



Extensive serous ciliochoroidal detachments and macular subretinal and intraretinal fluid following laser peripheral iridotomy

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ABSTRACT

Purpose: We present multimodal imaging of an interesting case of a 78-year-old man who developed large ciliochoroidal detachments and macular subretinal and intraretinal fluid in the right eye following bilateral neodymium-doped yttrium aluminium garnet (Nd:YAG) laser peripheral iridotomies (LPIs).

Observations: The ciliochoroidal detachments developed in the absence of documented post-procedure hypotony or intraocular pressure fluctuation. Ultrasound biomicroscopy (UBM) confirmed serous ciliochoroidal detachment. There are a small number of cases of ciliochoroidal detachments developing after peripheral iridotomy, but these have involved either argon laser, significant decrease in intraocular pressure, or underlying ocular conditions or structural abnormalities, such as Vogt-Koyanagi-Harada (VKH) or nanophthalmos.

Conclusions: Serous ciliochoroidal detachments following the relatively non-invasive procedure of LPI are rare occurrences. We present our case in hopes of increasing awareness of this potential acute complication. We also discuss the diagnostic challenges of this unique case, the extensive work up, and current status of the patient.

1. Introduction

Laser peripheral iridotomy (LPI) can be used as both a curative and diagnostic procedure in a variety of clinical scenarios. Commonly cited indications include primary angle closure, plateau iris configuration, and combined-mechanism glaucoma.^{1,2} While argon LPI was previously standard of care, over the last several decades, it has largely been replaced by neodymium-doped yttrium aluminium garnet (Nd:YAG) LPI.^{1,3} Advantages of Nd:YAG laser iridotomy include fewer laser applications and less likelihood of closure of a previously patent iridotomy.² Overall, LPI is a well tolerated and safe procedure. One of the most devastating complications is a transient rise in intraocular pressure, which can, in rare circumstances, lead to irreversible vision loss.^{3,4,5} Other complications include anterior uveitis, corneal opacification, hyphema, and cataract formation.^{4,5,6} A recent study showed that upwards of 40% of patients who underwent LPI went on to have cataract surgery within the following 3 years.⁶

There are a small number of case reports of serous ciliochoroidal detachments following LPI. The pathophysiology in these cases has been suggested to relate to either significant intraocular pressure fluctuation, severe inflammation following argon laser iridotomy, or an underlying

pathologic process, such as Vogt-Koyanagi-Harada (VKH).^{7,8,9} Here we present an interesting case of serous ciliochoroidal detachments following an otherwise uncomplicated Nd:YAG LPI, and compare this to the available literature on the topic.

2. Case report

A 78-year-old man presented to the emergency eye clinic with a three-week history of intermittent headache, right eye pain, and bilateral conjunctival injection. His past ocular history was significant for amblyopia in the left eye. His general medical history included dyslipidemia, hypertension, TIA, myocardial infarction, rosacea, COPD, asthma and lung cancer. Current medications included metoprolol, clopidogrel, ASA, rosuvastatin, perindopril, fluticasone/salmeterol, and ipratropium bromide/albuterol. On exam, his best corrected visual acuity (BCVA) was 6/7.5 in the right eye, and 6/120 in the left. Intraocular pressures (IOPs) were 18 mmHg in both eyes using Goldmann applanation tonometry. Gonioscopic examination showed occludable angles bilaterally, and he was found to have very shallow anterior chambers, right eye more so than left. There was no anterior chamber cell in either eye. He had a mixed cortical and posterior subcapsular

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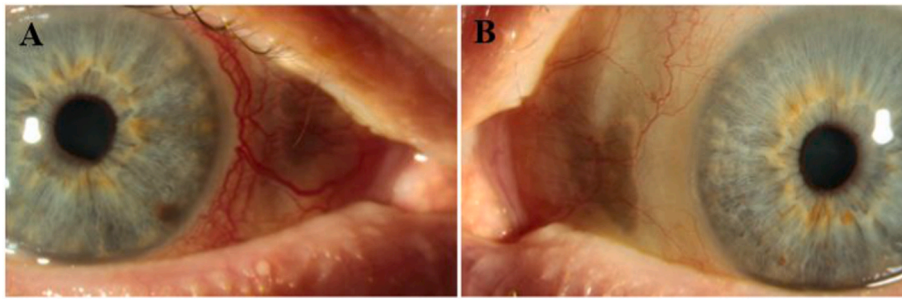


Fig. 1. Bilateral anterior segment photos showing prominent episcleral vessels at initial presentation.

