

Brainstem anesthesia: A rare complication after peribulbar anesthesia

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ABSTRACT

Brainstem anesthesia (BSA) is a rare but serious complication of the peribulbar block. Fewer than ten cases have been reported in the literature. We report a case of BSA after the peribulbar block for cataract surgery. The patient developed convulsion and respiratory arrest 5 min after injection of 7 ml of 2% lignocaine using a 25 mm (mm) length of 24 gauge (G) needle. The patient was successfully managed with prompt recognition and mechanical ventilation for about 1 h. Cataract surgery was performed 3 months after the adverse event. This time, the patient had an uneventful recovery. The patient was well at the 6-month follow-up.

Key words: Brainstem anesthesia, Convulsion, Local anesthesia, Peribulbar block

The peribulbar block is one of the regional anesthesia techniques used in ophthalmic surgery. As local anesthetic agents are administered into the extraconal compartment of the eye in a peribulbar block, the risk of optic nerve damage is avoided. The peribulbar block is frequently preferred for its low rate of complications despite its disadvantages such as requiring more than one injection and a larger volume of the local anesthetic agent [1]. The rate of major complications after peribulbar anesthesia was reported to be 0.006% in patients undergoing ophthalmic surgery [2,3]. Although the peribulbar block is theoretically considered to be much safer and easier to apply, some cases of brainstem anesthesia (BSA) after peribulbar block have been reported in the literature [4-10]. Herein, we report a case of respiratory depression and convulsion caused by probable BSA due to peribulbar block that was administered before cataract surgery.


CASE REPORT

A 55-year-old man with bilateral immature senile cataract was admitted at our institution for phacoemulsification of the right eye under local anesthesia (LA). He was hemodynamically stable at admission and had no comorbid condition. Preliminary laboratory parameters were within normal limits.

The peribulbar block was performed with a 25 mm 24 gauge (24G) needle. A total 7 ml of 2% lignocaine (mixture

of lidocaine 2%, with hyaluronidase 50 IU/ml, and adrenaline 1:200,000) was injected in primary gaze position after prior aspiration to see any blood or cerebrospinal fluid. Three minutes after the injection, the patient was not responding to verbal commands. His oxygen saturation was reduced to 70% and the radial pulse was 56/min. He was put on face mask oxygen at a rate of 5 l/min. He developed convulsion for about 5 min after the peribulbar block. We immediately sought the help of an anesthetist. The patient was intubated after intravenous injection of 2 mg of midazolam. The patient was shifted to the intensive care unit for mechanical ventilation. Arterial blood gas showed a pH of 7.34, PCO₂ of 35 mm of Hg, and PO₂ of 95 mm of Hg. Blood sugar was 85 mg/dl. The patient was extubated (about 60 min after intubation) once the patient was conscious and hemodynamically stable with normal oxygen saturation. There was no residual neuro deficit. Moist oxygen inhalation was continued for 2 h and monitoring for the rest of the day.

Subsequently, cardiology, and neuro-medicine consultation were sought. Electrocardiography and echocardiography reports were within normal limits. Magnetic resonance imaging of the brain showed cerebral cortical atrophy (Fig. 1) and there was no mass lesion or intracranial hemorrhage. After complete evaluation and exclusion of other causes of convulsion and respiratory arrest, the patient was diagnosed as a case of BSA. Cataract surgery was performed 3 months after the previous adverse event. Post-operative recovery was uneventful. The patient was well at the 6-month follow-up.

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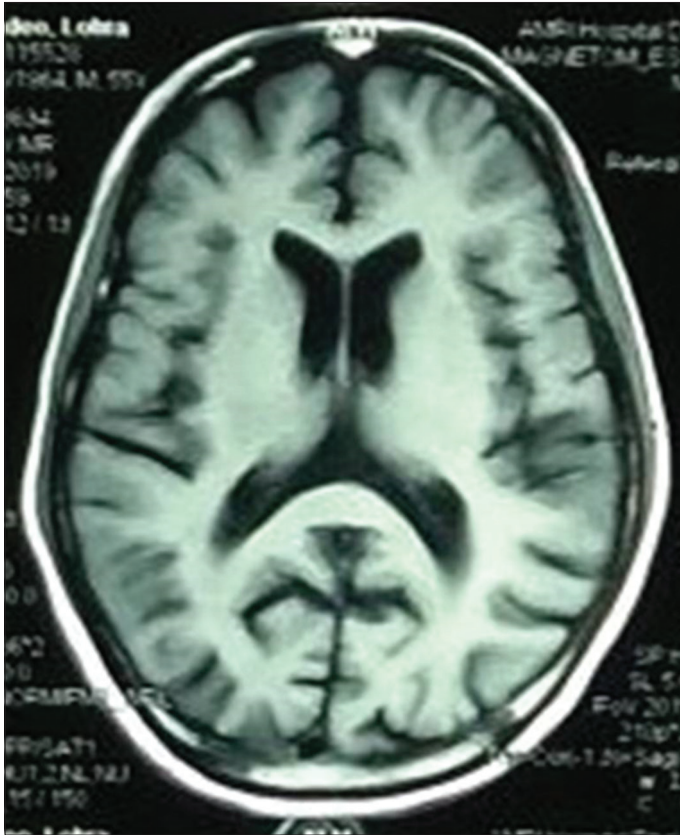


Figure 1: Magnetic resonance imaging of the brain showing cerebral cortical atrophy

DISCUSSION

BSA is a rare but serious complication of the peribulbar block. To the best of our knowledge, fewer than ten cases have been reported in the literature [4-10].

