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An exceptional presentation of pituitary apoplexy in thyrotoxicosis. A rare case report

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Keywords
pituitary apoplexy, pituitary haemorrhage, hyperthyroidism, diagnosis, management

ABSTRACT

Pituitary apoplexy (PA) is a rare and potentially life-threatening condition characterized by severe headaches, vomiting, visual disturbances, ophthalmoplegia, altered mental status, and possible pan-hypopituitarism. A macroadenoma-induced pituitary haemorrhage is the primary cause. Various factors such as systemic hypertension, altered intracranial pressure, head trauma, anticoagulation, and pregnancy can trigger PA. A 35-year-old non-smoking male presented with headaches and an enlarged thyroid gland. Initial imaging revealed a haemorrhage in the sellar area, prompting a clinical diagnosis of pituitary apoplexy with T3 thyrotoxicosis. Despite the absence of visual impairment, conservative management was chosen, including anti-hypertensive therapy and follow-up MRI after three months. PA diagnosis can be challenging without prior identification of a pituitary tumour. Imaging modalities like MRI are crucial for diagnosis, and radiological follow-up is recommended. The relationship between PA and hyperthyroidism remains unclear, necessitating further investigation in patients presenting with PA symptoms.

INTRODUCTION

Pituitary apoplexy (PA) is a rare and potentially life-threatening condition characterized by acute hemorrhage or infarction of the pituitary gland, typically occurring in the setting of a pre-existing pituitary adenoma. It manifests clinically with sudden-onset severe headaches, vomiting, visual disturbances, ophthalmoplegia, altered mental status, and potential pituitary hormone deficiency [1,2]. The condition poses a diagnostic challenge due to its varied presentation and potential overlap with other intracranial pathologies. Various predisposing factors, such as systemic hypertension, altered intracranial pressure, head trauma, anticoagulation therapy, pregnancy, and hemodialysis, have been implicated in triggering PA [3]. Prompt recognition and management are essential to prevent potentially devastating complications such as pituitary hormone deficiency, visual impairment, and neurological sequelae.

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Case report

A 35-year-old man presented to the neurosurgery department with headaches. He was a non-smoker. There was no evidence of ophthalmoplegia, although the thyroid gland was somewhat enlarged as a diffuse goitre on palpation. Hypopituitarism was not present. The patient had a 150/90 mmHg blood pressure, a heart rate of 90 beats per minute, a breathing rate of 18 breaths per minute, and a body temperature of 36.7 oC.Glasgow coma scale (GCS) was 15/15, and there were no additional cranial nerve impairments found. Computer tomography (CT) angiography revealed no evidence of an aneurysm. After ruling out traumatic brain damage and subarachnoid hemorrhage caused by an aneurysm, we were alerted to a suspicious lump in the sellar area of the head on CT. The pituitary gland seemed to be hemorrhaging (Fig 1).

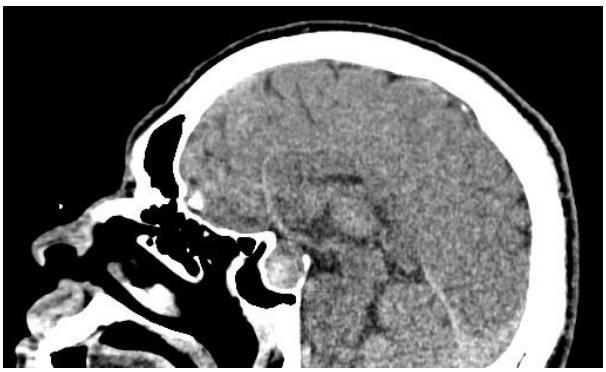

Figure 1. CT Head Saggital shows sign of hemorrhage in the pituitary.

Since the sellar area was of particular interest, we conducted an improved brain magnetic resonance imaging (MRI). Slightly hyperintense T1-weighted and hypointense T2-weighted images were seen in the sellar area of the brain, which indicated a bleeding in the lesion (Fig 2). Laboratory investigations revealed decreased thyroid stimulating hormone (TSH) levels (0.010 IU/mL) but increased free triiodothyronine (T3) (20.80 pg/mL) and thyroxine (T4) levels (> 6.00 ng/dL).

