Maternal and foetal outcomes in Pregnancy Induced Hypertension -A hospital based study.

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ABSTRACT: Introduction: Pregnancy induced hypertension (PIH) includes gestational hypertension, pre eclampsia and eclampsia. It is one of the leading cause of maternal and foetal morbidity and mortality. The study was undertaken to measure the risks and outcomes of PIH in mother and baby.

Materials and methods: A prospective study was conducted in 94 cases and 80 controls over a period of 2 years. The coagulation profile was carried out on the fully automatic STA COMPACT coagulometer and the patients were classified into pre eclamptic and eclamptic categories based on the clinical and hematological parameters.

Observations: The maternal and the foetal outcomes were studied and correlated with the coagulation profile. Maternal complications included HELLP seen in 10 patients, PPH in 8 cases, Infections in 3 cases, Ascites in 6 cases, Acute Renal Failure in 2 cases, Disseminated intravascular coagulation (DIC) in 3 cases and Maternal death in 2 cases. Foetal complications include IUGR seen in 18 cases and perinatal death in 27 cases. Out of 94 cases 62 had normal coagulation profile and 32 had deranged picture. Thus out of 32 patients with deranged coagulation profile, 27 (84.37%) women had adverse maternal outcome and 30 (93.75%) had unfavourable fetal outcome.

Conclusion: Thus suspecting a deranged coagulation status early in the course of the disease helps to plan preemptive management strategies that has been proven to have a crucial role in reducing the morbidity and mortality of both mother and fetus.

KEY WORDS: Coagulation, DIC, HELLP, IUGR, Mortality.

I. INTRODUCTION

1.1 Hypertensive disorders in pregnancy is one of the commonest medical disorders in pregnancy diagnosed by obstetricians in clinical practice and is one of the major causes of maternal & perinatal morbidity and mortality.[1]

1.2 Approximately 1,00,000 women die worldwide per annum because of eclampsia.[1] It is said that preeclampsia and eclampsia contribute to death of a woman every 3 minute worldwide.[2] Preeclampsia is a common dangerous condition for both mother and baby and is also predictable in onset and progression, cured only by termination of pregnancy.

1.3 In India – Gestational Hypertension continues to be responsible for the largest proportion of perinatal deaths resulting from prematurity and IUGR and is a major contributor to perinatal and maternal morbidity and mortality.[1] Majority of these conditions are preventable with good antenatal care, but looking at rural areas in country like India or many other Asian and sub-Saharan continents, the scene is still gloomy.
II. AIM AND OBJECTIVES
2.1 To correlate coagulation profile in patients of pregnancy induced hypertension with maternal and fetal morbidity and mortality.
2.2 To correlate the severity of pregnancy induced hypertension with the coagulation parameters.

III. MATERIALS AND METHODS
A prospective study titled ‘Coagulation profile in pregnancy induced hypertension’ was conducted at the Department of Pathology at Dr. Shankarrao Chavan Government Medical college & hospital, from January 2010 to June 2012.

3.1 Selection of control: Healthy normotensive pregnant females in the third trimester of pregnancy, without any signs and symptoms of pregnancy induced hypertension
3.2 Selection of cases: Pregnant females in the third trimester with symptoms and signs of pregnancy induced hypertension.

3.3 Exclusion Criteria:- In this study, following patients were excluded:
1) Hydatidiform mole
2) Twin pregnancy
3) Epilepsy
4) Chronic hypertension
5) Renal disorders

3.4 Total 174 cases were included in the study. The study groups were divided as follows:
A. Healthy normotensive pregnant controls- 80
B. Patients with preeclampsia- 58
C. Patients with eclampsia- 36

Detail history, important clinical findings and relevant investigations were noted as per the case proforma.

3.5 Whole blood sample was obtained by venepuncture of the Anterior cubital vein. The blood sample was obtained without a pressure cuff, allowing blood to enter the syringe by continuous free flow by the negative pressure from an evacuated tube. The 22 Gauge size needle and good quality 10 ml disposable plastic syringe was used for the collection of blood.

3.6 The hematological investigations were performed on a fully automated Orphee Mythic-18 three part differential cell counter. The coagulation parameters were carried on the STA COMAPCT fully automated coagulometer. All the details were recorded in the case proforma.

