



Sheehan's Syndrome with Reversible Cardiomyopathy: A Case Report

KEYWORDS

Sheehan's syndrome, Hypopituitarism, secondary amenorrhea

Dr. K.B.R. SASTRY

PROFESSOR, DEPARTMENT OF MEDICINE
GANDHI MEDICAL COLLEGE, SECUNDERABAD,
TELANGANA, INDIA

Dr. SUNIL KUMAR

ASSISTANT PROFESSOR,
DEPARTMENT OF MEDICINE,
GANDHI MEDICAL COLLEGE
SECUNDERABAD, TELANGANA,
INDIA

Dr. P. ANURADHA

ASSISTANT PROFESSOR,
DEPARTMENT OF MEDICINE,
GANDHI MEDICAL COLLEGE
SECUNDERABAD, TELANGANA,
INDIA

Dr. M.A. MUJEEB AFZAL

JUNIOR RESIDENT, DEPARTMENT OF MEDICINE
GANDHI MEDICAL COLLEGE, SECUNDERABAD,
TELANGANA, INDIA

Dr. P. PRIYADARSHINI

JUNIOR RESIDENT, DEPARTMENT OF MEDICINE
GANDHI MEDICAL COLLEGE, SECUNDERABAD,
TELANGANA, INDIA

ABSTRACT *Sheehan's syndrome is postpartum pituitary apoplexy leading to panhypopituitarism which can occur as acute syndrome in the immediate postpartum period or may take some time to manifest as a chronic syndrome. A 34-year-old woman presented with breathlessness, generalized anasarca, features of hypothyroidism with past history of lactation failure, secondary amenorrhea, and a hypocortisol state following severe postpartum hemorrhage after her last twin child birth with obstructed labour of the second twin delivery at home. Hormonal investigations showed evidence of panhypopituitarism and magnetic resonance imaging revealed empty sella. Meanwhile echocardiography revealed evidence of dilated cardiomyopathy (DCM). The patient was given replacement therapy in the form of glucocorticoids and levothyroxine. She improved and on follow-up over a period of 4 months, the DCM completely reversed. There are very few other such reports of reversible DCM in a patient with Sheehan syndrome.*

Introduction

Sheehan's syndrome is postpartum pituitary apoplexy leading to panhypopituitarism which can occur as acute syndrome in the immediate postpartum period or may take some time to manifest as a chronic syndrome. This is a case report of a Sheehan's syndrome presenting with congestive cardiac failure.

A 34 year old female came to our outpatient department with chief complaints of Breathlessness since 15 days of insidious onset and progressive, associated with orthopnea and Paroxysmal nocturnal attacks, and pedal edema of 15 days duration which progressed to anasarca over the last 3 days. She had a history of lethargy and amenorrhea since 4 years, on further probing she gave the history of amenorrhea ever since her last delivery which was complicated by the obstructed labor of second twin of her twin pregnancy. The delivery of the first twin was uncomplicated and spontaneous at home but the second twin had hand prolapse and was in obstructed labour for hours. While she was being moved to hospital 12 hours later to the delivery of first twin, the second twin got delivered spontaneously as still birth on the way. She had lots of bleeding in the whole process and had to be bedridden for a month. She could not feed her first and sole live twin baby as there was lactation failure. The infant succumbed 2 ½ month later to pneumonia. On examination she had apathetic dull look with generalized anasarca. There was no goiter. Her skin was dry, pale and thin. Her breasts were atrophied with depigmented areolae. Axillary and pubic hair were absent. The pulse was 84/min and blood pressure was 90/70mmHg. On cardiovascular examination proper, the apical impulse was displaced ½ inch lateral to midclavicular line and on auscultation there was S₃ in mitral area with grade 3 pansystolic murmur. Examination of the lungs

revealed bilateral basal crepitations and decreased breath sounds in both the infraaxillary areas. There was some free fluid in abdomen with shifting dullness but no fluid thrill. Examination of nervous system revealed delayed relaxation of the ankle reflexes. Fundoscopy examination was normal.

