Case Report

Acute iridocyclitis and cystoid macular edema related to kinked Hydrus® Microstent in advanced glaucoma

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Abstract

A 58-year-old male who underwent cataract extraction with combined intraocular lens and Hydrus® Microstent (Ivantis Inc, Irvine, CA, US) implantation 2 years ago in the right eye (OD) due to advanced glaucoma presented with blurry vision in right eye (OD) for 3 months. The visual acuity was 20/60 and slit-lamp examination indicated mild anterior chamber inflammation with unexposed, functioning tube shunt superotemporally in OD. Optical coherence tomography demonstrated cystoid macular edema (CME) with subretinal fluid. Fluorescein angiography demonstrated petaloid pattern leakage of CME. Gonioscopy revealed a kinked appearance of a Hydrus® Microstent protruding into the anterior chamber and causing iris chafing. Topical ketorolac tromethamine and prednisolone acetate were started. At the 2nd month of follow-up, the anterior chamber was quiet, and the CME resolved completely. Protruded kinked Hydrus® Microstent may lead to acute iridocyclitis and CME through iris chafing, which may be responsive to topical anti-inflammatory drops.

Keywords:

Cystoid macular edema, Hydrus® Microstent, ocular inflammation, protruding, subretinal fluid

Introduction

Cystoid macular edema (CME) is characterized by macular thickening with cystic spaces in the outer plexiform and inner nuclear layers, which occurs secondary to breakdown of the inner and outer blood–retinal barriers. Sometimes, abnormal permeability of perifoveal capillaries can also present with concurrent subretinal fluid accumulation.[1] CME is usually considered to be associated with ocular inflammation. Several risk factors have been established for CME, including cataract surgery, uveitis, diabetic retinopathy, retinal vein occlusion, retinitis pigmentosa, medications like prostaglandin analogs, vitreomacular traction syndromes, intraocular tumors, and optic nerve head abnormalities.[1] Although glaucoma itself is not a known risk factor for CME following cataract surgery, Lee *et al*.[2] demonstrated that untreated increased intraocular pressure (IOP) with glaucomatous optic nerve damage, retinal nerve fiber layer and/or visual field loss is related to a higher risk of postoperative pseudophakic CME.

Glaucoma is a progressive disease and involves various treatment modalities for the reduction of IOP to a target level.[3] Recently, micro-or minimally invasive glaucoma surgeries (MIGS) evolved rapidly with the advent of biomaterials and micro-assembly technologies. Usually indicated for treating mild-to-moderate glaucoma, they are promoted to result in shorter surgical times, and less severe complications as compared to traditional glaucoma surgery.[4] The Hydrus® Microstent (Ivantis, Inc., Irvine, CA) is a MIGS device that acts through bypassing trabecular outflow.[4] It was approved by the FDA in 2018 as a safe and efficacious device for use in a combined procedure with phacoemulsification in the treatment of open-angle glaucoma.[5]

Herein, we present a case with acute iridocyclitis and CME as a long-term complication related to kinked Hydrus® Microstent in the anterior chamber causing iris chafing syndrome.

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Case Report

A 58-year-old male presented to a tertiary eye center with blurry vision in the right eye for 3 months. The medical history was unremarkable except for atrial fibrillation which was well controlled with apixaban. The visual acuity (VA) was 20/60 and 20/30 in the right (OD) and left eye (OS), respectively. The patient had advanced glaucoma with a cup-to-disk ratio of 0.9 in both eyes (OU). The IOPs were 19 mmHg in OD and 12 mmHg in OS, for which the patient was using travoprost once daily OU, timolol maleate twice daily OU, and dorzolamide hydrochloride twice daily OD. The patient had undergone Ahmed glaucoma tube shunt implantation 5 years ago, and cataract extraction with combined intraocular lens (IOL) and Hydrus® Microstent (Ivantis, Inc., Irvine, CA) implantation 2 years ago in OD. On slit-lamp examination (Figure 1a), there were 1+ white anterior chamber cells, posterior synechiae and unexposed, functioning tube shunt superotemporally along with clear cornea and posterior chamber IOL in OD. Fundus examination showed 1+ pigmented cells in the anterior vitreous without any haze or snowballs, along with slightly elevated appearance of the macula and a circular area of pigmentary change superotemporally [Figure 1c]. Gonioscopy revealed kinked appearance of Hydrus® Microstent protruding into the anterior chamber with iris contact [Figure 1b]; there was no evidence of IOL dislocation. Optical coherence tomography (OCT) demonstrated CME with subretinal fluid in OD [Figure 1e]. Fluorescein angiography (FA) showed mild staining of optic disc along with increased hyperfluorescence at the macula in late phase which was consistent with a petaloid pattern of leakage of CME [Figure 1d]. Retinal vessels did not show any leakage suggestive of vasculitis.

