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Acute iris vascular tuft hemorrhage treated successfully with intravitreal bevacizumab and pressure patching in a patient with branch retinal vein occlusion

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ARTICLE INFO	A B S T R A C T
Keywords: Branch retinal vein occlusion Hyphema Iris vascular tuft Microhemangioma Vascular endothelial growth factor	Purpose: To report the presentation and management of active iris vascular tuft (IVT) hemorrhage and sponta- neous hyphema in an eye with previous branch retinal vein occlusion (BRVO). Observations: A 74-year-old male with a history of BRVO in the left eye presented with spontaneous hyphema and blurred vision. Clinical examination confirmed the presence of an actively bleeding IVT at the pupillary margin in the left eye. Sustained hemostasis was achieved following intravitreal bevacizumab injection and pressure patching of the eye. Conclusions and Importance: This is the first case report to demonstrate pressure patching as a non-invasive, effective method of achieving hemostasis in the acute setting of IVT hemorrhage. Intravitreal vascular endo- thelial growth factor antagonists such as bevacizumab may also be useful in decreasing the risk of IVT hemor-

1. Introduction

Iris vascular tufts (IVT), also known as Cobb tufts or microhemangiomas, are benign capillary outgrowths from the pupillary margin that range from 15 to 150 μ m in size.^{1,2} Patients with IVT are typically asymptomatic but in rare cases present with spontaneous bleeding into the anterior chamber, which may result in sudden blurring of vision and elevated intraocular pressure (IOP).^{2–6} Given the rarity of IVT, there is no supported consensus in the literature regarding IVT management, particularly in the setting of active IVT bleeding. However, treatment approaches such as laser photocoagulation^{2,7} and iridectomy^{2,8} have been described.

Herein, we document a case of spontaneous hyphema secondary to a bleeding IVT in an eye with previous branch retinal vein occlusion (BRVO), and we outline a minimally invasive approach to achieving hemostasis in the setting of active IVT bleeding.

2. Case report

A 74-year-old male with a remote history of BRVO of the left eye in 2013 presented to clinic with acute, painless monocular vision loss of the left eye. He had no preceding trauma, surgery, or anticoagulant use. He

previously had neovascularization of the iris and retina following the BRVO, which resolved with intravitreal bevacizumab injections in 2013 and pan-retinal photocoagulation in 2015 and 2017. He described a history of hypertension that was well-controlled on medication, and there was no personal or family history of bleeding disorders. Visual acuity on arrival was 20/25 (20/20 with pinhole) in the right eye and 20/20 in the left eye. IOP was slightly elevated in the left eye (22 mmHg) relative to the right eye (14 mmHg). Initial external evaluation demonstrated diffuse conjunctival injection of the left eye with what appeared to be an active iris bleed within the anterior chamber (Fig. 1). On slit-lamp examination, a 4-mm layering hyphema was noted inferiorly in the anterior chamber of the left eye. This hyphema was fed by a continuous filiform hemorrhage originating from an IVT at the 1 o'clock position of the pupillary margin (Video 1). There was no obvious neovascularization of the iris (NVI), although subtle neovascularization of the iridocorneal angle (NVA) was noted on gonioscopy. Dilated fundus examination demonstrated findings consistent with a previous BRVO in the left eye, including tortuosity of the superior retinal vessels without active edema or retinal neovascularization.

Supplementary video related to this article can be found at htt ps://doi.org/10.1016/j.ajoc.2022.101780

The IVT continued to bleed despite pupillary dilation with

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Fig. 2. Fluorescein angiography (FA) of the right (A) and left (B) iris demonstrating hyperfluorescent iris vascular tufts (IVTs) and late staining along the pupillary margin in both eyes. (C) A magnified FA photo of the left iris shows multiple hyperfluorescent IVTs, including an IVT at the 1 o'clock position of the pupillary margin (arrow) in the area of prior hemorrhage.

