Nausea, vomiting and thyroid function before and after induced abortion in normal pregnancy

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Summary: Thyroid function in early pregnancy has been reported to be slightly different from that in second or third trimester. We assessed thyroid function before and after induced abortion in normal pregnant women. A significant increase in serum Free T4 and a decrease in serum TSH were observed before abortion and these changes, apart from the contemporary significance of serum hCG- β , were especially marked in pregnant women with nausea and vomiting. On the other hand, an increased level of Free T4 and a reduced level of TSH returned to the normal ranges 7-10 days after induced abortion. Futhermore, serum hCG- β was significantly reduced.

These results suggest that, in normal early pregnancy, thyroid function may be related to serum $hCG-\beta$ concentration and its increased level, which induces gestational emesis.

Key words: Nausea; Vomiting; Thyroid function; Pregnancy.

INTRODUCTION

Nausea and vomiting are common symptoms in pregnancy and occur in over 50% of pregnant women (¹). A significant increase in serum Free Thyroxine (FT4) and a decrease in serum Thyroid Stimulating Hormone (TSH) were observed in subjects in early pregnancy with nausea and vomiting in relation to the levels in non-pregnant controls (²). These changes returned to the normal ranges after improvement of emesis or 1 week after in-

duced abortion (3). In order to distinguish slight transient hyperhyroidism associated with nausea and vomiting in normal early pregnancy from pregnancy associated with pre-existing thyroitoxicosis or hyperemesis gravidarum we have measured the serum levels of FT3, FT4, TSH and β subunit of human Chorionic Gonadotropin (hCG- β) before and after abortion in normal women in early pregnancy.

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MATERIALS AND METHODS

We measured the serum levels of Free T4, Free T3. TSH and hCG- β in 19 normal women in early pregnancy. Serum samples were obtained before and 7-10 days after induced abortion and the method used was radioimmunoassay (RIA). Seven out of 19 pregnant women showed gestational emesis with nausea and vomiting. We exclude subjects with autoimmune thyroid disease, thyrotoxicosis, and hidatiform moles. The results were expressed as the mean $\pm SD$ and the Student's paired T test was used for statistical testing.

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Table 1 Mean serum levels of FT4,	FT3, TSH and hCG-β before and after induced abortion in
patients with nausea and vomiting and	d in patients with no symptoms.

	Before induced abortion	After induced abortion	Before induced abortion	After induced abortion	
N.		7		12	
Age (yrs)	26.3 ± 4.2		27.0 ± 5.4		
Gest. Weeks	9.2 ± 1.3		9.6 ± 1.5		
FT4	1.66 ± 0.52	$1.25 \pm 0.17*$	1.35 ± 0.30	1.15 ± 0.14	
FT3	3.51 ± 0.55	3.41 ± 0.66	3.26 ± 0.45	3.19 ± 0.18	
TSH	0.59 ± 0.55	$1.33 \pm 0.68**$	1.03 ± 0.88	$1.28 \pm 0.79**$	
hCG-β	212 ± 159	$6.5 \pm 4.5***$	123 ± 112	6.9±5.1***	

RESULTS

Table 1 lists the mean serum levels of Free T4, Free T3, TSH and hCG-β in normal pregnant women before and after induced abortion. To clarify the relation of nausea and vomiting with change in serum Free T3, Free T4, TSH and hCG-β we divided women into two groups: those with nausea and vomiting and those with no symptoms. Serum hCG-β levels after and 7-10 days after induced abortion were reduced significantly (P<0.01) compared with the levels before induced Serum TSH levels were inabortion. creased significantly (P<0.02) 7-10 days after induced abortion compared with the levels before induced abortion in all pregnant women. A significant increase in serum levels of the Free T4 (P<0.001) was observed before induced abortion and were especially marked in pregnant women with nausea and vomiting. The values of both Free T4 and TSH returned to normal levels after induced abortion and, moreover, all the symptoms disappeared.

DISCUSSION AND CONCLUSION

The increase in Free T4 observed in the first trimester could be due to thyroid stimulation by human choriogonadotropin, a concentration which peaks at 10-13 weeks of gestation, decreasing to a relatively stable value by 20 weeks of gestation. This decline in human choriogonadotropin concentration may lead to a decrease in TSH output from the pituitary. The mechanism of transient hyperthyroidism in hyperemesis gravidarum is not clear. It has been suggested that it may be due to hCG (Norman 1981, Amir 1984).

We have observed an association between hCG and tyroid hormone levels (4,5) in individual patients and a significant correlation between hCG and Free T4 in hyperemetic pregnancy.

However, the concentration of hCG in hyperemesis subjects showed considerable overlap with normal pregnancy and therefore it is postulated that a molecular variant of hCG may be produced with exagerated TSH-like activity (6). Our results confirm our previous report of an increase of Free T4 in the first trimester of a normal pregnancy. An associated decrease in serum TSH suggests a physiologic activation of the thyroid gland. The most important finding in recent reports (7, 8) was that the combined changes in Free T4 increase and TSH decrease in early pregnancy were related to the severity of nausea and vomiting. Recently, Bober (9) also reported that 40% of their patients with hyperemesis gravidarum had elevated levels of Free T4 and an impaired TSH response to thyrotropin- releasing hormones, but stated that this "biochemical thyrotoxicosis" was transient. Our findings are compatible with the concept that hyperemesis gravidarum represents an extreme degree of the normal nausea and vomiting of pregnancy. The causal mechanism of gestational emesis is still unknown. Emesis usually occurs in the first trimester, when the level of hCG is high. In this study, we found that the mean level of hCG was highest in the group with nausea and vomiting. The results confirm those reported by Masson (10) of higher serum hCG levels in patients with emesis than in asymptomatic women.

The significant correlation found in this study between the serum concentration of hCG and Free T4 strongly suggests that the thyroid gland is physiologically activated in early pregnancy by hCG or a related substance, which may also induce gestational emesis.

Nevertheless, it is important to also keep psychological factor into account which could be causal factors or play a role in worsening symptoms.

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