

## Hypoglycemia as presenting manifestation of Sheehan's syndrome

Mandeep Singla, Geetika Garg

Government Medical College and Hospital, Chandigarh, India

### Abstract

Sheehan's syndrome is caused by ischemic necrosis of the pituitary gland from massive postpartum uterine bleeding. Despite advances in obstetric care, it is still an important cause of hypopituitarism in developing countries. Sheehan's syndrome has an insidious course with variable presentation. Here, we present a case having recurrent hypoglycemia and was later found to have hypopituitarism due to Sheehan's syndrome. Despite the presence of lactation failure and secondary amenorrhea following the delivery, she was diagnosed ten years later. Awareness of these clues and prompt evaluation for pan hypopituitarism can significantly reduce the morbidity and mortality in these patients.

**Keywords:** sheehan's syndrome, hypoglycemia, hypopituitarism, empty sella

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**Correspondence email:** [drmandeep2007@gmail.com](mailto:drmandeep2007@gmail.com)

### Introduction

Sheehan's syndrome is caused by ischemic necrosis of the pituitary gland from massive postpartum uterine bleeding. It was first described by Sheehan in 1937(1). With advancements of obstetrical care, Sheehan's syndrome has become uncommon in the western countries. However, it is still an important cause of hypopituitarism in developing countries. Sheehan's syndrome has an insidious course with variable presentations. The majority of cases remain undiagnosed for many years after delivery. Here, we present a case of Sheehan's syndrome with recurrent hypoglycemia as the presenting manifestation.

### Case

A 40-year-old female presented to the emergency department in a state of altered mentation. She was confused and sweating profusely. Her blood glucose level was 37 mg/dl. She regained consciousness after the administration of dextrose infusion. She had episodes of palpitations, blurred vision, and confusion for the past four years, which were relieved on eating. She had weight loss of twelve kilograms during the same period. She

was non-diabetic, not on any medication, and had no known malignancy. General physical examination revealed pallor, dry and coarse skin, facial wrinkling, absent axillary and pubic hair, and breast atrophy, suggestive of hypopituitarism. Her body mass index was 18.5 kg/m<sup>2</sup>. On eliciting a detailed history, she had lactation failure and secondary amenorrhea since her last child birth ten years back. She had received six unit of blood due to postpartum haemorrhage. Biochemical investigations revealed low haemoglobin (9.7g/dl) and hyponatraemia (124meq/l). Hormonal profile revealed panhypopituitarism: freeT3 1.2 pg/ml ( 2.3–4.2), freeT4 0.6 ng/ml (0.89–1.76), thyroid-stimulating hormone(TSH) 0.84mU/l ( 0.35–5.5), follicle-stimulating hormone(FSH) 1.8 IU/l ( 2.5–10.2), luteinizing hormone(LH) <0.1 IU/l ( 1.9–12.5), prolactin 1.3ng/ml ( 4–25), insulin-like growth factor-1(IGF-1) 78.6 ng/ml ( 109–284) , cortisol 68 nmol/l ( 100–550), and adrenocorticotrophic hormone(ACTH) 2.3 ng /l (5–60). Magnetic resonance imaging of the brain revealed thinning of anterior pituitary gland with extension of suprasellar cistern CSF into sella (Figure 1 & 2), thus confirming the diagnosis of Sheehan's syndrome. She was treated with oral hydrocortisone followed by levothyroxine supplementation. Hormone replacement therapy was instituted for gonadotropin

deficiency. Following treatment, she started gaining weight and the hypoglycemic symptoms abated.

## Discussion

Sheehan's syndrome is defined as pituitary hormone deficiency due to ischemic necrosis of the pituitary gland from massive postpartum uterine bleeding. Enlargement of pituitary gland, small sellar size and disseminated intravascular coagulation are considered to play a role in its pathogenesis. Anti-pituitary antibodies have also been demonstrated in patients with Sheehan's syndrome, suggesting an underlying autoimmune etiology (2). Extensive destruction of cells results in varying degrees of anterior pituitary dysfunction. It is one of the common causes of hypopituitarism in developing countries. An epidemiological study from Kashmir valley of India reported the prevalence to be around 3% for women above 20 years of age, almost two-thirds of whom had delivered babies at home(3).

