

therapy are at increased risk for visually significant spontaneous hyphema after pupillary dilation.

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Corticosteroid-Induced Glaucoma Attributable to an Adrenocorticotropin-Secreting Malignant Carcinoid Tumor of the Thymus

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PURPOSE: To describe the clinical and histopathologic findings in a patient with corticosteroid-induced open-angle glaucoma attributable to an adrenocorticotropin-secreting malignant carcinoid of the thymus.

METHODS: Case report. In a 33-year-old man, the clinical course, laboratory findings, and imaging results as well as the histopathologic findings are described.

RESULTS: Increased intraocular pressure in this patient represented a manifestation of severe hypercortisolism attributable to a malignant adrenocorticotropin-secreting carcinoid tumor. Surgical removal resulted in return of the intraocular pressure values to normal levels.

CONCLUSION: Thymic carcinoid is a rare cause of Cushing syndrome, which can lead to increased intraocular pressure. (*Am J Ophthalmol* 1999;128:100-101. © 1999 by Elsevier Science Inc. All rights reserved.)

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WE REPORT AN UNUSUAL CASE OF BILATERAL OPEN-angle glaucoma that was an early sign of a life-threatening systemic condition. A 33-year-old man, in perfect health until 1 month before initial examination and not taking any medications, was examined with ocular discomfort and dull pain. Upon ocular examination, uncorrected visual acuity was 20/20 in both eyes. The anterior and posterior segments were entirely normal, including perfectly clear corneas. Intraocular pressure was RE: 53 mm Hg and LE: 47 mm Hg. The patient denied any history of ocular disease. Family history for glaucoma as well as other eye conditions was negative.

Both pupils were fully reactive, and the anterior chambers were deep. No pseudoexfoliation, anterior chamber reaction, pigment dispersion, iris transillumination, heterochromia, posterior synechia, or keratoprecipitates were seen. The lenses were clear. The conjunctiva showed no evidence of dilated or tortuous vessels. No bruit, globe pulsation, or proptosis were present. Anterior chamber angles were open 360 degrees to the level of the ciliary body band in both eyes, without angle or iris neovascularization, excessive pigmentation, or peripheral anterior synechia. Axial length was 23 mm in both eyes. The optic nerve heads were normal and symmetric. Automated visual field tests showed mild defects, compatible with early glaucoma, in both eyes. The patient was placed on intensive pressure-lowering medications, including timolol, dipivefrin, and systemic acetazolamide, which was later discontinued because of its metabolic side effects.

From a state of perfect health 1 month earlier, this patient deteriorated rapidly, with fatigue, anorexia, weight loss, polyuria, and polydipsia of several weeks' duration. Upon physical examination, blood pressure was 140/100 mm Hg. A white tongue thrush consistent with oral candidiasis was found. Neurologic examination disclosed generalized weakness but no focal neurologic deficits.

A biochemistry examination disclosed the following abnormalities: potassium level was 2.0 mmol per liter (normal, 3.5 to 5.0 mmol/l), posing an immediate risk of life-threatening cardiac arrhythmias; metabolic alkalosis (pH = 7.54; HCO_3^- = 46 meq/l); and fasting glucose level of 11 mmol per liter (normal, 3.6 to 6.1 mmol/l).

The initial findings suggested the possibility of an endocrine condition. Urinary free cortisol measured 35,438 nmol per 24 hours (140 times the upper limit of normal). A free plasma cortisol level of 5804 nmol per liter (eight times the upper limit of normal) was found. Plasma adrenocorticotropin level measured 1,500 pmol per liter (130 times the upper limit of normal), proving that the hypercortisolism was secondary to an adrenocorticotropin-secreting tumor.

Total body computed tomography was nondiagnostic. Radiolabeled somatostatin analogue scintigraphy localized the secreting tumor to the anterior mediastinum. The clinical course was concurrently complicated by uncontrolled diabetes, pulmonary embolism, and bacterial sepsis.

