World Journal of Research and Review (WJRR)
ISSN:2455-3956, Volume-2, Issue-6, June 2016 Pages 21-23

Case Report: A Rare Case of Central Hyperthyroidism During Pregnancy- Diagnostic And Therapeutic Challenge

Dr. Hassan Abuzaid, Dr. Khalid Farouki, Dr. Akshay Athreya, Dr. Parag Mahajan

***Abstract*— Central hyperthyroidism is a rare cause of hyperthyroidism. It causes autonomous TSH secretion and is refractory to the negative feedback of thyroid hormones.**

Most patients with TSH secreting adenomas are diagnosed around the fifth decade of life. Here we present a case of a TSH secreting adenoma detected during the second trimester of pregnancy. We also present the challenges faced in diagnosing and treating the case because the patient refused neurosurgery till she delivered the baby.

She was managed medically and successfully delivered a healthy baby via normal vaginal delivery. She was referred to neurosurgery after delivery.

***Index Terms*—Central hyperthyroidism, pituitary adenoma, pregnancy**

I. INTRODUCTION

Thyrotropin (TSH) secreting pituitary adenomas are a rare cause of hyperthyroidism. Typically, it presents with signs and symptoms of hyperthyroidism and rarely can be asymptomatic. They account for 1% of all pituitary adenomas. In this situation, TSH secretion is autonomous and refractory to the negative feedback of thyroid hormones (inappropriate TSH secretion) and TSH itself is responsible for the hyper stimulation of the thyroid gland and the consequent hyper secretion of T4 and T3. Therefore, this entity can be appropriately classified as a form of "central hyperthyroidism". The first case of TSH-oma was documented in 1960 by measuring serum TSH levels with a bioassay.[1],[2] The presence of a TSH-oma has been reported at ages ranging from 8 to 84 years. However, most patients are diagnosed around the fifth-sixth decade of life. TSH-omas occur with equal frequency in men and women, in contrast with the female predominance seen in other more common thyroid disorders. Familial cases of TSH-oma have been reported as part of multiple endocrine neoplasia type 1 syndrome (MEN 1) and in familial isolated pituitary adenoma (FIPA) family with AIP mutation.[1]-[4]

These rare tumors make too much thyroid-stimulating hormone (TSH), which then causes the thyroid gland to make too much thyroid hormone. This can cause symptoms of hyperthyroidism such as: • Rapid heartbeat • Tremors • Weight loss • Increased appetite • Feeling warm or hot • Sweating • Trouble falling asleep • Anxiety • Frequent bowel movements • A lump (enlarged thyroid) in the front of the neck.[5]

Early diagnosis and correct treatment of TSH-omas may prevent the occurrence of neurological and endocrinological complications, such as visual defects by compression of the optic chiasm, headache and hypopituitarism, and should improve the rate of cure.[4],[6]

Here, we report a rare case of central hyperthyroidism first detected during pregnancy. The patient was asymptomatic till the 2nd trimester of pregnancy when she started having symptoms suggestive of thyrotoxicosis. T3, T4 and TSH were elevated which raised the suspicion of pituitary adenoma leading to hyperthyroidism.

II. CASE HISTORY

38 year old female primigravida, 20 weeks pregnant, presented to outpatient medicine clinic with complaints of headache, fatugue, non-specific abdominal discomfort, decreased appettite and increased bowel frequency for past 3 weeks.

She had no history of vision abnormalities, vomitting or other signs of increased intracranial tension. She had no background medical illnesses. Family history was non-contributory. On examination, she looked anxious, she was conscious and oriented to time,place and person. General examination revealed no pallor, icterus, cyanosis , clubbing or lymphadenopathy. Her hands were found to be moist and warm , and there were fine tremors. Neck examination revealed diffuse non-tender thyroid swelling on palpation. Visual fields by perimetry and visual acuity were normal, eye exam did not show any signs of Grave's ophthalmopathy. Fundus examination did not show any papilledema. Vital signs on admission showed tachycardia with a pulse rate of 120 , heart beat was regular and blood pressure was 126/80 mm Hg.

Systemic examination revealed tachycardia with a ejection systolic murmur best heard in pulmonary area. Respiratory , Neurological and gastrointestinal system examination was normal.

Initial investigations revealed elevated T3, T4 and TSH levels as shown in Table I.

Dr. Hassan Abuzaid, Consultant, Department of Internal Medicine, Hamad Medical Corporation, Doha, Qatar.

Dr. Khalid Farouki, Specialist, Department of Internal Medicine, Hamad Medical Corporation, Doha, Qatar.

Dr. Akshay Athreya, Resident, Department of Internal Medicine, Hamad Medical Corporation, Doha, Qatar.

