*Case Report*

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# Sheehan's Syndrome and Psychosis

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**Abstract**

Postpartum hypo-pituitarism is also termed as Sheehan's Syndrome. Heavy bleeding during or after delivery leading to further necrosis of the pituitary gland are all characteristic seen in Sheehan's Syndrome. It is commonly associated with hypo-volemic shock. The presentation of Sheehan's Syndrome most commonly include amenorrhoea, hypoglycaemia, hypothyroidism along with agalactorrhea, also psychiatric manifestations like commonly as psychosis are seen though they are not uncommon to be seen. In developed countries owing to advances in services of obstetric care the occurrence of Sheehan's Syndrome is seen relatively rare and is overall gradually decreasing worldwide. However, it can still be considered as common and relatively frequent seen in underdeveloped and developing countries. The diagnosis of Sheehan's Syndrome is commonly late as it evolves slowly, reporting of psychoses are quite rarely seen in cases of Sheehan's Syndrome. Herein in our study we discuss a case of 37 year old woman, diagnosis was psychotic disorder along with Sheehans Syndrome, diagnosed years later, with etiological aspects, follow-ups period and the details treatment with thyroxine, glucocorticoids which has shown a tremendous results with complete remission after attaining euthyroid and eucortisolemic state.

**Keywords**: Thyroxine; Psychosis; Sheehan's Syndrome; Post-Partum Hypo-Pituitarism.

## Introduction

Sheehan's syndrome (SS) is a quite rare and uncommon but serious postpartum complication; it was described by Sheehan in the year 1937. Defining of Sheehan Syndrome by varying degrees of anterior pituitary deficiency which is due to postpartum ischemic necrosis of the pituitary gland seen usually after massive bleeding. Sheehan's syndrome rarely encountered, but still can be considered as one of the commonest causes of hypo-pituitarism commonly seen in developing country like India. Adrenal pituitary insufficiency accoutring from hypo-volemia which is secondary to excessive blood loss during or after delivery is Sheehan syndrome. Sheehan syndrome some time may also present in post-partum period or years and decades after the delivery [1].

In Sheehan's syndrome implications of abnormalities of hypophyseal arteries which include external compression, thrombosis and as well vascular spasm are done. Various factors had been proposed in Pathogenesis of Sheehan's syndrome viz, DIC, Small sized sella, autoimmunity and enlargement of pituitary gland [2]. Sheehan's Syndrome is being diagnosed which is based on various clinical features which are like associated hormone deficiency, history related with obstetric quite suggestive, findings of laboratory of decreased hormone levels and related with radiological features. Treatment of Sheehan's Syndrome usually needs lifelong replacement of hormones which are deficient.

From Indian subcontinent study of Sheehan's Syndrome made by Zargar A H et al. from Kashmir valley, the prevalence of Sheehan's Syndrome was estimated to be around 3% in the women who are above 20 years of age and it was almost two-thirds

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wherein the delivery of babies had occurred at home [3]. However, the same is a quite rare cause of hypo-pitutarism commonly in developed countries.

In another study, the findings reveal that among 1034 hypo-pituitary adults, sixth most frequent cause of growth hormone deficiency (GHD) was Sheehan's Syndrome, it was being considered responsible for around 3.1% of cases in the study [4]. In this study, we discuss a case in a 37 year old woman, that in which the diagnosis was a psychotic disorder and Sheehan's Syndrome, diagnosed years later, and the etiological aspect, including the follow-up period and treatment with thyroxine and glucocorticoids resulted in complete remission after attaining euthyroid and eucortisolemic state.

**Case Report**

Female Patient of 37 years age, Muslim by Religion, housewife, presented to the department of psychiatry along with her mother with the complaints of hearing voices, she lives in self with poor self care, she had no work and is not involved in any kind of activity, she had sleep disturbance and used abusive language for friends and family and all this since past two decades.

Patient's illness is continuous and progressing gradually. On further detail examination patients Mother revealed that she delivered her second child which was vaginal delivery but there had been excessive bleeding but it was hospital delivery. As informed by mother, the mattress was filled with blood and she was administered with haemostatic injections. D & C occurred 1 month after pregnancy. Immediately after the pregnancy within one month she stopped lactation and she had amenorrhea. Relatives and parents noticed drastic change in patient's behaviour which was apparently began after around two years post delivery of second child. The complaints include used to sleep 2-4 hrs at night and used to shout. She had complaints of hearing voices, contents were derogatory and threatening. She lived with fearfulness. Whole day she sat on one place, did not do any work and always used to mutter.

Patient had tendency to neglect her personal hygiene also she was negligent in her child's care. She started excessive use of tobacco chewing so that she has decreased mouth opening. In initial few years she lived with her husband but later she lived many times in parental home. She was looked after by her mother since last 4 years. Her husband and two children live separately. She does not take meals properly, so her general physical condition becomes worsen. Patient took regular multiple consultations from many psychiatrists for abnormal behaviour. Currently she is on antipsychotic medication but her symptoms persist. There was not any significant past psychiatry and neurological history. Patient husband drink alcohol in dependence form, so quarrel between patient and her husband very common, her grandfather also suffered from seizure disorder. Patient never had any kind of smoking nor had any drinks of alcohol nether abused any kind of illicit drugs, overall there was not any kind of past forensic record related to her.

