OPIS PRZYPADKU CASE REPORT

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Ocular decompression retinopathy solely following medical treatment in steroid induced glaucoma

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HIGHLIGHTS

An aggressive reduction of intraocular pressure medically can occasionally cause ocular decompression retinopathy but can still be exercised in crucial situations, in order to preserve vision and prevent blindness.

ABSTRACT

An 18-year-old male developed bilateral, steroid induced glaucoma and steroid induced cataract following topical steroid usage. Best corrected visual acuity was 6/60 and perception of light in right eye and left eye respectively. Both eyes had clear cornea, 7–8 mm sluggishly reacting pupil with an immature cortical cataract and advanced glaucomatous optic neuropathy in right eye and a near mature cortical cataract in left eye. Intraocular pressure (IOP) was ≥50 mmHg and angles were open on gonioscopy in both eyes. Tablet acetazolamide 250 mg q.i.d. for 1 week along with brimonidine 0.15% and timolol 0.5% fixed combination eye drops b.d, brinzolamide 1% eye drops t.d.s and netarsudil 0.02% eye drops o.d. were prescribed for both eyes. At 2 weeks follow up, IOP reduced to 14 mmHg and 18 mmHg in right eye and left eye respectively. However, surprisingly, the right eye showed multiple intraretinal blot haemorrhages, in greater numbers temporally and extending towards the equator in all quadrants. Post phacoemulsification, the left eye fundoscopy revealed very advanced glaucomatous optic neuropathy with two intraretinal blot haemorrhages. The best corrected visual acuity improved only marginally upto 6/36 (post phacotrabeculectomy) and hand movement (post phacoemulsification) in right eye and left eye respectively. At 8 weeks post right eye phacotrabeculectomy, all the haemorrhages resolved completely in both eyes without any intervention. This case developed ocular decompression retinopathy (ODR) solely following oral and topical IOP lowering therapy which is rare. It shows that an aggressive IOP reduction, meant to reduce the risk of postoperative ODR, can occasionally itself become the cause of ODR. This case also demonstrates that, such a reduction can still be exercised in crucial situations, in order to preserve vision and prevent blindness, since ODR is usually a self-resolving condition having a benign course.

Key words: ocular decompression retinopathy, steroid induced glaucoma, phacoemulsification, mitomycin-C, phacotrabeculectomy

INTRODUCTION

Fechtner et al. in 1992 coined *ocular decompression retinopathy* to describe the hemorrhages due to a sudden lowering of intraocular pressure (IOP) and decompression of the eye following glaucoma filtering surgery [1]. But, now known, ocular decompression retinopathy (ODR) is a rare complication that can occur after any intervention, medical or surgical, causing an abrupt reduction of IOP.

