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ROLE OF T3, T4 AND TSH levels IN HYPEREMESIS GRAVIDARUM PATIENTS

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ABSTRACT

Introduction: *Hyperemesis gravidarum is defined as vomiting sufficiently severe to produce weight loss, dehydration, acidosis from starvation, alkalosis from loss of HCl, and hypokalemia. Various life-threatening complications occurs including acute renal failure, Mallory–Weiss tears, oesophageal rupture, pneumothoraces etc. The cause of Hyperemesis appears to be unknown and it is proposed to be related to high levels of HCG, estrogens, or both and thyroid hormones.* ***Aims and Objectives:*** *To estimate the T3, T4 and TSH levels in hyperemesis gravidarum patients.* ***Methods and Materials:*** *40 pregnant women less than 20 weeks of gestation, who have excessive vomiting and require hospital admission due to vomiting, are taken as cases and 40, age matched pregnant women less than 20 weeks of gestation without vomiting are taken as controls. Serum T3, T4 and TSH levels were estimated by automated chemiiluminiscence immunoassay.* ***Result:*** *The mean serum T3 level is 2.53±2.1 ng/ml in cases and 1.56±0.8 ng/ml in controls. The serum T3 level is high in cases but not statistically significant (p value 0.009). The mean serum T4 level is 13.59±4.3 µg/dl in cases and 9.22±3.1 µg/dl in controls. The serum T4 level is high in cases, which is statistically significant (p value< 0.001). The mean TSH level is 1.23±1.8 µIU/ml in cases and 2.97±1.8 µIU/ml in controls. The serum TSH level in cases is low, which is statistically significant (p value<0.001).* ***Conclusion:*** *Altered thyroid function may be the cause of vomiting and may attribute to its prolongation to second trimester.*

KEY WORDS

Hyperemesis Gravidarum, T3, T4, TSH.

INTRODUCTION

Nausea and vomiting are the most common symptoms experienced by a pregnant woman especially during first trimester.[1,2,3,4] It occurs in almost 50 to 90 % of pregnant women.[1,2,3,4] Normally nausea and vomiting may subside after first trimester, but 20 % women with symptoms may continue throughout the pregnancy.[4,5]

Hyperemesis gravidarum is a life threatening condition defined as persistent vomiting accompanied by weight loss of at least 5 % of pre-pregnancy body weight, which is unrelated to other causes and requires hospital admission for severe vomiting or any other complications like dehydration, electrolyte and metabolic disturbances, nutritional deficiencies [4,6,7,8,9,10].

The incidence of hyperemesis gravidarum is varying from 0.3 to 2 % of all pregnancies and affecting 0.5 % of live births. [4,7,8,11,12] Hyperemesis gravidarum occurs between 4th and 10th week of gestation, peaks at 10th week and resolves by 20th week of gestation. [2,7,13]

However, it may persist throughout the pregnancy in 10 % cases.

Hyperemesis gravidarum has both fetal and maternal adverse outcome; adverse fetal outcomes are low birth weight, preterm birth and small for gestational age.[8,14,15,16] Adverse maternal outcomes malnutrition, electrolyte imbalance, thrombosis, wernicke's encephalopathy, depression and muscle weakness etc.[7,14,15,17,18,19]

The etiology is still unknown. Some hormones like human chorionic gonadotropin hormone that has thyroid stimulating activity, estrogen and progesterone may have role in the hyperemesis gravidarum.[4,6,7,11,20]

Thus, this study was done to see the relation between thyroid status and hyperemesis gravidarum.

MATERIALS AND METHODS

This, case-control, study was conducted on randomly selected 80 pregnant women at less than 20 weeks of gestation from Bapuji Hospital, Davangere. Out of these 80 women, 40 women, less than 20 weeks of gestation, taken as cases, who were admitted in the hospital due to excessive vomiting. Forty pregnant women, less than 20 weeks of gestation, without any vomiting or with normal pregnancy were included as controls. Women more than 20 weeks of gestation, twin pregnancy, hydatiform mole, known case of thyroid disorder and any other chronic disorder, vomiting due to any other cause, were not included in the study.

In women of both groups 5 ml of venous blood was withdrawn, serum separated to estimate T3, T4 and TSH levels. Serum T3, T4 and TSH levels were estimated by chemiiluminiscence immunoassay.

RESULTS

As shown in Table No. 1 the mean age of cases and controls were 23.8 ± 2.4 and 23.42 ± 2.7 years respectively. All cases and controls were having sufficient intake of iodine and in euthyroid state. 57.5% (23) of cases were nulliparous and 42.5% (17) cases were multipaous. 55% (22) controls were nulliparous while 45% (18) controls were multiparous. Only 32.5 % (13) cases were having high T3 values of which 61.5% (8) were nulliparous. As shown in Table no. 2 and in the graph the mean T3 level in cases was 2.53 ± 2.1 ng/ml and in controls was 1.56 ± 0.8 ng/ml with the p value of 0.009, which is not statistically significant, although the value is higher in cases than in the controls.

70% (28) cases were having high T4 levels out of which 67.8% (19) were nulliparous. As shown in Table no 2 and in the graph the mean T4 levels were 13.59 ± 4.3 µg/dl and 9.22 ± 3.1 µg/dl in cases and controls respectively with the p value of <0.001, which is statistically significant.