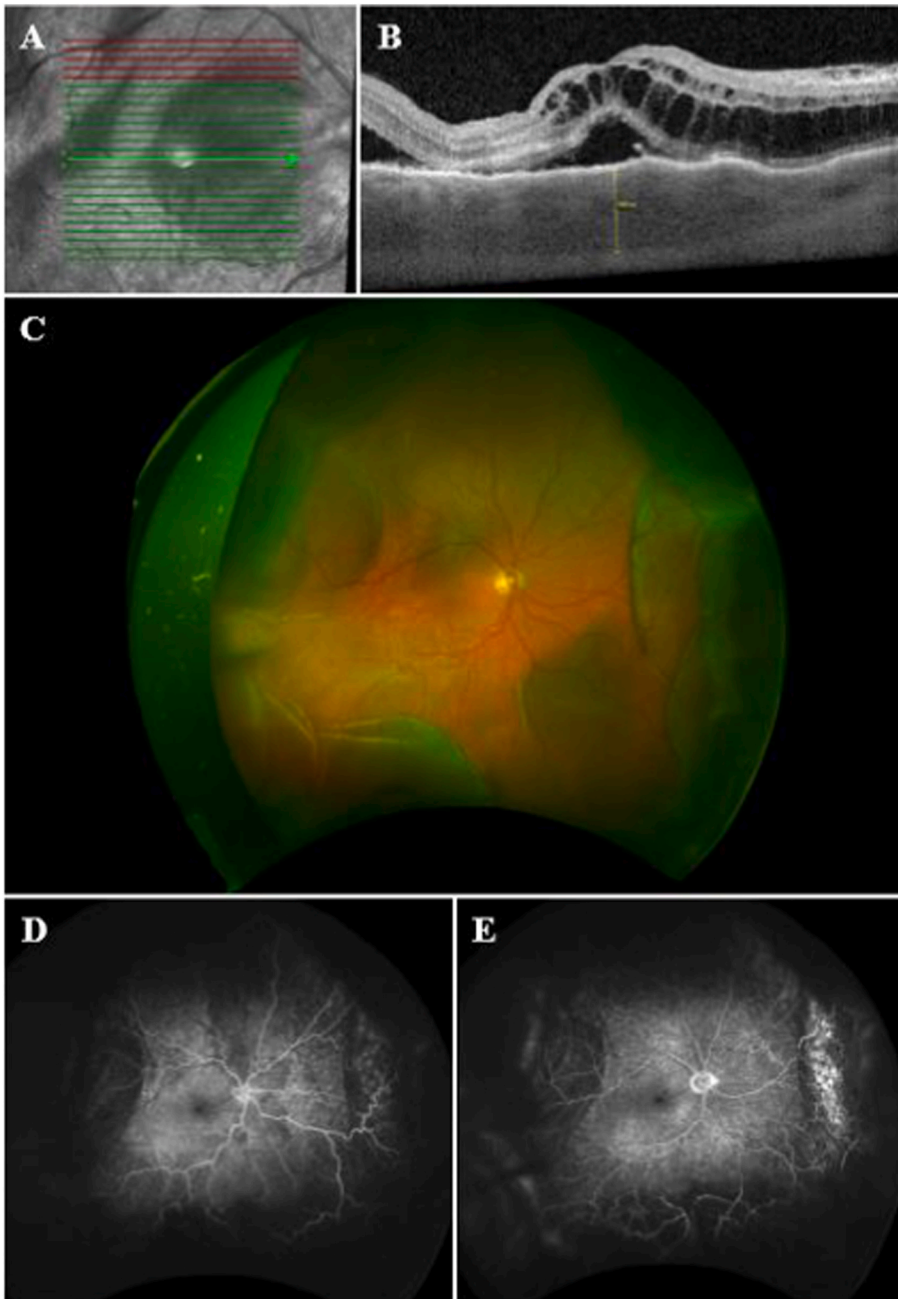


Fig. 2. Near-infrared image, enhanced depth imaging optical coherence tomography (EDI-OCT), Optos pseudocolour ultra-widefield fundus photograph, and ultra widefield fluorescein angiography (UWF-FA) of the right eye at initial presentation showing serous choroidal detachments and macular edema.