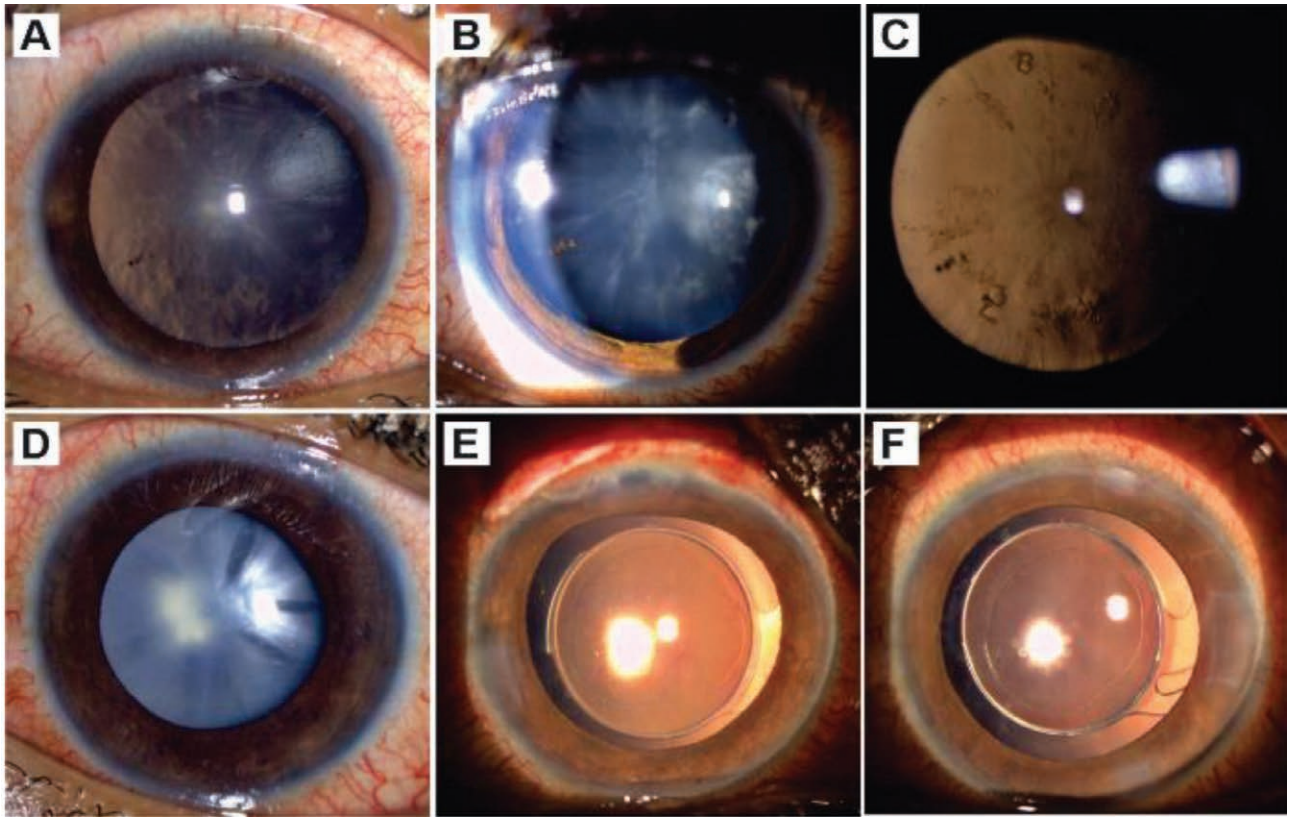
Most of the published ODR reports are secondary to some surgical or laser procedure [2]. Reports of development of ODR, solely following medical therapy for uncontrolled glaucoma are exceedingly rare [2–4]. This paper describes a unique case of steroid induced glaucoma (SIG) in a young male who developed ODR solely following oral and topical anti-glaucoma medications.

CASE PRESENTATION

An 18-year-old male presented to us with bilateral gradual progressive diminution of vision, greater in left eye (LE), since 2 months. History of unprescribed use of moxifloxacin 0.5% and dexamethasone 0.1% fixed combination eye drops b.d. in both eyes, off and on, since 2 years, for itching and redness, was present. The best corrected visual acuity (BCVA) was 6/60 and perception of light with accurate projection in all 4 quadrants, in right eye (RE) and LE respectively. Both eyes had clear cornea, deep anterior chamber and 7–8 mm sluggishly reacting to light pupil. In addition, the RE had anterior subcapsular, posterior subcapsular and peripheral cortical cataract while the LE had a near mature cortical cataract (fig. 1 A–D). On fundoscopy, the RE showed an optic disc with vertical cup disc ratio of 0.9, pallor and thinning of the superior neuroretinal rim, absent inferior neuroretinal rim with a generalized retinal nerve fibre layer (RNFL) loss inferiorly. General fundus was within normal limit. Fundus view of the LE was very hazy but an enlarged optic disc cup was barely appreciable on dilated pupil.

FIGURE 1

A. Slit lamp photograph (diffuse illumination) of RE with dilated pupil.
B. Slit lamp photograph (slit illumination) of RE with dilated pupil.
C. Slit lamp photograph (retro illumination) of RE with dilated pupil showing good fundus glow.
D. Slit lamp photograph (diffuse illumination) of LE with semidilated pupil.
E. Slit lamp photograph (diffuse illumination) of RE (post-operative) with superior bleb and releasable suture, superior surgical iridectomy and dilated pupil.
F. Slit lamp photograph (diffuse illumination) of LE (post-operative) showing posterior chamber intraocular lens and dilated pupil.



