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Case Report

Silent sella, silent glands: a case of secondary adrenal insufficiency in partial empty sella

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ABSTRACT

Empty sella syndrome (ESS) is a rare cause of hypopituitarism and may lead to secondary adrenal insufficiency (SAI). Atypical presentations without classic signs such as hypotension or electrolyte imbalance can delay diagnosis. We report a 41-year-old woman presenting with recurrent generalized tonic-clonic seizures and persistent hypoglycemia unresponsive to dextrose. Her medical history included anemia, alopecia, prior bariatric surgery, and bilateral oophorectomy. Laboratory tests revealed low adrenocorticotropic hormone (ACTH) levels with normal thyroid function. Brain magnetic resonance imaging (MRI) showed partial empty sella without mass lesions, indicating pituitary dysfunction and central adrenal insufficiency. The patient received intravenous hydrocortisone and glucose, leading to rapid resolution of seizures and hypoglycemia. She was transitioned to oral corticosteroid therapy and discharged with endocrine follow-up. No further hypoglycemic or seizure episodes occurred during follow-up. This case highlights the importance of considering central adrenal insufficiency in adults with unexplained seizures and refractory hypoglycemia. Partial empty sella may cause subtle pituitary hormone deficiencies that are easily overlooked. Early recognition and prompt glucocorticoid therapy are essential to prevent adrenal crisis and improve outcomes.

Keywords: Empty sella syndrome, Central adrenal insufficiency, Hypopituitarism, Refractory hypoglycemia, Seizures

INTRODUCTION

Empty sella syndrome (ESS) represents only about 1.2% of hypopituitarism cases, with tumors in the hypothalamic-pituitary region being the most frequent cause.[1] The prevalence of primary empty sella, i.e., empty sella without any discernible cause, is not precisely known; estimates range from 2% to 20%. Technical advances in neuroradiology have made empty sella an increasingly common incidental finding.[2] The prevalence of hypopituitarism in patients with primary ESS varies widely, with studies reporting a range between 2% and 32%. This variability underscores the importance of thorough hormonal evaluation in individuals diagnosed with ESS.[3] In a retrospective analysis of 46 patients with primary ESS, approximately 24% exhibited deficiencies in one or more pituitary hormonal axes, emphasizing the potential for significant endocrine abnormalities in this population.[4]

Although hormone disorders were not common in a study, patients with empty sella still exhibited higher rates of secondary adrenocortical insufficiency, which demonstrates that patients with empty and partial empty sella should undergo hormone tests as part of a routine assessment protocol.[5] Partially empty sella can complicate the diagnosis of pituitary disorders by obscuring adenomas on imaging. Though rare, its association with endocrine conditions like Cushing's disease highlights the need for thorough hormonal evaluation. Recognizing this relationship is essential for accurate diagnosis and timely management.[6]

CASE REPORT

Patient information

The patient, a 41-year-old female from Pakistan, admitted at Thumbay University Hospital. She has a medical history notable for hypoglycemia and seizures, with the latest episode being her fourth seizure in six months.

Medical history

She presented after experiencing a 30-minute tonic-clonic seizure with an 8-minute period of unconsciousness followed by postictal confusion. Symptoms included palpitations before the seizure, tongue biting, oral secretions, and a fall from bed resulting in a right hematoma. She also reported bilateral arm pain and persistent hypoglycemia unresponsive to dextrose. Past medical history includes anemia, hypoglycemia, fatigue, and poor sleep. She is a known case of alopecia. She has undergone bilateral oophorectomy in the past due to cyst rupture and bypass bariatric surgery.

Clinical findings

The patient was admitted with a suspected case of Addison's disease, presenting with a history of seizures. On examination, the patient was alert, oriented, and vitally stable. No further seizures were observed during the hospital stay.

