

# Sheehan's Syndrome: A Case Report

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Received December 23, 2013; Revised February 23, 2014; Accepted March 24, 2014

**Abstract** Sheehan's syndrome is defined by varying degrees of anterior pituitary deficiency due to postpartum ischemic necrosis of the pituitary gland after massive bleeding. Sheehan's syndrome, though rare, is still one of the commonest causes of hypopituitarism in developing countries like ours. The clinical presentation is variable with abrupt or insidiously developing pituitary insufficiency after a heavy intra-partum or postpartum haemorrhage. We present an young lady with this syndrome who had slowly progressive panhypopituitarism 6 years after a severe haemorrhage associated with the delivery of a male baby.

**Keywords:** Sheehan's syndrome, hypopituitarism, post partum ischaemic necrosis

**Cite This Article:** Mohammed Nasir Uddin, Mohammed Abbas Uddin Chy, and Taslima Khan, "Sheehan's Syndrome: A Case Report." *American Journal of Medical Case Reports*, vol. 2, no. 3 (2014): 50-51. doi: 10.12691/ajmcr-2-3-2.

## 1. Introduction

Sheehan's syndrome is a rare but potentially serious postpartum complication. It was first described in 1937 by Sheehan. Sheehan's syndrome is defined by varying degrees of anterior pituitary deficiency due to postpartum ischemic necrosis of the pituitary gland after massive bleeding. Sheehan's syndrome, though rare, is still one of the commonest causes of hypopituitarism in developing countries like ours. Although decreasing in frequency in recent years, it is still one of the commonest causes of hypopituitarism in developing countries owing to the lack of effective management of postpartum bleeding. The clinical presentation is variable with abrupt or insidiously developing pituitary insufficiency after a heavy intra-partum or postpartum haemorrhage. Its diagnosis is based on the clinical features of associated hormonodeficiency, a suggestive obstetric history, laboratory finding of decreased hormone levels, and related radiological features. Its treatment requires lifelong replacement of the deficient hormones.

## 2. Case Presentation

A 30 years young lady, married for 15 years having 3 child, from Satbaria, Chandanaish presented to OPD clinic with lethargy, loss of weight and appetite for a duration of eight months. The patient had noticed progressively increasing weakness, skin pallor and gradual loss of weight for the past 6 years before she came to OPD clinic.

Patient attendant complaints that she had been suffering from frequent attacks of unconsciousness for last several years, improved after administration of intravenous fluid. Repeated time she was diagnosed as Depressive illness,

Psychosis, Epilepsy. There was no history of meningitis, head injury, tuberculosis or diabetes mellitus in the past.

On further inquiry, the patient volunteered the history of heavy vaginal bleeding with retention of placenta following home delivery of male baby 6 years back. Immediately after the event the patient had become very sick; she was unconscious for hours and hospitalized and manual removal of placenta with 3 unit blood transfusion. The patient recalled that she had no menstruation after that incidence.

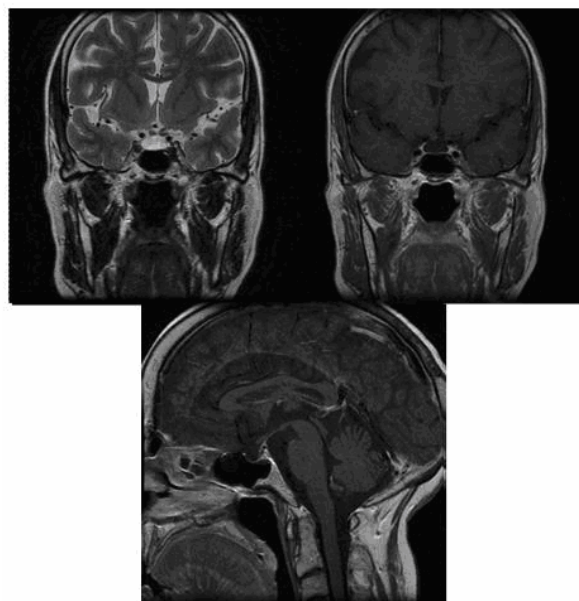


Figure 1. MRI showing an empty sella turcica

On examination, the patient was ill looking, thin, pale. The pulse was 88/min and blood pressure was 90/70mmHg (supine) and 70/60mmHg (2 mins after standing). The skin was pale with absence of hair from

skin. Examination of the lungs, heart and abdomen were normal. Examination of the central nervous system revealed generalized muscle and fat wasting and delayed relaxation of the ankle reflexes, the remaining examination was normal including fundoscopy.

