Pituitary apoplexy after spinal anaesthesia

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Summary

We report the case of a previously healthy 51-yr-old male who underwent an uneventful total hip replacement under spinal anaesthesia. His immediate postoperative course was complicated by the development of a severe frontal headache. Initial conservative treatment included oral analgesics and an epidural blood patch. The headache persisted and was followed by progressive vision loss and a right partial third nerve palsy. The patient was almost blind at the time of transfer to our neurosurgical unit. Relevant investigations revealed marked hyponatraemia (serum sodium concentration 122 mmol litre\(^{-1}\)) and second-degree heart block (Mobitz I). A CT scan showed a pituitary tumour and a large necrotic pituitary adenoma was excised. The postoperative course was uneventful with return of near normal vision at the time of discharge. Clinicians should consider this diagnosis when focal neurological deficits occur with post-dural puncture headache. (Br. J. Anaesth. 1998; 81: 616–618)

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Pituitary apoplexy is a rare clinical syndrome resulting from sudden infarction of a normal or adenomatous pituitary gland, which, if left untreated, may result in permanent neurological deficit.\(^1,2\) Symptoms follow rapid enlargement of the gland, compression of the optic chiasma and neighbouring structures, with associated endocrinopathy from pituitary necrosis.

Pituitary apoplexy has been reported after several surgical procedures.\(^3\) Most cases have been associated with cardiopulmonary bypass under general anaesthesia.\(^4\) Our case highlights complications associated with spinal anaesthesia, the clinical presentation of pituitary apoplexy and the potential aetiological factors in this particular patient.

Case report

A 51-yr-old male with osteoarthritis underwent elective total hip replacement in another institution. His past medical history was unremarkable; he received only oral analgesics. His preoperative physical examination was normal and there were no neurological deficits. A spinal anaesthetic technique was used. Intrathecal 0.5\% bupivacaine 3 ml was administered via a 25-gauge Whitacre needle in the L3–4 interspace.

Systolic arterial pressure decreased from 120 to 90 mm Hg and remained at this level for the duration of the procedure. Estimated intraoperative blood loss was 970 ml. A total of 3000 ml of crystalloid fluid and 1 u. of blood were infused. The immediate postoperative systolic arterial pressure was 90–95 mm Hg.

The patient developed a severe frontal headache 6 h after surgery. Initial conservative management included opioid analgesics. An epidural blood patch was administered 18 h after surgery because of persistence of the headache. Subsequently, the patient began to complain of blurred vision. Twenty-four hours after surgery it was noted that he had developed a partial right third nerve palsy. At this time, he was transferred to our neurosurgical department for further investigation.

On presentation to this institution, the patient had a complete right third nerve palsy, marked ptosis, a fixed dilated pupil and divergent strabismus. Visual acuity was limited to counting fingers on the left and perception of light on the right. He also had a bitemporal visual field defect. Systolic arterial pressure was 95/60 mm Hg and heart rate was 40–60 beat min\(^{-1}\). The electrocardiogram (ECG) showed a Mobitz type I secondary heart block. Haemoglobin concentration was 8.9 g litre\(^{-1}\) and he was markedly hyponatraemic (serum sodium concentration 122 mmol litre\(^{-1}\)) with normal serum potassium (3.8 mmol litre\(^{-1}\)).

A clinical diagnosis of pituitary apoplexy was made. Computed tomography (CT) revealed the presence of a pituitary tumour with enlargement of the gland into the cavernous sinus bilaterally, and suprasellar extension compressing the optic chiasma. An urgent right frontotemporal craniotomy and decompression of the pituitary adenoma was undertaken. Hydrocortisone 100 mg was administered i.v. before induction of anaesthesia to treat potential adrenal insufficiency. I.v. fluids and vasopressor support were required during the procedure to maintain systolic arterial pressure at 80–90 mm Hg. The procedure was otherwise uneventful.

Hyponatraemia (sodium 126 mmol litre\(^{-1}\)), plasma hypo-osmolarity (262 mmol litre\(^{-1}\)), oliguria and urinary hyperosmolarity (645 mmol litre\(^{-1}\)) complicated the early postoperative period. Spot urinary sodium was 71 mmol litre\(^{-1}\). Fluid and electrolyte imbalance was treated with supplemental corticosteroids and balanced salt i.v. administration. A diagnosis of panhypopituitarism was confirmed by reduced hypothalamic pituitary function tests.

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plasma concentrations of thyroid stimulating hormone (TSH) (0.1 mIU litre$^{-1}$), total tri-iodothyronine (T3) (<0.5 mmol litre$^{-1}$) and prolactin 17 mIU litre$^{-1}$; serum gonadotrophin concentrations were also low. Visual acuity was virtually normal despite residual partial third nerve palsy and a bitemporal visual field defect on discharge 11 days later.

Discussion

Headache, with an overall incidence of less than 1%, is a well documented complication of spinal anaesthesia since its first introduction by Bier in 1899.5 Rarely, more serious central neurological sequelae have been reported, including III, IV, VI, and VIIth cranial nerve palsies. Death resulting from bilateral subdural haematomas has been described after lumbar puncture.6,7 To the best of our knowledge, there have been no reports of pituitary apoplexy complicating spinal anaesthesia.

Pituitary adenomas represent 10–15% of all intracranial tumours. They are often clinically silent and have been found in up to 20–30% of all subjects in some autopsy series.8 Although 16.6% of all pituitary tumours removed surgically show histological evidence of infarction, only 6.8% of patients with pituitary adenomas develop the clinical manifestations of pituitary apoplexy. Thus it is a relatively rare complication.9 Most cases occur spontaneously and it is frequently the first manifestation of a pituitary adenoma, as occurred in our patient.

