

# Case Report: Pituitary Apoplexy: An Unusual Cause of Stroke

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**A case of pituitary apoplexy complicated by hemiparesis and dysphasia is reported. Investigations revealed complete obstruction of the left internal carotid artery due to haemorrhagic pituitary expansion. This case and the four previously reported suggest that early diagnosis of this rare complication is essential if surgery is to prevent cerebral infarction.**

Pituitary apoplexy is an important, well-recognised and treatable complication of pituitary macro-adenoma presenting typically with a sudden onset of headache and vomiting, often with visual loss, extra-ocular palsies and a depressed level of consciousness, (Wakai *et al.*, 1981). Focal signs in the limbs suggesting a hemisphere abnormality occur only rarely and are usually attributed to hypotension or arterial spasm consequent upon blood in the sub-arachnoid space.

We report a potentially correctable complication of pituitary apoplexy in which haemorrhagic expansion of the pituitary resulted in occlusion of the left carotid artery with consequent dysphasia and right hemiparesis.

## CASE REPORT

A previously fit 40-year-old man was admitted following the sudden onset of severe frontal headache, photophobia and vomiting. Although initial examination revealed no abnormality, 1 hour later he became suddenly obtunded, with dysphasia, a right homonymous hemianopia, a right upper motor neuron facial palsy and a dense right hemiplegia. Computed tomography of the brain, performed 16 h after presentation, showed an enlarged pituitary fossa containing a haemorrhagic pituitary tumour (Fig. 1) with supra-sellar extension. In addition there was a large, non-enhancing, low attenuation area extending posteriorly from the region of the left frontal lobe (Fig. 2), with a further low attenuation area in the head of the caudate nucleus. These findings were compatible with pituitary apoplexy and cerebral infarction in the territories of the left anterior cerebral artery and the lenticulo-striate branches of the left middle cerebral artery. Three vessel angiography, with hydrocortisone cover, revealed complete occlusion of the supra-clinoid portion of the left internal carotid artery and marked narrowing of the supra-clinoid segment of the right internal carotid artery. The discrete nature of the narrowing, adjacent to the known tumour, was more suggestive of encasement rather than spasm or atheroma (Fig. 3). The anterior cerebral arteries were elevated bilaterally due to the supra-sellar extension of the pituitary. There was spontaneous cross flow from right to left and via the posterior communicating arteries. The left common carotid bifurcation was normal but there was marked stasis in the internal carotid artery with no filling of the intracranial component.

Although during the next 24 h the patient's conscious level rapidly improved the focal neurological signs persisted. However, it was thought that surgical decompression of the pituitary fossa with establishment of flow in the left internal carotid artery might precipitate haemorrhage into the extensive area of cerebral infarction, as previously described by Rosenbaum *et al.* (1977). The management, therefore, was mainly conservative with radiotherapy to the pituitary tumour and endocrine replacement therapy.

A further computed tomography examination after 3 months con-

firmed infarction in the medial aspect of the left frontal lobe and in the head of the caudate nucleus, but the pituitary tumour by now had involuted into the pituitary fossa (Fig. 4).

## DISCUSSION

The occurrence of hemiplegia in patients with pituitary apoplexy is extremely unusual and in eight previously reported cases, reviewed by Rosenbaum *et al.* (1977), possible aetiological factors included hypotension, cerebral embolisation complicating carotid arterial puncture and arterial spasm caused by blood in the sub-arachnoid space. In this case there was no hypotension, the infarct had occurred before angiography and there was no evidence of sub-arachnoid blood or associated arterial disease.

Mechanical obstruction of the intra-cerebral arteries by pituitary expansion, with consequent hemiplegia, has been previously described in only four cases (Schnitker and Lehnert, 1952; Rosenbaum *et al.*, 1977; Majchrzak *et al.*, 1983; Bernstein *et al.*, 1984). However, the management of such patients is important since, by emergency decompression of the pituitary fossa, flow in the internal carotid artery may be restored and cerebral tissue preserved. Schnitker and Lehnert (1952) described a patient known to have a pituitary adenoma who died 16 days after developing pituitary apoplexy with focal hemisphere signs. Autopsy revealed a large haemorrhagic pituitary tumor causing compression of the right middle and anterior cerebral arteries with associated encephalomalacia. A further patient, reported by Rosenbaum *et al.* (1977), developed pituitary apoplexy with a left hemiplegia 12 h after bilateral common carotid angiography. Repeated angiography confirmed occlusion of the right internal carotid artery due to expansion of the pituitary tumour. Although the pituitary fossa was decompressed by surgical intervention the patient deteriorated and died 2 days later from haemorrhagic cerebral infarction, probably consequent upon revascularisation of the previously infarcted area. Majchrzak *et al.* (1983) described a 29-year-old man presenting with a severe headache, visual loss and transient left hemiparesis in whom computed tomography revealed a haemorrhagic pituitary tumour. In addition there was an ischaemic area in the distribution of the right anterior cerebral artery, which was shown by subsequent angiography to be completely occluded. Decompressive surgery performed 3 weeks after admission resulted in improved visual acuity but flow was not re-established in this artery. The transient nature of the neurological signs was thought to reflect good collateral circulation. Bernstein *et al.* (1984) reported a case in which a left hemiplegia followed pituitary apoplexy, apparently caused by a pre-operative triple bolus test with thyrotropin releasing hormone, luteinising hormone releasing-hormone and insulin. Angiography