Investigations revealed a haematocrit of 35% (with normocytic normochromic red cells), total WBC was 7800/ $\mu$ L with a normal differential count, urea was 8 mg/dL, creatinine 0.8 mg/dL, Na<sup>+</sup> 129 mEq/L, K<sup>+</sup> 4.3mEq/L, calculated serum osmolality 272, and urinary specific gravity 1.005.

Other investigations showed thyroid stimulating hormone (TSH) 0.448  $\mu$ lu/ mL, serum cortisol (at 9 am) 0.16 mcg/dL (when blood sugar was 52 mg/dL), follicle stimulating hormone (FSH) 0.66  $\mu$ lu/mL, leutinizing hormone (LH) 0.216  $\mu$ lu/mL. The diagnosis of Sheehan's syndrome was established and pituitary magnetic resonance images showing empty sellaturcica. She received replacement therapy of hydrocortisone 15 mg in morning and 5 mg at night, thyroxine 150 mcg daily.

### 3. Discussion

Sheehan's syndrome refers to postpartum hypopituitarism as a result of pituitary necrosis occurring during severe hypotension or shock secondary to massive bleeding during or just after delivery. Though first described by HL Sheehan in 1837, it was known as Simmond's disease until 1939 when Sheehan described the disease was due to postpartum necrosis of the anterior pituitary following postpartum haemorrhage. [1] Owing to improved obstetric care and effective management of post-partum haemorrhage in more developed countries, the prevalence of Sheehan's syndrome is decreasing. However, in a developing country like ours, it is still encountered at times as postpartum bleeding is common and timely intervention is not possible in many remote and rural areas.

The underlying process leading to Sheehan's syndrome is the infarction of the physiologically enlarged pituitary gland, particularly anterior lobe, secondary to the grossly decreased blood supply during intra-partum or postpartum events. Though vasospasm, autoimmunity, small sella size, and disseminated intravascular coagulation may also have role in the development of Sheehan's syndrome, none has been conclusively proven [2].

The clinical presentation of Sheehan's syndrome from long-standing non-specific features such as weakness, fatigue, and anaemia to profound abrupt hypopituitarism resulting in coma and death. The mean duration between postpartum bleeding and the subsequent development of symptoms varies from 1 to 33 years; [3] this period lasted more than six years in our patient. Clinical features are the result of the deficient hormones that may be single or many, but symptoms due to GH deficiency usually appear earliest. Our patient had clinical features consistent with GH, ACTH and TSH deficiency. Although failure of postpartum menstruation due to deficiency of FSH and LH is quite common, spontaneous pregnancies have been reported [4].

The diagnosis of Sheehan's syndrome is based on the features of hormone deficiency, a suggestive obstetric history, and decreased basal hormone levels (freeT3, free T4, TSH, cortisol, ACTH, FSH, LH, oestrogen, prolactin and insulin like growth factor-1). The presenting symptoms, though vague, were suggestive of hypopituitarism in our

patient. The finding of a normal pulse rate in the presence of significant postural drop was noteworthy as she had coexisting hypothyroidism and hypocortisolism. The delay in diagnosis was probably due to her vague symptoms and inadequate obstetric history. A dynamic pituitary function test like insulin tolerance test (ITT) is helpful to assess the pituitary reserve of GH and ACTH. We did not carry this test out in our patient as she already had a low blood sugar. MRI or CT of pituitary often shows an empty sella. Criteria have been suggested for the diagnosis of Sheehan's syndrome.

The treatment of Sheehan's syndrome is replacement of the deficient hormones. ACTH and TSH deficiencies should be replaced with glucocorticoids and thyroxine respectively; mineralocorticoid replacement is usually not required. Sex hormone replacement is important in premenopausal patients and GH replacement has shown improved lipid profile and quality of life in these patients [5].

### 4. Conclusion

Postpartum pituitary necrosis is a known complication, but it is now rarely seen. Even if postpartum hemorrhage has been well managed, this complication cannot be excluded, and it can be life threatening. It is necessary to consider this diagnosis in all patients having presented with a cardiovascular collapse during childbirth, whatever the cause, and in the presence of the classic signs of pituitary insufficiency. Multiple anterior pituitary hormone deficiencies in Sheehan's syndrome can be responsible. A simple replacement therapy with thyroid and cortisol hormones results in complete recovery.

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