Thyroid Storm Complicating Pregnancy, A Case Report and Management

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The incidence of hyperthyroidism in pregnancy is between 0,05 % and 0,2%. Graves disease is the most common cause of hyperthyroidism in pregnancy. Decrease in Thyroid Stimulating Hormone(TSH) levels and increase in free thyroxine levels are used for diagnosis of thyrotoxicosis. If hyperthyroidism is not treated during pregnancy, these pregnancies are prone to both maternal and fetal complications. Here we present a case of thyrotoxicosis with some maternal and fetal complications.

37 year old woman ,G4P2A1 with 28 - 29 weeks of pregnancy admitted to our clinic with complaints of back pain, painful uterine contractions and low pelvic pressure.

Her blood pressure was 200/120mmHg, pulse rate was 120/min. She had tremor and exolphtalmic eyes, diffuse edema on legs. On suspect of hyperthyroidism she was told to uncover her neck. A diffuse goiter on neck was examined. Her thyroid hormones were sent for control. Thyroid hormone levels were TSH:0,009 uIU/ml, Free T3 :17,2 pg/ml (2-4,4),Free T4:>6 ng/ml (0,9-1,7). On emergency consultation, she had the diagnosis of Basedow Graves complicated with thyroid storm and undertaken to emergency treatment. Her blood glucose levels were checked four times in a day regularly and she had fasting blood glucose levels >120mg/dl and postbrandial blood glucose levels >200mg/dl and with the diagnosis of gestational diabetes, she has been started on ınsuline therapy. After 3 months of treatment, she gave birth to 2700gr, 44cm, Apgar 7-8 fetus.

Postpartum no maternal or fetal complications were seen. Postpartum at the first week the baby had hyperthyroidism symptoms with the placental transport of autoantibodies, therefore breast feeding was stopped and checked for hormone levels regularly. The baby is now under control for the possibility of expected hypothyroidism.

In uncontrolled hyperthyroidism, preeclampsia, premature birth, abruptio placenta, intrauterine growth retardation, fetal hyperthyroidism, stillbirth rates increase. These complication rates fall with the treatment of hyperthyroidism. Not only IUGR, we think gestational diabetes mellitus and macrosomic or LGA(large for gestational age) fetus could be one of the results of hyperthyroidism and high metabolic state in pregnancy. Hyperthyroidism in pregnancy is more frequent in our country and may mimic many pregnancy related conditions and should be differentiated and managed carefully.

Key Words: Hyperthyroidism, Pregnancy, Maternal, Fetal, Complications

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Introduction

Thyroid disease is present in 2-5 percent of all women and 1-2 percent of women in the reproductive age group. Several of thyroid disorders which tend to occur during pregnancy are autoimmune in nature. Graves disease is the most common cause of hyperthyroidism in pregnacy, an organ specific autoimmune process is usually associated with thyroid-stimulating antibodies. These autoantibodies mimic thyrotropin or thyroid-stimulating hormone (TSH) in activity and cause thyroid hyperfunction and growth. Other causes of hyperthyroidism is subacute thyroiditis, toxic noduler goiter and toxic solitary nodule.[1,2,3] Free thyroid hormone levels are used for testing thyrotoxicosis in pregnancy because thyroid binding globuline levels increase with high estrogen in pregnancy and this results in increase of total levels of thyroid hormones. Decrease in TSH levels and increase in free thyroxine levels are used for diagnosis of thyrotoxicosis.[4,5,6] But this should be differentiated from physiologic TSH decrease and free thyroxine and triiodothyronine increase (approximately 20%) in early pregnancy due to high chorionic gonadotropin (HCG) levels.[7,8,9] These levels are maintained within a narrow normal range in second trimester with constant HCG levels and thus there is not overt functional hyperthyroidism. İf hyperthyroidism is not treated during pregnancy , these pregnancies are prone to both maternal and fetal complications. Here we present a case of thyrotoxicosis with some maternal and fetal complications treated successfully.

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Case Report

A 37 year old woman, G4,P2,A1 with 24-25 week of pregnancy according to last menstrual period (LMP) admitted to our clinic with complaints of back pain and painful uterine contractions and low pelvic pressure. On transabdominal ultrasonography, a single, alive fetus, concordant with 28-29 weeks of pregnancy with polyhydramnios was observed. On NST she had regular, high intensity contractions with frequency of 3 in 10 minutes.

She had 2 cm cervical dilatation and 30% effacement on examination.

