THYROID FUNCTION IN PREGNANCY

RAMALAKSIIMI A. BARU • RAMARAJU A. LAKHAMRAJA • RAMAN LEELA

SUMMARY

The present study was undertaken to evaluate the thyroid function in normal and abnormal pregnancies in relation to maternal nutritional status. The study population consisted of 55 normal pregnant women, 19 women with pregnancy induced hypertension and 28 non pregnant non lactating (NPNL) women from a general hospital and 46 pregnant women from a nursing home. The assessment of thyroid function were done during 1st, 2nd and 3rd trimester of pregnancy by estimating Triodothyronine (T3), thyroxine (T4) and thyroid stimulating hormone (TSH) by radioimmunoassay. The results indicate that during normal pregnancy T3, T4 levels were increased without altering thyroid stimulating hormone levels throughout pregnancy. In contrast high TSH levels were seen in pregnancy induced hypertension with no change in T3, T4 levels. Neither the socioeconomic status nor the body mass index (BMI) showed any relationship with the thyroid function. Thus the thyroid function appears to be same throughout pregnancy irrespective of the differences in the socioeconomic group and the nutritional status.

INTRODUCTION

During pregnancy there is moderate enlargement of the thyroid caused by hyperplasia of the glandular tissue and increased vascularity. The basal metabolic rate (BMR) increases progressively during normal pregnancy as much as by 25%.

Nat. Inst. of Nutri. Ilyderabad. Accepted for Publication in March95 Most of the increase being in oxygen consumption due to high metabolic activity of the products of conception. Thyroid function is closely ralated to BMR and oxygen consumption (Prout 1975). In undernutrition and chronic starvation, Triodothyronine (T3), and Thyroxine (T4) are known to decrease as an adaptive mechanism. T4 increase in normal pregnancy is due to increase in thyroid binding globulin. Both undernutrition (Cavallo et al 1990) and abnormal thyroid function in pregnancy are known to lead to poor outcome (Thomas R et al 1987).

The present study was undertaken to evaluate the thyroid status in normal and abnormal pregnancies in relation to maternal nutritioinal status.

MATERIAL AND METHODS

Fifty five normal pregnant women in different trimesters of pregnancy, 28 non pregnant non lactating women and 19 third trimester pregnant women with pregnancy induced hypertension (PIH) were recruited from low income group (LIG) attending a general hospital and 46 pregnant women from high income group (HIG) attending a nursing home with different nutritional status. Data was collected on height, body weight, arm circumference, fat fold thickness. Detailed obstetric examination was carried out. Fasting blood was drawn for

T3, T4 and thyroid stimulating hormone (TSH). All the hormones were estimated by RIA kits from BRIT, Bombay. Statistical analysis was done by students 't' test.

RESULTS

The mean anthropometric parameters i.e. body weight, height and BMI in HIG were significantly higher as compared to LIG women. Higher percentage of LIG subjects had body weight less than 42 kg, (31.4%) BMI <19 (22.8%) as compared to HIG women 15.2% and 13.0% respectively.

T4 levels were significantly higher (P< 0.001) in pregnancy, values being 124 ± 24.5 nmol/1 in first trimester 145 ± 23.2 nmol/1 in in second and 161 ± 30.6 nmol/1 in third trimester as compared to NPNL (109 ± 27.3 noml/1) (Table I).

The mean T3 levels showed a significant increase in the second trimester followed by a decrease in the third trimester of preg-

Table IThyroid function in pregnancy.

Group	T4 nmol/L	T3 nmol/L	TSH mu/L
NPNL (28)	109 ± 27.3a	2.15 ± 0.445a	1.9 ± 1.01a
lst trimester (11)	124 + 24.5	2.15 + 0.384	1.3 <u>+</u> 0.65
2nd trimester (13)	145 <u>+</u> 23.2b	2.76 ±0.445b	1.4 <u>+</u> 0.64
3rd trimester (77)	161 ± 30.6b	1.84 ± 0.430a	1.5 <u>+</u> 0.76a
PIH (19)	166 <u>+</u> 32.3	2.00 <u>+</u> 0.476	2.6 ± 1.52b

Values are means \pm SD

Number of subjects are given in parenthesis

The values with different superscripts indicate significant difference a vs b p < 0.001

THYROID FUNCTION IN PREGNANCY

Group	T4 nmol/L	T3 nmol/L	TSH mu/L
BMI			
>19	162 ± 32.7 (46)	1.69 ± 0.384 (49)	
<19	163 <u>+</u> 31.8		. ,
	(13)	(13)	
Weight			
> 50 kg		1.84 ± 0.430 (38)	
< 50 kg		1.69 <u>+</u> 0.323 (41)	1.6 <u>+</u> 0.74
Income			
HIG	156 ± 32.2 (44)	1.84 ± 0.061 (46)	
LIG		1.69 <u>+</u> 0.046 (36)	1.4 <u>+</u> 0.65 (35)

			Labl	le II			
Thyroid	function	in	third	trimester	of	pregnancy	in
	relatio	n	to nut	tritional st	tatu	IS	*

Values are means \pm SD

Number of subjects are given in the parenthesis

nancy. The T3 levels are within the normal range. TSH did not show any change throughout pregnancy.

