

Contralateral Amaurosis After Retrobulbar Block For Vitrectomy Presenting Solely with Hypertension and Tachycardia

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Citation

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Abstract

Retrobulbar block is a technique to provide local anesthesia for ophthalmologic surgery. Complications of this technique may be serious and potentially life-threatening. We report the rare complication of contralateral amaurosis presenting solely with hypertension and tachycardia and discuss its differential diagnosis and evaluation, the proposed mechanism of central spread of local anesthetic, and the technical factors of the block that may contribute to this complication. Immediate recognition of the clinical signs of central nervous system spread of the local anesthetic is essential. This immediate recognition allows the rapid treatment and support of the patient in the case of progression from contralateral amaurosis to more severe complications.

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INTRODUCTION

Retrobulbar block is a local anesthetic technique used for a variety of ophthalmologic procedures. Potential complications of this technique range from injury to the eye during injection to the spread of local anesthetic to the central nervous system. Many anesthesiologists are unaware of these potential complications, since most of them are published in the ophthalmic literature. Immediate recognition of the clinical signs of central nervous system spread of the local anesthetic is essential. This immediate recognition allows the rapid treatment and support of the patient in the case of the progression from contralateral amaurosis to more severe and life-threatening complications such as cardiorespiratory arrest.

We report the rare complication of contralateral amaurosis after retrobulbar block for vitrectomy presenting solely with hypertension and tachycardia and we discuss its differential diagnosis and evaluation, the proposed mechanism of its occurrence, and the technical factors of the block that may contribute to this complication.

CASE REPORT

A 43-year-old man with a left eye vitreous hemorrhage and

tractional retinal detachment underwent a left eye pars plana vitrectomy and membranectomy under monitored anesthesia care. The patient reported a history of hypertension, diabetes mellitus, diabetic retinopathy, and hyperlipidemia. The patient reported no allergies and his medications consisted of insulin, atorvastatin, pioglitazone, valsartan, lansoprazole, and aspirin. The patient had undergone two prior right eye vitrectomies for vitreous hemorrhage and proliferative diabetic retinopathy without complications, one and two years previously. Physical examination was essentially normal except for his ophthalmologic findings.

In the preoperative holding area, the patient received one milligram of midazolam intravenously, and 1% cyclopentolate (Cyclogel®) and 2.5% neosynephrine eye drops to the left eye (one drop every five minutes x three). The patient was then brought to the operating room where standard noninvasive ASA monitors were placed, as well as a nasal cannula connected to a capnograph. The eyes were placed in the neutral position with the gaze directed straight ahead. A left retrobulbar block was performed by the ophthalmology service using 5cc of a 1:1 mixture of 0.75% bupivacaine and 2% lidocaine with 50 units of hyaluronidase and was administered with a 25-gauge, 1.5 inch, Atkinson needle. After evaluating the eye for akinesia, the ophthalmology service repeated the retrobulbar block. A total of 7.5 milliliters of the above mixture was used. A plastic shield was placed over the contralateral eye. A total

of 67.5 micrograms and 37.5 micrograms of remifentanyl were titrated intravenously prior to the initial and repeated retrobulbar blocks, respectively. Shortly after the block, the patient's blood

pressure and heart rate increased (blood pressure 199/108, pulse 98). The elevation of blood pressure and heart rate were treated with dexmedetomidine and labetalol. The blood pressure and heart rate subsequently returned to baseline (blood pressure 140's/ 80's, pulse 70-80's) and the vitrectomy was completed uneventfully in ninety minutes. In the recovery room, the patient informed the ophthalmologist that he could not see out of his right eye, the nonoperative eye. This finding was confirmed by an indirect ophthalmologic evaluation. The right pupil was fixed at 4 millimeters and was non-responsive to light. The patient was alert, awake, hemodynamically stable, and had no other signs and/or symptoms. However, after discussion with the anesthesiologist, the ophthalmologist reexamined the patient and found that he also had anesthesia of his right eye. This reassured the ophthalmologist that a more severe cause of blindness was unlikely, but prompted the thought that central spread of the local anesthetic had occurred. A thorough ophthalmologic evaluation was carried out to rule out other causes of postoperative blindness. Indirect ophthalmoscopy showed good ocular blood flow and no signs of vitreous hemorrhage; dynamic ophthalmodynamometry showed good ocular blood flow; tonometry showed intraocular pressure 15mmHg; and fluorescein angiogram showed a 17 second arterio-venous transit time and good perfusion. In addition, there were no signs of hemiarterial or hemiretinal vein occlusions. Once the results of all the examinations proved to be normal, the patient was placed under observation. The patient began to see shadows and shapes, and his vision returned to its preoperative baseline three hours after the administration of the retrobulbar block.

