Purtscher-like retinopathy: A rare complication of peribulbar anesthesia

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Purtscher and Purtscher-like retinopathy is a distinctive retinal syndrome characterized by ischemic retinal whitening in a peripapillary pattern. We report a case of Purtscher-like retinopathy in a healthy 64-year-old man after a routine peribulbar anesthetic injection for cataract surgery. Although peribulbar anesthesia is considered to be a safer alternative to retrobulbar anesthesia, it has been associated with unusual but grave complications including central retinal artery occlusion.

Key words: Cataract surgery, peribulbar anesthesia, Purtscher-like retinopathy

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Purtscher's retinopathy is an occlusive vasculopathy which, in 1912, was first described by Dr. Otmar Purtscher as a syndrome of sudden blindness associated with a distinctive fundoscopic picture characterized by multiple patches of superficial retinal whitening and intraretinal hemorrhages surrounding the optic disc in patients who have sustained head trauma.[1] Since its original description, Purtscher's retinopathy has been associated with traumatic injury, primarily blunt thoracic and head trauma, and numerous nontraumatic diseases. When there is a nontraumatic etiology, the correct designation is Purtscher-like retinopathy. Purtscher-like retinopathy has been reported in several diverse conditions including acute pancreatitis, childbirth, fat embolism, and connective tissue diseases such as systemic lupus erythematosus.[2-5]

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Case Report

A 64-year-old healthy man underwent phacoemulsification with intraocular lens implant on the left eye. Preoperative fundus examination was normal and his best-corrected visual acuity in the left eye was 6/24 because of nuclear sclerosis. The patient was administered a single uneventful peribulbar injection consisting of 5 ml of lidocaine 2% and epinephrine 1:200,000 using a 25-gauge 25 mm peribulbar needle through the temporal portion of the inferior lid. No external compression was applied to the eye. On the first postoperative day, the patient had no complaints and his visual acuity had improved to 6/6. However, on examination, a left afferent pupillary defect was noted. Dilated ophthalmoscopy of the left eye revealed several large confluent peripapillary patches of retinal whitening mimicking soft exudates (Purtscher Flecken) [Fig. 1a]. A diagnosis of Purtscher-like retinopathy was made based on the classical clinical picture. Fundus fluorescein angiography revealed multiple hypofluroscent areas in the peripapillary region corresponding to the areas of the soft exudates [Fig. 2]. Swept-source optical coherence tomography of the left eye revealed edema of the inner retinal layers with hyperreflectivity of the nerve fiber layers corresponding to the areas of retinal whitening [Fig. 3]. As the patient was symptomless, no treatment was initiated for the treatment of the retinopathy. The patient underwent a comprehensive medical screening including liver function tests, pancreatic enzyme assay, Carotid Doppler, and transthoracic echocardiography to rule out other causes of Purtscher-like retinopathy. Subsequent follow-ups at 2 and 4 weeks revealed gradual disappearance of the cotton-wool spots [Fig. 1b and c], resolution of the peripapillary scotoma and decrease in the afferent papillary defect.

Discussion

Unilateral Purtscher's retinopathy has been reported following facial trauma, periorbital steroid injection, and retrobulbar anesthesia. There have been two previous case reports of Purtscher-like retinopathy after retrobulbar anesthesia.[6,7] To the best of our knowledge, this is the first case to be reported following peribulbar anesthesia. Peribulbar anesthesia is known to be safer, but it has still shown to have the tendency to cause potentially dangerous complications including central retinal artery occlusion through the remote effects of the anesthetic agent, amount injected, speed of injection, and postinjection mechanical compression.[8]

The pathogenesis of Purtscher's retinopathy is still a subject of debate with several theories being forwarded since the initial mechanical theory proposed by Purtscher. The most currently accepted theory is microembolization, causing arteriolar precapillary occlusion and microvascular infarct of retinal nerve fiber layer, forming cotton-wool spots. Although bilateral Purtscher-like retinopathy has been linked to the activation of complement and formation of leukoemboli, it is difficult to correlate this systemic mechanism to a local event. In this unilateral case, which was triggered by a local event of peribulbar anesthetic injection, the pathogenesis is largely hypothetical. The peripapillary distribution of the cotton-wool spots suggests an infarction of the peripapillary arterioles in an area of no collaterals. It could be hypothesized that the sudden increase in orbital volume might have increased the hydrostatic

