Multimodal imaging findings in Purtscher-like retinopathy after retrobulbar anesthesia

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Introduction: This is a case of Purtscher-like retinopathy with an unusual etiology of retrobulbar anesthesia, evidenced by optical coherence tomography-angiography (OCT-A) images.

Patient and Clinical Findings: After cataract surgery with a retrobulbar block, a 65-year-old woman experienced sudden dramatic visual reduction with superficial white retinal patches on fundus examination and corresponding capillary bed infarcts on fluorescein angiography and OCT-A.

Diagnosis, Intervention, and Outcomes: Using advanced OCT-A, fluorescein angiography imaging, and fundus photography, the patient was diagnosed with Purtscher-like retinopathy. The patient was treated with topical steroids, nonsteroidal anti-inflammatory drops, and antibiotic drops, with gradual and nearly complete resolution of visual acuity.

Conclusions: This case represents a rare but possible complication of retrobulbar anesthesia associated with cataract surgery. Despite dramatic changes on OCT-A, and in contrast to other reports with similar characteristic findings, our patient regained significant vision.

Purtscher retinopathy is a vaso-occlusive syndrome characterized by ischemic areas of retinal whitening in a peripapillary pattern occurring after severe trauma, such as chest compressions, long-bone injury, or head injury.1,2 While the pathophysiology is still debated, embolism and precapillary arteriolar occlusion by complement-mediated white blood cell aggregation have been proposed as possible mechanisms.3 Purtscher-like retinopathy describes this phenomenon from causes other than concussive trauma, such as fat or amniotic fluid embolism, acute pancreatitis, pre-eclampsia, or systemic vasculitides.3 In this case report, we describe a case of Purtscher-like retinopathy after uneventful retrobulbar anesthesia during routine cataract surgery. This case is the first of retrobulbar anesthetic origin to support its findings with unique advanced imaging modalities. Optical coherence tomography-angiography (OCT-A) images allow for definition of characteristic patterns in Purtscher-like retinopathy and therefore a deeper understanding of its pathogenesis.

CASE REPORT

A 65-year-old woman with hypertension, hypothyroidism, and no ocular history presented to resident clinic with symptomatic bilateral cataracts and an initial visual acuity (VA) of 20/50 in the right eye and 20/40 in the left eye. The patient underwent uncomplicated cataract surgery in the right eye, which improved to a corrected distance VA of 20/20.

3 months later, the patient underwent cataract surgery in the left eye. Intravenous sedation was given to the patient prior to a retrobulbar block solution consisting of 5 mL of lidocaine 2%, 5 mL of bupivacaine 0.75%, and 200 units of hyaluronidase. The solution was injected with a disposable 25-gauge, 1.25-inch blunt needle into the left inferotemporal retroorbital space. Gentle external pressure was then applied to the eye. Cataract extraction with phacoemulsification and posterior chamber intraocular lens implantation was performed without complication. The wounds sealed easily with minimal stromal hydration at the conclusion of the surgery. There were no intraoperative clinical signs of a stark rise in intraocular pressure (IOP).

On postoperative day 1, however, the patient was found to have VA of counting fingers and a relative afferent

Patient Consent Statement

The medical team takes full responsibility in the confidentiality of the patient’s data. The authors have followed HIPAA regulations.
pupillary defect in the left eye. Anterior slitlamp examination revealed mild corneal edema and trace cell reaction with unremarkable incisions and a well-centered intraocular lens. Fundus examination showed numerous areas of retinal whitening, intraretinal hemorrhages, and massive macular edema extending temporally from the optic nerve (Figure 1, A). OCT was performed and demonstrated subretinal fluid and intraretinal edema (Figure 1, B). Fluorescein angiography (FA) revealed large areas of capillary dropout with late leakage, corresponding to areas of retinal whitening. OCT-A showed inner retinal capillary bed ischemia and outer retina/choriocapillaris hypointensity in a honeycomb-like pattern (Figure 2, A–C), similar to a case in the literature.4 A presumed diagnosis of Purtscher-like retinopathy was made, and the patient was given topical steroids, nonsteroidal anti-inflammatory drops, and antibiotic drops.

