Case Report

Hyponatremia associated with Chronic Pituitary Apoplexy Masquerading as SIADH

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Abstract:

Pituitary apoplexy, while classically acute, can present insidiously as chronic apoplexy, posing significant diagnostic challenges. We report a 60-year-old male with multiple comorbidities who presented with progressive weakness and neck pain following a gastrointestinal illness. He was found to have severe symptomatic hyponatremia (Na+ 115 mmol/L) with a biochemical profile suggestive of the Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH). Brain MRI demonstrated sellar–suprasellar pituitary mass with heterogeneous signal intensity and hemorrhagic components, showing peripheral rim enhancement on gadolinium-enhanced. Further evaluation revealed a profoundly low serum cortisol (< 1.8 µg/dL) and low prolactin. This case underscores that a patient with adrenal insufficiency secondary to chronic pituitary apoplexy may present as hyponatremia with SIADH-like features.

Keywords: *Adrenal insufficiency, Hyponatremia, Pituitary apoplexy, SIADH.*

Introduction

Pituitary apoplexy is an acute clinical syndrome caused by hemorrhage or infarction of a pituitary gland, usually within an adenoma. It usually presents with sudden headache, vomiting, visual deficits, and hypopituitarism.[1] In contrast, "subclinical" or "chronic" apoplexy refers to a less symptomatic event where imaging reveals hemorrhagic or degenerative changes without the classic acute clinical syndrome.[2] Patients with chronic apoplexy often present insidiously with symptoms of hypopituitarism, which may be unmasked by physiological stress. Secondary adrenal insufficiency is a common and dangerous sequelae, frequently manifesting as severe hyponatremia that mimics SIADH. Distinguishing between primary SIADH and hyponatremia due to cortisol deficiency is critical, as the management diverges fundamentally; fluid restriction versus glucocorticoid replacement.[3] We present a case where chronic pituitary apoplexy presented with life-threatening hyponatremia, highlighting the diagnostic pitfalls and the imperative for timely endocrine evaluation.

Case Presentation:

A 60-year male with a history of insulin-dependent diabetes mellitus, hypertension (On Indapamide followed by hydrochlorothiazide and irregular beta-blocker use), ischemic heart disease and primary hypothyroidism on levothyroxine 50 mcg once daily presented with a two-week history of progressive, debilitating generalized weakness and intermittent, non-specific neck ache. His symptoms began after an episode of diarrhoea and vomiting, for which he was hospitalized at a local facility and discharged after symptomatic improvement. He denied of fever, visual changes, diplopia, headache or any history of head trauma. On further inquiry, it was noted that he had a prior episode of hyponatremia, with a documented serum sodium level of 131 mmol/L, a month back.

On admission to our centre, the patient was euvolemic, lethargic, drowsy but communicating and oriented to time, place and person. On examination, Blood pressure 150/90 mmHg, heart rate 98 bpm, afebrile, oxygen saturation 98% on room air. Neurological examination revealed a Glasgow Coma Scale (GCS) of 14. Cranial nerve assessment showed no visual field defects on confrontation, with full extraocular movements without nuchal rigidity.

The admission serum electrolytes revealed Sodium 115 mmol/L, Potassium 4.41 mmol/L. Initially, the hyponatremia was suspected to be secondary to Hydrochlorothiazide and Indapamide and they were discontinued after admission.

Serum osmolality was 245 mOsm/kg with an elevated urine osmolality (275 mOsm/kg). Spot urinary sodium was 56.1 mmol/L. All these lab findings were compatible with a SIADH-like biochemical profile. Blood tests for renal function, serum liver enzymes, and complete blood count were within normal limits.

Magnetic Resonance Imaging (MRI) of the brain with pituitary protocol was done and it demonstrated a >15 mm sellar–suprasellar pituitary mass with heterogeneous T2-weighted signal intensity and hemorrhagic components, showing peripheral rim enhancement on gadolinium-enhanced T1-weighted images, consistent with pituitary apoplexy. (Figure 1, 2).

