American Journal of Ophthalmology Case Reports 7 (2017) 138–139

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: http://www.ajocasereports.com/

Case report

Reversal of cilioretinal artery occlusion with intra-arterial tissue plasminogen activator

Andrew J. McClellan [a], Harry W. Flynn Jr. [a, *], Eric C. Peterson [b], Norman J. Schatz [a]

[a] Department of Ophthalmology, Bascom Palmer Eye Institute, University of Miami, Miller School of Medicine, 900 NW 17th Street, Miami, FL 33133, USA
[b] Department of Neurological Surgery, University of Miami, Miller School of Medicine, 1095 NW 14th Terrace, Miami, FL 33136, USA

ARTICLE INFO

Article history:
Received 1 January 2017
Received in revised form
12 June 2017
Accepted 6 July 2017
Available online 8 July 2017

Keywords:
Cilioretinal artery occlusion
Tissue plasminogen activator

ABSTRACT

Purpose: To present the reversal of a cilioretinal artery occlusion with the use of intra-arterial tissue plasminogen activator.
Observations: A monocular 74 year old male presented with a cilioretinal artery occlusion. Treatment with intra-arterial tissue plasminogen activator 7 hours after the onset of symptoms led to a complete restoration of vision.
Conclusions and importance: Early intervention with tissue plasminogen activator reversed acute onset loss of vision from cilioretinal artery occlusion.

1. Introduction

Retinal arterial occlusion is a devastating disease that often leads to permanent and severe loss of vision. Although many treatment options have been proposed, including ocular massage, anterior chamber paracentesis, hemodilution, and hyperbaric oxygen, none have been shown to consistently improve visual outcomes.[1] It has been reported that with vascular occlusion of greater than 4 hours, irreversible damage may occur to the retina.[2] The use of tissue plasminogen activator (TPA) to treat vascular occlusions in other fields of medicine has led to an increased interest in ophthalmology as a potential treatment for retinal vaso-occlusive disease. The patient presented herein received such treatment with successful visual recovery.

2. Case report

A 74 year old male presented with symptoms of intermittent vision loss in his right eye (amaurosis fugax) one hour prior to presentation. The symptoms progressed to a persistent, dense central scotoma with a visual acuity of 20/400 eccentrically. His recent symptoms began one week after discontinuing aspirin therapy in anticipation of a surgical procedure. Ocular history included a central retinal vein occlusion in the left eye, which had progressed to neovascular glaucoma, LP vision, and intermittent bouts of extreme ocular pain. Medical history is significant for hypertension and hyperlipidemia. The posterior segment examination and angiography demonstrated widely scattered dot hemorrhages consistent with an evolving central venous occlusion in the symptomatic eye, with concomitant cilioretinal artery occlusion (Fig. 1). Choroidal and arterial filling was reduced and delayed as well (not shown). The patient was immediately transferred to the stroke team. Cerebral angiography revealed delayed filling of the ophthalmic artery, and the patient was subsequently treated with intra-arterial recombinant tissue plasminogen activator (TPA) at 7 hours after the onset of symptoms (Figs. 2 and 3). Immediately following this treatment, the central scotoma and visual acuity in the right eye began to improve. Secondary systemic management included the initiation of clopidogrel, resumption of aspirin, and evisceration of the blind, painful fellow eye. Following cataract surgery six months after treatment, the visual acuity improved to 20/15 in the right eye.

3. Discussion

The significance of this report is that early intervention reversed acute onset loss of vision from cilioretinal artery occlusion. Acute presentation of retinal arterial occlusion is not often seen, although it is a crucial aspect of treatment with TPA. Similar to stroke, there are some reports of the use of TPA for retinal occlusive disease that suggest there is a similar “window” of time after the onset of symptoms that treatment must be administered for it to be

* Corresponding author.
E-mail address: hflynn@med.miami.edu (H.W. Flynn).

http://dx.doi.org/10.1016/j.ajoc.2017.07.002

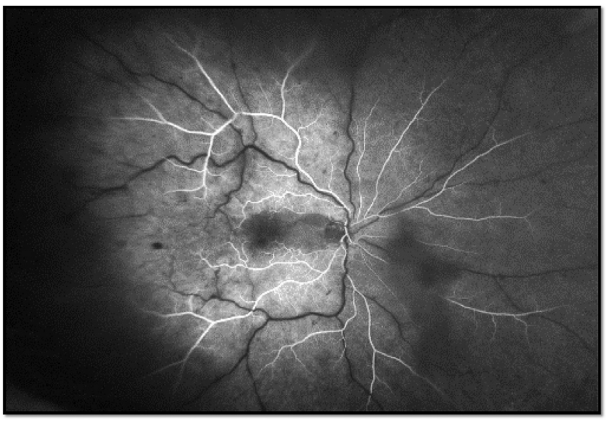

Fig. 1. Fluorescein angiography of the right eye demonstrating nonperfusion in the distribution of the cilioretinal artery.