**General Physical Examination**

Patient appeared anaemic and looked pale; on examination breast atrophy was appreciated as the important features of hypothyroidism. Her vital signs were seen abnormal and she was febrile. Her blood pressure was 75/55 mm of Hg, pulse rate 60/min. Skin has Icthysosis, no pubic and axillary hairs. She has difficulty in hearing and sub-mucous fibrosis present in oral cavity. Patient was clear and well oriented to time place and person. Bilateral up going plantars was revealed during Neurological examination of the patient. Gait appeared to be normal. Patient appearance was unkempt apathetic, revealed during mental status examination. She had a monotonous speech, brief reply and a very high reaction time was appreciated. Patients thought process had persecutory delusions, with auditory hallucinations, with intact memory. However she had unsatisfactory performance in attention and concentration task with verbal fluency test. Patient had no insight and understanding regarding why she had been to JNU Hospital in psychiatry department along with poor social judgement.

No abnormalities revealed on laboratory investigation which include Liver function, renal function as well as workup for inflammatory and infectious conditions. Patients profile for Hormones revealed very low levels of serum T3, T4, cortisol, FSH & LH and is being depicted in the Table 1. below normal range came for all electrolytes. Post menopausal shrunken ovaries were seen in Ultra sonography of abdomen and pelvis, whereas Magnetic resonance imaging of brain showed prominent supra-tentorial ventricular system with commensurate enlargement of sylvian fissure and cortical sulci suggestive of cerebral and cerebeller atrophy. Bilateral moderate to severe hearing loss was present on Audiometry.

**Table 1:** Laboratory Findings in the Patient presented

| Investigation | Status | Result | Unit | Bio Ref Int | At Discharge |
|---|---|---|---|---|---|
| HB | L | 8.3 | Gm% | 12 -15 | 8.4 |
| TLC | L | 2.9 | Thousand/cu mm | 4-11 | 8.4 |
| N | | 46 | % | 40 -80 | 71 |
| L | | 50 | % | 20 – 40 | 26 |
| M | | 02 | % | 2 – 10 | 03 |
| E | | 02 | % | 1 – 6 | 00 |
| B | | 00 | % | 0 -2 | 00 |
| TRBC | L | 2.85 | Millions/Cumm | 4.5 – 5.5 | 2.95 |
| PLATELETS | L | 111 | Thousand/ Cumm | 150 - 500 | 155 |
| MCV | L | 79.3 | FL | 83 - 101 | 84.9 |
| MCH | | 29.1 | PG | 27 – 32 | 29.4 |
| MCHC | H | 36.7 | G/DL | 30 - 35 | 34.6 |
| PCV | L | 22.6 | % | 38 - 45 | 22.9 |
| IRON | | 61 | UG/DL | 50 - 170 | |
| PBS - RBCs | Normocytic Normochromic Picture, Marked Anisopoikilocytosis Microcytes ++ Hypochromasia + Target Cell ++ | | | | |
| WBCs | | | Reduced On Smear | | |
| PLATELETs | | | Reduced On Smear | | |
| RBS | | 75 | MG/DL | 74 - 140 | |
| SE UREA | | 21 | MG/DL | 15 - 45 | |
| SE CREATININE | | 0.8 | MG/DL | 0.5 – 1.1 | |
| SODIUM | LL | 115 | MMOL/L | 135 - 145 | 127 |
| POTASSIUM | L | 3.2 | MMOL/L | 3.5 – 5.1 | 3.9 |
| CHLORIDE | L | 83 | MMOL/L | 98 - 110 | 93 |
| CALCIUM | L | 6.9 | MG/DL | 8.6 – 10.20 | 7.8 |
| BILIRUBIN T | | 0.5 | MG/DL | 0.0 – 2.0 | |
| DI | | 0.2 | MG/DL | 0.0 – 0.25 | |
| INDI | | 0.3 | MG/DL | 0.2 - 10 | |
| SGOT | H | 113 | U/L | 5-46 | |
| SGPT | | 47 | U/L | 0-49 | |
| ALP | H | 288 | U/L | 42-141 | |
| TOTAL PROTEIN | | 6.5 | G/DL | 6.0 – 8.0 | |
| ALBUMIN | | 3.8 | G/DL | 3.5 – 5.5 | |
| GLOBULIN | | 2.7 | G/DL | 2.3 – 3.6 | |
| A/G | | 1.4 | | 1.0 – 2.0 | |
| VIT D | | 56.1 | NG/ML | 30 - 100 | |
| VIT B 12 | H | >2000 | PG/ML | 239 - 931 | |
| FREE T3 | L | 0.71 | PG/ML | 2.7 – 5.27 | |
| FREE T4 | L | 0.07 | NG/ML | 0.78 – 2.19 | |
| TSH | | 1.75 | uIU/ML | 0.465 – 4.68 | |
| LH | L | 0.87 | mIU/ML | 15.9 – 54.00 | |
| FSH | L | 3.42 | mIU/ML | 21.5 - 131 | |
| CORTISOL | L | 0.67 | ug/dl | 5 - 23 | |
| ACTH | L | <5 | Pg/ml | 9 - 52 | |
| PROLACTIN | L | <0.6 | NG/ML | 5.18 – 26.53 | |
| DENGUE SEROLOGY | | | Non Reactive | | |
| MP CARD | | | Negative | | |
| CHEST X RAY (PA VIEW) | | | Normal | | |
| USG (ABDOMEN – PELVIS) | | | Post Menopausal Ovary Contracted | | |
| ECG - | | | T Wave Flattening In Lead 1,Avl, V4,V5,V6 | | |