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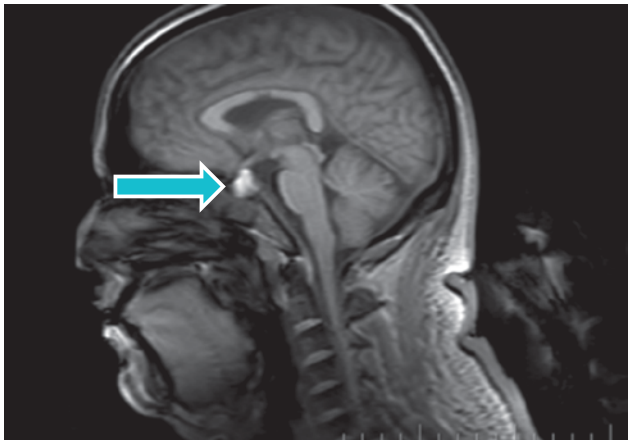

Figure 1: MRI of brain (T1-weighted)

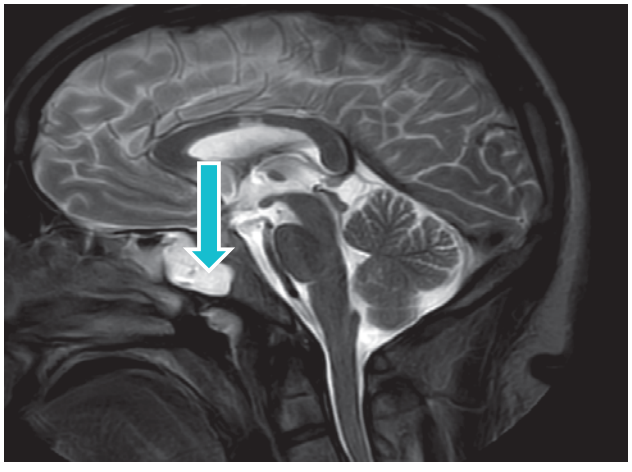

Figure 2: MRI of brain (T2-weighted)

The endocrine workup revealed low serum cortisol levels (9 AM cortisol: <1.82 µg/dL [normal: 5–25 µg/dL]; 5 PM: 1.81 µg/dL [normal: 5–25 µg/dL]). Prolactin was markedly reduced at 1.9 ng/mL (normal: 4.0–15.2 ng/mL). Thyroid functon tests demonstrated a low TSH of 0.008 mIU/L (normal: 0.4–4.0 mIU/L), while the free T4 level remained within the normal range at 1.2 ng/dL (normal: 0.8–1.8 ng/dL). These findings were consistent with ongoing levothyroxine replacement therapy.

Measurement of growth hormone (GH) and adrenocorticotropic hormone (ACTH) levels could not be performed as the assays were not available.

Given the severity of hyponatremia, intravenous 3% NaCl was initiated cautiously, aiming for a sodium correction rate of no more than 6–8 mmol/L in the first 24 hours. Following treatment with hypertonic saline and oral sodium replacement, the next day the serum sodium level became 118 mmol/L. Upon receiving the critically low cortisol result, a diagnosis of adrenal insufficiency secondary to hypopituitarism from chronic pituitary apoplexy was made. Intravenous hydrocortisone was immediately administered at a stress dose (100 mg bolus, followed by 50 mg every 6 hours).

A repeat prolactin measurement using serial dilution to evaluate for a possible Hook effect also yielded a low prolactin level. The biochemical evaluation revealed a low serum testosterone level of 0.35 ng/mL (normal: 2.5–8.4 ng/mL) with an inappropriately normal luteinizing hormone (LH) level.

After clinical and biochemical improvement- evidenced by resolution of lethargy, restoration of normal mental status, hemodynamic stability, and normalization of serum sodium to 135 mmol/L, the patient was transferred to the neurology inpatient department. After two days of observation, the patient was discharged in stable condition with advice to carry a steroid treatment card and to attend follow-up appointments with the Neurology and Endocrinology out patient departments two weeks later.

