Bacterial meningitis after spinal anesthesia can be caused by three possible mechanisms: hematogenous spread, equipment and anesthetic drug contamination, and break in the sterile technique. The source of the bacteria in our patient remained unclear. *S. salivarius* is a commensal of the skin, gastrointestinal tract, genitourinary tract, and oral cavity and paranasal sinuses. It is unlikely that surgery resulted in bacteremia. Even though an aseptic procedure was carefully followed, it is obvious, that the bacterium was introduced to the spinal space during the spinal puncture.

Meningitis is a serious complication and its early diagnosis and effective treatment is essential. Meningitis should always be considered as a possible differential diagnosis in patients suspected of having postspinal headache. A thorough knowledge and practice of aseptic techniques is crucial in performing spinal and extradural anesthesia.

Anesthesiology
1998; 89:1580–2

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Pituitary Apoplexy following Mitral Valve Repair

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POSTOPERATIVE neurologic or neuropsychologic deficits, or both, remain a common cause of disability after otherwise successful cardiac surgery. However, not all deficits are caused by gaseous or atheromatous emboli.

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Key words: Cardiac surgery; cardiopulmonary bypass; complications; pituitary apoplexy.

Case Report

A 56-yr-old, 90-kg man presented for elective mitral valve repair. Mitral valve prolapse was diagnosed initially 4 yr previously. One month before admission, a loud systolic murmur was heard at the cardiac apex; nevertheless, the patient continued to be asymptomatic. Medical history included chronic low-back pain and three lumbar laminectomies. He took no medications other than naproxen. A transthoracic echocardiography revealed normal left ventricular function, left atrial enlargement, mild tricuspid regurgitation, and severe mitral regurgitation, possibly from a torn posterior chorda tendinea.

On the day before surgery, physical examination revealed a fit male, with pupils equally reactive to light and accommodation. The electrocardiogram showed a normal sinus rhythm of 86 beats/min and findings consistent with left ventricular hypertrophy. There was an occasional premature ventricular beat.

After receiving preoperative antibiotic prophylaxis for bacterial endocarditis, anesthesia was induced using intravenous ketamine, fentanyl, midazolam, and succinylcholine. Anesthesia was maintained with isoflurane and fentanyl. Paralysis was maintained with pancuronium. Transesophageal echocardiography confirmed severe mitral regurgitation with a flail posterior leaflet. Venous and arterial cannulae were placed after heparin anticoagulation. Extracorporeal circulation was

initiated, and the patient was cooled to a blood temperature of 29°C. The posterior leaflet of the mitral valve exhibited mild myxomatous changes. The torn fan chordae to the anterior leaflet at the aortic commissure was excised and the anterior leaflet was repaired. Ring annuloplasty was performed. Cardiopulmonary bypass time totalled 1 hr 34 min; the aortic cross-clamp time was 57 min.

Transesophageal echocardiography before separation from bypass showed a satisfactory mitral valvular repair, and transesophageal echocardiography was used to monitor the completeness of air evacuation from the left atrium and its tributaries and the left ventricle. After rewarming, the rhythm spontaneously converted to a sinus mechanism, and, after as much intracardiac air had been evacuated as was feasible, the patient was weaned from bypass without inotropic drug support. Heparin was reversed with protamine, bypass cannulae were removed, and the chest was closed. The patient was transported to the cardiac intensive care unit in stable condition.

Approximately 5 min after arrival, the patient's eyes were untaped, revealing that the right pupil's diameter was 4 to 5 mm and unreactive to light and that the left pupil's diameter was 1 to 2 mm and reactive to light. Six hours later, when the trachea was extubated, the patient complained of blurred vision in his right eye. Further examination of the right eye revealed total ophthalmoplegia, lack of accommodation and pupillary response, and ptosis. There was also complete lack of sensation on the right forehead, eyebrow, eyelid, and upper nose. The initial presumptive diagnosis was perioperative stroke.

By postoperative day 2 the cranial nerve palsies remained unchanged, but the patient, making an otherwise normal recovery, was eating, drinking, and ambulating. On postoperative day 3, a neuroophthalmology consultant confirmed profound cranial nerve deficits of III, IV, VI, and the ophthalmic division of V on the right side, suggesting a cavernous sinus process. Multiple brain stem infarcts could not adequately explain the findings. Magnetic resonance imaging was deferred because of the presence of ferrous cardiac pacing wires. Cranial computed tomography revealed a mass lesion within the sella turcica extending to the suprasellar and sphenoid sinus areas, eroding through bone into the right sphenoid and cavernous sinuses (fig. 1). This finding narrowed the differential diagnosis to pituitary adenoma *versus* mucocele. A consulting neurosurgeon prescribed dexamethasone. On the fourth postoperative day, pacing wires were removed permitting magnetic resonance imaging. This study confirmed extension of the mass into the right cavernous and sphenoid sinuses and into the clivus, consistent with a necrotic, infarcted pituitary adenoma, craniopharyngioma, or less likely, a clivus chordoma.

Laboratory analysis of plasma revealed hypersecretion of prolactin (38.1 pg/dl, normal range, 1.8–14.4 pg/dl), and hyposecretion of thyrotropin (0.36 μIU/dl, normal range 0.4–5.5). The patient also showed biochemical signs of hypothyroidism (triiodothyronine = 37 ng/dl, normal range, 62–194; thyroxin = 4.8 ng/dl, normal range, 5.5–11.8), and of cortisol hyposecretion (cortisol = 1.7 μg/dl, normal range, 5.5–20). These findings were consistent with a prolactin-secreting (chromophobe) pituitary adenoma.

