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Proteinuria is not a Prequisite Criteria for Preeclampsia Diagnosis

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Abstract: Preeclampsia is now to be diagnosed by decreased blood platelets, trouble with the kidney or liver, fluid in the lungs or signs of brain trouble such as seizures and/or visual disturbances. This is an cohort prospective study conducted in H. Adam Malik hospital. All preeclampsia mother with or without proteinuria were included in this study. Data about the proteinuria, thrombocyte count, liver function (AST and ALT) renal function (ureum and creatinine) pulmonary edema (clinical diagnosis) and cerebral dysfunction (confuse, visual disturbance, severe headache) were collected. Statistical comparisons were made using SPSS 17. In this study, there was no association found between the number of complications between the groups with or without proteinuria ($\chi^2 = 1.777, p = 0.777$). Likewise, no difference was found between the severity of proteinuria and number of complications ($\chi^2 = 11.852, p = 0.754$). Proteinuria is not a prequisite for the diagnosis of preeclampsia.

Key words: Proteinuria, preeclampsia, organ dysfunction, prequisite, severity

INTRODUCTION

An estimated 50,000 women worldwide die annually from preeclampsia. The incidence of preeclampsia is 2-10%, depending on the population studied and definitions of preeclampsia (Shamsi et al., 2013). Preeclampsia is responsible for approximately 50,000 maternal deaths worldwide annually out of which 25% of cases are due to Intra Uterine Growth Restrictions (IUGR) and 15% are outcome of preterm birth in developed countries (Bell, 2010).

The American College of Obstetricians and Gynecologists (ACOG) Task Force on Hypertension in Pregnancy changed preeclampsia diagnosis criteria on November 2013. This report built by 16-member of ACOG task force for almost 2 year changes the paradigm about using proteinuria to diagnose preeclampsia (ACOG, 2013). They omitted proteinuria as a prequisite for preeclampsia and preeclampsia is diagnosed if hypertension occurs with any of the following: thrombocytopenia, impaired liver function, new-onset renal insufficiency, pulmonary edema or new development of cerebral or visual disturbances. This changing is due to the presence of significant protein in the urine would indicate multisystemic involvement, the degree of proteinuria correlates poorly with short-term adverse outcomes and long-term maternal renal prognosis (Duhig and Sherman, 2015).

The presence of proteinuria is not essential to the diagnosis of pre-eclampsia under many diagnostic consensus statements. Rather the task force includes evidence of maternal hepatic, renal, pulmonary and cerebral end-organ disease in the diagnostic criteria even in the absence of proteinuria. The aim of the present study was to assess preeclampsia complication in renal, liver, lung and cerebral between proteinuric pre-eclampsia compared with other non-proteinuric disease presentation.

MATERIALS AND METHODS

This is an cohort prospective study conducted in H. Adam Malik hospital. All preeclampsia mother with or without proteinuria were included in this study. Researchers obtained informed consents from all the subjects who were willing to join this study. Any subjects with other malignancies and delivered stillbirth or died newborns were excluded from this study.

The New ACOG Task Force suggests that the diagnostic criteria for preeclampsia be changed as: systolic or diastolic blood pressures values = 160 or 110 mm Hg, respectively occurring twice, 4h apart at bed rest; thrombocytopenia (platelet counts <100,000 µL); impaired liver function defined as either otherwise unexplained right-upper-quadrant-epigastric pain unresponsive to medications or hepatic transaminase levels twice normal; progressive renal insufficiency as defined above; pulmonary edema, new-onset cerebral or visual disturbances (ACOG, 2013).

Data about the proteinuria status, thrombocyte count, liver function (AST and ALT) renal function (ureum and creatinine) pulmonary edema (clinical diagnosis) and cerebral dysfunction (confuse, visual disturbance, severe headache) were collected. Statistical comparisons were made using SPSS 17 between proteinuric and non proteinuric group with
thrombocytopenia, impaired liver function, impaired renal function, pulmonary edema and cerebral dysfunction. A significance of 0.05 was taken.

