Sheehan`s Syndrome: A Case Report

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Abstract
Hypopituitarism developing after severe post partum haemorrhage (PPH) is called Sheehan`s Syndrome. With good obstetric care Sheehan`s Syndrome became a rare entity in developed countries. In Indian context it is still an uncommon serious problem. Presentation depends on the extent of the pituitary necrosis; severe cases presenting immediately and less severe cases presenting years or decades after episode. An young woman had Pre eclampsia, ante partum haemorrhage, post partum haemorrhage at her premature twin pregnancy delivery. Three years later she presented with secondary amenorrhoea. Associated symptoms of weakness, hypotension, cold intolerance, loss of sexual hair, breast and vulval atrophy, loss of libido clinched the diagnosis. Low levels of ACTH, TSH, FSH, LH, Prolactin, and also of Estradiol, Cortisol, Sodium confirmed the diagnosis. Hormone replacement therapy with Cortisone, Thyroxine, Estrogen and Progesterone resulted in improvement of her general condition. Her menstrual cycles are established, genital organs rejuvenated and libido regained.

Key Words: Sheehans Syndrome; Post partum haemorrhage; Hypopituitarism.

Introduction
Sheehan in 1937 described hypopituitarism developing in a woman after severe PPH. Earlier acquired pan hypopituitarism developing after infections, tumours, PPH was named Simmond`s disease. With good obstetric care, effective management of PPH, Sheehan`s Syndrome became a rare entity in developed countries. But in developing countries, Sheehan`s Syndrome is a real clinical entity even now. In Kashmir valley in India, projected demographic data in 2005 showed a prevalence rate of 2.7% in parous women aged 20-39 years; 3.9% in parous women aged 40 or more.

Case Report
Mrs xxxxx, aged 28 years presented at Obstetrics & Gynaecology department of Vinayaka Mission`s Medical College & Hospital for non resumption of menses after her last delivery three years ago. She is from a low socio economic status, rural, agricultural background. Her first
pregnancy ended in a spontaneous first trimester abortion and was evacuated. Second pregnancy—severe PET, full term, normal delivery, alive baby, 7 yrs. Third pregnancy—mild PET, full term, normal delivery, alive baby, 5 yrs. In the fourth pregnancy, she was diagnosed as twins, mild PET, low lying placenta. At 32 weeks had ante partum bleeding, premature delivery, retained placenta; had specialist medical help after two hours; manual removal of partially adherent placenta done, had PPH, blood transfusion; both babies died in a few hours; had drugs for suppression of lactation. She complained weakness, giddiness; became lean. On direct eliciting of history, she revealed that the breast size became less and she don’t have any sex desire.

Dull looking, pale, lean woman; anemic; parchment paper like dry, wrinkled skin; Temp 98.2 F; pulse rate 64 per minute; BP 100/66 mm of Hg supine, 90/60 mm of Hg sitting; small sagging breasts, areola pale; scaphoid abdomen; poor pubic hair; vulva shriveled, atrophied; no labial fat; labia minora pale. Vaginal wall dry with less rugosity. Cervix and uterus slightly undersized.

Hb 9.4 gms%; hematocrit 29%; ESR 45 mm first hour.

Fasting blood sugar 74 mg/dl; cholesterol 124 mg/dl.

Sodium 135 mEq/L
TSH 0.5 miu/L; FT3 1.1 pg/ml; FT4 0.7 ng/dl; prolactin 9 ng/ml; FSH 0.5 miu/ml; LH 0.6 miu/ml; estradiol 8 pg/ml; ACTH 6 pg/ml; cortisol 4 mcg/ml.

Ultrasonography: uterus 72x43x32 mm; endometrium thin streak; right ovary 25x18 mm; left ovary 24x16 mm; no follicular pattern.

Clinical diagnosis of Sheehan’s Syndrome is made.

She was given Prednisolone 20 mg and L Thyroxine 100 g for a month; from second month cyclical Ethinyl estadiol 50 mcg 21/28 and Progesterone 5 mg 10/28 was given. After 3 months, general condition improved, healthy looking, no giddiness; regained weight and skin turgor partially; vulva rejuvenated; had a minimal with drawl bleeding. For the next 6 months Estrogen, Progesterone, Thyroxine supplementation and Prednisolone 10 mg continued. One mg of Testosterone gel was given for six months. After 9 months of hormones, normal looking active female; axillary and pubic hair grown; vulva is almost normal looking; regularly menstruating. Advised to continue the same drugs.

**Discussion**

During pregnancy hypertrophy and hyperplasia of pituitary gland makes its volume almost double, without a corresponding increase in blood supply. In severe PPH with hypotension, shock, sluggish blood flow in the portal system of anterior pituitary leads to anoxia, thrombosis and necrosis of the anterior pituitary tissue. It is presumed that 32% of women with severe PPH develop some amount of pituitary damage. Necrosis of 50 % of pituitary tissue won’t cause any clinical problem. Destruction of 70-90 % of tissue results in symptoms of anterior pituitary hormone deficiency.

The manifestations of the disease depends upon the extent of the pituitary necrosis and damage to the individual trophic hormone areas of pituitary. Growth hormone secreting cells are in the periphery and are subjected to severe damage; but in adult women the symptoms of growth hormone deficiency are not pronounced. The order of the damage is Growth hormone, Prolactin, Gonadotrophins and lastly ACTH and TSH. Posterior pituitary secretions are neuro endocrines and are transported from hypothalamus. Hence post partum pituitary necrosis rarely involves posterior pituitary hormones. Diabetes insipidus is seen in only 5 % of cases of severe Sheehan’s Syndrome.

Sheehan’s Syndrome symptoms can present immediately after PPH or may present decades after the incident of PPH. Total pituitary necrosis manifests in the immediate post partum period. Hypotension, hypoglycemia, hyponatremia may
lead to coma and death. If the woman survives-hypotension, spells if giddiness, lactational failure is seen. Gonadotrophin deficiency manifests as non resumption of menses even after many years of delivery; lack of sex desire; poor axillary and pubic hair; less hair all over the body; loss of eye brows; atrophy of breasts; atrophy of external genitalia. Areola of breast and labia minora lose their dark colour. Vagina looks postmenopausal. Uterus may become atrophic. In less severe cases menses can resume, but oligomenorrhetic. Anovulation is common. In minimal gonadotrophic damage--women can conceive. Cortisol deficiency manifests as weakness, spells of giddiness, hypotension; loss of subcutaneous fat, loss of skin turgor results in pale, parchment paper like skin; wrinkles around mouth and eyes appear. Poor fat reserve and hypothyroidism leads to cold intolerance. Pituitary damage less than 50% is asymptomatic.

The management depends upon the condition of the patient at diagnosis. Revival of the woman and hormone replacement therapy is the treatment. Cortisone and Thyroxine replacement is lifelong. Estrogen and progesterone supplementation can be done till the age of natural menopause. Depression is to be managed with antidepressants. Low dose testosterone supplementation is helpful to revive sex drive.

Conclusions
Acute, fulminant hypo pituitarism is diagnosed and treated effectively. Less severe forms of Sheehan’s syndrome are insidious in onset, vague in presentation and so are under diagnosed. Eliciting history of PPH and high index of suspicion will unearth more cases of Sheehan’s Syndrome.

Conflict of interest: Nil

References