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(CASE REPORT)

From an obstetric history to severe hyponatremia: Late diagnosis of Sheehan's syndrome presenting as hypopituitarism. Case report

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Abstract

Sheehan's syndrome is a rare but potentially underdiagnosed cause of acquired hypopituitarism, resulting from ischemic necrosis of the pituitary gland following severe obstetric hemorrhage. Due to the nonspecific and progressive nature of its clinical manifestations, diagnosis may be delayed for years.

We present the case of a 49-year-old woman who presented with a 15-day history of persistent vomiting, asthenia, and paresthesias in the extremities. Initial workups revealed severe hyponatremia and hypochloremia. During the etiological evaluation, central hypothyroidism, hypogonadotropic hypogonadism, and hyperprolactinemia were documented. Physical examination revealed bradypsychia, cold and dry skin, and absence of body hair. Given the suspicion of hypopituitarism, a targeted interview revealed a history of severe postpartum hemorrhage 26 years prior. Magnetic resonance imaging (MRI) of the sella turcica showed findings consistent with an empty sella. Based on the integration of clinical, biochemical, and radiological findings, a diagnosis of hypopituitarism secondary to Sheehan's syndrome was established.

Keywords: Hyponatremia; Hypothyroidism; Hypopituitarism; Sheehan's syndrome

1. Introduction

Hypopituitarism (HP) was first described in humans by Simmonds in 1914 (1) and constitutes a complex endocrine syndrome resulting from insufficient secretion of one or more anterior pituitary hormones, with variable involvement of the posterior pituitary and functional abnormalities that may present acutely or insidiously. (2,3) Its clinical spectrum depends on the magnitude, rate of onset, and sequence of hormonal loss, with growth hormone and gonadotropin deficiency being the most common in the early stages, while ACTH and TSH deficiency is typically associated with more severe clinical presentations and higher morbidity and mortality. (2,3) In recent decades, the pathophysiological understanding of HP has evolved considerably, with recognition of a heterogeneous etiology that includes sellar and parasellar tumor lesions, infiltrative processes, autoimmune diseases, vascular causes, traumatic brain injury, infectious sequelae, and obstetric complications. (2,3)

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Among these, SS continues to be one of the classic and potentially underdiagnosed causes of acquired hypopituitarism in women, particularly in low- and middle-income countries where limitations in timely access to specialized obstetric care persist. (4,5) SS occurs secondary to ischemic pituitary necrosis following severe obstetric hemorrhage and/or hypovolemic shock during labor or the immediate postpartum period, a condition exacerbated by physiological pituitary hyperplasia during pregnancy, which significantly increases its metabolic demands and vulnerability to hypoperfusion. (3,4) Although historically described as a condition characteristic of postpartum hemorrhage, it is now recognized that even moderate hemodynamic events can trigger pituitary damage in predisposed patients. (4)

SS accounts for approximately 6% to 13.8% of the cases of hypopituitarism reported in various contemporary series. (2,4) However, its true prevalence is likely underestimated due to the nonspecific and progressive nature of its clinical manifestations, as well as the long interval between the triggering obstetric event and the definitive diagnosis, which can span years or even decades. (3,5) Classic initial manifestations include inability to lactate, secondary amenorrhea, and loss of axillary or pubic hair; however, many patients present with nonspecific symptoms such as chronic fatigue, hypotension, neuropsychiatric abnormalities, hypoglycemia, or fluid and electrolyte imbalances, leading to significant diagnostic delays. (4,5)

Among the most severe metabolic complications associated with pituitary insufficiency, hyponatremia stands out, particularly in the presence of adrenocorticotropic insufficiency and, to a lesser extent, concomitant central hypothyroidism. Decreased circulating cortisol leads to inappropriate increases in antidiuretic hormone secretion and alterations in renal excretion of free water, promoting the development of potentially severe euvolemic hyponatremia. (2,3) In this context, delayed identification of SS can lead to severe, life-threatening endocrine-metabolic decompensations, especially when the patient's distant obstetric history is not adequately integrated into the differential clinical diagnosis.

This case report describes a patient who presented to the emergency department with severe hyponatremia and a history of postpartum hemorrhage, suggesting hyponatremia secondary to SS.

2. Case presentation

A 49-year-old woman with no known medical history presented with symptoms that had been present for 15 days, initially characterized by episodes of self-limiting diarrhea lasting 24–48 hours, followed by persistent vomiting, asthenia, and paresthesia in the extremities.

