



Bitemporal hemianopsia secondary to ischemic chiasmopathy following mechanical thrombectomy

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ABSTRACT

Purpose: To report a case of bitemporal hemianopsia due to ischemic chiasmopathy after mechanical thrombectomy of a right distal internal carotid artery (ICA) occlusion.

Observations: A 60-year-old female presented with left sided weakness and difficulty speaking and was found to have suffered a right internal carotid artery occlusion 10 days after tricuspid valve replacement for severe symptomatic tricuspid valve disease. She underwent mechanical thrombectomy and in hospital and at further follow ups was noted to have a bitemporal hemianopsia, consistent with an ischemic optic chiasmopathy.

Conclusions and importance: The optic chiasm is vascularized by multiple arteries of the Circle of Willis. As such, ischemic optic chiasmopathy is rare. Clinicians should consider ischemic chiasmopathy following cardiac and other surgical procedures including mechanical thrombectomy of the ICA or its branches.

1. Introduction

A bitemporal hemianopic visual field defect typically indicates a lesion of the optic chiasm due to involvement of the crossing nasal fibers in both eyes. The most common causes of a bitemporal hemianopsia are compressive lesions (e.g., pituitary adenoma) but ischemic etiologies have been reported previously as a rare cause of chiasmopathy. We report a case of presumed ischemic chiasmopathy after mechanical thrombectomy of the internal carotid artery. To our knowledge, this is the first such case to be described in the English language, ophthalmic literature.

2. Case report

A 60-year-old female with severe, symptomatic tricuspid valve disease underwent surgical tricuspid valve replacement (TVR). Ten days

post-operatively, she presented with acute left-sided hemiplegia, dysarthria, right gaze preference, and a left facial droop. Past medical history included idiopathic hypertrophic subaortic stenosis (IHSS) for which she had undergone pacemaker placement. Following the TVR, she developed atrial fibrillation requiring anticoagulation with apixaban.

Computerized tomography (CT) and CT angiogram (CTA) of the head revealed a right-sided distal internal carotid artery (ICA) occlusion and acute right middle cerebral artery (MCA) territorial infarction involving the insula and temporal lobe. She did not receive intravenous thrombolysis as she was anticoagulated with apixaban. Mechanical thrombectomy was performed with stent retriever and suction. Fig. 1 illustrates the presence of an occlusion of the right cavernous segment of the ICA. There was an anomalous origin to the right MCA with no proper M1 segment, and the anterior and posterior divisions of the MCA originating separately from the bifurcation. Fig. 2 shows complete revascularization of the right ICA following thrombectomy. One day after

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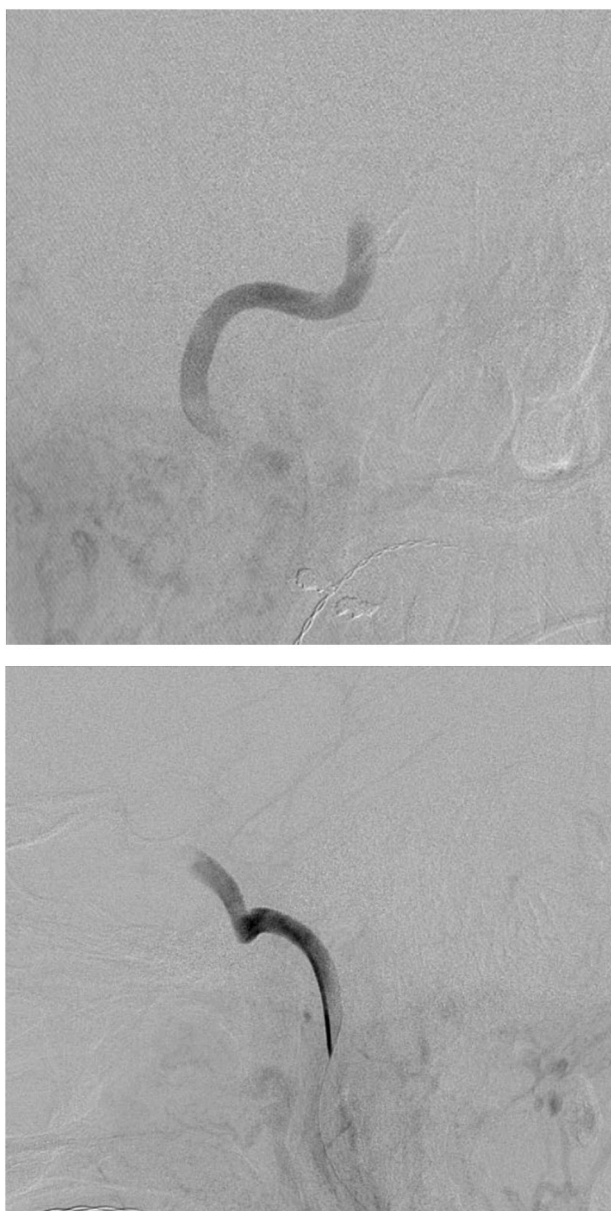