There were no abnormal neurological features in our patient before operation. The frontal headache that developed after operation was initially treated conservatively with routine analgesics. However, the patient failed to improve and in the absence of other neurological deficits at this time, a diagnosis of postdural puncture headache was made. Epidural blood patch has been reported to produce almost immediate relief in up to 89% of post-dural puncture headaches.10,11 With the onset of visual symptoms, other diagnoses considered included subarachnoid aneurysm haemorrhage, meningitis, pituitary apoplexy, midbrain infarction, encephalitis, cavernous sinus thrombosis, carotid artery aneurysm and parasellar tumour. A CT scan was the initial investigation and confirmed the presence of a large pituitary tumour with lateral extension into the cavernous sinus and suprasellar compression of the optic chiasma.

Pituitary apoplexy may follow sudden infarction or haemorrhage into the gland, which causes it to swell and compress adjacent structures. The clinical presentation of this patient was typical of fulminating pituitary apoplexy. The symptoms arise as a result of three processes: (1) leakage of blood or necrotic tissue into the subarachnoid space may be associated with headache, neck stiffness and photophobia; (2) pressure effects—superior extension of the pituitary gland may compress the optic chiasma resulting in loss of visual acuity and visual field defects. Hypothalamic dysfunction may be associated with alterations in temperature, respiration and cardiac function. This hypothalamic dysfunction may account for the transient Mobitz I secondary degree heart block noted in this case. Lateral extension of the gland may cause ocular palsies in up to 40% of cases as a result of compression of III, IV and VIIth cranial nerves in the cavernous sinus12; (3) endocrinopathy—acute adrenal insufficiency, observed in up to 66% of such cases, is the most important endocrine dysfunction after pituitary apoplexy.13 Intraoperative hypotension and hypothermia persisting into the postoperative period may have been manifestations of acute adrenal insufficiency in this case. Glucocorticoids are necessary for maintenance of pressor tone and glomerular filtration glucocorticoid insufficiency affects the ability of the renal tubules to excrete water. In addition, normal values for serum potassium were consistent with acute secondary adrenal insufficiency. Mineralocorticoid function may be unaffected because the renin–angiotensin–aldosterone axis is relatively independent of the ACTH–adrenal axis.14 The syndrome of inappropriate anti-diuretic hormone secretion was considered as an alternative cause but this has been reported rarely in association with pituitary apoplexy.15

Aetiological factors in the pathogenesis of pituitary apoplexy are not well understood. Elevated oestrogen states, bromocriptine therapy, irradiation and diabetes have been implicated,15–17 but the majority occur spontaneously without identifiable precipitating factors.15 It has been suggested that the adenomatous gland may “outstrip” its own blood supply at a variable critical time and produce ischaemic necrosis followed by haemorrhage into the gland.15

Intraoperative hypotension could be considered as an aetiological factor in our patient because systolic arterial pressure was reduced from 120 to 90 mm Hg after administration of spinal anaesthesia. There is some evidence to suggest that pituitary tumours may be particularly susceptible to changes in systemic arterial pressure. Bevan and colleagues18 described a case of pituitary apoplexy after administration of oral isosorbide mononitrate in which a patient developed severe headache within 90 min of ingesting the drug. A month later, severe headache, this time associated with persistent neurological deficits, followed attempts to restart drug administration. The patient was subsequently diagnosed as having pituitary apoplexy. The association of the vasodilator properties of the drug and the onset of apoplexy seemed likely.

Lever and colleagues19 reported on the onset of apoplexy after a thyrotrophin releasing factor (TRF) stimulation test and Bernstein, Hegel and Gentili20 also reported a case of apoplexy after a “triple bolus” test (a pituitary stimulation test using TRH, LHRH and insulin). Both attributed significance to the nor-epinephrine (noradrenaline) releasing properties of TRH which may induce vasospasm precipitating pituitary infarction.

The pituitary gland is supplied by end arteries which are poorly innervated and operate at a low hydrostatic pressure.21 The vessels within the tumour itself are abnormal. They are typically smaller, variable in number, poorly fenestrated and have irregular, ruptured basement membranes. In addition, the expanding mass may directly compress the infundibulum or the superior hypophyseal artery and impair blood supply to the entire anterior lobe. As a result of these anatomical features, an adenomatous pituitary is susceptible to infarction with relatively
minor changes in perfusion pressure. Low perfusion pressure was the suggested cause of apoplexy in the cases associated with cardiopulmonary bypass. Other considerations included embolization of platelet aggregates, atherosclerotic plaques or gas bubbles, and anticoagulation with inadequate reversal. Pituitary tumours are known to be susceptible to haemorrhage, observed in approximately 17% of adenomas. This is a variable finding in apoplexy and was not present in our patient.

Another, although perhaps less likely, mechanism of pituitary apoplexy was a reduction in CSF pressure secondary to CSF leak. Minor alterations in pressure gradients within the skull have been associated with the onset of apoplexy. These include lumbar puncture for pneumoencephalography, repetitive coughing and sneezing secondary to upper respiratory tract infections, minor head trauma, mechanical ventilation and scuba diving.

In summary, we believe this is the first case report of pituitary apoplexy complicating spinal anaesthesia. Pituitary adenomas are frequently asymptomatic and are thus difficult to diagnose before operation. However, this case highlights the importance of prompt assessment of patients with postdural puncture headache and clinicians should consider the diagnosis of pituitary apoplexy when focal neurological deficits are also present.

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References