Dr. Parag Mahajan, Department of Radiology, Hamad Medical Corporation, Doha, Qatar.

Investigation	Observed value	Normal range
Haemoglobin	11 gm%	12- 16 gm%
T3(Triodothyronine)	10.1	2.60-5.70
T4 (Thyroxine)	42 MIU/L	9-20
Prolactin	1588 MIU/L	109 – 557 MIU/L
Cortisol	127 NMOL/L	55 – 386 NMOL/L
Growth hormone	0.16 mcg/L	0 – 10 mcg/L
TSH	40.20 MIU/L	0.45 – 4.5 MIU/L

The peculiar results which showed elevated thyroid hormones (T3 and T4) in presence of elevated TSH raised suspicion for TSH secreting pituitary adenoma. Interestingly, other pituitary hormones- Growth hormone and prolactin were normal, pointing to an exclusive TSH secreting adenoma. An ultraosund of the thyroid showed a coarse echotexture of the thyroid gland and no definite focal lesion was noted.

An MRI head was done to find the lesion .Sagittal post contrast T1-weighted (Figure1) and coronal pre-contrast T2-weighted (Figure2) images reveal enlarged pituitary fossa (arrows in Image1) with a large pituitary adenoma (macroadenoma) involving the left cavernous sinus and extending into the left paracavernous region and the and suprasellar cistern on left. It encases the intracavernous and supra cavernous parts of the left inetrnal carotid artery (arrow in Figure2).

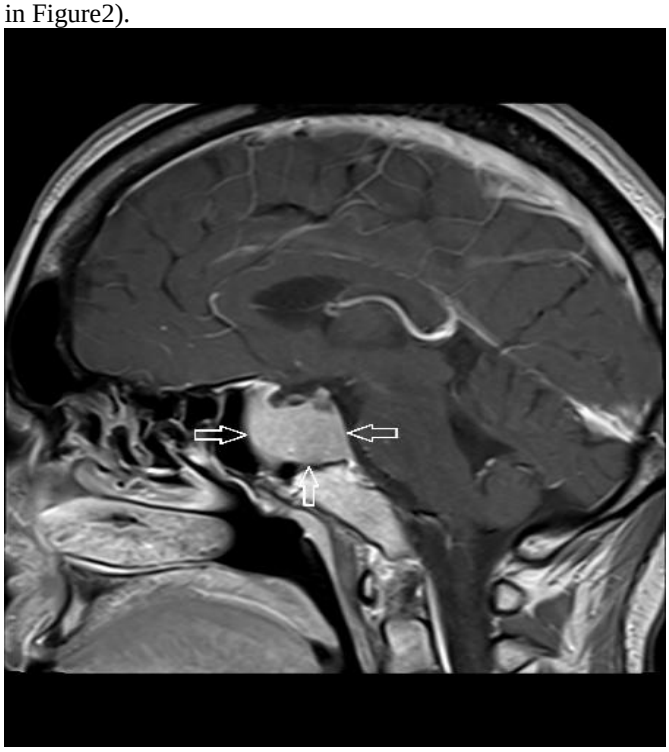

Figure 1

The patient was offered neurosurgical intervention to remove the adenoma but the patient was reluctant to undergo surgery while she was pregnant. She was therefore managed conservatively. Initially, she was started on propylthiouracil

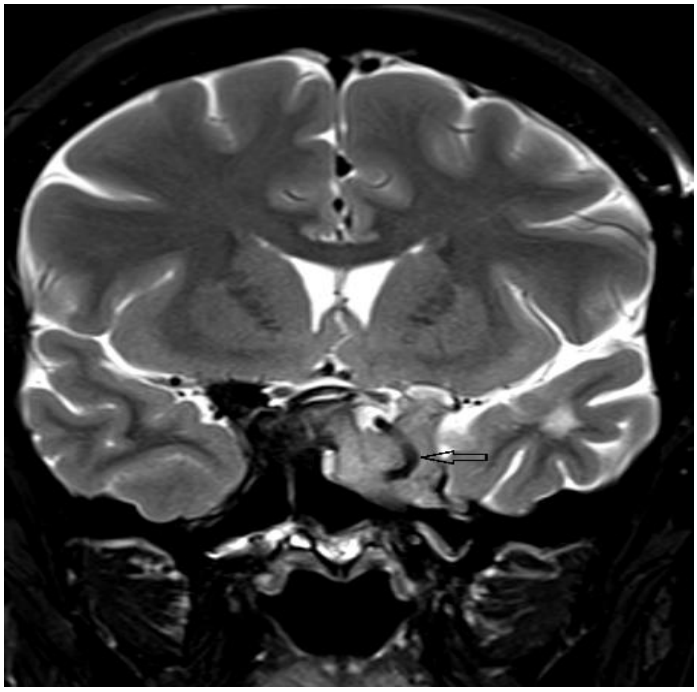

Figure 2

100 mg thrice daily till the end of second trimester and then switched to carbimazole 30 mg/day till delivery.

