



FIGURE 1. Left miosis and ptosis consistent with Horner's syndrome.

lights were turned off. Left pseudoenophthalmos was noted. After 10% cocaine was instilled into both eyes at 0 and 5 minutes, the right pupil dilated to 9 mm but the left only to 7.5 mm after 40 minutes. Because the postcocaine difference in anisocoria was greater than 1.0 mm, this was considered a positive test for Horner syndrome. On follow-up examination 2 weeks later, the Horner syndrome had improved but was still present (Figure 1). One week after that, the anisocoria and ptosis resolved.

Recognized causes of acquired Horner syndrome in children include spinal cord neoplasm, neuroblastoma, chest surgery, injury of brachial nerve roots or plexus, nasopharyngeal tumor, and carotid artery occlusion.<sup>3-5</sup> Our literature review disclosed no other cases of Horner syndrome after vagus nerve stimulator implant. The origin of Horner syndrome in this patient is likely transient dysfunction of third-order oculomotor fibers within the carotid sheath. Patients undergoing vagus nerve stimulator implant should be observed for Horner syndrome.

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## Serous Retinal Detachment and Cystoid Macular Edema in Hypotony Maculopathy

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**PURPOSE:** To report the foveal structural findings of hypotony maculopathy imaged with optical coherence tomography.

**METHODS:** Case report of a 39-year-old white woman with a 20 year history of uveitis, who underwent pars plana vitrectomy and pars plana Baerveldt implant for intractable glaucoma. Four months postoperatively, decreased vision and hypotony maculopathy ensued. Optical coherence tomographic (OCT) images were taken horizontally through the macula.

**RESULTS:** OCT demonstrated large intraretinal cysts, serous macular detachment, and choroidal folds, which resolved upon resolution of hypotony.

**CONCLUSION:** Cystic macular changes and serous macular detachment can be prominent features of hypotony maculopathy. (*Am J Ophthalmol* 2001;131:384-386. © 2001 by Elsevier Science Inc. All rights reserved.)

**HYPOTONY MACULOPATHY** WAS FIRST REPORTED BY Dellaporta in 1955.<sup>1</sup> The characteristic fundus findings include marked irregular stellate folding of the retina around the foveal center.<sup>2</sup> While Dellaporta theorized intraretinal edema as the cause,<sup>1</sup> Gass believed that thickening of the scleral wall and choroidal congestion caused central displacement of the normally thick retina surrounding the thin fovea, producing the characteristic central stellate retinal wrinkling. Thus, folds of the retina, retinal pigment epithelium and choroid were present, but cystoid macular edema was usually not present.<sup>3</sup> We report a case of hypotony maculopathy, in which cystic macular changes and serous macular detachment documented by optical coherence tomography (Humphrey Systems, Dublin, California) were prominent features.

• **CASE:** A 39-year-old white woman with a 20-year history of uveitis presented with increased intraocular pressure of the left eye as high as 48 mm Hg, despite maximal medical treatment. She had previous cataract surgery with posterior chamber lens implant. Visual acuity was counting fingers at 1 ft. Biomicroscopy revealed corneal epithelial and stromal edema, and 360° peripheral anterior synechiae, which prevented glaucoma seton placement in the anterior segment. The implant was in position without pupillary block. A pars plana vitrectomy was performed with placement of a pars plana Baerveldt glaucoma implant (Pharmacia and Upjohn Co, Kalamazoo, Missouri). Postoperatively, inflammation was minimal and controlled with topical anti-inflammatory medications. The intraocular pressure remained 3 mm Hg to 4 mm Hg. The macula

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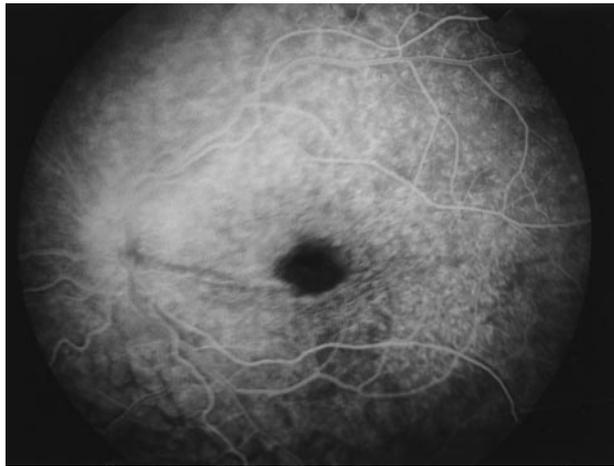


FIGURE 1. Arteriovenous phase of the fluorescein angiogram shows disk leakage and diffuse microvascular leakage in the macula and midperiphery.