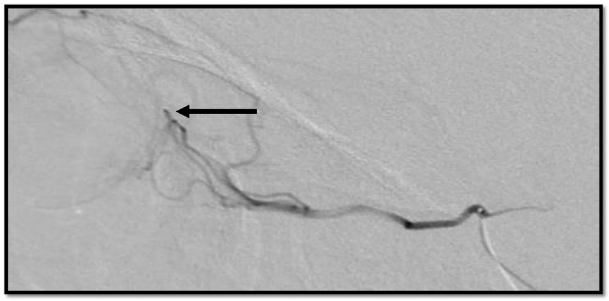

Fig. 2. Angiography of the right ophthalmic artery prior to tissue plasminogen activator infusion. Arrow identifies the point of obstruction.

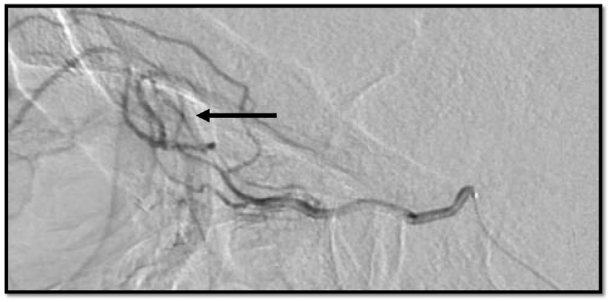

Fig. 3. Angiography of the right ophthalmic artery after tissue plasminogen activator infusion. Arrow shows arterial blood flow beyond point of obstruction.

effective, which may also be affected by the route of administration.[3] This patient's monocular status likely increased awareness of symptoms, leading to an earlier presentation than a patient with intact binocular vision. In the setting of stroke, TPA is commonly administered via intravenous or intra-arterial infusion. Studies by Hattenbach and Chen have shown improvement in visual acuity if administered systemically within 6 hours of the onset of symptoms, whereas a study by Aldrich et al. showed improvement in visual acuity if locally administered intra-arterially within 15 hours of the onset of symptoms.[4–6] Although the treatment "window" may be prolonged by intra-arterial delivery, it is possible that this more invasive route may increase the risk of adverse events while lending no or minimal advantage in visual outcome when the patient is treated up to 20 hours after onset.[7]

4. Conclusions

Though cilioretinal artery occlusion can be reversed with TPA, potential adverse events include hemorrhagic stroke and death.[8] Therefore, assessment of the risk to benefit ratio for the individual patient is important. For the patient presented herein, his monocular status factored heavily into the decision to pursue treatment with TPA.

Patient consent

The patient orally consented to the publication of this case.

Funding

National Institute of Health Center Core Grant P30EY014801 (Bethesda, Maryland), Research to Prevent Blindness Unrestricted Grant (New York, New York), and the Department of Defense W81XWH-09-1-0675 (Washington, DC).

Conflicts of interest

The following authors have no financial disclosures: AM,HF,EP,NS.

Authorship

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

Acknowledgements

None.

References

1. Fraser SG, Adams W. Interventions for acute non-arteritic central retinal artery occlusion. *Cochrane Database Syst Rev*. 2009:Cd001989.
2. Hayreh SS, Zimmerman MB, Kimura A, Sanon A. Central retinal artery occlusion. Retinal survival time. *Exp Eye Res*. 2004;78:723–736.
3. Schrag M, Youn T, Schindler J, Kirshner H, Greer D. Intravenous fibrinolytic therapy in central retinal artery occlusion: a patient-level meta-analysis. *JAMA Neurol*. 2015;72:1148–1154.
4. Hattenbach LO, Kuhli-Hattenbach C, Scharrer I, Baatz H. Intravenous thrombolysis with low-dose recombinant tissue plasminogen activator in central retinal artery occlusion. *Am J Ophthalmol*. 2008;146:700–706.
5. Chen CS, Lee AW, Campbell B, et al. Efficacy of intravenous tissue-type plasminogen activator in central retinal artery occlusion: report from a randomized, controlled trial. *Stroke*. 2011;42:2229–2234.
6. Aldrich EM, Lee AW, Chen CS, et al. Local intraarterial fibrinolysis administered in aliquots for the treatment of central retinal artery occlusion: the Johns Hopkins Hospital experience. *Stroke*. 2008;39:1746–1750.
7. Schumacher M, Schmidt D, Jurklies B, et al. Central retinal artery occlusion: local intra-arterial fibrinolysis versus conservative treatment, a multicenter randomized trial. *Ophthalmology*. 2010;117:1367–1375. e1.
8. Xian Y, Federspiel JJ, Grau-Sepulveda M, et al. Risks and benefits associated with prestroke antiplatelet therapy among patients with acute ischemic stroke treated with intravenous tissue plasminogen activator. *JAMA Neurol*. 2016;73:50–59.