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Case Report

Myxoedemic coma: an uncommon presentation of Sheehan syndrome

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ABSTRACT

This is a rare case of a 35 year old multiparous female who presented with electrolyte abnormality, hyponatremia in a setting of seizure and moderate pallor. She had a significant past history of childbirth complicated with post-partum haemorrhage after which she developed secondary amenorrhoea and lactation failure. Workup showed suppressed levels of all pituitary hormones and was treated as myxoedemic coma. A diagnosis of Sheehan's syndrome presenting as myxoedemic coma - a rare but emergency presentation was made.

Keywords: Sheehan's syndrome, Hyponatremia, Myxoedemic coma

INTRODUCTION

Sheehan syndrome is characterized by postpartum pituitary necrosis following post-partum hemorrhage. It has varied presentations which have been described in literature. In our case report, patient presented with myxoedemic coma. Myxoedemic coma of central origin (Sheehan syndrome) is exceedingly rare, but should be kept in mind if any female patient presents with altered sensorium along with features of post-partum pituitary dysfunction after excluding other metabolic, infective and focal causes.

CASE REPORT

A 35 year old multiparous female from Firozabad district of Utter Pradesh presented with generalized swelling all over the body for last six months and had multiple episodes of generalized tonic-clonic seizures for 1 month. There was loss of consciousness for last 4 days. She also had history of amenorrhoea and failure of lactation after delivery of last child. She took treatment in the form of tablets on and off. She had no history of fever, trauma, prior headache, visual disturbance and any loss of power of limbs. She had no prior history of thyroid disorder & migraine. There was no similar illness in the other family members. At the time of admission patient was comatosed. Her Glasgow coma scale was 3/15. Her pulse was 90/min. regular & blood pressure was 80 mm of Hg of right arm supine position & respiratory rate was 12/min & regular. Pallor was present. Clubbing, cyanosis & icterus were absent. Facial puffiness & non-pitting edema was present. Pubic and axillary hairs were absent. On neurological examination pupil was bilaterally normal & reacting to light. There was no facial asymmetry. Light &gag reflex were normal. Neck rigidity& Kernig's sign were absent. Planter reflex was bilateral nonellicitable. Deep tendon reflex were normal & other systemic examinations were within normal limits. On investigation blood picture revealed haemoglobin 8 gm%, total leucocyte counts 4500/mm^3, platelet count 1.4 lakhs/mm^3, differential counts (polymorphs 71%, lymphocytes 21%, eosinophils 2%, monocytes 2%) , ESR 24 mm/Hr, RBS 61 mg%, urea/cr. 23.9/0.78 mg%, serum sodium 118 mg%, serum potassium 4.86 mg%. Liver profile was normal. Peripheral smear & malarial serology were

negative. Widal test, HBsAg & HCV serology were non-reactive. Urine exam, CXR PA view, USG abdomen were within normal limits. On arterial blood gas analysis no hypoxia & hypercapnoea was present. CT Brain revealed normal study, CSF examination were within normal limit.

During this period electrolyte abnormality (hyponatremia) was corrected with hypertonic saline & antibiotic ceftriaxone was given. On the sixth day of admission corrected electrolyte profile was serum sodium - 138 meq/L but the patient Glasgow (GCS) coma scale did not improve up to the mark & her GCS was 6/15. This time planter reflex was bilateral withdrawal. Other examination findings were similar to previous findings. This case was further reviewed to find out the cause of encephalopathy, and other investigations with hormonal evaluation were planned. Her blood ammonia was 33µg/dl (N - 30.86). USG finding was suggestive of atrophic uterus. Her hormonal profile was done which were as follows - ACTH - <5 pg/ml (N - 7.2 to 63), S. cortisol 18.50 (N - 3.09 to 16.6) after steroid therapy. Serum prolactin - 1.01 ng/ml (non-pregnant - 2.8 to 29.2), S. FSH - 5.16 mIu/ml (N 3.08 to 8.08) S. LH 2.46 IU/ml (N 1.8 to 11.78) FT_4/TSH - 0.4/1.67. Serum calcium was 8.9 mg% (N 9- 11), HIV serology was non-reactive. ANA was negative, anti TPO antibody were within normal limit (30 IU/ml). EEG characteristically revealed diffuse slow wave pattern. MRI of brain was not done. Her hormonal profile & characteristic clinical presentation was consistent with the diagnosis of myxoedemic coma due to postpartum pituitary dysfunction.