The patient was a gardener, but there was no known history of ocular trauma or any prior episode of intraocular inflammation. Uveitis evaluations for both infectious and noninfectious causes were negative except for positive Toxoplasma IgG titers, and HLA-B51 positivity. A diagnosis of CME and acute iridocyclitis associated with iris chafing syndrome secondary to kinked Hydrus® Microstent in the anterior chamber. The patient was started on topical ketorolac tromethamine four times daily and prednisolone acetate four times daily in OD. Travoprost was stopped in OD, and brimonidine tartrate three times daily OU was added to his regimen due to higher IOP levels than target IOP during this treatment course. At the 2nd month of follow-up [Figure 2], the anterior chamber was quiet. Gonioscopy confirmed the persistent irido-touch of the Hydrus® Microstent with peripheral anterior synechiae (PAS) over portions of the microstent [Figure 2b]. The CME and subretinal fluid resolved completely on OCT [Figure 2e], and the FA did not show any leakage at the optic disc, macula, or peripheral vessels [Figure 2d]. VA improved to 20/25, and IOP was 12 mmHg in OD. Anterior and posterior segment

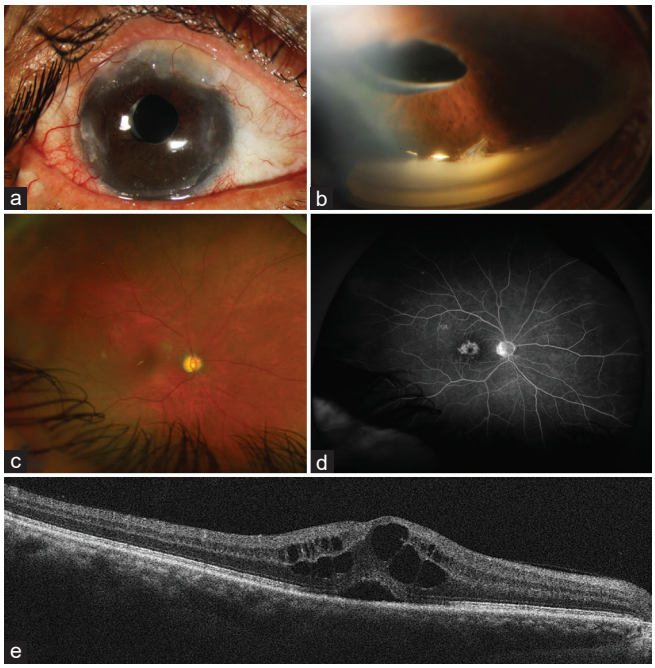

Figure 1: Initial findings of the index patient with Hydrus® Microstent presenting with acute iridocyclitis and cystoid macular edema (CME). (a) Slit-lamp photography showed injected conjunctiva; (b) gonioscopy showed kinked microstent in iridocorneal angle superotemporally with iris chafing; (c) colored fundus photography demonstrated cupped optic nerve; (d) fluorescein angiography demonstrated petaloid type leakage consistent with CME; (e) optical coherence tomography revealed intraretinal cystic changes consistent with CME

