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Case Report

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AN ELDERLY PATIENT DIAGNOSED QUITE LATE AS SHEEHAN'S SYNDROME: A CASE REPORT

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ABSTRACT

Purpose: Sheehan's syndrome (SS) is a disorder resulting secondary to ischemic pituitary insufficiency due to excessive blood loss during and after delivery. Patients with SS may be presented with various symptoms ranging from nonspesific symptoms (fatigue and weakness) to severe pituitary insufficiency that can lead to coma and even death. We presented in this report an elderly patient who admitted to our clinic with panhypopituitarism diagnosed as SS nearly 40 years after delivery.

KEYWORDS: Sheehan's syndrome, fatigue and weakness.

INTRODUCTİON

Sheehan's syndrome (SS) is a disorder resulting secondary to ischemic pituitary insufficiency due to excessive blood loss during and after delivery. Although SS is a rare cause of hypopituitarism in developed countries due to improved obstetrical techniques; it is still a common cause in developing and under developed countries. Although the pathogenesis and the natural history of SS remain unclear; some factors which have been considered in pathogenesis of SS are enlargement of pituitary gland, small sella size, disseminated intravascular coagulation and autoimmunity. SS diagnosis is often delayed due to slow progression.[1-5] During pregnancy because of lactotrop hyperplasia pituitary volume rises up to 36% of average normal size.[6] Because prolactin (PRL) and growth hormone (GH) are the first two hormones to be lost in patients with SS the most presenting symtoms are failure of lactation and amenorrhea. But the patients who suffer from SS may be presented with various symptoms ranging from nonspesific symptoms (fatigue and weakness) to severe pituitary insufficiency that can lead to coma and even death.[4,7] As a result of partial hypopituitarism, most patients have a mild disease and undiagnosed and untreated for a long time. Diagnosis of SS is based on medical history, physical findings and pituitary hormone levels and radiological findings. The last one vary according to the stage of the disease; an enlarged pituitary gland is evident in the early stages, while pituitary atrophy and an empty sella due to arterial necrosis can be seen in the later stages.[8] The treatment goal for SS is replacement of the absent hormones; glucocorticoid hormone replacement should be performed first, then thyroid hormone replacement, and finally sex steroid administration until the onset of menopause.[4] Diagnosis could be delayed due to the slow development of clinical disease. In the literature, the average year of diagnosis is 13 years. We presented in this report an elderly patient who admitted to our clinic with panhypopituitarism diagnosed as SS nearly 40 years after delivery.

CASE REPORT

A 63 year-old female patient suffered from weakness and fatigue for a long time was referred to our out patient clinic. She had excessive bleeding during child birth when she was 24 year-old and she had gone to bilateral oophorectomy and hysterectomy, so that she did not breast feed her child. However she has not applied any health care units. On physical examination, blood pressure was 100/65 mmHg and heart rate was 55 / min and she had pronounced pallor and fine wrinkles around the mouth and eyes, other physical examination findings were normal except for lost of pubic and axillary hairing. However, she spoke and moved slowly. We suspected from secondary hypothyroidism due to low FT4 and FT3 levels with insufficiently high levels of TSH. Other anterior pituitary hormones were also measured. Serum cortisol was 2.3 (normal range 3-19) µg/dL and ACTH was<5 (normal range 15-50) pg / ml; FT3 was 2.51 pmol/L (normal range 2.63-5.7 pmol/L), FT4 was 8.0 pmol / L(normal range 9-19pmol/L), TSH was 2.78 µIU / ml(normal range 0.35 to 4.4µIU/ml); E2 (estradiol) was <10 pg/ml (normal range10-28pg / ml), antiTPO (antithyroid peroxidase) was 0.34 (normal range 0-5.6) IU / ml; antiTg (antithyroglobuline) was 4.31 (normal

