

MOGAD Masquerading as Mucormycosis

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Background

Orbital apex syndrome (OAS) is comprised of a unique set of symptoms derived from the affected structures passing through the apex, namely cranial nerves II, III, IV, V₁, and VI [1].

Lesions here present with vision loss, ophthalmoplegia or paresis, elevated intraocular pressure, and often with additional signs of increased pressure i.e. chemosis, proptosis, periorbital swelling etc.

Typical culprits include infectious, inflammatory, neoplastic, traumatic, and vascular processes [1].

OAS due to a demyelinating process is exceedingly rare with only 2 previously documented cases [2].

Here we present a case of OAS in a young uncontrolled diabetic in which the leading differential was mucormycosis infection versus an autoimmune demyelinating condition

Clinical Presentation

30-year-old female presented with what was initially bilateral eye pain that then lateralized to the right eye. She reported concomitant right sided blurry vision that progressed to complete vision loss with accompany periorbital swelling and tenderness. Prior to arriving to our facility, the patient was believed to be suffering from periorbital cellulitis.

Neurological examination revealed right sided proptosis, near-complete ophthalmoplegia (Figure 1), and diminished visual acuity with no light perception and relative afferent pupillary defect. Previously noted elevated intraocular pressure at outside facility had normalized on arrival.

Leading differential included a Mucormycosis rhinocerebral infection in the setting of newly diagnosed uncontrolled diabetes mellitus vs an autoimmune demyelinating or inflammatory process. The presented dilemma being that the latter would respond to high dose steroids vs the former would acutely worsen.

She was started on a 5-day course of high dose IV Solu Medrol and amphotericin, with subsequent discontinuation of the antifungal. Upon completion of steroid course as well as a 5 day course of intravenous immunoglobulin (IVIG), the patient had full resolution of her vision loss and ophthalmoplegia.

Imaging/Examination

Figure 1: Testing of ocular motility revealed impaired abduction, supraduction, adduction, and infraduction respectively. Note also periorbital edema and slight proptosis on right.

