CASE REPORT

Massive Cerebral Infarction as a Feature of Pituitary Apoplexy

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Summary

A patient with pituitary apoplexy is reported who, in addition to the clinical features of apoplexy, developed a cerebral infarct secondary to compression of the internal carotid artery. The mechanisms of a cerebral infarct associated with pituitary apoplexy are discussed.

Key words : Cerebrovascular accident, Internal carotid artery, Pituitary tumour.

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Introduction

Pituitary apoplexy is a clinical syndrome characterised by sudden onset of headache, signs of meningeal irritation, visual impairment and ophthalmoplegia caused by enlargement of a pituitary adenoma, due to tumour infarction or haemorrhage.[1] The incidence of apoplexy in pituitary adenomas has been reported to be 0.6-10%.[1,2] We report a patient with pituitary apoplexy, who in addition to the above clinical features, developed a massive cerebral infarct due to direct compression of the internal carotid artery caused by the sudden enlargement of a pituitary tumour.

Case Report

A 40 year old male presented with history of sudden onset of bifrontal headache, vomiting and rapid deterioration of vision, first in the right eye followed by the left eye, 24 hours prior to admission at our hospital. He developed total loss of vision and altered sensorium within 12 hours of the onset of the symptoms. He was confused, obeying commands and attempting to open eyes to call. He had bilateral ptosis. There was pupillary asymmetry with the right pupil measuring 2.5 mm and left pupil 2 mm, reacting sluggishly to light. There was a total external ophthalmoplegia on the right side and all eye movements were restricted on the left side. He had a left upper motor neuron 7th nerve paresis and left hemiparesis. Neck stiffness and Kernig's sign were present. A plain CT scan showed a hyperdense sellar mass with suprasellar and right parasellar extension (Fig 1). Contrast CT showed mild uptake of the contrast by the tumour (Fig. 2). There was hypodensity seen in the right anterior cerebral artery territory. Following admission, the left hemiparesis progressed to a left hemiplegia within two hours. He was started on steroids and underwent an emergency

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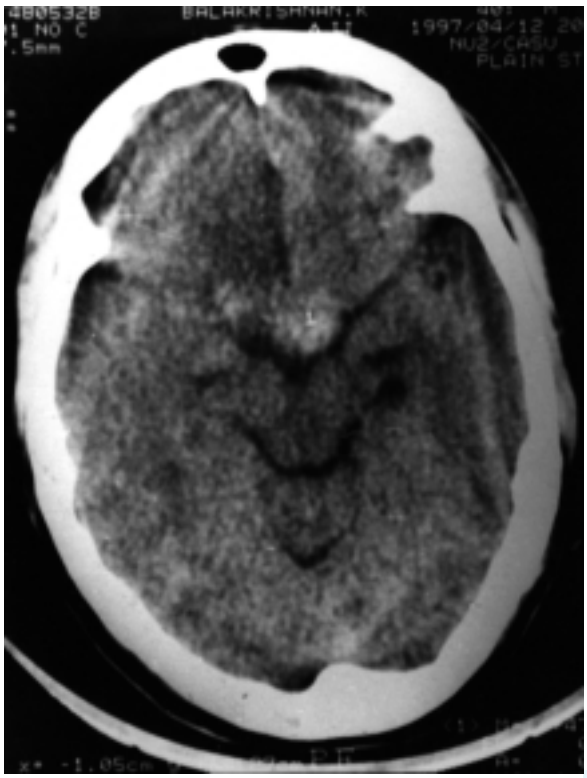

Fig. 1 : Plain CT scan showing blood within the pituitary adenoma and extension into the right parasellar region.

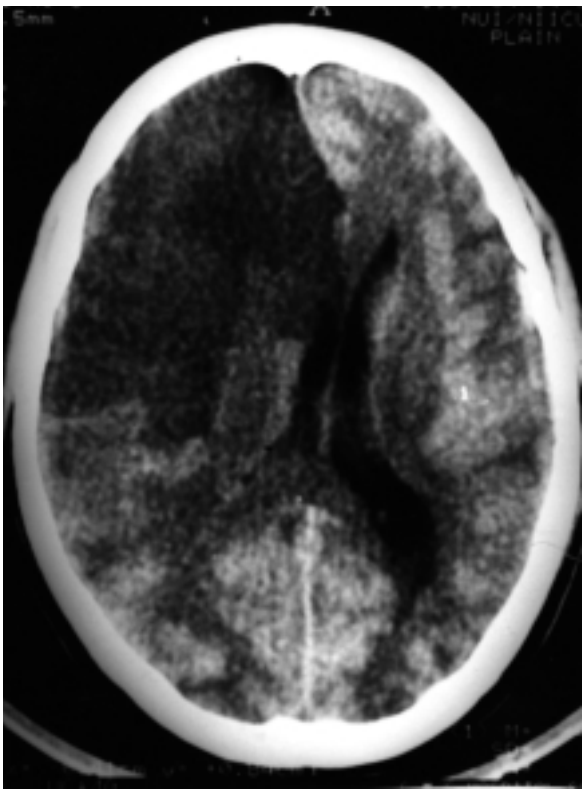

Fig. 3 : Postoperative CT showing massive cerebral infarct in the right internal carotid artery territory.