Fig. 1. External photograph of the left eye demonstrating diffuse conjunctival injection and a filiform iris hemorrhage feeding into an inferior layering hyphema.

tropicamide 1% and phenylephrine 2.5%. Given the presence of NVA on gonioscopy and the patient's prior success with anti-vascular endothelial growth factor (VEGF) therapy, the decision was made to proceed with intravitreal injection of bevacizumab. The patient was positioned and prepped in a sterile manner, and the injection was performed without complications. IOP in the left eye immediately following this injection was 48 mmHg and decreased to 35 mmHg within 10 minutes of the injection. On re-evaluation at the slit-lamp, a steady hemorrhage continued to flow from the same IVT. To promote hemostasis, a pressure patch was then formed by placing two eyepads over the closed left eye and applying tape firmly over the patch to hold it in place. After 30 minutes the pressure patch was removed, and bleeding from the IVT had ceased. The patient was provided with prednisolone 1%, atropine 1%, and dorzolamide/timolol eye drops for management of the hyphema, and he was sent home in stable condition.

After 1 day, IOP of the left eye had improved to 10 mmHg, the hyphema had decreased in height to 1 mm, and a quiescent IVT was noted at the location of the previous hemorrhage. Three weeks after initial presentation, the hyphema had completely resolved. An iris angiogram was obtained at 1 month and demonstrated no active bleeding or NVI; irregular hyperfluorescence was present at the pupillary margins of both eyes suggestive of bilateral IVTs (Fig. 2). Over the course of 2 months, the patient's visual acuity and IOP remained stable, and there was no recurrence of IVT hemorrhage.

3. Discussion

This case of spontaneous hyphema secondary to IVT hemorrhage is unique due to its occurrence in an eye with prior BRVO and its noninvasive management with pressure patching. While IVTs are known to occur more frequently in elderly, diabetics, and myotonic dystrophy patients,² few reports have described IVTs in association with acute or chronic retinal vein occlusion.^{3,9} The presence of *bilateral* IVTs in this

patient with a left-sided BRVO strongly suggests against a causal relationship between retinal vein occlusion and IVT development. However, some have suggested that the ischemic and hemodynamic changes caused by vascular occlusions may contribute to alterations in IVT structure that make them more prone to spontaneous bleeding.³ In our case, given the presence of NVA in the setting of chronic retinal ischemia of the left eye, it is possible that upregulation of VEGF (which promotes angiogenesis and increased vascular permeability) heightened the risk of IVT bleeding in this eye relative to the non-BRVO eye.¹⁰

When active IVT hemorrhage presents in clinic, it may pose a therapeutic challenge of arresting active bleeding in a timely manner to prevent adverse sequelae such as ocular hypertension and corneal blood staining. Therefore, understanding efficient and effective methods of arresting such bleeding is vital. Argon laser photocoagulation has been described for the treatment of actively bleeding IVTs.^{2,7,11} While this method has been shown to successfully stop active IVT hemorrhage, it entails its own set of risks including iris damage, anterior chamber bleeding, and corneal decompensation.^{2,12} In addition, timely access to an argon laser may vary in settings with limited resources. Given these potential barriers and complications, laser photocoagulation is typically reserved for patients with recurrent IVT hemorrhage or as a prophylactic measure for patients undergoing intraocular surgery.^{2,12,13} Limited trials of compression gonioscopy or digital compression have also been described, although these reports have shown unclear and inconsistent effectiveness in achieving adequate hemostasis.^{11,14} In our case, we demonstrate successful cessation of a large, active IVT hemorrhage directly following the application of a pressure patch over the involved eye. While it is possible that the preceding bevacizumab injection also contributed to hemostasis through additional elevation of the IOP, full arrest of the bleeding was not achieved until after steady, prolonged pressure was applied to the eye.

4. Conclusions

In summary, we report a case of spontaneous hyphema from an actively bleeding IVT in an eye with a previous BRVO. Hemostasis was successfully achieved in this case following intravitreal bevacizumab injection and pressure patching of the eye. We propose closely

monitored pressure patching as a first line, minimally invasive and accessible treatment to achieve IVT hemostasis in the acute setting—particularly in patients with no prior history of IVT bleeding. For recurrent spontaneous IVT hemorrhage, more invasive and definitive modalities such as laser photocoagulation may be considered. Intravitreal bevacizumab may also be useful in the rare setting of concurrent IVTs and retinal vein occlusion, however further investigation is needed to better characterize the relationship between VEGF activity and the risk of spontaneous IVT hemorrhage.

Patient consent

Consent to publish this case was verbally obtained from the patient. Additionally, this report does not contain any personal information that could lead to the identification of the patient.

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