e - ISSN - 2349 - 8005

INTERNATIONAL JOURNAL OF ADVANCES IN CASE REPORTS

Journal homepage: www.mcmed.us/journal/ijacr

DELAYED PRESENTATION OF SHEEHAN SYNDROME ASSOCIATED WITH HYPOGLYCEMIC CRISIS: A CASE REPORT

Homaid Alsahafi*, Bndar Damanhori, Hasan Almatrafi

Endocrinology Department, King Faisal General Hospital, Makkah, Saudia Arabia.

Corresponding Author: - **Homaid Alsahafi**
E-mail: mezo106@yahoo.com

Article Info
Received 03/09/2014
Revised 12/09/2014
Accepted 19/09/2014

Key words:
Sheehan's syndrome, Hypopitutrism, Hypoglycemic attacks

ABSTRACT

Sheehan's syndrome is rare put still one of a common cause of hypopitutrism in developing countries. Its presentation is variable and may delay for many years after the onset of post-partum hemorrhage. In our report we report an elderly Lady who presented with Sheehan 's Syndrome in the form of recurrent severe hypoglycemic attacks 32 years after her last delivery which was complicated with sever post-partum hemorrhage and inability to feed her son.

INTRODUCTION

Sheehan syndrome (SS) or post-partum pituitary necrosis is an adeno-pituitary insufficiency result from severe hypotension secondary to excessive blood losses during or after the delivery [1].

The clinical presentation of this syndrome is variable; the patient can present abruptly with acute hypopituitarism or insidiously with non-specific features. Its diagnosis is based on the clinical features of associated hormone deficiency, 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 report

66 years old elderly Sudanese lady was seen in endocrinology clinic at king Faisal general hospital Makkah in December 2012 with history of recurrent attacks of symptomatic severe hypoglycemia for the last 6 month reaching 38mg/dl associated with coma, that obligate her to visit emergency department for intravenous dextrose resuscitation.

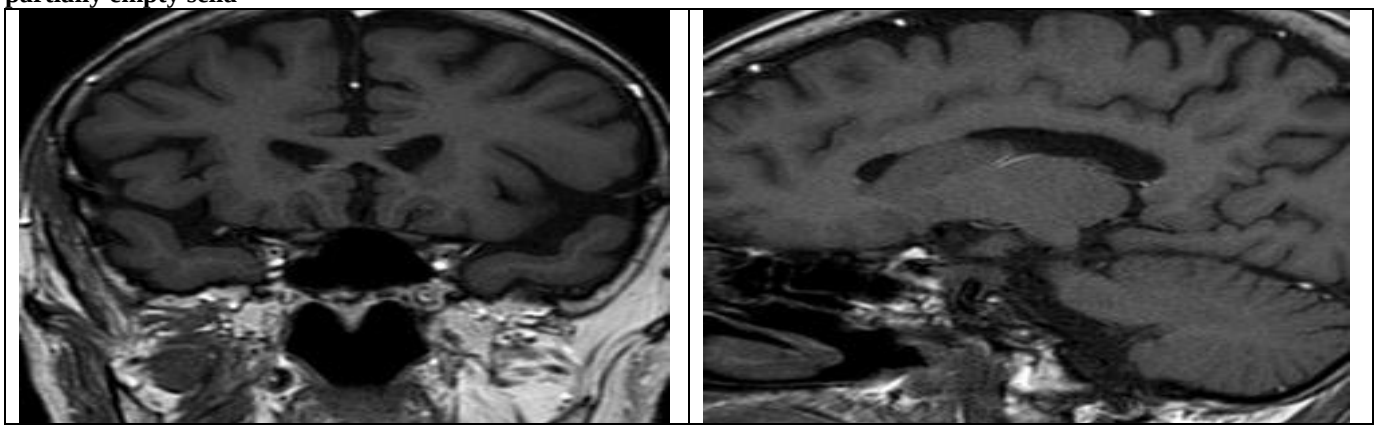
She had history of malaise, lethargy, cold intolerance and constipation, no history of polyuria or polydipsia.

Her last delivery was at age of 33 year, which was complicated with severe post-partum hemorrhage and in ability to breast feed her son; she did not menstruate since that time. On examination she was conscious oriented, she had dry skin, no skin hyperpigmentation, Bp 110/70mmHg no postural drop, Pulse 80 b/min, no thyroid enlargement. Chest, heart, abdomen were normal. Her Investigations showed: Glu 38.5 mg/dl, Na 134mEq/L, K 3.5mEq/L. Cr 1.47mEq/L, Urea 26.3mEq/L, WBC 4600/µL, Hb 9.5 G/dl, MCV 81fL. MCH 26.1pg Plt.189000/µL.

Random serum cortisol 0.10 ug/ml, ACTH <0.5pg/ml, Ft4 0.3 ng/dl, TSH 0.47 uIU/ml, Estradiol 10.0 pg/ml, LH. 0.8mIU/ml, FSH 5.13 mIU/ml, Prolactin 4.8 pg/ml.

