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Case Report

Pituitary Apoplexy Presenting as Acute Altered Sensorium in a Non-Diabetic Middle-Aged Man

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Abstract

Background: Pituitary apoplexy (PA) is a rare endocrine-neurosurgical emergency that is the result of acute bleeding or an infarction of the pituitary gland. Sudden headache, visual loss and cranial nerve palsies are typical presentation, but altered sensorium is an underemphasized but clinically important event which may present as a stroke or meningitis, and slow down the process of diagnosis.

Case: A non-diabetic 52-year-old man came to the emergency department with abrupt altered sensorium (Glasgow Coma Scale 9/15) without any history of headache or vomiting. There was no history of diabetes, hypertension, trauma, or any use of anticoagulants. On examination, he was drowsy but arousable, with stable vital signs and no focal neurological deficit. Initial non-contrast CT brain was unremarkable, but MRI revealed a 2.1 × 1.8 × 2.0 cm sellar-suprasellar mass with heterogeneous signal intensity and evidence of recent haemorrhage, consistent with pituitary apoplexy. Laboratory evaluation showed serum sodium 128 mEq/L, serum cortisol 1.8 µg/dL (low), ACTH <5 pg/mL, free T4 0.6 ng/dL (low) with inappropriately normal TSH, indicating secondary adrenal insufficiency and central hypothyroidism.

Management and Outcome: The patient received hydrocortisone 100 mg IV stat, then 50 mg IV every 6 hours, along with isotonic saline. In the following 24 hours, his mental condition improved significantly, and GCS returned to 15/15. Since there was no progressive loss of visual functions, he was treated in a conservative manner using hormone replacement (hydrocortisone and levothyroxine). Three-month follow up revealed that he was still neurologically intact, MRI revealed that haemorrhage resolved partially and he was stable clinically on pituitary hormone replacement.

Conclusion: Even in patients with no headache and unremarkable CT in middle-aged patients with acute altered sensorium, one should consider to evaluate for pituitary apoplexy even in the absence of headache or visual symptoms. MRI is the gold standard of diagnosis, and early treatment with glucocorticoids is life-saving, with definitive management being tailored.

Keywords: Pituitary apoplexy; altered sensorium; non-diabetic; adrenal insufficiency; MRI; hydrocortisone.

Introduction

Pituitary apoplexy (PA) is a rare but potentially fatal endocrine crisis caused by an acute haemorrhage or infarction in the pituitary gland. The disorder is most common in preexisting adenomas and is commonly the initial result of an undiagnosed pituitary tumor [1,2].

Classically, PA is characterized by acute severe headache, visual field deficit, ophthalmoplegia, nausea or vomiting, and a range of altered consciousness level [6,7]. Altered sensorium however is poorly identified whilst it can be present in up to 20-40% of patients with mild lethargy to stupor and coma in this disease, even though it is not widely recognized, particularly in its milder forms of presentations and manifestations of this condition [5,6]. The unusual manifestations may closely resemble meningitis, meningoencephalitis, or subarachnoid haemorrhage, and therefore, may be mistakenly diagnosed and treated later on [7,8]. The epidemiology of PA is characterized by most prevalence in the fifth to sixth decades of life, male predominance, and a 0.6-9% surgical treatment rate of pituitary adenomas with PA [2,3]. Despite precipitating factors being reported including hypertension, anticoagulation, dynamic pituitary testing, or major surgery, up to 40% of cases have no known precipitating factors [1,6].

Non-contrast CT detects only 25 – 50% of pituitary haemorrhages, whereas MRI is the diagnostic gold standard. The management needs urgent hemodynamic stabilization and high dose intravenous glucocorticoids to avoid adrenal crisis,

with a subsequent individualized decision between early transsphenoidal surgery and conservative treatment basing on neuro-ophthalmic status [1,4,6].

In this case, we are reporting about a non-diabetic middle-aged man whose first presentation was acute altered sensorium without headache or visual complaints, which makes it necessary to consider PA as a part of the differential diagnosis of sudden impaired consciousness.

Case Presentation

The patient is a non-diabetic 52-year-old man with no reported comorbidities who was brought to the emergency department with acute altered sensorium. As informed by relatives, he was oriented to time place and persons, until a few hours just before admission when he was observed to be unusually drowsy and incoherent in speech. No history of previous headache, vomiting, seizures, fever or visual complaints like blurring or diplopia. He was not using any anticoagulants, no known history of hypertension, no prior pituitary disease, no head trauma, no recent surgery, and no family history of endocrine disorder. Upon arrival, the patient was drowsy with a Glasgow Coma Scale (GCS) of 9/15 (E2V2M5). His vital signs were stable, including blood pressure 118/74 mmHg, pulse 86/min and regular, respiratory rate 18/min, temperature 36.9°C, and oxygen saturation 98% on room air. There were no signs of dehydration and meningeal irritation the patient was responsive to light, eye movements were complete and fundus examination revealed no papilledema. Motor and sensory systems were grossly intact. Higher mental function and cranial nerve examination could not be assessed.

