

was intentionally created at the lower part of the detachment pocket using a microvitrectomy blade passed through the paracentesis. Introducing a balanced salt solution and injecting air into the anterior chamber, the sub-Descemet hematoma and viscous fluid were washed out from the break in the Descemet membrane. A bimanual infusion-aspiration technique was also used to avoid expanding the detachment. Sulfur hexafluoride (SF₆) gas was then injected into the anterior chamber to restore the membrane.

One month after descemetopexy, the pupillary area was transparent (Figure 2). Postoperative best-corrected visual acuity was 0.6. Specular microscopy revealed a central endothelial cell count of 1,900 and 2,600 cells/mm² in the fellow eye. The intraocular pressure was 20 mm Hg without medication. The Descemet membrane remained attached at follow-up examination.

An intracorneal hematoma with the Descemet membrane detachment occurred as an unusual complication after viscocanalostomy. For our patient, hyaluronate 2.3% was selected as the viscoelastic material, which is reported to have an advantage over hyaluronate 1.4% for dilatation of the Schlemm canal.⁴ Our clinical impression is that lysis of the Descemet membrane may have occurred as a result of the high pressure during the injection of the hyaluronate. The postoperative blood reflux from the Schlemm canal may then have caused the large intracorneal hematoma with the Descemet membrane detachment. High-viscosity hyaluronate seems to require greater care to inject than does lower viscosity hyaluronate.

When a hematoma is sufficiently extensive to cover the pupillary area, it is best to perform a descemetopexy immediately. Hematoma are organized within a few days and thus become difficult to aspirate. Surgery for repair includes creation of a break in the Descemet membrane, alternating injections of a balanced salt solution and air into the anterior chamber to wash out the hematoma, followed by injection into anterior chamber of a long-lasting gas such as SF₆ to facilitate reattachment.

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Neodymium:yttrium-aluminum-garnet Laser Arteriotomy With Embolectomy for Central Retinal Artery Occlusion

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PURPOSE: To describe a patient with central retinal artery occlusion successfully treated with neodymium:yttrium-aluminum-garnet laser arteriotomy and embolectomy.

DESIGN: Interventional case report.

METHODS: A 77-year-old woman noted sudden loss of vision after vigorous coughing. A central retinal artery occlusion was diagnosed. Four hours after symptoms appeared, arteriotomy and embolectomy with neodymium:yttrium-aluminum-garnet laser was performed.

RESULTS: Displacement of embolus outside the artery with return of retinal perfusion and recovery of vision. Laser treatment also resulted in vitreous hemorrhage and false aneurysm formation of the central retinal artery.

CONCLUSIONS: Neodymium:yttrium-aluminum-garnet laser arteriotomy in a patient with central retinal artery occlusion resulted in extrusion of the embolus, reopening of the central retinal artery, and return of vision. This technique warrants further study as a primary treatment for this blinding disorder. (*Am J Ophthalmol* 2004;137:196-198. © 2004 by Elsevier Inc. All rights reserved.)

CENTRAL RETINAL ARTERY OCCLUSION IS A POTENTIALLY devastating visual disorder usually caused by closure of the vessel by atheroma or emboli. Sustained blockage of blood flow to the eye may lead to irreversible blindness and secondary ocular complications. Current treatment options for central retinal artery occlusion are usually ineffective and do not significantly influence the poor visual prognosis associated with this disorder.

To the best of our knowledge, this is the first report of successful neodymium:yttrium-aluminum-garnet (Nd:YAG) laser treatment of embolic central retinal artery occlusion.