RESULTS AND DISCUSSION

A total of 71 subjects with and without proteinuria preeclampsia included in this study. Characteristics of research subjects can be seen in Table 1. In the group without proteinuria, 2 (7.1%) subjects experienced thrombocytopenia, 9 (32.1%) subjects had cerebral dysfunction, 9 (32.1%) subjects had liver disorders, 7 (25%) subjects had renal insufficiency and 1 (3.6%) subjects had pulmonary edema. In preeclampsia group with proteinuria, 2 (4.7%) subjects had thrombocytopenia, 8 (8.6%) subjects had cerebral dysfunction, 14 (16.3%) had liver disorders, 7 (16.3%) had renal insufficiency and 14 (32.6%) had pulmonary edema. There is no difference in complications of thrombocytopenia, cerebral dysfunction, liver disorders, renal insufficiency and pulmonary edema both in preeclampsia group with or without proteinuria (p = 0.0656; p = 0.191; p = 0.971; p = 0.0367; p = 0.0153, respectively) (Table 2).

Analysis of number of complications between both groups, this study showed no differences between them (χ² = 1.777; p = 0.777). Majority of subjects in the group with and without proteinuria had no complications (57.1 and 55.8%, respectively). About 4% subjects in each group experienced <4 complications. The mean number of complications experienced by subjects in each group were 1-2 complications (Table 3). In fact, the number of complications was slightly higher in group proteinuria (0.96 vs. 0.79) but this was not statistically significant (p = 0.555) (Table 4). There was also no association between proteinuria severity and number of organ dysfunctions (χ² = 11.852; p = 0.754) (Table 5).

Evidence shows organ problems in the kidneys and livers can occur without signs of protein and that the amount of protein in the urine does not predict how severely the disease will progress (Waugh et al., 2005). Eliminated from measures of severe preeclampsia are massive proteinuria (i.e., >5 g day⁻¹) and fetal growth restriction because the extent of proteinuria does not predict morbidity and fetal growth restriction occurs commonly in the absence of associated preeclampsia (ACOG, 2013). One group of authors reported that hypertension and proteinuria were absent in 38% of women who presented with an eclamptic fit (Ouglas and Redman, 1994).

Homer et al. (2008) study showed that whether proteinuria or non proteinuria preeclampsia had different risk factors of complication. Women with proteinuric pre-eclampsia were more likely to have severe hypertension (39 versus 30%, p = 0.003) deliver preterm infants (39 versus 30%, p = 0.007) and had a higher perinatal mortality rate (25.2 versus 5.7 per 1000, p = 0.02) than those with non-proteinuric pre-eclampsia, who were more likely to have thrombocytopenia and liver disease. Women with non-proteinuric-eclampsia were more likely to have multiple pregnancies (3.9 versus 9.9%, p<0.001) experience severe hypertension (8.9 versus 29.7%, p<0.001) and deliver preterm infants (11.3 versus 30.2%, p<0.001) who were small for gestational age (12.7 versus 20.9%, p<0.001) than those with gestational hypertension.

In the other side, Chan et al. (2005) showed an adverse maternal outcome was significantly associated with higher spot urine protein/creatinine ratio at diagnosis (p<0.0001) with an odds ratio of 1.03 per mg/mmol (95% Confidence Interval [CI] 1.002-1.04) and with older maternal age (p = 0.014) with OR 1.06 per year (95% CI 1.01-1.11). An increased risk of adverse fetal outcome was associated with higher spot urine protein/creatinine (p = 0.013 or 1.44 per log [mg/mmol], 95% CI 1.08-1.92) gestation at diagnosis <34 weeks (p<0.0001; OR 3.60, 95% CI 1.90-6.82) and early pregnancy systolic blood pressure < or = 115 mmHg (p = 0.0002, OR 3.41, 95% CI 1.90-6.82).
Table 2: Multiorgan dysfunction in non proteinuric and proteinuric preeclampsia group

<table>
<thead>
<tr>
<th>Proteinuria</th>
<th>Thrombocytopenia</th>
<th>Neurologic</th>
<th>Liver</th>
<th>Renal</th>
<th>Pulmonary</th>
</tr>
</thead>
<tbody>
<tr>
<td>No proteinuric</td>
<td>2 (7.1%)</td>
<td>9 (32.1%)</td>
<td>9 (32.1%)</td>
<td>7 (25%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Proteinuric</td>
<td>2 (4.7%)</td>
<td>8 (18.6%)</td>
<td>14 (32.6%)</td>
<td>7 (16.3%)</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>X²</td>
<td>0.198</td>
<td>1.707</td>
<td>0.001</td>
<td>0.815</td>
<td>2.940</td>
</tr>
<tr>
<td>p-value</td>
<td>0.656</td>
<td>0.191</td>
<td>0.971</td>
<td>0.367</td>
<td>0.153</td>
</tr>
</tbody>
</table>

Table 3: Relationship between number of organ dysfunctions and proteinuric preeclampsia