remained flat without cystic changes for the first 4 postoperative months. After 4 months, large macular cysts, fine radiating macular striae, choroidal folds, disk swelling, and periarteriolar sheathing were observed. Fluorescein angiography showed disk leakage and retinal microvascular leakage in the macula and midperiphery (Figure 1). Optical coherence tomography demonstrated retinal thickening with large intraretinal cysts and serous macular detachment with an underlying choroidal fold (Figure 2, top). Because of persistent hypotony, the Baerveldt implant was removed 7 months after initial placement. Three weeks after removal of the implant, the intraocular pressure increased to 9 mm Hg. Improvement occurred in visual acuity to 20/400, retinal wrinkling and cystic changes decreased on fundus examination, and marked resolution of the cystic changes and serous detachment were noted on optical coherence tomography (Figure 2, bottom).

This case demonstrates that prominent features of hypotony maculopathy can be cystic spaces within the retina and serous retinal detachment. Cystoid macular edema occurs in varied clinical settings, including uveitis, postoperative macular edema after cataract surgery, and diabetic macular edema. Recently, similar intraretinal cystoid spaces and serous macular detachment have been documented in diabetic macular edema by optical coherence tomography.<sup>4</sup> The transudation of fluid in diabetic macular edema is primarily related to microvascular alterations. Inflammation also predisposes an eye to developing cystoid macular edema. This patient presented with an antecedent history of uveitis; yet, the preoperative and postoperative findings showed no evidence of recurrent inflammation. The cystic changes also did not develop until 4 months postoperatively.

Hypotony may also be a significant factor in resultant leakage from the perifoveal retinal vasculature. Hydrostatic pressure and osmotic pressure in both the retinal

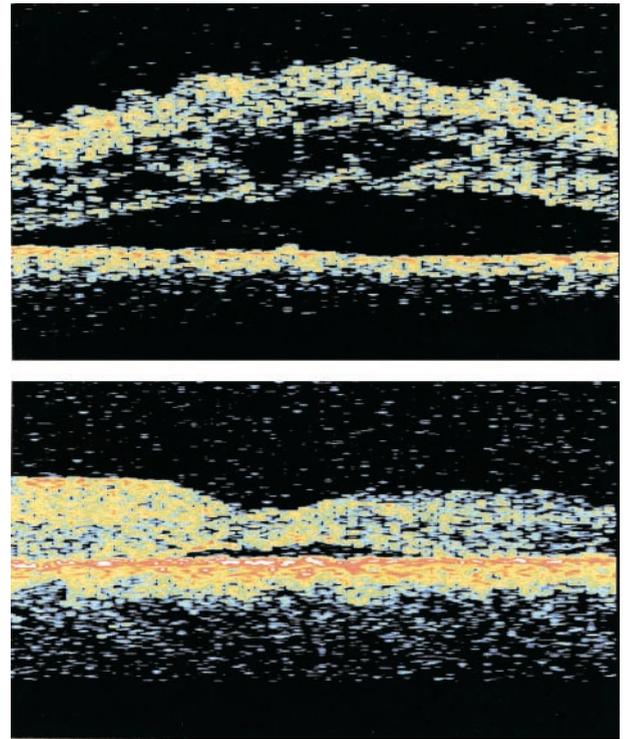


FIGURE 2. (Top) Optical coherence tomography demonstrates intraretinal cysts, serous macular detachment, retinal striae, and a choroidal fold. (Bottom) Resolution of cystic changes and marked decrease in serous fluid following increase in intraocular pressure.

capillaries and extracellular tissues determine the net fluid flow across fluid barriers, as presented in a modified concept of the hypothesis by Starling<sup>5</sup> -  $F = C \times [(P_{hc} - P_{ht}) + (P_{colt} - P_{colc})]$ , where  $C$  = constant,  $P_{hc}$  = mean hydrostatic pressure in capillary,  $P_{ht}$  = hydrostatic pressure in tissue fluid,  $P_{colt}$  = colloid osmotic pressure in tissue fluid, and  $P_{colc}$  = mean colloid osmotic pressure in capillary. Low intraocular pressure represents low tissue hydrostatic pressure, resulting in a higher hydrostatic pressure gradient across retinal capillaries, which promotes a net movement of fluid into the extracellular spaces. This may be exacerbated in eyes with preexisting microvascular disease, such as this case with uveitis. Restoring the intraocular pressure by removal of the shunt in this case resulted in prompt resolution of the cystic changes and serous macular detachment, showing that restoration of a more normal intraocular pressure and, thus, higher hydrostatic tissue pressure in the Starling law resulted in less outflow of fluid from retinal capillaries. Less fluid outflow allowed reabsorption of extracellular fluid in the retina and improvement in vision.

