

CASE REPORT

Hypopituitarism following eclampsia without significant post partum hemorrhage

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ABSTRACT

Deficits of anterior pituitary hormones in the peripartum period may occur due to infarction, hemorrhage within the gland or autoimmune destruction of the gland. Ischemic damage is limited to the anterior lobe sparing the posterior lobe usually. Sheehan's syndrome or postpartum pituitary infarction was classically described after severe postpartum hemorrhage (PPH). We report an interesting case of a 37 year old female who insidiously developed panhypopituitarism after eclampsia without significant PPH. [IJEM 2008;12(8): 43-45]

Keywords: Panhypopituitarism, eclampsia, postpartum hemorrhage.

INTRODUCTION

Impaired synthesis of one or more anterior pituitary hormones may result from heritable genetic factors, acquired anatomic insults, inflammation and vascular damage. Hypothalamic lesions can also result in similar type of defect. Hypopituitarism in peripartum period may be due to lymphocytic hypophysitis, postpartum pituitary infarction due to Sheehan's syndrome classically described after severe postpartum hemorrhage and pituitary apoplexy in a preexisting pituitary tumor in association with pregnancy induced hypertension. We, here, report a patient developing deficits of anterior pituitary hormones insidiously with a past history of eclampsia without postpartum hemorrhage.

Case report

A 37 year old lady (P3+0) married for 19 years presented with repeated episodes of vomiting, loss of appetite and generalized weakness for one month preceding admission. There was no history of pain abdomen, irregular food habit, lump in abdomen, passage of frank or altered blood or mucus or worm in stool. Though the patient lost weight in last month but that was not quantified.

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There was no history of headache persisting over days in the past including her peripartum period, visual disturbance, behavioral or higher functional abnormality, sudden loss of consciousness, features of cranial nerve palsy, sensorimotor abnormality or imbalance or sphincteric abnormality.

She denied history of any chronic drug intake.

In 1995, she had eclampsia without PPH. She delivered a dead fetus at that time and for obvious reason did not lactate and nor did she require any drug to suppress the same. She became oligomenorrhoeic after that and didn't conceive again. Currently she is oligo-hypomenorrhoeic. Gradually she lost her axillary and pubic hairs. She also had decreased libido following that event.

The lady is non diabetic, non hypertensive. There was no past history of tuberculosis or contact with a known case of tuberculosis. There was no history of trauma to head, joint pain, dysphagia.

On examination, the patient had mild pallor. There was no increased muco-cutaneous pigmentation. Her BMI (body mass index) was 21.2kg/m². Thyroid gland was not palpable. There was scanty axillary and no pubic hair. Pulse was 84/min, regular in rhythm, normal volume. Blood pressure was 104/64 mm Hg in supine and 100/62 mm Hg in standing. She had no clinical signs suggestive of raised intracranial tension or cranial nerve abnormality. Other systemic examination was also noncontributory.

After admission her hemoglobin was 10.3g%. Red blood

cell indices revealed packed cell volume of 30.5%, mean corpuscular volume-79 fl, mean corpuscular hemoglobin - 26.4 pg, mean corpuscular hemoglobin concentration - 33.4%. Peripheral smear revealed hypochromasia and anisocytosis. Total leucocyte count, differential leucocyte count, platelet count were within normal limits.

Blood biochemistry showed normal renal status, glucose tolerance, lipid profile. Liver function test revealed that serum albumin was 2.9g% and total protein was 5.8 g%. Patient had hyponatremia (Na-123 mEq/L). Serum potassium was 3.8mEq/L, serum calcium was 8mg% and serum phosphate was 3.2mg%.

24 hour urine output was 1.6 L. Routine stool and urine examination along with chest X ray and upper GI endoscopy did not reveal any abnormality. ANF(anti nuclear factor) was negative. Automated perimetry also was normal. Ultrasonography of abdomen suggested chronic calculus cholecystitis.

Her hormonal evaluation revealed that morning (at 8 AM) serum cortisol was 3.70 mcg/dL (3.7-19.4mcg/dL) and plasma ACTH was 12.90 pg/mL (upto 46 pg/mL). Serum cortisol one hour after ACTH stimulation was 5.30mcg/dL. Serum T3 was 0.48 ng/mL (0.8-2.0), T4 was 3.44ug/dL (5.1-14.1) and TSH was 2.29uIU/mL (0.27-4.2). Serum FSH was 5.64mIU/mL (3.35-21.63) and serum LH was 2.06mIU/mL (2.39-6.6); both of them were done on 3rd day of menstruation. Serum Prolactin was 3.80ng/mL (1.2-29.9). GH dynamic study (by insulin induced hypoglycemia) could not be done as the patient had history of seizure & basal serum cortisol was very low. Patient had BMD (bone mineral density) T score (by DEXA-dual energy X ray absorpsiometry) of -1.7 at lumbar spine and -1.9 at both hip joints.

MRI of pituitary showed partially empty sella with thinned out pituitary parenchyma annealed to the sellar wall. Posterior pituitary showed normal fatty signal. Infundibulum and optic chiasma were normal as were the parasellar areas. The impression of the radiologist was that of partially empty sella(Fig. 1).

Final diagnosis: Panhypopituitarism in patient with past history of eclampsia without PPH.

