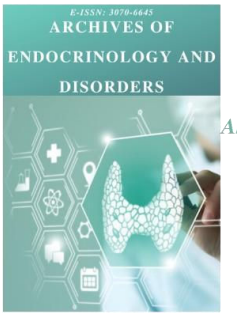

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Case Report

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The Woman in a Veil of Sheehan's Syndrome

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Abstract

Introduction and Importance: Sheehan's Syndrome (SS) is a condition arising due to postpartum necrosis of the pituitary gland. It is usually the result of severe hypotension or shock caused by massive hemorrhage during or after delivery. Patients with SS have varying degrees of anterior pituitary hormone deficiency. Advanced obstetrical care have reduced its incidence. However, it is still a cause of concern in less developed countries. SS often evolves slowly and hence, diagnosed late. History of postpartum hemorrhage, failure to lactate and cessation of menses are important clues to the diagnosis. Early diagnosis and appropriate treatment are important to reduce morbidity and mortality of the patients.

Case Presentation: Here, we describe the case of a 36 year-old female who presented in a drowsy state, in the outpatient department, with sudden onset of 6-7 episodes of clear, colourless vomitus since first day. Initial investigations revealed severe hyponatremia with persistent hypoglycemia. Endocrine studies and pituitary magnetic resonance scan revealed Sheehan's syndrome. She improved significantly once the treatment with hormone replacement therapy was started.

Clinical Discussion: Hyponatremia is an initial symptom of acute Sheehan's syndrome in approximately 60% of the reported cases. Acute hyponatremia can lead to life-threatening cerebral edema. Interestingly, our patient also had recurrent hypoglycemia, a rare presentation. The initial diagnosis depends upon detailed clinical history and physical examination, complemented by relevant laboratory tests and imaging studies.

Conclusion: Physicians should be suspicious of hyponatremia and hypoglycemia as it could be an initial manifestation of hypopituitarism of SS. Hence, the causes of persistent hypoglycemia and recurrent hyponatremia should be worked upon in young females.

Keywords: Hypoglycemia; Hyponatremia; Lactation Failure; Pituitary Necrosis; Postpartum Hemorrhage; Sheehan

Introduction

Sheehan's Syndrome (SS) is a condition arising due to postpartum necrosis of the pituitary gland. It mainly occurs as a result of Post-Partum Haemorrhage (PPH), making the pituitary gland unable to produce hormones. It is often diagnosed retrospectively, sometimes after several years of onset [1]. The first and most common symptom is lactation failure or agalactorrhea. Other symptoms include amenorrhea or oligomenorrhea, hot flashes, fatigue, bradycardia, hypotension, weight gain, constipation, which mimic hypothyroidism and hypocortisolism, besides, loss of axillary and pubic hair [2].

Case Report

A 36 year-old female presented in a drowsy state, in the outpatient department, with sudden onset of 6-7 episodes of clear, colourless vomitus since first day. There is no associated loose stools or fever. Also, there is no past history of similar episode leading to her hospitalization, except for a Total Abdominal Hysterectomy with Bilateral Salpingo-Oophrectomy (TAH-BSO), about 7 years ago. Her physical examination, at the time of admission revealed a confused female patient with stable vitals, without any neurovascular deficit but having mild pallor. She was admitted and treatment with intravenous (iv) normal saline and Ondansetron were started.

A regular workup for blood revealed normocytic, hypochromic anemia with severe hyponatremia and persistent hypoglycemia with plasma glucose ranging from 45 milligram per decilitre (mg/dL) to 52 mg/dL, with a high normal potassium (K+) level. She was started on dextrose saline for hypoglycemia, after sending the critical sample for insulin, c-peptide, growth hormone cortisol and random plasma glucose, her initial Random Plasma Glucose (RPG) was 45 mg/dL. Her hyponatremia, sodium (Na+) of 107 milliequivalent per litre (mEq/L) was corrected with 100 millilitres (mL) of 3% hypertonic saline given iv over 4 hours once along with 2 grams (g) of oral extra salt thrice daily. Her urine spot Na+ was 197 millimoles per litre (mmol/L) with K+ of 4.2 mEq/L. From next day onwards, her vomiting was controlled and hence, Ondansetron was omitted. Soon, her Na+ level returned to normal and hence, fluid was omitted. However, her Na+ level started falling again, hence, 15 mg of Tolvaptan, a Vasopressin Receptor (V-2-R) antagonist was started orally to maintain Na+ level.