range 0-4.11) IU / ml; PTH (parathormone) was 139 pg/ml (normal range 10-72 pg / ml); LH (luteinizing hormone) was 0.64 mIU / ml (normal range 5.1-61.9 mIU / mL), FSH (follicle stimulating hormone) was 2.63 mIU / mL (normal range 26.7-133.4 mIU / ml) Prolactin was 9.78 ng / mL (normal range 5.1-26.5) and IGF1 (somatomedin-C) was 51µg / L (normal range 70-210 mg / L) were detected. Serum Na(sodium) was 138 (normal range 136-145) mmol / L and potassium was 4.1 (normal range 3.5 to 5.1) mmol / L, glucose was 110 (normal range 80-100) mg / dL; serum creatinine level was 1.2 (normal range 0.5-0.9) mg / dl; calcium was 9.9 (normal range 8.8-10.2) mg / dl; 25-OH vitamin D was <7.0 ng / mL (normal range 30-100); evaluation of urinary density was 1025; respectively. In thyroid ultrasonography thyroid parenchyma was homogeneous; in right lobe 7x4 mm size nodule and in left lobe 5mm and 2 mm diameter hypoechoic nodules were present. Electrocardiography was present in the sinus bradycardia. Bone mineral density was consistent with osteoporosis; lumbar totally t-score: -3.4 femoral totally t-score: -2.3 was. Short ACTH stimulation test, 250 µg by intravenous was administrated; basal cortisol level was 3.9 µg/dl; 30. min cortisol was 7.7 µg/dl and 60 min cortisole was 10.2 µg/dl; there was not any response to the ACTH stimulating test. Ovaries and uterus were not showed in the pelvic ultrasonography. Adeno hypophysal pituitary MRI imaging was taken; adenohypophysis was not shown; it was consistent with empty sella but neurohypophysis was normal and bright spot was available. (Figure1). Replacement for the treatment ofpatient's was primarily started with hydrocortisone, then thyroid replacement was given and her symptoms resolved. Risedronate, calcium and D- vitamine were prescribed for osteoporosis. The amount of fluid taken and urinary output in the interests of patient follow-up were normal. Blood pressure, serum sodium level and urine density were normal. She was informed about steroid replacement medication without interrupting during lifetime.

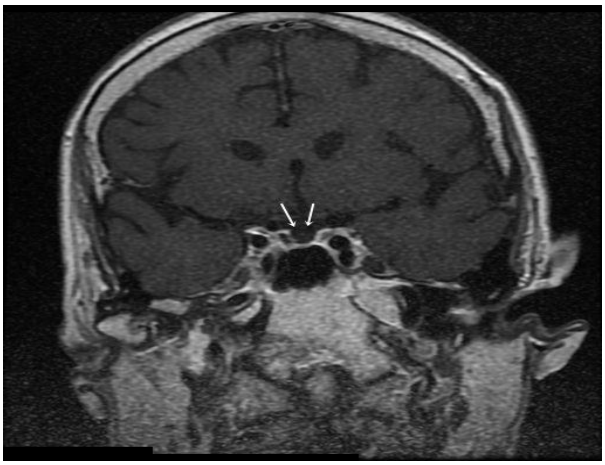

Figure 1 Pituitary magnetic resonance imaging shows empty sella, normal neurohypophisis and bright spot

DİSCUSSİON

Atrophy of the pituitary gland was interpreted in our patient's case as a likely outcome of late-onset SS, a consequence of a post-partum hemorrhage that our patient was able to recall. Sheehan first described pituitary infarction and panhypopituitarism after a post-partum hemorrhage in 1938, but the mechanism of ischemia is still not entirely clear. Hypotension and subsequent pituitary arterial vasospasm seem to compromise blood perfusion in the pituitary gland, causing its necrosis. Signs of hypopituitarism are reported in 32% of women with severe post-partum hemorrhage. The rapidity of onset and degree of pituitary insufficiency depend on the extent of the damage. The gland, however has a high reserve and more than 75% of the pituitary needs to be damaged before clinical manifestations are evident. The first of these appears to be the absence of lactation after delivery. Although a small percentage of patients with Sheehan's syndrome present with severe hypopituitarism immediately after labor, in many patients it remains undiagnosed and the pituitary failure is only recognized and treated after many years[5,9], as in our patient's case. CT and MRI in most cases indicate an etiologic diagnosis. Imaging results show an empty sella and an atrophic gland, as in our patient. The MRI results also showed signs of chronic flow deficits, which could to some extent justify the syndrome manifestation as a result of the progressive loss of residual functional reserve. Another condition considered was lymphocytic hypophysitis, related to pregnancy in most of the women studied. There are in fact many similarities between Sheehan's syndrome and lymphocytic hypophysitis, which is an autoimmune disease seen in both sexes. However, in our patient's case the absence of clinical signs and serological markers of autoimmunity, together with the obstetric history and the imaging results, excluded this diagnosis.[3]

A diagnosis of secondary hypothyroidism was suggested by the low FT3 and FT4 levels, with values inappropriately within the normal range or below the standard in the case of TSH.[10]

The hormonal evaluation suggested a solely pituitary genesis and the accuracy of MRI indicated the organic etiology of the syndrome event. Regarding the therapeutic approach, replacement therapy re-established a satisfactory physical and mental well-being.

The present report shows that Sheehan's syndrome might have to be late diagnosed as a consequence of slow progress years after excessive bleeding during childbirth. And also the present work suggests the need for an adequate diagnostic approach based on simple and inexpensive tests (including serum and urine laboratory tests along with imaging procedures) that focus on the early identification of the cause and its correct treatment.

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All authors declare that there is no conflict of interest.

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