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POST CARDIAC SURGERY OPHTHALMOPLEGIA DUE TO PITUITARY APOPLEXY

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BACKGROUND

Pituitary apoplexy is a clinical syndrome caused by the rapid enlargement of a pituitary adenoma due to hemorrhage or infarction. Because of associated destruction of normal pituitary tissue, both mass effect and hypopituitarism can combine to cause a fulminant and potentially fatal clinical illness. Hemorrhage into these lesions can be of variable degrees and thus there exists a spectrum of clinical manifestations ranging from an asymptomatic to a potentially fatal illness. The clinical syndrome of pituitary apoplexy occurs due to both local anatomic compression by a rapidly enlarging pituitary tumor and sudden loss of pituitary hormonal secretion due to compression and infarction of normal pituitary tissue.

Pituitary apoplexy in a pre-existing pituitary tumor can result in serious and permanent neurologic deficits following cardiac surgical procedures. Several factors related to the altered physiology of cardiopulmonary bypass (CPB) contribute separately or in combination to the development of this syndrome. Pituitary apoplexy is a neurosurgical emergency because of impending progressive visual deficits and widespread metabolic derangement due to pituitary insufficiency in the immediate postoperative period.

We describe a case report to emphasize the urgency of diagnosis with a high index of suspicion to avoid a life-threatening catastrophe in case the condition goes undiagnosed. Pituitary apoplexy itself is a completely treatable condition without any long term effects provided there is timely surgical intervention and appropriate medical therapy.

CASE REPORT

A 65 years old male who was a known case of dyslipidemia and ischemic heart disease was admitted electively in cardiothoracic service with history of exertional chest pain. Angiography revealed two-vessel coronary artery disease with proximal left anterior descending artery (LAD) disease. He underwent Coronary Artery Bypass Grafting (CABG). Post operatively he came out of the operating room without inotropic support and was shifted to Coronary ICU (CICU). Clinical evaluation in the CICU in the immediate postoperative period revealed that he had a right sided dilated pupil with absent light reflex. He was extubated the same evening. On regaining consciousness, he was noted to have right-sided ptosis along with a dilated fixed pupil. He remained hemodynamically stable throughout this period.

Neurology consult was given and patient was advised to have an urgent CT scan brain with the provisional diagnosis of ischemic stroke. This CT scan brain (plain) revealed a hypo dense area on the right side, just inferior to the head of the caudate nucleus, suggestive of acute infarction. Patient was started on antiplatelet therapy.

On the fourth post operative day, patient became drowsy and developed ptosis of the left eye as well. On examination the pupil on the left was reactive but extraoccular movements were severely restricted bilaterally. There were decreased pin-prick sensations over the left half of the body. Serum electrolyte showed that serum sodium was low (S. Na=121).

At this stage MRI brain and pituitary fossa were done which showed a cystic mass in the pituitary fossa with suprasellar extension. This mass was causing pressure on the optic nerves and chiasma. There was extension into the cavernous sinuses on both sides as well as erosion of sellar floor and extension into the sphenoid sinus. There was a small hypo intense area within it representing hemorrhage. There was also a small area of infarction below the head of the caudate on right side. Hormonal

profile revealed a low serum cortisol (3.84), low prolactin (0.83), and low LH (0.88). Remaining hormone profile was within normal range. Serum sodium however continued to remain low. Patient was immediately started on hydrocortisone and thyroxin. On 7th post CABG day, patient underwent trans-sphenoidal removal of the pituitary mass and it was found that mass was cystic and suckable with evidence of some foci of old hemorrhages. Histopathology confirmed it to be a pituitary adenoma with marked pituitary necrosis consistent with pituitary apoplexy. Immunohistochemistry revealed non-secretory pituitary adenoma. Patient showed improvement in his visual symptoms and was discharged on fifth post-operative day. At one month follow up, neurological examination showed no residual neurological deficits although he continued to require hormone replacement therapy.

DISCUSSION

Pituitary apoplexy was first described in 1898 by Bailey, a physician who noted that devastating hemorrhage could occur within a pituitary adenoma. It is a clinical syndrome caused by the rapid enlargement of a pituitary adenoma due to hemorrhage or infarction. Because of associated destruction of normal pituitary tissue, both mass effect and hypopituitarism can combine to cause a catastrophic clinical condition. Hemorrhage into these lesions can be of variable degrees, and thus there exists a spectrum of clinical manifestations ranging from asymptomatic to potentially fatal illness.[1,2]

The syndrome of acute, symptomatic pituitary apoplexy is rare and its presentation is highly variable.[3,4] Whereas early investigators suggested that pituitary apoplexy occurred primarily in patients with large macroadenomas with suprasellar extension,[5] it is now evident that tumors of almost any size may undergo hemorrhage and apoplexy.[2,4-8] Transient increase in intracranial pressure with resultant hypoperfusion of the pituitary gland, as caused by coughing, sneezing, or positive pressure ventilation, has been reported as a precipitant of apoplexy in patients with pituitary adenoma.[2,4,5]

Post CABG apoplexy is now a well recognized entity and several cases have been reported previously. Various presentations of post CABG pituitary apoplexy include post-operative ptosis, Addisonian crisis, malaise, decreased libido, persistent hyponatremia with headache and even deep coma with dilated pupils.[2,4,9-13] However some patients have been reported to be asymptomatic till they presented several months later with signs and symptoms of pituitary insufficiency.[2]

Pituitary apoplexy in a pre-existing pituitary tumor can result in serious and permanent neurologic deficits following cardiac surgical procedures. Several factors related to the altered physiology of cardiopulmonary bypass (CPB) contribute separately or in combination to the development of this syndrome.[14] Such factors include ischemia, hemorrhage, edema, and positive pressure ventilation.[2,4] Other precipitating factors may be related to the extracorporeal bypass apparatus, anticoagulation, low cerebral blood flow, and even anesthetic agents.[14,15] Reduced blood flow in the pituitary gland may result from fluctuations in blood pressure. Indeed, hypotension in the setting of cardiac surgery, lumbar laminectomy or haemodialysis has been associated with pituitary apoplexy of both normal and adenomatous glands. Some authors have suggested that stimulation of the pituitary gland by "stress", and fluctuations in blood pressure may play a more important part in the pathophysiology of pituitary apoplexy following cardiac surgery.[15] Current recommendation is to consider operating on patients with pituitary adenoma who need coronary artery bypass grafting operation off pump, so as to prevent pituitary apoplexy that cardiopulmonary bypass may cause.[15] Transsphenoidal surgical decompression in the present case and those previously reported appears to be safe after cardiac surgery and may be helpful in amelioration of compression of nearby structures.[1]

CONCLUSION

Pituitary apoplexy should be considered as a diagnostic possibility in patients who develop visual disturbances or ophthalmoplegia following open heart surgery.

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