It can manifest in the postpartum period or after months to years following the inciting delivery. The majority of cases remain undiagnosed for many years after delivery. In a study of 60 patients, the mean duration between the inciting delivery and the diagnosis was 13 years (4). Characteristic manifestations include failure to lactate or to resume menstruation, genital and axillary hair loss, asthenia and weakness, dryness and wrinkling of the skin, signs of premature aging and hypopigmentation(5). Uncommonly, it

can present acutely with circulatory collapse, hyponatraemia, diabetes insipidus, hypoglycemia, or psychosis. Hypoglycaemia in Sheehan's syndrome is attributed to the deficiency of glucose counterregulatory hormones, including cortisol and growth hormone. Deficiency of these hormones results in glycogen depletion and impaired gluconeogenesis.

The characteristic radiological finding is the presence of an empty sella or partially empty sella. Treatment is aimed at replacing the deficient hormones and to reduce the mortality due to hypopituitarism. In patients with both secondary hypocortisolism and hypothyroidism, glucocorticoids should be replaced first followed by thyroid hormone supplementation. Gonadotropin deficiency should be treated with a hormone replacement therapy. Replacement of growth hormone should be considered in patients with GH deficiency to maintain insulin-like growth factor-1 levels within the age-appropriate range for the patient.

## Conclusion

Sheehan's syndrome is still an important cause of hypopituitarism in developing countries. This case highlights the diverse manifestations of Sheehan's syndrome. History of postpartum hemorrhage, failure to lactate and cessation of menses are important clues to the diagnosis. Awareness of these clues and prompt evaluation for panhypopituitarism can significantly reduce the morbidity and mortality in these patients.

## References

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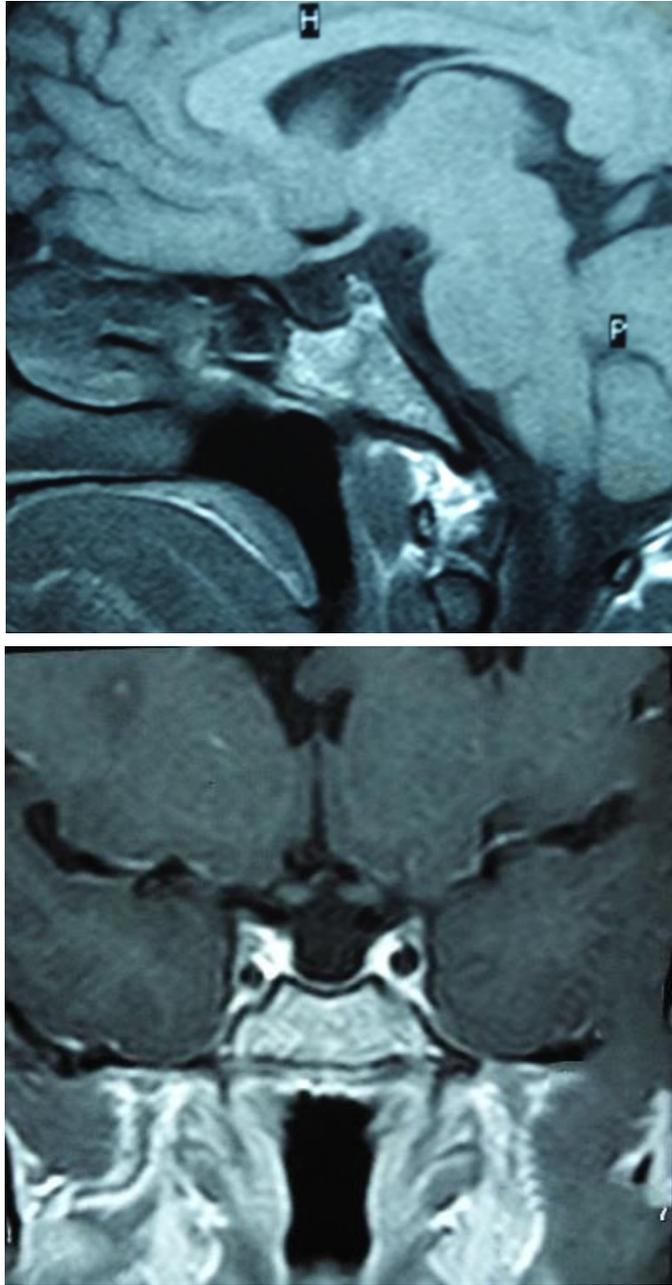


Figure 1, Figure 2: Saggital and coronal sections of Magnetic resonance imaging of the brain showing thinning of anterior pituitary gland with extension of suprasellar cistern CSF into sella