Myxedema Coma, Pancytopenia, and Hypocoagulopathy: A Rare Presentation of Sheehan's Syndrome

Sir,

Sheehan's syndrome is postpartum hypopituitarism caused by necrosis of the pituitary gland, as a result of massive hemorrhage or severe hypotension during delivery. It is still a frequent cause of hypopituitarism in developing countries like India.[1] Clinical manifestations are often subtle, leading to a delay in diagnosis.[2] Herein, we report a case of Sheehan's syndrome presenting as myxedema coma with hematological abnormalities.

A 36-year-old lady presented in the month of January with complaints of vomiting and drowsiness for 1 day. Her relatives also told that she had symptoms of cold intolerance and asthenia for the last 1 year. On further probing, it was found that she had postpartum hemorrhage seven years back requiring multiple blood transfusions during her last child birth. This was followed by lactation failure and amenorrhea. On examination, she had pallor, facial puffiness, coarse dry skin, and nonpitting bilateral pedal edema. She was altered with a Glasgow coma scale (GCS) score of 8, her systolic blood pressure was 80 mm Hg, pulse rate was 44/min, respiratory rate was 10/min, and body temperature was 35°C.

Blood investigations revealed pancytopenia with Hb of 8 g/dL, total leucocyte count of 1,600/μL, platelet count of 29,000/μL, and MCV of 93 fL. Peripheral smear showed normocytic-normochromic anemia, leucopenia, and thrombocytopenia. Serum iron profile, vitamin B12, and folate levels were normal. Blood coagulation profile showed prolonged prothrombin time of 15.1 s (normal: 11–13.5 s) and prolonged aPTT of 55.9 s (normal: 25–35 s). She had hyponatremia (serum sodium level was 121 mg/dL) and hypoglycemia (random blood sugar level was 54 mg/dL). Hormonal profile revealed the following: TSH - 0.19 (normal: 0.4–6) μIU/mL, Free T4 - 1.89 (normal: 4.8–11.6) μg/dL, Free T3 - 0.2 (normal: 0.5–1.85) ng/dL, and low serum cortisol level - 3.1 μg/dL. Serum luteinizing hormone was low at 3.1 mIU/mL and serum follicle-stimulating hormone was also low at 9.04 mIU/mL. ECG was suggestive of sinus bradycardia with low-voltage complexes. Echocardiography revealed moderate pericardial effusion with normal left ventricular function.

A clinical diagnosis of myxedema coma with adrenal crisis of central origin (Sheehan's syndrome) was made. She was immediately shifted to ICU, intubated in view of low GCS and started on inotropes. She was started on 200 mg of intravenous hydrocortisone in divided doses; loading dose of levothyroxine 300 μg via Ryle's tube was given followed by daily 100 μg of supplementation along with other supportive measures. Subsequently contrast enhanced magnetic resonance imaging of brain showed partial empty sella, thus confirming our diagnosis of Sheehan's syndrome [Figure 1]. The patient exhibited improvement with reversal of shock, resolution of bradycardia, bradypnea, pericardial effusion, correction of metabolic parameters, improvement in sensorium, and was extubated. Coagulation profile normalized in a week and pancytopenia resolved completely in 4 weeks. She was henceforth managed with incremental dose of levothyroxine (titrated according to FT4) and maintenance dose of oral hydrocortisone.

Myxedema coma is a rare life-threatening endocrine emergency with a high mortality rate. Sheehan's syndrome presenting as myxedema coma is extremely rare. Dutta *et al*. studied 23 patients of myxedema coma and only 3 patients had secondary hypothyroidism due to Sheehan's syndrome. The most common precipitating event for myxedema crisis was cold exposure in their study.[3] Pancytopenia is rare and is associated with hypocellular marrow. The cytopenias improve after steroid and thyroxine supplementation.[4] A hypocoagulable state, as evident in our case, with increased tendency for bleeding is attributed to acquired factor VIII and vWF deficiency in severe hypothyroidism.[5]

To conclude, myxedema coma can be rarely secondary to Sheehan's syndrome and is reversible cause of pancytopenia and hypocoagulable state. Early diagnosis and appropriate treatment can result in favorable outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other

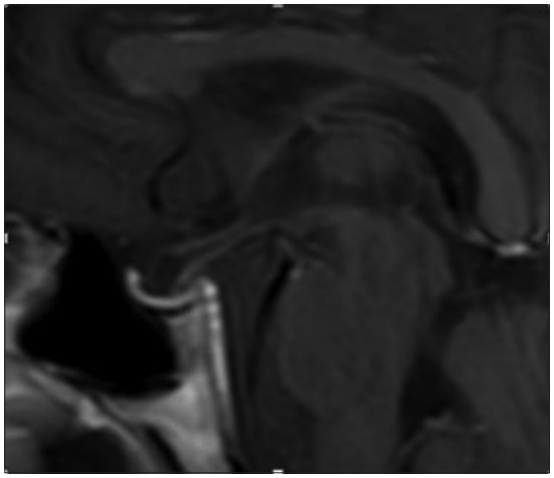

Figure 1: Sagittal postcontrast T1 magnetic resonance imaging showing thin enhancing tissue along the floor of the pituitary fossa with cerebrospinal fluid (CSF) occupying the rest of the fossa suggesting a partially empty sella

clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

Badari Mylliemngap, Satish Swain, Surabhi Vyas[1], Prabhat Kumar

Departments of Medicine and [1]Radio-diagnosis, All India Institute of Medical Sciences, New Delhi, India

Address for correspondence:
Dr. Prabhat Kumar,
Department of Medicine, Third floor, Teaching Block,
AIIMS, New Delhi - 110 029, India.
E-mail: drkumar.prabhat@gmail.com

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