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CLINICAL REPORT

Pituitary apoplexy during general anesthesia in beach chair position for shoulder joint arthroplasty

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Abstract Pituitary apoplexy is a rare but potentially life-threatening clinical syndrome caused by the sudden enlargement of pituitary adenoma secondary to infarction and/or hemorrhage. It may be the first presentation of previously undiagnosed pituitary adenoma. Although various precipitating factors of pituitary apoplexy are indicated, the pathogenesis remains unknown. In this report, we describe for the first time a case of pituitary apoplexy developed explicitly during general anesthesia supplemented with interscalene brachial plexus block in beach chair or barbershop position for shoulder joint arthroplasty.

Keywords Beach chair position · Complication · Interscalene brachial plexus block · Pituitary apoplexy

Introduction

Pituitary apoplexy (PA) is a clinical syndrome resulting from acute hemorrhage or necrosis or both of a preexisting pituitary adenoma. PA was first described in 1898 [1], but the concept of the disease was only established in 1950 [2]. The precipitating factors have been categorized into four components [3], namely, those associated with reduced blood flow, an acute increase in blood flow, stimulation of the pituitary gland, and the anticoagulated state.

In this report, we describe for the first time a case of PA in a patient that developed explicitly during general anesthesia supplemented with an interscalene brachial plexus block in the beach chair or position during shoulder joint arthroplasty.

Case report

A 60-year-old male patient with persistent shoulder pain and stiffness was admitted to our hospital for shoulder joint arthroplasty. The patient was 183 cm tall and had a body weight of 72 kg. He had a 5-year history of diabetes mellitus and was being treated with insulin and glimepride. Four years prior, the patient had undergone anterior transvertebral herniotomy for cervical disc herniation. Physical examination at admission revealed that the patient still had numbness in his left upper limb. He had no history of headache.

Preoperative blood pressure (BP) was 130/75 mmHg in the supine position. After the patient had been administered an interscalene brachial plexus block with 30 ml of 0.5% ropivacaine, general anesthesia was induced by the administration of 8% sevoflurane, 50 mg propofol, 0.25 μ/kg/min remifentanil, and 45 mg rocuronium. Anesthesia was maintained with 1.5–2% sevoflurane and 0.15–0.05 μ/kg/min remifentanil. Immediately after the patient was placed in the beach chair position, BP measured in the upper arm decreased to 60/36 mmHg for <5 min; this was treated with ephedrine administration. After the episode, the BP remained in the 90- to 100-mmHg systolic range for the remainder of the anesthesia. Oxygen saturation was 100%,

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and end tidal CO_2 values were in the 30 s range throughout the anesthesia. There was little estimated blood loss, and a total of 1250 ml of crystalloid fluid was infused during the 6 h and 1 min of anesthesia. Immediately upon emerging from anesthesia, the patient's BP was 140/87 mmHg in the supine position.

Upon arrival in the post-anesthesia care unit (PACU), the patient complained of a severe headache limited to the right side. The headache was persistent even after the administration of the nonsteroidal anti-inflammatory drug flurbiprofen. Despite the severe headache, the patient was free of neurological symptoms, such as anisocoria, visual disturbance, nuchal rigidity, abnormal reflex, ocular palsy, visual disturbance, nausea, and vomiting. He was able to walk in a straight line unaided. After a 2.5-h stay in the PACU without remission of headache, the patient was moved to the ward. The headache persisted at 3.5 h after emergence from anesthesia, and the patient complained of nausea and vomited. At 4 h after anesthesia, the patient underwent a computed tomographic scan (CT) screening to rule out intracerebral hemorrhage, subarachnoid hemorrhage, or infarction. A supraseller mass was visible on the CT scan (Fig. 1). On post-operative day (POD) 1, a follow-up magnetic resonance imaging (MRI) study revealed a 2.5 × 1.5-cm supraseller mass extending into the right cavernous sinus (Fig. 2), leading to a diagnosis of PA. The results of laboratory tests indicated mild hyponatremia (133 mEq/L). The results of endocrine studies performed on POD 1, including thyroid function tests, luteinizing hormone, follicle stimulating hormone, cortisol, and prolactin, were all within normal limits, indicating the adenoma was non-functional. On POD 2, the patient developed anisocoria (Rt 2.5 mm; Lt 2.0 mm), right ptosis and ophthalmoplegia, and palsy of the third nerve without any general abnormality, such as hemiparesis. On POD 16, the patient underwent successful transsphenoidal hypophysectomy. Pathological evaluation of the surgical specimen revealed an infarcted pituitary adenoma with hemorrhage surrounding the tumor. The finding led to the final diagnosis of PA due to tumor infarction followed by hemorrhage of a non-functional pituitary adenoma. The subsequent clinical course and recovery were uneventful. The patient was discharged day 34 following the development of the PA. At the 2-month follow-up, he showed a gradual recovery of ophthalmoplegia.

