



## Sheehans Syndrome – Atypical Presentation - Case Report

Authors

**Dr Aiswarya Kosaraju<sup>1</sup>, Dr Vengada Krishnan Krishnamoorthy<sup>2</sup>**

<sup>1</sup>Final Year Resident in the Dept of General Medicine, SRMC, Chennai

<sup>2</sup>Professor in the Dept of General Medicine, SRMC, Chennai

### Abstract

*Sheehans Syndrome (SS) is postpartum hypopituitarism caused by ischemic necrosis of the pituitary gland. Usually it results from severe hypotension or shock caused by massive haemorrhage during or after delivery. Enlargement of pituitary gland, small sellar size, DIC and autoimmunity are considered as other causal factors. Its incidence is currently decreasing in developed countries owing to improved obstetric care. We are here by presenting a case report of 53 year old female who had history of PPH during her second pregnancy, presented to casualty with complaints of fever, vomiting, loose stools and shortness of breath. On evaluation she was diagnosed as SCRUB typhus and found to have pancytopenia which did not improve with the treatment of underlying infection. In due course patient developed hyponatremia. On subsequent evaluation she was diagnosed to have hypocortisolism and hypothyroidism. MRI Pituitary was suggestive of SHEEHANS SYNDROME. In view of non specific presentation and little importance paid to the menstrual history there was a delay in the diagnosis and treatment.*

### Case Report

A 53 years old female, who is known case of old PTB during her childhood (completed treatment course), G3P2A1L2, post hysterectomy due to PPH during her second pregnancy, reported to the casualty with complaints of fever for 5 days, vomiting for 3 days, loose stools for 3 days and shortness of breath for 1 day. Clinical examination revealed that patient has pallor, with bilateral crepitations in the lungs on auscultation. Assessment of vitals revealed that patient has normal blood pressure and pulse rate with a low room air saturation of 93%. Blood investigations sent showed low HB (7.6 gm/dL), low totals counts (1900cmm), low platelets (0.46 lakhs /cmm), with normal renal function tests and serum electrolytes. Liver function test showed mild

elevation in Total bilirubin (2.13mg/dl), with normal liver enzymes and a low albumin (2.7mg/dl). X ray chest was done and it showed features suggestive of ARDS and blood gas analysis revealed hypoxia. Diagnosis of? ARDS, ? H1N1,? SCRUB fever was made and the patient was admitted to intensive care unit and was started on T.OSELTAMIVIR and T DOXYCYCLINE and patient was connected to non invasive ventilation. On subsequent evaluation it was found out that the patient was SCRUB positive, hence treatment with antibiotics was continued and later after the patient improved symptomatically was shifted back to ward.

In view of persisting pancytopenia on 5<sup>th</sup> day of hospitalization (HB -7.8mg/dl, TC 2200cmm, PLT - 0.71 lakhs/cmm) further work up was done

to rule out the cause of pancytopenia. Iron studies and Vitamin B 12 was sent which was within normal limits, USG abdomen done was normal, peripheral smear was done and showed normocytic normochromic anemia with thrombocytopenia and leucopenia, ANA-negative, DS DNA negative, RF factor within normal limits , thyroid function test were normal (FT4 – 0.60 ng/dl, FT3 – 2.67pg/ml, TSH – 2.66 MIU/dl ) sr cortisol 13 microgram/dl, ESR – 30 mm/mg % . CT THORAX was also done and it showed old PTB changes and no evidence of interstitial lung disease.

During the evaluation of thrombocytopenia patient developed vomiting and altered sensorium in the ward and urgent serum electrolytes were sent which showed hyponatremia (NA – 108mmol/litre, CL- 78mmol/litre, K – 3.3mmol/litre) hence the patient was shifted to ICU and was started with 3% NACL correction. Repeat cortisol and TFT were sent and it was found that 8 am cortisol was only 0.8 microgram / dl and thyroid hormones were found to be low and hence the patient has been started on IV Hydrocortisone 50 mg iv tds and the sodium improved gradually to 128 over the next three days and patients sensorium improved. In view of hypocortisolism and low FT3, FT4 levels and with underlying history of PPH, MRI of the pituitary gland was done and reported as empty sella and on evaluation other pituitary hormones were also found to be decreased. Diagnosis of SHEEHANS SYNDROME was made and the patient was continued on steroid and thyroid supplementation. During the treatment patient developed Acute Stress Psychosis secondary to steroid therapy and received psychiatric counselling. Patient was discharged with oral steroids and blood counts were repeated after 6 days and it showed normal HB, TC, PLATELETS.



MRI Pituitary Showing Empty Sella

### Discussion

Sheehans syndrome (SS) is postpartum hypopituitarism caused by ischemic necrosis of the pituitary gland. It usually occurs as a result of severe hypotension or shock caused by massive hemorrhage during or after delivery.<sup>(1)</sup> Enlargement of pituitary gland, small sellar size, disseminated intravascular coagulation (DIC) and autoimmunity have been suggested to play a role in the pathogenesis of SS. Its frequency is decreasing in Developed countries owing to advances in Obstetric care.

SS can present with lactation failure during postpartum period, failure to resume menses, genital and axillary hair loss, generalized weakness, fine wrinkles around the eyes and lips, signs of premature aging, dry skin and hypopigmentation and other evidence of hypopituitarism.

Studies have suggested that the Anterior Pituitary Hormones were found to be more involved than the Posterior Pituitary Hormones. GH deficiency is very common because somatotrophes are located in the lower and lateral regions of the pituitary gland and are most likely to be damaged by ischemic necrosis of the pituitary gland. The

order of frequency of hormone loss has generally been found to be growth hormone and prolactin, gonadotropins, ACTH and thyrotropin . At least 75% of pituitary must be destroyed before clinical manifestations become evident.<sup>(2)</sup>

\*Hyponatremia is the most common electrolyte disturbance.<sup>(3)(4)</sup>

Hypothyroidism and glucocorticoid deficiency by decreasing free water clearance independent of vasopressin cause hyponatremia. SIADH and volume depletion are the other factors leading to hyponatremia, which might have caused low sodium in our case .

Hematological abnormalities like anemia, thrombocytopenia, pancytopenia associated with hypocellular marrow occur because of hypopituitarism and complete recovery have been shown to occur after achieving eucortisolemic and euthyroid state .

\*Hypopituitarism also causes neuropsychiatric symptoms, by increased neural excitability and increased conduction velocity along peripheral axons while prolonging conduction across synapses. This probably delays the arrival of signals from the periphery to the central nervous system, resulting in a deficit between perception and integration of sensory signals, and is a possible mechanism for hallucinations and psychosis.

Radiologically hypopituitarism manifests as emptysella or partially empty sella, pathologically there is central ischemic necrosis in an enlarged gland followed by pituitary gland atrophy and empty sella. Hormonal essays show a low level of T4, Estrogen, Gonadotropin, Cortisol and ACTH depending on the extent of necrosis .

Treatment of hypopituitarism is to replace deficient hormones life long, acute psychosis occurring during steroid therapy reverts back to normal once eucortisol state is reached.

Delayed diagnosis in this syndrome is mostly due to the fact that symptoms are non-specific, lack of awareness in the medical fraternity regarding the condition and the little importance that is paid to

the menstrual history in a woman presenting with such symptoms. Physicians must be aware that Addison's disease may present solely with psychiatric symptoms and maintain a high index of suspicion for this potentially fatal condition<sup>(5)</sup>.

### References

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