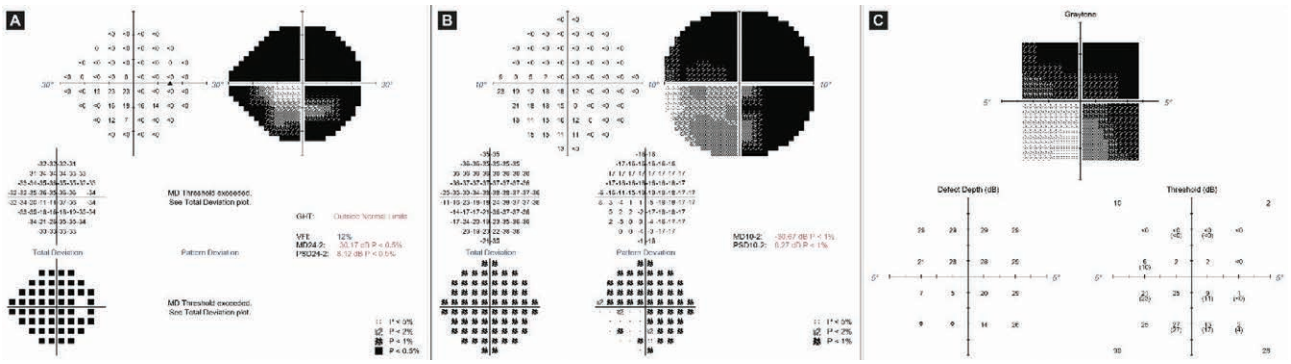
IOP on Goldmann applanation tonometry was 52 mmHg and 50 mmHg in RE and LE respectively. Angles were open upto ciliary body on gonioscopy bilaterally. B scan of the LE confirmed optic disc cupping. The central corneal thickness on ultrasound pachymetry was 540 µm and 536 µm in RE and LE respectively. Humphrey field analysis of the RE displayed a deep superior altitudinal field defect and an inferior arcuate scotoma with involvement (greater temporally) of central 10° and 5° fields (fig. 2 A–C). The patient was diagnosed as SIG with steroid induced cataract in both eyes. As the patient refused admission, tablet acetazolamide 250 mg q.i.d., syrup potassium chloride 1 teaspoon q.i.d., tablet pantoprazole 40 mg o.d. for 1 week along with brimonidine 0.15% and timolol 0.5% fixed combination eye drops b.d., brinzolamide 1% eye drops t.d.s. and netarsudil 0.02% eye drops o.d. were prescribed for both eyes.

cupping and 2 blot hemorrhages outside macular area, superiorly and inferiorly, one each were visible (fig. 3 B, 4 C). On third postoperative day, with treatment, the LE cornea cleared, IOP reduced to 20 mmHg and BCVA improved to hand movement with accurate projection of light. Optical coherence tomography (OCT) performed for both eyes showed a lower average inferior RNFL thickness compared to superior in both eyes, RNFL thinning of inferior quadrant in RE, and marked RNFL thinning in all except nasal quadrant in LE (fig. 5).

After one week, under guarded visual prognosis, RE mitomycin-C augmented trabeculectomy with phacoemulsification and intraocular lens implantation, was performed under local peribulbar anesthesia. Mitomycin-C 0.4 mg/mL was used for 3 min. On postoperative day one, the RE showed a low, diffuse non-leaking filtration bleb, well-formed

FIGURE 2

Standard automated perimetry results of the RE (Humphrey Field Analyzer-3, SITA-Standard, Carl Zeiss Meditec, Dublin, CA USA).
A. 24-2 test grid.
B. 10-2 test grid.
C. Macular threshold test with target V.



At 2 weeks follow-up, there were no fresh complaints and except conjunctival congestion, both eyes anterior segment examination was as before. IOP with Goldmann applanation tonometry was 14 mmHg and 18 mmHg in RE and LE respectively. However, unexpectedly, the RE fundus revealed multiple intraretinal blot hemorrhages located within as well as outside the temporal vascular arcade but away from the fovea and optic disc, (in greater numbers temporally than nasally) and extending towards the equator (fig. 3 A, 4 A–B). The LE general fundus was not clearly visible.

Axial length of both eyes was 24.10 mm. Under guarded visual prognosis, LE phacoemulsification with intraocular lens implantation (fig. 1 F) was performed under local peribulbar anesthesia. On first postoperative day, BCVA remained perception of light with accurate projection, mild corneal oedema, anterior chamber reaction was present and IOP was 28 mmHg. A pale optic disc with near total

anterior chamber, mild anterior chamber reaction with posterior chamber intraocular lens (fig. 1 E). The BCVA was 6/60 and IOP was 10 mmHg on Goldmann applanation tonometry. Fundus examination was as before. At 2 weeks follow up post phacotrabeculectomy, the RE BCVA reduced to counting finger 1 feet, IOP was 2 mmHg, and well formed, non-leaking bleb, deep anterior chamber with an intense fibrinous membrane over posterior chamber intraocular lens was present. However, there were no fresh retinal hemorrhages. The patient was started on oral prednisone 40 mg per day in tapering dose for 2 weeks, prednisolone acetate 1% eye drops every 2 h, and atropine 1% eye drops t.d.s. in RE. At 4 weeks postoperative visit, BCVA improved to 2/60, IOP was 6 mmHg and the anterior chamber reaction reduced. No fresh retinal hemorrhages were seen. On topical steroid treatment at 6 weeks postoperative visit, the RE BCVA further improved to 6/36 and IOP was 11 mmHg with quiet anterior chamber.