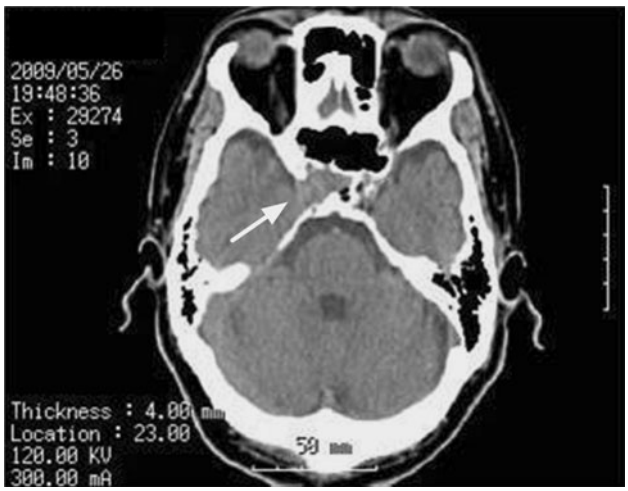

Fig. 1 Sagittal computed tomographic image scanned on the showing a supraseller mass (*arrow*) in a 60-year-old man who underwent shoulder joint arthroplasty

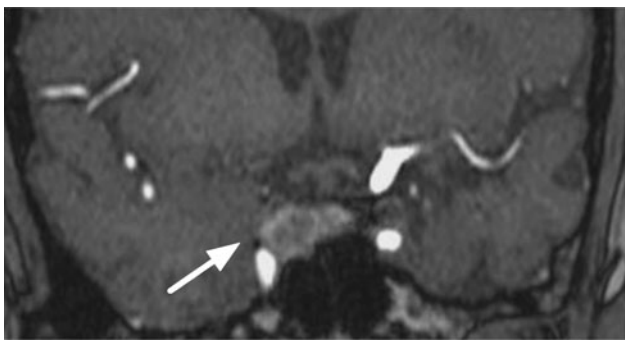

Fig. 2 Coronal T2-weighted magnetic resonance imaging scan showing a 2.5 × 1.5-cm supraseller mass (*arrow*) extending into the right cavernous sinus in a 60-year-old man who underwent shoulder joint arthroplasty

Discussion

Pituitary apoplexy is a clinical syndrome resulting from acute hemorrhage or necrosis, or both, of a preexisting pituitary adenoma. The pathological syndrome is characterized by headache, visual activity, visual field defect, ocular palsy, nausea and vomiting, altered mental status, and hemiparesis. During an acute episode, the expanding pituitary mass may compress the surrounding structures, namely, the cavernous sinus and neurovascular bundle, resulting in cranial nerve palsies and ophthalmoplegia. A review of published case reports in the English literature from 1990 to 2006 revealed 97 reported cases of PA. The precipitating factors were divided into stimulation of the pituitary gland/tumor, surgery, anticoagulant therapy, head injury, radiation therapy, and other miscellaneous causes. Among the 40 patients in whom surgery was implicated as a precipitating factor, 22 had undergone cardiac surgery, six had pituitary surgery for large tumors, 11 had undergone other surgical procedures, and one had spinal anesthesia [1, 4–7]. Patients undergoing cardiac surgery seem to be at higher risk than those undergoing other types of surgery [5], possibly due to the frequent use of anticoagulant therapy, cardiac bypass, and more severe hypotension among this patient group.

Intraoperative cerebral hypoperfusion is considered to be due to systemic hypotension, and the beach chair positioning was an etiological factor in our case. The vessels within the tumor itself are abnormal, being generally smaller than normal vessels, variable in number, and poorly fenestrated with irregular and ruptured basement membranes. In addition, the expanding mass may directly compress the infundibulum or the hypophyseal artery and impair blood supply to the entire anterior lobe. As a result of these anatomical features, a pituitary adenoma with poor auto-regulation of blood flow is susceptible to infarction or hemorrhage with relatively minor changes in perfusion pressure [1]. The volatile anesthetics [8] and vascular dysfunction due to diabetes mellitus disturb cerebrovascular auto-regulation, causing perfusion to become pressure-dependent. Placing the anesthetized patient in the beach chair position conveys some risk of impaired cardiovascular function, particularly hypotension, at the same time. Mean arterial pressure should be corrected to head level to obtain a meaningful index of cerebral perfusion pressure. The BP should be maintained at a minimum value of 60 mmHg in healthy patients who are assumed to have normal cerebral vasculature [9]. Four cases of ischemic brain injury after shoulder surgery in the beach chair position have been reported [10], suggesting that hypotension during the anesthesia may be one of the causes of PA, especially in patients in the beach chair position even though the duration of the procedure is very short.

A transient increase in intracranial pressure could be another cause of PA in our case [1]. This could result from lumbar puncture for pneumoencephalography, repetitive coughing and sneezing secondary to upper respiratory infections, minor head trauma, mechanical ventilation, and scuba diving. In fact, at the time of extubation, our patient coughed a few times. However, the patient never moved and coughed during the operation under anesthesia.

Although headache is the most commonly reported symptom and presents in up to 100% of patients affected with PA, it is almost universally described as severe and abrupt and located retro-orbitally or bi-frontally. Because headache located on the right side and neurological deficits did not occur simultaneously in our patient, the diagnosis was difficult. Moreover, in our patient, ophthalmoplegia, which is one of the most frequently observed clinical symptoms in PA, did not develop until 2 days after the incidence of PA. We assume that the hemorrhage after infarction of the pituitary adenoma progressed slowly, although the exact mechanism of the discrepancy is unknown.

In summary, we report a case of PA. Asymptomatic pituitary adenomas are difficult to be diagnosed preoperatively. The case presented here demonstrates that PA can occur even after shoulder joint arthroplasty surgery with the patient in the beach chair position. To the best of our knowledge, this is the first reported case of PA during shoulder joint arthroplasty. Because headache is a nonspecific symptom, the diagnosis was difficult in our case. We would like to emphasize the importance of a high index of suspicion for early diagnosis and management of this potentially fatal neurological perioperative complication.

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