Case report

Ocular decompression retinopathy after laser iridotomy in a patient with acute angle closure

Tzu-Te Hu, Shao-Chun Chen, Po-Chen Tseng, Chun-Chen Chen, Shiow-Wen Liou

Department of Ophthalmology, Taipei City Hospital, Renai Branch, Taipei City, Taiwan

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ABSTRACT

A 65-year-old female developed elevated intraocular pressure (IOP) with angle closure in the right eye. The fundus examination revealed vitreo-retinal hemorrhage that had occurred after neodymium-doped yttrium aluminum garnet (Nd:YAG) laser iridotomy. The retinal hemorrhage resolved spontaneously. This is a rare complication that occurs when the IOP is lowered rapidly. Ocular decompression retinopathy (ODR) was identified. We reviewed several cases of ODR that were treated by various interventions. We also discuss the possible pathogenesis, prognosis, and treatment strategy.

1. Introduction

Angle-closure glaucoma (ACG) is a leading cause of irreversible blindness in the world.1 It is estimated that it will affect up to 5.3 million people by 2020.1 Several methods lower intraocular pressure (IOP) such as medical treatment, laser treatment, paracentesis, and surgery. Vitreo-retinal hemorrhage after rapid IOP reduction is a rare complication that is also known as ocular decompression retinopathy (ODR).2 In 1992, Fechtner et al3 defined ODR as an iatrogenic complication that occurs after the abrupt lowering of IOP after glaucoma filtering surgery. In this paper, we present a case of ODR that occurred after the rapid lowering of a patient’s IOP by laser iridotomy.

2. Case Report

A 65-year-old woman experienced a right temporal headache and right ocular pain for 1 day. She had no other systemic diseases, except asthma. Her IOP level was 60 mmHg oculus dexter (OD) and 14 mmHg oculus sinister (OS) at presentation. Slit-lamp examination showed an extremely shallow angle with a fixed mid-dilated pupil and microcystic corneal edema (OD). The vertical optic cup–disc ratio was approximately 0.5 in both eyes. The initial best corrected visual acuity (BCVA) was 3/60 (OD). She was treated with oral acetazolamide (250 mg), topical pilocarpine (2%), topical dorzolamide (2%), topical brimonidine (0.15%), and intravenous injection of 20% mannitol (500 mL). However, the IOP level (OD) remained > 35 mmHg; therefore, we performed neodymium-doped yttrium aluminum garnet (Nd:YAG) laser iridotomy (LI) using a power of 5 mJ on her right eye. The IOP level suddenly decreased to 17 mmHg after LI treatment.

The day after LI, the patient complained of a dark shadow floating in front of her right eye. Fundus examination (Fig. 1) showed scattered retinal hemorrhages combined with vitreous hemorrhage (OD). Mildly tortuous retinal vasculature was also present. The fundus finding of the left eye was unremarkable. Fluorescein angiography showed blocking defects in the area of the retinal hemorrhages, but neither retinal ischemia nor retinal edema was detected (Fig. 2).

During the follow-up period, the IOP was maintained within normal limits under topical antiglaucomatous medications and the vitreous and retinal hemorrhages resolved gradually over time. At the 4-month follow-up, the BCVA had returned to 18/20.

3. Discussion

Ocular decompression retinopathy presents as a vitreo-retinal hemorrhage after rapid IOP reduction. This may be a complication
Several hypotheses have been proposed to explain the pathogenesis of ODR, but two stand out. The first hypothesis is the Mechanical Theory holds that a sudden decrease in IOP level changes the globe shape, which then shears fragile capillaries. Furthermore, the expansion of the lamina cribrosa may compress the central retinal vein, which may also lead to diffuse retinal hemorrhaging. The second hypothesis is the Vascular Theory, which holds that a sudden decrease in IOP level may reduce retinal arterial resistance and results in extravascular fluid leakage. In addition, among the patients who have impaired autoregulation of the retinal vasculature, there may be a rapid increase in retinal capillary blood flow, which could result in multiple focal retinal dot hemorrhages.

Ocular decompression retinopathy often has a fair prognosis. Mukkamala et al found the average resolution time is 13 ± 12.4 weeks (64 eyes of 55 patients) and the mean final visual acuity improved to 20/65. Only 14% of patients required advanced surgical treatment such as vitrectomy.

According to statistics, nearly 80% of ODR patient are asymptomatic and only 4% of patient have the symptom of floaters, which accounts for a very small proportion. Our patient complained of distinct floaters by the next day, and we discovered a vireo-retinal hemorrhage at that time. The patient had no history of systemic diseases such as diabetes mellitus or hypertension. The fluorescein angiography examination also excluded the possibility of diabetic retinopathy. The sudden reduction of IOP after LI presumably directly damaged fragile retinal capillaries and contributed to the subsequent retinal hemorrhages. In addition, the mildly tortuous retinal vessels may have resulted from the compression of the central retinal vein. Unlike the more usual case of central retinal vein occlusion, which is caused by persistent elevated vascular resistance, ODR only exerts an impact on the central retinal vein for a short time. This may explain why patients with ODR often have a good prognosis, compared to individuals with true central retinal vein occlusion. After 4 months follow up in our patient, the retinal hemorrhage had resolved and the BCVA had returned to 18/20.

In conclusion, although ODR is a complication of IOP-lowering procedures, it does not usually cause permanent visual impairment. In a patient with acute ACG, especially after LI or other surgical intervention, the condition of the retina should not be ignored, even when miotics-related small pupil or hazy media is evident. It is important to determine the etiology of hemorrhage in these patients, especially in symptomatic patients without previous retinopathy or major systemic diseases. Ocular decompression retinopathy needs to be distinguished from other retinal vascular diseases such as diabetic retinopathy or retinal vascular occlusion because a different treatment protocol is used for this disease and a better prognosis is associated with it. The visual prognosis of our patient was fortunately rather good with conservative treatment only—perhaps because of the early treatment of acute angle-closure glaucoma and profound damage to the ganglion cells had not occurred. We present this case of ODR to remind clinicians of this rare complication and so that clinicians can explain more thoroughly to patients prior to performing the laser procedure.

References


