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#### PITUITARY APOPLEXY: REPORT OF TWO CASES

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## PITUITARY APOPLEXY: REPORT OF TWO CASES

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### SUMMARY

**Pituitary apoplexy is a common clinical syndrome characterised by acute headache, ophthalmoplegia, diminished visual acuity and altered mental status caused by the sudden haemorrhage or infarction of the pituitary gland. The two cases reported highlight the variability in presentation which underscores the need for a high index of suspicion and the need for MRI in patients presenting with a thunderclap headache and 'normal' initial investigations.**

### INTRODUCTION

Pituitary apoplexy is an uncommon yet potentially life threatening clinical syndrome caused by the rapid enlargement of a pituitary adenoma because of haemorrhage or infarction (1). The clinical features are typically sudden in onset, usually evolving over hours and include: headache, vomiting, visual disturbance, ophthalmoplegia and altered consciousness (2, 3).

Bailey was the first to describe a clinical case of pituitary apoplexy resulting from catastrophic haemorrhage of a pituitary adenoma in 1898 (4, 5). Subsequently, there have been numerous published case reports and case series dealing with pituitary apoplexy, resulting in improved understanding of the clinical syndrome and its pathologic features.

*Case 1:* A 42 year old right-handed married teacher, who while singing with the children in her kindergarten class, was struck by an intense occipital headache, nausea and one episode of non-projectile vomiting. She also reported having briefly 'passed out'. Persistence of a dull headache that was aggravated by sitting up prompted a medical referral to a local outpatient clinic where a blood pressure of 150/100mmHg was recorded and an injection of metoclopramide and hydralazine were administered. She was subsequently referred to the Accident and Emergency unit of Aga Khan University Hospital.

She was a para 3+ 1 with recall of previous episodes of elevated blood pressure. She was on a

combined oestrogen progesterone preparation for contraception. Her past medical history was significant for an appendectomy 10 years ago.

Physical examination at the time of admission revealed an alert female with a Glasgow coma scale of 15/15 and vitals: BP 140/90mmHg; Temp 36.5 °C; breathing at 18 breaths per minute and a regular pulse rate of 88 beats per minute. Neurological examination revealed no signs of meningeal irritation, pupillary size 2mm bilaterally with good light response direct and consensual, visual acuity 6/9 RE and 6/9 LE and normal visual perimetry on confrontation testing. She had normal optic discs and no papilloedema in both eyes. She had no other neurological findings. A diagnosis of sub-arachnoid haemorrhage was suspected with a differential diagnosis of vertebrobasilar insufficiency.

Head CT however did not reveal any haemorrhage and a lumbar tap performed about twelve hours after initial occurrence of symptoms failed to reveal xanthochromia.

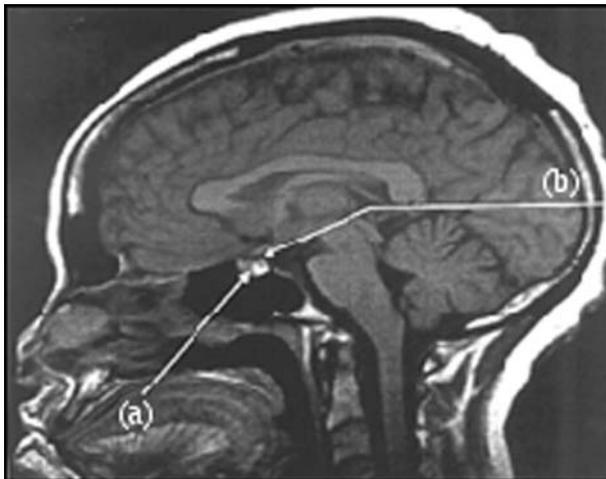
Brain MRI scan showed a hyperintense area in the posterior pituitary with decreased signals on gradient T2 weighted sequences consistent with pituitary apoplexy.

Pertinent laboratory values included a low serum cortisol at baseline; however, we could not establish concurrent ACTH levels, but on administration of intramuscular ACTH, demonstrated a significant rise in serum cortisol levels half and one hour later. She had normal TSH, FSH, LH, prolactin and electrolytes on presentation.

The patient was managed conservatively with corticosteroids and on a follow up hormonal profile was noted to have hypothyroidism necessitating thyroxine replacement. She is currently back to her kindergarten class, asymptomatic, on a regular endocrine follow up on corticosteroids and thyroxine.

**Figure 1**

- (a) T1 weighted sagittal view of the pituitary gland demonstrating bright signal intensity in the anterior pituitary indicative of haemorrhage  
(b) Normal posterior pituitary



*Case 2:* A married 32 year old African female presented seven days after delivery with a history of having had a sudden onset thunderclap headache at its worst at onset two days after delivery. The headache was worse on lying down, was relieved somewhat by over-the counter analgesia and was associated with neck pain and stiffness. She had also reported the presence of fever and episodic non projectile vomiting two days prior to admission. There was no history of visual disturbances.

