Ghrelin does not change in hyperemesis gravidarum

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ABSTRACT

Objectives: Ghrelin levels can play an important role in maintaining the energy balance of pregnant women. Therefore, we investigated the relationship between HG and Ghrelin.

Material and methods: 50 female patients admitted to the VAN Yüzüncü Yıl University, Gynecology and Obstetrics Department were evaluated. The patients were divided into two groups: Group 1 included 25 pregnant women with HG, Group 2 included 25 healthy pregnant women.

Results: The two groups showed similarities in terms of age, gravidity, B-HCG and gestational age. There was no statistically significant difference between the two groups in terms of the Ghrelin levels (p = 0.867).

Conclusions: This study shows that there is no difference between Ghrelin levels and HG during pregnancy. Increased Ghrelin in previous studies was attributed to low oral intake. Another study reported lower Ghrelin levels are not the result of, but are rather the cause of, reduced oral intake during. The balancing of these two conditions does not lead to a change in the level of Ghrelin.

Key words: Ghrelin; Hyperemesis Gravidarum; Etiopathogenesis

INTRODUCTION

Nausea and vomiting can be seen in 80% of pregnant women in the first months of pregnancy. Hyperemesis gravidarum (HG), a severe form of nausea and vomiting, occurs in about 0.3–3% of all pregnancies. Severe nausea and vomiting occurring more than three times a day in patients with ketonuria and more than 5% weight loss are diagnosed with HG. Its aetiology has yet to be fully elucidated [1–3].

Ghrelin, which is a gastric hormone that regulates food intake, energy metabolism and growth hormone secretion, attracts attention as an appetizing hormone. Ghrelin levels increases before a meal and decreases after a meal [4, 5].

There have been many studies in the literature between HG and Ghrelin. Ghrelin levels can play an essential role in maintaining the energy balance of pregnant women. Therefore, we investigated the relationship between HG and Ghrelin.

MATERIAL AND METHODS

In this study, 50 female patients admitted to the VAN Yüzüncü Yıl University, Gynecology and Obstetrics Department were evaluated. The patients were divided into two groups: Group 1 included 25 pregnant women with HG, Group 2 included 25 healthy pregnant women.

Patients with comorbid diseases, such as trophoblastic diseases, gestational diabetes, preeclampsia, thyroid diseases, infectious diseases, inflammatory diseases, renal diseases, hepatic diseases and psychiatric disorders, as well as patients with smoking and alcohol habits, those with chronic medication use and those with pregnancies over 10 weeks were excluded from the study. The gestational
weeks were determined according to the patient’s last menstrual period and by ultrasonography.

Blood is taken from the antecubital vein and centrifuged at 1500 rpm for 15 minutes (core and NF 800, Turkey,) is obtained after Sera removed to Eppendorf tubes and stored at -80°C. Serum samples prepared in this way were sent to Bolu Abant Izzet Baysal University Faculty of Medicine in order to study. Samples were measured using the Biovendor Human Acylated Ghrelin ELISA kit (No: RD194062400R, Biovendor GMBH, Heidelberg ). Results are given in pg/mL.

Written and verbal informed consent was obtained from all of the volunteers. The ethical principles for medical research involving humanitarian issues envisaged in the Declaration of Helsinki were applied.

Statistical analysis
Numerical variables were figured as mean plus/minus standard deviation. For the comparative analysis between the groups (case vs control), a χ² test was used for the categorical variables and either a Student’s t-test or the Mann–Whitney U test was used for the continuous variables. Differences were considered significant at p < 0.05.

RESULTS
Group 1 consisted of 25 patients with ketone positive (HG), Group 2 consisted of 25 healthy pregnant women who were admitted to the hospital for routine controls. Clinical and laboratory results are shown in Table 1. No significant differences were found between the groups in terms of the maternal age, gestational age, gravidity and B-HCG. There was no statistically significant difference between the two groups in terms of the Ghrelin (p = 0.867).

Table 1. Baseline criteria and treatment outcome of the groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n = 25)</th>
<th>Group 2 (n = 25)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) (mean ± SD)</td>
<td>27.60 ± 4.8</td>
<td>28.92 ± 5.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>7.96 ± 1.3</td>
<td>7.2 ± 1.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Gravidity</td>
<td>4.04 ± 1.45</td>
<td>4.22 ± 1.34</td>
<td>0.8</td>
</tr>
<tr>
<td>Ghrelin (pg/mL)</td>
<td>55.2 ± 14.7</td>
<td>54.52 ± 13.7</td>
<td>0.86</td>
</tr>
<tr>
<td>B-HCG</td>
<td>94662 ± 72040</td>
<td>71099 ± 65061</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation

DISCUSSION
In this study, we investigated Ghrelin changes between pregnant women with HG and healthy pregnant control group. Ghrelin levels were similar in both groups.

Although many studies have been conducted on HG, a clear decision on its etiology has not been disclosed. Some studies have investigated other peptides, such as leptin, that affect food intake and appetite. Nausea and vomiting and changes in leptin levels have been studied [6, 7], but these studies have failed to explain the etiology. For this reason, the hormone Ghrelin has gained importance with the thought that it may be involved in the etiology of HG, and many studies have been done on this subject.

Oruç et al. [8] found that Ghrelin levels were significantly increased in patients with HG compared to normal pregnant women, which may be due to the need for the maternal organs to react with a compensatory mechanism to recover energy. The level of Ghrelin increases before eating and decreases after a meal [9], which means plasma Ghrelin and oral intake correlate negatively. In contrast, Kaygusuz et al. [10] found higher Ghrelin levels in the control group than in the HG group. This suggests that the lower Ghrelin levels are not the result of, but are instead the cause of, reduced oral intake during HG. Wibowo et al. [11] compared Ghrelin levels in patients with HG treated with vitamin B6 supplementation divided into two groups. However, it found no difference between two groups. In a study by Chittumma et al. [12], vitamin B6 was given to women with hyperemesis, B 6 was useful for the treatment of HG. Since nausea and vomiting were reduced in patients receiving vitamin supplements, their energy requirement would be reduced, and Ghrelin levels would be expected to decrease, but the Wibowo study, found no difference in Ghrelin levels between HG patients given B6. We think this is a situation that supports our study. Increased Ghrelin in previous studies was attributed to low oral intake. Another study reported lower Ghrelin levels are not the result of but are instead the cause of, reduced oral intake during [8, 10]. The balancing of these two conditions does not lead to a change in the level of Ghrelin. This suggests that the aetiology of HG requires more comprehensive studies.

CONCLUSIONS
The limitation of this study was that we had a small number of patients. Secondly, we could not measure the pre-pregnancy Ghrelin of these pregnant women. Thirdly, we did not investigate other factors that affect Ghrelin levels in maternal serum.

This study shows that there is no difference between Ghrelin levels and HG during pregnancy. We do not know the exact cause of this mechanism. However, we think that many mechanisms balance this situation. We believe that more extensive studies are needed to investigate the role of Ghrelin in HG pathology.

REFERENCES


