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Recurrent Annular Peripheral Choroidal Detachment after Trabeculectomy

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Key Words

Annular peripheral choroidal detachment · Trabeculectomy · Steroid withdrawal ·
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Abstract

We report a challenging case of recurrent flat anterior chamber without hypotony after trabeculectomy in a 54-year-old Black male with a remote history of steroid-treated polymyositis, cataract surgery, and uncontrolled open angle glaucoma. The patient presented with a flat chamber on postoperative day 11, but had a normal fundus exam and intraocular pressure (IOP). Flat chamber persisted despite treatment with cycloplegics, steroids, and a Healon injection into the anterior chamber. A transverse B-scan of the peripheral fundus revealed a shallow annular peripheral choroidal detachment. The suprachoroidal fluid was drained. The patient presented 3 days later with a recurrent flat chamber and an annular peripheral choroidal effusion. The fluid was removed and reinforcement of the scleral flap was performed with the resolution of the flat anterior chamber. A large corneal epithelial defect developed after the second drainage. The oral prednisone was tapered quickly and the topical steroid was decreased. One week later, his vision decreased to count fingers with severe corneal stromal edema and Descemet's membrane folds that improved to 20/50 within 24 h of resumption of the oral steroid and frequent topical steroid. The patient's visual acuity improved to 20/20 following a slow withdrawal of the oral and topical steroid. Eight months after surgery, the IOP was 15 mm Hg without glaucoma medication. The detection of a shallow anterior choroidal detachment by transverse B-scan is critical to making the correct diagnosis. Severe cornea edema can occur if the steroid is withdrawn too quickly. Thus, steroids should be tapered cautiously in steroid-dependent patients.

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Introduction

Shallow or flat anterior chamber occurs as an early postoperative complication in about 10% of glaucoma filtration surgery patients [1–3]. Failure to reform the normal chamber can cause vision-threatening problems such as cataract and corneal decompensation [4]. Identifying the cause as soon as possible is crucial to avoiding severe complications [5–7]. Treatment for flat anterior chamber is directed at eliminating the cause(s). The differential diagnosis for flat anterior chamber with increased intraocular pressure (IOP) includes suprachoroidal hemorrhage, aqueous misdirection glaucoma or pupillary block, while the causes of flat anterior chamber with low IOP include over-filtration, wound leak, cyclodialysis or serous choroidal detachment [5–10]. In some cases, the underlying cause of the flat chamber can be difficult to treat and identify. Here we report the case of recurrent flat anterior chamber with normal IOP after trabeculectomy due to annular peripheral choroidal detachment. This case reinforces the awareness of this complication as one of the differential diagnoses of flat anterior chamber after glaucoma surgery, the importance of ultrasonography in making the diagnosis, and the need for accommodating therapeutic modification to treat complications arising in cases with underlying inflammatory disease.

Case Report

A 54-year-old African-American male with a remote history of prednisone treated polymyositis developed bilateral open angle glaucoma. His past ocular history included trabeculectomy in the right eye, bilateral cataract surgery and the maintenance of topical steroid drops to manage his rebound iritis. On the day of surgery, he reported some mild cold symptoms, but he insisted on having the surgery done. He had an uneventful trabeculectomy with mitomycin C performed on his left eye and did well during the first postoperative week with deep anterior chamber and an IOP in the 7–9 mm Hg range. He was on Vigamox 0.5% (Alcon, Fort Worth, Tex., USA) and prednisolone acetate 1% 4 times a day. He returned for a follow-up on postoperative day 11 with a complaint of persistent left eye pain for the last 3 days. On examination, his visual acuity was 20/80, pin holed (PH) to 20/30, and IOP was 10 mm Hg. His anterior chamber was flat with a complete iris-cornea touch except a 1/5 cornea thickness space between the cornea and the intraocular lens (IOL). The superior fornix-based bleb was slightly elevated without leakage and the peripheral iridectomy was patent. There were severe fibrin exudates in the anterior chamber with a 360° posterior synechiae of the pupil. The retina was flat on fundus exam without scleral depression. The initial differential diagnosis included: overfiltration, pupillary block, aqueous misdirection, choroidal effusion or choroidal hemorrhage. Inflammation was considered to play a role because of his history of steroid dependency, eye pain and copious fibrin in the anterior chamber. He was given oral prednisone 60 mg daily, topical Atropine 1% (Falcon Pharmaceuticals, Ltd., Fort Worth, Tex., USA) and phenylephrine 2.5% (Akorn, Inc., Lake Forest, Ill., USA) to dilate the pupil. Prednisolone was discontinued and Durezol (Difluprednate Ophthalmic Emulsion 0.05%; Alcon) was given one drop every hour [11]. In addition, he was given a 2-mg dexamethasone subconjunctival injection. Following the application of dilating drops in the clinic, the anterior chamber began reforming and posterior synechiae began to break down. He was sent home with Atropine 1% and phenylephrine 2.5% 4 times a day as

