

Frequency of Deranged Coagulation Profile in Patients having Pregnancy Induced Hypertension

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Abstract.....Object: is to evaluate the frequency of deranged coagulation profile in patients having pregnancy induced hypertension. **Methodology:** A total number of 540 patients were involved in the study. Sample was collected with the help of non-probability consecutive sampling technique. Patients were divided into three groups, pregnancy induced hypertension, preeclampsia and eclampsia. Following variables were calculated and assessed, age, gravidity. Parity, gestational age, diagnosis of eclampsia or preeclampsia, platelet count, prothrombin time, activated partial thromboplastin time, serum fibrinogen and serum albumin. **Results:** Frequency of low platelet count, low serum fibrinogen level, prolonged PT and prolonged aPTT in PIH group was 45.7%, 4.3%, 2.9% and 2.9% respectively. Frequency of low platelet count, low serum fibrinogen level, prolonged PT and prolonged aPTT in preeclampsia group was 39.4%, 8.7%, 5.1% and 4.2% respectively. Frequency of low platelet count, low serum fibrinogen level, prolonged PT and prolonged aPTT in eclampsia was 52.8%, 12%, 7.5% and 5.1% respectively. **Conclusion:** This study concludes that derangement in clotting profile occurred in certain percentage of patients suffering from preeclampsia and frequency of derangement was significant

Keywords: Coagulation Profile, Pre-eclampsia, Eclampsia, Pregnancy, Hypertension

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INTRODUCTION

Preeclampsia and eclampsia are major health problems occurring during pregnancy and are most common hypertensive disorders along with pregnancy induced hypertension¹. These disorders are reported to cause complications in 3 to 8 percent pregnancies in terms of maternal morbidity and mortality hypertension. It poses great threat not only to the mother but the baby as well. Coagulations profile is diagnostic of any changes in blood hemostasis³. In coagulation profile of an individual activated partial thromboplastin time (aPTT), prothrombin time⁴, full blood count, platelet count, thrombin time, fibrin degradation products, Fibrinogen and D-dimer are assessed to check for any coagulation disorders present in human blood⁵.

Pathophysiology of preeclampsia is not known yet. Underlying mechanism has been stated as, decreased invasion of trophoblastic cells of maternal vascular bed which initiated the development of preeclampsia as a result of abnormal immunological interactions^{6, 7, 8}. Resulting placental hypo perfusion causes endothelial damage along with higher vascular permeability.

In kidneys glomeruloendotheliosis occurs, a specific finding in preeclampsia⁹. It is associated with loss of proteins in urine of the women with preeclampsia.

Very little data has been found regarding evaluation of frequency of coagulation derangements among the patients presenting as cases of preeclampsia or eclampsia. In this study focus will be on the coagulation profiles of the women presenting in gynecological emergency. It will help clinicians in determining the possible coagulation related complication in hypertensive pregnant women.

Methodology

This study was conducted in department of gynecology and obstetrics Nishtar Hospital Multan from July 2016 to December 2017. It is a descriptive cross sectional study. A total number of 540 patients were involved in the study and sample size was calculated by an online computer software openepi.com using 95% confidence interval and 80% power o study. All they were presented in department of Gynecology and Obstetrics Nishtar Hospital Multan. Reference for our study was taken from a previous study¹⁰. Sample was collected with the help of non-probability consecutive sampling technique and all the patients were involved in the study after taking informed consent. Ethical approval for our study was taken from Hospital Ethics Committee. Inclusion criteria were set as; patients diagnosed with the help of history, clinical examination and investigations as confirmed cases of preeclampsia and eclampsia were involved in the study.

Patients diagnosed as anti-phospholipid syndrome and any acute or chronic liver disease or cases of congenital blood clotting disorder were excluded from the study after thorough evaluation of clinical and laboratory findings. Pregnant women with history of grand multiparity, past history of post partum hemorrhage, multiple pregnancies and hydromnias were also excluded from the study. Patients were divided into three groups, pregnancy induced hypertension, preeclampsia and eclampsia. Pregnancy induced hypertension was defined as systolic blood pressure of greater than 130mmHg while eclampsia as systolic blood pressure greater than 14mmHg. Normal platelet count was taken as more than $150000/\text{mm}^3$. Data thus obtained was subjected to statistical analysis which was performed by a computer software SPSS version 23. Frequency and percentage was calculated for qualitative variables while mean and standard deviation was calculated for quantitative variables. Chi square test was applied to assess the association among different variables and p value less than or equal to 0.05 was taken as significant.

Results

A total number of n=540 patients were enrolled in this study. This study was further divided into three groups i.e. 40% (n=216) pregnancy induced hypertension (PIH), 47% (n=254) preeclampsia and 13% (n=70) eclampsia. The mean age, gravidity, parity, gestational age, TLC and Hb of the PIH patients was 26.50 ± 3.52 years, 2.74 ± 1.20 , 1.36 ± 0.86 , 29.50 ± 4.85 weeks, $11000/\text{mm}^3$ and 9.48 ± 2.03 respectively. The mean age, gravidity, parity, gestational age, TLC and Hb of the preeclampsia patients was 27.25 ± 4.09 years, 2.68 ± 1.13 , 1.28 ± 0.87 , 30.33 ± 4.20 weeks, $10500/\text{mm}^3$ and 8.41 ± 1.85 respectively. While, the mean age, gravidity, parity, gestational age, TLC and Hb of the eclampsia patients was 28.37 ± 2.82 years, 2.80 ± 1.11 , 1.24 ± 0.95 , 28.98 ± 2.21 weeks, $10000/\text{mm}^3$ and 8.73 ± 0.85 respectively. The differences were statistically significant of age ($p=0.001$), gestational age (0.024), TLC ($p=0.000$) and Hb ($p=0.000$) with regard to pregnancy induced hypertension, preeclampsia and eclampsia (Table. I).

