HYPERHOMOCYSTEINEMIA AT 16th WEEK GESTATION AND SUBSEQUENT PREECLAMPSIA

Abstract

This study was done on 197 pregnant women. Fasting plasma total homocysteine (Hcy) was measured in each woman at 16th week of pregnancy. They were divided into 2 groups according to development of preeclampsia. Women who did not develop preeclampsia (n = 179) comprised control group. Women who developed preeclampsia (n = 18) comprised the study group.

Fasting plasma Hcy at 16th week gestation was significantly higher in preeclampsia group compared to control group (P<0.05). Also the number of cases with Hcy ≥ 90th percentile of controls (≥5.37 ng/dl) was significantly higher in preeclampsia group compared to control group.

It could be concluded that hyperhomocysteinemia at 16th week gestation may be an indirect risk factor for placental vasculopathy predisposing the study group.

Introduction

Homocysteine is not present naturally in the diet, but it is an essential intermediate in metabolism of methionine. Each compound, methionine or homocysteine, is the precursor of the other and the synthesis of one is the mechanism of detoxification of the other (Finkelstein & Martin, 2000). Homocysteine metabolism...

Hyperhomocysteinemia, a known vascular disease risk factor, is associated with placental vascular syndromes of preeclampsia, placental abruption, thrombocytopenic
mass index (BMI), blood pressure and body
mass, and then on the basis of the
determined to determine the
2. Complete a general and a

rectal examination.

detect

defects. Defects were subsequently

identified in the medical records of the

participants.

1. Full history was taken with the


were


subjects to the following:

All study participants were

subjects for the purposes of

the 16th week of gestation.

were collected to give blood samples at

hospital and attendance clinic until

antenatal care.

This study comprised 197

Subjects and Methods

The aim of the work

were the authors of this study are

to determine the prevalence of

homocysteine levels in

maternal plasma, as compared with

plasma of non-pregnant women.

In late gestation, levels of ho-

1997).

with this process (Kraftkova et al.,
cytochrome may be associated

been presented that hypertension

of pre-eclampsia, a hypothesis has

fetion and increased platelet age-

1999) as endothelial cell dysfunc-

stress (Hamka et al., 1999).

in pre-eclampsia by oxidative

advanced endothelial dysfunction

concentration in pregnancy may

so, 1998). Increased homocysteine

no information (Welch and Lascal-

endothelial cell injury and throm-

fully understood. It may include

endothelial dysfunction on vascular health is not

damaging effect of hyperhomocys-

Although the mechanism of

2000).

Fetal death in utero (Wang et al.,

intrauterine growth retardation or

events, and/or fetal syndromes of

(Sorenson et al., 1999; Hultén et

et al., 2001).
2. Ultrasound examination for the preser-

preservation, edema and occurrence of my-

and the decrease of lower limb reser-

t to the occurrence. formal examination with

monthly for:

baseline data collection and

obviation of follow up period (be-

ered hypothyroidism). The

levels of TSH > 5.27 μg/dl were consid-

erined as those with plasma

cysteine, IgG or total plasmino-

< 2000 mg/dl (Table 6). Co-

on level of the control group

90% of baseline plasma le-

rate. The plasma was sepa-

ted from all women into ci-

blood samples were collect-

b. Blood samples: Various

by immunometric method,

cooled to measure protein

- Lifetime of 24 hours was col-

- Exclusion urination.

- a. Complete urine analysis.

- b. Laboratory investigations.

- c. Urinary antigen examinations.

- d. Congenital abnormalities.

- e. Total age, and to exclude le-

- f. To confirm the results.