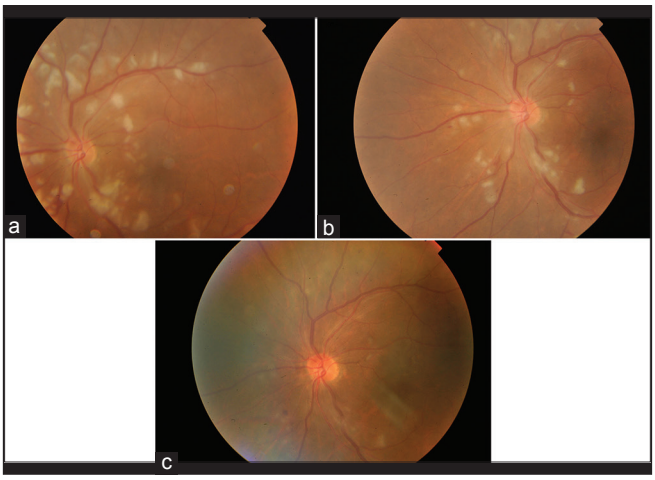

Figure 1: Fundus photograph of the left eye. (a) Postoperative day 1. Numerous, peripapillary, soft exudates and superficial hemorrhages. (b) Follow-up at 2 weeks. (c) Follow-up at 4 weeks

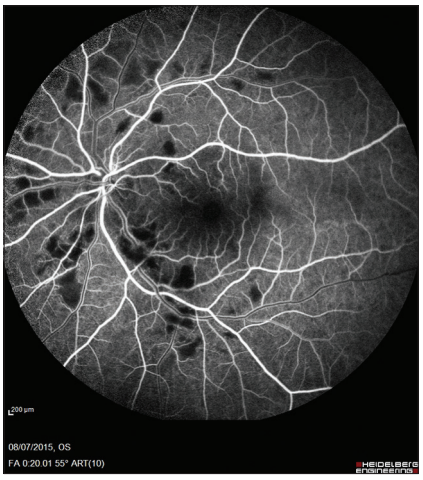

Figure 2: Fundus fluorescein angiography of the left eye (postoperative day 4) revealed blocked fluorescence in the sites corresponding to the retinal and preretinal hemorrhages, capillary dropout in the areas of Purtscher flecken and cotton-wool spots

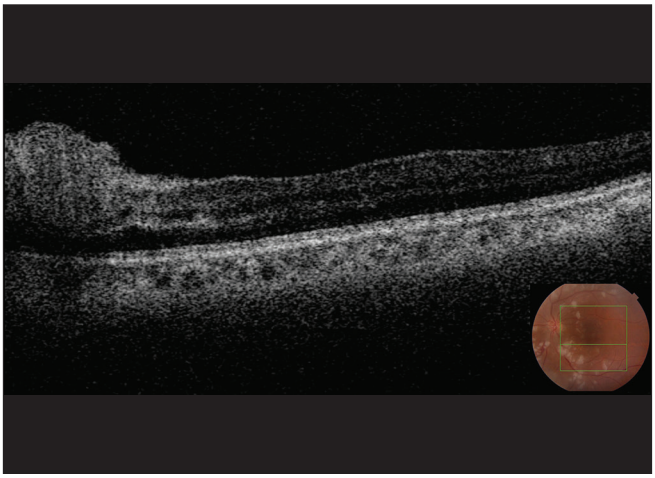

Figure 3: Optical coherence tomography of the left eye (postoperative day 4) revealed edema of the inner retinal layers with hyperreflectivity of the nerve fiber layers corresponding to the areas of retinal whitening

pressure and reproduced the conditions of a Valsalva maneuver. However, this theory is challenged by the fact that considering the significantly larger volume of the extraconal space in comparison to intraconal space, 5 ml of anesthetic agent is unlikely to produce significant hydrostatic pressure to produce vascular occlusion. Another possibility is that the infarction might have been caused by the accidental embolization of the central retinal artery or a posterior ciliary artery by either some residual air bubbles in the syringe or an orbital fat embolus mobilized by the needle. Most likely, the infarction may have been caused by the vasospastic effects of adrenaline accentuated by the intrinsic vasoconstrictive properties of lidocaine. Findl *et al*. reported a decrease in retinal blood flow velocity by 10–15%, 1–5 min, respectively, following peribulbar anesthesia without a vasoconstrictive agent like adrenaline.[9]

Purtscher-like retinopathy in this patient had a favorable outcome without treatment. We report this case to inform ophthalmic surgeons and anesthetists that although peribulbar anesthesia avoids direct optic-nerve injury, indirect injury may occur from vasospasm in response to the injection.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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