BSA may result from the spread of local anesthetic agent into the central nervous system (CNS) due to inadvertent perforation of the optic nerve sheath (which are extensions of the cerebral meninges) by the needle tip or by puncture of the orbital artery where the retrograde flow of anesthetic agent from orbital artery to the internal carotid artery can affect the midbrain [8,11]. The route of spread was demonstrated by Drysdale in a cadaver model by injecting 3 ml radiopaque dye after puncture of the optic sheath [12]. The contrast material could be traced along the nerve sheath to the optic chiasm, ultimately reaching the pons and midbrain. Another mechanism that has been postulated is the absorption of LA by the arachnoid villi and spread to cerebral structures [13] which may occur due to the manual compression after the block and the use of hyaluronidase along with LA.

Irrespective of the mechanism of BSA, the patient may present with shivering, vomiting, irregularities in respiratory pattern, respiratory arrest, temporary hemiplegia, bilateral brainstem nerve palsies, aphasia, and generalized convulsion [3,5]. The CNS manifestations after inadvertent LA injection depend on the quantity of injected LA, the depth of needle insertion, force with which it is injected, concentration of LA, and the area into which it spreads. Typically, symptoms of BSA appear 5–10 min after injection of LA but presentation may be delayed up to 40 min.

In the majority of the cases, a transient and self-limited course has been observed. Initially, features of the parasympathetic blockade and sympathetic hyperactivity may be more evident. Signs of sympathetic blockade may appear later on. Serious neurological symptoms such as agitation, confusion, convulsion, nystagmus, apparent shivering, and loss of consciousness may occur due to the passage of LA through the blood–brain barrier and blockage of inhibitory pathways. Sympathetic hyperactivity may develop due to the involvement of medulla oblongata leading to excitatory stimulation of vasomotor, respiratory, and vomiting centers resulting in arrhythmias, hypertension, and vomiting [14]. Later, sympathetic blockade along with hypotension and apnea may follow [15]. The common presenting features are usually loss of consciousness, respiratory depression, hypotension along with contralateral ophthalmoplegia, and amaurosis. Other manifestations such as aphasia, vertigo, hearing loss, slurred speech [16], dysarthria with paralysis of contralateral sixth nerve [17], paralysis of the third cranial nerve of the contralateral side [10], and localized convulsions of the ipsilateral face followed by hemiparesis [18] have also been reported in the literature.

In our patient, the presentation was a loss of consciousness and convulsion which developed within 5 min followed by respiratory arrest. Other causes of convulsion after LA were excluded such as hypoglycemia, medication errors, stroke, severe hypoxia caused by deep sedation, or following a cardiac arrest complicating cardiac ocular reflex. He did not develop any serious cardiovascular event. Moreover, the patient had complete recovery including full neurological functions 1 h after the adverse event. The sequence of events and their timing points toward a case of BSA. In this respect, our case was similar to that of Bensghir *et al.* [8]. Paul *et al.* [9] reported a case of BSA after the peribulbar block where the presentation was convulsion similar to ours, without respiratory arrest followed by prolonged hypotension requiring vasopressor agents.

As BSA is a potentially life-threatening complication of the peribulbar block, every attempt should be made to prevent it. The incidence of the CNS complication with a 38 mm retrobulbar needle is between 0.2 and 0.3% [19]. Needle size shorter than 30 mm can decrease the risk of complications [8]. However in our case, BSA occurred after peribulbar block with a 25 mm (24G) needle, similar to that of Gomez *et al.* [5] and Kazancıoğlu *et al.* [6]. The complication can be, to some extent prevented by avoiding injection too deeply in the eye, aspiration before injection of LA, infiltration of LA in primary gaze position (optic nerve is slack in this position [1], which allows the nerve to be pushed aside), wiggle test [20] (needle is moved from side to side after the needle is introduced into the orbit, but before any LA is injected, any movement of the globe suggests penetration of the sclera, optic nerve, or an extraocular muscle) and the use of ocular ultrasonography to visualize the orbital structure and the spread of local anesthetics [21].

Once the patient develops BSA, prompt recognition and supportive treatments are essential to save the patient. Symptoms

of BSA appear usually 5–10 min after the administration of peribulbar block up to 60–90 min, after which recovery starts. There may be prolonged residual effect for a few hours; therefore, the patient should be monitored in the intensive care unit. Specific management depends on the manifestations and may include intravenous fluid resuscitation, inotropic circulatory support, use of benzodiazepine or barbiturates for seizure control, intubation, and mechanical ventilation in case of respiratory arrest. Vital parameters and oxygen saturation should be maintained with supportive therapy till recovery. In severe cases, the patient may need airway management and cardiopulmonary resuscitation. Our patient also required intravenous fluid, intravenous midazolam, a short-acting benzodiazepine, and ventilatory support as he developed convulsion and respiratory arrest.

