Journal of Health and Aging
Published online at https://tcsuckhoelaohoa.vn

Case Report

SHEEHAN SYNDROME PRESENTING WITH SEVERE HYPONATREMIA: A CASE REPORT

Bao Gia Tran[1,*], Nhu Thao Tran[1], An Thi Hoang Nguyen[2], Vy Thanh Nguyen[1,2], Binh Hoa Pham[1,2], Hong Thi May Nguyen[2]

1. Department of Geriatrics and Gerontology, School of Medicine, University of Medicine and Pharmacy at Ho Chi Minh City, Ho Chi Minh City, Vietnam
2. Department of Endocrinology, Thong Nhat hospital, Ho Chi Minh city, Vietnam

* *Corresponding author: Bao Gia Tran* ✉ *bs.trangiabao99@gmail.com*

ABSTRACT: Sheehan syndrome, characterized by pituitary necrosis secondary to postpartum hemorrhage, results in partial or complete hypopituitarism. Diagnosis is often delayed due to its nonspecific clinical manifestations; however, the condition can lead to life-threatening complications such as hyponatremia. We present the case of a 56-year-old woman admitted with symptomatic severe chronic hyponatremia, who was subsequently diagnosed with panhypopituitarism caused by Sheehan syndrome. The patient was successfully managed with hypertonic saline infusion and hormone replacement therapy (levothyroxine and hydrocortisone). This case emphasizes the importance of obtaining a thorough obstetric history and maintaining a high index of suspicion for Sheehan syndrome in middle-aged women presenting with unexplained hyponatremia.

Keywords: Sheehan syndrome, hyponatremia, hypopituitarism

Received: 12/09/2025 Accepted: 20/10/2025 Published: 01/11/2025

1. INTRODUCTION

Sheehan syndrome was first described in 1937 by Harold Sheehan and refers to pituitary necrosis leading to hypopituitarism as a consequence of postpartum hemorrhage [1]. Sheehan syndrome accounts for approximately 6 – 8% of all causes of hypopituitarism [2]. Its prevalence ranges from 0.005% to 3%, depending on the study population and the quality of obstetric care [3, 4]. The clinical spectrum is diverse, varying from agalactia, amenorrhea, and chronic fatigue to life-threatening emergencies such as acute adrenal insufficiency or severe hyponatremia [5]. Therefore, early recognition and timely treatment of Sheehan syndrome are crucial to prevent life-threatening complications and to improve patients' quality of life.

2. CASE REPORT

A 56-year-old woman was admitted to the hospital due to nausea, with a long-standing history of generalized fatigue, poor appetite, and recurrent nausea that had significantly impaired her daily activities. Her past medical history included gastritis, constipation, and vestibular disorder. Current medications were omeprazole and betahistine. On physical examination, vital signs were stable: blood pressure 110/70 mmHg, heart rate 60 bpm, respiratory rate 18 breaths/min, temperature 36.8°C, and oxygen saturation 98% on room air. The patient was alert but slow to respond, with pale skin and mucous membranes. No other abnormalities were found on systemic examination.

Initial laboratory results revealed hyponatremia (serum sodium 104 mmol/L), hypokalemia (serum potassium 3.3 mmol/L), hypochloremia (serum chloride 74 mmol/L), and random plasma glucose 6.9 mmol/L. Complete blood count showed normocytic normochromic anemia (Hb 112 g/L, MCV 80 fL, MCH 27 pg) with normal white blood cell and platelet counts. Renal and liver function tests were within normal limits.

The patient was admitted to the Department of Endocrinology with a diagnosis of severe hyponatremia. Further testing demonstrated low serum osmolality (214.9 mOsm/kg), urinary sodium 41 mmol/L, and urinary potassium 17 mmol/L. Endocrine hormone assays (Table 1) supported a diagnosis of hyponatremia secondary to hypopituitarism. On further questioning, the patient reported a history of massive postpartum hemorrhage at age 35 that required transfusion of five units of blood. After delivery, she had no lactation and experienced permanent amenorrhea. She denied diarrhea or vomiting during the current illness.