Fig. 1. Anterior to Posterior (AP) and lateral projection from arteriogram (A, B) show occlusion of the internal carotid artery (ICA) in the cavernous segment.

thrombectomy, the patient reported ptosis of the right eye (OD), diplopia, and loss of peripheral vision in both eyes (OU).

On examination at the bedside, near vision was 20/60 OU. There was no anisocoria or relative afferent pupillary defect (RAPD). There was a mild ptosis (1 mm) OD and a mild abduction and supraduction limitation OD. Anterior segment examination showed mild conjunctival chemosis OD and dilated fundus examination was normal OU. A CT scan of the orbit with contrast was unremarkable. CT head demonstrated no change in the prior MCA infarct, specifically, no chiasmal lesion was identified. Magnetic resonance imaging (MRI) could not be obtained due to patient's non-MRI compatible cardiac pacemaker.

Eight months after discharge from the hospital, the patient was seen in the Houston Methodist Hospital neuro-ophthalmology clinic. The ptosis OD, ophthalmoplegia, and diplopia had resolved spontaneously.



Fig. 2. AP and Lateral views (A,B) show complete revascularization following thrombectomy. The M1 segment is anomalous with early bifurcation from the carotid terminus.

The bitemporal visual field loss OU remained unchanged. On examination, her best corrected visual acuity was 20/25 OD and 20/40 in the left eye (OS). Color vision tested with Ishihara plates was 14/14 OU. The pupils were isocoric with no RAPD. The extraocular motility exam, intraocular pressure, and slit lamp biomicroscopy were normal OU. Automated perimetry (Humphrey visual field 24-2), [Fig. 3](#), demonstrated an incomplete bitemporal hemianopsia, denser inferiorly. There was no evidence of a left homonymous hemianopsia due to the prior

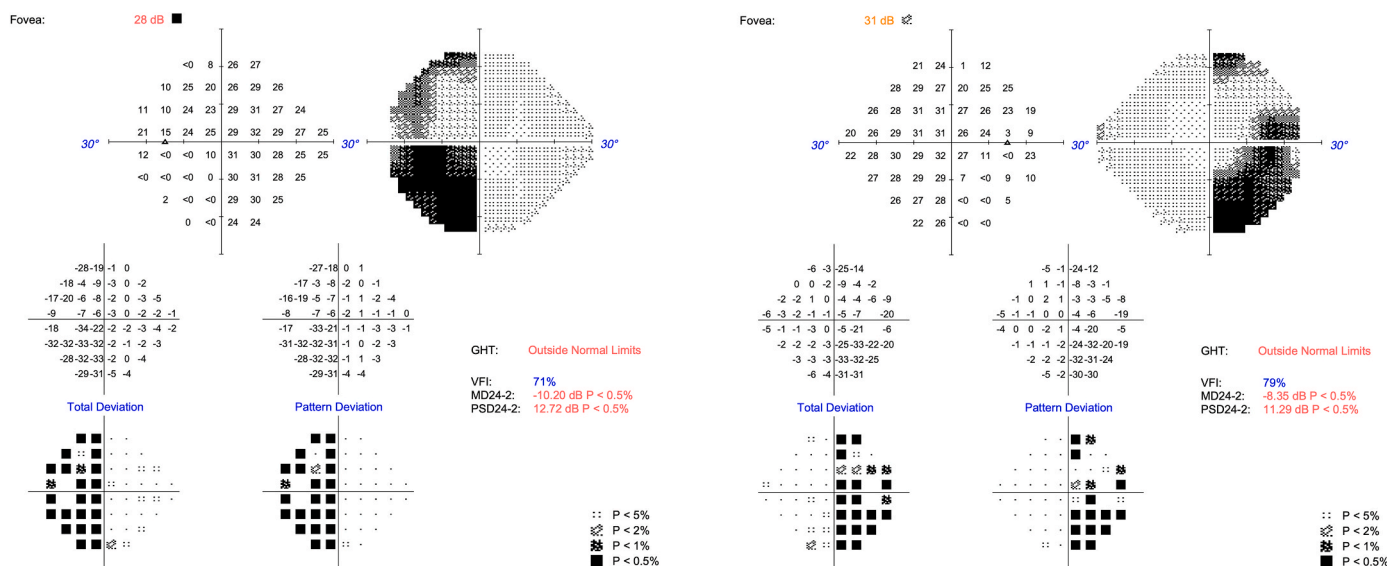


Fig. 3. Automated perimetry (Humphrey visual field 24-2) demonstrates bitemporal hemianopsia denser inferiorly.

