Nasser A, Raja F. Local Anesthetic Systemic Toxicity following Peribulbar Block: A Case Report. J Med Acad 2020;3(2):67–68.

Source of support: Nil

Conflict of interest: None

mm Hg. Lipid emulsion infusion was continued for 2 hours at 0.25 mL/kg/minute. Over the next 1 hour, the patient improved symptomatically as well. The vision and speech improved, with no evidence of globe perforation or optic nerve damage. The patient was shifted to intensive care unit (ICU) for 24 hours of continuous monitoring and was discharged after 48 hours.

Discussion

The plasma concentration of LA depends on the dose and the vascular supply at the site of drug deposition. The well-perfused organs such as the brain and the heart receive the bulk of the drug and the free portion in the plasma determines the clinical and toxic effects. These toxic effects are caused due to the sodium (Na^+), potassium (K^+), and calcium (Ca^{2+}) channels, thereby inhibiting neuronal transfer and depolarization.[10]

Local anesthetic can spread to the CNS after ophthalmic regional blocks via a conduit formed by the fusion of the optic nerve epineurium with the tubular sheath of the dura mater which is continuous with the sclera. While injecting the drug if the sheath is perforated, the central spread can occur either by accidental intra-arterial injection or into the subdural and subarachnoid spaces. The incidence of central spread is higher with retrobulbar blocks than with peribulbar or sub-Tenon's blocks.[3]

In case of accidental intra-arterial injection, retrograde flow of anesthetic agent from a branch of the ophthalmic artery through the internal carotid artery to the midbrain can occur. In such cases, the CNS toxicity is almost immediate, and a seizure would result which may lead to cardiovascular instability. This complication can be avoided by aspirating to check blood flow before injecting the drug. When the drug spreads into the CSF, the onset of symptoms and the symptomatology is varied; largely depending on which part of CNS is affected.

The classic symptoms of LAST are subjective such as auditory changes, circumoral numbness, metallic taste, and agitation that may progress to seizures and/or CNS depression. Cardiac toxicity generally does not occur without preceding CNS toxicity, except when LAST occurs secondary to direct intra-arterial injection, while following seizures, cardiac excitation develops (tachycardia, hypertension, arrhythmias). If the drug concentration is large cardiac depression will follow. This classic description, however, presents with extreme variability in terms of the onset and the duration of symptoms.[7] In our case, the patient developed CNS symptoms along with cardiac depression.

The sequelae of brainstem anesthesia and cardiac toxicity will usually be apparent in the first 15 minutes after the injection. It may affect the temperature regulation and the patient may experience disorientation, amaurosis fugax, aphasia, hemiplegia, unconsciousness, convulsions, and respiratory or cardiac arrest.

Prompt recognition and early institution of treatment will ensure a more favorable outcome. Close initial observation with monitoring of vital signs will help in deciding the further course of treatment. Airway control and respiratory support with 100% oxygen supplementation and possible cardiac intervention with circulatory support in the form of fluids and vasopressors may be required. Rosenblatt et al. reported the first clinical application of 20% lipid emulsion for the treatment of LAST.[11] Reversal of LA toxicity can be achieved by the so-called "lipid sink" phenomenon of the intralipid emulsion therapy. The intravascular lipid mass binds the toxin and pulls it from the target tissue, thereby reversing the neurologic and cardiac toxicity.[9] The present recommendation is a large intravenous bolus of approximately 1.5 mL/kg over 2–3 minutes followed by infusion of 0.25–0.5 mL/kg/minute for 10 minutes. A maximum dose of 10 mL/kg can be used.[7,9] In our patient as well, we observed significant improvement after the initiation of 20% lipid emulsion therapy.

The accidental penetration of the needle tip into the nerve sheath can be prevented by adequate patient counseling and instructing them to maintain a neutral gaze while performing the block. The risk can also be decreased by using needles shorter than 30 mm. In our case, the length of the needle was 25 mm, and the complications might have resulted from advancing the needle too far. Ultrasound-guided blocks are known to reduce the risk of LAST by 60–65%. "Wiggle test" may be performed and the needle aspirated for blood before injecting the drug.

In all the cases of ophthalmic regional blocks, the patients should never be left unattended for at least 15 minutes after the injection, and monitoring including pulse oximetry, electrocardiogram, and noninvasive blood pressure should be established along with peripheral intravenous access. This should be made mandatory and part of the hospital protocol for early recognition and favorable outcome.

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