Pituitary magnetic resonance imaging with contrast was consistent with an empty sella (Figure 1). The patient was treated with hypertonic saline (3% sodium chloride) and initiated on hormone replacement therapy, including hydrocortisone 20 mg in the morning and 10 mg in the afternoon. After 4 days of adrenal hormone replacement, levothyroxine was introduced at a dose of 25 µg daily. Following treatment initiation, her mental status markedly improved, and she became more responsive. Laboratory results also improved, with gradual correction of serum sodium, reaching the normal range (138 mmol/L) at the time of discharge.

Table 1. Patient's hormone test results

Blood test	Result	Unit	Ref
Cortisol 8 AM	1,3	µg/dL	3,7 – 19,4
ACTH 8 AM	20,3	pg/mL	7,2 – 63,6
TSH	0,97	µIU/mL	0,35 – 4,94
Free T4 (fT4)	0,09	ng/dL	0,932 – 1,71
Total T3	0,195	ng/dL	0,846 – 2.02
FSH	2,78	IU/L	25,8 – 134,8
LH	0,28	IU/L	7,7 – 58,5
IGF – 1	15,8	ng/mL	67.3 – 201
Estradiol	< 5	pg/mL	< 138

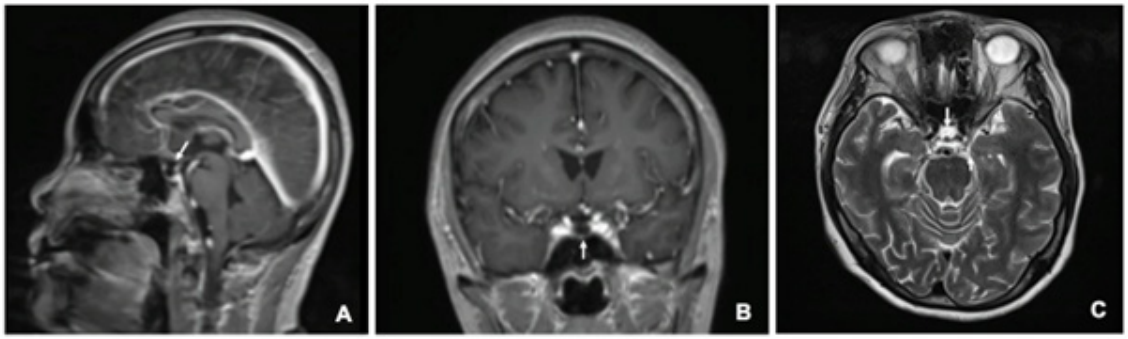

Figure 1. Sagittal T1-weighted brain MRI with gadolinium contrast shows an empty sella: enlarged sella turcica filled with cerebrospinal fluid, with a thin rim of flattened pituitary tissue along the sellar floor (arrow), and no evidence of mass lesion (A). Coronal T2-weighted pituitary MRI demonstrates an enlarged sella completely filled with cerebrospinal fluid, with a slender, midline pituitary stalk and no mass effect or displacement (B). Axial T2 FLAIR TSE pituitary MRI reveals a widened sella turcica with homogenous CSF-like hyperintensity, compressed pituitary tissue flattened against the sellar floor, and no space-occupying lesion (C).

3. DISCUSSION

3.1. Diagnosis of Sheehan syndrome

In most cases, Sheehan syndrome presents with nonspecific symptoms that contribute to a delayed diagnosis, often occurring 7 – 19 years after the postpartum hemorrhage [3, 6, 7]. Diagnosis requires integration of medical history, clinical features, endocrine testing, and pituitary imaging. The diagnostic criteria proposed by Zuleyha Karaca [5] are summarized in table 2. Patients typically report a history of severe postpartum hemorrhage requiring blood transfusion, followed by failure of lactation and amenorrhea. Clinically, the disease may manifest acutely soon after delivery with headache, altered consciousness, agalactia, and signs of acute adrenal insufficiency such as hypotension, hyponatremia, hypoglycemia, and nausea. However, in the majority of cases, symptoms appear years later with chronic, nonspecific complaints such as fatigue, myalgia, constipation, cold intolerance, muscle weakness, and weight loss. Characteristic features include sparse axillary and pubic hair, breast atrophy, dry hypopigmented skin, and premature aging appearance.