She had a history of fasting intolerance for over six months. Remarkably, she had relatively thin, unpigmented extremities and short stature, without any pubic or axillary hair. On further questioning her relatives, it was revealed that she had an episode of PPH during the her last child birth, 7 years ago, leading to TAH-BSO. Since then, she is amenorrhoeic. Also, she does not have any history of steroid intake.

Suspecting differential diagnosis as secondary adrenal insufficiency or Sheehan's syndrome, hormonal assays were conducted. It turned out that she had a low Thyroid Stimulating Hormone (TSH) at 0.45 micro international unit per mL (mcIU/mL), a low free Tetraiodothyronine (fT4) at 0.125 nanogram per dL (ng/dL) and a low 8 am serum cortisol at 4.2 mcg per decilitre (mcg/dL) with low Adrenocorticotropic Hormone (ACTH). However, the result of ACTH or Cosyntropin stimulation test confirmed Sheehan's syndrome. Here critical sample at the time of hypoglycaemia (RPG 45 mg/dl) showed low growth hormone and low cortisol (4.9 mcg/dL) along with suppressed insulin (1.2 pmol/L) and c-peptide levels (0.07 nmol/L). She also had a low prolactin, estradiol and FSH level suggesting pan hypopituitarism. Her Magnetic Resonance Imaging (MRI) brain showed empty sella. Her treatment commenced with oral 25 mg levothyroxine before breakfast, hydrocortisone 10 mg after breakfast and 5 mg after dinner for her low cortisol levels, besides, 2 g extra salt thrice daily for her low Na+ levels. A follow up for several weeks post hospital discharge showed a full restoration of her hormonal and electrolyte imbalance within 6 weeks. The relevant findings of her laboratory and radiological investigations are depicted in Table 1.

Laboratory Investigations	Values	Reference Values
Haemoglobin (Hb)	8.8g/dL	12-15 g/dL
Sodium (Na+)	110 mEq/L	135-145 mEq/L
Potassium (K+)	4.5 mEq/L	3.5-5 mEq/L
Random Plasma Glucose (FPG)	45 mg/dL	70-99 mg/dL
Free Tetriodothyronine (fT4)	0.125 ng/dL	0.7-1.9 ng/dL
Thyroid Stimulating Hormone (TSH)	0.45 mcIU/mL	0.5-5 mcIU/mL
Follicle Stimulating Hormone (FSH)	1.56 mIU/mL	25.8-134.8 mIU/mL (surgical menopause)
Luteinizing Hormone (LH)	0.35 mIU/mL	15.9-54 mIU/mL (surgical menopause)
Estradiol (E2)	12 pg/ml	50-200 pg/ml
Prolactin	2 mIU/L	40-530 mIU/L
Growth Hormone (GH)(critical sample)	0.05 mcg/L	0-5 µg/L
Insulin (critical sample)	1.2 pmol/L	14.35-143.5 pmol/L
Cpeptide (critical sample)	0.07 nmol/L	0.27-1.19 nmol/L

Cortisol (critical sample)	4.9 mcg/dL	5-25 mcg/dL
Adrenal Corticotropic Hormone (ACTH)	7.30 pg/mL	10-60 pg/mL
Insulin like Growth Factor-1 (IGF-1)	<15 ng/mL	70-300 ng/mL
Cosyntropin test: Cortisol @ 8am Cortisol @ 8.30 am (30" after administration of ACTH/ Cosyntropin (250 mcg)) Cortisol @ 9am (60" after administration of ACTH/ Cosyntropin (250 mcg))	2.44 mcg/dL 5.93 mcg/dL 7.40 mcg/dL	5-25 mcg/dL
Urine spot Na^+	197 mEq/L	>20 mEq/L

Table 1: Relevant investigations.

