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RESEACH ARTICLE

SHEEHAN'S SYNDROME: A CASE REPORT

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Abstract

Background: Sheehan's syndrome also known as postpartum hypopituitarism is a rare complication of postpartum haemorrhage which was first described by Sheehan in 1937. It occurs as a result of ischaemic necrosis of the pituitary gland due to severe postpartum haemorrhage. It is one of the most common causes of hypopituitarism in developing countries. **Objective:** we aimed to present a rare case of Sheehan's syndrome from our centre. Case presentation: **Case Presentation:** We present the case of 40 –year-old woman, with partial hypopituitarism after a home delivery associated with a massive postpartum haemorrhage nine years previously. She presented with amenorrhea and fatigue. Complete clinical evaluation, hormone profile revealed partial hypopituitarism. A remarkable improvement was noted after commencement of glucocorticoid replacement. **Conclusion:** Clinicians should have high index of suspicion of the condition and the possibility of late presentation in order to diagnose and treat promptly.

Key words: Hypopituitarism, Pituitary gland, postpartum haemorrhage, Sheehan's syndrome.

INTRODUCTION

Sheehan's syndrome also known as postpartum hypopituitarism is a rare complication of postpartum haemorrhage (PPH) which was first described by Harold Leeming Sheehan in 1937 (Gardner & Shobach, 2018). It occurs as a result of ischaemic necrosis of the pituitary gland due to severe postpartum haemorrhage and vascular collapse leading to varying levels of hypopituitarism (total or partial) and the presentation may be delayed (Gardner & Shobach, 2018). It is reported that Sheehan's syndrome accounts for 0.5% of all known cases of hypopituitarism in females (Genetu *et al.,* 2021). It occurs in 5 out of every 100,000 births (Schury & Adigun, 2019; Shivaprasad, 2011). The prevalence is much higher in developing countries and the prevalence is as high as 3.1% in Kashmir region of India, where more than half of the affected individuals had home deliveries (Karaca *et al.*, 2016). The mechanism for the ischemia in Sheehan's syndrome is not certain. Hypotension along with vasospasm of the hypophysial arteries is currently believed to compromise arterial perfusion of the anterior pituitary (Karaca *et al.,* 2016). During pregnancy, the pituitary gland is more sensitive to hypoxemia because of its increased metabolic needs and more susceptible to the prothrombotic effects of the hyperoestrogenic state. Some

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investigators have noted that the hypopituitarism does not always correlate with the degree of haemorrhage but that there is good correlation between the pituitary lesion and severe disturbances of the clotting mechanism (as in patients with placenta Previa) (Karaca *et al.,* 2016). Characteristic manifestations include failure to lactate, amenorrhea, pubic and axillary hair loss, as well as other evidence of hypopituitarism (Otsuka *et al.,* 1998).

Recent progress in obstetric care has greatly reduced the incidence of Sheehan's syndrome as such new cases are seldom encountered (Gardner & Shobach, 2018). Diagnosis of Sheehan's syndrome is done based on important physical examination findings, including breast atrophy, loss of axillary or pubic hair and myxoedema; relevant laboratory investigations such as pituitary hormone profile, complete blood cell count and biochemical parameters (including glucose, sodium and potassium).

Treatment of hypopituitarism includes hormone replacement therapy. It involves replacement of adrenal hormone first and then subsequent replacement of thyroid hormone and gonadotropins. Hydrocortisone is replaced first because thyroxine therapy can exacerbate glucocorticoid deficiency and may induce an adrenal crisis (Lamberts et al., 1998; Orrego & Bargan 2000). Replacement of growth hormone is necessary in children with hypopituitarism but is controversial in adults. Some people with severe growth hormone deficiency derive great benefit from replacement, but standard recommendations are not available (Davies *et al.,* 2000).

CASE REPORT

A 40 year old Para7 whose last childbirth was 9 years ago presented with complaints of amenorrhea of 9 years duration, fatigue, occasional dizziness and constipation. There was also history of lactation failure following her last delivery. She had unsupervised home delivery that was complicated by severe PPH as a result of which she had four units of blood transfused. Soon she developed lactation failure for which she resorted to formula feeding of her baby. The patient visited several healthcare facilities where she was often given Intravenous fluid and combined oral contraceptive pills.

Physical examination on presentation revealed a hyper pigmented young lady who was mildly pale, with scanty axillary hair and blood pressure of 80/50mmHg. The combination of history (PPH, lactation failure), clinical examination findings (hypotension, hyperpigmentation, scanty hair) and biochemical evidence (low cortisol, LH, FSH, Prolactin) confirms the diagnosis of Sheehan's syndrome. MRI was not done due to financial constraint. The result of the investigation is shown in table 1. The patient was commenced on 30mg of hydrocortisone (20mg am and 10mg pm) following which her condition gradually improved. She was advised to wear a Medic Alert bracelet, to indicate that, she is a patient with adrenal insufficiency that may require assistance.