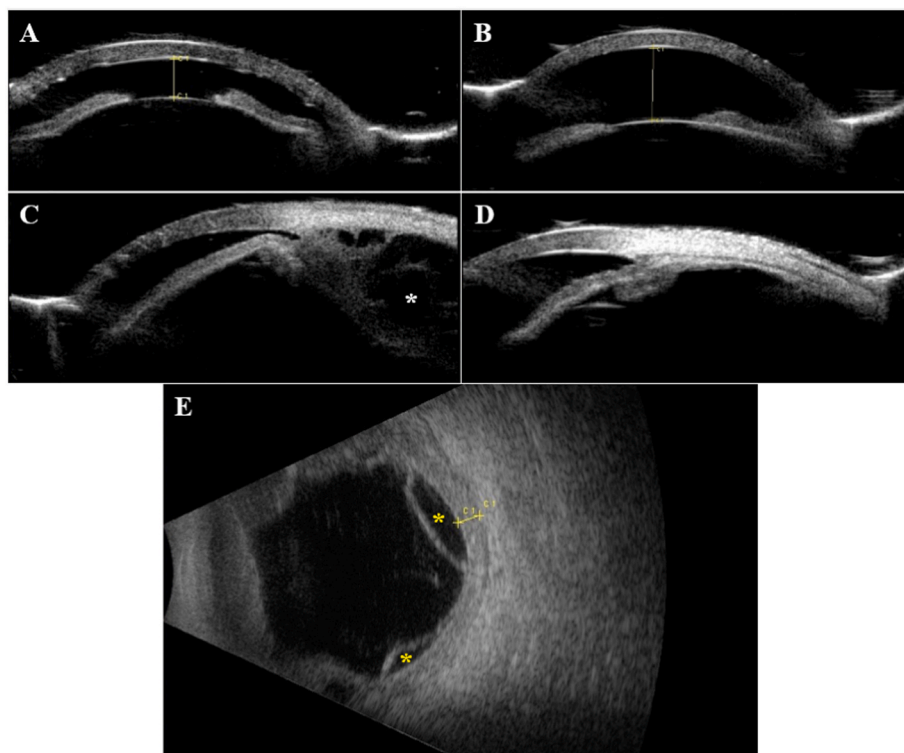


Fig. 3. Ultrasound biomicroscopy (UBM) and B-scan ultrasound of the right eye at initial presentation, as well as 4 months later showing resolution of the serous ciliochoroidal detachment.

cataract in the right eye, and mild nuclear sclerotic changes in the left lens. The optic disc, macula, and vessels were within normal limits in both eyes. The patient's presentation was consistent with plateau iris, and therefore bilateral Nd:YAG LPs were performed. Despite his amblyopia, it was felt that this would be a beneficial prophylactic measure in the left eye to preserve his vision, given that he did have an anatomically narrow angle. A total of 99 mJ and 180 mJ were delivered to the right and left eyes, respectively. The following day, visual acuities and IOP remained stable.

Three days later, the patient returned with ongoing headaches and progressively worsening vision in the right eye. Repeat examination showed a BCVA of 6/60 in the right eye, and 6/60 in the left eye. IOPs were 13 mmHg and 18 mmHg in the right and left eyes, respectively. His anterior chambers remained shallow. Both LPs were patent. There was 1+ anterior chamber cell in the right eye, and the left anterior chamber was quiet. Posterior segment examination revealed elevated retina in the right eye, and the patient was therefore referred to the retina service. He was also started on prednisolone drops four times per day in the right eye to address the ocular inflammation.

At his subspecialist appointment, his visual acuity and IOP were unchanged. The exam was consistent with prominent episcleral vessels in the right eye more than the left (Fig. 1). Dilated fundus exam (DFE) in the right eye showed extensive serous choroidal detachments throughout the fundus, as well as serous retinal detachments. This was confirmed on Optos pseudocolour ultra-widefield (UWF) fundus photography (Fig. 2C). Enhanced depth imaging optical coherence tomography (EDI-OCT) demonstrated subretinal fluid and cystoid edema in the macula and a thickened choroid (Fig. 2 A/B). Ultra widefield fluorescein angiography (UWF-FA) showed hypofluorescence in the areas of peripheral serous choroidal detachment, as well as late pooling nasally in the sub-RPE space (Fig. 2 D/E). There was no vitritis, optic disc edema, or mass lesions identified. From this visit, consults were made to ocular oncology, neuro-ophthalmology, and a uveitis specialist to rule out malignancy (given his history of lung cancer), carotid cavernous fistula, and posterior scleritis, respectively, as underlying

etiologies for his serous ciliochoroidal detachments. Persistent anterior chamber inflammation and worsening pain also lead to increasing the frequency of his prednisolone to one drop every hour, and initiating oral steroids at a dose of 60 mg once daily.

