

Preservation of Thyrotroph Function in Sheehan's Syndrome

Sir,

Although Sheehan's syndrome (SS) is now rarely described from developed nations, physicians from developing nations need to keep the entity in mind wherein with modern hormone assays, and imaging diagnosis is not difficult. Atypical or partial presentation of SS is described from India; with the preservation of gonadotroph and corticotroph function,^[1,2] preservation of thyrotroph has not been described.

A 38-year-old female was suspected to have primary hypothyroidism a year back on the basis of clinical features, and thyroid function tests revealed total T3 of 0.51 ng/ml (range: 0.8–1.8), T4 of 1.2 µg/dl (range: 4–13), and thyroid-stimulating hormone (TSH) of 100 mIU/L (range: 0.5–6.5). She was put on thyroxine 50 µg/day which she was taking intermittently. Recently, she presented to our hospital and detailed history revealed that she was married at the age of 31 years. She conceived 5-year postmarriage and terminal event was intrauterine death followed by postpartum hemorrhage. She required blood transfusion during the postpartum period. She also had history of easy fatigability, regression in breast size, and loss of axillary and pubic hair following the delivery of the dead fetus. Examination revealed blood pressure of 90/60 mmHg with no postural drop, respiratory rate of 18/min, and oral temperature of 97.6°F (36.4°C). There was no goiter or lymphadenopathy. She had atrophy of breasts and sparse axillary and pubic hair. Chest, cardiovascular, and abdominal examinations were noncontributory. Central nervous system examination revealed slow mentation in the form of delayed response to verbal commands. Examination of motor system did not reveal any focal neurodeficit, but deep tendon jerks were markedly delayed. Evaluation revealed hypoglycemia (blood glucose of 38 mg/dl), normal hemogram, kidney, and liver function, with normal serum sodium and potassium; electrocardiogram revealed bradycardia and low-voltage complexes. Abdominal ultrasound was normal. Hormonal investigations revealed TSH of 23 mIU/L with T3 of 1.64 ng/mL, T4 of 8.5 µg/dl and anti-thyroid peroxidase (TPO) antibody titer of 46 IU/L (normal value <35). Basal (8 am) serum cortisol

0.07 mcg/dl, adrenocorticotrophic hormone <5 pg/ml (range: 9–52), prolactin 19.4 ng/ml (range: 4–23), follicle-stimulating hormone 16 IU/ml, luteinizing hormone 3.74 IU/ml, and serum insulin-like growth factor-1 was 39 ng/ml (range: 109–284). Magnetic resonance imaging of pituitary revealed empty sella. On the 2nd day of admission, the patient developed acute adrenal crisis and was managed with intravenous (IV) fluids and IV hydrocortisone. Levothyroxine was restarted. The patient improved clinically. Appetite improved and there was an improvement in general well-being. The patient was given oral prednisolone, and levothyroxine was continued at the same dose. She was discharged on the 5th day of admission.

Preservation of various anterior pituitary functions in SS has been reported previously. Of all the cell lines in the anterior pituitary, gonadotroph and corticotroph cell lines are the most often preserved. Because of the location of somatotroph and lactotrophs in the lateral and posterior portion of the pituitary, the location is supposed to be the most susceptible to ischemic necrosis.^[3,4] However, some previous studies have reported thyrotroph involvement in 90%–100% of women with SS;^[1] at the same time, preservation of thyrotroph has not been described. Some elevation in TSH has been reported in these women; it usually does not cross >20 mIU/L.^[5] In the present, patient's initial TSH was more than 100 mIU/L suggesting preservation of thyrotroph function which responds to failing thyroid by marked elevation of TSH as is seen in primary hypothyroidism. The possibility of autoimmune polyglandular disease is less likely in view of the absence of the elevation of TPO antibodies.

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Conflicts of interest

There are no conflicts of interest.

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