

Anterior segment ischemia following three muscle strabismus surgery in a young adult with prior carotid artery injury

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Abstract

Anterior segment ischemia (ASI) is a rare but vision-threatening complication of strabismus surgery. We present a case of ASI following three muscle strabismus surgery in a young, healthy woman with a history of mild left carotid artery injury secondary to head trauma. Three weeks after left medial rectus recession and total transposition of the left superior and inferior recti to the left lateral rectus to treat a traumatic left sixth nerve palsy, she had left pupil peaking and inferonasal iris atrophy. She had no change in her visual acuity and no anterior segment inflammation. Iris fluorescein angiography demonstrated delayed filling and neovascularization of the superotemporal iris. She was diagnosed with ASI and treated with prednisolone acetate 1% four times a day in the left eye. Her exam remained stable over the next month. To our knowledge, this is the first report of ASI after three muscle strabismus surgery in a young, healthy patient with prior mild unilateral carotid injury.

History

A 27-year-old woman presented to a pediatric ophthalmologist complaining of constant diplopia eight months after suffering traumatic brain injury (TBI) from being struck by a vehicle while riding a bicycle. Relevant injuries included basilar skull fracture, subdural hematoma, and brainstem and subarachnoid hemorrhages. MRA of the head and neck following her accident showed luminal narrowing and irregularity of the left carotid artery at the proximal petrous and petrous-cavernous junction.

Examination

Her visual acuity was 20/100 in the right eye and 20/70 in the left. Her intraocular pressure was 10 in both eyes. She has no afferent pupillary defect. She had incomplete blink in the right eye. The inferior 1/3 of the right cornea had an irregular surface with neovascularization. Her right optic disc had mild pallor. Her slit lamp exam and dilated fundus exam were otherwise normal. Sensorimotor exam showed limited abduction of both eyes as well as limited supraduction, adduction, and infraduction of the right eye. She had an 85 prism diopter esotropia in primary gaze and near and a 35 prism diopter left hypertropia. She had increased adduction on attempted elevation and depression of the right eye. She was diagnosed with esotropia and right hypotropia from traumatic bilateral 6th nerve palsy and right 3rd nerve palsy with aberrant regeneration of the right superior rectus to the right medial rectus. Surgery to center the left eye was planned, followed later by surgery to align her right eye.

Surgical course

Utilizing three fornix incisions to preserve the limbal conjunctival circulation, she underwent 6.5mm left medial rectus recession on adjustable suture and total transposition of the left superior rectus and inferior rectus to the left lateral rectus, with a 3mm resection of both vertical recti. In the first week postoperatively, she complained of refractory left retrobulbar pain. On her first visit three weeks after surgery, she had superotemporal left pupil peaking and inferonasal iris atrophy (**figure 1**). Her best corrected visual acuity was stable and she had no anterior chamber cell or flare. She had residual esotropia and left hypertropia in primary gaze, as expected. Iris fluorescein angiography (FA) demonstrated delayed filling and neovascularization of the superotemporal iris (**figure 2**). She was diagnosed with anterior segment ischemia (ASI) and started on prednisolone acetate 1% four times a day in the left eye. Her exam remained stable over the next month.

Four months after left eye surgery, she underwent total transposition of right superior rectus to lateral rectus with 3mm resection to correct the left hypotropia and total transposition of the right inferior rectus to the lateral rectus. Right medial rectus recession was avoided to decrease the risk of ASI. She had no evidence of ASI after this procedure.

Discussion and diagnosis

ASI is a rare but vision threatening complication of strabismus surgery. ASI occurs in approximately 1 out of 13,000 strabismus operations.¹ ASI commonly presents with pupillary changes and anterior chamber cell and flare and can rarely lead to phthisis bulbi.²

Patient-related risk factors for ASI include age, compromised anterior segment circulation, hematologic disorders, atherosclerosis, and thyroid eye disease.² In this otherwise healthy young woman, carotid artery injury was the only patient risk factor. She suffered left carotid artery injury fourteen months before her initial left eye strabismus surgery, but she showed no clinical signs of left carotid insufficiency.

Greater numbers of muscles operated on are a risk factor for ASI. Simultaneous recession of 2-3 recti muscles in primates occasionally produced transient ASI, while simultaneous recession of all four recti muscles produced serious, permanent ASI.³ In adult humans, there is an additive effect on anterior segment circulation disruption when more than one rectus muscle is disrupted.⁴ Fornix incisions may decrease, but do not eliminate, the risk of ASI.^{2,5}

Vertical rectus muscle disruption impacts anterior segment circulation more than horizontal rectus surgery. In one study, all 17 adults who underwent primary vertical rectus surgery had angiographic ASI on postoperative day 1, and six had signs of ASI on slit lamp exam; in contrast, there were no angiographic or clinical signs of ischemia in the 15 patients who underwent secondary vertical rectus or primary or secondary horizontal rectus surgery.⁶ A study of anterior segment indocyanine green angiography (ICGA) after strabismus surgery found filling delays in all primary vertical recti surgeries but no delays after primary horizontal recti surgery.⁷

We confirmed the diagnosis of ASI using iris FA. Iris vasculature can be visualized using FA, ICGA, or OCT-A.^{7,8} Angiographic abnormalities often improve or resolve weeks to months after surgery,^{6,7} but they can persist years later.

Topical steroids are a common treatment for ASI, but it is unknown whether treatment changes the natural history.² Clinical signs typically resolve over time. Occasionally iris atrophy and pupil changes persist.⁹ Final visual acuity is typically unchanged from preoperative visual acuity.¹

To our knowledge, this is the first report of ASI in a young, healthy patient with prior unilateral carotid injury undergoing three muscle strabismus surgery. ASI following strabismus surgery was reported in a young man with prior left internal carotid artery ligation.¹⁰ He underwent left medial rectus recession and left lateral rectus resection for a 6th nerve palsy followed nine years later by lateral transposition of the vertical recti. He developed ASI after the transposition. Another healthy young man with abducens palsy following TBI developed severe ASI after medial rectus recession combined with temporal transposition of both vertical recti.⁹ That patient had no history of carotid disruption, but given his lack of other risk factors, it is possible that his trauma caused undiagnosed carotid injury. Our case demonstrates that ASI can occur in a healthy patient with mild carotid disease after three muscle surgery.

This case highlights the importance of obtaining complete records following traumas to identify ASI risk factors preoperatively. It is possible that preoperative iris angiography or OCT-A could have prompted modifications to the surgical plan.¹¹ The surgery could have been performed in a staged fashion, which may decrease the risk of ASI.² Alternatively, we could have preserved the anterior ciliary vessels² or considered ciliary sparing augmented adjustable transposition surgery¹² to reduce the likelihood of ischemia.

Conclusion

In conclusion, ASI can occur in a healthy, young patient with mild carotid injury undergoing three muscle strabismus surgery, even using fornix incisions. In patients with head trauma, anterior segment imaging may be helpful in guiding surgical planning.

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Figure 1: Left superotemporal pupil peaking and inferonasal iris atrophy three weeks after 3 muscle surgery on the left eye



Figure 2: Iris fluorescein angiography demonstrating delayed filling and neovascularization of the superotemporal iris three weeks after 3 muscle surgery on the left eye

