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Case Report

Resolution of pre-eclampsia following correction of hypothyroidism

Sahu T Meenakshi [1], Agarwal Anjoo [2], Das Vinita [3], Pandey Amita [2]

[1] Senior Research Associate, [2] Assistant Professor, [3] Professor & Head

Department of Obstetrics and Gynecology, Queen Mary's Hospital, K.G. Medical University, Lucknow

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Introduction

Hypothyroidism in pregnancy has adverse maternal and fetal effects. In this case, preeclampsia resolved following correction of maternal hypothyroidism.

Case report

A 30-year-old $G_3P_2L_0A_0$ was attending our antenatal out patient department since 14 weeks gestation. Her first trimester was uneventful. Both her previous pregnancies ended in preterm still birth one at 8 months and one at 7 months gestation and both babies were still born.. Her routine antenatal investigations showed Hb 10.5 g/dL screening blood sugar (50 gm glucose) 81 g/dL, blood group B positive and VDRL negative. However in the second trimester screening at 18 weeks, thyroid stimulating hormone (TSH) was raised to 7.94 IU/mL, while thyroid peroxidase (TPO) antibody, free T3 and T4 were normal. She was diagnosed to be a case of subclinical hypothyroidism at 18 weeks gestation and was advised a repeat TSH after 6 weeks. At 24 weeks of gestation she developed pregnancy induced hypertension (PIH) with blood pressure of 160/106 mm Hg in right arm in supine posture and urine albumin 30 mg/dL in a random urine sample. Her repeat TSH was also raised and free T4 was normal. Full blood count, liver and renal function tests, serum electrolytes, and fundus examination were normal. Ultrasonography revealed a single live fetus of 25 weeks gestation in breech presentation, no fetal goiter, placenta posterior in upper segment with adequate liquor, and normal maternal kidneys. She was advised admission which she refused. Tablet methyl alpha dopa 250 mg thrice a day and thyroxine 50 µg daily was started. After 1 week of therapy her blood pressure was still raised and therefore nifedipine 10 mg twice a day was added. In her next visit after 1 week, blood pressure was normal. She had no proteinuria.

At 29 weeks of gestation her alpha dopa was stopped as her blood pressure was 120/70 mm Hg; she was still on thyroxine and nifedipine. The pregnancy was continuing uneventfully and 5 weeks later tablet nifedipine too was stopped as blood pressure was 110/70 mm Hg and urine albumin was absent. Her TSH at 34 weeks was 4.15 IU/mL (normal range 0.5-5.5). Even with no antihypertensives at 34 weeks and beyond, hypertension did not recur.

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Correspondence :
Dr. Das Vinita
B-2, Sector-B, Aliganj
Lucknow, India
Tel. (R) 91-522-2385533, 2330759
Email : das_lko@yahoo.com

An elective cesarean section was done on 22nd November, 2005 at 38 weeks of gestation in view of cephalopelvic disproportion and a live female baby weighing 2800g was delivered. Cord blood TSH and day-3 neonatal TSH were normal. She was discharged on 30th November, 2005 after the stitch removal and advised to continue thyroxine and to come for follow up with post-delivery TSH report. Her TSH at postnatal visit was normal. She was advised to continue taking thyroxine. She conceived again 6 months after delivery and is continuing to take thyroxine.

Discussion

Preeclampsia is a multisystem disorder involving the placenta, liver, kidneys and other systems. The incidence is reported to be 5%, but it is markedly influenced by age, parity, race and genetic and environmental factors. Hypertension and proteinuria are the diagnostic signs. By the time symptoms such as headache, visual disturbances and epigastric pain develop the disorder is almost always severe. Therefore, the importance of antenatal care in the early detection and treatment is obvious [1]. In our case, there were no symptoms suggestive of preeclampsia but during her antenatal visit at 24 weeks we detected a raised blood pressure along with significant proteinuria. So we started antihypertensive therapy initially with methyl alpha dopa. After 1 week, we had to add nifedipine too as her blood pressure was not controlled.

For a research project, we are doing a routine antenatal TSH screening between 13-26 weeks for all consenting women. During such second trimester TSH screening test, she was detected to be suffering from subclinical hypothyroidism as seen by a raised serum TSH but a normal free thyroxine (FT4) without any symptoms. It represents a compensated state in which increased TSH output is required to maintain normal circulating thyroid hormone level. The prevalence of subclinical hypothyroidism among pregnant women is 8%. Various studies have confirmed an association of hypothyroidism, both clinical and subclinical, with adverse maternal and perinatal outcome; preeclampsia is one of them [2,3]. During the 1 year period, 500 women registered for antenatal care and delivery in our unit and 39 developed PIH. Of these 39, seven were clinically hypothyroid and four had subclinical hypothyroidism. In a study by Lao [4], subclinical hypothyroidism during pregnancy is considered harmful and screening and treatment is justified. With this background we gave her thyroxine supplementation and after 4 weeks of combined antihypertension treatment, we could discontinue alpha dopa and after another 5 weeks, at 34 weeks, even nifedipine. Since the blood pressure remained normal without any antihypertensives, probably in this woman the preeclampsia was due to subclinical hypothyroidism and its treatment cured PIH. However, according to the American College of Obstetricians and Gynecologists there is not enough evidence to determine whether screening and treatment for subclinical hypothyroidism in asymptomatic women is justified [5]. According to Spong [6] routine screening of all pregnant women remains unwarranted.

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