In PIH, no differences were seen in T3 and T4 levels as compared to normal pregnancy. However, TSH levels were significantly higher (P < 0.001) when compared to the levels in third trimester pregnant women and non pregnant non lactating women (NPNL).

Analysis of data with respect to anthropometric parameters and income in relation to LIG and HIG women did not reveal any significant differences (Table II).

None of the subjects showed evidence of thyroid deficiency as reflected in low T3 or T4 high TSH and all the values were within normal range.

DISCUSSION

The present study indicates that T3 and T4 are increased (though within normal physiological range) during pregnancy and TSH levels were unchanged except for a slight fall in the first trimester of pregnancy

JOURNAL OF OBSTETRICS AND GYNAECOLOGY OF INDIA

as compared to non pregnant women. Changes in T3 and T4 are similar to the results reported in the literature (Chan et al, 1975, Glinoer et al 1990, Gow et al, 1985, Sawhney et al, 1981, Smith et al 1983). These changes in T3 and T4 are known to be stimulated by hyperestrogenemia which increases serum thyroid binding globulin levels by inducing its sialylation and decreasing hepatic clearance (Ain et al, 1987).

The TSH response to Thyrotropic hormone (TRH) is suppressed in early pregnancy due to Human Chorionic Gonodotrophin (HCG) (Guillavine et al, 1985). There are controversial reports regarding TSH levels in pregnancy; some reporting no change (Chan et al, 1975) similar to present study levels and others an increase in levels (Glinoer et al, 1990) while significantly high TSH levels were seen in PIH, no changes were seen in T3 and T4 levels in the present study. High levels of T4 and subnormal T3 levels as compared to controls have been reported in PIH. The increase in TSH levels may reflect the severity of preeclampsia (Lao et al, 1988).

It is reported that T3 and T4 decrease inconditions of chronic malnutrition resulting in low BMR (Cavallo 1990). The lack of difference in the levels between the two socioeconomic group pregnant women either in relation to body weight or BMI may be due to the anabolic condition of the pregnancy requiring higher levels of T4 to cope up with the increased metabolic activities of the tissues and conceptus. The normal thyroid function in the present study could possibly be due to low endemicity of goitre in the area where our study was carried out.

Thus the thyroid function during normal pregnancy seems to be geared towards meeting the needs of growing conceptus.

ACKNOWLEDGEMENT

The authors are thankful to Dr. Vinodini Reddy, Director, National Institute of Nutrition, Hyderabad for her encouragement in the study. They are aos thankful to Mrs. Sudha Srinivasan for her secretarial assistance.

REFERENCES

- 1. Ain KB, Mori Y and Refetoff S : J. Clin. Endocrinol. & Metabol. 65; 689; 1987.
- Cavallo E, Armellini F, Zamboni M. Vicentini M, Milani P and Bosello O : Horm. Met. Res. 22; 632; 1990.
- 3. Chan V, Paraskevaides CA and IIalo JF : Brit J. Obstet & Gynec 82; 137; 1975.
- Glinoer D. Nayer PD, Bourdoux P, Lemone M, Robyn C, Steirteghem AV, Kin Thaert J & Lejevine B : J. Clin. Endocrinol. Metabol. 71; 276; 1990.
- 5. Gow SM, Kellet IIA, Seth J. Sweeting VM, Toft AD and Beckett GJ : Clin. Chim. Acta. 152; 325; 1985.
- Guillavine J, Schussler GC and Goldman J : J. Clin. Endocrinol & Metabol. 60; 678; 1985.
- 7. Lao TT, Chin RKII and Swaminathan R : Brit J. Obstet Gynec 95; 880; 1988.
- 8. Prout TE; Am. J. Obstet Gynec 122; 669; 1975.
- 9. Sawhney RC, Rastogi GK, Kumar M, Devi PK and Dash FJ : Ind. J. Med. Res. 73; 41; 1981.
- 10. Smith SCII & Bold AM : Brit. Obstet Gynae. 70; 532; 1983.
- 11. Thomas R. & Reid R.L. : Obstet & Gynec 70; 789; 1987.

310