By postoperative day 7, the VA had dramatically improved to 20/80-2 with reduction in macular edema and marked retinal reperfusion on FA. Dilated fundus examination continued to show characteristic patches of retinal whitening or capillary bed infarcts, also referred to as Purtscher flecken, with few intraretinal hemorrhages in a peripapillary pattern. The honeycomb ischemic pattern on OCT-A in the deeper layers corresponded anatomically with the overlying Purtscher flecken. Macular OCT showed gradual resolution of subretinal fluid each week, and serial FAs demonstrated improved perfusion. Additional OCT findings included hyperreflectivity of the inner nuclear layer nasal to the fovea, corresponding with the location of Purtscher flecken. OCT-A and FA continued to depict nasal choroidal nonperfusion, delayed arterial filling, and macular ischemia. Despite some generalized depression on Humphrey visual field testing, the corrected distance VA dramatically recovered to 20/25- over the next few weeks.

DISCUSSION

This case represents both an extremely rare complication of retrobulbar anesthesia and an unusual etiology of Purtscher-like retinopathy. As with previous reports of Purtscher-like retinopathy, the patient presented with sudden dramatic visual reduction to counting fingers.3 The fundus examination showed characteristic findings of superficial white retinal patches and corresponding capillary bed infarcts on FA and OCT-A. The pathogenesis of Purtscher flecken is attributed to the precapillary arterioles,
which stop 50 μm around the retinal arterioles. Occlusion of these arterioles leads to ischemic patches clearly demarcated by normal retina adjacent to the vessels. Imaging in this case shows the lobulated pattern of ischemia extending like a honeycomb stencil from superficial to deep retinal layers.

The advanced imaging supports 2 main theories for the pathogenesis of the infarction seen in Purtscher-like retinopathy. One plausible hypothesis is the introduction of air microemboli through direct contact of the needle with the posterior ciliary or central retinal artery, causing an intermittent occlusion of precapillary arterioles. Another possibility relates to ischemic intervals due to an increase in IOP. It has been established that peribulbar and retrobulbar anesthesia techniques can lead to a rise in IOP, resulting in a drop in pulsatile ocular blood flow for a short period. This creates a period of relative ocular ischemia, to which precapillary arterioles may be more vulnerable. It is possible in our patient that either an introduction of a small embolus to the nerve sheath or an increased pressure on the optic nerve leading to a period of relative ischemia, or both, led to either compression or occlusion of precapillary arterioles. In addition, the patient had a short axial length of 21.87 mm in the left eye, which could have predisposed contact of the needle with these anatomical structures or heightened the intraocular pressure.

While Purtscher-like retinopathy after administration of retrobulbar anesthesia has been infrequently documented in the literature, in this case, we further detail notable features on OCT and OCT-A in association with this condition. There are few case reports of Purtscher-like retinopathy accompanied by OCT-A images, but, to our knowledge, none in association with retrobulbar anesthesia.

Similar to the imaging in the study by Li et al., our OCT-A showed poor perfusion of the inner retinal vascular plexuses and a honeycomb-like hypointensity in outer retina/choriocapillaris layers. These ischemic areas corresponded to the Purtscher flecken noted in the inner retina. Li et al. conjectured that this choroidal lobular infarction was the causative pathogenesis for lack of visual recovery in their patient. However, our patient’s OCT-A showed reduced blood flow in a pattern that more closely resembled the infarction in corresponding superficial retinal layers. Our patient also showed significant ischemia on initial imaging but regained vision almost completely, thus misaligning any prognostic inferences that could be made from OCT-A.

In the study conducted by Sharma et al., their patient’s OCT showed disorganization of the inner retinal layers nasal to the fovea with atrophy of the ganglion cell layer. This corresponded with their OCT-A, which showed the presence of multiple areas of capillary dropout in the nasal macula, more pronounced in the deep capillary plexus than in the superficial capillary plexus. While we saw nasal hyperreflectivity on the OCT of our patient, capillary dropout was much more pronounced in the superficial capillary plexus. The patients reported in the studies conducted by Li et al. and Sharma et al. did not regain vision in the affected eye, while our patient did.