well as Durezol every hour. The next morning, his visual acuity was 20/200 (PH 20/60), IOP was 7 mm Hg, and his anterior chamber was flat with cornea-IOL touch. His fundus exam revealed no choroidal detachment or hemorrhage. Healon (Abbott Medical Optics Inc., Santa Ana, Calif., USA) was injected to reform the anterior chamber [12]. His oral prednisone and topical drops were continued.

Despite this treatment, the patient returned to the clinic 2 days later. He had a totally flat anterior chamber with a normal fundus and an IOP of 10 mm Hg. Aqueous misdirection was suspected, but peripheral choroidal effusion could not be ruled out. Transverse B-scan using a temporal approach identified a shallow annular anterior choroidal effusion (fig. 1). He was taken to the operating room the same day and the suprachoroidal effusion was drained from infero-nasal and infero-temporal sclerotomies [5]. A moderate amount of light yellow clear fluid was retrieved. The anterior chamber was reformed with Healon (Abbott Medical Optics Inc.) at the end of the surgery aiming at a high IOP to prevent choroidal effusion from recurring. He was continued on oral prednisone 60 mg daily and Durezol every 2 h and dilating drops 4 times a day.

His anterior chamber remained deep for 2 days with IOP in the mid-teens. However, on the third day after drainage of the choroidal effusion, the anterior chamber was flat with an IOP of 10 mm Hg. Aqueous misdirection was suspected, but repeat transverse B-scans showed recurrent peripheral choroidal effusion. The patient was taken to the operating room. One sclerotomy wound was reopened and a smaller amount of suprachoroidal fluid aspirated. The conjunctival incision for trabeculectomy was opened and additional 10–0 nylon sutures were placed on the sclera flap. Also, a half-thickness 3 × 3 mm donor scleral patch was placed to reinforce a thin area on the scleral flap. Postoperatively, his anterior chamber was deep with an IOP in the 40's. The IOP was lowered to the 30's over the next 2 days with paracentesis and ocular massage. Suture lysis was performed on the third postoperative day and his IOP decreased to the mid 20's. The patient developed a large corneal epithelial defect postoperatively. The oral prednisone was decreased by 20 mg every 2 days from the initial 60 mg/day, and topical Durezol was decreased from one drop every 2 h to 4 times a day to promote corneal healing.

The patient presented 7 days later with counting fingers vision due to diffuse corneal stromal edema and Descemet's membrane folds. The epithelial defect was completely healed and the IOP was 18 mm Hg with a deep anterior chamber. The corneal edema was thought to be due to the rapid steroid tapering. Oral prednisone 60 mg/day was resumed. The first dose of prednisone was given in the clinic and topical Durezol was increased to one drop every 2 h. The patient's corneal edema and Descemet's membrane folds cleared with vision improving to 20/50 the next morning. Oral prednisone was slowly tapered by 10 mg/week, while topical Durezol was continued at one drop every 2 h for 5 weeks, followed by a very slow taper. At this point, his anterior chamber depth stabilized and cornea remained clear. Suture lysis was performed cautiously and as needed. Visual acuity in the eye returned to 20/20 and IOP was 15 mm Hg, 8 months after the initial surgery, without glaucoma drops.

Discussion

Extremely high or low IOP can be of great assistance when trying to sort out the cause of a flat chamber following glaucoma surgery. However, a flat anterior chamber with normal IOP creates a more puzzling situation. The differential diagnosis usually includes conditions from both ends of the spectrum, including overfiltration, pupillary block, suprachoroidal

hemorrhage, choroidal detachment, aqueous misdirection, and annular peripheral choroidal detachment.

