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HYPERTHYROIDISM IN PREGNANCY AND LACTATION

Dr. Pooja Agrawal	MD/MS Obstetrics and Gynaecology, SR in OBGY department of GMC, Nagpur, India
Dr. Kanchan Golawar	MD/MS Obstetrics and Gynaecology, Speciality Medical Officer in OBGY department of GMC, Nagpur, India
Dr. Manjushree Waikar	MD/MS Obstetrics and Gynaecology, Professor in OBGY department of GMC, Nagpur, India
Dr. Mansi Shrigiriwar	MD/MS Obstetrics and Gynaecology, Associate Professor in OBGY department of GMC, Nagpur, India

ABSTRACT Hyperthyroidism in pregnancy is generally caused by Grave's disease which can cause thyroid crises, premature labour, abortion and fetal death. Graves disease often becomes more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period. The function of the thyroid gland in pregnancy is increased which is influenced by increased levels of thyroxin binding globulin (TBG), thyroid-stimulating factors (TSF) from the placenta and decreased iodine supply in the thyroid gland. These complaints are more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period. In inadequate treatment, hyperthyroidism complicated to maternal heart failure and fetal death. Hyperthyroidism can cause complications in the mother and fetus and increase morbidity and mortality. Proper initial treatment will improve outcomes.

KEYWORDS :

INTRODUCTION

Hyperthyroidism is hyperfunction of the thyroid gland characterized by a 15- 20% increase in basal metabolism, sometimes accompanied by a mild enlargement of the thyroid gland. About 90% of hyperthyroidism is caused by Grave's disease, both solitary and multiple toxic nodules and toxic adenomas. Grave's disease is generally found at a young age that is between 20 to 40 years and more often found in women than men with a ratio of 5: 1. (1) Hyperthyroidism is estimated at 2: 1000 of all pregnancies, but if it is not controlled it can cause thyroid crisis, premature labour , abortion and fetal death. disease often becomes more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period.(2)

Impaired release of milk, rather than impairment of milk synthesis, appears to be the greater obstacle with both hyper- and hypothyroidism. Without adequate milk removal, increased concentrations of feedback inhibitor of lactation protein in residual milk trigger downward regulation of milk synthesis, resulting in suppressed milk production and eventual involution of the gland. Thus, improving milk release may improve lactation when thyroid hormones are dysfunctional. Exogenous pitocin (i.e., pitocin nasal spray) might provide the oxytocin necessary to eject milk. Massaging the breast from the chest toward the nipple prior to feeding may make more milk available to baby. Breast compression, which mechanically increases internal pressure, may also help to propel milk from the breast during the feed.(3) Galactogogues would be effective only in the presence of a functioning milk ejection reflex and will work best when thyroid hormones are in balance. They should be considered supportive, not first-line therapy, but may be useful adjunct therapy when milk production has suffered. Greater recognition of the impact of thyroid dysfunction on lactation and timely, accurate diagnosis with appropriate treatment of affected mothers will enable mothers to provide sufficient milk to their babies. In time, a greater understanding will also lead to more effective treatments and improved outcomes.

Here we present, a 25-year-old primiravida 37 weeks of gestation,with intrauterine growth restriction came to the emergency department in active stage of labour and delivered vaginally a low birth weight female child of 1.7 Kg. . The patient also complained about excessive sweating and fatigue. On physical examination, the patient's general condition appeared weak with a blood pressure of 110/70 mmHg and pulse 120 beats per minute regular. The exophthalmos and the thyroid gland feel soft in the neck without pain. Laboratory tests found TSHs levels 0.085 μIU / mL with fT4 11.4ug/dl. Ultrasonograph of neck was suggestive of bulky, heterogenous and had increaded vascularity with provisional diagnosis of thyroiditis with cervical lymphadenopathy.The patient had been diagnosed as a case of thyrotoxicosis (Grave's disease) since 2018 on medical treatment, however she had not taken any medications since past two years.