On physical examination hyperemic, hot face and tremor in hands were noticed, she had difficulty in breathing and answering the questions. She had in crisis state on appearance. Her blood pressure was 200/120mmHg, pulse rate was 120/min. She had exolphtalmic eyes, on suspect of hyperthyroidism she was told to uncover her neck. A diffuse goiter on neck. was examined diffuse goiter on neck and diffuse edema on legs was seen. She reported this swelling of neck had been present from 3rd months of pregnancy and she did not continue her medications. On laboratory investigation, hemoglobine:9,5g/dl, hct: 28%, plt:285000, wbc:11000, blood glucose: 272mg/dl. On urine analysis, (+)proteinuri on dipstick, (++) ketonuria, (++) glucosuria were present. Liver enyzmes, electroytes, billuribine levels were normal . Her ECG documents were ritmically normal except tachycardy. On eye examination, eye lids were retracted, exophtalmic, unable to close when sleeping. Thyroid hormones were TSH:0,009 uIU/ml, Free T3 :17,2 pg/ml (2-4,4),Free T4:>6ng/ml (0,9-1,7). On emergency consultation she had the diagnosis of Basedow Graves complicated with thyroid storm and undertaken to emergency treatment. On emergency treatment, propyltiouracil (Propycil 50mg, Dr.F.Frik) 3x4, alfametyldopa (Methyldopa 250mg, İ.E Ulagay) for hypertension 3x2, propranolol HCL(Dideral 40mg,Sanofi) :3x1/2, were ordered with rapid intravenous hydration. We started magnesium tocolysis for contractions and steroid for pulmonary maturation. After 48 hours of this therapy, her thyroid storm crisis state was corrected. Her blood pressure levels were stabilised to 140/90mmHg, pulse rate 84/min. On 24 hour urine: 840mg proteinuri was detected The baby was controlled with daily CTG's for fetal wellbeing. After one week, propyltiouracil dose was decreased to first 3x4 and then to 3x2 (Propycil 50mg, Dr.F.Frik), propranolol HCL (Dideral 40mg,Sanofi) was stopped. Her blood glucose levels were checked four times in a day regularly and she had fasting blood glucose levels >120mg/dl and after feeding, she had postbrandial blood glucose levels >200mg/dl and with the diagnosis of gestational diabetes, she has been started on ınsuline therapy.(3x4Ü regular, 1x4 Ü NPH but increased to 10U NPH regularly because of resistant high glucose levels). After 7 weeks of hospitalisation for hyperthyroidism, preterm labor and gestatıonal diabetes mellitus, she had spontaneous contractions at 34-35 weeks and she gave birth vaginally to 2700 gr, 44cm female fetus with a 5 minute Apgar score of 8-9. The baby was large for gestational age.

On labor, in complete blood count hb:11g/dl, Hct:35%, Plt:254000, Wbc:8200, liver enzymes, electrolytes were normal, albumin:3.1g/dl(3.4-4.8), on urine analysis: protein was (+) on dipstick. Free T3:9,89pg/ml(2-4,4), FT4:1,85ng/ml (0,9-1,7), TSH:<0,005 uIU/ml (0,5-4,2), Antithyroidperoxidase:73,53IU/ml (0-34), thyroglobulin:457.6ng/ml(1-78). Her blood glucose levels were checked regularly and we used tamponade solution (1000cc Isotonic solution with 16U regular ınsulin) ınfusion on labor

After birth, propyltiouracil dose was decreased to 3x1(Propycil 50mg, Dr.F.Frik) and her blood glucose levels were checked, insulin doses were decreased regularly and after one week, the ınsulın therapy was stopped and called for regular controls in endocrine clinics Postpartum no maternal complication was seen.

The newborn did not have goiter on neck but had high levels of FT3 and FT4 levels due to maternal autoantibodies passed from placenta. On regular controls fortnightly, these levels did not fall and the baby had symptoms of hyperthyroidism, so newborn specialists recommended to stop breastfeeding, to end another source of autoantibodies. After this, the thyroid hormone levels become to normalize, the baby began to gain weight. and now 6 months old baby is under control for the possibility of occurence of hypothyroidism.

Discussion

The prevalence of hyperthyroidism differs according to ethnicities and regional differences. Thyrotoxicosis affects up to 0.2% of pregnant women. If left untreated it is associated with increased fetal mortality and morbidity. In our country ,the incidence of hyperthyroidism was found 4% which is greater than that reported in literature.[10,11]. In hyperthyroidism, fertility does not decrease but abortus rates increase. In uncontrolled hyperthyroidism, preeclampsia, premature birth, abruptio placenta, intrauterine growth retardation, fetal hyperthyroidism, stillbirth rates increase.[10] Mestman reported the incidence of preeclampsia as 22% in thyrotoxicosis.[12] Miller reported the incidence of preeclampsia 33%, growth retardation incidence 12,2%, premature birth incidence 31,6% and added that these complication rates fall with the treatment of hyperthyroidism.[13]

The treatment of choice for thyrotoxicosis during pregnancy is antithyroid medication, either propylthiouracil or methimazole, since radioactive iodine cannot be used.

