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Pathology

COAGULATION PROFILE IN PREECLAMPSIA AND ECLAMPSIA

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ABSTRACT Pregnancy induced hypertension (PIH) is hypertension that develops as the direct result of gravid state.1,2 There is distinct possibility of accentuation of hypercoagulable state of pregnancy during eclampsia and preeclampsia and it contributes to death of woman every 3 minutes worldwide.3,4,5 Deranged coagulation profile in PIH is a significant predictor of increased adverse maternal & perinatal outcome. Such cases should be thoroughly investigated and followed closely to prevent complications. Coagulation profile can be assessed by tests such as prothrombin time (PT), activated partial thromboplastin time (APTT) & platelet count.4,6,7

KEYWORDS : PIH, Eclampsia, Preeclampsia, Platelet count, Coagulation profile

INTRODUCTION

Over half a million women die each year from pregnancy related causes.[8,9,10] In many low-income countries complications of pregnancy and childbirth are the leading cause of death amongst women of reproductive years.[6,11]

Normal pregnancy is associated with remarkable changes in uteroplacental intravascular coagulation mechanism leading to a hypercoagulable state and suppression of fibrinolysis.[12]

Pregnancy –induced hypertension includes incidence of HTN > 20 weeks of pregnancy.

American College of Obstetrics and Gynecology Classifications for Hypertensive Disorders of Pregnancy.[3,11,13]

1. **Preeclampsia:**

a) Mild pre-eclampsia- BP between 140/90 to <160/110 mm Hg without proteinuria.
b) Severe pre-eclampsia- BP >160/110 mmHg proteinuria, 5 gm/24 hours (>3+ on dipstick) and presence of headache, visual disturbances, oliguria and thrombocytopenia.

2. **Eclampsia-** Criteria of pre-eclampsia with convulsions that cannot be attributed to another cause.

Due to low socioeconomic status, apathetic attitude, poor health education and lack of regular antenatal supervision the incidents of preeclampsia are more in developing countries like India.[6,14,15]

Coagulation profile can be assessed by tests such as Prothrombin time (PT), Activated partial thromboplastin time (APTT) and platelet count.[4,6,7]

Early identification of such high-risk women and monitoring derangements in their coagulation system are surely pivotal in the prevention of complications.

Aims & Objective

a. To study the coagulation profile in pregnancy induced hypertension (preeclampsia and eclampsia) and normotensive pregnancies.
b. To establish the relationship of coagulation parameters (PT, APTT and D-dimer) and platelet count with severity of pregnancy induced hypertension.

MATERIAL AND METHODS

This two years record based, descriptive, cross sectional and observational study was carried out in Pathology Department. Coagulation profile was done on patients admitted in Department of obstetrics and gynecology at a tertiary care center, from December 2020 to September 2022.

The study group was divided into 4 groups.

a) Healthy normotensive pregnant controls- 351 **b)** Women with mild preeclampsia- 307 **c)** Women with severe preeclampsia- 254 **d)** Women with eclampsia- 219

Exclusion Criteria:

1. Known case of hypertension and on anticoagulation therapy.
2. Incidence of HTN < 20 weeks.
3. Patients with history of known bleeding disorders.
4. Hydatidiform mole.
5. Epilepsy.

Detailed history, important clinical findings such as blood pressure, proteinuria were noted.

Analysis of the collected samples was done on fully automated 5 part hematological analyzer and fully automated coagulation analyzer.

RESULT & DISCUSSION

Total 780 cases of pregnancy induced hypertension and 351 controls were included in the study.

Age Distribution: Maximum number of patients was seen in age group of 20-24yrs in all three groups. This finding was in agreement with Chaware S A et al [16]. PIH occurring in younger age signify to early age of marriage and first pregnancy in developing countries.[6]

Parity Wise Distribution: In present study, 68.08% of mild preeclampsia, 62.20% of severe preeclampsia & 66.54% of eclamptic patients were primigravidae.Our study was in concordance with Suresh Arjunrao Chaware et al[4] and Priya MJ et al[17].

Gestational Age: The mean gestational age in healthy pregnant control was 34.69 + 3.01 wks. While the mean gestational age in mild preeclampsia, severe preeclampsia, and eclampsia was 34.79 + 2.57 wks, 33.85 + 3.16 wks and 33.17 + 2.85 wks respectively. Thakur Bhava na et al[18] reported mean gestational age of 36.36 wks, 34.41wks & 32.38 wks in mild preeclampsia, severe preeclampsia & eclampsia respectively.

Clinical Features:

a. Blood Pressure (BP):

The BP in mild preeclampsia, severe preeclampsia & eclampsia were 146.38/96.76, 165.98/112.89 & 169.82/110.23 respectively. The BP in different groups of our study is well correlating with Chaware S A et al[16] and Naveed Tamboli et al[19].

b. Edema:

In present study, pathological edema in mild (4.56%), severe preeclampsia (11.42%) and eclampsia (18.72%) was correlated with Naveed Tamboli et al[19].

c. Proteinuria:

Proteinuria is an important indicator of severity because it usually develops late in the course of disease.[16]

In present study, the significant proteinuria (+2 to +4) was seen only in 109 cases (35.56%) of mild preeclampsia, 188 cases (74.02%) of severe preeclampsia & 198 (90.41%) cases of eclampsia. Our finding was correlated with Suresh Chaware et al[4] and Naveed Tamboli et al[19].