On the sixth postoperative day, the patient underwent successful transsphenoidal hypophysectomy. Pathologic evaluation of the surgical specimen revealed an infarcted pituitary adenoma (pituitary apoplexy) with coagulative necrosis. Postoperative course was uneventful and he was discharged to home 5 days after brain surgery and 13 days after mitral valve repair.

Four months after surgery, the patient had greatly improved and experienced only mild blurring of vision in his right eye. He continued to have mild weakness of the right lateral rectus muscle, causing diplopia with extreme right gaze. He continued to require maintenance levothyroxine.

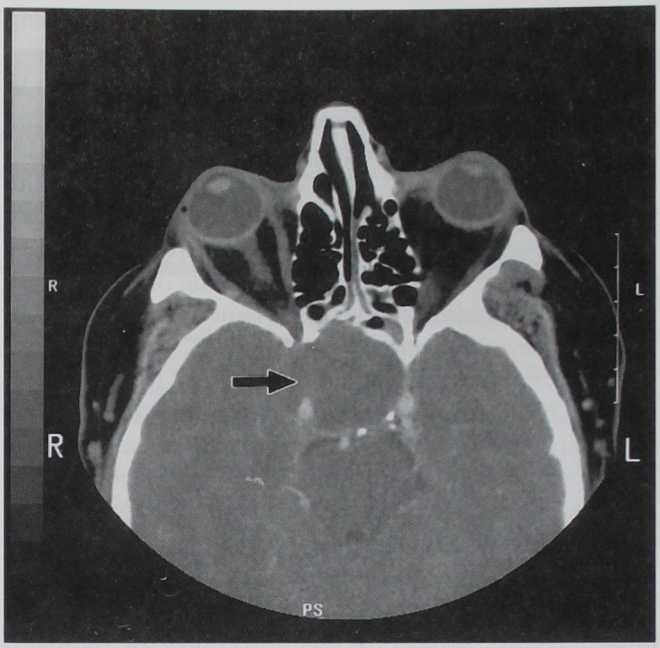

Fig. 1. Enhanced cranial computed tomography scan shows a large mass in the sella turcica with erosion of the right sphenoid bone. Arrow indicates erosion of right sphenoid bone.

Discussion

Pituitary apoplexy is a clinical syndrome resulting from acute hemorrhage or necrosis of the pituitary gland. The syndrome, first described by Bailey in 1898,[1] was named by Brougham in 1950.[2] Pituitary apoplexy often is associated with pituitary adenoma. Pituitary adenomas are identified in 1.4%[3] of adult necropsies; however, only 14% of these show evidence of hemorrhage or infarction. In a retrospective study of 799 patients with known pituitary adenoma, clinical symptoms of pituitary apoplexy occurred in 5%.[4]

The clinical syndrome of pituitary apoplexy includes headache (76%), visual acuity and visual field defects (62%), ocular palsies (40%), nausea and vomiting (21%), altered mental status (19%), hemiparesis (4.3%), and fever (2.4%).[5] Compression of surrounding structures by the expanding pituitary tissue may result in palsies of cranial nerves II, III, IV, V, and VI and hemiparesis caused by occlusion of the carotid artery and altered sympathetic autoregulation secondary to hypothalamic compression.[5] Endocrinopathies are typically present

due to compression of normal pituitary by surrounding tumor or to the effects of tumor hypersecretion.

Pituitary apoplexy has been described previously in nine cases after cardiopulmonary bypass.[6–10] Possible mechanisms of injury during cardiopulmonary bypass include acute pituitary edema secondary to hemodilution from crystalloid priming of the cardiopulmonary bypass tubing.[6] Edema might also result from inadequate superior vena cava drainage or from drug or blood product-induced histamine release.[6] Tissue ischemia can result from low flow states ($< 2.2\ l \cdot min^{-1} \cdot m^{-2}$) or low mean arterial pressure (< 50 mmHg) and from cerebral emboli during cardiopulmonary bypass.[6] Compression from rapid tumor expansion may further compromise pituitary tissue perfusion.[9] Finally, hemorrhage into the adenoma can occur because of heparin anticoagulation or other clotting abnormalities.[6]

In this patient, dissimilar to many previously described, signs of pituitary apoplexy were apparent immediately after surgery. In this procedure, the right and left atria were open to the atmosphere. As a consequence, despite evidence that air was removed adequately from the heart and cardiopulmonary bypass circuitry, the initial diagnosis after surgery was that of possible brainstem infarction. Stroke after cardiopulmonary bypass occurs in 2 to 6% of patients and more subtle neuropsychiatric changes occur in up to 66% of patients, particularly within the first few days after surgery.[11] Initial therapy of presumed embolic brain ischemia is conservative. However, despite the patient's rapid recovery from cardiac surgery, the cranial nerve palsies remained, mandating further evaluation. A diagnosis of the rare disorder pituitary apoplexy was soon determined.

Although conservative management of pituitary apoplexy has been advocated in selected patients,[12] prompt surgical decompression, especially in patients with impairment of vision or consciousness, is recommended by other authors.[4,5] Some recommend that patients with known pituitary adenoma and cardiac disease should undergo pituitary adenoma resection before cardiac surgery if stable; however, this recommendation must be tempered by the acknowledgment that the total number of patients undergoing cardiac surgery with occult pituitary adenoma is unknown.[10]

In summary, we present a case of pituitary apoplexy presenting with new and persistent cranial nerve palsies immediately after mitral valve repair surgery. Although our patient experienced infarction of brain tissue in the form of the pituitary gland, the case is a reminder that all new neurologic deficits after cardiac surgery do not result from emboli, thrombosis, transient ischemic attacks, or systemic hypotension.

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