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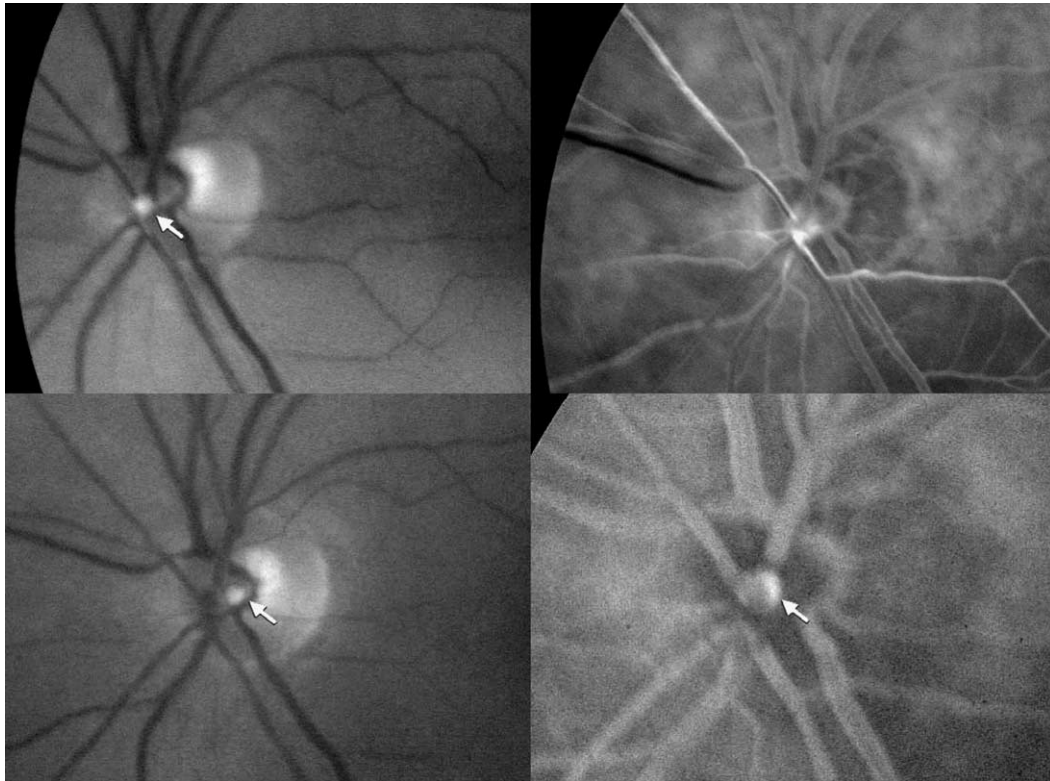


FIGURE 1. (Upper left) Embolus at bifurcation of central retinal artery. (Upper right) Fluorescein angiography demonstrates occlusion of the central retinal artery. (Lower left) Eighteen days following neodymium:yttrium-aluminum-garnet laser arteriotomy. Embolus has shifted temporally and is outside retinal vessel. (Lower right) Fluorescein angiography reveals perfused retinal vessels. Hyperfluorescence at treated area may represent a false aneurysm.

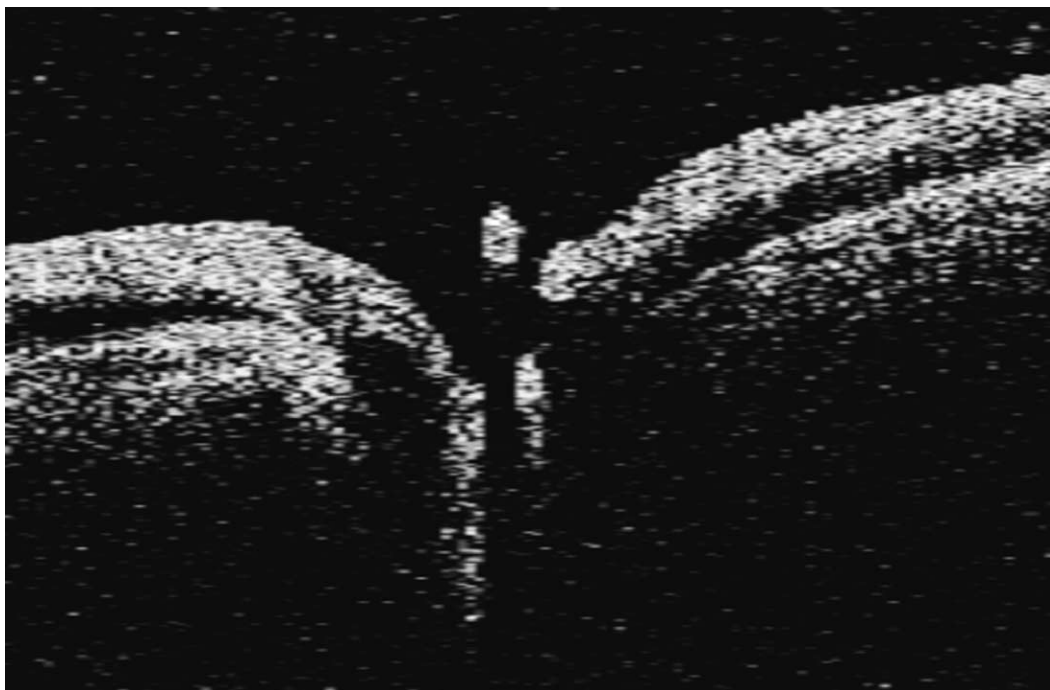


FIGURE 2. Ocular coherence tomography of the optic nerve head post treatment. Opacified structure located anterior to the optic nerve is consistent with the extruded embolus.