right MCA infarction. Optical coherence tomography (OCT) and optic nerve head photos revealed nasal and temporal atrophy (“band atrophy”) OD greater than OS consistent with a chiasmal etiology (Fig. 4A and 4B). OCT of the macular ganglion cell layer also demonstrated loss in the papillomacular bundle corresponds with a chiasmal lesion (Fig. 4C). Repeat CT imaging of the sella showed no chiasmal or pituitary lesion. There was no evidence for suprasellar hemorrhage or chiasmal or pituitary apoplexy. No demyelinating lesions were seen on neuroimaging to suggest multiple sclerosis (MS). Serum antibody tests for syphilis, tuberculosis, aquaporin 4 in neuromyelitis optica (NMO) and myelin oligodendrocytic glycoprotein (MOG) were negative. A presumptive diagnosis of bitemporal hemianopsia following ischemic chiasmopathy following mechanical thrombectomy of the right ICA was made. Serial follow up of the patient has shown no change over time.

3. Materials and methods

Approval from the Institutional Review Board (IRB) was not required, however informed consent was obtained from the patient, and this report is in accordance with HIPAA regulations. The patient consented to publication of the case in writing.

4. Discussion

A bitemporal hemianopsia is typically due to a compressive lesion at the optic chiasm. The most common acquired chiasmal lesions in adults are neoplastic (e.g., pituitary adenoma, craniopharyngioma, or meningioma). However, the differential diagnosis includes vascular (e.g., parasellar ICA aneurysm), infectious (e.g., syphilis, tuberculosis, sellar abscess), demyelinating (e.g., NMO, MOG, MS), inflammatory (e.g., systemic lupus erythematosus, sarcoidosis), traumatic, and iatrogenic (e.g. post surgical). Rarely some medications have been reported to cause chiasmopathy (e.g., ribavirin, interferon, ethambutol, and isoniazid).¹⁻⁸ Ischemic chiasmal syndrome has been described in dolichoectatic tortuous parasellar arteries (e.g., ICA) and other ischemic mechanisms include atheromatous plaque formation occluding the chiasmal vascular supply, arachnoiditis with fibrosis, arteritis, and postpartum necrosis.⁹

The chiasm receives its blood supply from the arteries of the circle of Willis. The lateral surface of the chiasm is supplied by branches of the ICA and anterior cerebral arteries (ACA) The superior hypophyseal arteries, which originate from the ICA (at the level of the ophthalmic

artery), supply the inferomedial aspect of the chiasm. Superiorly, the chiasm is supplied by branches of the ICA, the small superior chiasmal arteries, the first segment of the ACA, superior hypophyseal artery, and anterior communicating artery.¹⁰⁻¹² The denser inferiorly, bitemporal visual field defect in our patient localizes to the superior portion of the optic chiasm, an area supplied by the superior hypophyseal artery, a branch of the ICA.

Embolic chiasmal infarction has been described previously, following open heart surgery, secondary to atrial fibrillation, and also following treatment of an ophthalmic artery aneurysm, leading to occlusion of the superior hypophyseal artery.¹³⁻¹⁵ Our patient presented with bitemporal hemianopsia following successful mechanical thrombectomy with stent retriever. We hypothesize that the bitemporal hemianopsia was secondary to ischemia of the chiasm due to occlusion of multiple feeding arterial branches, including the superior hypophyseal artery. The ophthalmoplegia and ptosis in this case also suggests the possibility of transient ischemia involving the cavernous sinus from branches of the internal carotid artery. The endovascular approach for the mechanical thrombectomy was via the right ICA and a thrombus in that location could also have produced the right MCA infarct in addition to the presumed superior hypophyseal artery ischemia to the optic chiasm. Embolization in a new (previously unaffected) territory is a known complication of thrombectomy and there have been reports of occlusion of the ophthalmic artery, internal maxillary and superficial temporal artery leading to orbital infarction syndrome. A second hypothesis is that the initial embolic stroke, and not the thrombectomy, was the cause of the ischemic chiasmopathy – the patient only reported symptoms 1 day after her mechanical thrombectomy, however in the instance of more severe neurological deficits such as hemiplegia, as well as dysarthria limiting her communication, her bitemporal hemianopsia may not have been initially identified. As part of the initial stroke workup, the patient underwent a National Institute of Health Stroke Scale (NIHSS) assessment, which includes brief assessment of visual fields, and no field defect was noted, although it is possible it was not identified in the setting of her dysarthria and her gaze preference.