Laboratory investigations revealed a low adrenocorticotropic hormone (ACTH) level (<5.0 pg/ml) and intermittent hypoglycemic episodes. GRBS values ranged from 109 to 205 mg/dl, with one noted value of 186 mg/dl. Electrolyte and hormonal imbalances were mild; complete blood count (CBC), liver function test (LFT), renal profile, thyroid function, and abdominal imaging were unremarkable.

Diagnostic assessment

Brain magnetic resonance imaging (MRI) with contrast showed an attenuated anterior pituitary height, consistent with partial ESS, without any mass or abnormal enhancement as seen in Figures 1 and 2. These findings supported a central cause of ACTH deficiency, suggestive of secondary adrenal insufficiency. Figure 1 demonstrates a partially empty sella with a thinned and flattened pituitary gland lining the floor of the sella turcica. The sella is partially filled with cerebrospinal fluid (CSF), giving it a "flattened" appearance. The pituitary stalk is midline and intact, with no evidence of mass lesion or compression. These features are consistent with partial ESS.

Figure 2 again shows a partially empty sella, with a reduced volume of the pituitary gland and prominence of the suprasellar cistern extending into the sella. The normal anatomical structures of the brainstem, cerebellum, and corpus callosum are preserved. The overall appearance supports the diagnosis of partial empty sella without secondary structural pathology.

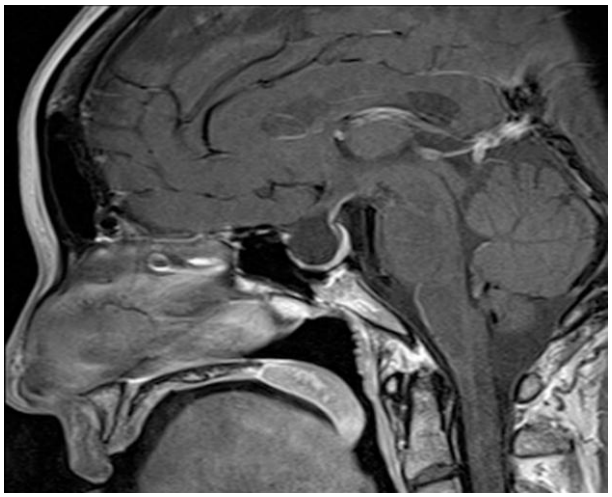

Figure 1: Sagittal T1-weighted post-contrast MRI of the brain.

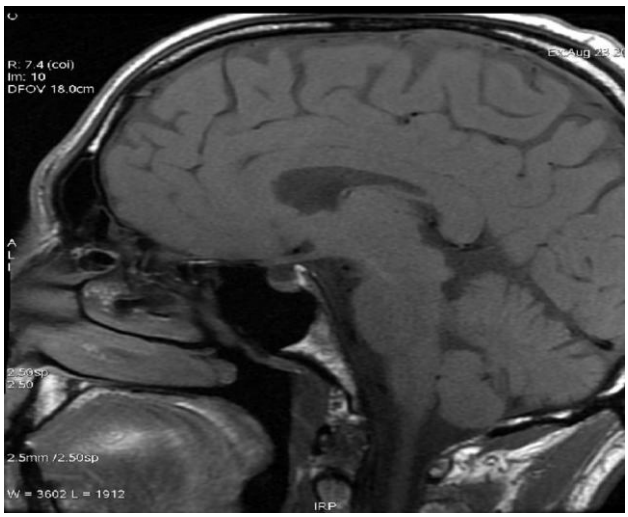

Figure 2: Sagittal t1-weighted MRI without contrast.

Final diagnosis

The diagnostic assessment confirmed secondary adrenal insufficiency likely due to pituitary dysfunction, with partial empty sella as the probable underlying etiology. The diagnosis was supported by hormonal assays, neuroimaging, and consistent clinical findings of hypoglycemia, fatigue, and improved response to steroid therapy.

Therapeutic interventions

The patient was managed with a multidisciplinary approach targeting hypoglycemia, seizures, and adrenal insufficiency. She received IV dextrose (10% and 50%) in varying volumes, with glucose levels closely monitored. Seizures were controlled with clonazepam 0.5 mg.

