CASE REPORT

A case of pituitary apoplexy masquerading as subarachnoid hemorrhage

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Key Clinical Message
Pituitary apoplexy may cause xanthochromia and mimic the clinical presentation of subarachnoid hemorrhage.

Keywords
Endocrine, headache, pituitary, subarachnoid, xanthochromia.

Introduction
Headaches are a common presenting complaint to the medical admissions unit and the differential diagnosis is large. It is important to remember that pituitary apoplexy is a cause of sudden onset headache as to miss the diagnosis may lead to life-threatening consequences. Subarachnoid hemorrhage (SAH) is a more commonly recognized cause of acute onset headache and is often diagnosed through imaging and blood or xanthochromia in the cerebrospinal fluid (CSF). However, it is also important to appreciate that SAH can precipitate pituitary apoplexy and that the latter may in itself produce xanthochromia. We present a case of a patient who presented with acute onset headache due to pituitary apoplexy that was initially attributed to SAH due to the presence of CSF xanthochromia.

Case History
A 75-year-old man presented to the medical admissions unit 4 days after an acute onset, severe frontal headache. There was no associated nausea, vomiting or photophobia. He had a past medical history of atrial fibrillation, bronchiectasis, lumbar stenosis, previous cerebral infarction, and ischemic heart disease. His medications included Aspirin, Ramipril, Doxazosin, Bisoprolol, Simvastatin, and Lantanoprost eye drops.

On admission, his Glasgow Coma Scale (GCS) was 15. He had a temperature of 38°C and a blood pressure of 135/85 mmHg. The only abnormalities of note were mild neck stiffness with cardiovascular, respiratory, abdominal, and neurological examination otherwise normal.

A provisional diagnosis of subarachnoid hemorrhage was made and further investigations undertaken.

Investigations
The patient underwent a CT brain scan, which demonstrated a pituitary adenoma with the initial report suggesting no evidence of hemorrhage. A lumbar puncture was performed. The CSF demonstrated a protein concentration of 1.44 g/L (0.15–0.45), glucose 4.8 mmol/L (2.2–3.9) and the gram stain did not demonstrate any organisms. There was xanthochromia in the CSF and a diagnosis of subarachnoid hemorrhage (SAH) was assumed.

The following morning, the patient had a sudden fall in his GCS, from 15 to 13. In addition, his blood pressure
fell to 66/46 mmHg. Biochemical data revealed a low sodium level at 131 mmol/L and serum potassium within the reference range at 3.5 mmol/L. On review of the original overnight CT scan by a consultant neuroradiologist, there was felt to be hemorrhage consistent with pituitary apoplexy within a pituitary macroadenoma. The patient was also assumed to have hypoadrenalism and commenced intravenous hydrocortisone 100 mg thrice daily.

**Differential Diagnosis**

Differential diagnoses of pituitary apoplexy and SAH were considered based on the history and the presence of xanthochromia. A subsequent neurosurgical review recommended CT angiography. This confirmed the pituitary macroadenoma yet no aneurysm was identified.

**Outcome and Follow Up**

The patient underwent a pituitary MRI scan, which confirmed the presence of a pituitary macroadenoma, discrete from the optic chiasm (Fig. 1). There was no visual field defect present and the patient was managed conservatively with switch to oral hydrocortisone.

Baseline pituitary endocrine function (presented in Table 1) revealed a low prolactin and testosterone concentration. The short synacthen test demonstrated a 30-minute cortisol measurement 704 nmol/L, yet as this was performed shortly after apoplexy, the patient continued on hydrocortisone 10 mg bd with a view on repeating the short synacthen test 6 weeks after discharge from hospital.

The patient was discharged home following a 10 day in-patient stay on hydrocortisone 10 mg bd in addition to his regular medication. Further outpatient review and pituitary profile tests demonstrated thyroid function test and testosterone level within normal limits. A follow-up short synacthen test revealed a 30 min cortisol 579 nmol/L.