FIGURE 3

Wide field colour fundus photograph (Optos, UK).
A. Right eye (pre-operative) showing multiple peripheral hemorrhages with normal macula.
B. Left eye (post-operative) showing 2 hemorrhages with normal macula (white arrow pointing at superior hemorrhage).

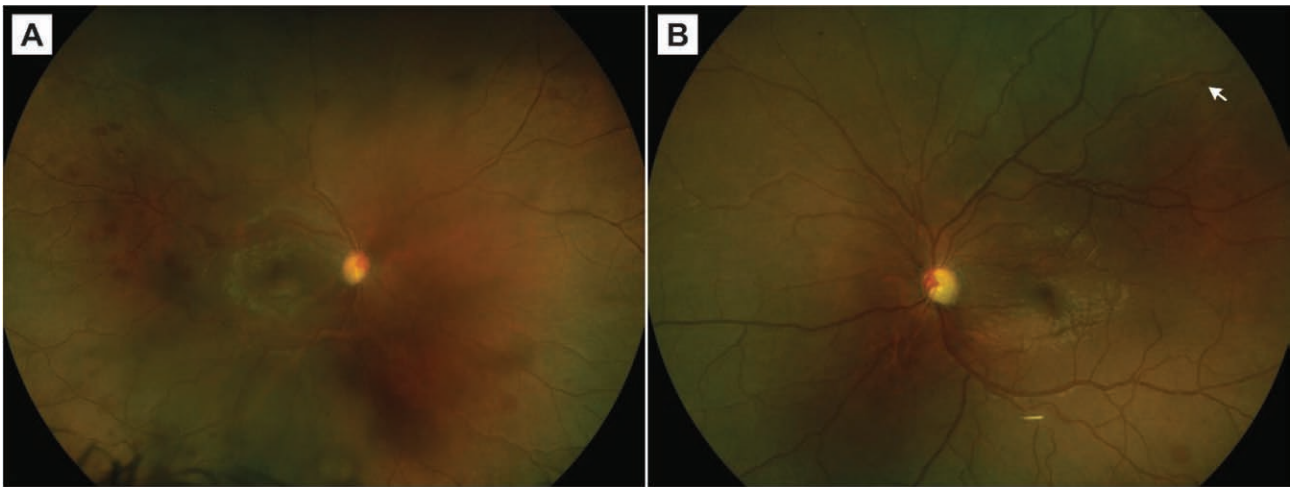
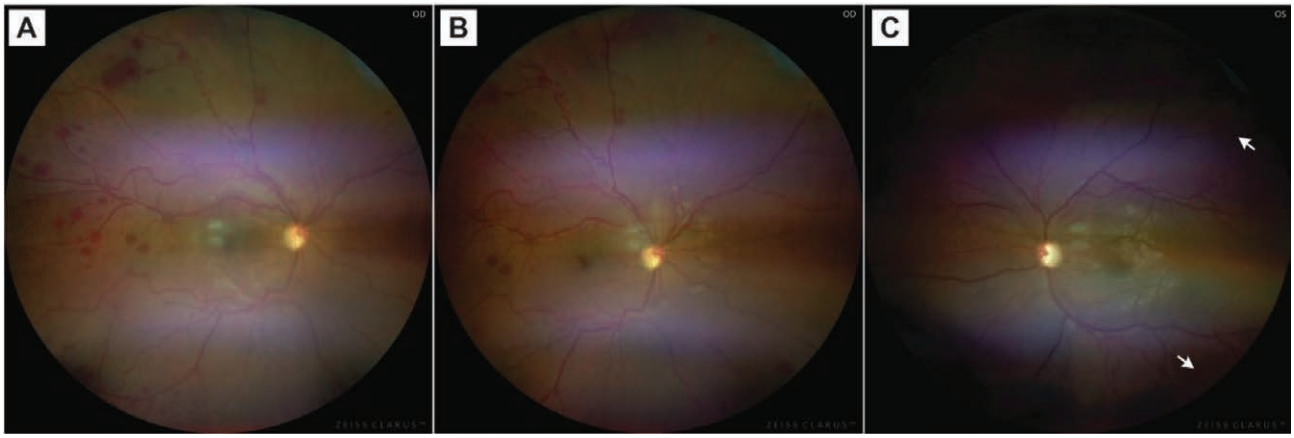


FIGURE 4

Colour fundus photograph (Clarus, Carl Zeiss Meditech, Dublin, CA, USA).
A. Right eye (pre-operative) showing multiple peripheral hemorrhages with normal macula.
B. Right eye (pre-operative) showing few additional nasal hemorrhages along with the temporal hemorrhages.
C. Left eye (post-phacoemulsification) showing 2 hemorrhages (white arrows) with normal macula.