Hydrocortisone (100 mg every 6 hours) and dexamethasone (4 mg) were administered for suspected adrenal insufficiency. High-dose vitamin D3 (300,000 IU) and iron supplementation (Ferinject 500 mg weekly) were given for metabolic support.

Supportive treatments included pantoprazole for gastric protection, clomipramine for systemic symptoms, and 20% human albumin infusions for low albumin levels. Hormonal and biochemical tests guided medication adjustments, with regular input from endocrinology and neurology teams.

Follow up and outcomes

After initiating intravenous hydrocortisone, the patient's hypoglycemia and seizures resolved, confirming secondary adrenal insufficiency. She was stabilized and discharged on oral corticosteroids, with endocrine follow-up planned for long-term hormone replacement. Supportive treatments like vitamin D and iron supplementation addressed underlying deficiencies. No further seizures or hypoglycemic episodes were reported during follow-up. Patient education covered stress-dose steroid use and emergency precautions. Regular endocrinology reviews ensured therapy adjustments and monitoring. This case underscores the value of early recognition and treatment of pituitary-related adrenal insufficiency, particularly in atypical presentations like seizures and refractory hypoglycemia due to partial ESS.

DISCUSSION

This case underscores the diagnostic and therapeutic challenges of secondary adrenal insufficiency (SAI), especially when the initial symptoms—such as seizures, lightheadedness, and episodes of low blood sugar—are vague and non-specific. The combination of reduced ACTH levels and imaging findings consistent with partial empty sella points toward an underlying pituitary disorder. This is consistent with previous reports where structural abnormalities in the pituitary region have been identified as the root cause of SAI.[7]

The patient's condition improved notably following the immediate administration of intravenous hydrocortisone and glucose, highlighting the critical role of early detection and intervention. Comparable cases have demonstrated that swift initiation of corticosteroid therapy can lead to rapid clinical stabilization in individuals with adrenal insufficiency.[8]

In contrast, postponing diagnosis and treatment may lead to serious outcomes, such as adrenal crisis and a higher risk of death.[9]

Effective SAI management goes beyond steroid replacement; modified-release hydrocortisone better mimics natural cortisol rhythms and improves metabolic control and quality of life, as shown by Isidori et al.[10] However, individual responses vary, underscoring the need for personalized dosing and regular monitoring.

This case is unique due to the rare presentation of secondary adrenal insufficiency with seizures and persistent hypoglycemia as primary symptoms—without classic signs like hypotension or severe electrolyte imbalance. Unlike typical cases of partial ESS, which are often asymptomatic or mild, this patient's condition manifested with refractory hypoglycemia and seizures, only responsive to corticosteroid therapy. This highlights the under-recognized link between cortisol deficiency and neuroglycopenic symptoms. The case emphasizes the importance of considering central adrenal insufficiency in adults with unexplained seizures and hypoglycemia, as supported by literature.[11-13]

ESS, though rare, can cause pituitary hormone deficiencies like adrenal insufficiency. In this case, the patient's seizures and hypoglycemia were linked to pituitary dysfunction due to partial empty sella seen on MRI. Imaging showed a small pituitary gland without tumors, confirming a central cause of low adrenal hormone levels. Rapid improvement with hydrocortisone and glucose highlighted the importance of early diagnosis and treatment. Long-term management requires tailored hormone replacement to mimic natural cortisol rhythms, improving patient outcomes and quality of life.

CONCLUSION

This case shows how challenging it can be to diagnose secondary adrenal insufficiency when it presents with vague symptoms like seizures and low blood sugar. Reporting this from a tertiary care hospital in the UAE is important because it highlights the need for doctors in the region to consider pituitary problems in similar patients. It also emphasizes how useful brain imaging and teamwork between specialists are in making the right diagnosis and providing the best care. By sharing this case, we hope to raise awareness among healthcare providers in the Middle East and help improve early detection and treatment of such rare but serious conditions.

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