IV. RESULTS
There were total 174 subjects out of which 80(46%) were controls and 94(54%) were diagnosed PIH cases.

4.1 In controls, the maximum number were seen in the age group of 21-25 yrs i.e. 46 (57.5%) Mean age was found to be 24.41 yrs. Similarly maximum number of patients with pre eclampsia and eclampsia were in the age group of 21-25 years Mean age of patients with pre-eclampsia was 24.55 and that with eclampsia was 24.30 yrs.
4.2 Out of 80 controls, maximum i.e. 51 (63.75%) were primigravida. Out of total 94 cases, most of the cases i.e. 62 cases (66.50%) were primigravidae and 32 cases (33.50%) were multigravidae.
4.3 The mean gestational age in control was 37.30 wk with the range of 34-39 wks. While the mean gestational age in preeclampsia, and eclampsia were 34.03 wks and 32.38 wks respectively.
4.4 The mean blood pressure in controls was 127.05/82.35. In pre eclampsia it was 167.55/115.72 and that of eclampsia was 171.33/119.5 mm Hg.

Fig.1: Distribution of subjects.

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Table 1: Hemoglobin, platelet count and coagulation parameters in all groups.

<table>
<thead>
<tr>
<th>Tests</th>
<th>Control (n=80)</th>
<th>PE (n=58)</th>
<th>Eclampsia (n=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Hb (gm%)</td>
<td>10.03</td>
<td>9.08</td>
<td>9.85</td>
</tr>
<tr>
<td>Platelet count (lac/cmm)</td>
<td>2.42±0.62</td>
<td>1.60±0.51**</td>
<td>1.51±0.68**</td>
</tr>
<tr>
<td>Thrombocytopenia cases</td>
<td>--</td>
<td>17 (29.31%)</td>
<td>16 (44.44%)</td>
</tr>
<tr>
<td>BT (min)</td>
<td>2.43±0.21</td>
<td>2.66±1.35</td>
<td>2.70±1.44</td>
</tr>
<tr>
<td>Prolonged BT cases</td>
<td>--</td>
<td>4/6.89%</td>
<td>3/8.33%</td>
</tr>
<tr>
<td>CT (min)</td>
<td>5.28±0.91</td>
<td>5.60±1.06</td>
<td>5.65±1.18</td>
</tr>
<tr>
<td>Prolonged CT cases</td>
<td>--</td>
<td>4/6.89%</td>
<td>3/8.33%</td>
</tr>
<tr>
<td>PT (sec)</td>
<td>13.67±1.06</td>
<td>13.73±2.24</td>
<td>13.82±2.02</td>
</tr>
<tr>
<td>Prolonged PT cases</td>
<td>--</td>
<td>4/6.89%</td>
<td>3/8.33%</td>
</tr>
<tr>
<td>APTT (sec)</td>
<td>33.17±3.24</td>
<td>37.44±6.01**</td>
<td>37.69±5.61**</td>
</tr>
<tr>
<td>Prolonged APTT cases</td>
<td>--</td>
<td>19 (32.75%)</td>
<td>13 (36.11%)</td>
</tr>
</tbody>
</table>

†**- very significantly lower- P<0.01 †***-Very significantly higher- P<0.01

4.5 It has been seen that the platelet count in preeclampsia and eclampsia was very significantly lower than that in normal healthy pregnant controls. The mean platelet count in preeclampsia and eclampsia was 1.60± 0.51 lakh/cumm with P<0.001 and 1.51±0.68 lakh/cumm with P<0.001. Reduced platelet count was seen in 17 cases (29.31%) of preeclampsia and 16 cases (44.44%) of eclampsia.

4.6 It has been seen that prothrombin time was not significantly prolonged (P>0.05) in various severity of pregnancy induced hypertension. Prothrombin time was prolonged in 7 cases (4 of severe preeclampsia and 3 of eclampsia).

4.7 The mean activated partial thromboplastin time in preeclampsia was 37.44±6.60s with P< 0.001 which is significantly prolonged when compared with healthy controls. Similarly, in eclampsia the mean activated partial thromboplastin time was 37.69± 5.61s with P value < 0.001 which is again significantly prolonged as compared to controls. Activated partial thromboplastin time was prolonged in 19 cases (32.75%) of preeclampsia and 13 cases (36.11%) of eclampsia.