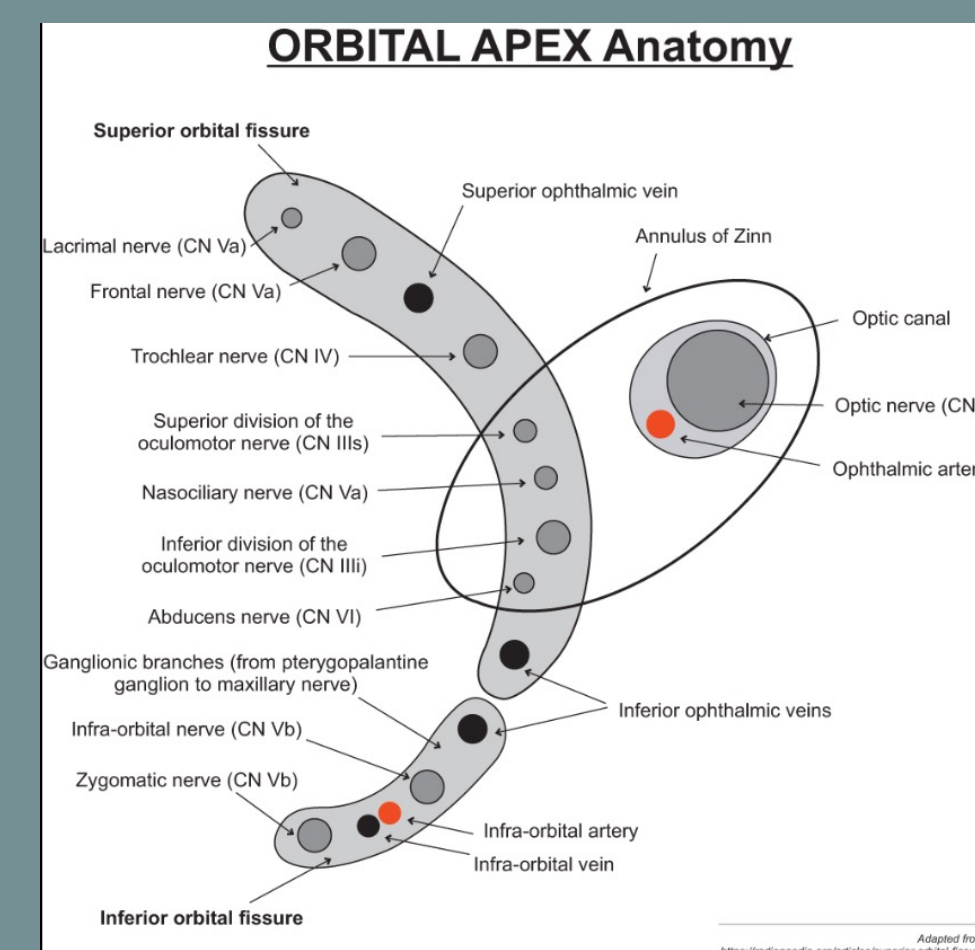


Figure 2: initial MRI orbits revealing fat stranding with post contrast enhancement of the perineural sheath bilaterally, right > left.