Discussion:

Acute pituitary apoplexy is a clinical entity resulting from acute hemorrhage or infarction of the pituitary gland typically within a pre-existing adenoma.[2,4] It may present with sudden onset of headache, visual deficits, ophthalmoplegia, and hormonal dysfunction caused by rapid sellar expansion. In contrast, chronic pituitary apoplexy refers to a subacute or indolent form characterized by a slower, often insidious progression of symptoms over weeks to months, without the dramatic neuro-ophthalmic crisis.[5]

This case is a quintessential example of how chronic pituitary apoplexy can present not with neurological catastrophe, but with a severe metabolic disturbance—like hyponatremia which dominates Syndrome of Inappropriate Anti Diuretic Hormone (SIADH) like clinical picture. The patient's pre-existing pituitary macroadenoma likely underwent prior silent hemorrhage or infarction (chronic apoplexy) at an indeterminate time, resulting in partial hypopituitarism. The preadmission gastrointestinal illness acted as a physiological stressor, precipitating an acute adrenal crisis in the setting of inadequate cortisol reserve.[6]

The biochemical profile was classic for SIADH, but the etiology was secondary adrenal insufficiency. Cortisol exerts a tonic inhibitory effect on hypothalamic vasopressin (ADH) secretion. In its absence, unopposed ADH action leads to water retention and impaired free water excretion.[7] Furthermore, the patient's diarrhoea, vomiting and probable concomitant mineralocorticoid deficiency contributed to hypovolemia, another potent stimulus for ADH release. The ongoing use of indapamide followed by hydrochlorothiazide, which impairs urinary dilution in the distal tubule, served as a significant exacerbating factor.[3]

In our case, the key to diagnosis was MRI demonstrating a sellar mass with altered signal intensity and suprasellar extension, thereby confirming the anatomical substrate underlying the endocrine dysfunction.[2,4] Based on these findings, a comprehensive hormonal evaluation was undertaken, which revealed low serum cortisol and prolactin levels. The reduced prolactin level, (a pituitary-derived hormone), together with an inappropriately low thyroid-stimulating hormone despite a low-normal free

thyroxine level (on replacement therapy) suggested central hypothyroidism. These findings also provided further supportive evidence of panhypopituitarism.[8]

The persistently low prolactin level even after sample dilution in this case represents a critical and diagnostically challenging finding that extends beyond the classic high-dose Hook effect. While serial dilution is the standard method to unmask the Hook effect and confirm true hyperprolactinemia.[9,10] Its failure to elevate the prolactin level necessitates consideration of alternative pathologies. These scenario strongly suggests the presence of a non-functioning pituitary macroadenoma causing stalk compression and secondary hypoprolactinemia. Or it may also be suggestive of a hemorrhagic or infarcted (apoplectic) prolactinoma where the causing secretory tissue to be destroyed.[5,11]

The management in our case successfully highlighted two critical tenets. Cautious use of hypertonic saline is indicated for severe, symptomatic hyponatremia to mitigate cerebral edema. The definitive therapy for hyponatremia of hypothalamic-pituitary-adrenal (HPA) axis failure is glucocorticoid replacement. The patient showed rapid and sustained clinical improvement following hydrocortisone administration, before significant correction of serum sodium with saline. This response strongly supports our final diagnosis and highlights that corticosteroid therapy in this setting was not only supportive but was also disease-modifying.[6,8]

Conclusion

Chronic pituitary apoplexy is a deceptive clinical entity that can present with severe, life-threatening hyponatremia mimicking SIADH. A history of prior, milder hyponatremia may represent a prior, subclinical episode of hormone deficiency unmasked by minor stress. Since diuretics are a common cause of hyponatremia, their intake should not prematurely close the diagnostic search. Severe hyponatremia in patients with pituitary pathology should raise immediate suspicion for adrenal insufficiency until it is formally excluded.

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