Finally the diagnosis of Sheehan's syndrome was finalised and patient was started with Aripiprazole 5 milligram for one week followed by 10 mg per day with tablet Clonazepam 0.5 mg 1HS or SOS, along with Injection Hydrocortisone 50 mg Intra venous QID for three days, followed by 25 mg/day orally, tablet Thyroxin 50 ug OD after five day 100 ug, Capsule Calcitriol 0.25 mg- 1 OD. With this overall treatment patient had a drastic improvement within few weeks for all of her physical and psychiatric symptoms. She was convinced for continuation of her treatment for her life time with counselling and the importance of continuous treatment and regular follow-up was made understood.

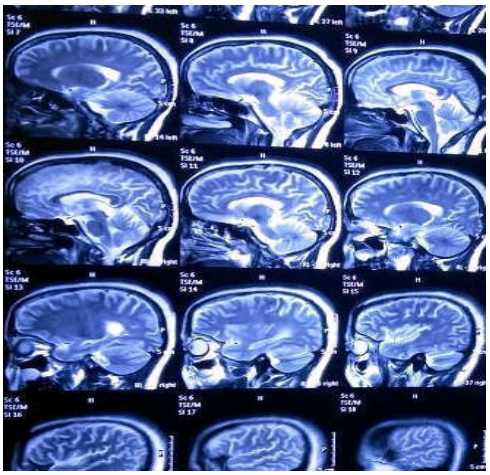

**Fig. 1:** MRI BRAIN reflecting marked atrophy of Anterior Pituitary with partially empty sella

## Discussion

Physician's diagnosis of Sheehan's syndrome is purely based on the history given by the patient regarding postpartum haemorrhage which is usually followed by anterior pituitary failure which is complete along with MRI findings demonstrated as empty sella. Anterior pituitary which results in ischemic necrosis and hypo-pituitarism occur is commonly seen during pregnancy with enlargement of pituitary gland and postpartum haemorrhage [5].

Following the delivery the Sheehan's Syndrome can be presented soon or later even after few months or years later. In a study with sixty numbers of patients that revealed the average time between the earlier obstetric history and coming to diagnosis of Sheehan's syndrome shall be around thirteen years [6].

Failure to lactate, amenorrhoea, genital and Axillary hair loss, weakness, asthenia, wrinkles around eyes, lips which were very fine, along with prominent signs of aging with dry skin, hypo-pigmentations and altogether evidence suggestive of hypo-pituitarism were the usual features patient presented with. However. it is also to note that just mere presence of postpartum lactation or absence of amenorrhea is not the criterion to rule out the diagnosis of Sheehan's syndrome. It can also present as an emergency with severe hypo-natremia, hypoglycaemia, circulatory collapse, congestive cardiac failure or some time with psychosis [7].

In a study made by Ozkan and Colak, revived around twenty cases of Sheehan's Syndrome, around fifteen percent presented with hypoglycaemia and hypothyroidism, whereas hyponatremia seen in five percent of cases, six cases showed empty sella and in nine cases showed partial empty sella [1].

In Sheehan's syndrome very little attention was made towards psychoses, the reason for this neglect may be due to rarity of the disorder especially in western countries. First postpartum psychosis in a case of Sheehan's syndrome was being reported in the study of Kale K et al. [8]. In our case also the patient presented with psychosis along with Sheehan's syndrome. Our patient developed Sheehan's syndrome which proceeded by postpartum haemorrhage. Sudden drop of hormones level which leads to relative deficiency of such hormones during postpartum was appreciated and these may be responsible for some related psychiatric symptoms [9]. It is very clear from the study that either in initial stages or when the patient shows psychotic symptoms, antipsychotics may need to be administered to the patients as an adjunct [10]. Complete recovery resulted when the patient had treatment with Thyroxine and Gluco-corticoids soon after attaining euthyroid and eucortisolemic state.

## Conclusion

Sheehan's syndrome patient presenting with psychosis is relatively uncommon. Treating physicians in case of postpartum- psychosis, with significant and prominent obstetric history must have a strong index of suspicion. With this case report presentation we emphasize the clear importance of meticulousness in history taking and patient's examination with postpartum psychosis.

*Competing Interests*

Authors have declared that no competing interests exist.

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Dr. SKG made the diagnosis and was involved in the management of the patient. Dr. NAS wrote the manuscript and per- formed the literature search and corrected the manuscript. Both authors read and approved the final version of the manuscript.

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