

Pituitary Apoplexy: A Rare Cause of Middle Cerebral Artery Infarction

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SUMMARY

Pituitary apoplexy is a well-known complication of a pituitary adenoma. However, an ischaemic event caused by pituitary apoplexy is rare. We reported a case of pituitary apoplexy with middle cerebral artery infarction in a 44-year old man who presented with a sudden onset of altered sensorium. Vasospasm is the most likely underlying cause of the infarction in this case secondary to trans-diaphragmatic rupture of the sella tumour into the subarachnoid space.

KEY WORDS:

Pituitary apoplexy, stroke, middle cerebral artery infarction, pituitary adenoma, tumours

INTRODUCTION

Pituitary apoplexy is a life threatening condition caused by sudden haemorrhage or infarction of the pituitary gland and characterized by severe headache, neuro-ophthalmological symptoms and signs, and altered mental status¹. Cerebral infarction associated with pituitary apoplexy is a rare occurrence. We reported a case of pituitary apoplexy with middle cerebral artery territory (MCA) infarction in a 44-years old man presented with altered consciousness.

CASE REPORT

A 44-year old man who had no underlying medical or surgical illness was found unconscious at home. He complained of severe headache with few episodes of vomiting one day prior to admission. There was no history of trauma, fever or fitting episode. On assessment, there was no eye opening (E1), no verbal response (V1) and the patient was only localizing pain (M5) giving a Glasgow Coma Scale of E1V1M5 which was a total of 7. This indicated severe brain injury. Vital signs were normal. The right pupil was reactive to light but the left pupil was dilated with sluggish reaction to light. Left side of the body did not move and hypotonic. He was intubated and admitted to intensive care unit. Computed tomography of the brain and subsequent magnetic resonance imaging (MRI) revealed a large pituitary tumour with haemorrhagic components within it (Figure 1). There was minimal subarachnoid haemorrhage on the ipsilateral brain and an acute infarction involving the right MCA territory. The right internal carotid artery (ICA) was

compressed by the mass but maintains its flow with good opacification of its distal branches (Figure 2). Emergency craniotomy, decompression and excision of tumour were done. Pathological examination confirmed apoplexy of a pituitary adenoma. The patient had a poor post-operative recovery and succumbed five days later. management.

DISCUSSION

Pituitary apoplexy is a rare medical emergency characterized by sudden onset of headache, vomiting, visual impairment and decreased consciousness caused by haemorrhage and/or infarction of the pituitary gland¹. It can occur in both adenomatous and nonadenomatous pituitary glands². Pituitary adenomas are particularly prone to haemorrhage and necrosis with histological evidence of infarction seen in 17% of the cases, but only 7% were symptomatic³. It is important to note that pituitary apoplexy does not occur only in patients with large macroadenoma but in tumours of any size. Most importantly, most cases (80%) of pituitary apoplexy occur in patients who have as of yet undiagnosed pituitary adenomas; with the apoplectic episodes often the presenting symptoms of the pituitary tumour as in our patient².

The occurrence of ischaemic events in patients with pituitary apoplexy is rare⁴. The attributed factors are cerebral vasospasm and mechanical compression. The pathophysiology of vasospasm following pituitary apoplexy remains unclear. Several hypotheses have been proposed to account for vasospasm such as vasospasm of the vessel from extravasated blood into the subarachnoid space and/or possible interaction of vasoactive agent released from the pituitary tumour in the hypothalamo-hypophyseal area⁴.

In cases where ischaemic events were attributed to the mechanical compression of the cerebral artery by pituitary apoplexy, the occlusion sites of the compromised vessels were primarily located in the supra-clinoid or cavernous portion due to its local vascular anatomy⁴. In these cases, the internal carotid artery (ICA) and anterior cerebral artery (ACA) are commonly affected due to mechanical compression of these vessels at the 'Circle of Willis' by rapid tumour expansion due to haemorrhage or infarction. The middle cerebral artery (MCA), not being part of the closed loop systems is usually not affected by this phenomenon.

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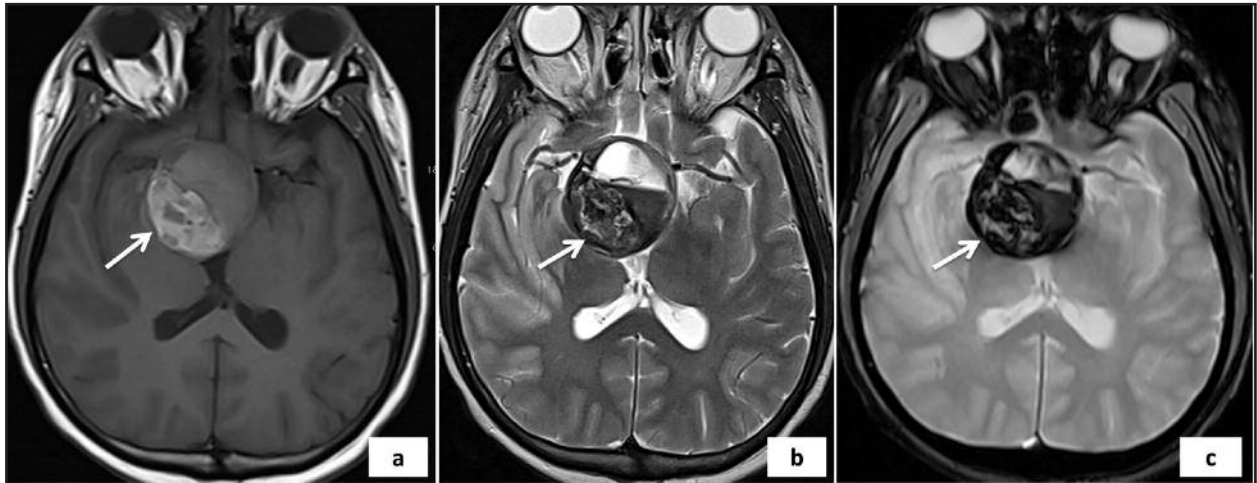


Fig. 1 : Pituitary mass at the sellar region with intra-tumoral haemorrhage seen on axial MRI (a) T1WI, (b) T2WI and (c) GRE sequences (arrows). The haemorrhagic component is hyperintense on T1-weighted image, heterogenous on T2-weighted image with hypointense rim and showed blooming artifact on GRE sequence.