She had had a spontaneous vertex delivery after induction at 38 weeks gestation and blood loss was noted to be appropriate. Of note was the presence of pregnancy induced hypertension in both the recently ended and her first pregnancy.

Initial assessment at admission revealed a young female who was sick looking and in pain. Blood pressure was 158/105mmHg (range 140 - 190/90 - 120), temperature of 38.1 °C, pulse rate of 95 beats per minute and a respiratory rate of 18 breaths per minute.

She was alert with a Glasgow coma scale of 15/15, oriented in time place and person. Neurological examination was positive for neck stiffness, a positive Kernig's sign and bitemporal hemianopia to red pin. Her pupils were 3mm each and reacting to light. There were no other neurological deficits. The rest of

systemic exam was normal.

Sub-arachnoid haemorrhage was entertained as the diagnosis with differentials of meningitis and cerebral venous thrombosis. Head CT was reported normal and MRI brain revealed pituitary area haemorrhage in an expanded enhancing pituitary gland suggestive of pituitary apoplexy.

Her serum electrolytes remained normal and her hormonal profile was consistent with the lactating state. Her febrile state was attributed to a culture proven urinary tract infection.

Treatment included supportive care and a neurosurgical review suggested continuation of conservative treatment with possibility of surgery if the visual field defects worsened. The patient improved on the conservative approach and at the time of discharge the visual field defect had resolved completely. She is currently symptom-free and on regular follow up.

**Figure 2**

- (c) Coronal FLAIR sequence demonstrating minimal hyperintense signal in the anterior pituitary gland



## DISCUSSION

The definition of pituitary apoplexy has not always been uniform. The majority of authors however agree that it is a clinical syndrome resulting from the haemorrhage, infarction or haemorrhage infarction of a pituitary tumour that results in its sudden and fulminant expansion. Both patients conformed to this definition of clinical apoplexy.

Pituitary apoplexy typically occurs in pituitary macro adenoma which may be hyperfunctional or clinically unfunctional. There is no subtype of tumour that confers a higher risk of apoplexy. The two patients did not have a previously diagnosed pituitary tumour which presents a challenge in making a diagnosis and is a common phenomenon in a number of case series.

Sub-arachnoid haemorrhage is the most common differential diagnosis, and although SAH can be a presenting feature of pituitary apoplexy, it is important to differentiate the two and a sub-arachnoid haemorrhage from an intracranial aneurysm, vascular malformation and cavernous sinus thrombosis. Since a sub-arachnoid bleed is usually the first differential, a CT brain is almost always mandatory; however, a negative scan should raise the suspicion of pituitary apoplexy. Both cases had an initial differential diagnosis of sub-arachnoid haemorrhage and on negative CT scanning, pituitary apoplexy confirmed on MRI. MRI is undoubtedly the imaging modality of choice, because it clearly demonstrates the features of haemorrhage and infarction, the suprasellar extension, optic chiasm compression and extension into the cavernous sinuses (6-8).

Although majority of the cases present spontaneously, Biousse *et al* (9) reduced the multiple factors reported as precipitants into four categories: (a) reduced blood flow in the gland, (b) acute increase in blood flow into the gland, (c) stimulation of the pituitary gland, and (d) the anticoagulated state. The first case is particularly interesting from an aetiological standpoint because contrary to the common belief that pituitary apoplexy occurs in the pregnant or post partum state, this patient had no obvious precipitating factor. This case highlights the importance of having a high index of suspicion especially for patients who do not have clear precipitants and appropriately investigating such patients. Stimulation of the gland and reduced blood flow are possible stimulants in the second case.

The clinical manifestations from a clinicopathological view have been divided into three groups: (a) destruction or compression of the pituitary gland leading to hypopituitarism, (b) sudden enlargement leading to the compression of adjacent neural structures and, (c) leakage of blood resulting in meningism and symptoms similar to sub-arachnoid haemorrhage. The first patient had evidence of hypocortisolism just above 24 hours of the event demonstrated by a low baseline cortisol and subsequent stimulation on administration of ACTH. The second patient however, did not demonstrate any signs of hypopituitarism but had visual symptoms and signs suggesting compression of the adjacent optic chiasma.

The two pillars of management of pituitary apoplexy are transphenoidal decompression and corticosteroid replacement (10-12). It is important to recognise hypocortisolism since if untreated can result in a fatal Addisonian crisis. Surgical decompression is indicated in patients with reduced level of consciousness, hypothalamic disturbances and sudden severe visual impairment or blindness. Both patients were treated conservatively with high dose corticosteroids, correction of fluid and electrolyte imbalances. Although the second patient had visual impairment this resolved spontaneously and may be attributed to the slow resorption of blood following haemorrhage.

The purpose of this presentation is to raise the index of suspicion for pituitary apoplexy due to its varying presentations as described in the two cases, the need for an MRI when the initial investigations are 'normal' and the caution about the potentially fatal hormonal disturbances, especially hypocortisolism, that may follow. In addition, we did not find case reports in local literature.

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