Ultrasound biomicroscopy (UBM) showed a shallow central anterior chamber depth of 1.3 mm (Fig. 3A). There was a large echo-free supraciliary space extending into the suprachoroid in all four quadrants, in keeping with serous ciliochoroidal detachment for 360° (Fig. 3C). B-scan ultrasonography showed diffuse choroidal thickening, with an average thickness of 1.7 mm (Fig. 3E). No evidence of a retinal or choroidal lesion was identified on ultrasonography. The patient had normal axial lengths of 23.16 mm and 23.47 mm in the right and left eyes, respectively.

A CT angiogram of the head and neck did not show any evidence of carotid cavernous fistula to explain the patient's dilated episcleral vessels and ciliochoroidal detachments. Clinically, he had no proptosis or cranial nerve palsies.

At the time of his initial visit in uveitis clinic, he had been on topical and oral steroids for about 2 weeks. He reported subjective improvement in vision and right eye pain since starting prednisolone. On exam, his BCVA had improved to 6/9 in the right eye, and remained stable at 6/60 in the left. IOPs remained within normal limits at 10 mmHg in the right eye, and 14 mmHg in the left. He continued to have prominent episcleral vessels in the right eye. His anterior chamber reaction had resolved, and his serous ciliochoroidal detachments had significantly improved. Bloodwork including creatinine, electrolytes, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), anti-cyclic citrullinated peptide (CCP) antibody, rheumatoid factor (RF), syphilis enzyme-linked immunosorbent assay (EIA), anti-myeloperoxidase (MPO), and anti-glomerular basement membrane (GBM) were all unremarkable. Complete blood count (CBC) showed a slightly elevated platelet count of $374 \times 10^9/L$, normal white blood cell count, and decreased hemoglobin of 131 g/L. The anti-proteinase level was equivocal at 0.4 AI.

Three months after his initial presentation, his BCVA remained stable at 6/9 in the right eye. Repeat EDI-OCT and fundus photos showed

