University Journal of Medicine and Medical Specialities

ISSN 2455- 2852 2018, Vol. 4(1)

UNUSUAL CAUSE OF HYPOTHYROIDISM - A CASE REPORT

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Abstract :

Hypothyroidism is the most prevalent endocrine disorder after diabetes mellitus. Most common cause of hypothyroidism is the primary pathology in thyroid gland. One among the rare causes of the secondary hypothyroidism is the empty sella syndrome. Empty sella refers to the radiological appearance of an enlarged sella turcica that is partially or completely filled with cerebrospinal fluid.(2) The purpose of this paper is to report an unusual cause of hypothyroidism and to review the clinical and radiological features of the empty sella syndrome and to discuss the pathogenetic mechanisms involved.

Keyword : Hypothyroidism, Empty sella

CASE REPORT:

A 38 year old female (figure1) was admitted with a history of excessive lethargy, feeling of excessive sleepiness, cold intolerance, weight gain and decreased appetite for the past one year duration with the symptoms being intensified for the last three months before presentation. Patient has two female children delivered by natural labour, both are hale and healthy. Patient gave a history of post partum haemorrhage during her last delivery which occurred eighteen years back and was transfused six units of whole blood for the same. She also reported decreased lactation for her second child and has been amennorheic since her last delivery. There was no past history of any surgeries or drug intake for any comorbid illnesses.

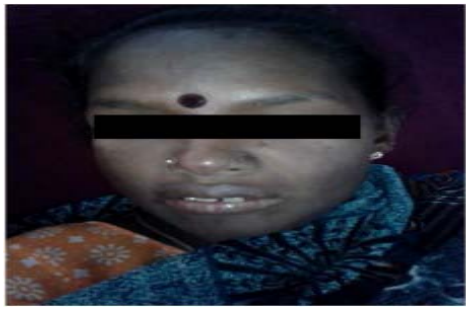

Fig.1.patient's photograph on admission

On examination patient was conscious, oriented, had coarse facial features, slow laboured speech, dry puffy skin, madarosis, sparse dry hair, macroglossia with pallor and non pitting pedal edema. BP was 100/60 mmHg, PR-56/mt, RR-14/ mt,temperature of 36.8 degrees. Cardiovascular, respiratory and abdominal examination were normal. Central nervous system examination revealed sluggish deep tendon reflexes with delayed relaxation (pseudo myotonic reflexes) with normal visual field, normal acuity and normal fundus. Breast examination was normal with no nipple discharge. Sparse axillary and pubic hairs were present.

So by this clinical presentation, we diagnosed as HYPOTHYROIDISM. Investigations: Hb : 8.6 g%, TC: 4600/ mm3, DC: N65 L30 E5(%), platelets: 2.8 lakhs/mm3, ESR 30/60 mm. Renal Function Tests were within normal limits, BLOOD SUGAR: 68mg/dl.

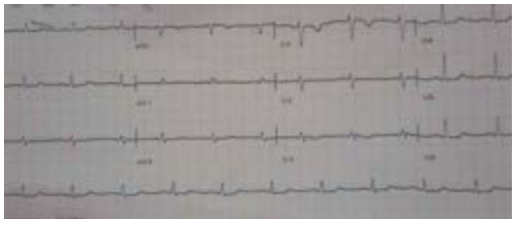

Fig.2 ECG of the patient showing low voltage complex

ECG (figure 2) showed low voltage complex, X ray chest : increased CT ratio, USG neck was normal. ECHO revealed minimal pericardial effusion.

THYROID FUNCTION TESTS: TSH: 0.55 µIU/ml (normal 0.30 – 5.5 µIU/ml) free T3: 16ng/dl (normal 60 – 200ng/dl), freeT4: O.3 µg/dl (normal 4.5 – 12 µg/dl) suggestive of SECONDARY HYPOTHYROIDISM. We suspected secondary hypothyroidism probably due to post partum pituitary necrosis or Sheehans syndrome from the patient's history and the TSH level. On reviewing the literature, it was found out that thyroxine supplementation increases the rate of cortisol metabolism and can lead to adrenal crisis and therefore corticosteroids should be supplemented first, before thyroid hormone replacement therapy. So, we proceeded on to check for the cortisol levels. The 8 am serum cortisol was 0.95µg/dl (normal 6.2 – 19.4 µg/dl).