Hyponatremia is the most common biochemical abnormality and often the primary reason for hospital admission. Endocrine evaluation demonstrates varying degrees of hypopituitarism: decreased prolactin and gonadotropins leading to agalactia and amenorrhea; reduced TSH and free T4 consistent with secondary hypothyroidism; and low cortisol due to secondary adrenal insufficiency. In some cases, dynamic stimulation tests may be necessary for confirmation. Additional findings may include anemia or electrolyte disturbances, among which hyponatremia is the most frequent.

Neuroimaging, particularly magnetic resonance imaging (MRI), plays a crucial role. In the chronic stage, the typical finding is a partially or completely empty sella, reflecting pituitary atrophy following necrosis [5].

Table 2. Diagnosis of Sheehan syndrome in the chronic period

	Hypopituitarism (ranging from one hormone deficiency to panhypopituitarism)
Essential criteria for the diagnosis	- Partial or complete empty sella appearence on pituitary MRI - History of postpartum hemorrhage
Strongly suggestive criteria	- Severe hypotension or shock which may necessitate blood transfusion or fluid replacement during and after delivery - Postpartum agalactia - Failure to resume regular menses after delivery

3.2. Severe hyponatremia in Sheehan syndrome

Hyponatremia is the most common electrolyte abnormality in patients with Sheehan syndrome, with reported prevalence ranging from 21% to 59% [8, 9], and in some series reaching nearly 70% among elderly patients [10]. This is a serious complication that may result in altered consciousness, seizures, or even death if not promptly recognized and treated. The underlying pathophysiology involves cortisol and thyroid hormone deficiency, both of which impair free water excretion, combined with secondary antidiuretic hormone (ADH) hypersecretion triggered by hypotension or increased corticotropin-releasing hormone (CRH) in adrenal insufficiency [5].

In our case, a 56-year-old woman presented with severe hyponatremia (serum sodium 104 mmol/L) accompanied by chronic fatigue, nausea, and impaired daily functioning. Laboratory testing revealed multiple pituitary hormone deficiencies, and MRI showed an empty sella, consistent with chronic Sheehan syndrome. After treatment with hypertonic saline and appropriate hormone replacement therapy, her serum sodium gradually normalized and her clinical symptoms markedly improved.

This case highlights that hyponatremia is not only a common biochemical abnormality but also a critical diagnostic clue guiding the recognition of Sheehan syndrome.

3.3. Magnetic resonance imaging in the diagnosis of Sheehan syndrome

Pituitary MRI is the imaging modality of choice in the diagnosis of Sheehan syndrome, especially for differentiating it from other causes of hypopituitarism [5]. MRI findings vary according to the stage of the disease: in the acute phase, non-hemorrhagic central necrosis with pituitary enlargement may be observed; in the chronic phase, pituitary atrophy leads to a partially empty sella (30%) or completely empty sella (70%) [11]. MRI also plays an important role in differential diagnosis. In the acute stage, the imaging appearance of Sheehan syndrome may mimic pituitary apoplexy secondary to an adenoma. However, the persistence of a mass, erosion of the sellar floor, or deviation of the pituitary stalk is more suggestive of an adenoma. Additionally, postpartum lymphocytic hypophysitis may also evolve into an empty sella resembling Sheehan syndrome, highlighting the need for repeated clinical and radiologic follow-up [12].

Therefore, MRI not only confirms the diagnosis of Sheehan syndrome by demonstrating partial or complete empty sella but also helps exclude other potential causes such as pituitary adenoma, pituitary apoplexy, or lymphocytic hypophysitis. In current diagnostic criteria, the presence of a partial or complete empty sella on MRI is considered an essential criterion for establishing the diagnosis of Sheehan syndrome [5].

