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Review

Surgical treatment of pituitary apoplexy in association with hemispheric infarction

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ABSTRACT

We report a patient with pituitary apoplexy in whom cerebral infarction developed, possibly secondary to vasospasm. Pituitary apoplexy is a clinical syndrome caused by acute hemorrhage or infarction of the pituitary gland. Our patient's clinical symptoms and radiographic findings greatly improved after surgical resection of the apoplectic pituitary gland. An extensive literature review was performed, including all previously reported cases of pituitary apoplexy leading to cerebral infarction. The clinical features, pathophysiological mechanisms, management and outcome of cerebral infarction following pituitary apoplexy are discussed. We show that cerebral infarction following pituitary apoplexy is associated with much poor prognosis. Early surgical decompression of the tumor and hemisphere should be performed in patients with severe or progressive neurological deficits, however, those with less severe presentations may be treated conservatively or with delayed elective surgery.

1. Introduction

Pituitary apoplexy (PA) is a clinical syndrome caused by acute hemorrhage or infarction of the pituitary gland, and is characterized by the sudden onset of severe headache, visual impairment, ophthalmoplegia and altered mental status. The incidence of apoplexy in patients with pituitary adenoma is approximately 2–7% [1]. Cerebral infarction is a rare complication of PA, and only isolated patients have been reported. In this report, we describe a patient with PA who developed ischemic stroke and improved significantly after pituitary and hemisphere decompressive surgery. We also review similar patients reported in the literature and summarize their clinical characteristics.

2. Case report

A 23-year-old man presented with severe headache, nausea, fever and decreased visual acuity of his right eye for 1 day, followed by a sudden loss of consciousness. He had no significant previous medical history. On examination, his blood pressure was 130/80 mmHg, and body temperature was 37.1 °C. Signs of acromegaly, including frontal bossing and hand enlargement were identified, and neck stiffness was present. Spontaneous and reflex movements to painful stimuli were present in his right limbs but absent on the left side. The left Babinski's sign was present. Emergency brain CT scans showed an enlarged pituitary fossa containing a hemorrhagic mass within the pituitary gland (Fig. 1A). Brain MRI showed a giant sellar and supersellar mass demonstrating heterogeneous hyperintensity on both T1- and T2-weighted images, indicative of hemorrhage (Fig. 1B). Hormonal tests revealed hypopituitarism and a high level of growth hormone (GH), supporting the diagnosis of a pituitary adenoma.

In addition, MRI with fluid attenuated inversion recovery and diffusion-weighted images showed a large stroke involving the right frontal, parietal and temporal lobes, and the right basal ganglia, corresponding to the territories of the right middle cerebral artery (MCA) and the artery of Heubner branch of the right anterior cerebral artery (ACA; Fig. 1C, D). Magnetic resonance angiography (MRA) of the brain showed decreased diameters of the right internal carotid artery (ICA) trunk in the cavernous portion, the M1 segment of the right MCA and the A1 segment of the right ACA. Transcranial Doppler (TCD) sonography demonstrated an increase

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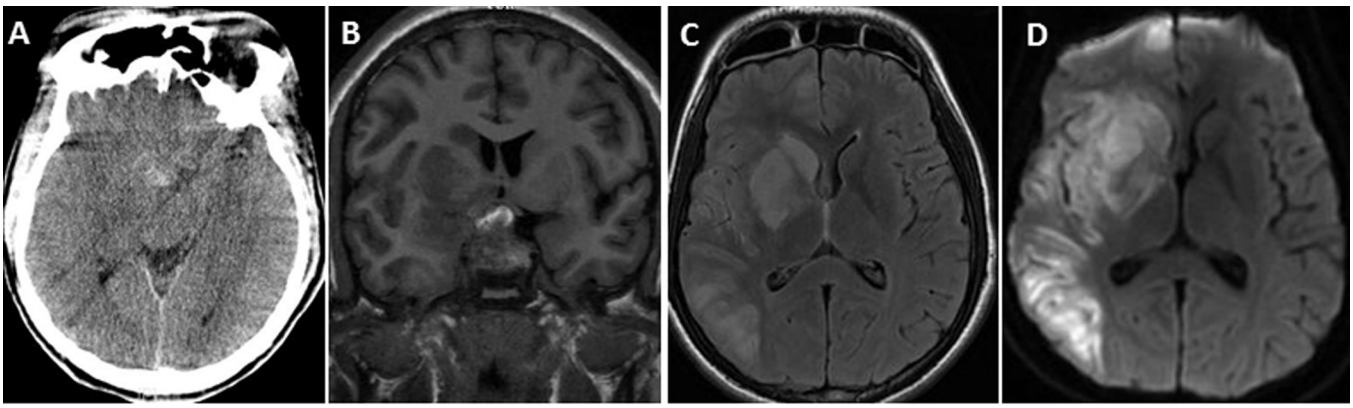

Fig. 1. Preoperative imaging of our patient: (A) axial brain CT scan showed an enlarged pituitary fossa containing a hemorrhagic mass within the pituitary gland; (B) T1-weighted coronal MRI showed a giant, partially hyperintense pituitary adenoma extending to the suprasellar. The tumor invaded the bilateral cavernous sinus, causing compression of the optic chiasm and intracavernous internal carotid artery (ICA) on the right side; (C) fluid-attenuated inversion recovery and (D) diffusion-weighted axial MRI showed hyperintense signal at the right frontal, parietal and temporal lobes, the right basal ganglia, corresponding to the territories of the right middle cerebral artery (MCA) and the artery of Heubner branch of the right anterior cerebral artery (ACA).

of the mean and peak flow velocities in the right ACA and MCA consistent with vasospasm (Supp. Fig. 1A).