Post delivery baby was admitted to NICU . Mother had complete lactation failure not responding to any galactogogues. In postnatal period endocrinology opinion was taken and patient was started on neomercazole tablet 10mg once a day and propranolol(beta blocker) 40 mg once a day.

During treatment at home, patients are advised to monitor thyroid function to the clinic every once a month.

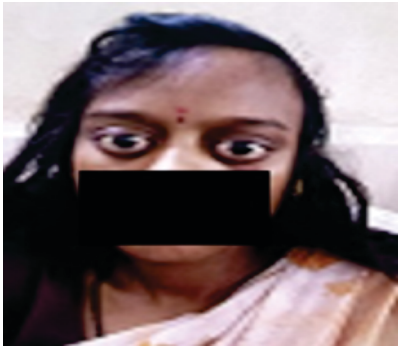

Figure 1: exophthalmos

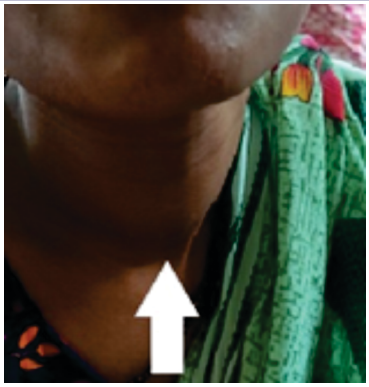

Figure 2: neck swelling

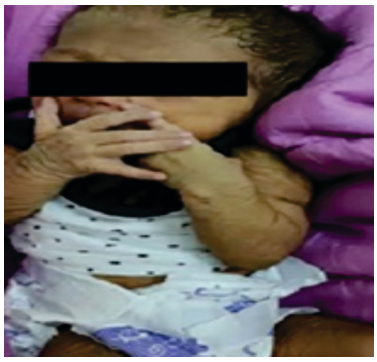

Figure 3: neonate with signs of intrauterine growth restriction

DISCUSSION

According to Glamour, pregnancy is a unique condition, in which the thyroid gland's physiology is affected by 3 changes, 1) a change in thyroid size due to increased levels of thyroxin binding globulin (TBG) in response to increased estrogen levels and increased levels of iodine binding protein starting weeks 12th which reaches 2 times normal levels which will increase T4 and T3 levels in serum; 2) an increase in the secretion of thyroid-stimulating factors (TSF) from the placenta especially human chorionic gonadotropin (HCG); and 3) pregnancy is accompanied by a decrease in iodine supply in the thyroid gland due to an increase in renal clearance of iodine and iodine loss through the photo-placental complex at the end of pregnancy so that it will cause a relative iodine deficiency state.(4)

Clinical signs that can be used as a guide for diagnosis of Graves disease are the presence of tremors, non-infiltrative or infiltrative eye disorders, weight loss without knowing why, local myxedema, myopathy and onycholysis. All of these conditions never occur in a normal pregnancy. If the resting pulse exceeds 100 times per minute and does not slow down with Valsalva manoeuvring, this gives a strong possibility of hyperthyroidism. Pregnant hyperthyroidism patients can experience hyperemesis gravidarum which can only be treated with anti-thyroid medications.(5) At an older gestational age, Grave's disease has a tendency to remission and will experience an exacerbation in the postpartum period.(4) In pregnancy, there will be a decrease in the maternal immune response which is thought to be caused by increased fetal suppressor T cell activity that exerts suppressor factors. These suppressor factors cross the placental barrier thereby suppressing the mother's immune system. After the placenta is released, these suppressor factors will disappear. This can explain why the exacerbation of hyperthyroidism occurs in the postpartum period. After delivery, there is an increase in TSAb levels which peak 3 to 4 months postpartum. This increase can also occur after abortion. A survey conducted by Amino et al. (1979-1980) showed that 5.5% of Japanese women suffer from postpartum thyroiditis. The clinical features of postpartum thyroiditis are often unclear and difficult to detect. Postpartum thyroiditis usually occurs 3-6 months after delivery with clinical manifestations of transient hyperthyroidism followed by hypothyroidism and then spontaneous recovery.(6)