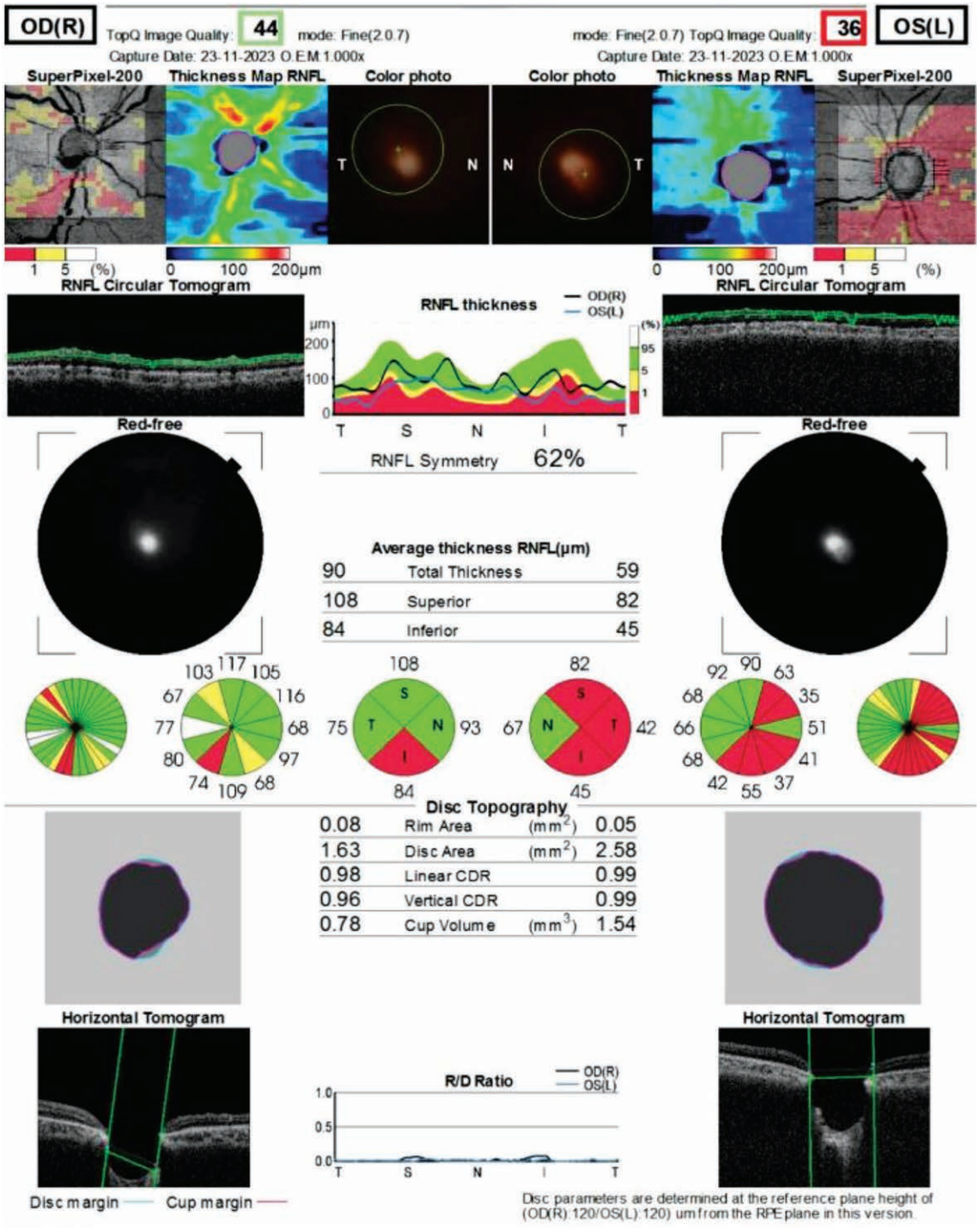
The LE IOP with Goldmann applanation tonometry at all these visits was >21 mmHg each time on the previous 3 topical anti-glaucoma drugs. With visual prognosis explained, LE trabeculectomy was advised and performed using mitomycin C 0.4 mg/mL for 3 min. The BCVA of hand movement with accurate projection of light in all quadrants was retained. At 8 weeks, post RE phacotrabeculectomy/2 weeks post LE trabeculectomy, on tapering topical steroid treatment (both eyes), IOP was 11 mmHg and 15 mmHg in RE and LE respectively. The BCVA was 6/36 (-1.0 cylinder 10°) and hand movement with accurate projection of light in RE and LE respectively. All the retinal hemorrhages were cleared. At the last follow-up till date, 16 weeks post RE phacotrabeculectomy/10 weeks post LE trabeculectomy, on tapering topical steroids (LE), IOP was 10 mmHg and 13 mmHg in RE and LE respectively. The BCVA was 6/36 with correction (-1.0 cylinder 10°) and hand movement with accurate projection of light with correction (-1.0 cylinder 20°) in RE and LE respectively.