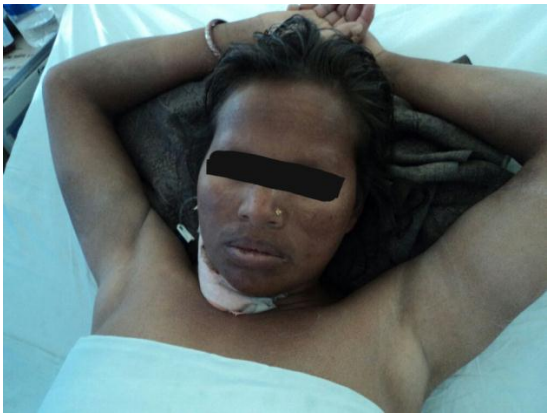

Figure 1: Patient with myxoedemic coma.

Patient was started on IV dexamethasone (8 mg TDS). Few hours after first dose of dexamethasone, 500 µg levothyroxine was given through naso-gastric tube & she responded dramatically. Initially eye opening returned and gradually motor response improved. On 7th day of steroid and thyroxin therapy, eye opening & motor function returned to withdrawal, but the patient was still more talkative, verbal response improved after 12th day of steroid & thyroxin therapy. Simultaneously antiepileptic was also added. Patient was discharged with oral prednisolone 5 mg OD, Tab eltroxine 100 µg 1 OD, tab. Cal. 500 mg, tab. carbamazepine 100 mg BD. Antiepileptic was gradually withdrawn and the patient was seizure free for last 8 months during follow up.

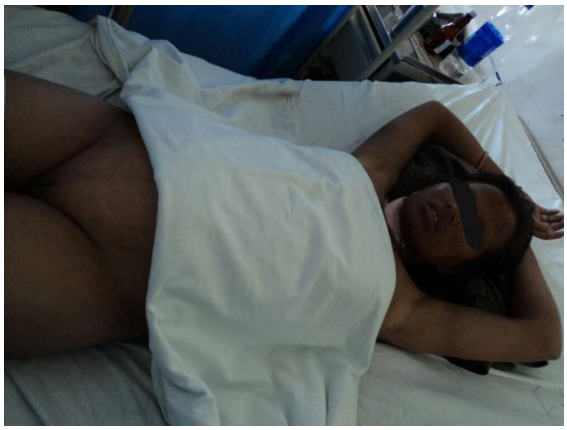

Figure 2: Gradually improvement of motor response after dexamethasone treatment.

DISCUSSION

Sheehan syndrome is postpartum pituitary necrosis following postpartum hemorrhage. Enlargement of pituitary gland, small sellar size, DIC, autoimmunity has been suggested to play a role in pathogenesis of Sheehan syndrome. Sheehan syndrome is characterized by varying degrees of anterior pituitary dysfunction[1]. Sheehan syndrome can present in the postpartum period with lactation failure or after many months to year following the events. In many affected woman anterior pituitary dysfunction is not diagnosed for many years. A study of 60 patients shows the average time between the previous obstetrical event & diagnosis of Sheehan syndrome was 13 years[2]. In our case it was approximately 11 years. Characteristic manifestation of Sheehan syndrome include failure to lactate, amenorrhea, genital & axillary hair loss, asthenia, weakness, sign of premature ageing , dry skin , hypo pigmentation & other evidence of hypopituitarism. The absence of amenorrhea or the presence of postpartum lactation, however doesn't rule out the diagnosis. Uncommonly it can present actually with circulatory collapse, hyponatremia, hypoglycemia, diabetes insipidus, CCF, psychosis. In our case patient had presented with hypotension, hyponatremia, & hypoglycemia as well.[3] Hypothyroidism & glucocorticoid deficiency by decreasing free water clearance independent of vasopressin causes hyponatremia. SIADH & volume depletion are the other factors leading to hyponatremia (A rare emergency presentation of Sheehan syndrome). Myxoedemic coma is a rare, often fatal endocrine emergency that concerns elderly patient with long standing primary hypothyroidism. Myxoedemic coma of central origin is exceedingly rare.[4]

Our case had progressive features, initially it was suspected to be seizure disorder secondarily to electrolyte

disturbance due to suspected Sheehan syndrome but later on diagnose to be a case of myxoedemic coma of central origin. This case highlights that any female patient who presented with feature of post-partum pituitary dysfunction with unconscious state, diagnosis of myxoedemic coma should be consider after excluding infective, metabolic & focal cause of unconsciousness.

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