POPULAR ARTICLE *28th December2022*

Prevalence and Consequences of Hypothyroidism among Maternal Aged Group Women (15-49 years)

Tripti Verma and Alka Gupta

Department of Food Nutrition and Public Health, Ethelind College of Home Science, SHUATS Prayagraj, 211007

Corresponding Author*

Tripti Verma

Email
tripti.verma100@gmail.com

MS No. 21211

FIGURE 1: Pregnancy and motherhood (Image Courtesy: freepik.com)

KEYWORDS: Hypothyroidism, Maternal aged group, Fetal, Diet, Grave's disease, Pregnancy

SUMMARY

Pregnancy-related hypothyroidism is harmful to both the mother and the foetus. Future intellectual development of children born to women who received inadequate care or no care at all is greatly impacted. Women with a low thyroid reserve or an iodine shortage may develop hypothyroidism during pregnancy because this is a demanding time for the thyroid gland. During a typical pregnancy, the levels of thyroid stimulating hormone (TSH), total thyroid hormone, and thyroxine-binding globulin all vary. Different aspects of pregnancy outcome are impacted by thyroid disease. Autoimmune thyroid illness increases the risk of miscarriage. Children born to severely hypothyroid mothers may have permanent neurological deficits. GD (Graves' disease) Pregnancy can be viewed as a state in which a combination of events concurs lead to pregnancy loss as well as fetal thyroid dysfunction. All women who already have thyroid dysfunction are advised to undergo thyroid function tests as soon as the pregnancy is confirmed in addition to following a healthy diet. During pregnancy, the medication needs to be carefully monitored. To find pregnant women who are at risk, one should utilise the trimester-specific reference intervals for thyroid hormones that were developed for the pregnant Indian population after serially observing the pregnant women. Regarding the iodine nutritional status of pregnant women in India, very little information is known. As a result, the current inquiry will be conducted to determine the prevalence and risk factors that are linked to an increased risk of producing hypothyroidism in expectant moms.

INTRODUCTION

Iodine is essential for the production of maternal and fetal thyroid hormones that regulate the development of the fetal brain and nervous system. A woman's iodine requirements increase substantially during pregnancy to ensure adequate supply to the fetus. Globally, although nearly a third of school-age children (246 million) are estimated to have insufficient iodine intake, this commonly-used surrogate measure likely greatly underestimates the number of pregnant and lactating women with inadequate iodine nutrition status. Depending on the timing and severity, insufficient iodine intake increases the risk of negative reproductive outcomes, such as prenatal and infant mortality, and intellectual impairment, the most extreme form of which is cretinism (WHO 2015). Iodine deficiency disease (IDD) is the most common cause of preventable mental deficiency in the world today as iodine plays a critical role in infant brain development and hence this nutrient has immense importance during pregnancy and lactation. Most of the 1,572 million people worldwide, estimated to be at risk of IDD, live in developing countries of Africa, Asia and Latin America; however, large parts of Europe are also vulnerable.IDD was recognized as a public health problem in India after the pioneering work of Prof. V. Rama linga swami and others and led to the formation of National Goitre Control Program (NGCP) in 1962. The implementation of NGCP continued till 1983 with limited success. In 1984, the Govt. of India decided to adopt the programme of Universal Salt Iodization (USI) under which all salt meant for human consumption was to be fortified with iodine. In 1992, the NGCP was renamed as National Iodine Deficiency Disorder Control Programme (NIDDCP). A ban on the sale of no iodized salt was lifted in September and the ban was reemployed on 27th May 2005. The factors responsible for a higher requirement of iodine are:

(1) Increased requirement of thyroxin (T_4) to maintain a normal global metabolism in the mother.

(2) Transfer loss of (T_4) and iodide from the mother to the fetus.

(3) Increased loss of iodide through the kidney due to an increase in the renal clearance of iodide in pregnancy.

The recommended dietary intake of iodine during pregnancy is therefore higher than the value 100 µg/day which is recommended for non-pregnant adults and adolescents. Iodine balance is negative during pregnancy below a daily intake 150 µg/day. The iodine intake of an exclusively breastfed infant is dependent on the iodine intake of the mother during pregnancy and lactation. Pregnant and lactating women and neonates are the main targets of the effects of iodine deficiency because of the impact of maternal, fatal and neonatal hypo thyroxinemia on neonatal brain development. In this study, we have evaluated the prevalence of iodine deficiency among pregnant and lactating mothers, almost three decades after the adoption of USI program by the Indian Government. The recommended iodine intake during pregnancy was increased from 200 to 250 µg/day and median UIE concentration cut off was increased from 100µg/l to 150 µg/l.