Recent animal studies have shed some light on the impact of hyperthyroidism on lactation. Excessive thyroid hormone seems to accelerate mammary growth. While hypothyroid rats have smaller litters and longer gestations, hyperthyroid rats have larger litters, prolonged labors, and earlier onset of labor and lactogenesis. When severe hyperthyroidism was induced before mating and maintained through pregnancy, Rosato et al.(7) noted good lobulo-alveolar growth and evidence of lactogenesis II, yet complete lactation failure occurred. This suggests a problem with oxytocin release and milk ejection. The authors also observed defects in maternal behavior among many of the mother rats which were likely related to the oxytocin deficit. A study by Varas et al.(8) examined the effects of induced moderate hyperthyroidism in rats. The rats were able to lactate, but impairment of milk ejection led to apoptosis, involution, and litter death over time. Histological studies again showed functional mammary tissue with "distended alveoli" but "almost total absence of adipose tissue." In an acute suckling test at 21 days postpartum, significantly smaller increases in serum prolactin and oxytocin concentration were found in treated rats compared to controls after 30 minutes of suckling. Two recent human cases have involved multigravid women who delivered prematurely secondary to poorly controlled hyperthyroidism. In each case, lactation was severely suppressed with neither woman able to express colostrum. Standard lactation management strategies were tried without success.(9) When lactogenesis stage II seems to have occurred, yet the milk "just isn't coming out," the possibility of hyperthyroidism should be considered, in the absence of other explanations. If diagnosed, the first line of treatment is to lower the level of thyroid hormones. Lao recommends propylthiouracil (PTU) as the drug of choice for breastfeeding mothers because it is excreted in insignificant amounts in breast milk and does not depress neonatal thyroid function. Methimazole is suggested as an acceptable option if the infant can be monitored frequently.(10) Improving lactation outcomes.

REFERENCES:

1. Shahab A. Hipertiroidisme Dalam Kehamilan. Fak Kedokt Univ Sriwijaya, 2012.
2. Garry D. Penyakit Tiroid pada Kehamilan. 500 CDK. 2013;40(7):500-503.
3. Newman,J.BreastCompression.http: //www.drjacknewman. com/index. php?option=com_content&task=view&id=5&Itemid=6#BreastCompression. Accessed December 12, 2005. Page last edited 2 007-10-14 09:34:37 UTC.
4. Kahaly GJ, Bartalena L, Hegedüs L. 2018 European Thyroid Association Guideline for the Management of Eur Thyroid J. 2018;7:167-186. doi:10.1159/000490384
5. Working F, Clinical G, Fetal M. Good clinical practice advice: Thyroid and pregnancy. Int J Gynecol Obs. 2019;144:347-351. doi:10.1002/ijgo.12745
6. Clinic O, Hospital CDA, Muscel C, Hospital M, Arges C De, Hospital M. Maternal and fetal complications of the hypothyroidism-related pregnancy. Mædica. 2010;5(2):116-123.
7. Rosato, R., M. Gimenez, and G. Jahn. Effects of chronic thyroid hormone administration on pregnancy, lactogenesis, and lactation in the rat. Acta Endocrinol (Copenh) 1992; 127(6):547-54.
8. Varas, S., E. Munoz, M. Hapon et al. Hyperthyroidism and production of precocious involution in the mammary glands of lactating rats. Reproduction 2002; 124:691-702.
9. Betzold, C. Personal communication with Lisa Marasco. December 7, 2005.
10. Lao, T. Management of hyperthyroidism and goitre in pregnancy, and postpartum thyroiditis. J Paediatr Obstet Gynaecol 2005; 31(4):155-64.