4. CONCLUSION

This case underscores the importance of carefully obtaining obstetric history in middle-aged women who present with unexplained hyponatremia. Sheehan syndrome is frequently diagnosed late—sometimes decades after delivery—but early recognition and timely initiation of appropriate hormone replacement therapy can significantly improve clinical outcomes and enhance patients' quality of life.

REFERENCES

[1] Sheehan H.L. Postpartum necrosis of the anterior pituitary. American journal of obstetrics and gynecology. 1971;111(6):851. Epub 1971/11/01. PubMed PMID: 4944349.

[2] Regal M, Paramo C, Sierra SM, Garcia-Mayor RV. Prevalence and incidence of hypopituitarism in an adult Caucasian population in northwestern Spain. Clin Endocrinol (Oxf). 2001;55(6):735-40. Epub 2002/03/16. doi: 10.1046/j.1365-2265.2001.01406.x. PubMed PMID: 11895214.

[3] Zargar AH, Singh B, Laway BA, Masoodi SR, Wani AI, Bashir MI. Epidemiologic aspects of postpartum pituitary hypofunction (Sheehan's syndrome). Fertil Steril. 2005;84(2):523-8. Epub 2005/08/09. doi: 10.1016/j.fertnstert.2005.02.022. PubMed PMID: 16084902.

[4] Kristjansdottir HL, Bodvarsdottir SP, Sigurjonsdottir HA. Sheehan's syndrome in modern times: a nationwide retrospective study in Iceland. Eur J Endocrinol. 2011;164(3):349-54. Epub 2010/12/25. doi: 10.1530/EJE-10-1004. PubMed PMID: 21183555.

[5] Karaca Z, Kelestimur F. Sheehan syndrome: a current approach to a dormant disease. Pituitary. 2025;28(1):20. Epub 2025/01/26. doi: 10.1007/s11102-024-01481-1. PubMed PMID: 39863703; PubMed Central PMCID: PMC11762620.

[6] Diri H, Tanriverdi F, Karaca Z, Senol S, Unluhizarci K, Durak AC, et al. Extensive investigation of 114 patients with Sheehan's syndrome: a continuing disorder. Eur J Endocrinol. 2014;171(3):311-8. Epub 2014/06/12. doi: 10.1530/EJE-14-0244. PubMed PMID: 24917653.

[7] Ramiandrasoa C, Castinetti F, Raingeard I, Fenichel P, Chabre O, Brue T, et al. Delayed diagnosis of Sheehan's syndrome in a developed country: a retrospective cohort study. Eur J Endocrinol. 2013;169(4):431-8. Epub 2013/07/19. doi: 10.1530/EJE-13-0279. PubMed PMID: 23864341.

[8] Gei-Guardia O, Soto-Herrera E, Gei-Brealey A, Chen-Ku CH. Sheehan syndrome in Costa Rica: clinical experience with 60 cases. Endocr Pract. 2011;17(3):337-44. Epub 2010/11/03. doi: 10.4158/EP10145.OR. PubMed PMID: 21041170.

[9] Lim CH, Han JH, Jin J, Yu JE, Chung JO, Cho DH, et al. Electrolyte Imbalance in Patients with Sheehan's Syndrome. Endocrinol Metab (Seoul). 2015;30(4):502-8. Epub 2015/10/21. doi: 10.3803/EnM.2015.30.4.502. PubMed PMID: 26485467; PubMed Central PMCID: PMC4722405.

[10] Kurtulmus N, Yarman S. Hyponatremia as the presenting manifestation of Sheehan's syndrome in elderly patients. Aging Clin Exp Res. 2006;18(6):536-9. Epub 2007/01/27. doi: 10.1007/BF03324855. PubMed PMID: 17255644.

[11] Schury MP, Adigun R. Sheehan Syndrome. StatPearls. Treasure Island (FL) ineligible companies. Disclosure: Rotimi Adigun declares no relevant financial relationships with ineligible companies.2025.

[12] Dejager S, Gerber S, Foubert L, Turpin G. Sheehan's syndrome: differential diagnosis in the acute phase. J Intern Med. 1998;244(3):261-6. Epub 1998/09/25. doi: 10.1046/j.1365-2796.1998.00370.x. PubMed PMID: 9747750.