Propylthiouracil (PTU) remains the drug of choice, since it does not cross the placenta. The initial goal is to control the hyperthyroidism and then use the lowest medication dose possible to maintain the serum thyroid hormone levels in the high normal range. In this way the smaller doses of medications are used, and there seems to be little risk to the baby.[14]

Our patient firstly mimicked severe preeclampsia on attendance with high blood pressures and severe edema but after the history and examination, she had the diagnosis of thyrotoxicosis. On pregnancy course, she had been diagnosed to have mild preeclampsia with blood pressures changing 150/90-140/90mmHg and mild proteinuria. She had premature contractions as expected in thyrotoxicosis and gave response to magnesium tocolysis that is best for this high metabolic state. We did not detect intrauterine growth retardation because she had accompanying gestational diabetes and a large baby for gestation was born. The hyperthyroidic metabolism from the beginning of gestation and crisis state may have triggerred the occult diabetes and made gestational diabetes overt. So we think, not only SGA babies but also LGA babies may be expected in hyperthyroidism

Fetal thyrotoxicosis (hyperthyroidism) occurs occasionally due to transfer of maternal thyroid-stimulating antibodies across the placenta. Most often, the mother herself has hyperthyroidism which is being treated with antithyroid drugs that also passively treat the baby by crossing the placenta.[13,14]

The baby did not have hypothyroidism postpartum as expected due to medications of propyltiouracil in pregnancy but in contrast, hyperthyroidism was seen due to transport of maternal autoantibodies. Clues to the presence of fetal hyperthyroidism are fetal heart rate consistently above the normal limit of 160 beats per minute and the presence of high levels of thyroid stimulating antibodies in the mother's blood. Cessation of breastfeeding is one of the alternative ways of controlling hyperthyroidism in babies having the signs of thyrotoxicosis.

As a result, we think not only IUGR but also with triggering of high metabolic state and with exposure to antiinsuliner hormones longer periods, gestational diabetes mellitus may be provoked and LGA babies could be expected in hyperthyroidic mother babies as in our case.

Hyperthyroidism in pregnancy is more frequent in our country and may mimic many pregnancy related conditions and should be differentiated and managed carefully to prevent maternal and fetal complications.

Gebeliği Komplike Eden Bir Tirotoksikoz Olgusu; Olgu Sunumu ve Yönetimi

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Gebelikte hipertiroidizmin prevalansı %0,05-0,02'dir. Tanıda en değerli testler Tiroid Stimülan Hormon (TSH) düşüşü ve Serbest T4 (fT4) yükselmesinin saptanmasıdır. Ancak daha önce de belirtildiği üzere ilk trimesterde fizyolojik olarak TSH'da %20 oranında düşme görülebilir. Graves hastalığı gebelerde en sık hipertiroidi nedenidir. 37 yaşında SAT'a göre 28-29 gebelik haftasında bir gebe erken doğum sancıları ile başvurdu. USG'de 28-29 hafta tekiz, FKH (+), 1300 gr fetus mevcuttu. Kollum:bir parmak, minimal efasman mevcuttu. Yapılan muayene TA: 200/120, nds:100 ritmik, cilt hiperemik, ellerde tremor, bacaklarda sert (+++) ödem mevcuttu. Hastanın gözleri eksolftalmik, boynunda hiperplazik guatr mevcuttu. Anamnezinde gebeliğin 3. ayından itibaren guatr öyküsü mevcut olan hasta dahiliye konsültasyonu sonucunda tirotoksikoz ve gebelik tanısıyla yatırıldı. Tetkiklerinde hipermetabolik duruma bağlı olarak KŞ:272, idrar miktarı azalmış konsantre idi. FT3:17,2 pg/ ml, fT4: 6 ng/ml, TSH: 0.009 uIU/ml. ,Acilen Propisil 3x4, Alfamet 3x2, Dideral 3x1/2 başlanılıp hidrasyon yapıldı, hastanın tüm bulguları, hipertansiyonu, erken doğum sancıları tiroid krizine bağlandığı için, ağrılar için sadece Magnesium önerildi. Tedavi başlandıktan 48 saat sonra tiroid krizi düzeldi, TA:140/90, nds:84. Esbach: 840mg. Kan şekeri profilinin AKŞ>120mg/dl, tokluk >200mg/dl olması üzerine gestasyonel diabet tanısı ile 3x4 İnsülin önerildi. Hasta 34-35. gebelik haftasında spontan vajinal yoldan 2300gr, kız, Apgar 8-9 fetus, doğurdu. Bebekte de tirotoksikoz bulguları saptanması nedeniyle anne sütü kesilip, tedaviye başlandı.
Hipertiroidide en sık gözlenen maternal komplikasyonlar; abortus, prematür doğum, dekolman plasenta, preeklampsi, konjestif kalp yetmezliği ve hiperemezis gravidarum ve en sık fetal komplikasyonlar olarak; neonatal tirotoksikoz, intrauterin gelişme geriliği, prematürite, ölü doğum ve konjenital anomaliler olarak bildirilmektedir. Bizim olgumuzda da hastada aynı zamanda gestasyonel diabet saptanmıştır, buna bağlı LGA bebek doğmuştur.Biz hipertiroidide sadece SGA bebek değil, hipermetabolik durum ve hipertiroidiye bağlı olarak gestasyonel diabetin indüklenebileceğini ve LGA bebek doğabileceğini düşünmekteyiz. Doğum sonu, bebekte beklenildiği gibi hipotiroidi değil anneden geçen antikorlar nedeniyle neonatal tirotoksikoz saptanmıştır.

Anahtar Kelimeler: Hipertiroidi, Gebelik, Maternal, Fetal, Komplikasyonlar

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