MATERNAL SCREENING OF THYROID FUNCTION

Dr. M.A. Taher

Pregnant women comprise the most vulnerable population group with respect to iodine deficiency, because of its causative link with cretinism, an irreversible defect resulting from severe iodine deficiency in utero. For this reason, the elimination of cretinism is one of the most important aims and monitored indices of success of community iodine supplementation programs.[1]

Iodine deficiency in young children can compromise their mental development. Two studies in Bolivia and Bangladesh did not show any effect, while in two in Malawi and Benin did. It may well be that the iodine deficiency was less severe in the populations in which no effect was found compared with those in which it was.[2-5]

The fetus cannot make T_4 (thyroxine) in the first half of pregnancy when neuron proliferation and migration are taking place in the brain. Pop et al correlated maternal T_4 levels during 12 weeks of pregnancy with impaired infant psychomotor performance at 10 months.[6]

Azizi et al published a study on 14 children who had a transient increase in TSH (thyroid stimulating hormone or thyrotropin) and were completely normal after the 3rd month of life, and then between 4 and 6 years of age they had a much lower I.Q. (intelligence quotient) than normal children.[1]

Transiently elevated TSH (or TSH surge) was found during the first 24th year of life is considered to be entirely physiological. It is at least partly due to the cooling of the neonate immediately after birth and to the cutting of the cord. This is why we have two kinds of normal levels. If the blood sample is collected right after birth or from the cord, the cut off point is about 20 mU/L., however, if you take it as a usual screening test 3 days after birth, the cutoff point will be about 5mU/L.

In conditions of mild iodine deficiency, serum free T_4 slightly decreases during gestation, while in iodine sufficiency there is only a slight (15%) decrease by the end of gestation. In mild iodine deficiency, serum TSH and thyroglobulin are still higher in neonates than in their mothers. The frequency distribution of neonatal TSH on day 5, at the time of systematic screening for congenital hypothyroidism is shifted towards elevated values. In moderate iodine deficiency, the frequency of a neonatal TSH above 20-25, mU/L blood that is above cutoff point used for recalling neonates due to suspicion of congenital hypothyroidism in programs of systematic screening for congenital hypothyroidism, is increased. The hypersensitivity of neonates to iodine deficiency is explained by their very small intrathyroidal iodine pool, which requires increased TSH stimulation and a fast turnover rate in order to maintain a normal secretion of thyroid hormones.[8]

In mild iodine deficiency, preterm infants have low total and free T_4 elevated TSH and exaggerated TSH response to TRH. This picture of primary subclinical hypothyroidism is in contrast with the picture of tertiary hypothyroidism evidenced in preterm infants in iodine replete areas, characterized by the fact that TSH remains normal in spite of low free T_4.

Director, Center for Nuclear Medicine and Ultrasound, Rangpur, Bangladesh.

In humans, T_4 is already found in the first trimester coelomic fluid from the 6th week of gestational age, a long time before the onset of fetal thyroid function, which occurs at the 24th week of gestation. The number of T_3 (triiodothyronine) receptors and the amount of T_3 bound to the receptors in the whole brain increase about 10-fold between 10 and 18 weeks, also before the onset of fetal thyroid function. At term, about 20-50% of cord serum T_4 is still of maternal origin. Maternal screening of thyroid function should be considered seriously as a part of routine antenatal check up.

REFERENCES

1. Allen L, Gillespie S. What works? A review of the efficacy and effectiveness of nutrition interventions. UN ACC/SCN Nutrition policy paper No. 19. New York, UN Administrative Committee on Coordination, 2001.
2. Bautista A, Barker PA, Dunn JT, Sanchez M, Kaiser DL. The effects of oral iodized oil on intelligence, thyroid status and somatic growth in school-age children from an area of endemic goitre. Am J Clin Nutr 1982; 35:127-134.
3. Huda SN, Grantham-mcGregor S, Tomkins A. Cognitive and motor function of iodine-deficient but euthyroid children in Bangladesh do not benefit from iodized poppyseed oil (Lipiodol) J Nutr 2001; 131:72-77.
4. Shrestha RM. Effect of iodine and iron supplementation on physical, psychomotor and mental development in primary school children in Malawi; Ph D thesis Wageningen Agricyltural University, 1994.
5. Van den Briel T, West CE, Bleichrodt N, van der Vijer FJR, Hautvast JGAJ, Ategbo EA. Improved iodine status is associated with improved mental performance of school children in Benin. Am J Clin Nutr 2000; 72: 1179-1185.
6. Pop VJ Kuijpens JL, Van Baar AL, et al. Low maternal free thyroxine concentrations during early pregnancy are associated with impaired psychomotor development in infancy. Clin Endocrimol 1990; 50:149-155.
7. Azizi F, Khoshniat M, Bahrainian M, Hedayati M: Thyroid function and intellectual development of infants nursed by mothers taking methimazole. J Clin Endocrinol Metab 2000; 85: 3233-3238.
8. Delange FM. Iodine deficiency disorders in mothers and infants. In Delange FM, West KP Jr. (eds.) micronutrient Deficiencies in the First Months of life, Nestle Nutrition workshop series pediatric programs Vol. 52. pp. 89-102, Nestec Ltd. Vevey/S. Karger AG, Basel, 2003.