Initial laboratory investigations revealed serum sodium 128 mEq/L and potassium 4.0 mEq/L. Random blood glucose was 104 mg/dL, effectively excluding hypoglycaemia as the cause of altered sensorium. Renal and liver function tests were normal. Hormonal profile showed a low morning cortisol of 1.8 µg/dL with suppressed ACTH (<5 pg/mL), consistent with secondary adrenal insufficiency. Free T4 was 0.6 ng/dL (low) with an inappropriately normal TSH of 1.1 mU/L, suggesting central hypothyroidism. Serum prolactin and gonadotropins were marginally reduced. Complete blood count was within normal limits.

Table 1: Laboratory findings at presentation

Parameter	Patient Value	Reference Range	Interpretation
Serum sodium	128 mEq/L	135–145 mEq/L	Hyponatremia
Serum potassium	4.0 mEq/L	3.5–5.0 mEq/L	Normal
Random blood glucose	104 mg/dL	70–140 mg/dL	Normal
Morning cortisol	1.8 µg/dL	6.7–22.6 µg/dL	Low (adrenal insufficiency)
ACTH	<5 pg/mL	10–60 pg/mL	Suppressed
Free T4	0.6 ng/dL	0.8–1.7 ng/dL	Low (central hypothyroidism)
TSH	1.1 mU/L	0.4–4.0 mU/L	Inappropriately normal
Prolactin	3.8 ng/mL	4–15 ng/mL	Mildly reduced
LH/FSH	Low	Age/sex dependent	Hypogonadotropic state

A non-contrast CT scan of the brain performed at admission was unremarkable, showing no obvious evidence of infarct or acute haemorrhage. Given the strong clinical suspicion of an intracranial event, an MRI of the brain and sella was obtained. This revealed a sellar-suprasellar mass measuring 2.1 × 1.8 × 2.0 cm with heterogeneous T1 hyperintensity, areas of subacute haemorrhage, and compression of the optic chiasm, without hydrocephalus. These findings were diagnostic of pituitary apoplexy in a macroadenoma. Despite suprasellar extension contacting the optic chiasm on MRI, there was no objective visual field deficit on formal assessment.

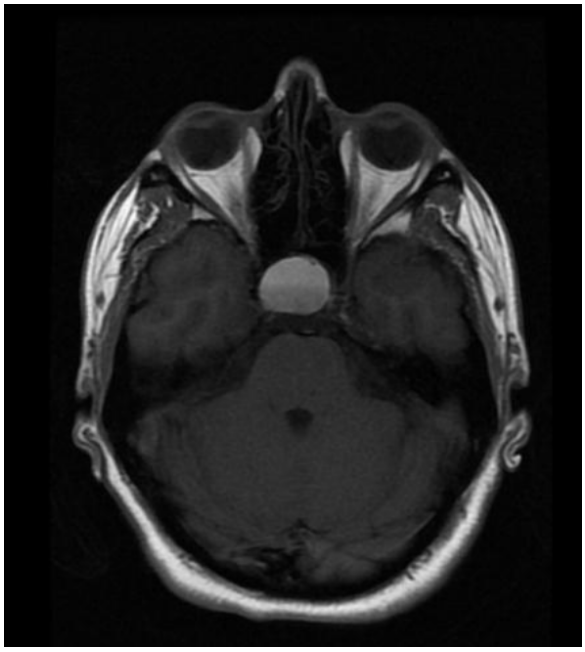

Figure 1: MRI brain (T1-weighted axial image) demonstrating a 2.1 × 1.8 × 2.0 cm sellar–suprasellar mass with heterogeneous hyperintensity and intralesional haemorrhage, consistent with pituitary apoplexy. Mild superior displacement of the optic chiasm is noted.