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## Tamoxifen Retinopathy in a Male Patient

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**PURPOSE:** To report a case of tamoxifen retinopathy in a male patient.

**METHODS:** Case report. A 68-year-old man, who had received a cumulative tamoxifen dose of 60 g over 33 months for unresectable hepatocellular carcinoma, was evaluated.

**RESULTS:** A peculiar, bilateral, symmetric, inner retinal crystalline deposition associated with mild macular edema was discovered. No other ocular toxicity of tamoxifen was observed.

**CONCLUSION:** To our knowledge, this is the first report of tamoxifen retinopathy in a male. (*Am J Ophthalmol* 2001;131:386–387. © 2001 by Elsevier Science Inc. All rights reserved.)

TAMOXIFEN, A NONSTEROIDAL ANTIESTROGEN, HAS been used most commonly in the treatment of estrogen receptor–positive tumors, such as breast and endometrial carcinoma. However, it has also been used at higher doses in the treatment of estrogen receptor–negative tumors, such as hepatocellular carcinoma.<sup>1</sup> Because of the antiestrogenic properties of tamoxifen, it has been used almost exclusively in women. Additionally, its ocular toxicity has only been reported in women. To the best of our knowledge, this is the first reported case of tamoxifen retinopathy in a man.

• **CASE:** A 68-year-old Egyptian, male physician was referred to our clinic for bilateral decrease in vision over the past 2.5 years. Cataract surgery with posterior chamber intraocular lens implantation had been performed without complications in each eye 9 (left eye) and 11 (right eye) months before

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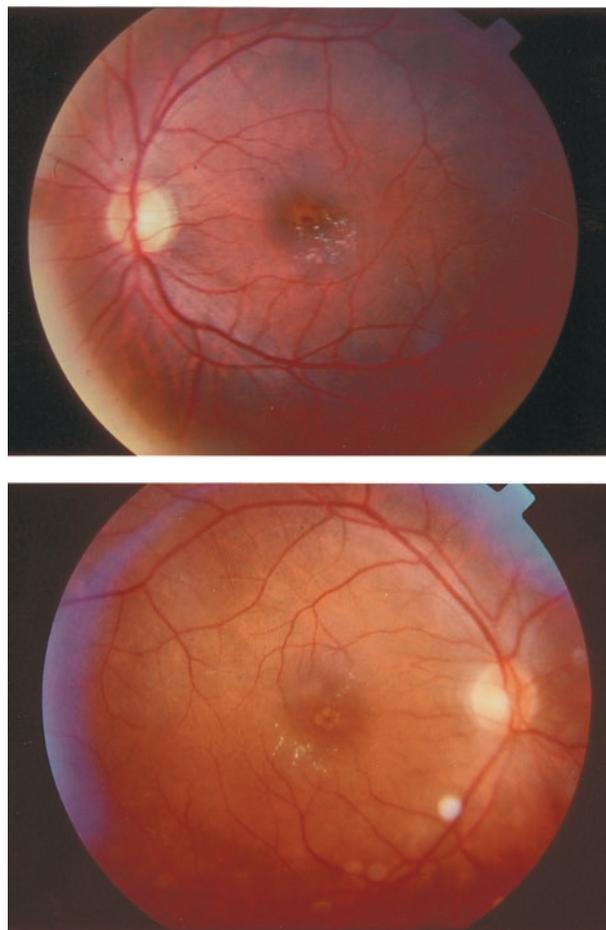


FIGURE 1. (Top and Bottom) Fundus photographs with inner retinal crystalline deposition most prominent in the inferotemporal fovea of each eye.

presentation. No family history of ocular disease existed. Thirty-four months previously, he had been started on tamoxifen, 20 mg, 3 times a day, for unresectable hepatocellular carcinoma. He had stopped taking tamoxifen 5 weeks before presentation. Therefore, he had received a cumulative dose of 60 g over 33 months. He also had hypertension, which was diet controlled.

Visual acuities at presentation were RE: 20/60 and LE: 20/40. Anterior segment examination showed posterior chamber intraocular lens implantations with slight posterior capsule opacification in both eyes. No corneal opacity existed, and intraocular pressures were normal in both eyes. Funduscopy disclosed a peculiar, bilateral, symmetrical, inner retinal crystalline deposition that was predominantly in the temporal juxtafoveal region (Figure 1). Fundus fluorescein angiography showed mild bilateral cystoid macular edema.

Ocular toxicity in patients taking tamoxifen appears to be dose related, and it is uncommon in long-term, low-dose tamoxifen use (20 mg/day). Various ocular findings, such as lens opacities and whorl-like superficial corneal opacities, inner retinal crystalline deposition, macular edema, retinal