Transient Orbital Infarction Syndrome in an Otherwise Healthy Male

Collin Anderson MD MS, Isaac Kim MD, Patrick Oellers MD,
Robert Hill MD, Justin Dexter MD

Introduction

- Orbital ischemia may result from thromboembolism, coagulopathy, trauma, surgery, compression, or vasculitis[1]
- Central retinal and/or ophthalmic artery involvement may produce blindness, and ophthalmoplegia may occur from blockage of extraocular muscle arterial supply[1]
- Ophthalmic artery occlusion alone is not reported to induce global orbital ischemia, likely due to rich anastomoses between branches of ophthalmic and external carotid arteries[1]
- Global orbital infraction is extremely rare, previously reported in select few case reports[1,2,3]

Case

- 42-year-old otherwise healthy male presents with sudden onset, painful vision loss and near total ophthalmoplegia in left eye
- OS: VA HM, +APD, IOP 15, EOMs -3 all directions, mild proptosis compared to OD
- DFE notable for diffuse macular and fundus pallor, normal anterior segment exam and nerve
- Neuroimaging: non-con CT head + MRI orbit w/wo read OS>OD proptosis with “mild prominence of bilateral EOMs sparing tendons,” CTA head/neck + MRA head/neck all negative
- Broad differential considered: ophthalmic artery occlusion, nonspecific orbital inflammation, IgG4, SLE, GPA, orbital apex syndrome, superior orbital fissure syndrome, Tolosa Hunt, TED, syphilis, sarcoid, TB, Lyme
- Positive labs: ANA screen, anti-ds DNA, p-ANCA
- Negative labs: ESR, CRP, Quant gold, Lyme, Syphilis IgG/IgM, ACE, IgG subpanel

Figures

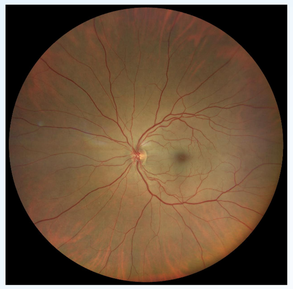

Figure 1: Color fundus photo on presentation, left eye. Diffuse macular pallor.

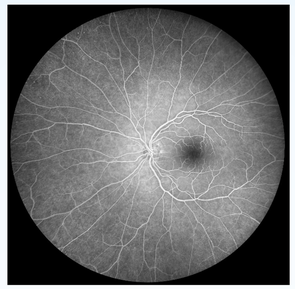

Figure 2: IVFA on presentation, left eye. Peak phase ~30 seconds. No clear delay in retinal arterial filling, unlike CRAO.

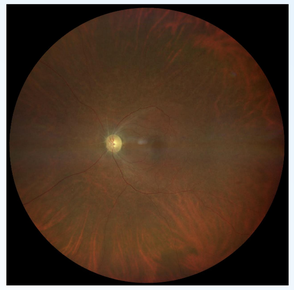

Figure 3: Color fundus photos at 1 month, left eye. Notable nerve pallor, vascular attenuation and peripapillary traction.

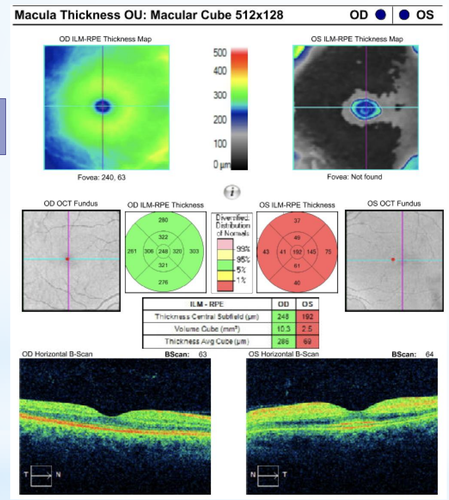

Figure 4: OCT Mac on presentation, central foveal thickness decreased OS>OD.

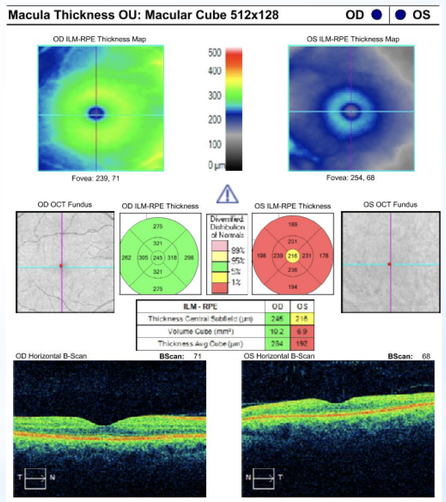

Figure 5: OCT Mac 1 month after onset, more prominent inner retinal thinning OS.

Case continued

- Testing: initial fundus photos show macular pallor (Figure 1), no clear filling defect on IVFA (Figure 2). One month later, fundus exam demonstrates increased nerve pallor, more attenuated vasculature, and peripapillary retinal traction (Figure 3).
- Initial OCT Mac shows central foveal thinning OS (Figure 4), and at 1 month with shows progressed inner retinal atrophy consistent with possible retinal ischemic event (Figure 5).
- Started on PO prednisone and Valtrex, referred to heme/onc for hypercoagulability workup and rheumatology
- Follow-up at 1 month: OS VA improved to 20/60, persistent APD, EOMs now full

Conclusion

- Orbital infarction syndrome is a rare phenomenon, necessitating a thorough workup and broad differential to determine etiology and patient risk factors

References

1. Borruat FX, Bogousslavsky J, Uffer S, Klainguti G, Schatz NJ. Orbital infarction syndrome. *Ophthalmology*. 1993;100(4):562-568. doi:10.1016/s0161-6420(93)31606-4
2. Vergez, A. Syndrome oculaire rare ua cours d'une thrombose carotidienne spontanée (thrombose de l'artère ophtalmique) [A rare ocular syndrome in spontaneous carotid artery thrombosis (thrombosis of ophthalmic artery)]. *Ann Ocul (Paris)*. 1959;192(5):376-384.
3. Bogousslavsky J, Pedrazzi PL, Borruat FX, Regli F. Isolated complete orbital infarction: a common carotid artery occlusion syndrome. *Eur Neurol.* 1991;31(2):72-76. doi:10.1159/000116650

This work was funded in part by an unrestricted grant from Research to Prevent Blindness, Inc. New York, New York and by the Lions District 20-Y1, Syracuse, New York. No other significant financial interests or relationships to disclose.

SUPPORTED BY
Research to Prevent Blindness