DISCUSSION

Retrobulbar block is an anesthetic technique used for a variety of ophthalmologic procedures. It provides anesthesia, akinesia, analgesia, control of intraocular pressure, and postoperative analgesia. Complications may be serious and potentially life-threatening. These can range from injury to the eye during injection to the spread of local anesthetic to the central nervous system. Thus, potential complications from a retrobulbar block can include the following: retrobulbar hemorrhage¹, ecchymosis², globe penetration or perforation³, optic nerve atrophy, penetration of the optic

nerve sheath, oculocardiac reflex, ocular myotoxicity, amaurosis of the operative eye, seizures, retinal artery occlusion, bilateral hearing loss⁴, globe ischemia, and brainstem anesthesia. Brainstem anesthesia, the most serious complication of a retrobulbar block, can present with the following signs and symptoms: confusion, dizziness, blurred vision, ophthalmoplegia, tinnitus, dysphagia, respiratory depression to apnea, limb paralysis, shivering⁵, dysarthria⁶, nausea/vomiting, sudden changes in cardiovascular vital signs, and/or amaurosis of the contralateral eye. The onset of these symptoms after retrobulbar injection is variable and can range from 2 to 40 minutes after injection⁷. Our patient did have a transient, sudden increase in blood pressure and heart rate and, in retrospect, this may have been an initial sign of central spread of local anesthetic. Fortunately, the local anesthetic did not spread more centrally and progress to more severe and life-threatening complications. Hamilton proposed that the symptoms of hypertension and tachycardia with brainstem anesthesia occur when local anesthetic tracks beneath the dura of the optic nerve sheath and enters the cerebrospinal fluid at the cranial end of the neuroaxis, causing neuroaxial blockade⁸. In

addition, he speculated that there is vagal blockade at the brainstem and abolition of the carotid sinus reflex⁸. This is, to the best of our knowledge, the only reported case of contralateral amaurosis after retrobulbar blockade presenting solely with hypertension and tachycardia.

Nearly 16 out of 6000 patients have been reported to develop signs and symptoms, including contralateral transient amaurosis, caused by local anesthetic directly spreading to the central nervous system after retrobulbar block⁷.

Contralateral transient amaurosis is, thus, a rare complication of retrobulbar block^{7,9-11,15-21}. However, its true incidence may be higher than previously thought. For example, it is difficult to recognize this complication when the patient is under a combined general anesthetic and retrobulbar block, or when the nonoperative

eye has a preexisting visual loss. Furthermore, since the contralateral eye is usually draped or shielded, a nonprogressive amaurosis may go unrecognized by the patient if the amaurosis resolves by the end of surgery.

The proposed mechanism responsible for this complication is the injection of local anesthetic into the subdural space of the optic nerve sheath, passing along the ipsilateral optic nerve sheath to the optic chiasm. As the anesthetic passes to

the area of the optic chiasm, it

interferes with the conduction in the contralateral optic nerve¹⁵. An air bubble in the ipsilateral optic nerve sheath after retrobulbar block shown under computed tomographic scan supports this intra-nerve sheath injection mechanism¹⁶. The mechanism of local anesthetic spreading into the optic nerve sheath, under the dural sheath of the optic nerve¹⁵ has been demonstrated by

contrast radiography¹³ and by the recovery of lidocaine and bupivacaine from the cerebrospinal fluid of patients who developed respiratory arrest¹⁴.

Technical factors that may contribute to this complication are the following:

- 1) Not withdrawing the needle if greater resistance is felt on placement of the needle. When this occurs, one must expect to have entered the optic nerve sheath.
- 2) The total dosage injected; however, after respiratory arrest, blood levels of local anesthetics have been measured at intervals and have been shown to be similar to those subjects who had a normal response²².
- 3) The administration of hyaluronidase which might enhance the spread of
4. local anesthetics centrally²³; however, the spread of local anesthetic into the central nervous system after retrobulbar block has been reported even without hyaluronidase⁹.
- 4) The type of needle. A blunt needle may be less likely to penetrate the optic nerve sheath; however, central spread of local anesthetic has been reported with a blunt needle⁹;
- 5) The gaze on placement of the retrobulbar block to a straight ahead fixation can reduce the incidence of this complication but it is not foolproof⁷.

It is unclear why some anesthetics enter the subdural space of the optic nerve sheath and others enter the subarachnoid space. The depth of needle insertion, force, concentration, volume of injection, and other anatomic variations may play a role.

CONCLUSION

In this patient, amaurosis was noted at the end of surgery and contralateral amaurosis suggested that central spread of the local anesthetic occurred. For a complete evaluation of the

patient and to rule out other causes, we considered other differential diagnoses including stroke, central retinal artery occlusion, retrobulbar hemorrhage, seizures, hysteric reaction, and transient cardiac arrest^{9,10-11,17}. The patient, however, had no signs that suggested a stroke, a seizure, transient cardiac arrest, or a hysteric reaction. The patient was alert, awake, and hemodynamically stable. Further ophthalmologic evaluation excluded other ophthalmologic etiologies. The patient's vision returned to his preoperative baseline three hours after the administration of the retrobulbar block. We know of no reports of permanent contralateral visual loss after injection of local anesthetic in the optic nerve sheath.

In summary, complications of ophthalmologic nerve blocks are rare yet can have serious and life-threatening consequences. A thorough understanding and the immediate recognition of these potential complications will allow the rapid treatment and support of the patient.

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