Case Report

With a pinch of salt: The importance of history in evaluation of hyponatremia

Rashmi Prakash[1], Smitha Bhat[2], Tushar Gopalakrishna[3], K Sudeep[4]

From [1]PG Resident, [2]Professor, [3]Senior Resident, Department of Medicine, [4]Associate Professor, Department of Endocrinology, Father Muller Medical College Hospital, Kankanady, Mangalore, Karnataka, India

ABSTRACT

Fatigue, a non-specific symptom, is often dismissed as trivial or functional, however, an underlying malignant, endocrine, or metabolic disorder may be the cause of fatigue. We present the case of an elderly lady who presented with tiredness, anorexia, and vomiting for a month. Physical examination revealed pallor, facial puffiness, and coarse crackles in the left mammary area. Investigations revealed hyponatremia and hypokalemia. The patient denied a history of amenorrhea and lactation failure, but further questioning revealed that she had severe post-partum hemorrhage necessitating a hysterectomy. Investigations revealed normal TSH, low normal fT4, inappropriately low FSH, and LH. The patient was treated with thyroid and adrenal hormone replacement. We present this case to emphasize the importance of detailed history taking for a medical diagnosis, especially in conditions like Sheehan's syndrome with relatively non-specific presenting complaints.

Key words: Fatigue, Females, Sheehan syndrome, PPH

Fatigue is a non-specific symptom often neglected by health-care professionals especially when the patient is a female. However, certain features in the medical history of the patient such as bony pain, altered bowel habits, menstrual disturbances, weight gain, and history of blood loss would point to a malignant or endocrine cause of the fatigue.

This case history describes an elderly female patient who presented with chronic fatigue and was found to have hyponatremia. We present this case to emphasize that even when the presenting complaint is non-specific or appears trivial, physical, and laboratory evaluation yields diagnoses that are amenable to treatment.

CASE REPORT

A female hypertensive patient, aged 68 years, presented to the medicine OPD with complaints of fatigue, reduced appetite, and vomiting for the past year. However, these symptoms have increased over the preceding three weeks. The medical history of the patient revealed that she was on amlodipine for hypertension as well as 2.5 mg per day of prednisolone, which had been prescribed 21 years ago following an episode of fever, weakness, and unresponsiveness. There was a history of contact with a tuberculosis patient. The family history was significant for postpartum hemorrhage (PPH) in her mother, following which she expired a year later.

Physical examination revealed pallor and facial puffiness. Blood pressure was 150/90 mm Hg and heart rate was 82/min. Systemic examination revealed coarse crackles in the left mammary area. There was no skin or mucosal hyperpigmentation, lymph node enlargement, or papilloedema.

Laboratory evaluation revealed hyponatremia (Na^+ 118 mEq/L) and hypokalemia (K^+ 3.0 mEq/L). In view of the contact history, respiratory findings, and hyponatremia, tuberculosis with syndrome of inappropriate antidiuretic hormone secretion (SIADH) was considered. Sub-acute Addison's disease was also considered, however, the dose of steroid which she was receiving was unlikely to cause hypothalamic-pituitary-adrenal axis (HPA) suppression.

Further investigations revealed random serum cortisol of 200 nmol/l, thyroid-stimulating hormone (TSH) of 0.7 mIU/ mL (0.5–4.5 mIU/ mL), free thyroxin (fT4) of 0.8 (0.7–1.8 ng/dL), inappropriately low follicle-stimulating hormone (FSH) of 4 IU/L, and luteinizing hormone (LH) of 3 IU/L (as the patient was post-menopausal).

With the laboratory findings of low serum cortisol, inappropriately low TSH, FSH, and LH, as well as the lack of hyperpigmentation, a central cause of hypocortisolism was considered. When questioned directly, the patient denied a history of amenorrhea and lactation failure. However, on further

Access this article online	
Received - 07 April 2021 Initial Review - 23 April 2021 Accepted - 30 April 2021	Quick Response code
DOI: 10.32677/IJCR.2021.v07.i05.004	

Correspondence to: Smitha Bhat, Department of Medicine, Father Muller Medical College Hospital, Kankanady, Mangalore, Karnataka, India. E-mail: doctorsmitha@yahoo.co.in

questioning, she revealed that she had severe PPH following her last child's birth, necessitating a hysterectomy; the question of lactation failure was irrelevant in view of the unfortunate stillbirth of the child.

Magnetic resonance imaging (MRI) brain was ordered and it showed a partially empty sella (Fig. 1). The patient was diagnosed to have Sheehan's syndrome based on hyponatremia, low TSH, FSH, LH as well as neuroimaging revealing partial empty sella. She was treated with thyroid, adrenal hormone replacement, and correction of electrolytes.

