Generalized seizures during cataract surgery following peribulbar block: A case report

Gautam Paul¹, Ankita Narula², Praveer K Srivastava², Mohit Bharambe², Zakir Hussain Laskar³

From ¹Departments of Ophthalmology, Silchar Medical College and Hospital, ²Department of Ophthalmology, Trainee, Silchar Medical College and Hospital, Uttar Krishnapur Pt III, Assam, India

Correspondence to: Dr. Ankita Narula, Department of Ophthalmology, Silchar Medical College and Hospital, Uttar Krishnapur Pt III - 788 014, Assam, India. E-mail: aas28thehope@gmail.com

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ABSTRACT

Local anesthesia techniques are commonly used to perform the cataract surgery. Among these techniques, peribulbar anesthesia (PBA), when compared to retrobulbar anesthesia, is more effective and safe. However, PBA is not without risk. Complications may arise which if not recognized early and resuscitated, can be fatal. A 70-year-old Indian female was admitted for cataract extraction and lens implant in her left eye. Lignocaine sensitivity was done and showed no abnormal reaction. She developed generalized tonic-clonic convulsions following peribulbar block. This case report attempts to highlight the need for a trained personnel, compliance with safety standards in anesthesia, awareness, and early recognition of the possible complications and their management.

Key words: Cataract surgery, Complications, Convulsions, Peribulbar anesthesia

Local anesthetic techniques are widely used for cataract surgery. In patients undergoing ophthalmic surgeries under local anesthesia, major complications are rare [1]. From these techniques, peribulbar anesthesia (PBA) has been very successful over the retrobulbar anesthesia seen its effectiveness and safety [2,3]. Despite this success, varying complications during PBA have been described [1,4,5]. Life-threatening complications include myocardial ischemia, cardiovascular depression, respiratory arrest, and convulsions [6-9]. We present a case in which generalized tonic-clonic convulsions developed during PBA and we discuss the possible mechanisms of central nervous system (CNS) spread and possible means of prevention.

CASE REPORTS

A 70-year-old Indian female was admitted for cataract extraction and lens implant in her left eye. There was no history of diabetes mellitus, hypertension, or any other significant medical illness in the past, and there was no personal or family antecedent of allergy, epilepsy, or taking food or toxic drug. A routine preoperative evaluation showed blood pressure of 130/78 mmHg and heart rate of 82 beats/min, bilaterally clear chest. Urine dipstick test for sugar was non-reactive.

Preoperative topical antibiotic drop and oral administration of a prophylactic antibiotic (ciprofloxacin 500 g) were given to her. Lignocaine sensitivity was done and showed no abnormal reaction. On the operating table, after swabbing and explanation of the procedure, PBA was given with two injections of 3 ml each (anesthetic mixture of lidocaine 2%, mixed with hyaluronidase 50 IU/ml and adrenaline 1:200,000) in primary gaze position. The first injection was given in her lower temporal peribulbar space and the second injection in her upper nasal peribulbar space with a needle of 24 GA, 25 mm, ¾ inch (Dispo van®, Hindustan syringes and medical devices, India) after a negative aspiration test and followed by manual compression of her globe for 5 min. The eye was draped.

About 5 min after local anesthetic injection, some movement was noted in patient’s hand; the drape was removed and up rolling was noticed in her contralateral eye which proceeded to a generalized tonic-clonic seizure within a minute. The seizure subsided after few seconds and blood pressure (BP) was 170/100 mmHg with the irregular heartbeat of 98/min. The physician was called and she was immediately shifted to medicine ward. Her CNS examination revealed bilaterally reduced plantar response with dilated contralateral (right) pupil not responding to light and a diagnosis of generalized tonic-clonic seizure was made. The seizures were controlled with injection phenytoin and maintenance phenytoin therapy was started.

However, her BP was started falling continuously even after receiving 3 boluses of normal saline. Injection dobutamine infusion was started but her condition did not improve. Electrocardiogram showed sinus bradycardia and she was shifted to the intensive care unit so as to provide the critical care with continuous monitoring of her vitals. Her capillary blood glucose was 136 mg/dL, axillary temperature was 36.5°C, and her serum electrolytes were normal. Her complete blood counts were within normal limits, arterial blood gasses were in normal range and she was semi-conscious.

As BP was not maintained with dobutamine, dopamine was also added with the gradual increment in doses. However, there was no improvement, so noradrenaline infusion was also added,
and BP started showing improvement. These three vasopressors were continued for the next 3 days with the dose modifications as per the requirements. After 5 days of continuous vasopressor therapy and oxygen inhalation, patient starting recovering gradually. The drugs were tapered off and she was shifted to the general ward. A brain computed tomography was unremarkable. On monitoring, the patient for 2 days in general medicine ward and confirming her stability, she was discharged.

**DISCUSSION**

Peribulbar block produces optimal conditions for the performance of eye surgery. A rare but serious systemic complication related to PBA is generalized seizures. Davis and Mandel found only 1 major complication (0.006%), a grand mal seizure that might have been related to systemic toxicity in 16,224 patients who received a peribulbar block [1]. During surgery under local anesthesia, convulsions may have several causes such as hypoglycemia, medication errors, stroke, and severe hypoxia caused by deep sedation or following a cardiac arrest complicating cardiac ocular reflex and CNS intoxication by the spread of local anesthetic agent (Table 1). Several mechanisms may lead to CNS spread of local anesthetics during PBA.