We recognize that the lack of MR imaging in our case (due to non-MR compatible pacemaker) is a limitation of our report. Future cases may benefit from MRI to further characterize any acute or chronic structural changes in the chiasm after ischemia.

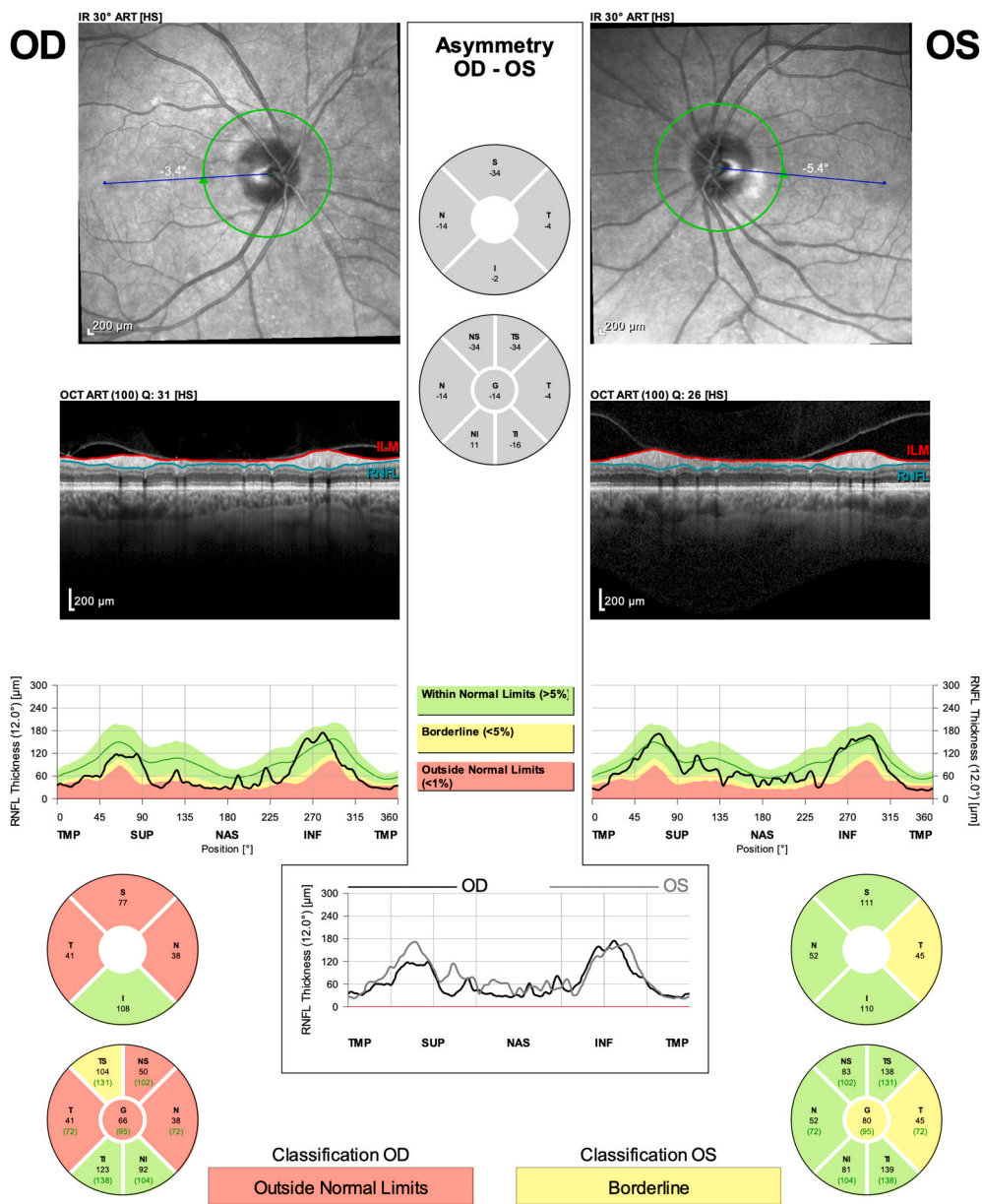


Fig. 4A. Optical Coherence Tomography (OCT) of the Retinal Nerve Fiber Layer (RNFL) demonstrating band atrophy in the right greater than the left eye.