Table 2: Maternal outcome in patients with PIH (Total=94)

<table>
<thead>
<tr>
<th>Normal coagulation profile (n=62)</th>
<th>With deranged coagulation profile (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HELLP</td>
<td>0</td>
</tr>
<tr>
<td>PPH</td>
<td>3</td>
</tr>
<tr>
<td>Infection</td>
<td>1</td>
</tr>
<tr>
<td>Ascites</td>
<td>2</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>0</td>
</tr>
<tr>
<td>DIC</td>
<td>0</td>
</tr>
<tr>
<td>Maternal Death</td>
<td>1</td>
</tr>
</tbody>
</table>

4.8 Out of 32 patients with deranged coagulation profile, 27 (84.37%) women had adverse outcome while remaining 5 (15.62%) women had normal outcome.

4.9 Out of 62 cases with normal coagulation profile, only 7 (11.29%) women had unfavorable outcome while 55 (88.71%) women had normal course.

4.10 Chi square value is 48.83 i.e. P < 0.0001, with df=1 which suggests that derangement in coagulation profile is statistically very significantly associated with maternal morbidity and mortality.

Table 3: Fetal outcome in patients with PIH(Total=94)

<table>
<thead>
<tr>
<th>Normal coagulation profile (n=62)</th>
<th>With deranged coagulation profile (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perinatal death</td>
<td>11</td>
</tr>
<tr>
<td>IUGR</td>
<td>4</td>
</tr>
</tbody>
</table>

4.11 Out of 32 cases with deranged coagulation profile, 30 women (93.75%) had unfavourable fetal outcome while out of 62 women with normal coagulation profile, 15 (24.19%) had unfavourable fetal outcome.

4.12 Chi square value is 40.92 i.e P<0.0001, with df=1, which suggests that derangement in coagulation profile is very significantly associated with fetal morbidity and mortality.
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V. DISCUSSION

5.1 The incidence of HELLP syndrome in cases of PIH (n=94) found in present study was 10.63%. The values of present study are consistent with that of Sibai (1990)[3] who reported an incidence of 9.8% and J.Prakash (2006)[4] reported a rate in the range of 2-12%.

5.2 The incidence of Post Partum Hemorrhage in PIH (n=94) is found to be 8.5% in our study. There were total of 8 cases, out of which 5 were found in severe pre eclampsia and 3 were found in eclampsia. The incidence rate correlates with 10.20% for Ludec et al (1992)[5].

5.3 Acute Renal Failure was found in only 2 cases of pre eclampsia. No case was found in eclampsia. Its incidence was found to be 2.12% in present study

5.4 The incidence of Disseminated Intravascular Coagulation incidence rate found in our study was 3.19% which is consistent with 3% conducted by Ludec et al (1992)[5].

5.5 Sepsis was found in 3 cases of PIH comprising of 3.1%. Ascites as a complication of PIH is found in 6 cases

5.6 The incidence of maternal mortality in our study was 2.12% which well co-relates with that conducted by Hagragi (2006)[6] was found to be 2.3%.

5.7 The incidence of IUGR in pre eclamptic patients in our study was found to be 19.14% which was comparable to Ludec et al (1992)[5] 21% and Samantha et al (2006)[7] 15.5%.

5.8 The incidence of perinatal mortality in severe pre eclampsia patients in our study was 28.72% which is comparable to Gaddi et al (2001)[8] 22.3%.

5.9 Leduc et al (1992)[5] reported significant association between thrombocytopenia and maternal complications and reported that platelet nadir is the best predictor of maternal outcome.

5.10 P.W. Howie et al (1976)[9] reported a significant correlation between coagulation factors and fetal outcome. He and his co-workers came to the conclusion that the coagulation indices help to predict the severity of fetal morbidity and hence helps to take appropriate measures before the stage of irreversibility arises.

VI. VI CONCLUSION

6.1 The hypertensive diseases complicating pregnancy still remains the major problem in developing countries. The fact that pregnancy induced hypertension is largely a preventable condition is established by observing the negligible incidence of pre-eclampsia and eclampsia with the institution of early management.

6.2 The early detection of compromised status combined with the institution of prompt treatment has been proven to have a crucial and definite role in reducing the morbidity and mortality of both mother and fetus.

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