In our case, overfiltration was not a significant factor. The bleb never became highly elevated and the eye never hypotensive. Based on the presence of a patent peripheral iridectomy, pupillary block was ruled out. Suprachoroidal hemorrhages are usually located posterior to the equator and associated with severe pain, but neither were present. In typical cases of choroidal detachment, IOP is low with choroidal detachment being easily recognized on the fundus exam. However, not all cases of choroidal detachment are associated with a low IOP [3]. Aqueous misdirection, a diagnosis of exclusion, is characterized by flattening of the anterior chamber, increased IOP despite a patent iridectomy, the absence of choroidal detachment, and suprachoroidal hemorrhage or bleb overfiltration. IOP is usually, but not always, elevated [1]. The pathophysiology of aqueous misdirection glaucoma is not fully understood. A key mechanism is believed to be abnormal aqueous dynamics leading to vitreous expansion. The expanding vitreous then pushes the iris-lens diaphragm forward, causing secondary angle closure [10]. In addition, an inherent permeability defect in the anterior vitreous body as well as choroidal expansion may also play a role in the pathophysiological process [7].

Our case of annular peripheral choroidal detachment was difficult to diagnose because it presented with clinical characteristics similar to aqueous misdirection. Dugel et al. [8] reported 18 patients diagnosed initially as aqueous misdirection, but were found to subsequently have annular peripheral choroidal detachment. Annular peripheral choroidal detachment is usually not recognized by fundus examination because it typically does not extend posterior to the equator [8]. A peripheral retinal exam with sclera depression was not feasible for fear of wound dehiscence or leak in the early postoperative stage. It is easy to misdiagnose annular peripheral choroidal detachment as aqueous misdirection if the choroidal detachment is not detected. The important point is that annular peripheral choroidal detachment can be identified with standard echography, with a transverse approach as a smooth, or scallop-shaped thick membrane in the periphery with little aftermovement [8, 13]. Ultrasound biomicroscopy (UBM) is helpful to illustrate the anatomical changes associated with annular peripheral choroidal detachment. However, it is not available to every practice and requires an emersion technique that may unduly stress the postoperative eye, especially those with a flat anterior chamber and a weak bleb wound. Supraciliary fluid has been detected in patients by UBM and associated with aqueous misdirection glaucoma [1]. As the correct diagnosis, an alternative view to this finding could be that supraciliary fluid found on the ultrasound exam lends itself to annular choroidal detachment. If supraciliary fluid is detected by either ultrasound or UBM, the proper diagnosis should be annular peripheral choroidal detachment.

IOP is less helpful in diagnosing annular peripheral choroidal detachment as it can be high, low, or normal. In our case, IOP was always in the normal range. However, low IOP readings could be caused by enhanced aqueous outflow from the suprachoroidal space in the areas of detachment [14]. On the other hand, if the flat chamber is caused by anterior rotation of the ciliary body-iris-lens diaphragm, it may lead to secondary angle closure glaucoma and IOP might be normal or high [8]. Another hypothesis for normal IOP in our case is the possibility of an error in IOP measurement. Because the chamber was totally flat, there was direct contact between the cornea and the iris/IOL, a false high IOP may have been obtained.

Little is known about the pathogenesis of annular peripheral choroidal detachment. It is not clear why some cases have large choroidal effusion located posterior to the equator, and some cases develop annular peripheral choroidal detachment in which only a small amount

of fluid is needed to create a flat anterior chamber. Usually, choroidal effusion is a consequence of hypotony from overfiltration in the immediate postoperative period, which can be followed by a vicious cycle of hypotony and choroidal effusion. In our case, the patient did not have overfiltration within the first postoperative week. We believe that inflammation played a major role in his disease process. The patient had a history of steroid dependence and mild cold symptoms on the day of surgery and that may have made him prone to more postoperative inflammation, perhaps necessitating more frequent topical steroid use with or without oral prednisone. Although his bleb was never highly elevated to suggest true overfiltration, there could have been some relative overfiltration as he required bleb revision to resolve the choroidal detachment.

It is controversial whether annular peripheral choroidal detachment should be considered as a separate diagnosis [3, 8]. We deem it worthwhile to list it as a distinct entity to reinforce awareness of this condition. Once the diagnosis of annular peripheral choroidal detachment is confirmed by ultrasonography, the management should aim at resolving the choroidal detachment.

Some cases of annular peripheral choroidal detachment have responded well to treatment with topical cycloplegics and corticosteroids, but our case did not. Dugel et al. [8] reported that 10 out of 18 patients with annular peripheral choroidal detachment treated only medically had complete resolution of detachment with a mean time to resolution of 19.6 days. The authors hypothesized that this treatment tightens the zonules, rotates the ciliary body-iris-lens diaphragm backward and relieves the secondary angle closure.