Because of the relative adrenal insufficiency, hydrocortisone 50 mg iv qid was started. LThyroxine of dose 100 µg/OD was added per orally.Patient's symptoms did not improve much and worsened by the third day of admission. Patient went in for coma with BP 70/40 mmHg with associated hypoglycaemia ,hyponatremia (125 meq/l), hypothermia(34 degree celcius) and subsequently was diagnosed as MYXOEDEMA COMA.

We had planned for MRI to find out the central cause of pan hypopituitarism. But patient could not be mobilized as the patient was in a critical state and hence we proceeded to manage myxoedema coma.

She was treated with intravenous fluids, saline and dextrose,and measures to combat hypothermia. Intravenous Levothyroxine was started with 600µg as a loading dose followed by 100µg/day was given along with the steroids. Patient's condition got improved four days later and metabolic parameters got normalised. Eltroxine150µg OD per orally was started by fifth day and steroid dose was tapered and kept with a maintenance dose of hydrocortisone 20 mg/day intravenously.

We also checked for the other hormone levels- Growth hormone level 0.7 ng/ml (normal 1-10 ng.ml), Prolactin 31ng/ml (normal 3-27ng/ml), ACTH 1.3 pg/ml (normal 5-60pgm/ml) FSH 0.77 mIU/ml (normal 1.5-12.4mIU/ml) LH 0.84 mIU/ml(normal 1.7-8.6mIU/ml). Levels of oxytocin and vasopressin were normal. We proceeded to do a MRI (fig 3,4) which revealed the empty sella with compressed pituitary gland thus confirming the diagnosis of post partum pituitary necrosis (sheehan's syndrome).Further course in the hospital was uneventful. During discharge patient was put on eltroxine – 100 µg OD and prednisolone 5 mg – 2.5 mg (2/3rd dose in morning , 1/3rd in the evening).

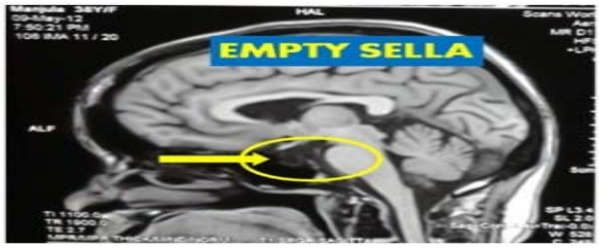

fig 3: MRI brain T1 weighted image shows the empty sella or the empty pituitary.

Patient was reviewed six weeks later with thyroid function tests free T3 85ng/dl (60 – 200 ng/dl), free T4 5 µg /dl (4.5 – 12 µg/dl), TSH 0.5 µIU/ml (0.30 – 5.5 µIU/ml) and cortisol levels 7µ g/dl (6.2 – 19.4 µg/dl). Eltroxine and steroid doses were continued. We adviced the patient to increase the dose of prednisolone to 15mgs in the morning and 5 mg in the evening during periods of acute illnesses. Since the patient does not have features of hyperprolactinemia or growth hormone deficiency, patient was advised to follow up with repeat hormonal assay after 6 months. Considering the age of the patient, hormone replacement therapy with estrogen and progesterone was started mainly to prevent premature osteoporosis.

fig 5: Patient's photograph after 12 weeks of treatment.