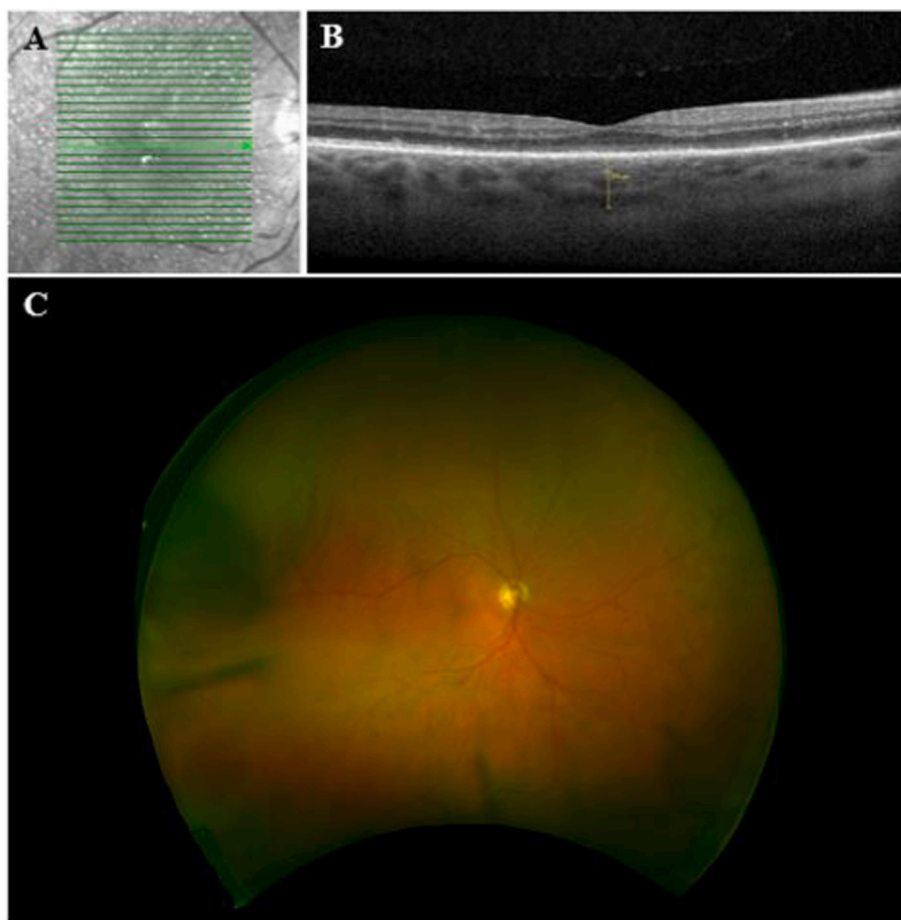


Fig. 4. Near-infrared image, EDI-OCT, and Optos pseudocolour ultra-widefield fundus photograph of the right eye 3 months after initial presentation showing near-complete resolution of the serous choroidal detachments and macular edema.

resolution of the macular subretinal fluid and cystoid edema (Fig. 4A/B) and near-complete resolution of the choroidal and retinal detachments (Fig. 4C). There was also normalization of the choroidal thickness from 650 μm at initial presentation to 316 μm . Repeat UBM done approximately 4 months after initial presentation showed normalization of the anterior chamber depth (Fig. 3B) and resolution of the serous ciliochoroidal detachment (Fig. 3D). At his most recent follow up, 5 months after initial presentation, his EDI-OCT was stable (Fig. 5A/B) and fundus photos showed near-complete resolution of the serous choroidal detachments (Fig. 5C).

3. Discussion

Sakai et al. demonstrated by UBM that small and limited ciliochoroidal detachment following LPI is common.¹⁰ However, large serous ciliochoroidal detachments following LPI have also rarely been reported. Kaden et al. describe a case of a woman in her 60s developing serous ciliochoroidal detachments two days following an uncomplicated laser iridotomy for narrow angle glaucoma.⁸ The ciliochoroidal detachments resolved after five days of treatment with oral prednisone and topical cycloplegia. The mechanism was felt to be related to the acute decrease in intraocular pressure that accompanied the LPI, which had also been described in a 2017 publication by Sakai et al.¹¹ This differs from our case in that there was no documented elevated IOP prior to the LPI, and only a modest decrease of 5 mmHg following the procedure. It is possible that the patient experienced more drastic fluctuations in IOP outside of what was captured during clinic visits, and that the laser iridotomy led to ciliary body detachment, transient hypotony, and subsequent ciliochoroidal detachments. However, it seems less likely

that acute changes in IOP were the sole underlying mechanism for the development of ciliochoroidal detachments in our patient, given the lack of documented IOP variability.

Cases of choroidal and retinal detachment have been documented after laser iridotomy performed with specifically argon laser.^{7,10,11} A study by Robin et al. found that a mean of 12 ± 11 and 0.033 ± 0.025 J were required during LPI for argon and Nd:YAG lasers, respectively.³ Because argon laser delivers more laser energy to the eye compared to Nd:YAG, it can result in comparably more intraocular inflammation. This inflammation, in turn, could account for the development of serous ciliochoroidal detachments. While our patient, in contrast, underwent uneventful Nd:YAG laser iridotomy, he did develop mild anterior chamber inflammation. Our patient's presentation, like that of Kaden et al.'s, resolved with systemic steroids. The benefit of steroids in treating this complication may be twofold: both by decreasing inflammatory burden and potentially raising intraocular pressure in a condition that can be precipitated by hypotony. There have been documented cases of patients developing ciliochoroidal detachments following Nd:YAG peripheral iridotomies, but these were in the unique setting of known VKH in one case, and nanophthalmos in the other.^{9,12}

Our case is unique in that extensive subretinal fluid and cystoid edema developed in the macula, which was not documented in the aforementioned cases.