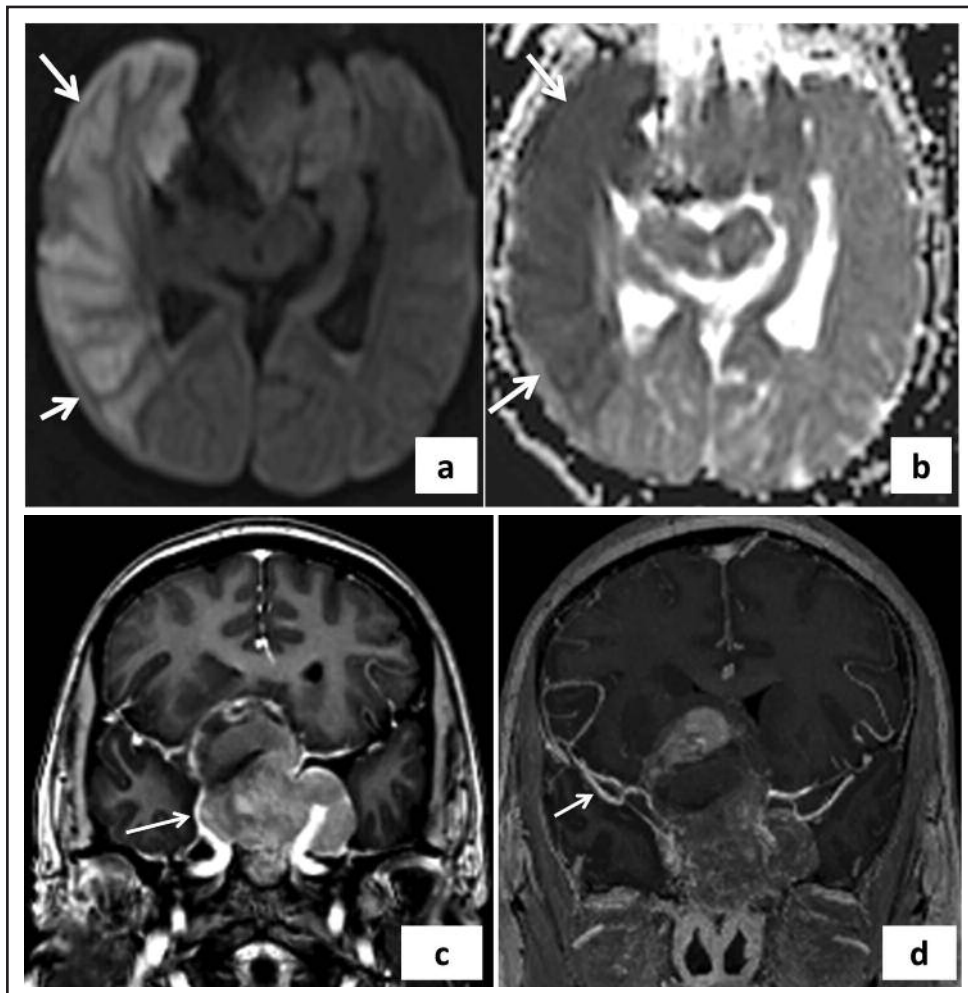


Fig. 1 : Neurovascular event in this patient. Acute cerebral infarction involving the right MCA territory showed on axial MRI as (a) hyperintensity on DWI sequence and (b) hypointensity on corresponding ADC map (arrows). Coronal reformatted images post contrast (c) showed the tumour compressed and pushed the right ICA (arrow). The vessel is still patent with no evidence of thrombus within. (d) The distal MCA branches on both sides were opacified but there were irregularities of the right MCA outline (arrow) suggestive of vasospasm.

However, the sudden vascular compression could also cause trauma to the vascular walls that resulted in thrombus, which then disintegrated and embolized into the right MCA and ACA.

In our case, we hypothesized that vasospasm was the cause of ischaemic event due to the involvement of unilateral MCA, irregularity of right MCA outline and presence of ipsilateral subarachnoid haemorrhage. Even though compression of ICA was demonstrated at the cavernous portion, the flow was maintained and there was no thrombus in the distal arteries (MCA) detected.

Owing to the rarity of the condition, there are no evidence-based criteria to justify the clinical decision between a conservative approach and neurosurgical intervention¹. A few retrospective observational studies suggested early decompression to restore the flow in the carotid artery, which may result in resolution of the neurological deficit⁵. Others reported delayed decompression following conservative therapy with steroid was associated with better outcomes in patients with cerebral infarct⁴. The combined effect of the infarct and brain oedema was usually the cause of mortality in these patients as in our case.

CONCLUSION

Imaging of pituitary tumour with apoplexy should not focus only on the tumour itself but should include assessment of its vascular complication. The presence of neurovascular event may change the choice of treatment and prognostication. Pituitary apoplexy with infarction is associated with poor outcome especially if the infarction involved a large cerebral territory as demonstrated in this case.

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