Discussion

The pituitary gland naturally enlarges ante-partum. This makes it more susceptible to low blood flow states caused by a major hemorrhage [1]. The vasoconstriction of hypothalamic-portal vessels following PPH leads to ischemic pituitary necrosis. This leads to a loss of somatotroph and thyrotroph cells, resulting in Sheehan's syndrome [1-6]. The pathogenesis of SS can be attributed to the enlargement of pituitary gland, small sellar size, disseminated intravascular coagulation and autoimmunity [1,2]. Hypothalamic cell Anti-Hypothalamus Antibodies (AHAb), but not against Arginine Vasopressin (AVP) secreting cells, have also been proposed. However, the significance of these antibodies remain unclear. It has been hypothesized that the sequestered antigens due to tissue necrosis could trigger autoimmunity and may cause delayed hypopituitarism in these patients [3]. Classical clinical features include failure to lactate or to resume menses, genital and axillary hair loss, asthenia and weakness, fine wrinkles around the eyes and lips, signs of premature aging, dry skin, hypopigmentation and other features of hypothyroidism [2]. Uncommonly, hyponatremia, hypoglycemia, congestive cardiac failure or psychosis may also occur [1-6]. Hyponatremia, as an initial symptom of acute Sheehan's syndrome, is seen in approximately 60% of the reported cases. Acute hyponatremia can lead to life-threatening cerebral edema. It may arise from diminished free water clearance caused by hypothyroidism and adrenal insufficiency [5]. Interestingly, our patient also had recurrent episodes of hypoglycemia, in the setting of panhypopituitarism, manifesting as central hypothyroidism, lactation failure and anemia. The recurrent hypoglycemic episodes might have been attributed to secondary adrenal insufficiency with defective counter-regulatory hormone responses. Though isolated cortisol deficiency is rarely sufficient to produce severe hypoglycemia, concomitant deficiencies of cortisol and growth hormone secondary to hypopituitarism resulted in profound impairment of glucose counter-regulation. Cortisol deficiency leads to depletion of hepatic glycogen reserves, reduced hepatic gluconeogenesis and blunting of sympathoadrenal activation during hypoglycemia. The markedly suppressed serum insulin and c-peptide concentrations, together with low anterior pituitary hormone levels, are consistent with appropriate suppression of pancreatic β-cell insulin secretion in response to hypoglycemia [6]. Sometimes, the diagnosis of SS may be delayed for several years until features of hypopituitarism (e.g., secondary hypothyroidism or secondary adrenal insufficiency) become more apparent [5]. The initial diagnosis depends on the detailed clinical history and physical examination, complemented by relevant laboratory tests and imaging studies. These include the assessment of anterior pituitary hormone levels and MRI of the brain. Early MRI findings include a non-hemorrhagic enlargement of the pituitary gland with a thin rim of peripheral enhancement after gadolinium, without pituitary gland enhancement. Subsequent pituitary shrinkage and an empty sella have been reported in late scans, as in our case [1-6]. The mainstay of treatment include hormonal replacement therapy. In patients who have both secondary hypothyroidism and hypocortisolism, glucocorticoids should be replaced before the replacement of thyroid hormone. If not diagnosed in time, the prognosis can be grave, resulting in death. Patients who wish to become pregnant may be directed to the service of fertility for ovulation induction followed by successful pregnancy [1-7].

Conclusion

Sheehan's Syndrome (SS) is a frequent cause of hypopituitarism in underdeveloped countries. The myriad clinical features are often non-specific but history of postpartum hemorrhage, failure to lactate and cessation of menses are important clues to the diagnosis. Both hyponatremia and recurrent hypoglycemia could also be an initial manifestation of SS. Early diagnosis and appropriate treatment are of utmost importance, in order to reduce the morbidity and mortality, besides, improving the women healthcare.

Conflict of Interest
The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement
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Ethical Statement
The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore, was exempt.

Informed Consent Statement
Informed consent was taken for this study.

Authors' Contributions
All authors contributed equally to this paper.

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