Sakai et al. commented on prominent episcleral vessels in their patient who developed ciliochoroidal detachments following LPI.¹¹ Our patient was found to have a similar clinical presentation, and more so in the eye that developed marked ciliochoroidal detachments. Choroidal venous congestion and increased ciliary venous pressure have been considered as risk factors for the development of uveal effusion, and it is

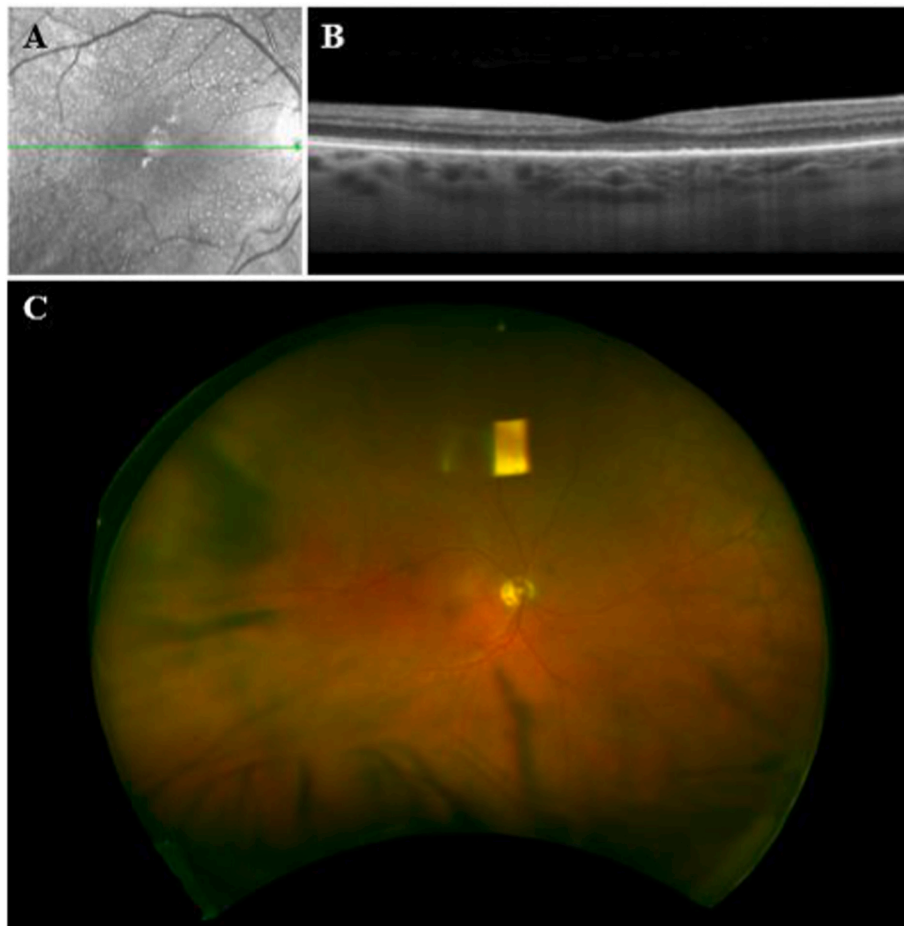


Fig. 5. Near-infrared image, EDI-OCT, and Optos pseudocolour ultra-widefield fundus photograph of the right eye 5 months after initial presentation showing complete resolution of the choroidal detachments and macular edema.

possible that dilated episcleral veins are a herald for this and should be specifically looked for and commented on prior to performing LPI.^{13,14}

Ciliochoroidal detachments have been described following other commonly performed ocular laser procedures, namely panretinal photocoagulation (PRP). The etiology is felt to be related to a thermally induced choroiditis that alters the normal fluid dynamics and oncotic gradients of the choroid.¹⁵ Risk factors for the development of serous ciliochoroidal detachment following PRP include using a higher number of laser applications, treating a larger area of retina, and having a shorter axial length.^{16,17} While the laser energy in LPI should in theory be more limited to the anterior segment compared to PRP, it is possible that similar processes could contribute in those undergoing LPI who have similar risk factors.