Treatment: The patient is on daily replacement dose of hydrocortisone 20mg in morning and 10 mg in evening, levothyroxine 75 mg (microgram) with 1000mg of elemental calcium.

DISCUSSION

Deficits of anterior pituitary hormones in the peripartum period may occur due to infarction, hemorrhage within the gland or autoimmune destruction of the gland.

Panhypopituitarism developing in and around pregnancy may present acutely as unconsciousness or may present insidiously with features due to lack of anterior pituitary hormones. Trophic hormonal failure associated

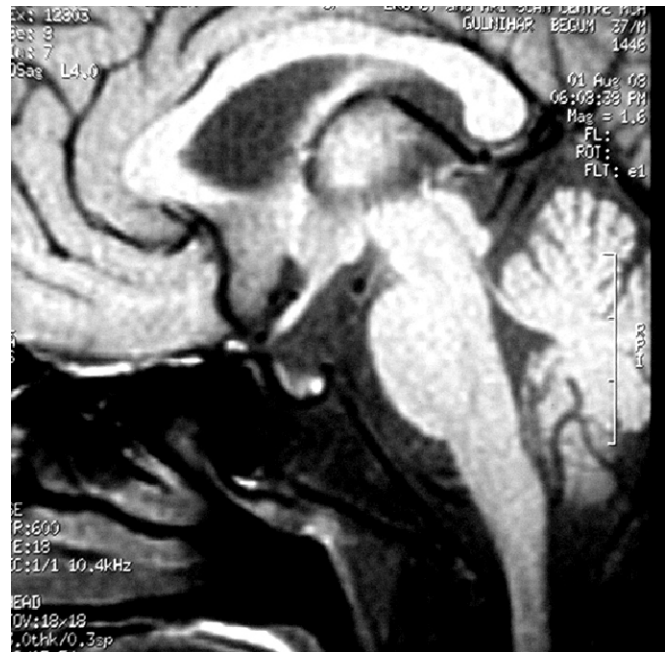


Figure 1: MRI of the pituitary showing partially empty sella.

with pituitary destruction occurs usually in the following order of GH followed sequentially by FSH, LH, TSH, and ACTH. In adults hypogonadism is the earliest symptom.

During pregnancy, the pituitary gland enlarges in response to estrogen stimulation. The hypervascular gland is vulnerable to blood pressure changes. Development of hypovolemic shock following postpartum hemorrhage results in adenohipophyseal vessel vasospasm and pituitary necrosis termed as Sheehan's syndrome. This syndrome occurs in approximately in 1-2% of women with significant PPH (Defined as 1-2 L of blood loss with hypovolemic shock) 1.Eclampsia is a risk factor for PPH.The extent of necrosis determines degree of severity and its presentation either acute shock like presentation or insidious onset characterized by lactational failure, secondary amenorrhoea and breast involution(2). Most patients of Sheehan's syndrome have a mild disease and go undiagnosed and untreated for a long time. It may result in partial or panhypopituitarism and GH is one of the hormones lost earliest. The great majority of the patients have empty sella on CT or MRI(2).

The blood pressure changes during eclampsia can cause damage to various organs as evidenced by infarction of those organs viz. placenta, liver, kidney, brain, heart. Vasospasm of arteries supplying the organs is thought to be a contributory factor of placental infarction. Both eclampsia and pre eclampsia have been found to be associated with increased risk of acute myocardial infarction(3). Cerebral vasospasm has been reported as the cause of ischemic cerebral symptoms in patients of eclampsia or pre eclampsia(4, 5).

Lymphocytic hypophysitis is a close differential diagnosis of Sheehan's syndrome when hypopituitarism

develops in and around pregnancy. Lymphocytic hypophysitis is a rare inflammatory lesion of the pituitary gland, commonly affecting young women during late pregnancy or in the postpartum period. This disorder is believed to have an autoimmune pathogenesis, with increased association with other autoimmune disorders, mainly Hashimoto's thyroiditis and Addison's disease. The clinical manifestations are mainly secondary to mass effects such as headaches and visual field abnormality or partial and total hypopituitarism(6). The corticotropin axis is the most frequently affected axis. Hyperprolactinaemia is also a common feature of this disorder and may represent endocrine marker of this disease(6). Though the natural history is unclear, but it is thought that, if left untreated gland may eventually be destroyed and on imaging partial or complete empty sella may be found in the late stage.

Our patient had insidious onset hypopituitarism as evidenced by history and investigations, following eclampsia without significant PPH. Therefore it is a rare presentation of Sheehan's syndrome. Sheehan's syndrome or panhypopituitarism developing after eclampsia without PPH has been reported rarely(8,9). She did not have visual field defect or hyperprolactinaemia suggestive of lymphocytic hypophysitis. On the contrary her prolactin level was in the lower side of normal range raising the possibility of infarction as this is the hormone deficit which appears first following pituitary infarction as compared to early growth hormone deficit in case of pituitary mass lesion. Considering the role of

vasospasm in infarction of various organs in eclampsia, probably our patient developed pituitary infarction due to vasospasm of feeding vessels of pituitary gland during her episode of eclampsia.

The idea behind presenting the case is that one should look beyond the commoner etiologies of insidiously developing panhypopituitarism developing following pregnancy.

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