International prevalence of hypothyroidism in pregnancy

The estimated prevalence of hypothyroidism in pregnancy is 2-3%. Of these, 0.3-0.5% is OH and 2-2.5% is SCH. Studies have demonstrated 60% risk of fetal loss and 22% risk of gestational hypertension with untreated OH. A firm association between OH and adverse risk to the maternal-fetal unit has been demonstrated. The miscarriage rate in SCH

Academic editor- Dr. Sandeep Singh, PhD, Kanpur, (208021) Uttar Pradesh, India.

is 6% vs. 3.6% in euthyroid women. A two-to threefold increased risk of pregnancy related complications was demonstrated in untreated women with SCH.

American Thyroid Association's (ATA 2011) recommendation is neither for nor against universal screening in the first trimester. It recommends treatment of OH (TSH>trimester specific values with low T_4 or TSH>10 irrespective of T_4) and SCH with positive TPO antibodies. ATA also recommends regular TSH monitoring of euthyroid TPO positive pregnant women (PW) throughout pregnancy. SCH in PW who have not been treated initially should be monitored every 4 weeks with serum TSH and FT_4 approximately, until 16–20 weeks of gestation and at least once between 26 and 32 weeks gestation. The Endocrine Society (2012) does not recommend universal screening of all PW but encourages TSH in "high risk" individuals and low dose thyroxine to target TSH to <2.5mIU/l. It recommends repeating the screening in the second trimester if initial screening is normal.

National prevalence of hypothyroidism in pregnancy

Prevalence of hypothyroidism in pregnancy in the Indian population is 4.8-12%. Intrauterine Growth Restriction (IUGR) prevalence is 25% in OH and 8% in SCH, while the incidence of pre-term delivery is 33% with OH and 11% with SCH. Indian Thyroid Society (ITS) recommends screening of TSH levels in all PW at the time of their first visit, ideally during prepregnancy evaluation or as soon as pregnancy is confirmed, although evidence for this is limited from studies that have already been carried out.

Pregnancy can be viewed as a state in which a combination of events concurs to modify the thyroidal economy. Hypothyroidism during pregnancy has an adverse effect on both mother and child. Children born to untreated or undertreated mothers have profound effect on future intellectual development. Pregnancy is a stressful condition for the thyroid gland resulting in hypothyroidism in women with limited thyroid reserve or iodine deficiency. There is change in the level of thyroxine-binding globulin, total thyroid-hormone level and change in the level of thyroid stimulating hormone (TSH) during normal pregnancy. Thyroid dysfunction has varied impact on pregnancy outcome. The risk of miscarriage is increased in autoimmune thyroid disease. Severe maternal hypothyroidism can result in irreversible neurological deficit in the offspring. Graves' disease (GD) can lead to pregnancy loss as well as fetal thyroid dysfunction.

The most common cause of maternal hypothyroidism is the autoimmune disorder known as Hashimoto's thyroiditis. In this condition, the body mistakenly attacks the cells of the thyroid gland, leaving the thyroid without enough cells and enzymes to make enough thyroid hormone to meet the body's needs. Radioiodine therapy and thyroid surgery, but also iodine, medicines or rare genetic disorders may cause hypothyroidism.

Maternal perspective of hypothyroidism

Hypothyroidism problems can be difficult to diagnose in pregnancy due to higher levels of thyroid hormone in the blood, increased thyroid size, fatigue, and other symptoms common to both pregnancy and thyroid disorders. Women with hypothyroidism have decreased fertility; even if they conceive, the risk of abortion is increased, and risk of gestational hypertension, anaemia, abruptioplacenta and postpartum haemorrhage is increased.

Fetal and neonatal perspective of hypothyroidism

The impact of severe iodine deficiency or congenital hypothyroidism on the foetus and new born is profound, as are the foetus is dependent on maternal trans-placental thyroid hormone supply in the first trimester or may be the effects of overt maternal hypothyroidism on pregnancy. The severity, timing of onset and duration, as well as postnatal management, all influence fetal and neonatal brain development. Because thyroid hormones are crucial to fetal brain and nervous system development, uncontrolled hypothyroidism especially during the first trimester can affect the baby's growth and brain development and It is now believed than even mild maternal hypothyroidism (from mild iodine deficiency, thyroid autoimmunity, or thyroid under-replacement) may affect fetal brain development.

Consequences of untreated hypothyroidism

Untreated hypothyroidism in pregnancy is associated with adverse maternal effects. During pregnancy, it is known to result in miscarriages (in early pregnancy), recurrent pregnancy losses, anaemia, pre-eclampsia, gestational diabetes, abruption placenta, postpartum haemorrhage, increased caesarean sections due to fetal distress, and rarely myopathy and even congestive heart failure (CHF) in severe cases. Hypothyroidism results in preterm births, intrauterine growth restriction, intrauterine fetal demise, respiratory distress and increased prenatal mortality (PNM). In newborns, it leads to cognitive, neurological and developmental impairment. Thyroid hormone is critical for fetal brain development.