She had an uncomplicated normal vaginal delivery at term. There were no complications in the post partum period and the baby was healthy. During the follow up period the patient was counselled, and referred for neurosurgery as per previous successful cases. [3],[7],[8].

III. Discussion

Central hyperthyroidism is known to be a cause for infertility as well as many other antepartum, intra-partum and post partum complications like placenta previa, abruptio placenta, pre eclampsia, eclampsia and many other fetal complications [9],[10].

Our case is unique because the patient presented to us in the second trimester and was previously asymptomatic.

Previous studies have shown that hormonal changes during pregnancy may activate a dormant pituitary adenoma and make it functional. [2],[4],[6]. This could be a possibility in this case as the patient had no previous history of hyperthyroidism, was able to conceive naturally and had no symptoms till the second trimester of pregnancy.

After confirming the diagnosis, a multi-disciplinary team comprising OBGYN, Medicine and Neurosurgery service was offered to the patient. The patient refused neurosurgery during her pregnancy and was therefore managed medically during the pregnancy. She was meticulously followed up. Regular follow up visits included testing for mass effects of the mass by routinely checking visual fields by perimetry and checking for signs of raised intracranial tension.

Patient successfully delivered via normal vaginal delivery. The baby was healthy and the patient was referred to the neurosurgery service for resection of the adenoma post –partum [2],[5],[6].

IV. Conclusion

Our case is a rare example of a pituitary adenoma that became symptomatic only during the second trimester and was managed successfully with oral medications, finally resulting in an uncomplicated vaginal delivery.

Acknowledgment

The authors would like to thank Hamad Medical Corporation, Doha, Qatar for their support.The patient gave informed consent to the publication of this article.

References

[1] Ónnestam L, Berinder K, Burman P, et al. National incidence and prevalence of TSH-secreting pituitary adenomas in Sweden. J Clin Endocrinol Metab 2013; 98:626.
[2] Song M, Wang H, Song L, et al. Ectopic TSH-secreting pituitary tumor: a case report and review of prior cases. BMC Cancer 2014; 14:544
[3] Bolz M, Körber S, Schober HC, 2013 TSH secreting adenoma of pituitary gland-rare cause of hyperthyroidism in pregnancy. Dtsch Med Wochenschr 138: 362-366
[4] Beck-Peccoz P, Brucker-Davis F, Persani L, Smallridge RC, Weintraub BD. Thyrotropin-secreting pituitary tumors. Endocr Rev 1996; 17:610-638
[5] Duarte FH, Jallad RS, Salgado LR, Bronstein MD. TSH-secreting pituitary tumors: two case reports and literature review. Arq Bras Endocrinol Metabol. 2009 Dec;53(9):1157-66..
[6]Siristatidis C, Mastorakos G, Vitoratos N, Gregoriou O, Iakovidou H,Sal amalekis E & Creatsas G Thyroid hormone resistance and enlargement of the sella turcica during pregnancy. Archives of Gynecology and Obstetrics 2004 **269**152–155.
[7]Chaiamnuay S, Moster M, Katz MR & Kim YN.Successful management of a pregnant woman with a TSH secreting pituitary adenoma with surgical and medical therapy. Pituitary 2003 **6** 109–113.
[8] Bronstein MD, Salgado LR, de Castro Musolino NR, 2002 Medical management of pituitary adenomas:the special case of management of the pregnant woman. Pituitary 5: 99-107.
[9] Lake MG, Krook LS, Cruz SV. Pituitary adenomas: An overview. Am Fam Physician. 2013;88:319-327.
[10] Molitch ME. Pituitary tumors and pregnancy. Growth Horm IGF Res. 2003;13 Suppl A:S38-44.

Dr Hassan Abuzaid is a Consultant in Internal Medicine at the Al Khor Hospital, Hamad Medical Corporation, Doha, Qatar

Dr. Khalid Farouki is a Specialistin Internal Medicne at the Al Khor hospital, Hamad Medical Corporation, Doha, Qatar.

Dr. Akshay Athreya is pursuing his residency in Internal Medicine from Hamad Medical Corporation, Doha, Qatar. He obtained his MBBS degree from Kasturba Medical College, Manipal, India in the year 2013.

Dr. Parag Mahajan is a specialist at the Al Khor hospital, Hamad Medical Corporation, Doha, Qatar.