![Figure 1. MRI with contrast. A coronal image of the pituitary macroadenoma as demonstrated by the arrow.](image1)

![Figure 2. MRI with contrast. A coronal view of the pituitary gland 3 months after the apoplectic event. The upper arrow demonstrates the optic chiasm, and the lower arrow demonstrates the pituitary gland with resolution of the adenoma following the event.](image2)

| Table 1. Pituitary function tests. |
|-----------------------------------|
| Baseline endocrine function tests | Results during hospitalization | Results at outpatient review 3 months later | Reference range |
| IGF 1               | 16.3                      | 23                   | 4–25 nmol/L |
| TSH                 | 0.25                      | 1.08                 | 0.3–4.40 mIU/L |
| T4                  | 11.9                      | 13.1                 | 9–19.1 pmol/L |
| Prolactin           | 43                        | –                   | 56–278 (mIU/L) |
| Testosterone        | 1.7                       | 8.6                  | 8–30 nmol/L |
| FSH                 | 15.4                      | 24                   | 1–12 IU/L |
| LH                  | 6.3                       | 10                   | 1–9 IU/L |
| Short synacthen (nmol/L) | 333                      | 298                  | 30 min cut-off |
| Cortisol 0 min      | 704                       | 579                  | reference ≥450 nmol/L |

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L (cut off 450 nmol/L) indicating adequate adrenal reserve and the hydrocortisone was discontinued (Table 1).

A repeat MRI pituitary scan demonstrated a considerable reduction in the size of the previously reported pituitary macroadenoma. The tumor appears to have shrunk considerably being confined to the right side of the pituitary fossa lying next to what appears as normal pituitary tissue (Fig. 2).

Discussion

Pituitary apoplexy is a rare cause of sudden onset headache and, as is often the case, there were no features to suggest the prior existence of a pituitary tumor. Similarly, the presentation with sudden onset headache mimics that of a subarachnoid hemorrhage. The presence of xanthochromia on CSF examination prompted further investigation of the patient for the source of a SAH.

This case illustrates the presence of CSF xanthochromia in association with pituitary apoplexy [1]. There is close proximity of the pituitary gland to the subarachnoid space, as the anterior surface of the pituitary gland is covered by the arachnoid membrane. Thus, in pituitary apoplexy it is assumed that blood can extravasate into the subarachnoid space following an apoplectic event and mimic the CSF features of SAH. In addition, there is a delay in the presence of xanthochromia in the CSF which can take up to 12 h to develop, based on the conversion of oxyhemoglobin to bilirubin in the CSF [2].

Although our case reveals pituitary apoplexy as the cause of xanthochromia, others have reported the association of apoplexy in conjunction with SAH [3–6]. Interestingly, there is some association between intracranial aneurysms and pituitary adenomas [7]. Apoplexy often occurs in an occult pituitary adenoma and the hemodynamic changes of cerebral flow associated with acute SAH may predispose to apoplexy. In our case, the possibility of coincident pituitary apoplexy and SAH was considered yet the absence of an abnormality on cerebral CT angiography argues against aneurysmal SAH as a cause. Although, we note cerebral angiography has a low sensitivity in the detection of small aneurysms [8].

Our case also illustrates the successful approach to conservative management of pituitary apoplexy. Not all cases of pituitary apoplexy require surgical resection [7]. In the absence of visual field impairment and without the tumor causing visual field defects, a conservative approach can be adopted [9, 10]. Repeat MRI of the pituitary, 3 months following admission revealed near complete resolution of the pituitary macroadenoma. This may be a consequence of auto-infarction of the tumor yet with residual disease, future monitoring with imaging will be required.

In summary, this case report reveals the rare event of pituitary apoplexy as a cause of sudden onset headache and the potential of pituitary apoplexy to mimic SAH through production of xanthochromia. While in this case, the diagnosis was pituitary apoplexy, our literature review reveals that SAH may precipitate apoplexy within a pre-existing pituitary tumor and that exclusion of the former may be required. We also show the success of a conservative approach to the management of pituitary apoplexy in certain cases.

Conflict of Interest

None declared.

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