One possible mechanism is an inadvertent intra-arterial injection in the ophthalmic artery or its branches. Reversal of the direction of blood flow in the artery due to injection pressure causes the anesthetic solution to flow back into the internal carotid artery and is delivered to the thalamus and other midbrain structures [10-12]. Because of its anomalous position of lying inferior to the optic nerve [20,21]. Absorption of the local anesthetic agent by the arachnoid villi may cause its spread to cerebral structures [22]. Thereby, an accidental injection through these envelopes may spread the local anesthetic under arachnoid or subdural space to brain structures can cause a direct intoxication of brain structures. The size of the needle used and the position of the eye at the time of injection are the main risk factors for this mechanism. With the globe in superonasal gaze during injection, the optic nerve is in close proximity to the introduced needle [9]. The risk of accidental puncture of the envelope of the optic nerve in superonasal gaze position is high as compared to the primary gaze position.

The distance between the temporal region and lower optical foramen is between 42 mm and 54 mm. With the needles of shorter size between 16 mm and 25 mm and high volume of anesthetic agent, the risk of puncture of the optic nerve is low in comparison with the use of long needles of 31 mm or more, which provide an immediate anesthesia with a low volume of the anesthetic agent but increase the risk of puncture of the optic nerve [20,21]. Absorption of the local anesthetic agent by the arachnoid villi may cause its spread to cerebral structures [22]. Manual compression and the use of hyaluronidase increase the risk for it.

Capillary blood glucose of 136 mg/dl in our patient ruled out hypoglycemia as the cause of the seizure. Injected drugs were verified again and showed no medication error. Laboratory analysis showed no metabolic disorder. There was no neurological deficit after awakening, and her brain computed tomography was normal eliminating a cerebral stroke. Hence, the seizures were probably related to the peribulbar technique. A negative aspiration

**Table 1:** Possible mechanisms, their signs and modes of prevention for convulsions after peribulbar block

<table>
<thead>
<tr>
<th>Causes</th>
<th>Symptoms</th>
<th>Management</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oculocardiac reflex</td>
<td>Bradycardia/cardiac arrest</td>
<td>Release of muscle tension</td>
<td>Gentle muscle tractions</td>
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<td></td>
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<td>Injection of atropine</td>
<td>Injection of atropine</td>
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<tr>
<td>Drug errors</td>
<td>Tachycardia/bradycardia/convulsions (depends on type of drugs injected)</td>
<td>Symptomatic treatment</td>
<td>Labeling of syringes</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Sweating, drowsiness, confusion, abnormal behavior, and convulsions</td>
<td>Administration of serum glucose (10%)</td>
<td>Verification of syringes before injection</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Desaturation, consciousness disorders ... convulsions</td>
<td>Oxygen therapy, liberation of superior airways, intubation</td>
<td>Avoid hypoglycemic drugs in the morning of surgery</td>
</tr>
<tr>
<td>Stroke</td>
<td>Consciousness disorders, respiratory distress, hemodynamic instability, and convulsions</td>
<td>Control of airway, control of hemodynamic parameters</td>
<td>Perioperative monitoring of blood glucose</td>
</tr>
<tr>
<td>Toxicity of local anesthetics</td>
<td>Somnolence, diplopia, shivering</td>
<td>Oxygen therapy</td>
<td>Monitoring of oxygen saturation</td>
</tr>
<tr>
<td></td>
<td>Difficult speaking, arrhythmias</td>
<td>Airway liberation</td>
<td>Systematic supplemental oxygen during sedation</td>
</tr>
<tr>
<td></td>
<td>Convulsions, coma</td>
<td>Anti-convulsants</td>
<td>Monitoring of sedation</td>
</tr>
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</table>

Other proposed mechanism is inadvertent brainstem anesthesia [14-17]. Studies have shown that there is communication between the cerebral structures and the optic nerve, through the three envelopes surrounding the optic nerve, which are an extension of the cerebral meninges [18,19]. Thereby, an accidental injection through these envelopes may spread the local anesthetic under arachnoid or subdural space to brain structures can cause a direct intoxication of brain structures. The size of the needle used and the position of the eye at the time of injection are the main risk factors for this mechanism. With the globe in superonasal gaze during injection, the optic nerve is in close proximity to the introduced needle [9]. The risk of accidental puncture of the envelope of the optic nerve in superonasal gaze position is high as compared to the primary gaze position.

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test during the technique and the delayed onset of convulsions after the block are in favor of the second mechanism (inadvertent brainstem anesthesia) and against the first mechanism (intraarterial injection). Whatever be the mechanism involved in this complication, prevention is necessary to avoid the occurrence of such major life-threatening complications.

The presence of anesthetists always remains required during the local anesthetic procedures for the management of complications. Standards of anesthetic safety in ophthalmic surgery should be maintained strictly [23,24]. Monitoring of vitals during ophthalmic local anesthesia should be there, and all technical and human resources must be available, in the operative room, to deal with the occurrence of any complications [25]. Regular practice of needle test aspiration before each injection, maintaining the globe in primary gaze during the injection and the use of shorter needles <25 mm are the other preventive measures to avoid such complications.

Ultrasound could help in guiding the direction of the needle during the technique, in seeing the injection sites, in reducing the volume of injected drug, and in reducing the complication rate and improving the safety and performance of these techniques. These benefits have already been demonstrated in local anesthesia for other surgeries; in ophthalmic surgery, the use of ultrasound for the realization of local anesthesia is in full development [26-28].

CONCLUSION

Complications may arise during local anesthetic techniques which if not recognized early and resuscitated, can be fatal. The mastering and perfecting of these techniques by practitioners need of trained personnel and compliance with safety standards in anesthesia are the only way to guarantee the prevention of such complications.

REFERENCES


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