

Case Report

Post-partum recurrent hypoglycemia

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ABSTRACT

Hypoglycemia can be effortless to treat. Its evaluation on the other hand, needs a thorough appraisal. The studied patient had recurrent episodes of hypoglycemia post-delivery which were unprovoked. These conspicuous episodes of hypoglycemia prompted an evaluation and a swift arrival at the diagnosis. In hindsight, she had multiple, typical risk factors predisposing her to develop a Sheehan's syndrome. This was an acute presentation of a familiar disease.

Keywords: Hypoglycemia, Sheehan's syndrome

INTRODUCTION

Although management and diagnosis of hypoglycemia is generally believed to be effortless, probing the reason for the same can be challenging. More so when a clinical situation arises when the hypoglycemia is recurrent and more importantly, unprovoked. The various causes of hypoglycemia are mentioned in Table 1.

This case is an example of rare etiologies causing a very common manifestation. In a study by Güven et al, Sheehans syndrome was responsible for 8.73% of patients with hypoglycemia.¹ Sheehan's syndrome is well described in the literature but has the propensity to herald in a lot of mortality.^{2,3}

The physician must be aware that although the sequence of events that was present in this case is typically described for Sheehan's syndrome, there is still a possibility of it getting missed.⁴⁻⁶

Apart from hypoglycemia, a subtle hyponatremia may not be obvious which must bring in the suspicion of a Sheehan's syndrome.⁷⁻¹⁰

CASE REPORT

We present a 37-year-old (gravidity 3, parity 2) patient at a period of gestation of 33 weeks, who was admitted in authors' tertiary care hospital with complaints of bleeding per vaginum for 3 days. She was found to be severely anemic and underwent an emergency cesarean section in view of antepartum haemorrhage (APH) with a low-lying placenta.

She was transfused packed cells peri-operatively. Post-operatively she had an atonic post-partum haemorrhage (PPH) which was successfully managed with uterotonics and transfused packed cells. Post-operative day 2, she complained of generalized malaise and an appetite loss which was initially attributed to her anemia. She had a symptomatic hypoglycemia and in spite of being initiated on a dextrose infusion, she had further recurrent episodes of hypoglycemia. She also complained of transient giddiness while getting up from a lying down position and was found to have orthostatic hypotension. Her blood reports also showed an unsuspected hyponatremia which was not evaluated initially.

Table 1: Causes of hypoglycemia.

Causes of hypoglycemia	
Lifestyle related	
Dietary error	
Excessive physical activity	
Weight loss	
Drug related	
Sulfonamide	
Insulin	
Ethanol	
Quinine	
Thiazide diuretics	
Haloperidol	
Salicylates	
Endocrinological disease	
Hypopituitarism	
Hypothyroidism	
Adrenal Insufficiency	
Nervous system	
Autonomic neuropathy	
Gastrointestinal neuropathy	
Renal failure	
Hepatic failure	
Sepsis	
Other causes	
Gastrointestinal dysfunction/Malabsorption	
Gastrointestinal surgery	
Insulinoma	

Table 2: Blood investigations.

Laboratory parameters	Values
Hemoglobin	5.3 g/dL
Total leucocyte count	9,800 cells/mm ³
Platelet count	1,34,000 cells/mm ³
Venous RBS	40 mg/dl
Blood urea	20 mg/dl
S. Creatinine	0.7 mg/dl
S. Sodium	127 mEq/L
S. Potassium	3.7 mEq/L
S. Chloride	90 mEq/L
S. Calcium	7.5 mg/dL
S. Phosphorus	2.4 mg/dL
Total protein	4.9 mg/dL
Albumin	2.3 mg/dL
Total bilirubin	0.6 mg/dL
Direct Bilirubin	0.2 mg/dL
SGOT	37 U/L
SGPT	11 U/L
ALP	180 U/L
GGT	10 U/L
HBA1c	5.7 %
Serum iron	62 µg/dL
Serum ferritin	368 ng/mL
Serum vitamin B12 levels	747
Direct Ccomb's test	Negative
HIV/ HBsAg/ HCV (ELISA)	Negative

With an initial presentation of recurrent hypoglycemia, hyponatremia, low blood pressure recordings, significant generalized malaise and loss of appetite, adrenal insufficiency was suspected. Basal cortisol was sent and a post 25µg cosyntropin stimulated serum cortisol was also estimated-both of which had come to be extremely low. She was started on Inj. Hydrocortisone, after which her clinical condition including her appetite and blood sugars improved. Incidentally TSH levels were also found to be high for which thyroid hormone replacements were given. In the setting of significant antepartum and postpartum haemorrhage and an already existing hypothyroidism, a possibility of a hypopituitarism developing after an apoplexy of the pituitary (Sheehan's Syndrome) was kept for which a hormonal assay and an MRI of the pituitary was done. Her FSH and LH were found to be extremely low, consistent with hypopituitarism and serum prolactin was high. An MRI of the pituitary confirmed a partial empty sella. The investigations are mentioned in Table 2, 3, 4.

