# Severe, sudden onset Headache in a young man: Sub-arachnoid Haemorrhage vs. Pituitary Apoplexy

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**Citation**

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**Abstract**

We describe the case of a young man who presented with an acute onset of headache and focal neurological deficits. There was significant diagnostic difficulty as initial investigations such as CT brain imaging and lumbar puncture results were normal. It was only when his metabolic and endocrine status was thoroughly assessed that we discovered he had developed pituitary tumour apoplexy. There is a large differential diagnosis for an acute severe headache in a young patient and pituitary apoplexy is only accurately assessed on MRI imaging.

## INTRODUCTION

Headaches are undeniably common. Any headache severe enough to warrant emergency admission to hospital needs to be investigated fully and thoroughly. The case we present represents a rare and potentially life-threatening disorder that can mimic several other significant intra-cranial problems. Clinical unawareness of the possible presence of a pre-existing pituitary mass often means that pituitary apoplexy is overlooked. Consequently the diagnosis is often delayed and the clinical signs are mistaken as produced by a different cause.

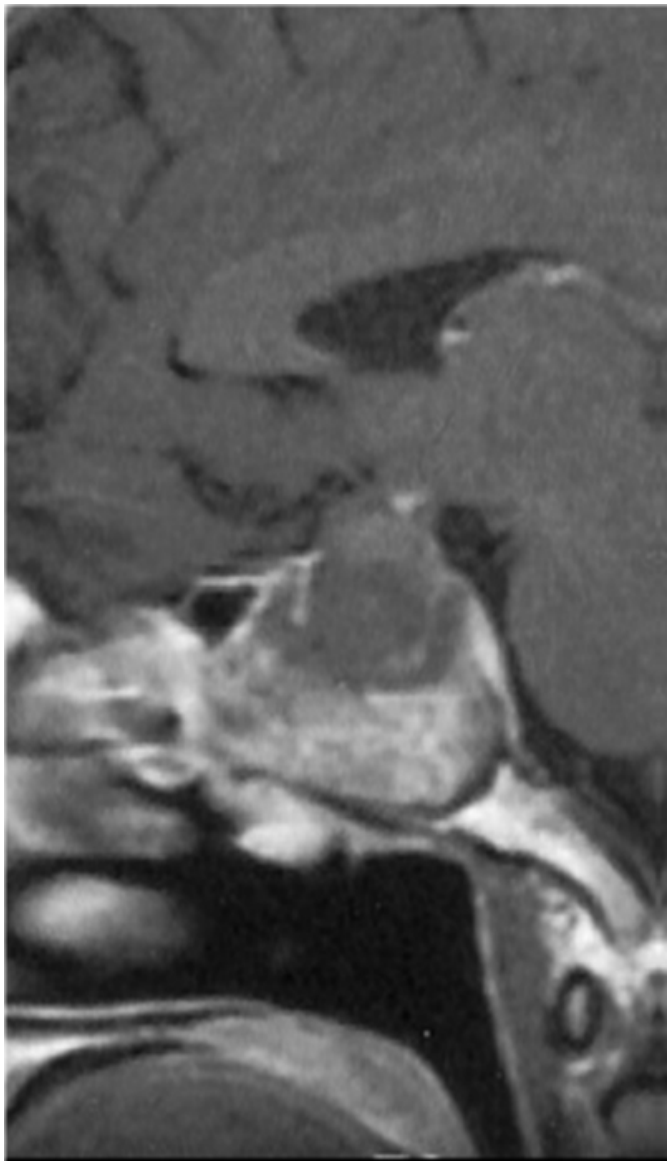
## CASE REPORT

A 27-year-old man presented to Accident & Emergency following a sudden onset of a severe unilateral frontal headache. In addition he described nausea, vomiting, vertigo, fever, lethargy and diplopia on horizontal gaze. On examination, he was pyrexial and sweaty. He had a reduced level of consciousness with a GCS of 14 out of 15, and there was bilateral restriction of his visual fields. He also demonstrated a right-sided sixth cranial nerve palsy but there were no other focal neurological deficits. On mobilising to the toilet he became dizzy and his blood pressure displayed a significant postural drop.

An urgent CT scan of his brain followed by a lumbar puncture was undertaken, both of which were normal. There was no evidence of a haemorrhage, meningeal inflammation or elevation of intracranial pressure. Laboratory investigations showed that he had a raised serum/blood potassium (5.8) and urea (12.2), and a slightly decreased sodium level (133). A random cortisol level was then checked as the suspicion of Addison's disease had been raised (in light of the electrolyte abnormalities, postural hypotension and clinical history) and was found to be 130 (140-560) indicating that he could be acutely hypoadrenal ([1]).

**Figure 1**

Figure 1: MRI Pituitary. Haemorrhage into tumour



An initial MRI of his brain was reported as normal, but after 2 days this was repeated in light of his unresolved symptoms. Subsequently a further MRI, 72 hours after admission showed the hypointense signal of a haemorrhage within the pituitary fossa. A short Synacthen test (tetracosactrin) was also performed and this demonstrated an appropriate cortisol response indicating that the adrenal glands were functioning and the reason behind the initial low cortisol and several of his symptoms was disruption of the pituitary-adrenal axis itself.

A diagnosis of pituitary apoplexy was reached and he was treated medically with high dose corticosteroids and referred to the neurosurgeons for urgent trans-sphenoidal decompression. He was found to have a large, previously undiagnosed pituitary adenoma. The patient consequently made a good recovery and is now on life long hormonal replacement therapy.

## DISCUSSION

Our patient essentially presented with two overlapping sets of symptoms. Firstly those, which were related to an acute, localised intra-cranial haemorrhage. And secondly, as a result this rendered the patient acutely hypopituitary with its associated endocrinological manifestations ([3]).

The non-specific nature of the presenting problem, variability of signs and delay in organising the correct investigations mean its recognition can be delayed. All of the following differentials can fit into a similar pattern ([4]);

Migraine,
Meningitis,
Subarachnoid haemorrhage,
Hypertensive encephalopathy,
Cavernous sinus thrombosis,
Midbrain infarction,
Diabetic cranial nerve palsy,
Optic neuritis,
Space occupying lesion,
Cerebral vasculitis.

Also, if the hormonal disturbances are not appreciated, then a hugely important aspect of the syndrome is missed. The association of pituitary apoplexy with surgery, childbirth and infection may reflect the greater activity of the pituitary gland in response to the increased need for stress hormones in these circumstances ([2],[5]).

Irrespective of the multiple possible diagnoses the initial investigations of choice upon admission to an emergency department with such a constellation of symptoms would always be a CT of the brain plus or minus a lumbar puncture to assess the CSF ([6]). The problem with pituitary tumour apoplexy is that CT is not an adequate imaging technique. Randeva et al reported that CT scanning only picked up 21% of cases of apoplexy in their series, as opposed to 100% with the use of MRI ([7]). Therefore, considering the low sensitivity of CT in these cases it is important not to ignore the possibility of apoplexy if the clinical scenario suggests it ([8]).

Swift recognition of this condition allows for prompt steroid administration and surgical decompression. Recovery of visual and oculo-motor symptoms commonly occurs and hypopituitarism can be compensated for with hormonal

treatment ([9]). In any patient with a dramatic, severe headache pituitary apoplexy needs to be considered and special attention needs to be paid to any electrolyte or metabolic disturbance.

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