Table 1: Laboratory characteristics

Laboratory investigations	Values	Reference range	Interpretation
WBC	5.48 x10^9/L	3.50 – 11.00	Normal
Haemoglobin	11.8 g/dL	11 – 17.5	Normal
Platelets count	242 x10^9/L	100 – 300	Normal
AST	43 i.u/L	5 -46	Normal
ALT	43i.u/L	6 – 49	Normal
ALP	92 i.u/L	90 -279	Normal
TSH	0.7 mIU/L	0.5 – 5.0	Low normal
T3	1.0 ng/dL	0.7 – 2.4	Normal
T4	54 nmol/L	43 -131	Normal
Prolactin	1.17 ng/dL.	3.3 – 26.7	Low
Random cortisol	30.14 nmol/L	240 – 618	Low
8 am Cortisol	41.5 nmol/L	100 – 550	Low

Keys: AST = Aspartate aminotransferase, ALT = Alanine aminotransferase, ALP= Alkaline phosphatase, TSH = Thyroid stimulating hormone, T3 = Triiodothyronine, T4 = Thyroxine, WBC =white blood cells count.

DISCUSSION

Sheehan's syndrome or necrosis of the pituitary gland, is a rare complication of postpartum haemorrhage initially described in 1937 (Karaca *et al.,* 2016; Gardner & Shobach, 2018). This case illustrates an example of Sheehan syndrome with delay in diagnosis. The pituitary gland is physiologically enlarged in pregnancy and is therefore very sensitive to the decreased blood flow caused by massive haemorrhage and hypovolemic shock. Women with Sheehan's syndrome have varying degrees of hypopituitarism, ranging from pan-hypopituitarism to only selective pituitary deficiencies (Iwasaki *et al.,* 1989; Schrager & Sabo, 2001). The anterior pituitary is more susceptible to damage than the posterior pituitary.

Diagnosis of Sheehan syndrome can be difficult. The diagnosis is based on clinical evidence of hypopituitarism in a woman with a history of a postpartum haemorrhage. Deficiencies of specific anterior pituitary hormones will cause varied symptoms. Corticotrophin deficiency can cause weakness, fatigue, hypoglycaemia, or dizziness. Gonadotropin deficiency will often cause amenorrhea, oligomenorrhoea, hot flushes, or decreased libido. Growth hormone deficiency causes many vague symptoms including fatigue, decreased quality of life, and decreased muscle mass (Shivaprasad, 2011). The patient in the current case report presented with similar symptoms suggestive of corticotrophin and gonadotropin deficiencies.

Secondary hypothyroidism is clinically indistinguishable from primary hypothyroidism, but patients with hypothyroidism caused by hypopituitarism have low T3 and T4 levels with normal or even inappropriately low TSH levels. Diagnosis of pan-hypopituitarism is straightforward, but partial deficiencies are often difficult to elicit (Dejager *et al.,* 1998). A woman with pan-hypopituitarism will have low levels of pituitary hormones (luteinizing hormone, corticotrophin, and thyrotropin) as well as the target hormones (cortisol and thyroxine). Recent report showed, that abnormal expression of HESX1, TLE1, TLE3 and MSX2 genes may cause a genetic predisposition to the development of Sheehan's syndrome (Diri *et al.,* 2016).

In this case, the diagnosis of Sheehan syndrome was suspected because of her history of PPH, skin hyperpigmentation, low blood pressure and low baseline cortisol and low normal TSH, in keeping with secondary hypothyroidism.

Radiologic imaging with either computed tomography or magnetic resonance imaging is usually not helpful in the acute phase and has not been used frequently in acute diagnosis (Lamberts *et al.,* 1998). However, with progression the radiological imaging may reveal empty sella, in women with Sheehan syndrome further (Otsuka *et al.,* 1998; Banzal & Ayoola, 1999). Imaging can be helpful in situations where the diagnosis is not apparent. Although the diagnosis in the current case is apparent, still a brain MRI was requested to confirm empty Sella and rule-out other pituitary pathology. However, the MRI was not done due the high cost of the investigation. The patient was commenced on 30mg of hydrocortisone (20mg am and 10mg pm) following which her condition gradually improved. She was advised to wear a Medic Alert bracelet, to indicate that, she is a patient with adrenal insufficiency that may require assistance.

Conclusion

Sheehan's syndrome is uncommon as a result of improved obstetric care. The diagnosis of this disease should be considered in any woman who reports signs or symptoms of pituitary deficiency in a background history of severe postpartum haemorrhage. Clinicians should have high index of suspicion of the condition and the possibility of late presentation in order to diagnose and treat promptly to prevent complications associated with the disease.

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