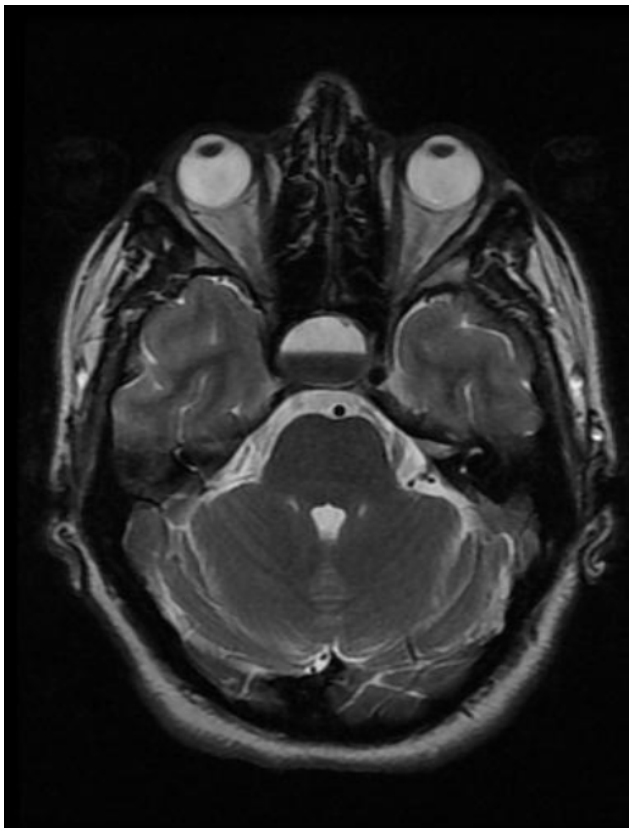

Figure 2: MRI brain (T2-weighted axial image) showing mixed signal intensity within the pituitary fossa, representing haemorrhagic areas.

Hydrocortisone 100 mg IV stat followed by 50 mg IV every 6 hours, with isotonic saline resuscitation, was started immediately. The next day (24 hours later) his neurological conditions became much better and his GCS was back to 15 /15. Ophthalmological examination was performed and visual acuity and visual field were found to be normal and no ocular motor palsy. Neurosurgical consultation was requested, but considering that there were no progressive visual or neurological

impairments, it was advised to be conservative. The patient was stable throughout the hospital stay. He was switched to oral hydrocortisone (20mg in the morning, 10mg in the afternoon) and levothyroxine (50 ug/day). The patient was educated on sick-day steroid rules.

The patient stated that he had no symptoms recurrence and was neurologically intact at the three months follow-up.

Repeat MRI indicated that there was partial improvement of the haemorrhagic component with attenuation of the size of the tumour. Re-examination of the endocrine revealed persistent secondary adrenal insufficiency and central hypothyroidism, which required further hormone replacement.

The patient is receiving long-term endocrinology follow-up of pituitary functioning.

Differential diagnoses considered:

Acute ischemic stroke, subarachnoid haemorrhage, meningoencephalitis, metabolic/toxic encephalo pathy.

These were reasonably excluded by normal non-contrast CT, absence of meningeal signs and inflammatory markers, and correction of hyponatremia/adrenal insufficiency with clinical improvement.

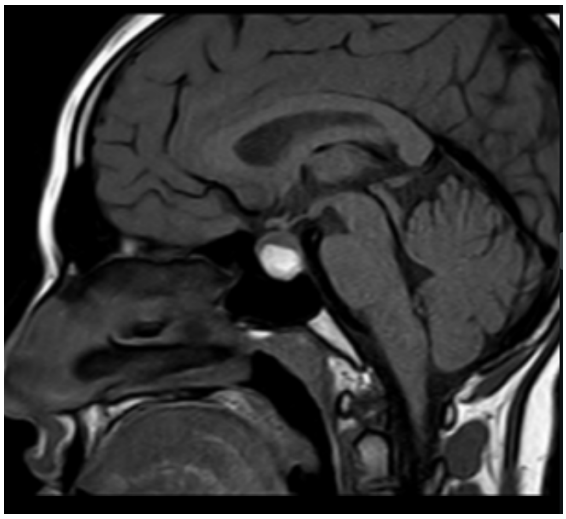

Figure 3: Follow-up T1 saggital brain MRI image at 3 months, showing resolving haemorrhage as compared to previous MRI.

Table 2: Clinical timeline of events

Day / Time	Clinical Event / Finding	Intervention / Outcome
Day 0 – Morning	Found drowsy, unresponsive at home	Brought to ED
Day 0 – Admission	GCS 9/15; vitals stable; no focal deficits	Initial labs, CT brain (normal)
Day 0 – Evening	Hyponatremia (Na 128 mEq/L), low cortisol, central hypothyroidism	Started IV hydrocortisone + fluids
Day 1	GCS improved to 15/15	Continued hydrocortisone, monitoring
Day 2	MRI: 2.1 × 1.8 × 2.0 cm hemorrhagic sellar–suprasellar mass	Neurosurgery consulted; conservative management advised
Day 5	Clinically stable, normal vision	Transitioned to oral hydrocortisone + levothyroxine
Discharge (Day 7)	Neurologically intact, stable labs	Discharged with replacement therapy
3-Month follow-up	No recurrence, stable clinically	MRI: partial resolution of haemorrhage, persistent hormonal deficiency

Discussion

Pituitary apoplexy (PA) is a serious but rare situation caused by the acute haemorrhage or infarction of the pituitary gland typically in a preexisting adenoma. In spite of the classical presentation of severe headache, visual impairment and cranial nerve palsies, the atypical manifestations are increasingly becoming identified. Amongst them, altered sensorium, is a very serious manifestation that is highly valued but underestimated.