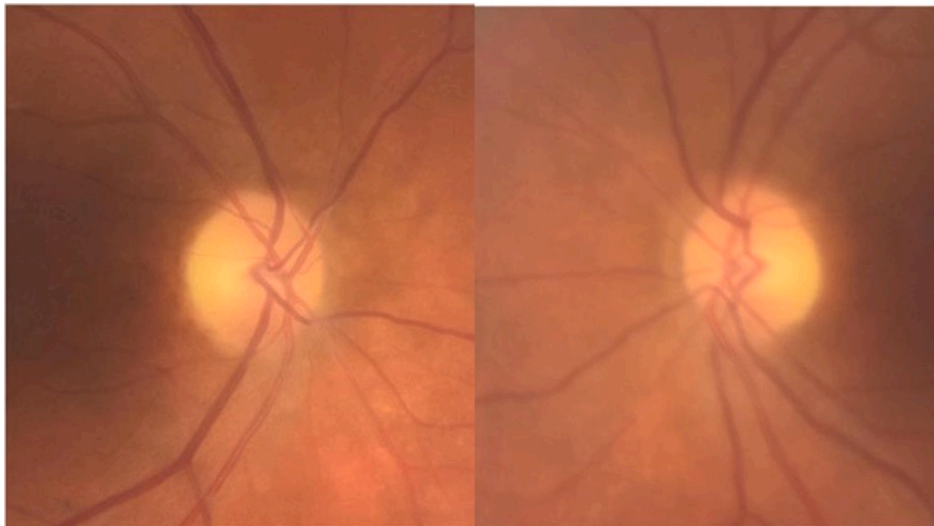


Fig. 4B. Optic nerve head photos illustrating nasal fiber loss and secondary pallor in both eyes, suggestive of band atrophy.

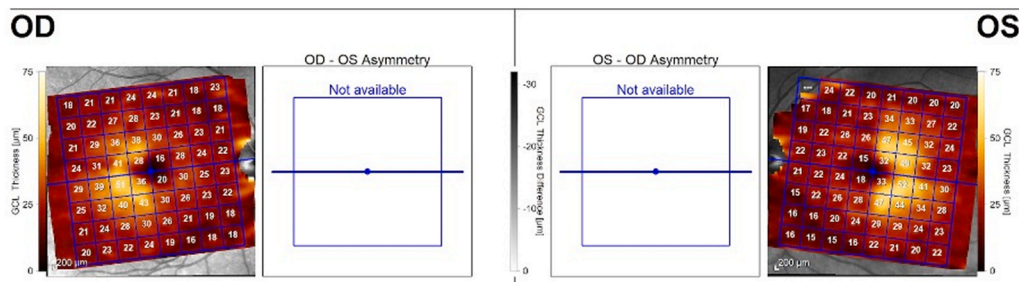


Fig. 4C. Optical Coherence Tomography (OCT) of the Macular Ganglion Cell Layer demonstrating nasal thinning in both eyes.

5. Conclusions

Ischemic chiasmopathy is a rare cause of bitemporal hemianopsia but clinicians should be aware of this possibility following cardiac and other surgical procedures including mechanical thrombectomy of the ICA or its branches. Neuroimaging should be directed to the optic chiasm to exclude an acute compressive lesion (e.g., pituitary apoplexy). To our knowledge, this is the first such case to be reported in the English language, ophthalmic literature.

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CRediT authorship contribution statement

Rohini Rao Sigireddi: Conceptualization, Writing – original draft, Writing – review & editing. **Nita Bhat:** Conceptualization, Writing – original draft, Writing – review & editing. **Subahari Raviskanthan:** Conceptualization, Writing – original draft, Writing – review & editing. **Peter William Mortensen:** Conceptualization, Writing – original draft, Writing – review & editing. **Shruthi Harish Bindiganavile:** Conceptualization, Writing – original draft, Writing – review & editing. **Richard Klucznik:** Conceptualization, Writing – original draft, Writing – review & editing. **Andrew Go Lee:** Conceptualization, Writing – original draft, Writing – review & editing.

Declaration of competing interest

None.

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