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Letter to the Editor

Gestational Transient Thyrotoxicosis Associated with Hyperemesis Gravidarum

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Quick Response Code:

Dear Sir,

A 28-year-old primigravida presented with history of amenorrhea for 2 months; complaints of nausea and vomiting since 1 week - aggravated since 1 day with weight loss of 3 kg. She was diagnosed to have hyperemesis gravidarum. The case was referred to General Medicine in view of altered thyroid profile. She did not have any symptoms of thyroid disorders at present or in the past and was apparently healthy thus far. There were no clınıcal sıgns of hyperthyroidism (no tachycardia, no eye signs, no goiter, etc.). Pulse rate was 86 bpm; Blood pressure was 100/60 mm Hg; she was afebrile and Systemic examination was unremarkable. Complete hemogram, random blood sugar, renal function test, liver function test and Urine routine examination - within normal limits; ketone bodies present in urine. USG was unremarkable; consistent with 2 months of gestation with single intra-uterine pregnancy. Her thyroid profile was consistent with thyrotoxicosis (Free triiodothyronine FT3 - 3.99 (2.3-4.2 pg/ml), Free thyroxine FT4 – 2.77 (0.89-1.76 ng/ml), Serum thyroid stimulating hormone – 0.008 (0.27-4.2 μIU/ml), Anti thyroid peroxide (TPOAb) – 7.80 (0-34 IU/ml) – Negative, TRAb (TSH Receptor Antibody) assay – Negative). Ultrasound of thyroid was normal, and radioisotope scan of thyroid was not considered in view of pregnancy. With a diagnosis of gestational transient thyrotoxicosis associated with hyperemesis gravidarum,[1] intravenous fluids and antiemetics were given and discharged with advice to get a thyroid profile in out patients department after 1 month which normalized.

Goldman *et al* use a broad term "transient nonimmune hyperthyroidism of early pregnancy" since there are many causes such as twin/multiple pregnancies, mutation in the TSH receptor, hyperplacentosis, hyperreactio luteinalis, hydatidiform mole, and choriocarcinoma, though the entity associated with hyperemesis gravidarum is the most common one.[2] Goldman and Mestman call it "transient thyrotoxicosis of hyperemesis gravidarum."[2] Goldman and Mestman define this entity as follows: "*Hyperthyroidism diagnosed for the first time in early pregnancy,transient, without evidence of thyroid autoimmunity, lack of physical findings consistent with Graves' disease, resolving spontaneously by the end of the first or early second trimester of pregnancy.*"[2]

The clinical challenge is to differentiate this from Graves' disease. Investigations usually reveal low or undetectable serum TSH associated with elevated serum FT4 and negative tests for autoimmunity (TPO and TRAb).[2]

With the increase in human chorionic gonadotropin levels during the first trimester of pregnancy, there is a concomitant reciprocal decrease in TSH that continues into the mid-trimester of pregnancy.[2,3] hCG levels being elevated, there is a weak binding of hCG with TSH receptors resulting in this phenomenon.[3] This may be associated with hyperemesis gravidarum.[3] Thyrotoxicosis has been attributed to the thyrotropic action/thyroid-stimulating activity of hCG (with either high levels of hCG or hCG molecule variant with high biological activity).[2] However, serum hCG levels are not used as a diagnostic marker, except in hydatidiform mole and choriocarcinoma.[2] Ultrasound should be done to exclude other etiologies such as multiple pregnancies and trophoblastic disease.[2] Antithyroid drugs are not indicated in this condition; Patients may be treated with only i.v.fluids and symptomatic supportive treatment.[2,3] There is no effect on obstetrical outcome while some studies have indicated lower birth weight of the baby.[2,4]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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