CONCLUSION

According to the data revealed by the report, India is facing a serious threat of under-nutrition where more than half of the women of reproductive age suffer from thyroid dysfunction. Having hypothyroidism can make feel exhausted and sluggish, and it can make it difficult to concentrate, among other things. Therefore, it is important to eat the right variety of foods in the correct proportions, a varied and healthy diet. For example, choose low fat, low calorie spread rather than butter or ordinary margarines avoid high salt intake and cut down on hidden fats & sugars (cakes, biscuits, chocolate) with calcium rich foods and / or supplements, and normal vitamin D levels. Along with the diet, it is recommended to all the women who already have known thyroid dysfunction should immediately go for thyroid function tests as soon as the pregnancy is confirmed. Careful monitoring of the medicine should be done during the course of pregnancy. The trimester specific reference intervals for thyroid hormones established for pregnant Indian population after serially following the pregnant women should be used to identify at risk women. Very limited data is available regarding the iodine nutritional status of pregnant women in India. Therefore, the present investigation will be carried out to know the prevalence and associated risk factor that increases the possibilities of causing hypothyroidism in pregnant mothers.

Acknowledgment: Image Courtesy of freepik.com, 2022, Republished under Creative Commons Attribution 3.0 License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

REFERENCES

1. L. M. De-Regil, K. B. Harding, J. P. Peña-Rosas and A. C. Webster (2015). Iodine supplementation for women during the preconception, pregnancy and postpartum period. Protocol. *Cochrane Database of Systematic Reviews*, Issue 6. Art. No.: Cd011761.

2. E. N. Pearce, M. Andersson and M. B. Zimmermann (2013). Global Iodine Nutrition. Thyroid. 23(5), 1-6.

3. E. M. Wong, K. M. Sullivan, C. G. Perrine, L. M. Rogers and J. P. Peña-Rosas (2011). Comparison of median urinary iodine concentration as an indicator of iodine status among pregnant women, school-age children, and non-pregnant women. *Food and Nutrition Bulletin*, S32 (3), 206-212.

4. M. B. Zimmermann (2012). The effects of iodine deficiency in pregnancy and infancy. Paediatric and Perinatal Epidemiology. 26(l1), 108-117.

5. M. B. Zimmermann and M. Andersson (2012). Update on iodine status worldwide. *Curr. Opin. Endocrinol Diabetes*, 19, 382-387.

6. M. B. Zimmermann and M. Andersson (2011). Prevalence of iodine deficiency in Europe in 2010. *Ann Endocrinol*, 72, 164-166.

7. V. Ramalingaswami, T. A. Subramanian and M. G. Deo (1961). The aetiology of Himalayan endemic goitre. *Lancet*, 1,791-794.

8. S. Srinivasan, A. Sinha, T. A. Subramanyan and M. G. Deo and V. Ramalingaswami. Himalayan Endemic Deafmutism. *Lancet*, 2, 176-185.

9. J. Matovinovic and V. Ramalingaswami (1958). Therapy and prophylaxis of endemic goitre. *Bull World Health Organ*, 18, 233-253

10. V. Ramalingaswami (1953). The problem of goitre prevention in India. *Bull World Health Organ,* 9, 275-281.

11. U. Kapil (2007). National iodine deficiency disorder control programme (NIDDCP) in India: Current status and failure strategies. *J Indian Thyroid Soc,* 4, 37-49.

12. U. Kapil (2000). Progress made in elimination of iodine deficiency disorders and possible impact of lifting ban on sale of non-iodised salt. *J Acad. Hosp Admin*, 12, 33-41.

13. R. Sankar and C. S. Pandav (2005). Ban on sale of non-iodized salt for human consumption: A step in the right direction. *Natl Med J India*, 18, 169-171.

14. H. J. Dworkin, J. A. Jacquez and W. H. Beierwaltes (1966). Relationship of iodine ingestion to iodine excretion in pregnancy. *J. Clin Endocrinol Metab*, 26, 1329-1342.

15. F. Delange (2007). Iodine requirements during pregnancy, lactation and the neonatal period and indicators of optimal iodine nutrition. *Public Health Nutr*, 10, 1571-1580.

16. A. M. Leung, E. N. Pearce and L. E. Braverman (2011). Iodine nutrition in pregnancy and lactation. *Endocrinol Metab Clin North Am*, 40, 765-777.

17. M. Qian, D. Wang and Z. Chen (2000). A preliminary meta-analysis of 36 studies on impairment of intelligence development induced by iodine deficiency. *Zhonghua Yu Fang Yi Xue Za Zhi,* 34, 75-77.

18. https://www.freepik.com/premium-vector/modern-illustration-about-pregnancy-motherhood-beautiful-young-woman-with-long-hair-minimal-design-illustration-cartoon-flat-style_11549514.htm

Citation: Verma and Gupta (2022). Prevalence and Consequences of Hypothyroidism Among Maternal Aged Group Women (15-49 years). International Journal of Medicine & Health Research, 8(1), 1-3.