DISCUSSION

Sheehan's syndrome is hypopituitarism due to pituitary infarction following severe PPH. Clinical features range from a mild presentation with insidious and, therefore undiagnosed fatigue and anorexia to a catastrophic acute adrenal insufficiency. Lactation failure and amenorrhea following childbirth associated with severe blood loss are important historical clues for diagnosis. On neuroimaging, the sella turcica appears larger due to pituitary necrosis- a sign called the empty sella sign, which has various causes [1].

Sheehan's syndrome constitutes 0.5% of all known cases of hypopituitarism in women [2]. It occurs in 1 per 100,000 births worldwide, and in 5 per 100,000 births in developing countries. It is the most common cause of hypopituitarism in developing countries. Up to 30% of cases follow PPH [3-6]. A study conducted in Iceland showed a prevalence of 5.1 per 100,000 women [7]. A prevalence of 3.1% has been reported in Kashmir valley in women aged 20 years and above [8]. Almost two-thirds of these women had delivered their babies at home. Famuyiwa *et al*. reported two new patients per year with Sheehan's syndrome in a teaching hospital in Nigeria [9]. The higher prevalence of Sheehan's in developing countries could be due to the dearth of accessible effective obstetric care [10].

Hyperplasia of the pituitary gland during pregnancy is not accompanied by an increase in the arterial supply, thus rendering the pituitary vulnerable to ischemia. Common symptoms of Sheehan's syndrome that do not immediately present in the postpartum period include fatigue, the decline in secondary sexual characteristics, anemia, hypoglycemia, and symptoms of hypothyroidism. Of these, our patient had fatigue, nausea, and vomiting. It must be emphasized that it is the non-specific nature of these symptoms that makes the diagnosis of Sheehan's syndrome so hard to pin down. In addition, symptoms such as lowness of mood and fatigue in a female patient are often dismissed as functional.

Hence, a diagnostic delay of up to 20 years often results. Sheehan's syndrome should always be ruled out in women with symptoms of adrenal insufficiency and a characteristic obstetric history. In a study published by Dr. Sheehan in 1965, among the 12 cases diagnosed before death, the mean duration of disease was 18.5 years, and the mean survival after diagnosis was 7.1 years [11].

Multiparity was a possible risk factor for PPH in our patient. Other reported risk factors include previous PPH. However, the familial association of PPH that we saw in our patient has not been previously reported. Other causes of hypopituitarism that we considered include lymphocytic hypophysitis (which is associated with pregnancy, though not PPH) [12], and granulomatous hypophysitis [13]. These two conditions, however, more commonly present with visual symptoms and not the fatigue and anorexia which were seen in our patient.

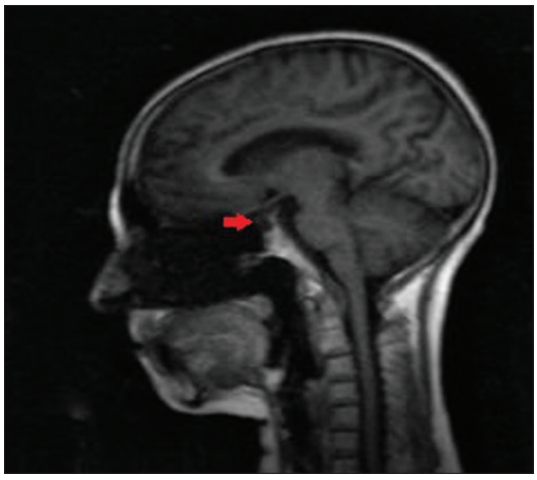

Figure 1: T1-weighted sagittal section showing partial empty sella

CONCLUSION

Sheehan's syndrome sometimes presents as acute adrenal insufficiency when unmasked by acute stressors. Hypertension, hyperglycemia, and hypokalemia, which our patient had, are not typical features of hypoadrenalism. These features may have been due to long-term steroid use. We surmised that the prior episode of unresponsiveness following fever might have been due to latent hypopituitarism made overt by infection. In view of the mother's PPH and subsequent sudden demise a year later, it is not unreasonable to wonder whether she was a case of Sheehan's syndrome as well. We present this case to emphasize the importance of patient narrative in medical diagnosis. Had we taken the negative history for lactation failure and amenorrhea at face value, this diagnosis would have remained elusive.

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Funding: None; Conflicts of Interest: None Stated.

How to cite this article: Prakash R, Bhat S, Gopalakrishna T, Sudeep K. With a pinch of salt: The importance of history in evaluation of hyponatremia. Indian J Case Reports. 2021;7(5):185-187.