DISCUSSION

ODR is a relatively rare entity with incidence of 3.05% in adults and 5.24% in children as reported by Jung et al. [5] and Fadel et al. [6] respectively.

FIGURE 5

Optical coherence tomography of the optic nerve head and retinal nerve fibre layer for RE and LE (Topcon 3D optical coherence tomography DRI OCT triton plus, Japan).



ODR has been shown to occur following trabeculectomy with mitomycin-C, trabeculectomy alone without antimetabolites, trabeculectomy with 5-fluorouracil, trabeculectomy with cataract surgery, trabeculectomy with trabeculotomy, trabeculotomy alone, glaucoma drainage device insertion, orbital decompression surgery, silicone oil removal, phacoemulsification, sutureless 25-gauge transconjunctival pars plana vitrectomy, anterior chamber paracentesis, and deep sclerectomy with mitomycin-C [2, 7]. ODR has also been reported secondary to sutured 23-gauge transconjunctival pars plana vitrectomy [8], canaloplasty [9], goniotomy [10], EXPRESS shunt implantation [11], bleb needling

and suturolysis [12], and anterior chamber paracentesis with intracameral bevacizumab injection [13]. It has also been documented after laser procedures like neodymium-doped yttrium aluminum garnet (Nd-YAG) laser iridotomy [2], argon laser peripheral iridoplasty [2] and Nd-YAG membranotomy [14]. In our patient, the ODR occurred prior to any surgical intervention.

ODR has been described in various glaucoma aetiologies like angle closure glaucoma, uveitic glaucoma, primary open angle glaucoma, congenital and juvenile glaucoma, neovascular glaucoma, SIG, pseudoexfoliation glaucoma, iridocorneal endothelial syndrome, posttraumatic glaucoma, post keratoplasty and pseudophakic glaucoma [2, 5]. Our patient was a case of SIG.

Reports of ODR specifically related to steroids (SIG, steroid induced hypertension) are few. In a review of 32 manuscripts by Mukkamala et al., out of total 64 eyes (55 patients) diagnosed with ODR, SIG was present in only 3 eyes (4.7%) [2]. In a retrospective case control study by Jung et al., out of the total 21 eyes developing ODR following glaucoma surgery in various glaucoma aetiologies, only 2 eyes (9.5%) had SIG [5]. Gil-Martínez et al. also reported bilateral ODR in bilateral SIG, following trabeculectomy with mitomycin-C in both eyes, in a 38-year-old woman with bronchial asthma, rhinitis and ocular allergy [15]. In another bilateral SIG case of a 20-year myopic male with allergic conjunctivitis, Mohammed et al. found ODR on first post-operative day in the LE with deep sclerectomy [16]. Fadel et al. too found the highest incidence of ODR (3 out of 4 eyes, 75%) after surgery amongst SIG cases, in children [6].

ODR has also been described in steroid induced hypertension (SIH). Abu Samra et al. reported unilateral retinopathy post implantation of EXPRESS implant in the LE in a case of unilateral SIH following steroid use, post-penetrating keratoplasty LE for keratoconus [11]. In another case of bilateral SIH associated with tubulointerstitial nephritis and uveitis syndrome in an 8-year girl, Muñoz-Negrete et al. reported bilateral ODR after deep sclerectomy with intra-scleral acrylic implant (Esnoper™, AJL, Vitoria, Spain) with mitomycin-C in both eyes [7].