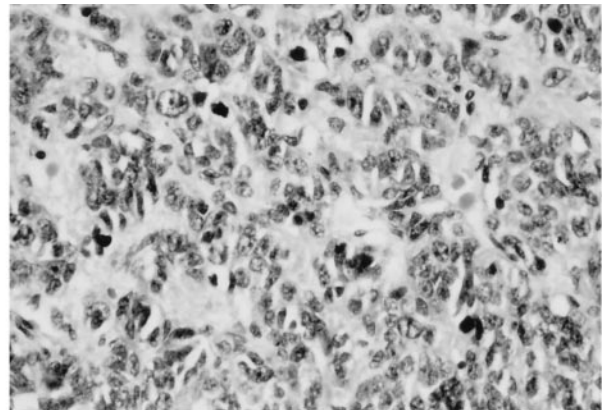
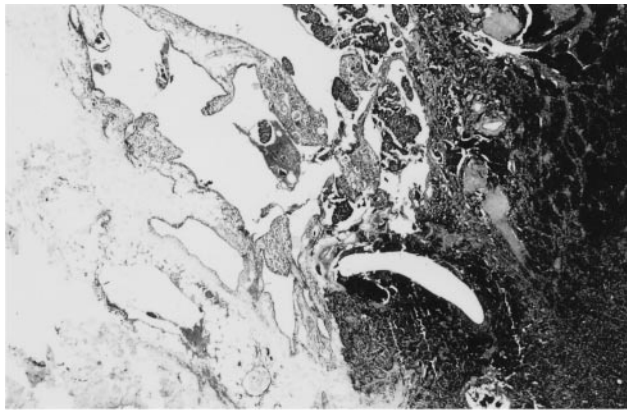


FIGURE. (Left) Under low magnification, the tumor (on the right) is invading normal adult thymus tissue (on the left) (hematoxylin and eosin). (Right) The tumor's appearance under higher magnification (hematoxylin and eosin).

Aggressive medical treatment in an intensive care unit enabled the patient to undergo definitive resection of the mediastinal tumor. Thoracotomy disclosed a $5 \times 6 \times 2$ -cm solid mass, weighing 45 g. No macroscopic evidence of local invasion was found. The histopathologic examination disclosed a malignant carcinoid neoplasm originating from the thymus (Figure). Immunohistochemical staining for adrenocorticotropin was positive.

After excision of the tumor, the patient's clinical condition, as well as the laboratory profile, improved markedly. Antiglaucoma therapy was discontinued, and intraocular pressure in both eyes reverted to normal. Several months later, intraocular pressures increased slightly to between 22 and 23 mm Hg. The patient is now being closely followed up without any pressure-lowering medication and with stable visual fields.

Malignant carcinoid of the thymus is a rare cause of Cushing syndrome. In a study by Wick and associates,¹ about one third of the patients with thymic carcinoids presented with Cushing syndrome caused by ectopic adrenocorticotropin production. The earliest description of the association between Cushing syndrome and increased intraocular pressure² appeared in 1937. Neuner and associates, in 1968, reported a series of 29 patients, of whom seven had intraocular pressure above 23 mm Hg.³ Haas and Nootens reported on a 34-year-old man who presented with intraocular pressures of 41 mm Hg in each eye, associated with a benign adrenal adenoma.^{2,4} Although extremely rare, increased intraocular pressure may be a result and a sign of an undetected corticosteroid-secreting tumor.

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Venous Collateral Remodeling in a Patient With Posttraumatic Glaucoma

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PURPOSE: To photographically document venous collateral development, remodeling, and regression in a patient with traumatic glaucoma.

METHODS: Consecutive fundus photographs were evaluated, labeled, and correlated with the clinical history of a patient with unilateral posttraumatic glaucoma.

RESULTS: This report photographically documents the appearance, remodeling, and subsequent disappearance of collateral vessels from venous occlusion on the surface of the optic disk in an eye with increased intraocular pressure and progressive glaucomatous cupping.

CONCLUSIONS: Asymptomatic chronic obstruction of a branch retinal vein on the optic disk may cause venous collaterals to develop in the absence of retinal hemorrhages or other signs of venous occlusive disease. Increased intraocular pressure, arteriolar sclerosis, and glaucomatous cupping are risk factors for these occlusions. (*Am J Ophthalmol* 1999;128:101-103. © 1999 by Elsevier Science Inc. All rights reserved.)

A 53-YEAR-OLD WOMAN WAS INITIALLY EXAMINED IN 1983 with a 3-year history of increased intraocular pressure in her right eye. Her highest recorded intraocular pressure in the right eye before treatment was 35 mm Hg.

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