Initial tests revealed severe hyponatremia and hypochloremia. During the etiological investigation, a pattern consistent with central hypothyroidism and hypogonadism was identified. (see Table 1)

Table 1 Initial blood work of patient

Hormones	Baseline	Normal levels
TSH, mUI/L	1.20	0.35 - 4.94
Free T4, ng/dL	<0.420	0.7 - 1.48
Total T4, µg/dL	2.41	4.87 - 11.72
Prolactin, ng/mL	1.92	5.18 - 26.5
LH, mUI/mL	0.5	5.16 - 61.99
FSH, mUI/mL	1.8	26.72 - 133.41
Cortisol, µg/dL	ND	
ACTH, pg/mL	ND	
Sodium, mmol/L	112.2	136 - 145
Chlorine, mmol/L	83.11	95 - 108
Fasting blood glucose, mg/dL	84	70 - 105

ND, not determined.

The physical examination revealed bradypsychia, cold and dry skin, and an absence of body hair. Given the suspicion of hypopituitarism, further questioning revealed a history of severe postpartum hemorrhage 26 years prior, requiring a transfusion of 7 units of blood products and a hysterectomy, which was subsequently associated with an inability to breastfeed and cold intolerance.

In addition, an MRI of the sella turcica was ordered, which revealed an empty sella turcica. (see Figure 1) These findings confirmed the diagnosis of hypopituitarism secondary to Sheehan's syndrome, and hormone replacement therapy for the corticotropin and thyroid axes was initiated.

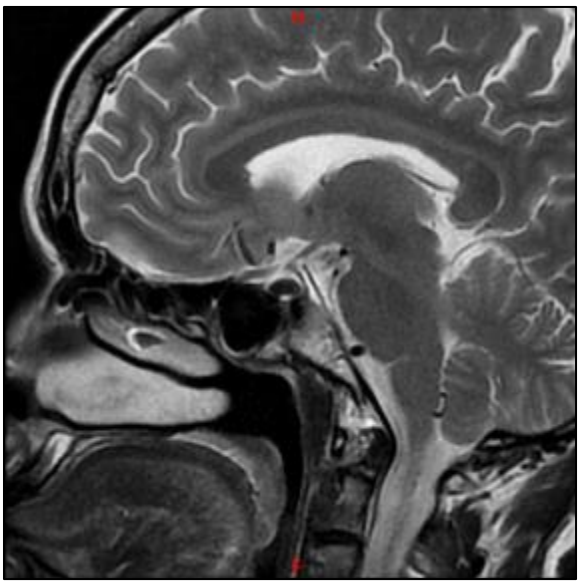

Figure 1 Magnetic resonance imaging of the case showing empty sella appearance

3. Discussion

Postpartum hypopituitarism was first described in 1937; improvements in obstetric care have significantly reduced its incidence. However, postpartum hypopituitarism should always be considered among the possible causes of hypopituitarism in women. A lack of awareness on the part of the physician can lead to delays in the diagnosis and treatment of PS. (6,4) Actual epidemiological data on postpartum hypopituitarism may be underestimated globally, with a higher incidence in countries with limited access to care. (5)

In our case, the patient initially presented with nonspecific symptoms, along with a striking history of postpartum hemorrhage, which led us to consider postpartum hypopituitarism as a possible cause. This highlights the importance of a targeted medical history for an appropriate diagnostic approach.

A significant diagnostic limitation in this case was the inability to obtain cortisol and ACTH levels prior to the initiation of glucocorticoid therapy, due to institutional constraints. Although it was not possible to biochemically confirm involvement of the adrenocorticotropic axis, given the coexistence of severe hyponatremia, central hypothyroidism, a history of obstetric hemorrhage, and failure to lactate, empirical initiation of glucocorticoid replacement was prioritized to prevent potentially fatal adrenal decompensation prior to starting levothyroxine.

Obstetric history, inability to breastfeed, and secondary amenorrhea are key indicators; sparse axillary and pubic hair and dry skin are typical signs of SS. Hyponatremia may be present in 21–59% of patients and should therefore be considered in the differential diagnosis of hyponatremia. (4) The diagnosis of SS is often delayed by up to 20 years due to its insidious onset and subtle manifestations, making a high index of suspicion essential. Consequently, timely recognition of SS continues to represent a multidisciplinary diagnostic challenge, highlighting the importance of a targeted obstetric history, early clinical suspicion, and a comprehensive endocrinological approach in women with unexplained fluid and electrolyte disturbances.

4. Conclusion

The diagnosis and the treatment of hypopituitarism are often delayed for a long time, which leads to a reduced quality of life for patients. Early diagnosis of hypopituitarism is essential. The typical presentation usually includes panhypopituitarism, a history of postpartum amenorrhea, and an inability to breastfeed. At presentation, hypoglycemia and hyponatremia may be observed, as in our case. Delayed diagnosis can lead to cardiovascular and metabolic disorders, bone loss, reduced quality of life, and cognitive dysfunction, despite adequate hormone replacement therapy, since these therapies do not fully mimic normal physiology.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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