Serious Complication of Pituitary Apoplexy

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ABSTRACT
A 50-year-old man suddenly collapsed at home following an acute episode of severe headache. At admission, his consciousness level was 4/15 on the Glasgow Coma Scale. Urgent computed tomography of the brain showed a large enhancing suprasellar mass. Follow-up computed tomography scan showed massive bifrontal and left inferior basal ganglion infarctions. Magnetic resonance imaging of the brain and pituitary gland confirmed the diagnosis of pituitary apoplexy and infarctions. The mass effect from the infarctions was not controlled by external drainage catheter alone and decompressive bifrontal craniectomy was required. Trans-sphenoidal debulking of the pituitary tumour was performed. The patient survived, but had severe disability. The mechanisms of cerebral infarct associated with pituitary apoplexy and the possible treatments are discussed.

Key Words: Cerebral infarction; Pituitary apoplexy

INTRODUCTION
Pituitary apoplexy is a clinical syndrome characterised by sudden onset of headache, signs of meningeal irritation, visual impairment, ophthalmoplegia, hormonal dysfunction, disturbance of consciousness, and sudden death. The condition is usually caused by sudden enlargement of a pituitary adenoma due to haemorrhagic infarction. Occasionally, pituitary apoplexy could result from haemorrhage within a non-adenomatous tumour or even within a normal gland. The incidence of pituitary apoplexy ranges from 0.6% to 16.6%.1,2 Asymptomatic haemorrhage or infarction may account for 14.0% to 22.0% of pituitary tumours, while clinical apoplexy is less common and accounts for 0.6% to 9.0%.3 Massive cerebral infarction is a rare life-threatening complication. This report describes a patient with this complication.

CASE REPORT
A 50-year-old man presented to the emergency department at the Prince of Wales Hospital, Hong Kong, in 2008 with a Glasgow Coma Scale score of 4/15. He was immediately intubated and mechanically ventilated. Urgent computed tomography (CT) of the brain showed a large well-defined round sellar mass with suprasellar extension and large right cavernous invasion (Figures 1, 2, and 3).

Figure 1. Axial non-contrast computed tomography scan of the brain on admission showing a large well-defined pituitary mass with suprasellar extension.

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The cavernous segments of the internal cerebral arteries were not seen on the vascular-phase contrast images (Figure 3). There was some pushing on a small precommunicating segment of the left anterior cerebral artery, which was patent (Figure 2). The vertebral basilar system and both posterior communicating arteries were patent. No vascular spasm was seen in the anterior, middle, or posterior cerebral arteries. No subarachnoid or intracerebral haemorrhage was evident. The patient was admitted to the intensive care unit.

Follow-up CT scan done the next day revealed a massive cerebral infarction symmetrically involving both frontal lobes and part of the left basal ganglion, where there was a small area of haemorrhage (Figure 4). Magnetic resonance imaging (MRI) of the brain showed subacute blood and haemosiderin deposition within a pituitary tumour, indicating different ages for the blood
Complication of Pituitary Apoplexy

products (Figures 5 and 6). A diagnosis of pituitary apoplexy was made. The cavernous segments of the internal cerebral arteries could not be seen on time-of-flight magnetic resonance angiogram (Figure 7). The stretching of the precommunicating segment of the left anterior cerebral artery was still apparent. Haemorrhagic foci were also seen within the infarcted areas.

External ventricular drainage was done after admission to hospital to control the raised intracranial pressure. As the intracranial pressure remained persistently elevated and uncontrolled, bifrontal decompressive craniectomy was performed (Figure 8). Trans-sphenoidal excision of the haemorrhagic pituitary tumour was performed on day 3 and histology confirmed an infarcted pituitary adenoma, consistent with apoplexy. The patient survived, but was severely disabled.

DISCUSSION

Only a few patients with pituitary apoplexy associated with cerebral infarction have been reported in the English-language literature. Six patients had narrowing of the intracranial vessels by mechanical compression from a mass, while 2 patients had narrowing of the vessels due to vasospasm. In most of the patients with compression, the narrowing was in the internal carotid artery, either in the cavernous segment or in the supraclinoid portion. In one patient, the middle cerebral artery was occluded and, in another, the anterior cerebral artery was occluded. In 2 patients, the internal carotid arteries were compressed bilaterally to varying degrees. Vasospasm can be induced by subarachnoid haemorrhage or by some chemical vasoactive substances liberated from the process of pituitary apoplexy. In this patient, the reason was bilateral internal carotid compression at the cavernous segment with the possibility of a contribution from stretching of the proximal left anterior cerebral artery. Ischaemia from the stagnation of flow or thromboembolism were both possible mechanisms.

Surgical decompression may or may not restore flow to the compressed and occluded vessel. Clark et al cautioned that decompression of the vessel can be harmful in the presence of an established infarct, as a
result of reperfusion injury and conversion into a space-occupying haemorrhage. There was reperfusion haemorrhage in this patient, but it was not space occupying, and did not cause the clinical deterioration. Four of the 6 patients described in the literature underwent operation; 3 via a transcranial approach and 1 via a transsphenoidal approach. Three patients had good outcomes, but 1 died. Of the non-operated patients, 1 died and the other improved with conservative management. From this limited number of patients, early surgery for patients with vascular compression seems beneficial. Even for patients with vascular spasm, early surgery may decrease the intratumoural pressure that causes subarachnoid leakage of blood or chemicals. Medical treatment with bromocriptin and corticosteroids may also shrink the tumour or stabilise the condition. If vascular spasm due to subarachnoid bleeding is the cause, treatments such as hypertensive-hypervolaemic-haemodilution therapy, oral or intravascular antispasmodics, and early mechanical intracranial angioplasty may be life saving. However, these hypotheses need further study.

In conclusion, massive ischaemic stroke caused by pituitary apoplexy is a rare event. Intensive monitoring and supportive management is required, but early intervention could be more beneficial. A controlled trial would be challenging because of the rarity of the situation and the multiplicity of the clinical condition and cause of the infarct. The logical approach is to decompress the mass as early as possible if there is mechanical vascular compression. For patients with vascular spasm, the use of mechanical decompression or institution of anti-spasmodic therapy is possible.

REFERENCES