Pituitary Apoplexy Causing Isolated Blindness After Cardiac Bypass Surgery

Pituitary apoplexy is a clinical syndrome that usually results from infarction of, or hemorrhage into, a pituitary macroadenoma. Typically, there is a rapid increase in tumor volume resulting in the abrupt onset of a variable combination of symptoms and signs that may include headache, meningismus, vomiting, visual loss, ophthalmoplegia, and stupor. Many factors have been implicated as precipitants, including major surgery. We describe 2 patients who developed blindness, without any other symptoms or signs to suggest pituitary apoplexy, due to infarction of undiagnosed pituitary macroadenomas during coronary artery bypass grafting (CABG).

Report of Cases. Case 1. A 79-year-old man with no prior symptoms to suggest neuro-opthalmologic or endocrine disease underwent CABG. Following extubation, he reported blindness but denied headache. On examination, he was alert and oriented and there was no meningismus. He had no light perception OU. His pupils were 2 mm in diameter and unreactive to light. Examination findings were otherwise unremarkable.

A large pituitary mass was evident on computed tomography (CT) (Figure, A). On magnetic resonance imaging (MRI), the mass was seen to be extending into the suprasellar cistern to compress the optic chiasm (Figure, B). Pituitary apoplexy was diagnosed and dexamethasone sodium phosphate (8 mg) was administered intravenously. Transsphenoidal decompression was performed 24 hours after CABG. Histopathologic examination of the tissue revealed a necrotic pituitary adenoma. Postoperatively, the patient’s pupils became dilated but remained unreactive, and optic atrophy developed. On follow-up, he remained blind with no light perception OU.

Case 2. A 64-year-old man with no prior symptoms to suggest neuro-opthalmologic or endocrine disease underwent CABG. Following extubation, he reported blindness but denied headache. On examination, he was alert and oriented and there was no meningismus. He had no light perception OU. His pupils were 2 mm in diameter and unreactive to light. Examination findings were otherwise unremarkable. Because it was thought that a stroke had produced his blindness, treatment with intravenous heparin sodium was commenced.

A large pituitary mass was visible on CT. On MRI, the mass was noted to be extending into the suprasellar cistern to compress the optic chiasm. There was no stroke. Pituitary apoplexy was diagnosed, heparin was stopped, and dexamethasone sodium phosphate (12 mg) was administered intravenously. Transsphenoidal decompression was performed 64 hours after CABG, following the correction of thrombocytopenia (platelet count, 72 × 10^9/L) and coagulopathy (international normalized ratio, 2.3; activated partial thromboplastin time, > 180 seconds). Histopathologic examination of the tissue revealed a necrotic pituitary adenoma with secondary hemorrhage. Postoperatively, the patient’s pupils became dilated but remained unreactive, and optic atrophy developed. On follow-up, he remained blind with no light perception OU.

Comment. Cardiac bypass surgery is rarely implicated as a precipitant for pituitary apoplexy. In one series reviewing 11 cases, 1 patient was known to have a macroadenoma and 3 had symptoms or signs of endocrine or neuro-opthalmologic disease prior to surgery. Postoperatively, all developed multiple symptoms and signs to suggest a diagnosis of pituitary apoplexy. In contrast, our patients developed blindness alone. To our knowledge, these are the first reported cases of pituitary apoplexy causing isolated blindness following cardiac bypass surgery. The blindness resulted from chiasmal compression, and loss of the pupillary reaction to light was...
the only localizing sign. The explanation for the initial miosis in our patients is not certain, although it was possibly a manifestation of bilateral Horner syndromes from hypothalamic compression.