As evident, most of the aforementioned reports are secondary to some surgical procedure. Reports of ODR, solely following medical treatment are sparse. In the review of 32 manuscripts by Mukkamala et al., only 2 out of 64 (3%) eyes were found to develop ODR due to medical treatment [2]. Paufique et al. report in 1965, on a patient with retinal hemorrhages following medical management of acute angle closure appears to represent the first recognized case of ODR [2, 3]. Alwitry et al. in 2006 described a patient of acute primary angle closure who developed bilateral pre-retinal hemorrhages, following a pronounced reduction in IOP in both eyes, 2 h after receiving intravenous acetazolamide and topical dexamethasone, apraclonidine hydrochloride 1%, and pilocarpine hydrochloride 4% [2, 4]. Our patient developed ODR solely following oral and topical anti-glaucoma medications usage without any intravenous drugs. The present case is an addition to this series and to the best of our knowledge, the first to be reported in a SIG patient.

The spectrum of fundus finding in ODR are variable. According to Mukkamala et al., hemorrhages involving all the retinal layers are most common and are present in 95%, sub-hyaloid hemorrhage in 33%, white centered retinal hemorrhages in 20% and vitreous hemorrhage in 5%. The retinal hemorrhages occur throughout the fundus; most commonly within the posterior pole (retinal area within the temporal vascular arcades) in 92%. These extend to the peripheral retina in 72% and into the peri-papillary area and/or optic nerve head in 22% [2]. In our patient too, the posterior pole and the retinal periphery, both were involved in the RE but there was no involvement of the vitreous, peri-papillary area and/or optic nerve head, or choroid in either eye. All the hemorrhages were intra retinal and none were white centered in either eye.

The differential diagnoses of ODR include central retinal vein occlusion, ocular ischemic syndrome, Terson syndrome, diabetic retinopathy, Valsalva retinopathy, shaken baby syndrome and coagulopathies [2]. Although fundus fluorescein angiography was not done in our case, the diagnosis of the condition was purely clinical, based on the following features; absence of old age, no history of systemic disease (like diabetes, hypertension, easy bruising or bleeding) or medication intake (like aspirin or anticoagulants), absence of venous dilatation and macular edema on fundus examination, and normal routine blood investigations.

In the review by Mukkamala et al., 84% of cases developed ODR in one eye and majority (80%) of ODR patients are symptomless except occasional complaints of diminution of vision, floaters, or central scotoma. The mean time to diagnosis was 1.5 (±2.0) days and the average time to resolution was 13 (±12.4) weeks (range 2–72 weeks) with 85% of eyes regaining baseline vision. In most patients, no intervention is required [2]. In our patient, the ODR was bilateral but could be detected in the LE only after phacoemulsification. There was no increase in complaint or symptoms like decreased vision, floaters, or scotoma, despite developing hemorrhages at 2 weeks following medical treatment. This was because of non-involvement of vitreous, fovea or optic nerve. However, it was not possible to ascertain the actual time of occurrence of ODR in our case since the patient presented 2 weeks after starting medical treatment and was not examined in-between during this interval. All the hemorrhages resolved by 8 weeks in both eyes without any intervention.

There is no consensus regarding the pathogenesis of ODR, and mechanical and vascular theories have been proposed. According to the mechanical theory, acute drop in IOP re-

sults in scleral collapse and globe deformity which causes mechanical shearing of capillaries leading to hemorrhage. The sudden drop in IOP also causes anterior movement of lamina cribrosa which might impede axoplasmic flow, leading to disc edema and central retinal vein compression, resulting in retinal hemorrhages mimicking central retinal vein occlusion [1, 2, 17]. The vascular theory is based on autoregulation and fragility of retinal capillaries. A breakdown of auto-regulation mechanism occurs in conditions like poorly controlled diabetes mellitus, hypertension, prolonged exposure to elevated IOP or long-standing inflammatory conditions like uveitic glaucoma. In these patients, acute transient reduction in IOP may reduce retinal arterial resistance, resulting in increased flow, which might overwhelm the resistance of capillaries, leakage through already fragile capillaries, leading to retinal hemorrhages [1, 2, 14, 18]. As our patient was of younger age (18 years), a low scleral rigidity leading to a greater scleral deformation/collapse following an acute drop in IOP could be a possible reason. Another reason could be a defective or total loss of autoregulation because of longstanding glaucoma, resulting in a sudden increase in retinal intravascular flow as a result of IOP drop ultimately leading to development of retinal hemorrhages.