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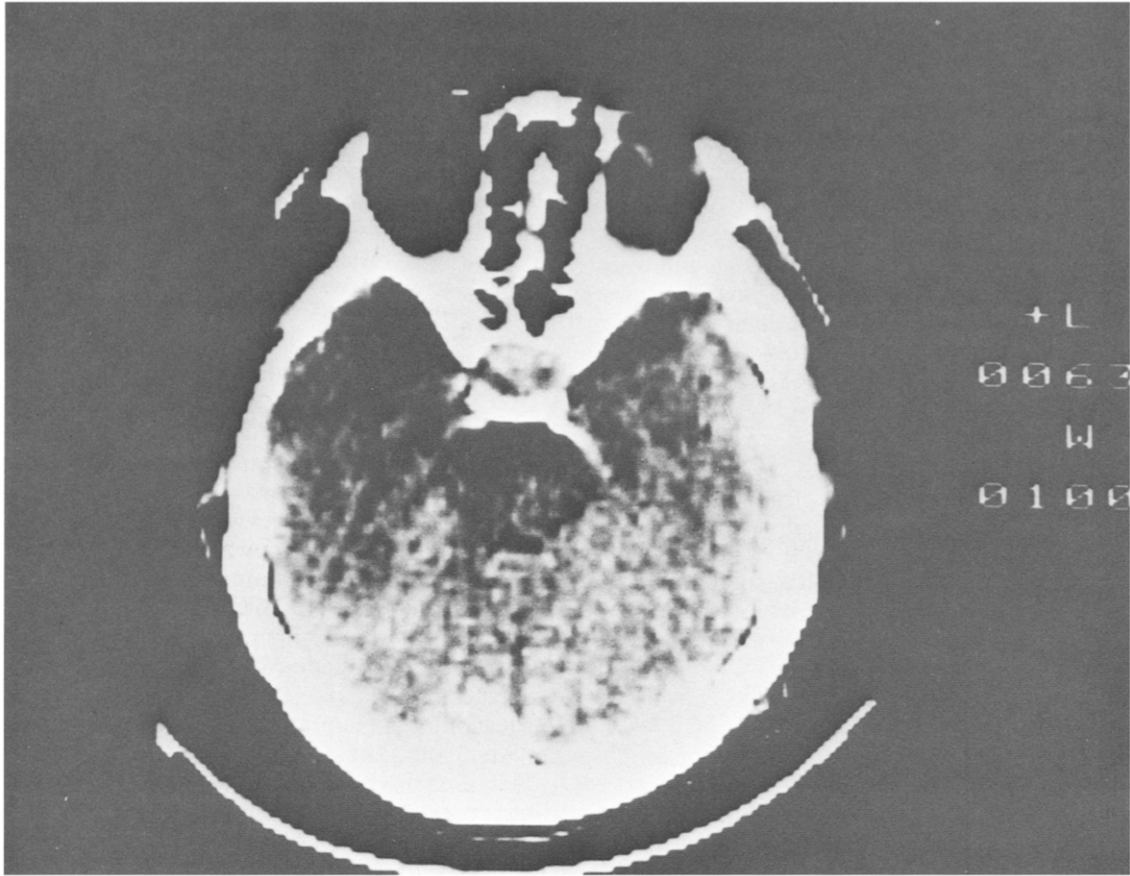


Fig. 1 - Initial unenhanced CT scan showing a pituitary tumour of mixed density compatible with pituitary apoplexy (L 63, W 100).



Fig. 2 - Initial CT scan showing a non-enhancing low density area in the territory of the left anterior cerebral artery (L 63, W 100).