Pituitary apoplexy could result from the interplay of several factors during cardiac bypass surgery. Macroadenoma infarction could result from hemodilution, hypotension, or microembolism from the heart or aorta, whereas hemorrhage could result from anticoagulation, thrombocytopenia, or platelet dysfunction.4

The imaging modality of choice for diagnosis is MRI because it clearly shows pituitary infarction and hemorrhage as well as compression of parasellar structures.1,3 If only CT is obtained, the diagnosis can be missed.1 However, in cases such as ours in which the lesion is obvious on CT, the diagnosis can be made and treatment with corticosteroids and transsphenoidal decompression can be initiated without MRI. Because there can be visual improvement in patients with severe visual loss who are not decompressed until several days after onset,6 the timing of surgery remains controversial. Although both of our patients remained blind despite undergoing decompression within 3 days, we believe that to maximize visual prognosis, urgent decompression is indicated in all patients with apoplexy who have rapid-onset blindness.

In summary, pituitary apoplexy is a rare complication of cardiac bypass surgery that usually occurs in patients with undiagnosed macroadenomas. When it produces isolated blindness, loss of the pupillary reaction to light is an important localizing sign. The diagnosis can be confirmed using CT (coronal images give the highest yield) or, ideally, MRI. When blindness develops rap-

Figure. Imaging from case 1. A, Postcontrast sagittal (left) and coronal (right) computed tomographic images show a nonenhancing mass (asterisk) arising from the pituitary fossa. B, Axial (left) and coronal (right) T2-weighted magnetic resonance images show the mass (asterisk) extending into the suprasellar cistern and compressing the optic chiasm (arrowheads).
idly, urgent decompression is recommended because visual prognosis is poor if intervention is delayed.

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Treatment of Iris Melanoma and Secondary Neovascular Glaucoma Using Bevacizumab and Plaque Radiotherapy

Iris melanoma is uncommon, representing only 2% of all uveal melanomas. In a series of 169 consecutive patients with iris melanoma, Shields et al. indicated that elevated intraocular pressure was found in 30% of cases. The main mechanism for the elevated pressure was mechanical obstruction of aqueous outflow from solid tumor or seeding. In that series, there were no eyes with secondary neovascular glaucoma. We report herein the unique association of neovascular glaucoma with chronic iris melanoma and discuss its management.

Report of a Case. A 64-year-old man was referred with a documented 12-year history of a slowly enlarging pigmented iris lesion in the left eye. The right eye had longstanding poor vision secondary to ocular histoplasmosis with a foveal scar diagnosed at age 15 years. At his initial visit, the visual acuities were 20/300 OD and 20/150 OS. On slitlamp examination of the left eye, a nodular, pigmented tumor with prominent intrinsic vascularity was noted in the inferior iris and measured 8 mm in the largest diameter and 1.9 mm in thickness, with corneal endothelial touch (Figure 1). There was no ciliary body component by ultrasound biomicroscopy. Associated ectropion uveae, cataract, and profound iris neovascularization of the entire iris were found. Intraocular pressures were 17 mm Hg OD and 24 mm Hg OS, despite using topical and oral antiglaucoma medications. Angle involvement with tumor seeding and neovascularization were noted on gonioscopy. Fundus examination showed bilateral peripheral histoplasmosis chorioretinal lesions with a large atrophic foveal scar in the right eye.

A diagnosis of enlarging iris melanoma in the left eye with secondary cataract and neovascular glaucoma was rendered. Due to contralateral amblyopia, enucleation was avoided, and because of extensive tumor seeding, surgical resection was not considered. Plaque radiotherapy combined with a single injection of 1.25 mg of intravitreal bevacizumab (0.05 mL at a concentration of 25 mg/mL) (Avastin; Genentech, Inc, San Francisco, California) was advised for treatment of the melanoma and resolution of the iris neovascularization. At the time of radiotherapy, fine-needle aspiration biopsy of the melanoma for genetic testing was performed.

Microsatellite array disclosed chromosome 3 monosomy. At 15 months' follow-up, the iris neovascularization and glaucoma were completely resolved with an intraocular pressure of 6 mm Hg while the patient was receiving no medications, and the residual melanoma scar remained regressed and flat (Figure 2).

Figure 1. A 64-year-old man was referred with an enlarging iris melanoma and secondary neovascular glaucoma in his only seeing eye. A, The melanoma occupied the inferior portion of the iris and diffuse neovascularization on the entire iris surface was noted. B, Ultrasound biomicroscopy depicted a solid iris mass touching the endothelium.