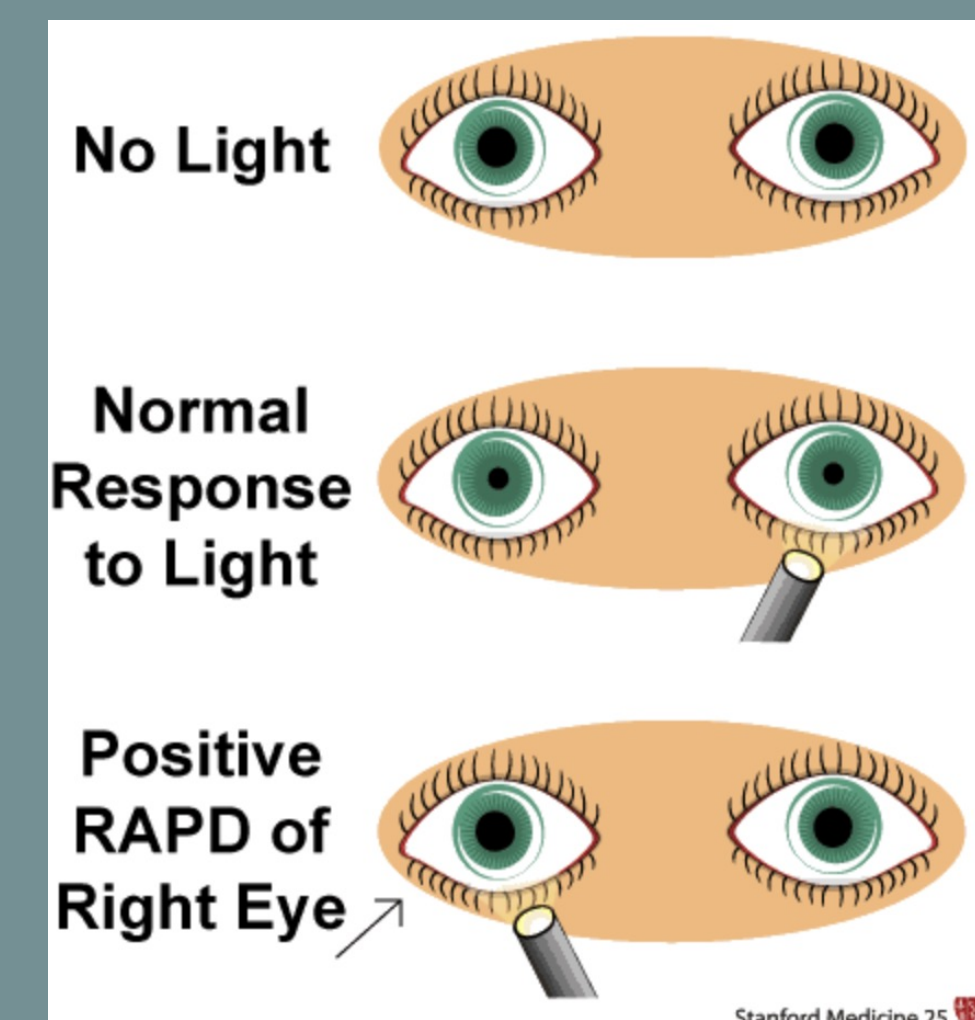
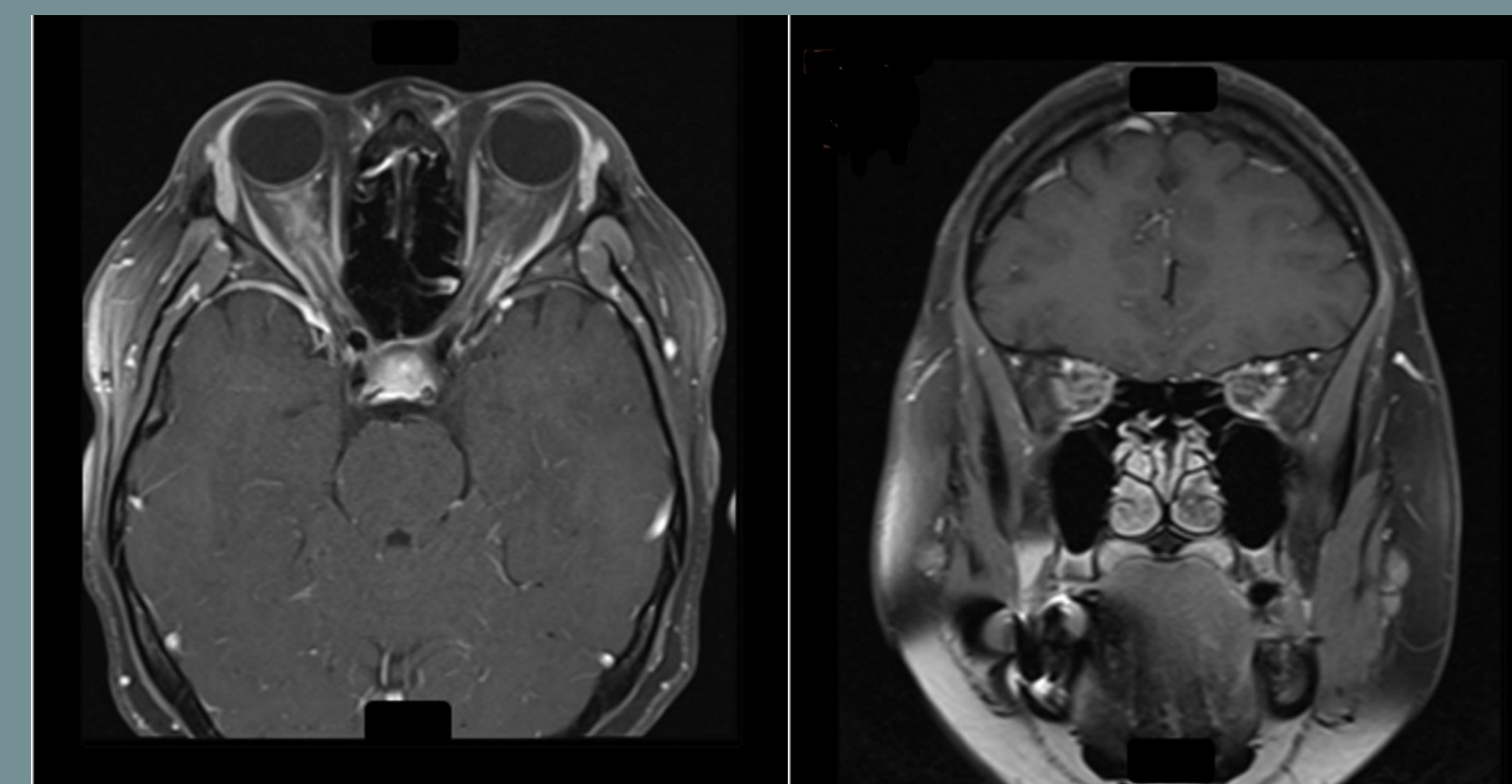
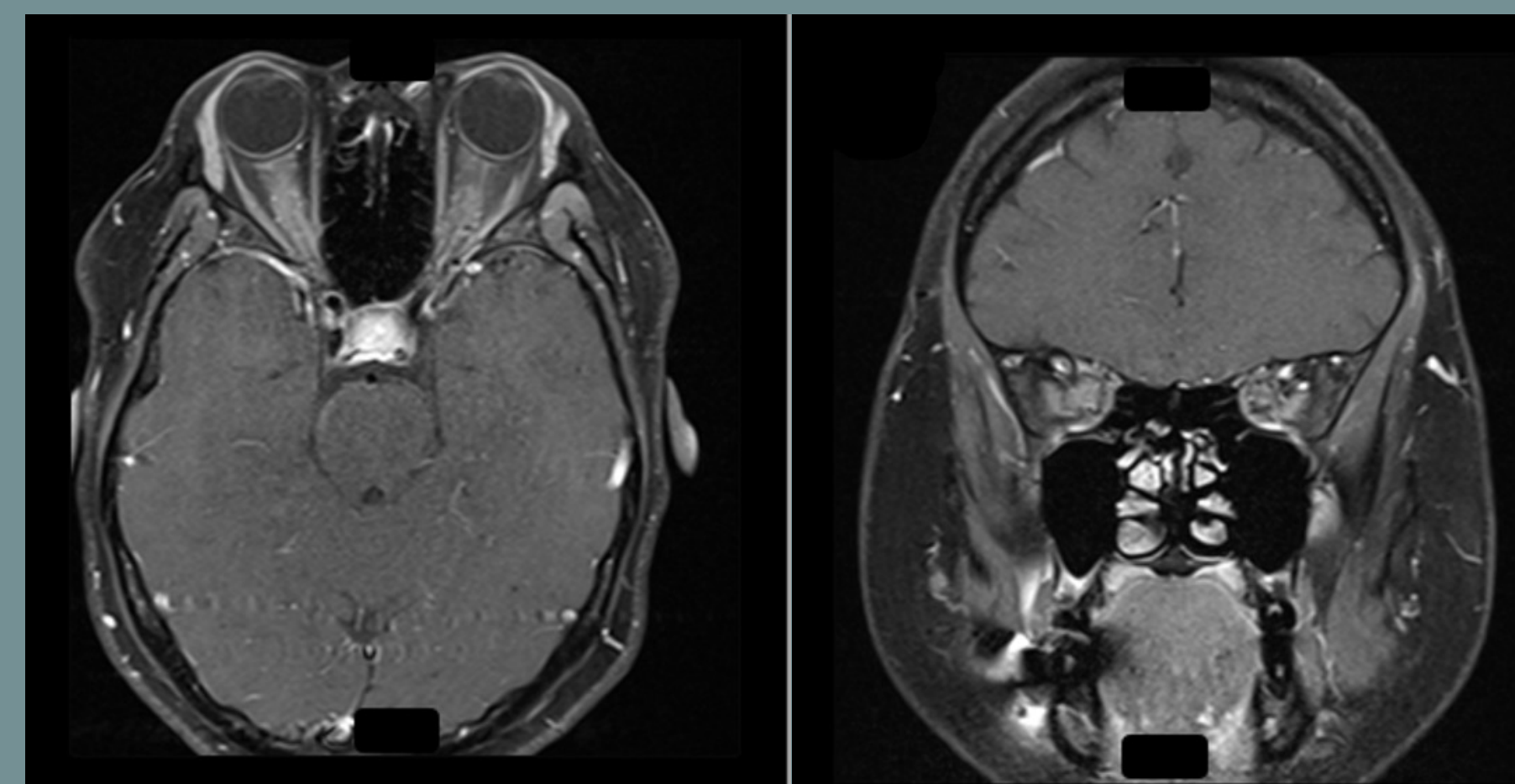


Figure 3: Repeat MRI orbits 4 days after the above revealing progression of optic nerve enhancement right > left with notable extension to involve the orbital apex.



Results

Magnetic Resonance Imaging (MRI) of the orbits done at an outside facility revealed findings consistent with optic perineuritis bilaterally, right worse than left. Repeat MRI done at our facility 4 days later revealed progression with optic nerve enhancement extending to the orbital apex on the right (Figure 2).

Lumbar puncture with a normal cell composition and opening pressure. Cerebrospinal fluid (CSF) studies largely unremarkable beyond elevated myelin basic protein indicating likely breakdown of myelin.

Infectious, autoimmune, and inflammatory panels returned without abnormalities except for a positive serum IgG against myelin oligodendrocyte glycoprotein (MOG).

Conclusion

MOG associated disease (MOGAD) is typically considered in patients with characteristic optic neuritis, longitudinally extensive transverse myelitis (LETM), and acute disseminated encephalomyelitis (ADEM).

In recent years however, numerous novel presentations have been reported including cortical encephalomyelitis, imaging-negative myelitis, and various ophthalmological presentations [2,3].

The aim of this presentation is to broaden the spectrum of cases in which MOGAD is considered while illuminating potential mimics and red herrings as was the case in this patient.

References