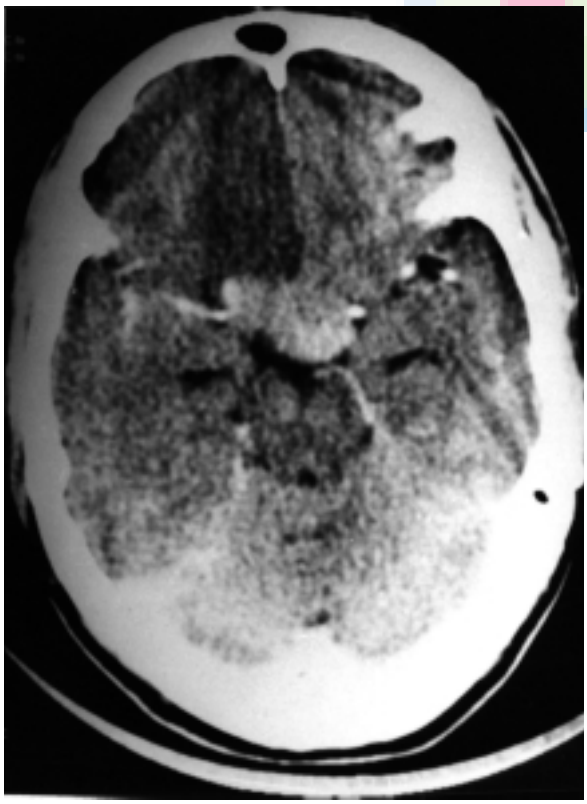

Fig. 2 : Contrast CT showing hypodensity in the right anterior cerebral artery territory.

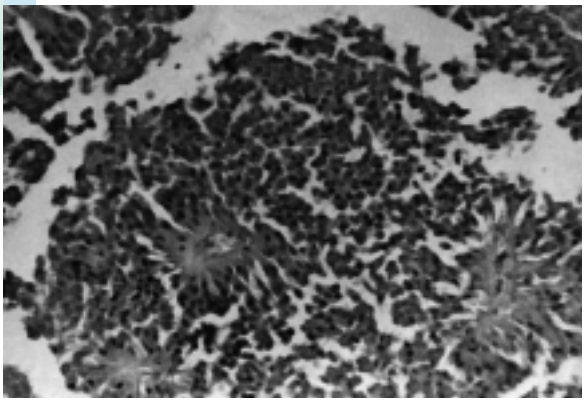

Fig. 4 : Photomicrograph of the tumour showing necrotic outlines of tumour cells in a pituitary adenoma. (H & E x 200).

transnasal transsphenoidal excision of the tumour. At surgery, the tumour was necrotic and was excised except for a small portion invading the right cavernous sinus. Following surgery, the patient was electively ventilated and a repeat CT scan (Fig. 3) showed a large infarct in the right internal carotid artery territory with mass effect and midline shift. He developed signs of progressive brainstem dysfunction and diabetes insipidus, which was treated with pitressin injections. He died on the second post operative day. The biopsy showed a pituitary adenoma with necrosis (Fig. 4). Autopsy showed an infarct in

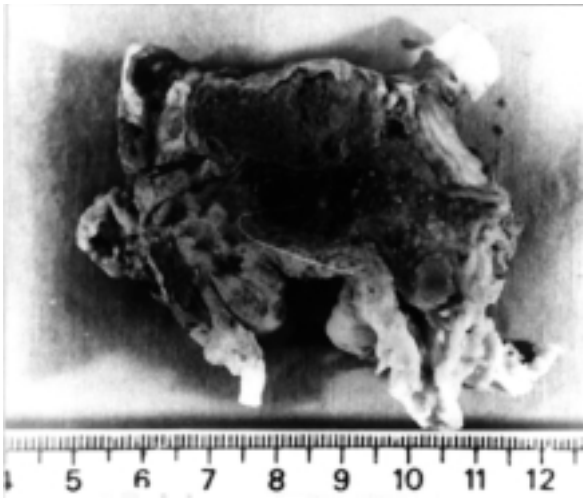

Fig. 5 : Coronal section through the sellar and parasellar region of the autopsy specimen. Note the extension of the tumour into the right cavernous sinus with compression and flattening of the internal carotid artery (arrow).

the right internal carotid artery territory. Coronal sections through the sella and parasellar region showed evidence of compression of the right intracavernous internal carotid artery by the tumour (Fig. 5).

Discussion

Pituitary apoplexy can cause narrowing of intracranial vessels by either mechanical obstruction due to an enlarged suprasellar mass[3] or by vasospasm.[4] Six cases of pituitary apoplexy resulting in direct compression of intracranial vessels have previously been reported in literature.[3,5-9] In most of these cases the internal carotid artery, either in the cavernous sinus or in the supraclinoid portion was occluded by the enlarged tumour. In one case, the middle cerebral artery[5] and in another the anterior cerebral artery[7] were found to be occluded. In two patients, the internal carotid arteries were compressed bilaterally to varying degrees.[8,9] Mukherjee et al[10] reported a patient who had a stroke and pituitary apoplexy, and hypothesised that ischaemia in the internal carotid artery was the primary event leading to infarction in the pituitary macroadenoma. It is likely that the sequence of events was the other way around, i.e. the pituitary apoplexy occurred first, leading to internal carotid artery stroke.

Surgical decompression may or may not restore flow in the occluded vessel. Clark et al[9] cautioned that decompression of the vessel might, in fact, be harmful in the presence of an established infarct on CT scan, by converting it into a haemorrhagic one. Most authors have advocated early surgery, although, even with early decompression mortality may be high. Four of the six patients mentioned above were operated upon, three via a transcranial approach[3,6,7] and one via the transsphenoidal approach.[8] Three of these[6-8] had good outcomes while one died.[3] Of the non-operated patients, one died[5] and the other improved[9] with conservative management.

In conclusion, stroke caused by occlusion of the arteries in the circle of Willis or cavernous segment of the internal carotid artery by a pituitary adenoma is a rare event. Early surgical decompression through the transsphenoidal route is advisable. The mass effect produced by the infarct and oedematous brain is usually the cause of mortality in these cases as it was in our patient.

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