67.5 % (27) were having low TSH level out of which 70.3 % (19) were nulliparous. As shown in Table no 2 and in the graph the mean TSH levels were 1.23 ± 1.8 µIU/ml and 2.97 ± 1.8 µIU/ml in cases and controls respectively, with the p value of <0.001, which is statistically significant.

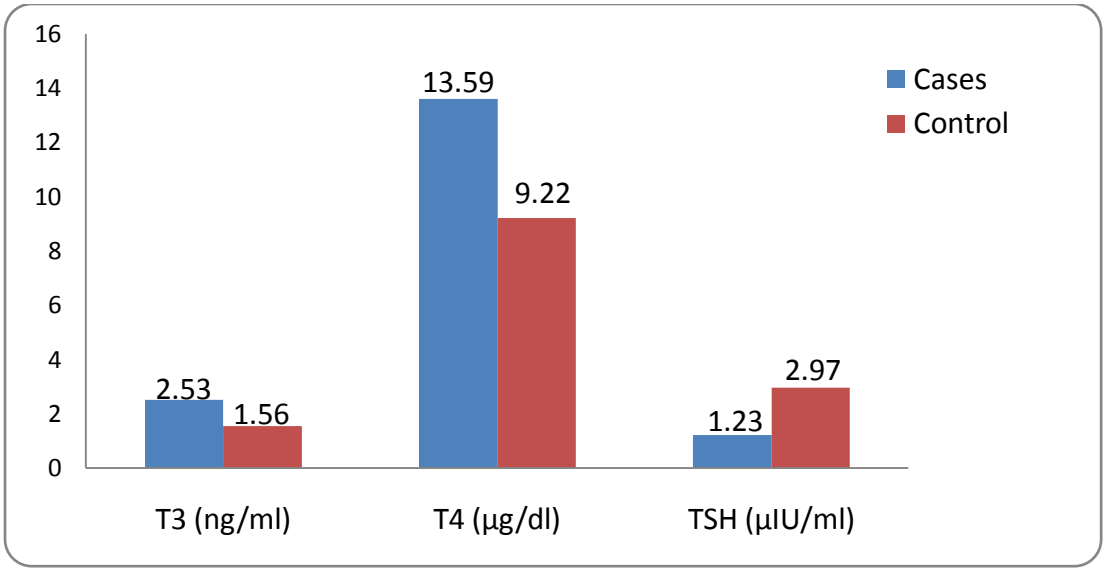
	Cases	Controls
Age (Years)	23.8 ± 2.4	23.42 ± 2.7
Nulliparous	23 (57.5 %)	22 (55 %)
Multiparous	17 (42.5 %)	18 (45 %)
Total	**40**	**40**

Table No. 1: Mean age, number of cases and controls in the study.

Parameters	Cases	Control	p value*
T3 (ng/ml)	2.53 ± 2.1	1.56 ± 0.8	0.009
T4 (µg/dl)	13.59 ± 4.3	9.22 ± 3.1	< 0.001
TSH (µIU/ml)	1.23 ± 1.8	2.97 ± 1.8	< 0.001

Table No. 2: Levels of T3, T4 and TSH in cases and controls.

***p value < 0.001 is statistically significant.**



Graph: Mean levels of T3, T4 and TSH in Cases and Controls

DISCUSSION

Although the exact pathogenesis of hyperemesis gravidarum is unknown, it is widely accepted that various metabolic and endocrine factors have some role in causing hyperemesis gravidarum. Human chorionic gonadotropin hormone is the one of the most important factor, which connects with hyperemesis gravidarum. The peak of hyperemesis gravidarum is between 12 to 14 weeks, as the peak of hCG production from placenta.[21,22]

The exact mechanism, how hCG can cause hyperemesis gravidarum is not clear, but it may be due to an effect on the secretory processes in the upper GI tract or by stimulation of the thyroid function due to structural similarity.[7,23,24]

hCG is a bipeptide and a glycoprotein, which has an alpha subunit that is identical to TSH and beta subunit which has sequence homology to TSH.[11,25,26,27] Thyroid function is increased due to activation of TSH receptor by hCG. During this time, T3 and T4 level may increase.[7,28,29,30,31] Under the influence of estrogen the thyroid binding globulin production will be more and clearance will be less, thus The thyroid gland increases production of T3 and T4 to meet the requirement.[6,7,25]

In our study, the T3 value is higher in cases compare to controls but not statistically significant, while T4 is significantly higher in cases compare to controls. The TSH value is significantly decreases in the cases compare to controls. The more significant increase in the T3 and T4 levels and significant decrease in the TSH levels in the nulliparous cases compare to multiparous cases are because of the first exposure of hCG to the nulliparous.

These results are consistent with the study done by Prince et al.[32] Ganguli et al also found out

significant raise in T4 and suppression of TSH in cases when compared with controls, while T3 is increased but not significantly.[33] A study done by Gill et al also was consistent with the results we got in our study.[11]

Tan et al had also reported transient hyperthyroidism in hyperemesis gravidarum.[34] Goodwin et al reported that hyperemesis gravidarum women had hyperthyroidism or suppressed TSH.[35]

CONCLUSION

In conclusion, hyperthyroidism is associated with hyperemesis gravidarum and hyperemesis can lead to several, severe complications, so routine investigation of thyroid function is necessary to prevent adverse fetal and maternal outcome.

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