3. Ultrasound examinations.
between fasting plasma HCGs was a significant positive correlation in the Preclampsia Group. There were no significant differences in levels of cases with hypertension compared to control groups. Group II preclampsia and control groups were divided into two groups based on whether or not the women were retrospectively allocated into two groups: I. Control Group (I): Women who completed the follow-up period without the development of preclampsia. II. Preclampsia Group (II): Women who developed preclampsia during the follow-up period. The prevalence of preclampsia was significantly higher in the Preclampsia Group (9.1%) compared to the Control Group (1.1%). Twenty weeks gestation, at least two occasions 6 hours apart > 140/90 mmHg plus significant proteinuria (≥ 300 mg) at each visit was diagnosed as Preclampsia. The correlation coefficient for follow-up of the case was 0.05. For all a correlation coefficient r, for all a correlation coefficient r, for all a correlation coefficient r, for all a correlation coefficient r, correlation coefficients were estimated using Pearson’s method. The correlations were analyzed by Z test. Correlation coefficients were estimated by number and percent and were used for paired t-test. Variability pre-
### Table 1: Plasma levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
<th>Group III (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>101.9 ± 14.8</td>
<td>78.4 ± 13.8</td>
<td>79.4 ± 5.7</td>
</tr>
<tr>
<td>DLB</td>
<td>153.6 ± 24.1</td>
<td>123.8 ± 30.2</td>
<td>123.8 ± 30.2</td>
</tr>
<tr>
<td>150.5 ± 12.4</td>
<td>120.3 ± 17.5</td>
<td>120.3 ± 17.5</td>
<td></td>
</tr>
<tr>
<td>29.7 ± 12.4</td>
<td>29.7 ± 12.4</td>
<td>29.7 ± 12.4</td>
<td></td>
</tr>
<tr>
<td>1.63 ± 0.05</td>
<td>1.63 ± 0.05</td>
<td>1.63 ± 0.05</td>
<td></td>
</tr>
<tr>
<td>78.3 ± 3.9</td>
<td>78.3 ± 3.9</td>
<td>78.3 ± 3.9</td>
<td></td>
</tr>
<tr>
<td>30.4 ± 2.8</td>
<td>30.4 ± 2.8</td>
<td>30.4 ± 2.8</td>
<td></td>
</tr>
<tr>
<td>%</td>
<td>77.2%</td>
<td>77.2%</td>
<td>77.2%</td>
</tr>
<tr>
<td>n (n=179)</td>
<td>20</td>
<td>11.2%</td>
<td>11.2%</td>
</tr>
<tr>
<td>p</td>
<td>0.05</td>
<td>(U=179)</td>
<td>(U=179)</td>
</tr>
</tbody>
</table>

#### Table 2: Plasma cholesterol levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>

#### Table 3: Plasma triglycerides levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
</tbody>
</table>

#### Table 4: Plasma insulin levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>

#### Table 5: Plasma glucose levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>

#### Table 6: Plasma FBS levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>

#### Table 7: Plasma creatinine levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>

#### Table 8: Plasma urea levels at 16th week gestation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (control)</th>
<th>Group II (pre eclampsia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>250 ± 30</td>
<td>250 ± 30</td>
</tr>
<tr>
<td>LDL</td>
<td>100 ± 20</td>
<td>100 ± 20</td>
</tr>
<tr>
<td>HDL</td>
<td>50 ± 10</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>150 ± 50</td>
<td>150 ± 50</td>
</tr>
</tbody>
</table>
FIG. 1: Correlation coefficient between some parameters and fasting plasma level of [HCVs (μg/dl) in preclampsia group.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>BMI (kg/m²)</th>
<th>Age (years)</th>
<th>Severity of Preclampsia</th>
<th>Occurrence of Preclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td>d</td>
<td>0.0120</td>
<td>1.0500</td>
<td>0.9700</td>
<td>0.8600</td>
</tr>
</tbody>
</table>

Table (3): Correlation coefficient between fasting plasma level of [HCVs and some parameters in preclampsia group.
levels were higher in preclampsia maternal plasma homocysteine
Volset et al. (2000) reported that
Rahman et al. (2000) and
Rahman et al. (2000). Relatation (Rubard et al., 2000) in vitro and intracellular growth
fears and perhaps with fetal death
be embryonic events, neural tube de-
the placental abruption, preecamp-
preclampsia and hypertension may be
the pregnancy. It appears that hyp-
ions has been made only recently
for several pregnancy complications
hyperhomocysteinemia may be responsible
The discovery that hyperhonio-
•
her disease (hyperleukocytosis and me-
the pathogenesis of hyperhomocystein-
are agents or merely a marker for
ction of homocysteine is the cause-
whether the increased concentration
risk factor for hyperhomocystein-
cysteinaemia is an independent
sional inadegacy. Hyperhonio-
guished pathology, toxicity and nu-
result from general variabilities, ac-
metabolic implications that may
Hyperhomocysteinemia is a

**Discussion**
The present study shows a negative correlation between maternal age and fasting plasma homocysteine levels. A significant correlation between maternal age and elevated plasma homocysteine levels was also observed in the pre eclampsia group. Moreover, the study showed that women with a higher body mass index and who also had other medical conditions were more likely to develop pre eclampsia. These results are in agreement with those reported by So et al. (1996) who found a significant correlation between maternal age and plasma homocysteine levels.

The results of the present study, along with those of previous studies, indicate that maternal age is a risk factor for pre eclampsia. Maternal age has been shown to be associated with an increased risk of pre eclampsia, and women with a higher body mass index are more likely to develop the condition. These findings provide a basis for further research into the potential role of maternal age in the development of pre eclampsia.
References

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