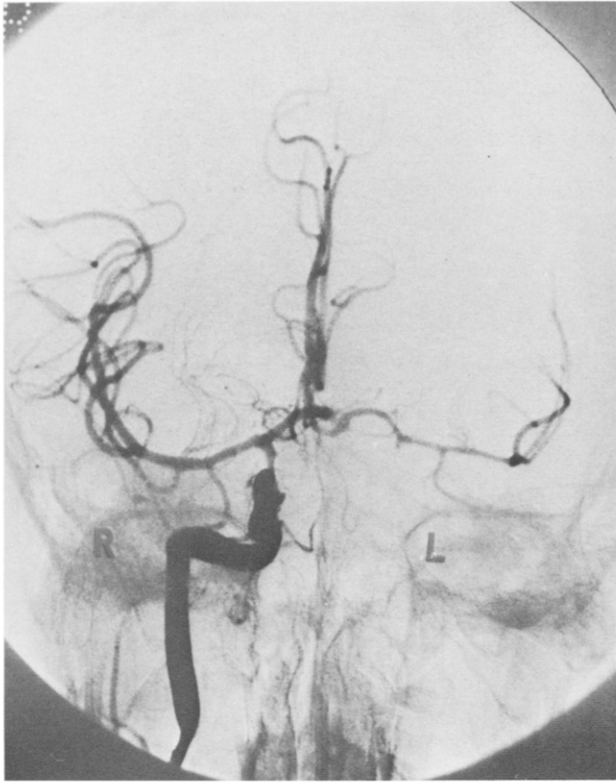


Fig. 3 - Right internal carotid angiogram (subtraction film) demonstrating severe narrowing of the supraclinoid portion of the right internal carotid artery, good crossflow from right to left and elevation of both anterior cerebral arteries.

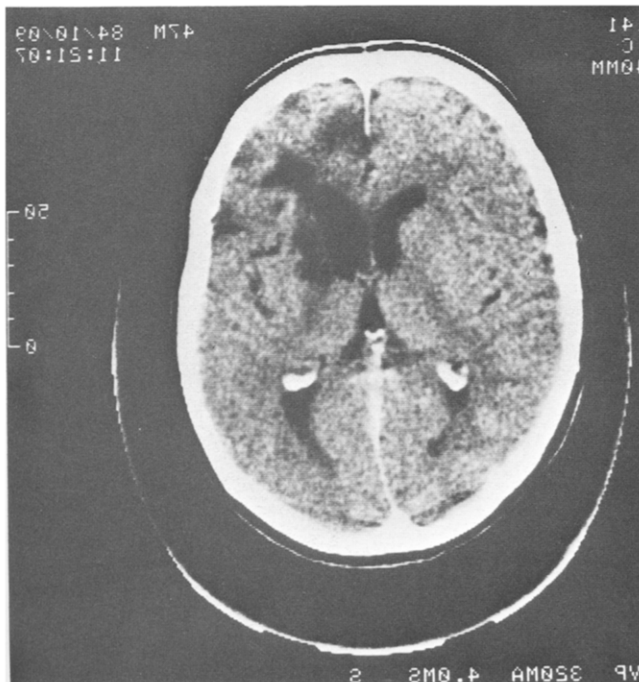


Fig. 4 - Enhanced CT scan 3 months after pituitary apoplexy. This shows extensive infarction in the left frontal lobe (L 40, W 100).

showed bilateral internal carotid artery occlusion but following immediate decompression of the pituitary fossa, internal carotid artery patency was restored and all neurological signs resolved.

The case reported here in which hemisphere infarction occurred due to internal carotid artery occlusion, following haemorrhagic infarction of the pituitary, raises several important clinical points with respect to diagnosis and treatment. The diagnosis of pituitary apoplexy, especially where there is a known tumour, is usually clear. However, when the presenting features are suggestive of subarachnoid haemorrhage or a stroke, the underlying diagnosis may be overlooked and it is necessary to consider pituitary apoplexy in these two groups of patients. When hemiplegia complicates apoplexy the early management is critical to prevent permanent neurological sequelae due to potentially reversible arterial compression. Extensive studies in patients with transient ischaemic attacks due to carotid artery stenosis have shown the safety of revascularisation procedures in patients without cerebral infarction (Chater and Popp, 1976; Gratzel *et al.*, 1976). However, surgical treatment of complete occlusion of the carotid artery is usually considered a hazardous procedure carrying a high risk of precipitating cerebral haemorrhage into ischaemic or infarcted brain tissue (Gonzalez and Lewis, 1966; Sundt *et al.*, 1975). Thus, we suggest that if, as in our patient, the computed tomography scan at the time of diagnosis already demonstrates cerebral infarction, surgical decompression may be dangerous and conservative management more appropriate. However if, as in the case of Bernstein *et al.* (1984), there is cerebral ischaemia rather than infarction, early operative management to restore carotid artery flow may result in resolution of the neurological deficits.

Further case reports are required to clarify the appropriate management of this rare but potentially correctable disorder.

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