Table 3: Hormonal and antibody assay.

Parameters	Values
FSH	0.3 ↓
LH	0.3 ↓
Prolactin	182 ↑
FT3	3.7 ↓
FT4	6.75 ↓
TSH	33 ↑
Anti TPO Antibodies	17.5 N
Anti TTG	Negative
ANA	Negative
Basal cortisol	0.76 mcg/dL
Cortisol post ACTH stimulation	1.7 mcg/dL

Table 4: Imaging.

Parameters	Description
MRI Pituitary	Features suggestive of a partial empty sella
Antenatal USG	Placenta low lying posterior, placenta previa
	Single live intrauterine pregnancy at approximate gestational age of 32 weeks 1 day in cephalic presentation
	AFI 13.0
Echocardiography	Chronic rheumatic valvular disease
	Mild MS with severe MR (MV Area 2.5 cm ²)
	Severe TR (RVSP 55mmHg)
	Dilated LA, RA
	Good LV and RV systolic function LVEF 55%

Historically, she had breathlessness on exertion for the last 15 years, along with palpitations. Initially

breathlessness was NYHA grade 1 but worsened to NYHA grade 3. At the time of her hospital stay, she was able to climb only one flight of stairs without any difficulty. She had no history of breathlessness at rest, chest pain, cough, vomiting, lower limb swelling, abdominal distension or facial puffiness. On cardiovascular examination, she had a heaving apex, with her apex at the mid clavicular line 5th intercostal space and a parasternal heave present. A grade 3 pansystolic murmur heard better on expiration. An echocardiography scan showed evidence of a rheumatic valvular heart disease. She was started on Inj. hydrocortisone, after which her clinical condition improved (Her appetite and her blood sugars improved). Her intravenous corticosteroids were gradually replaced by oral prednisolone. TSH levels were also found to be high for which thyroid hormone replacements were given.

DISCUSSION

Retrospectively, a pre-existing primary hypothyroidism and an elevated prolactin would have led to a hyperplasia of the pituitary cells causing an increase in vascularity of the pituitary gland thereby making her vulnerable to developing a pituitary apoplexy; with the APH and PPH triggering the cascade leading to an acute anterior hypopituitarism. The pathogenesis of Sheehan's syndrome in studied patient is described in Figure 1.

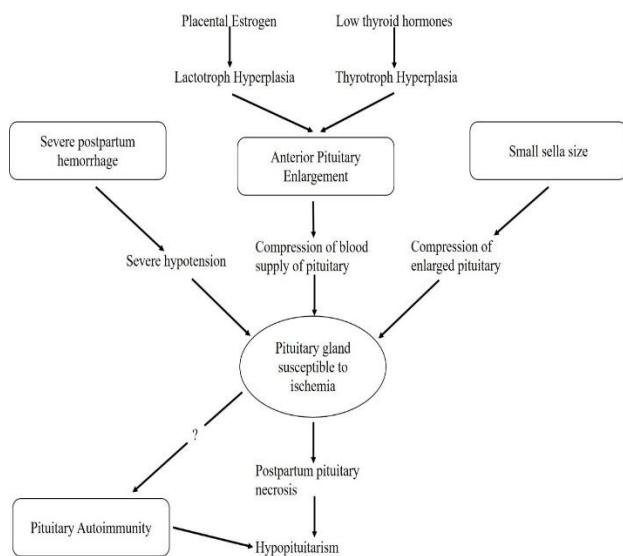


Figure 1: Pathogenesis of Sheehan's syndrome.

An unsuspecting hyponatremia should have prompted an evaluation consequentially leading to a timely diagnosis. Eventually, the recurrent episodes of unprovoked hypoglycemia raised a clinical suspicion of an acute hypopituitarism and timely intervention was instigated.

The common inceptive clinical feature provoking a suspicion of Sheehan's syndrome, are amenorrhoea and lactation failure. In a study by Diri et al, on patients with Sheehan's syndrome, 85.1% patients reported

amenorrhoea starting immediately after delivery and 42.1% patients had lactation failure.¹¹ The most familiar antecedent obstetric circumstance is obstetric hemorrhage (82%).¹² But it should not be forgotten like in our patient, hyponatremia and recurrent hypoglycemia, i.e. partial hormone deficiencies (which preservation of other hormones) can also be the presenting features. Due to a clinical lethargy, often there are delays in arriving at a diagnosis. This was an acute presentation of this familiar disease in a young patient.

CONCLUSION

In regions where Sheehan's syndrome is still common, we must be familiar about the atypical and partial presentations, which are also quite common. The clinical features are often subtle, and a certain delay may ensue before the diagnosis is made. A high index of suspicion and timely management makes its diagnosis and treatment straightforward.

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