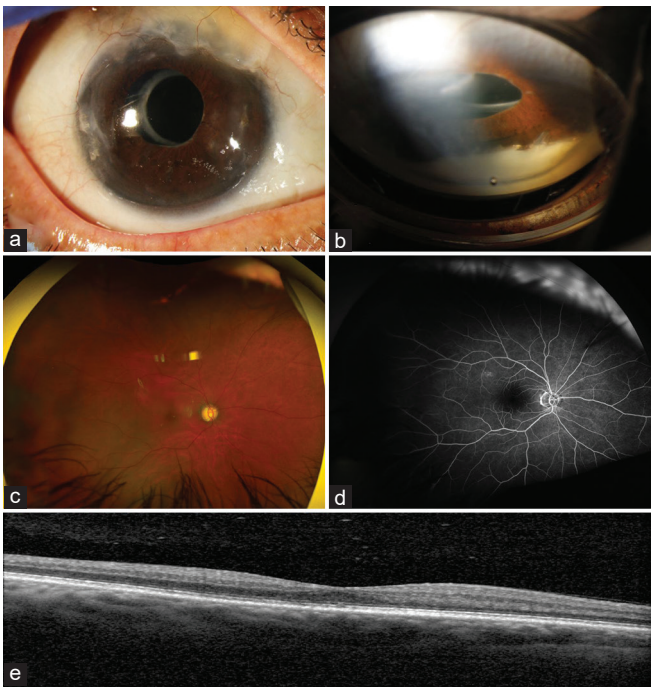

Figure 2: Clinical findings 2 months after treatment. (a) Slit-lamp photography revealed much less injected conjunctiva; (b) gonioscopy showed persistent but stable kinked microstent in iridocorneal angle superotemporally; (c) colored fundus photography revealed no new retinal pathology; (d) fluorescein angiography demonstrated no leakage from macula or optic disc; (e) optical coherence tomography revealed resolution of cystoid macular edema

examination was unremarkable in OS except for mild nuclear sclerosis during the follow-up examination.

Discussion

The Hydrus® Microstent (Ivantis, Inc., Irvine, CA), which is composed of nitinol (nickel-titanium) alloy, is an 8-mm-long curved, superelastic aqueous drainage MIGS device. It functions as a scaffold within the Schlemm's canal by expanding the canal to nearly 4–5 times of its physiological width. The Hydrus® Microstent is inserted into the Schlemm's canal under gonioscopic guidance through a preloaded injector, and spans 3 clock hours (90°, one quadrant) providing access to multiple collector channels.[5] In addition to rare intraoperative adverse events such as malposition and transient hyphema, the formation of PAS and iris adhesions is the most common postoperative complications of Hydrus® Microstent. However, those complications were not linked to IOP increase or device malfunction.[6,7] Postoperative inflammation was mostly transient and resolved within the 1st month. Persistent uveitis requiring steroid treatment for >3 months was only observed in 0.5% of cases during 2 years of follow-up.[6]

In our case, 2 years following the combined surgery, a kinked Hydrus® Microstent protruding into the anterior chamber provoked acute inflammation by iris chafing, which was only visible by gonioscopy. In the literature, iris chafing syndrome was usually linked to anterior chamber IOLs, and less frequently, sulcus-or scleral-fixated IOLs with resultant elevated IOP, anterior chamber inflammation, and recurrent hyphema, although any of these can occur in isolation.[8] Hou *et al*.[9] also reported an asymptomatic patient having persistent inflammation without evidence of iris atrophy or IOL dislocation related to EX-PRESS glaucoma filtration device 8 years after uneventful implantation. In their case, gonioscopy revealed localized iris atrophy under the shunt with surrounding iris billowing and layered hyphema, which resolved with a localized laser iridoplasty. On the contrary, there was no iris atrophy in our case which is likely due to the acute onset and relatively small area of iris chafing. Despite no identified ocular trauma history, the reason for the kinked microstent may be his occupation as a gardener, which may put him at risk for both microtrauma, and elevated venous pressure due to bending.

Given the increasing number of glaucoma device implantations with evolving MIGS technology, glaucoma device-associated ocular inflammatory complications such as iris chafing, and CME development may become more prevalent. These may adversely affect long-term outcomes if untreated. Iris chafing and related uveitis–glaucoma–hyphema syndrome are treated surgically to relieve the mechanical friction of IOLs, as the topical anti-inflammatory and antiglaucoma medications do not always treat the underlying cause. However, for the treatment of glaucoma stent-related ocular inflammation, stent repositioning or removal might not be efficient and/or possible due to postoperative PAS and iris adhesions.[6,7] Furthermore, surgery-related exacerbation of inflammation may lead to obstruction and subsequent failure of the stent, leading to elevated IOP and further glaucoma progression. Therefore, medical treatment with topical NSAIDs and steroids as a first-line approach seems reasonable for the treatment of such patients.[1,10] The present case also responded well to topical anti-inflammatory treatment with complete resolution of CME at the 2nd month of follow-up. Topical antiglaucoma medications sufficiently controlled IOP during his course. However, long-term follow-up is still needed to assess the efficacy of this treatment and associated outcomes. In addition, repeated gonioscopy is indicated due to persisting abnormal shape of microstent. To the best of our knowledge, the index case is the first report demonstrating kinked appearance of Hydrus® Microstent as a cause of acute iridocyclitis and CME.

In conclusion, late postoperative persistent or recurrent inflammation can be observed during the follow-up of patients who underwent Hydrus® Microstent implantation. Topical NSAIDs and steroids appear to be successful in the management of associated inflammation. Regular gonioscopic examination is essential to recognize and manage the subtle changes related to microstents and angle structures. Cumulative evidence from future reports will also be helpful.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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