Regarding the IOP changes related to ODR, Mukkamala et al., review found that mean of the IOPs before ODR was 45.7 (±16.9) mmHg, the mean decrease in IOP was 33.2 (±15.8) mmHg (range: 4–57 mmHg) and the IOP after ODR was 13.2 (±10.3) mmHg [2]. In our case too, similar to Mukkamala et al., the initial IOP before ODR was 52 mmHg and 50 mmHg, the IOP decrease was 38 mmHg and 32 mmHg and the IOP after ODR development was 14 mmHg and 18 mmHg, in RE and LE respectively. However, there was marked asymmetry in the presentation of ODR with only 2 hemorrhages in the LE compared to the multiple hemorrhages of the contralateral eye. This asymmetric presentation of ODR could probably be due to the difference in magnitude of IOP drop after treatment, with the LE having a lesser IOP reduction (32 mmHg) compared to the RE (38 mmHg) from an initial IOP of ≥50 mmHg in both eyes.

Postoperative hypotony (IOP <6 mmHg) is a common occurrence post-augmented trabeculectomy involving use of antiproliferative agents such as mitomycin-C or 5-fluorouracil. A steep IOP reduction and a low IOP post-operatively/hypotony were suggested as risk factors of ODR by Suzuki et al. [17] According to Yalvac et al. too, suddenly decreasing the IOP during surgery and hypotony in the early post-operative period, were thought to trigger ODR [18]. Fechtner also suggested that prevention of postoperative hypotony may be beneficial [1]. However, in contrast to Suzuki et al. [17], Yalvac et al. [18] and Fechtner et al. [1], Jung et al. in their retrospective case control study found that apart from low haemoglobin (P = 0.040), the only ocular factor that was significantly associated with the development of ODR between the ODR group and the control group (without ODR) was the preoperative maximum IOP (P = 0.031) in multivariate regression analysis. They also found that postoperative IOP itself did not play a crucial role in the occurrence of retinal hemorrhages after glaucoma surgery and that the frequency of postoperative hypotony was not significantly different (P = 0.467) between the ODR group and the control group (without ODR) [5]. Saricaoglu et al. in the first case of their two, also did not find postoperative hypotony as a factor in the pathogenesis of ODR [19]. In our case, the occurrence of hemorrhages with an IOP of 14 mmHg and 18 mmHg post medical treatment, and the non-occurrence of fresh retinal hemorrhages in the RE even with an IOP of 2 mmHg and 6 mmHg in post-operative period, following phacotrabeculectomy with mitomycin-C augmentation is relevant. This implies the findings of Jung et al. [5] and Saricaoglu et al. [19]. To reduce the risk of ODR, Yalvac et al. recommended lowering of IOP as much as possible preoperatively in young patients with high IOP [18]. Suzuki et al. suggested that the IOP should be decreased as slowly as possible preoperatively to prevent sudden, marked and prolonged hypotony and thereby reduce the risk of ODR [17]. In contrast to Suzuki et al., even though any intravenous drugs were not used, the IOP reduction was done aggressively in our patient so as to lower the IOP as fast and as much as possible. This was done to preserve vision as the patient was young, had a very high presenting IOP, advanced disc damage with poor visual fields involving macula and hence a very high risk of loss of vision too. The advanced stage of glaucoma disease in our patient is evident with only a marginal improvement in the final BCVA post-surgery in both eyes. However, this aggressive lone, oral and topical preoperative IOP lowering medical therapy itself became the cause of ODR in our case.

CONCLUSION

This is the first reported case of ODR, solely following an oral and topical IOP lowering therapy in a SIG patient and is a unique presentation. An aggressive reduction of IOP medically, meant to reduce the risk of postoperative ODR, can occasionally itself become a cause of this hemorrhagic retinopathy. However, it is well known that ODR usually is a self-limiting condition having a benign course with complete resolution of the hemorrhages subsequently. Hence, such an aggressive IOP reduction can still be exercised in crucial situations, in order to preserve vision and prevent blindness, as demonstrated through this case.

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Authors' contributions:
Navjot Singh Ahluwalia: concept and design, preparation of abstract, drafting of the article, manuscript writing and editing, final approval.
Aishwary M. Duddalwar: data acquisition, manuscript editing.
Rakesh Shakya: manuscript editing.

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A written informed consent authorizing publication of anonymized clinical data and images was provided by the patient.
The content presented in the article complies with the principles of the Helsinki Declaration, EU directives and harmonized requirements for biomedical journals.