DISCUSSION:

The term empty sella (also known as an empty pituitary fossa) was coined in 1951 by Busch as a result of autopsy study of 40 cadavers. Empty sella is present in 5% of normal subjects on autopsy studies. (4) However, the term empty sella is in fact a misnomer as, the sella is not completely empty but, the pituitary is always present both anatomically and functionally, though often it is compressed and displaced downwards which results from a combination of incomplete diaphragm sellae and an increased CSF fluid pressure. (3) An empty sella thus is usually an incidental anatomic finding and occasionally it results in abnormal pituitary function. It can occur at any age and in either sex. However, it is more common in women and increases in frequency with age. (5)

An empty sella syndrome (ESS) may be classified as primary when it occurs in patients who had not received pituitary irradiation or pituitary surgery while, an empty sella discovered following such procedures is classified as secondary empty sella.(6) Recently, shrinkage of the pituitary gland by antipituitary antibodies was advocated as another possible cause of primary ESS. (10)

Most persons with primary empty sella are asymptomatic and the detection of this abnormality may be incidental. Typically primary empty sella syndrome occurs in obese,
multiparous women. These patients sometimes may have headache and hypertension. Endocrine abnormalities are not a common occurrence. Hyperprolactinaemia occurs occasionally, possibly due to stalk stretching. (2) Children with an empty sella most commonly have GH deficiency, although other pituitary hormone dysfunction may occur. Spontaneous CSF rhinorrhoea and pseudotumour cerebri are two syndromes occasionally associated with an empty sella.

Secondary empty sella turcica is one which is associated with an iatrogenic event such as surgery, irradiation, or both; or with non-iatrogenic disease such as infarction and infection ofthe pituitary gland. The predominant clinical finding in these patients are visual abnormality, in the form of diminished visual acuity, peripheral field constriction, bitemporal hemianopia, or quadrantanopia occurring due to the herniation and traction of optic apparatus in to the empty sella.(6) Individuals with secondary ESS due to destruction of the pituitary gland have symptoms that reflect the loss of pituitary functions, such as the ceasing of menstural periods , loss of libido, impotence, fatigue, and intolerance to stress and infection.

Lateral skull x-ray appearances are indistinguishable from those of patients with a pituitary mass (e.g pitutary macroadenoma)with the fossa is variably enlarged. Computerised tomography scans will show the pituitary fossa to be occupied largely by substance of CSF or water density rather than a normal gland. (2)

MRI is the modality of choice for confirming the diagnosis of an empty sella. On T1 sagittal MR images, extension of CSF into the sella is easily identified and remaining gland is compressed along the floor. Typical central position of the infundibulum is a useful sign in an empty sella which helps to rule out a cystic lesion (eg., Rathke's cleft cyst, craniopharyngioma, cystic pituitary macroadenoma) in the suprasellar region which are the main differential diagnosis of empty sella. (9)

For primary empty sella syndrome, there is no treatment necessary if pituitary function is normal. Medications such as bromocriptine, which lower prolactin levels, may be prescribed if prolactin levels are high and interfering with the function of ovary and testes.

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For secondary empty sella syndrome, treatment involves replacement of the main life saving hormones, thyroxine and cortisol. Replacement of growth hormone is indicated particularly in children with increase in dose needed during puberty. Sex steroids therapy is indicated for women of reproductive age, if fertility is desired and in post menopausal women to prevent premature osteoporosis. In children, treatment involves beginning of replacement of
oestrogen or testosterone at the appropriate time of desired puberty.

Surgical indications for correction of symptomatic ESS remain controversy and rare. Visual disturbances and CSF rhinorrhea are the main indications for surgery.

Complications of primary empty sella syndrome include mild hyperprolactinemia, CSF rhinorrhoea. Complications of secondary empty sella syndrome are related to the effects of diminished level of pituitary hormones and visual field abnormality.

CONCLUSION:

The empty sella is usually an incidental anatomic finding and it rarely results in abnormal pituitary function. The rarity of our case is that the patient presented with features of hypothyroidism without any visual field abmormality. Empty sella syndrome is not a lifethreatening illness, if clinically diagnosed early. The prognosis is excellent and life expectancy can be normal with adequate lifelong hormone replacement. So, any female patient presenting with any endocrine abnormality, with history of lactation failure following post partum haemorrhage must be investigated for empty sella syndrome.

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