We acknowledge that our patient did not report a decrease in vision immediately after the retrobulbar block; however, she was given intravenous sedation before the block and throughout the case. While the patient may have started to experience visual changes during the block, she was likely not awake enough to verbalize them. In addition, alternative cataract surgery complications that could be implicated in Purtscher-like retinopathy include elevation of IOP. However, this patient’s surgery was uneventful and performed with routine phacoemulsification settings. IOP fluctuations were at a maximum setting of 65 mm Hg, with no overt clinical signs of intraoperative IOP spike, such as corneal edema or a firm globe. Therefore, we conclude that the retrobulbar block was the likely inciting factor in this case.

We report this case to bring awareness to Purtscher-like retinopathy as a rare but possible complication of retrobulbar anesthesia. This procedure was performed in a resident clinic setting, where retrobulbar anesthesia is routinely administered for cataract surgery. While the rise of topical anesthesia use in private practice mitigates the risk for such complications, we advocate for trend away from retrobulbar injections in teaching institutions as well.

**WHAT WAS KNOWN**

- Purtscher-like retinopathy describes a vaso-occlusive phenomenon caused by etiologies other than concussive trauma, such as fat embolism.
- Cases of Purtscher-like retinopathy after peribulbar and retrobulbar anesthesia have been infrequently described in the literature, believed to be due to the introduction of a small embolus to the nerve sheath or an increased pressure on the optic nerve, leading to a period of relative ischemia.

**WHAT THIS PAPER ADDS**

- Few case reports have described Purtscher-like retinopathy accompanied by OCT-angiography images, and, to the authors’ knowledge, this is the first to do so in association with an etiology of retrobulbar anesthesia. The images corroborate characteristic imaging features of Purtscher-like retinopathy.
- This study suggested that short axial length could be a contraindication to retrobulbar anesthesia use, recommending a need to dissuade this anesthetic practice.

**REFERENCES**

1. Purtscher O. Noch unbekannte Befunde nach Schadeltrauma. Ber Dtsch Ophthalm Ges 1910;36:294–307
2. Blodi BA, Williams CA. Purtscher-like retinopathy after uncomplicated administration of retrobulbar anesthesia. Am J Ophthalmol 1997;124:702–703
3. Agrawal A, McKibbon MA. Purtscher’s and Purtscher-like retinopathies: a review. Surv Ophthalmol 2006;51:129–136
4. Li B, Li D, Chen Y. Purtscher-like retinopathy presented a honeycomb-like pattern in optical coherence tomography angiography. BMC Ophthalmol 2019;19:232
5. Watkins R, Beigi B, Yates M, Chang B, Linardos E. Intraocular pressure and pulsatile ocular blood flow after retrobulbar and peribulbar anaesthesia. Br J Ophthalmol 2001;85:796–798

6. LeMagne JM, Michiels X, Van Causenbroeck S, Snyers B. Purtscher-like retinopathy after retrobulbar anaesthesia. Ophthalmology 1990;97:859–861

7. Lim BA, Ang CL. Purtscher-like retinopathy after retrobulbar injection. Ophthalmic Surg Lasers 2001;32:477–478

8. Cho HK, Lee D, Lee WK, Shin CH, Ryu JW. Purtscher-like retinopathy after retrobulbar anaesthesia in a patient with an intraconal mass. Can J Ophthalmol 2010;45:441–560

9. Sharma A, Reddy YC, Shetty AP, Kader SM. Electric shock induced Purtscher-like retinopathy. Indian J Ophthalmol 2019;67:1497–1500

10. Vezzola D, Allegrini D, Romano MR, Pagano L, Montericcio A, Fogagnolo P, Rossetti LM, De Cilla S. Optical coherence tomography angiography in Purtscher-like retinopathy associated with dermatomyositis: a case report. J Med Case Rep 2019;13:206

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