Surgical treatment of annular peripheral choroidal detachment by drainage of the suprachoroidal effusion has been employed successfully by others [1, 8]. Dugel et al. [8] reported surgical drainage of suprachoroidal fluid, which resulted in the immediate resolution of 8 cases concerning annular peripheral choroidal detachment. However, they did not mention recurrent cases. In our case, the annular peripheral choroidal detachment recurred 3 days after the first drainage and the patient underwent repeat effusion drainage due to total flat anterior chamber. The scleral flap was reinforced with a scleral patch during the second suprachoroidal drainage, in case overfiltration played a role in the causes of his flat anterior chamber. The revision of the scleral flap helped to resolve the choroidal effusion. Trope et al. [10] treated a similar case of flat anterior chamber with normal IOP by using pars plana vitrectomy to resolve the supraciliary effusion. However, we believe that the surgical procedure of choice should be drainage of suprachoroidal fluid, not vitrectomy, if the presence of fluid is proven. Bleb revision is generally not required for choroidal detachment surgery. The decision whether to repair the bleb is made based upon clinical judgment during surgery. If a significant elevation of the bleb is observed while using a balanced salt solution to form the chamber during surgery, it is probably wise to reinforce the bleb. The surgeon for this case was debating whether to place a trans-conjunctival suture over the bleb during the first drainage, but decided against it because the thick conjunctiva made it difficult.

Severe corneal edema developed after the second drainage of choroidal effusion when the patient's oral and topical steroids were tapered. When the dosage of both, oral and topical steroid, was resumed, his cornea cleared up dramatically. The reason for the corneal edema is most likely due to the patient's steroid dependence and postoperative inflammation.

Conclusion

Annular peripheral choroidal effusion can cause a flat chamber without an obvious visible choroidal effusion on a clinical exam. It should be emphasized that annular peripheral choroidal effusion can easily be misdiagnosed as an aqueous misdirection when the IOP is normal or high. A transverse B-scan can detect the presence of shallow anterior choroidal detachment, which is critical for the diagnosis. We strongly recommend transverse B-scans for all patients with flat chamber when the diagnosis is not straight-forward. It is essential to differentiate annular peripheral choroidal effusion from aqueous misdirection. The appropriate surgical procedure is drainage of the suprachoroidal fluid, once it is confirmed by B-scan. Rapid steroid withdrawal in steroid dependent patients can cause severe corneal edema, which can be a challenge to treat and diagnose. Thus, steroids should be tapered very slowly in those patients.

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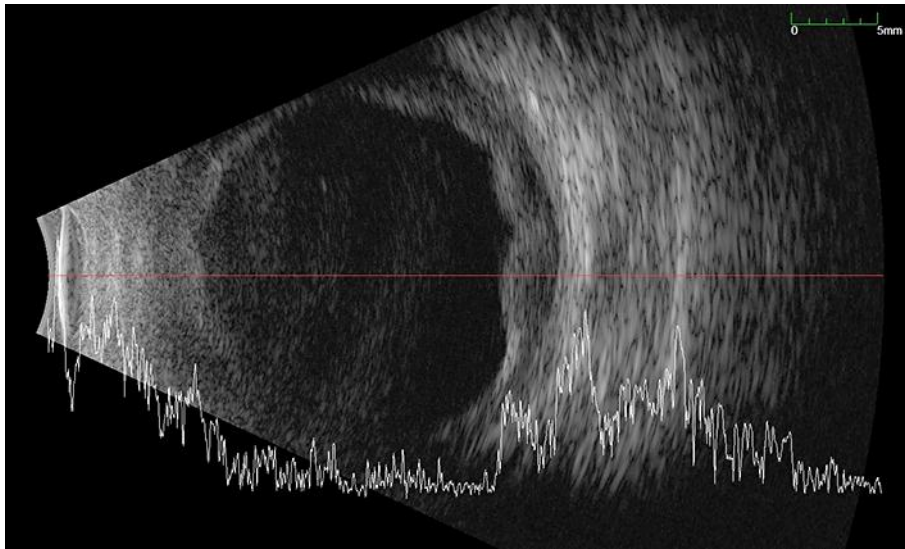


Fig. 1. Transverse B-scan from the temporal approach (probe placed on globe temporally and oriented vertically), showing an annular peripheral choroidal effusion [ring-shaped fluid accumulation between the choroid (arrow) and sclera (*)].