CONCLUSION

Although BSA after the peribulbar block is a rare event, its possibility should be kept in mind. In common ophthalmic surgeries such as cataracts, strabismus, and trabeculectomy, patient monitoring should not be disregarded due to the short duration of the surgery or a large number of patients to be operated on. The importance of adequate patient monitoring during common ophthalmic surgeries such as cataracts, strabismus, trabeculectomy, and vitrectomy should not be ignored due to the burden of a large number of patients to be operated on or short duration of these surgeries. The peribulbar block should be performed by an experienced surgeon taking all the precautions and the operation room should be equipped with basic resuscitation instruments.

REFERENCES

- Alhassan MB, Kyari F, Ejere HO. Peribulbar versus retrobulbar anesthesia for cataract surgery. *Cochrane Database Syst Rev* 2015;2:CD004083.
- Yasar T, Çağlar Ç. Local injection anesthetics in ophthalmic surgery review. *Turk J Ophthalmol* 2009;39:496-505.
- Davis DB 2nd, Mandel MR. Efficacy and complication rate of 16,224 consecutive peribulbar blocks. A prospective multicenter study. *J Cataract Refract Surg* 1994;20:327-37.
- Eke T, Thompson JR. Serious complications of local anesthesia for cataract surgery: A 1 year national survey in the United Kingdom. *Br J Ophthalmol* 2007;91:470-5.
- Boret H, Petit D, Ledantec P, Bénédicte S. Brainstem anesthesia after peribulbar anesthesia. *Ann Fr Anesth Reanim* 2002;21:725-7.
- Gomez RS, Andrade LO, Costa JR. Brainstem anesthesia after peribulbar anesthesia. *Can J Anaesth* 1997;44:732-4.
- Kazancıoğlu L, Batçık Ş, Kazdal H, Şen A, Gediz BŞ, Erdivanlı B, *et al.* Complication of peribulbar block: Brainstem anesthesia. *Turk J Anaesthesiol Reanim* 2017;45:231-3.
- Bensghir M, Badou N, Houba A, Balkhi H, Haimour C, Azendour H. Convulsions during cataract surgery under peribulbar anesthesia: A case report. *J Med Case Rep* 2014;8:218.
- Paul G, Narula A, Srivastava PK, Bharambe M, Laskar ZH. Generalized seizures during cataract surgery following peribulbar block: A case report. *Indian J Case Rep* 2017;3:128-30.
- Jaichandran VV, Nair AG, Gandhi RA, Prateeba-Devi N. Brainstem anesthesia presenting as contralateral third nerve palsy following peribulbar anesthesia for cataract surgery. *Acta Anaesthesiol Taiwan* 2013;51:135-6.
- Hamilton RC. Brain-stem anesthesia as a complication of regional anesthesia for ophthalmic surgery. *Can J Ophthalmol* 1992;27:323-5.
- Drysdale DB. Experimental subdural retrobulbar injection of anesthetic. *Ann Ophthalmol* 1984;16:716-8.
- Shantha TR. The relationship of retrobulbar local anaesthetic spread to the neural membranes of the eyeball, optic nerve, and arachnoid villi in the optic nerve. *Anesthesiology* 1990;72:A849.
- Palte HD. Ophthalmic regional blocks: Management, challenges, and solutions. *Local Reg Anesth* 2015;8:57-70.
- Hamilton RC. Brain stem anesthesia following retrobulbar blockade. *Anesthesiology* 1985;63:688-90.
- Palte HD, Hoa DP, Canseco AP. Surdity in the OR: An unusual case of brainstem anesthesia. *Case Rep Anesthesiol*. 2017;2017:4645381.
- Rosen WJ. Brainstem anesthesia presenting as dysarthria. *J Cataract Refract Surg* 1999;25:1170-1.
- Pragt E, van Zundert AA, Kumar CM. Delayed convulsions and brief contralateral hemiparesis after retrobulbar block. *Reg Anesth Pain Med* 2006;31:275-8.
- Carneiro HM, Oliveira B, Avila MP, Neto OA. Anestesia do tronco encefálico após bloqueio retrobulbar extraconal: É possível evitar? Relato de caso [Brainstem anesthesia after extraconal retrobulbar block: can it be avoided? Case report]. *Rev Bras Anesthesiol* 2007;57:391-400.
- Kimble JA, Morris RE, Witherspoon CD, Feist RM. Globe perforation from peribulbar injection. *Arch Ophthalmol* 1987;105:749.
- Luyet C, Eichenberger U, Moriggl B, Remonda L, Greif R. Real-time visualization of ultrasound-guided retrobulbar blockade: An imaging study. *Br J Anaesth* 2008;101:855-9.

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