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Hypoglycemia a rare manifestation of Sheehan syndrome

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Abstract

We present a 28 years FA, Para 4 who presented with recurrent episodes of syncopal attack for one month which used to improve after taking meals orally.

Past history was suggestive of post-partum haemorrhage due to retained placenta. Symptomatic hypo glycaemia was documented on several occasions during hospital stay. Laboratory findings revealed adrenal, thyroid and gonad otrophin hormone deficiency and MRI brain showed empty sella. Hypo glycemia is a rare presentation of Sheehan syndrome and high index of suspicion is required to diagnose this syndrome.

Keywords: MRI, Hypo glycemia, Blood.

Introduction

Sheehan's syndrome is a rare but still a common cause of hypopituitarism in the developing countries. The clinical presentation is variable depending upon the partial or complete deficiency of various hormones secondary to post pituitary necrosis due to bleeding during or after delivery. Hypo glycaemia as a presenting feature of Sheehan's syndrome is rare. Once the diagnosis is established lifelong replacement of deficient hormone is required.

Case

We present a 28 years FA who presented with generalised weakness for last 4 years and recurrent

episodes of syncopal attack for last one month. With these complaints patients had visited many health institutions before coming to us. She had been put on anti-depressants many times by physicians.

On inquiring further, she revealed she has had last child birth 4 years back which was a home conducted delivery complicated by postpartum haemorrhage due to retained placenta. The placenta was removed in hospital and she received blood transfusions. She could not breast feed her baby due to failure of lactation and she gave history of amenorrhea since last childbirth.

On examination patient was apathetic, pulse rate 70/min., BP 80/60 mmhg, loss of axillary and pubic hair and atrophy of breast was present. Systemic examination revealed delayed relaxation of ankle reflexes. Investi gations revealed RBS-18 mg, 53 mg, 45 mg, 57 mg and 65 mg on 1st, 2nd, 3rd, 4th and 5th day of admission, Na-126 and 128 meq and K-4.1and 4.7 on day 1 and 2. The base line serum cortisol of patient was 1.2 micro gram (at 7-9 am). Keeping the possibility of adrenal insufficiency in view of hypo tension, hypo glycaemia and hypo natremia, hydro cortisone was started.

After administering hydro cortisone, blood sugar level, blood pressure and serum sodium improved. The hormone levels revealed, FT4 -0.32 mcg/ dl (0.89- 1.76 mcg/dl), LH-5.0 mIU (15-54 mIU/ml), FSH-0.88 mIU/ml (23-116.3). ACTH-10.90 pg./ml (10-46 pg./ml) and serum prolactin- 0.83 ng/ml (1.82-20). Ultra sonography of abdomen and pelvic organs was normal and MRI brain revealed empty sella as shown in figure 1.

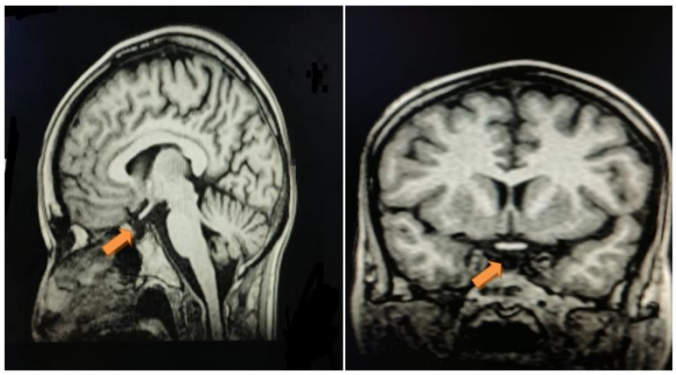

Figure 1: Left image*:* Saggital section suggestive of empty sella (orange arrow) showing absence of pituitary in sella turcica; Right image: coronal section of the same patient.

A dynamic pituitary function test like insulin tolerance test is helpful to assess the pituitary reserve of GH and ACTH. We did not carry this test in our patient as she already had low blood sugar level and low blood pressure and hydrocortisone was already started. Based on clinical, laboratory and imaging findings, diagnosis of presenting case is Sheehan's syndrome post-pituitary necrosis due to postpartum haemorrhage (retained placenta). The patient was started on thyroxin, oestrogen and progesterone in addition to hydrocortisone.

Discussion

Sheehan's syndrome or post-partum pituitary necrosis is a hypopituitarism state secondary to hypovolemia due to excessive bleeding during or just after delivery. It was first described by Sheehan in 1937[1]. During pregnancy, the pituitary gland enlarges two-fold due to hyperplasia of prolactin secreting cells as a result of elevated oestrogen. Enlarged pituitary gland may compress the blood vessels supporting it or there may be a pre disposition in pregnant women compared with non-pregnant women[2]. Partial or total hypopituitarism develops with necrosis of 70 % to 90% of the gland.[2] Sheehan's syndrome is a significant cause of maternal morbidity and mortality in developing countries

although it is rare in developed countries because of better obstetrical care. The clinical presentation of Sheehan's syndrome is variable. It may manifest either immediately after delivery or after a delay of several years, depending on the amount of pituitary tissue destruction. Clinical presentation may range from long-standing non-specific features such as generalised weakness, fatigue and anaemia to profound abrupt hypopituitarism resulting in coma and death[3]. Our case also presented with nonspecific features like generalised weakness and dizziness for which she had consulted many physicians. The cause of dizziness in presenting case may be due to hypo glycaemia; historically dizziness was episodic and used to improve after eating meal. Hypoglycemia as a presenting feature of Sheehan's syndrome is rare and cause may be due to adrenal insufficiency and growth hormone deficiency as a counter-regulatory mechanism[6]. Our patient had clinical features suggestive of gonadotropins (FSH, LH), ACTH and TSH deficiency.

The first presentation is lack of postpartum milk pro duction presumably because of low circulating prolactin followed by failure of resumption of menstruation as seen in our case. The adrenal and thyroid insufficiency occurs late in the disease process. The interval between post- partum bleeding and Sheehan's syndrome diagnosis is averaged 16.35 +- 4.74 years. It is believed that 32% of women with severe postpartum bleeding will develop hypopitutarism. The reason for delay in diagnosis is that most of the patients do not have symptoms suggesting the diagnosis in early period of disease and Sheehan's syndrome is not well known among physicians is another contributing factor in delay in diagnosis. So most of the patients will consult many physicians and several diagnosis will be made like anaemia, hypo thyroidism, depression. It is similar in presenting case who came from a rural remote area, had a home conducted delivery and developed post-partum haemorrhage due to retained placenta. She could not get treatment of post-partum bleeding well in time due to non-availability of good obstetrical services. The diagnosis of Sheehan's syndrome is based on the features of hormone deficiency, a suggesting obstetrical history, decreased basal hormone levels (FT4, TSH, CORTISOL, FSH, LH, PROLACTIN) and imaging study of brain suggestive of empty sella. A dynamic pituitary test like insulin tolerance test was not done in our case as she was already having low blood sugar level, low blood pressure and hydrocortisone was started after base line cortisol level analysis. Once diagnosis of Sheehan's syndrome is established lifelong replacement of hormone is required to restore normal functioning of the thyroid, adrenal and ovarian axis in premenopusal state.

Conclusion

High index of suspicion, detailed history and physical examination are important in recognizing Sheehan's syndrome. Patient education is very important because treatment requires lifelong hormone replacement.

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