Primary angle closure itself can be associated with ciliochoroidal detachment, though typically on a more mild scale.^{18,19,20,21} One study found that 58% of eyes with acute primary angle closure glaucoma had demonstrable serous ciliochoroidal detachments on UBM at their initial visit.¹⁸ Proposed mechanisms for this observation include choroidal circulation congestion secondary to ocular hypertension, marked IOP fluctuation and hypotony, and uveal vasculature exudation from inflammation.^{20,21} Medication use has also been linked to ciliochoroidal detachments, including acetazolamide, pilocarpine, and check point inhibitors, such as ipilimumab and nivolumab.^{20,21,22} Our patient was not taking any of these medications. His clinical picture was felt to be consistent with intermittent angle closure. It is thus possible he was predisposed to developing worsening ciliochoroidal detachments following his LPI if angle closure in and of itself initiated the process.

Our case is unique in that the patient developed extensive

intraretinal and subretinal fluid in the macula in addition to impressive serous ciliochoroidal detachments following Nd:YAG LPI, without documented significant fluctuation in IOP. Our patient underwent Nd:YAG LPI, whereas the majority of prior documented cases occurred following argon laser procedures. Likely, the development of serous ciliochoroidal detachments and macular edema was multifactorial, with potential ciliary body detachment, transient hypotony, inflammation, and vascular congestion playing a role.

4. Conclusions

Ciliochoroidal detachments following the relatively non-invasive procedure of LPI are rare occurrences. Those cases that have been documented previously have been in the context of argon peripheral iridotomy, or otherwise have been found in eyes with underlying structural abnormalities or disease states, such as nanophthalmos or VKH. The majority resolve with systemic and/or topical steroids. Here we present an interesting case of a patient developing serous ciliochoroidal detachments, macular edema and subretinal fluid following uncomplicated Nd:YAG LPI, in hopes of increasing awareness of this potential acute complication.

Patient consent

Written informed consent was obtained from the patient.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Intellectual property

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

Research ethics

We further confirm that any aspect of the work covered in this manuscript that has involved human patients has been conducted with the ethical approval of all relevant bodies and that such approvals are acknowledged within the manuscript.

IRB approval was obtained (required for studies and series of 3 or more cases).

Written consent to publish potentially identifying information, such as details or the case and photographs, was obtained from the patient(s) or their legal guardian(s).

Anterior segment photography showing prominent, dilated episcleral vessels, more so in the right eye (A) compared to the left (B).

The near-infrared image (A) indicates the location of the cross section shown in the OCT (B) with a highlighted green horizontal line. The EDI-OCT shows marked intraretinal and subretinal fluid, with loss of the normal foveal contour, as well as choroidal thickening (indicated by the yellow calipers, showing a thickness of 650 μm). The pseudocolour UWF fundus photo (C) shows 360° of serous choroidal detachments, with associated serous retinal detachments. The early (D) and late (E) UWF-FAs show hypofluorescence peripherally, in the areas of serous choroidal and retinal detachment. There is also late hyperfluorescence noted nasally, consistent with pooling.

The UBM (A) shows a shallow central anterior chamber depth (indicated by the yellow calipers, C1) of 1.3 mm. There is a large echo-free supraciliary space (indicated by the yellow asterisk) extending into the suprachoroid (C), in keeping with serous ciliochoroidal detachment. The B-scan ultrasound (E) shows diffuse choroidal thickening, with an average thickness of 1.7 mm (indicated by the yellow calipers, C1). Multiple shallow serous retinal detachments are also appreciated (indicated by the white asterisks).

The follow up UBM (B) shows a normal central anterior chamber depth (indicated by the yellow calipers, C1) of 2.38 mm. There is resolution of the large echo-free supraciliary space extending into the suprachoroid (D).

The near-infrared image (A) indicates the location of the cross section shown in the OCT (B) with a highlighted green horizontal line. The EDI-OCT shows resolution of the intraretinal and subretinal fluid, with a normal foveal contour appreciated. The choroid has returned to a normal thickness (indicated by the yellow calipers, showing a thickness of 316 μm). The pseudocolour UWF fundus photo (C) shows near-complete resolution of the serous choroidal and retinal detachments, with artifact noted superotemporally due to cortical cataract.

The near-infrared image (A) indicates the location of the cross section shown in the OCT (B) with a highlighted green horizontal line. The EDI-OCT shows resolution of the intraretinal and subretinal fluid, with a normal foveal contour appreciated. The pseudocolour UWF fundus photo (C) shows complete resolution of the serous choroidal and retinal detachments, again with artifact noted superotemporally due to cortical cataract.

Declaration of competing interest

The following authors have no financial disclosures: DB, AZ, JM, HL, RRG.

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