Third nerve palsy after coronary artery bypass surgery

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Third nerve palsy may occur after coronary bypass. It has been described in the setting of pituitary apoplexy. This article details a case of third nerve palsy after bypass in association with a nonapoplectic pituitary adenoma.

Clinical Summary

A 78-year-old man had originally presented with unstable angina. He underwent cardiac catheterization and angiography, which revealed severe triple vessel disease and moderately impaired left ventricular function. Other medical history included localized moderately differentiated carcinoma of the prostate treated with monthly goserelin acetate depot (Zoladex; Novartis, Basel, Switzerland), hypertension treated with enalapril maleate (Renitec; Merck Sharp & Dohme, Whitehouse Station, NJ), and inactive peptic ulceration. A left internal thoracic artery graft and 4 saphenous vein grafts were created with the use of cardiopulmonary bypass. The procedure was uneventful with a total bypass time of 93 minutes and a crossclamp time of 55 minutes. Systolic blood pressures during cardiopulmonary bypass ranged from 60 to 70 mm Hg. The patient was extubated 6 hours after the operation. Examination revealed a regular pulse rate of 80 beats/min and a blood pressure of 130/70 mm Hg. He was awake and fully orientated and had a complete ptosis of the right eye, which was abducted and depressed. There was complete loss of adduction and elevation of the eye, and the pupil was fixed and dilated. Superior oblique function was normal, as were movements of the left eye. He had no headache and visual fields were intact. The other cranial nerves and results of the limb examina-

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Figure 1. A, T1 weighted axial scan demonstrating pituitary adenoma with lateral extension into the cavernous sinus on the right. B, T2 weighted coronal magnetic resonance image through the pituitary gland showing a right-sided adenoma extending into the posterior cavernous sinus and abutting the right posterior clinoid process.

tion were within normal limits; in particular, there were no contralateral long tract signs. Both plantar responses were flexor. The findings were consistent with an isolated painless complete palsy of the right third nerve. The differential diagnosis was considered to be either an incidental microvascular ischemic oculomotor neuropathy or a previously asymptomatic pituitary adenoma undergoing silent infarction or hemorrhage.

A cranial computed tomographic scan targeting the sellar region a few hours later was suggestive of a possible lesion in the right side of the pituitary gland, and further evaluation with magnetic resonance imaging was recommended. This was performed 4 days later and demonstrated a mass in the right side of the pituitary fossa consistent with a pituitary adenoma (Figure 1). Suprasellar extension and cavernous sinus invasion were apparent on the right side but no evidence of hemorrhage. The third nerve palsy improved over the next week and by 6 weeks had completely resolved. Endocrine evaluation revealed normal levels of prolactin, growth hormone (including insulin-like growth factor-1), thyroid-stimulating hormone, and adrenocorticotrophic hormone. Levels of luteinizing hormone and testosterone were depressed consistent with goserelin acetate therapy.

Discussion

Pituitary infarction may present in different ways ranging from being entirely silent through to the florid features of apoplexy (hemorrhage into a macroadenoma). The latter includes retroorbital headache, nausea and vomiting, meningism, visual field defects (upward extension of mass), ophthalmoplegia (commonly third nerve palsy—lateral expansion), and facial pain and numbness (lateral expansion).¹ Pituitary adenomas that invade the cavernous sinus can present with a third nerve palsy in the absence of apoplexy.² In these cases, an isolated painless third nerve palsy may

be the sole presenting feature. Demere and associates³ (1997) reported on 6 patients with third nerve palsies occurring after coronary artery bypass surgery.³ These 6 cases were all attributed to ischemia, and in 4 cases there was evidence of associated small vessel cerebral disease. The lesions were painless and bypass time was not prolonged. Cooper and colleagues⁴ (1986) reported 3 cases of third nerve palsy due to pituitary adenomas in the immediate post-operative period after cardiac surgery.⁴ In 2 of the 3 cases there was evidence of hemorrhagic infarction in the adenoma after transphenoidal resection. In the third case neither hemorrhage nor necrosis was seen in the specimen.

The present pituitary neoplasm had not undergone hemorrhagic infarction but was invading the cavernous sinus in proximity to the third nerve. The mechanism of the neuropathy is speculative, but in view of the relatively rapid and complete resolution of the neuropathy, it is most likely that ischemia of the tumor mass during bypass surgery resulted in edema and expansion of the adenoma, directly compressing the nerve. An alternative possibility of coincidental ischemia of the oculomotor nerve is intuitively less plausible.

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