On evaluation, Investigations showed hemoglobin level of 11 gm% with WBC counts of 6100/μL with normal differential counts and normocytic normochromic picture on smear. RBS was 78 mg%, Blood urea 19 mg % and serum creatinine 1 mg%. The serum electrolytes were Na⁺ 136 mEq/dL, K⁺ 4.2 mEq/dL, and Cl⁻ 103 mEq/dL. Her ECG had normal sinus rhythm with low voltage complexes. Chest x-ray was suggestive of Cardiomegaly and bilateral minimal pleural effusion. Her LFT was in normal range.

Hormonal investigation revealed TSH of 1.09 μIU/ml with free T4 of 0.29 ng/dL. The 8 AM cortisol was 224 μg/dL and the other hormones were as FSH 6.99 μIU/ml, LH 3.31 μIU/ml, prolactin 1.01 ng/ml, and growth hormone 0.05 ng/ml.

Ultrasound abdomen and pelvis revealed mild ascites, mild bilateral pleural effusion, and atrophic uterus and small ovaries. 2D-Echo scan was suggestive of Global hypokinesia of LV with Ejection fraction of 36% and moderate Mitral Regurgitation. MRI Brain showed normal sized empty sella with no abnormalities of hypothalamus, suprasellar and parasellar regions. (Figure1 MRI brain here)



Figure1: MRI Brain sagittal view showing empty sella.

She was started on Diuretics, ACEI and iv hydrocortisone 50 mg 8th hourly for 2 days before we started her on Oral Levothyroxine 75 mcg OD and prednisolone 5 mg OD. With dramatic improvement in general well-being, as breathlessness decreased, and edema subsided, she was discharged on Prednisolone 5 mg OD along with Levothyroxine 75 mcg OD. On follow up 4 months later her 2D-echo scan revealed good LV function with ejection fraction of 58% and the Mitral regurgitation disappeared.

Discussion:

Sheehan's syndrome occurs as a result of ischemic necrosis of pituitary gland from severe postpartum hemorrhage because of uninstitutionalized deliveries like this case is not uncommon in developing countries like ours¹. The pathogenesis of the syndrome may involve enlarged pituitary gland, small sella size, vascular compromise of the hypophyseal vessels, thrombosis, vasospasms, disseminated intravascular coagulation and autoimmunity. The posterior pituitary is usually not affected due to its direct arterial supply. Most common initial symptoms are lactation failure and amenorrhea after delivery². The presentations can range from non-specific symptoms to coma and the clinical manifestation may change from one patient to another. A few patients present with abrupt onset of hypopituitarism acutely after delivery with hyponatremia, hypoglycemia and adrenal crisis². But the majority of the patients present late with secondary hypothyroidism and features

of secondary adrenal insufficiency, which, in the rather chronic case is similar to Addison's disease with symptoms including fatigue, weight loss, hypoglycemia, anemia and hyponatremia. Growth Hormone is one of the first hormone to be affected.

Cardiac abnormalities including Dilated cardiomyopathy (DCM) have been described in hypopituitarism cases. Laway et al. and Monwarul Islam et al. have described DCM in Sheehan's syndrome which responded to Levothyroxine and glucocorticoid replacement^{3,4}. Dilated cardiomyopathy in hypopituitarism can be attributable to deficiency of Growth hormone, thyroid hormone and corticosteroids in varying degrees. Reversal of Dilated and hypertrophic cardiomyopathies on replacement of levothyroxine have been reported in hypothyroidism⁵. In our case DCM was diagnosed in a Sheehan's syndrome and the underlying etiology could not be established but may be due to the panhypopituitarism and hormonal deficiencies as there was reversal of the DCM on Hormonal replacement. Sheehan's syndrome was readily evident from the history and examination and was confirmed by the hormonal investigation and empty sella on MRI. As for the treatment of Sheehan's syndrome is concerned, first glucocorticoids should be supplemented first before starting levothyroxine in order to avoid precipitating adrenal crisis. Sex hormones can be supplemented in reproductive age group as in our patient which can also prevent osteoporosis in long term. Growth hormone replacement is controversial at present⁶.

Simple and inexpensive levothyroxine and corticosteroid hormonal replacement has led to complete reversal of dilated cardiomyopathy in this case of Sheehan's syndrome. Hypopituitarism should be kept in mind when confronted with cardiomyopathy in postpartum females especially when associated with amenorrhea and lactation failure.

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