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 parasenal sinuses. It is unlikely that surgery resulted in bacteremia. Even though an aseptic procedure was carefully followed, it is obvious, that the bacteria 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.

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Pituitary Apoplexy following Mitral Valve Repair

Eric N. Meek, M.D.,* John Butterworth, M.D.,† Neal D. Kon, M.D.,‡ David A. Zvara, M.D.,§ George E. Ash, Jr., M.D.,* Timothy J. Martin, M.D.

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.

* Resident, Department of Anesthesiology.
† Professor and Head, Section of Cardiothoracic Anesthesiology.
‡ Associate Professor, Department of Cardiothoracic Surgery.
§ Assistant Professor, Department of Anesthesiology.

Received from the Departments of Anesthesiology, Cardiothoracic Surgery, and Ophthalmology, Wake Forest University School of Medicine, Winston-Salem, North Carolina. Submitted for publication April 17, 1998. Accepted for publication July 13, 1998.

Address reprint requests to Dr. Butterworth: Department of Anesthesiology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, North Carolina 27157-1009. Address electronic mail to: jbutser@wfubmc.edu

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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 mitral 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 sucnyclidine. Anesthesia was maintained with isoflurane and fentanyl. Paralysis was maintained with pancuronium. Transesophageal echocardiography confirmed severe mitral regurgitation with a flap 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;.T he aortic cross-clamp time was 57 min.

Transesophageal echocardiography before separation from bypass
showed a satisfactory mitral valvular repair, and transesophageal echo-
cardiography 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 mecha-
nism, 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 untape,
reveling 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 un-
changed, but the patient, making an otherwise normal recovery, was
eating, drinking, and ambulating. On postoperative day 3, a neuro-
ophthalmology 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 ade-
quately explain the findings. Magnetic resonance imaging was deferred
because of the presence of ferrous cardiac pacing wires. Cranial com-
tputed 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, craniopharyn-
gioma, or less likely, a clivus chordoma.

Laboratory analysis of plasma revealed hypersecretion of prolactin
(38.1 μg/dl, normal range, 1.8–14.4 μg/dl), and hyposecretion of
thyrotropin (0.36 μIU/ml, 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-secret-
ing (chromophobe) pituitary adenoma.

On the sixth postoperative day, the patient underwent successful
transsphenoidal hypophysectomy. Pathologic evaluation of the surgi-
cal specimen revealed an infarcted pituitary adenoma (pituitary apo-
plexy) 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

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.

to have mild weakness of the right lateral rectus muscle, causing
diplopia with extreme right gaze. He continued to require mainte-
nance levothyroxine.

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, was named
by Brougham in 1950. Pituitary apoplexy often is asso-
ciated with pituitary adenoma. Pituitary adenomas are
identified in 1.4% of adult necropsies; however, only
14% of these show evidence of hemorrhage or infarc-
tion. In a retrospective study of 799 patients with known
pituitary adenoma, clinical symptoms of pituitary apo-
plexy occurred in 5%. 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%). Compression of surrounding structures by
the expanding pituitary tissue may result in palsies of
cranial nerves II, III, IV, V, and VI and hemiparesis
carried by occlusion of the carotid artery and altered
sympathetic autoregulation secondary to hypothalamic
compression. Endocrinopathies are typically present

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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. Possible mechanisms of injury during cardiopulmonary bypass include acute pituitary edema secondary to hemodilution from crystalloid priming of the cardiopulmonary bypass tubing. Edema might also result from inadequate superior vena cava drainage or from drug or blood product-induced histamine release. 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. Compression from rapid tumor expansion may further compromise pituitary tissue perfusion. Finally, hemorrhage into the adenoma can occur because of heparin anticoagulation or other clotting abnormalities.

In this patient, dissimilar to many previously described, signs of pituitary apoplexy were apparent immediately after surgery. In this procedure, the right and left atra 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. 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, prompt surgical decompression, especially in patients with impairment of vision or consciousness, is recommended by other authors. 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.

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.

References