Therefore, a clinical diagnosis of pituitary apoplexy with T3 thyrotoxicosis was made. There were no signs of compression of the optic chiasm and no visual impairment, so surgical intervention was unnecessary. Given the absence of visual impairment or neurological deficits, conservative management was initiated, including anti-hypertensive therapy and close monitoring of hormone levels. Follow-up MRI imaging was scheduled after three months to assess for resolution of hemorrhage and monitor for any changes in pituitary morphology.

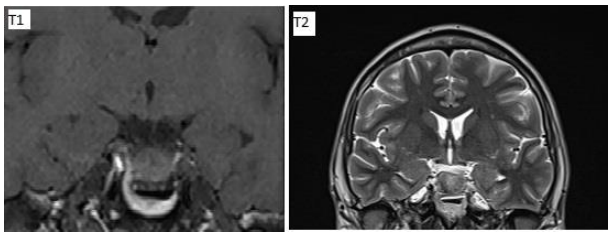

Figure 2. MRI of Brain T1 shows partially hyperintense and T2 shows partially hypointense.

Discussion

Pearce Bailey published the first description of pituitary apoplexy (PA) in 1898 [4]. If a pituitary tumor hemorrhages or infarcates, it may cause pituitary apoplexy, an uncommon but potentially deadly illness [5].

An apoplexy diagnosis might be challenging if a pituitary tumor has not been previously identified. Postpartum hemorrhage and the use of anticoagulants, as well as other bleeding diseases, diabetes, concussions, and other injuries to the brain, are all linked to an increased risk of apoplexy. The most common risk factor was hypertension. Additionally, thyroid dysfunction has been well proven to affect cardiac output, contractility, blood pressure, vascular resistance, and rhythm problems [6,7]. Thus, in individuals with pituitary adenoma, it is theoretically plausible that primary thyroid failure induces pituitary apoplexy. However, the clinical relationship between pituitary apoplexy and thyroid dysfunction has remained ambiguous.

Hyperthyroidism, particularly T3 thyrotoxicosis, might be overlooked in pituitary apoplexy due to normal free T4 and reduced TSH levels. TSH suppression together with increased free T3 but normal free T4 is referred to as T3 thyrotoxicosis [8]. Acute neuro-ophthalmological degeneration with headache is a sign of pituitary apoplexy. The clinical presentation is variable. Fever and headaches are symptoms of infarcted tissue leaking into the subarachnoid space. Ophthalmoplegia may develop if the pituitary enlarges or bleeds into the cavernous sinus [9].

Cortisol deficit is a biological indication of pituitary insufficiency that should be explored. Aside

from TSH, free thyroid hormones are required to diagnose secondary hypothyroidism. Imaging of the pituitary fossa, particularly CT, has low diagnostic sensitivity. So 'pituitary' MRI is the gold standard for detecting bleeding. It is critical to do radiological follow-up with an MRI in individuals who have PA. In pituitary apoplexy, fluid and electrolyte levels are monitored to prevent subsequent diabetes insipidus. In cases of reduced state of awareness, visual impairment, or hypothalamic disruption, immediate high dosage corticosteroid replacement and surgical decompression may be necessary [10].

The relationship between pituitary apoplexy and hyperthyroidism, particularly T3 thyrotoxicosis, remains unclear and warrants further investigation. Although hyperthyroidism has been implicated as a potential trigger for pituitary apoplexy, the underlying mechanisms and clinical implications remain poorly understood. Future research should aim to elucidate the pathophysiology of this relationship and identify potential therapeutic targets.

Conclusions

Pituitary apoplexy is a rare but potentially life-threatening condition that requires prompt recognition and management. Clinical presentation can vary widely, and imaging studies such as CT and MRI play a crucial role in confirming the diagnosis. Management is largely supportive and aims to stabilize the patient, control symptoms, and prevent complications. The relationship between pituitary apoplexy and hyperthyroidism remains unclear and warrants further investigation. Optimal management requires a multidisciplinary approach involving neurosurgery, endocrinology, and radiology, with close monitoring of hormone levels and imaging studies to assess for resolution of hemorrhage and monitor for any changes in pituitary morphology.

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