The patient was initially treated with fluid replacement, intravenous hydrocortisone and mannitol. His mental status improved but the weakness in his left limbs and poor vision in the right eye persisted 5 days into his admission. A right fronto-temporal craniectomy for sellar and suprasellar tumor resection was performed 7 days after admission. The right visual acuity improved to finger counting, and the muscle strength in the left limbs improved to grade 3 (Medical Research Council scale 0–5) on postoperative day 1. Histological examination of the surgical specimen revealed diffused necrosis and hemorrhage in a pituitary adenoma, and immunostaining was positive for GH, compatible with the diagnosis of an apoplectic GH-secreting adenoma.

The brain MRI, 1 week after surgery, showed a significant reduction of signal abnormality in the right MCA and ACA territory (Fig. 2A–C). On MRA, the calibers of right MCA, ACA and ICA had all improved. TCD sonography demonstrated that the blood flow velocities of the right ACA and MCA had returned to normal (Supp. Fig. 1B). The GH level had also returned to the normal range. One and a half months after surgery, the muscle strength on his left side normalized to grade 5 and his right visual acuity improved to 20/40.

3. Review

3.1. Methods

We conducted a literature search in both MedLine (National Library of Medicine, Bethesda, MD, USA) and EMBASE (Elsevier, Amsterdam, The Netherlands) using the following keywords: "pituitary adenoma" OR "pituitary apoplexy", AND "stroke" OR "cerebral infarct/infarction" OR "cerebral ischemia/ischaemia" OR "cerebral vasospasm" OR "cerebrovascular accident". Only English language literature was included in the review. A PA and cerebral infarction diagnosis must have been based on the acute onset of neurological deficits plus CT scans and/or MRI, digital subtraction angiography (DSA), or autopsy. We extracted the following information from the relevant papers: first author, year of study, sex, age, precipitating factors, clinical features, imaging findings, mechanism of stroke, treatment, pathological findings, and outcome.

3.2. Results

Overall, 35 patients with cerebral infarction associated with PA were identified. The main clinical features of the 36 patients, including the present study, are summarized in Supplementary Table 1. The average age was 45.2 years (range: 6–81). Thirty patients were male (83%). Only three patients (8%) had a known diagnosis of pituitary adenoma before the episode of acute apoplexy. Twelve patients (33%) had symptoms related to pituitary adenoma; headache (22%) and visual disturbance (18%) were the most common. Other symptoms included amenorrhea, sterility, photophobia, ptosis, change in appearance, mental confusion, and memory deficit. The precipitating factors for PA were identified in eight patients and included angiography, head trauma, pituitary surgery, endocrine stimulation tests, high fever, and anticoagulant therapy (22%; Table 1).

The most common symptoms of PA were decreased consciousness (97%), signs of intracranial hypertension such as acute severe headache or vomiting (86%), motor deficit (75%), visual disturbance such as decreased visual acuity and visual field defect (72%), ophthalmoplegia due to palsy of III, IV, or VI cranial nerves (44%), and meningismus (44%; Table 1).

CT scans and/or MRI were carried out in 28 of 36 patients (78%), MRA in 13 (36%), and DSA in 11 (31%). PA was identified by CT scans in 28 of 36 patients (78%), and by pathological findings in the other eight patients (22%). Cerebral infarction was confirmed by CT scans and/or MRI in 26 of 36 patients (72%), by DSA in eight (22%), and by autopsy in two (6%; Supp. Table 1). Intracranial vessel compression was visualized in 20 patients (56%), cerebral vasospasm in eight (22%), and subarachnoid hemorrhage (SAH) in five (14%). Cerebral infarction was unilateral in 24 (67%) and bilateral in 12 patients (33%). Twenty-four patients (67%) had non-watershed cerebral infarctions, five (14%) had watershed infarctions. By computed tomography angiography (CTA), MRA, angiography, or autopsy, ICA compromise was demonstrated in 20 patients (56%), ACA involvement in 14 (39%), and MCA involvement in 11 (31%; Supp. Table 1).

Mechanical compression of the intracranial vessels was the pathophysiological mechanism of PA-related cerebral infarction in 22 patients (61%). Cerebral vasospasm and hypotension were thought to be the mechanism for cerebral infarction in 15 (42%) and three patients (8%), respectively.

Neurological deficits completely recovered in two patients (6%) after treatment, greatly improved in 14 (39%), and moderately