A patient brought to us, a middle-aged, non-diabetic man, reported that he has acute altered consciousness, no prior headache or visual complaints, and this is where the diagnostic problem of PA that is based on typical findings can

be observed when such characteristics are not observed. Five to forty percent of patients with PA report an altered mental status; mild drowsiness to unconsciousness, although incidentally [1,2].

The mechanisms behind it are adrenal deficiency with compromised hemodynamics or hyponatremia, subarachnoid haemorrhage, elevated intracranial pressure, and compression of the hypothalamus [1,3]. In a study by Lee et al., 1 out of 16 surgically treated patients had stuporous mentality and this was attributed to hyponatremia [2]. Wang et al. described a patient with fever and meningism initially managed as meningitis; subsequent imaging identified pituitary haemorrhage consistent with apoplexy.

Salehi et al. reported an elderly man who presented with ophthalmoplegia and frequent instances of altered consciousness without headache; MRI revealed pituitary apoplexy and surgery resulted in complete neurological recovery [1]. Viola et al. reported a 63-year-old male on anticoagulants who developed headache, progressive visual impairment, and, ultimately, altered consciousness, as a result of adrenal crisis; high-dose hydrocortisone reversed his condition without surgical intervention [5].

Wang et al. highlighted another unusual mimic—PA presenting as acute meningitis with fever, photophobia, and altered sensorium, again emphasizing that absence of classical symptoms may delay diagnosis [6].

In contrast to these reports, our patient had neither headache nor visual loss at presentation, making altered sensorium the sole initial manifestation. Neuroimaging plays a crucial role in diagnosis. Non-contrast CT detects only 25 – 50% of pituitary haemorrhages, whereas MRI is the diagnostic gold standard [5]. MRI, however, is a reliable method to prove haemorrhagic or necrotic adenoma and is the gold standard of diagnosis [4,5].

The normal CT and diagnostic MRI findings of our patient are congruent with the observations and confirm the significance of early MRI in unexplained altered sensorium.

Acute management rests upon the provision of timely glucocorticoid therapy which will correct adrenal failure and cause a decrease in mass effect. Intravenous hydrocortisone in high dose results in fast enhancement of consciousness and hemodynamic stability, as seen in both in Viola et al. [5] and in our case.

The decision to surgically decompose or use conservative management is dependent on the extent of visual or neurological compromise. Although early transsphenoidal surgery is recommended in patients with slowly progressive loss of vision or sensorium deterioration, conservative therapy using steroids and hormonal replacement yields positive results in stable cases [1,2,5]. Our patient has been responsive to medical management, which is in line with evidence that the management can be equally effective when visual functioning is intact.

Our case demonstrates the broad clinical spectrum of PA due to the absence of diabetes, headache, or cranial nerve palsies. Misdiagnosis is an inherent condition since, as Ranabir and Baruah observed, PA may mimic stroke, meningitis or any other acute neurological disease [1].

The early identification is critical as the late introduction of steroids can complicate or even lead to death. Our report contributes to the scanty literature describing altered sensorium as the leading symptom of PA, and emphasizes the role of high index of suspicion in other presentations of this emergency.

Conclusion

Pituitary apoplexy is a rare, potentially life-threatening endocrine emergency with highly variable presentations. Although headache and visual deficit are classic features, altered sensorium may be the sole presenting symptom and the condition is often misdiagnosed as stroke or meningitis.

In our case, a non-diabetic middle-aged man presented with altered consciousness without headache or visual loss. MRI played a significant role in making the diagnosis after a non-contributory CT scan was taken. High-dose glucocorticoid administration immediately led to swift neurological recovery, and the patient was treated successfully through conservative management using hormone replacement. The given case illustrates the criticality of high index of suspicion regarding PA in patients with sudden altered sensorium despite the absence of typical features. MRI is to be taken promptly, and no action should be postponed with glucocorticoid replacement since proper intervention could be life-saving and show considerable effects.

Learning Points

- Pituitary apoplexy may present with altered sensorium without headache or visual symptoms, and can mimic stroke or meningitis.
- MRI is the diagnostic gold standard, whereas CT findings may be unremarkable.
- Prompt administration of intravenous hydrocortisone is critical for stabilization and recovery.
- Conservative management with hormonal replacement can be effective in patients

without progressive visual or neurological deterioration.
- A high index of suspicion is essential to prevent delays in diagnosis and treatment.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the consent is available for review by the journal's editorial office.

Author Contributions:

Dr. Sahil Sangwan – primary author, collected clinical data, drafted the manuscript, contributed to radiological interpretation and clinical management, critically revised the manuscript and provided overall supervision.

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