A 77-year-old woman noted sudden loss of vision in her left eye after vigorous coughing. Medical history was remarkable for a right carotid endarterectomy and resection of an abdominal aortic aneurysm. Ocular examination 2 months earlier revealed moderate cataracts and uncorrected visual acuities of 20/40 for each eye.

Within 2 hours of onset of symptoms, the visual acuity in the left eye was counting fingers. Examination of the left eye showed an afferent pupillary defect and extensive visual field loss. Ophthalmoscopy disclosed diffuse retinal whitening and a large refractile embolus within the central retinal artery (Figure 1). The clinical diagnosis was central retinal artery occlusion.

After receiving informed consent from the patient, treatment with three applications of a Zeiss Visulas Nd:YAG II laser (Carl Zeiss Ophthalmic Systems, Inc., Humphrey Division, Dublin, California, USA) was performed 4 hours after loss of vision. A Goldmann three-mirror contact lens was applied, with the laser in single-burst mode. The laser was focused slightly posterior to the visible arterial wall at the site of the embolus. The first two applications of 0.8 millijoule had no discernible effect. The third and final application at 1.1 millijoule appeared to move the embolus, and brisk hemorrhage from the central retinal artery into the vitreous was observed (Figure 2). Digital pressure applied to the globe resulted in cessation of bleeding. One hour later the visual acuity had improved to 20/100 in the treated eye.

The vitreous hemorrhage gradually reabsorbed, and 3 weeks after treatment the uncorrected vision in her left eye had improved to 20/40. The displaced embolus gradually dissolved within 3 months of treatment.

Our case study indicates that arteriotomy performed in a timely manner with Nd:YAG laser may favorably influence retinal blood flow and visual prognosis in patients with visible retinal emboli causing central retinal artery occlusion.

Prior attempts at laser treatment for retinal emboli have been met with mixed success. Dutton and Craig¹ and Ciulla and associates² reported failure of argon laser treatment for retinal emboli. Opremcak and Benner³ observed recovery of vision in a Nd:YAG laser-treated patient with branch retinal artery occlusion and another similarly treated patient with cilioretinal artery occlusion.

Neodymium:yttrium-aluminum-garnet laser treatment in our patient resulted in extrusion of the embolus through an arteriotomy and return of normal perfusion. Laser arteriotomy also caused a vitreous hemorrhage and "false aneurysm" formation. Although such lesions usually resolve, the prognosis of false aneurysms involving the central retinal artery is unknown. Further study is needed to confirm the efficacy and safety of laser arteriotomy in the treatment of embolic occlusion of the central retinal artery.

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Intraocular Pressure Profile of a Child on a Systemic Corticosteroid

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PURPOSE: To report the ocular hypertensive response to high-dose systemic corticosteroid in a pediatric patient.

DESIGN: Observational case report.

METHODS: A 9-year-old patient with leukemia received oral prednisolone at a dosage of 2.3 mg/kg/d for 5 weeks, followed by a 4-month break and then a 4-week course of oral dexamethasone at 10 mg/d. Detailed ocular examination was performed for both eyes before and regularly throughout the two courses of treatment.

RESULTS: The intraocular pressure in both eyes rose to almost 40 mm Hg after only 8 days of oral corticosteroid. On stopping systemic corticosteroid, the intraocular pressure rapidly returned to baseline level within 2 days. A similar intraocular pressure profile was recorded for both eyes during the course of oral dexamethasone. The patient remained largely asymptomatic throughout.

CONCLUSIONS: Systemic corticosteroid may give rise to significant but asymptomatic ocular hypertension in pediatric patients. (*Am J Ophthalmol* 2004;137:198-201. © 2004 by Elsevier Inc. All rights reserved.)

ONE OF THE MAJOR OPHTHALMIC COMPLICATIONS OF corticosteroid is ocular hypertension that may result in irreversible ocular damage. Our previous studies have shown that children have more frequent, more severe, and more rapid ocular hypertensive response to topical dexamethasone than adults.^{1,2} It is not known whether pediat-

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