<table>
<thead>
<tr>
<th>Proteinuria</th>
<th>0 (%)</th>
<th>1 (%)</th>
<th>2 (%)</th>
<th>3 (%)</th>
<th>4 (%)</th>
<th>γ²</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No proteinuric</td>
<td>16 (57.1)</td>
<td>4 (14.3)</td>
<td>2 (7.1)</td>
<td>5 (17.9)</td>
<td>1 (3.6)</td>
<td>1.777</td>
<td>0.777</td>
</tr>
<tr>
<td>Proteinuric</td>
<td>24 (55.8)</td>
<td>11 (25.6)</td>
<td>2 (4.7)</td>
<td>5 (11.6)</td>
<td>1 (2.3)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 4: Comparison between total complications between proteinuric and non proteinuric preeclampsia groups

<table>
<thead>
<tr>
<th>Proteinuria</th>
<th>Total complications</th>
<th>SD</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No proteinuric</td>
<td>0.96</td>
<td>1.32</td>
<td>1.729</td>
<td>0.555</td>
</tr>
<tr>
<td>Proteinuric</td>
<td>0.79</td>
<td>1.13</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 5: Association between severity of proteinuria and number of complications

<table>
<thead>
<tr>
<th>Proteinuria</th>
<th>0 (%)</th>
<th>1 (%)</th>
<th>2 (%)</th>
<th>3 (%)</th>
<th>4 (%)</th>
<th>γ²</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No proteinuric</td>
<td>7 (46.7)</td>
<td>3 (20.0)</td>
<td>1 (6.7)</td>
<td>4 (26.7)</td>
<td>0 (0)</td>
<td>11.852</td>
<td>0.754</td>
</tr>
<tr>
<td>+1</td>
<td>9 (69.2)</td>
<td>1 (7.7)</td>
<td>1 (7.7)</td>
<td>1 (7.7)</td>
<td>0 (0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>+2</td>
<td>12 (66.7)</td>
<td>4 (22.2)</td>
<td>0 (0)</td>
<td>2 (11.1)</td>
<td>0 (0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>+3</td>
<td>2 (40.0)</td>
<td>2 (40.0)</td>
<td>1 (20.0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>+4</td>
<td>2 (40.0)</td>
<td>2 (40.0)</td>
<td>1 (20.0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

The area under the Receiver Operating Characteristic (ROC) curve was 0.67 for adverse maternal outcomes and 0.72 for adverse fetal outcomes.

Thornton et al. (2010) study indicate that the presence of proteinuria denotes a group of women who have higher antenatal blood pressure, who deliver at earlier gestation and require operative delivery more commonly although it is not an indicator of other markers of maternal morbidity or perinatal mortality. The proteinuric cohort had higher systolic and diastolic blood pressure recordings than the non-proteinuric cohort (160/102 and 149/94 mmHg, respectively, p<0.001) and were also administered magnesium sulphate more frequently (44 vs. 22%, respectively; p<0.001) delivered at earlier gestation (37 vs. 38 weeks, respectively; p<0.001) required operative delivery more frequently (63 vs. 48%, respectively; p<0.001) and received more antihypertensive medications during the antenatal period (72 vs. 57%, respectively; p<0.001). Acute renal failure and acute pulmonary oedema were rare. About 4 cases of eclampsia all occurred in non-proteinuric women. The perinatal mortality rate was lower for the offspring of women with proteinuric pre-eclampsia compared with offspring of non-proteinuric women (13/1000 and 31/1000, respectively p = 0.006).

In this study, there was no association found between the number of complications between the groups with or without proteinuria (X² = 1.777; p = 0.777). Likewise, no difference was found between the severity of proteinuria and number of complications (X² = 11.852; p = 0754). The proportion of subjects experiencing or not experiencing complications in both groups was almost the same with the average of 1-2 complications. In fact, found the number of complications was slightly higher in the group without proteinuria (0.96 vs. 0.79) although, this was not statistically significant (p = 0.555).

CONCLUSION

Proteinuria is not absolutely required for the diagnosis of preeclampsia. This study concluded that proteinuria is not absolutely required for the diagnosis of preeclampsia. preeclampsia diagnosis may be established by the presence of hypertension as in association with thrombocytopenia (platelet count >100,000 µL) impaired liver function (elevated blood concentrations of liver transaminases to twice the normal concentration) the new development of renal insufficiency (serum creatinine concentration >1.1 mg dL⁻¹ or a doubling of the serum creatinine concentration in the absence of other renal disease) pulmonary edema or new-onset cerebral or visual disturbances.

REFERENCES