Coagulation Profile In Women With Preeclampsia And Eclampsia

It has been seen that platelet count in all three groups of PIH was significantly lower than that of normal pregnant control (P<0.05).

It has been seen that prothrombin time and activated partial thromboplastin time were significantly prolonged (P<0.05) in all three groups of PIH as compared to normotensive pregnant controls.

I. Platelet count:

Table 1: Comparison of mean platelet count in preeclampsia and eclampsia as compared to controls with other studies.

Authors	Normal pregnant control (lac/cumm)	Mild preeclampsia (lac/cumm)	Severe preeclampsia (lac/cumm)	Eclampsia (lac/cumm)
Priyamvada Singhal et al (2019)[20]	2.24	2.14	1.77	1.43
Naveed Tamboli (2021)[19]	2.38	2.25	1.75	1.44
Present study (2022)	2.48	2.24	1.74	1.01

Reduced platelet count occurs due to consumption of intravascular coagulation.[16]

II. Prothrombin time (PT):

Table 2: Comparison of mean prothrombin time (sec) as compared to controls with other studies

Authors	Haldar B et al (2020)[21]	Naveed Tamboli et al (2021)[19]	Present study (2022)
Control	13.8 ± 1.10	13.27±0.8 1	13.12 ± 0.96
Mild preeclampsia	14.81 ± 1.02	13.3±0.86	13.95 ±1.59
Severe preeclampsia	15.75 ± 1.61	13.30± 2.65	15.97 ± 2.93
Eclampsia	16.73 ± 3.14	13.40± 2.13	15.94 ± 3.05

The mean prothrombin time was increased in severe preeclampsia and eclampsia.

III. Activated partial thromboplastin time (APTT):

Table 3: Comparison of mean APTT (sec) as compared to controls with other studies

Authors	Thakur Bhavana et al (2018)[18]	Naveed Tamboli et al (2021)[19]	Present study (2022)
Control	30.54 ± 1.35	31.61±4.06	31.88 ± 3.47
Mild preeclampsia	32.08 ± 2.93	31.87±3.80	33.68 ± 7.16
Severe preeclampsia	34.73 ± 5.72	36.44± 13.15	39.46 ± 7.32
Eclampsia	35.67 ± 6.66	41.15± 9.37	44.53 ± 13.60

Mean APTT was increased in severe preeclampsia, eclampsia.

IV.D-dimer:

In our study plasma levels of D-dimer was raised in 25.73% cases of mild preeclampsia, 52.36% cases of severe preeclampsia and 55.71% cases of eclampsia while none of the healthy pregnant control showed raised D-dimer levels. Our finding was in agreement with Naveed Tamboli et al.[19]

Coagulation profile and Maternal & fetal complications:

Out of 254 cases of severe preeclampsia, 138 cases showed maternal and fetal complications and all of them showed deranged coagulation profile.

Maternal complications in patients of severe preeclampsia with deranged coagulation profile were abruptio placentae (7.48%) followed by post-partum haemorrhage (5.90%), acute renal failure (5.12%), HELLP syndrome (4.72%), pulmonary edema (2.76%) and decompensated DIC (1.97%).

Out of 138 cases with deranged coagulation profile, fetal complications were including prematurity (40.56%), IUGR (22.44%) and perinatal death (11.42%).

Out of 219 cases of eclampsia, 120 cases showed maternal and fetal complications and all of them showed deranged coagulation profile. Out of 219 cases of eclampsia status epilepticus were seen in 59 (26.96%) cases. Patients of eclampsia with deranged coagulation profile had HELLP syndrome (10.96%), abruptio placentae (10.50%), pulmonary complications (10.05%) (includes pulmonary edema in 11 cases, acute respiratory distress syndrome in 8 cases and pulmonary embolism in 3 cases), acute renal failure (7.76%), DIC (5,93%), postpartum haemorrhage (3.65%) and cerebrovascular accident (3.65%).

There was poor fetal outcome in cases of eclampsia with deranged coagulation profile. Prematurity was seen in 26.03%, intrauterine growth retardation was seen in 34.25% and perinatal death was seen in 24.65% in deranged coagulation profile.

CONCLUSION

Preeclampsia and eclampsia have remained a leading cause of maternal mortality and being higher in developing countries due to illiteracy, poor antenatal care and poverty.

Changes in haemostasis and coagulation cannot be assessed by a single test. Individual test results can vary from case to case. Hence combination of tests is needed. Platelet count, prothrombin time, activated partial thromboplastin time and D-dimer play important role in diagnosis of pregnancy induced hypertension. Raised APTT and D-dimer are fairly good indicators of preeclampsia, eclampsia and needs aggressive treatment.

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