MRI pituitary showed partially empty sell .She was diagnosed as case of delayed presentation of Sheehan's syndrome with pan hypopitutresim. She was given prednisone 5mg po am and 2.5mg po pm. She improved clinically and no further hypoglycemic attacks,

Figure 1. MRI of pituitary fossa without contrast .Coronal (*left*) and sagittal (*right*) T1-weighted MR images showed partially empty sella



two weeks later, she was started gradually on thyroxin 50 mcg replacement for secondary Hypothyroidism.

DISCUSSION

Sheehan syndrome (SS) or post-partum pituitary necrosis is an adeno-pituitary insufficiency result from severe hypotension secondary to excessive blood losses during or just after the delivery. It was first described by Sheehan in 1937.

Women with Sheehan syndrome have varying degrees of hypopituitarism, ranging from panhypopituitarism to only selective pituitary deficiencies. The anterior pituitary is more susceptible to damage than the posterior pituitary [3].

The diagnosis can be made reliably in the presence of lactational failure, prolonged amenorrhea and hypoglycemic crises [4, 5]. It is hard for pregnancy in Sheehan's syndrome. However, only a small proportion of patients with Sheehan's syndrome may have spontaneous pregnancy [6]. However, other signs of adenohypophysal insufficiency are often delayed and subtle leading to the diagnosis being missed. Hyponatremia, anemia, and low bone mass were frequently seen in patients with SS [7].

Our patient was presented with hypoglycemic crisis resulted from secondary adrenal insufficiency (low serum cortisol, low ACTH level) due to Sheehan syndrome, she had history of severe post-partum hemorrhage and in ability to breast feed her son, resulted in prolonged history of secondary amenorrhea, secondary hypothyroidism (low ft4, normal TSH), secondary hypogonadism (low FSH, low LH, low estradiol), and She had mild renal impairment .she had mild hyponatremia and normocytic normochromic anemia which often seen in patient of Sheehan syndrome.

The mean duration between postpartum bleeding and the subsequent development of symptoms varies from 1to 33 years, [8] in our patient was 32 years. The findings on MR imaging observed in Sheehan syndromeare not specific. During the acute phase there is more uniform contrast enhancement of sellar or suprasellar mass, pituitary atrophy resulting in an empty sellaobserved in the late-stage of Sheehan syndrome [9]. Pituitary MR in our patient showed partially empty sella.

The treatment of Sheehan's syndrome is replacement of the deficient hormones. ACTH and TSH deficiencies should be replaced with glucocorticoids and thyroxin respectively, mineralocorticoid replacement is usually not required, Cortisol replacement should precedethyroxin to avoid Addisonian crisis [10]. Our patient was given prednisone 5mg po am and 2.5mg po pm. She improved clinically and no further hypoglycemic attacks, two weeks later she was started gradually on thyroxin 50 mcg replacement for secondary Hypothyroidism.

Sex hormone replacement is important in premenopausal patients and GH replacement has shown improved lipid profile and quality of life in these patients. In conclusion, Sheehan syndrome is more common in especially in developing countries like our country. Considering the duration of disease, one may conclude that diagnosis and treatment are delayed. This may be originating from natural course of the disease. However, the fact that the physicians don't know SS well or don't give enough attention and time to the examination of the patients seems an important factor. Thus, we consider that SS should be emphasized in training of the physicians.

REFERENCES

1. Knobel B, Ben-Yosef S, Rosman P. (1984). Sheehan's syndrome and empty sella turcica. *Isr J Med Sci*, 20(3), 232-5.
2. Paudyal BP. (2005). Delayed presentation of Sheehan's syndrome: a case report. *Kathmandu University Medical Journal*, 3(2), 175-177.

3. Pokharel S, Shrestha A, Jha SC, Maskey D, Shrestha B, Poudel P et al. (2013). A case report on Sheehan`s' syndrome. *Journal of Chitwan Medical College*, 3(4), 49-50.
4. Errarhay S, Kamaoui I, Bouchikhi C, Châara H, Bouguern H, Tizniti S et al. (2009). Sheehan's Syndrome; A case report and literature review. *Libyan J Med*, 4(2), 81–82.
5. MGüven, F Bayram, K Güven, F Kelestimur. (2000). Evaluation of patients admitted with hypoglycaemia to a teaching hospital in Central Anatolia. *Postgrad Med J*, 76, 150–152.
6. Hao J, Liu M, Mo Z. (2012). The symptoms get worse after pregnancy in Sheehan's Syndrome: A case report. *Case Reports in Medicine*, 4.
7. Sunil E, Rajita D, Rajagopal G, Satish P, Suresh V, Laksmi P, Set al. (2013). Sheehan's syndrome: a single centre experience. *J Clin Sci Res*, 2, 16-21.
8. Sanyal D, Raychaudhuri M, (2012). Varied presentations of Sheehan's syndrome at diagnosis: A review of 18 patients. *Indian J Endocrinol Metab*, 16(2), S300–S301.
9. Kaplun J, Fratila C, Ferenczi A, Yang WC, Lantos G, Fleckman AM, et al. (2008). Sequential Pituitary MR Imaging in Sheehan Syndrome: Report of 2 Cases. *AJNR*, 29, 941-943.
10. Bhasin A, Singal RK. (2007). Panhypopituitarism. *JIACM*, 8(3), 256-9.