In group of patients with PIH platelet count, PT, aPTT, serum fibrinogen, serum albumin was 215.94 ± 60.45 $10^6/\text{mm}^3$, 12.46 ± 0.48 seconds, 31.69 ± 1.43 seconds, 422.88 ± 12.13 mg/dl and 2.8 ± 1.16 mg/dl respectively.

In preeclampsia group, platelet count, PT, aPTT, serum fibrinogen, serum albumin was 214.71 ± 82.11 $10^6/\text{mm}^3$, 11.98 ± 0.39 seconds, 31.35 ± 1.34 seconds, 420.25 ± 12.23 mg/dl and 2.82 ± 1.1 mg/dl respectively. In eclampsia group, platelet count, PT, aPTT, serum fibrinogen, serum albumin was 212.38 ± 47.38 $10^6/\text{mm}^3$, 11.48 ± 0.29 seconds, 31.04 ± 1.11 seconds, 417.21 ± 11.18 mg/dl and 2.54 ± 0.48 mg/dl respectively. Frequency of low platelet count, low serum fibrinogen level,

prolonged PT and prolonged aPTT in PIH group was 45.7%, 4.3%, 2.9% and 2.9% respectively. Frequency of low platelet count, low serum fibrinogen level, prolonged PT and prolonged aPTT in preeclampsia group was 39.4%, 8.7%, 5.1% and 4.2% respectively. Frequency of low platelet count, low serum fibrinogen level, prolonged PT and prolonged aPTT in eclampsia was 52.8%, 12%, 7.5% and 5.1% respectively (Table II).

Discussion

In this studied we assessed frequency of derangements in coagulation profile of women suffering from hypertensive disorder of preeclampsia and eclampsia and result showed that very little frequency of patients had deranged coagulation profile. In these patients platelet count was affected the most and 44.5 percent women had platelet count less than $150000/\mu\text{mol}$. No patient had low fibrinogen level or prolonged PT or APTT with normal platelet count. These results have shown that derangements do occur in conditions of preeclampsia and eclampsia. In PIMS Islamabad a study was conducted to assess the frequency of disseminated intravascular coagulation and they reported its prevalence to be 15% in patients with ante-partum hemorrhage¹². In an another study it was reported that mild preeclampsia does not affect coagulation profile or platelet count significantly as compared to normal pregnant women, where as severe preeclampsia and eclampsia impart their effect on coagulation profile and platelet count in the form of intravascular coagulation. HELLP (Hemolysis, Elevated Liver enzymes and Low platelet count) syndrome is a well known complication of severe preeclampsia and eclampsia¹³. Total platelet count in maternal blood and aPTT and is of diagnostic value in women suffering from severe pregnancy induced hypertension i.e. preeclampsia/eclampsia. These two tests can help in improving the early and timely diagnosis of coagulation profile derangement in patients of preeclampsia and eclampsia. This will ultimately prove helpful for the obstetricians by helping them prevent fatal disseminated intravascular coagulation¹⁴. A study conducted in recent years compared a control group with preeclampsia and eclampsia groups and results showed that BT (bleeding time), PT (prothrombin time), aPTT (activated partial thromboplastin time) and CT (clotting time) measurements were almost similar in all groups. But in those results platelet count showed significant lowering in number with a trend from normal to mild preeclampsia to severe preeclampsia and eclampsia. They also studied the level of D-Dimer and results showed that D dimer was raised in pregnancy induced hypertensive conditions with normal values in control group and very high values in eclampsia group¹⁵. Literature has showed that pregnancy can result in mild to moderate derangement in coagulation profile thus resulting in changes in haemostasis of pregnant -----

Table-1: Study Population Demographics Characteristics (N=540)

Characteristics	PIH* 40% (N=216)	Preeclampsia 47% (n=254)	Eclampsia 13% (n=70)	P Value
Age (years)	26.50±3.52	27.25±4.09 years	28.37±2.82	p=0.01
Gravidity	2.74±1.20	2.68±1.13	2.80±1.11	p=0.697
Parity	1.36±0.86	1.28±0.87	1.24±0.95	p=0.522
gestational age	29.50±4.85	30.33±4.20	28.98±2.21	p=0.024
TLC*	11000/m ³	10500/mm ³	10000/m ³	p=0.000
Hb*	9.48±2.03	8.41±1.85	8.73±0.85	p=0.000

Table-II: Hematological Parameters in different Hypertensive Disorders (N=540)

Characteristics	PIH* 40% (N=216)	Preeclampsia 47% (n=254)	Eclampsia 13% (n=70)	P Value
Platelet count	215.94±60.45	214.71±82.11	212.38±47.38	p=0.901
PT*	12.46±0.48	11.98±0.39	11.48±0.29	p=0.000
aPTT*	31.69±1.43	31.35±1.34	31.04±1.11	p=0.001
Serum fibrinogen	422.88±12.13	420.25±12.23	417.21±11.18	p=0.002
Serum albumin	2.8±1.16	2.82±1.1	2.54±.48	p=0.139
Low platelet count	45.7% (n=32)	39.4% (n=100)	52.8% (n=114)	p=0.015
Low fibrinogen level	4.3% (n=3)	8.7% (n=22)	12% (n=26)	p=0.131
Prolonged PT*	2.9% (n=2)	5.1% (n=11)	7.5% (n=19)	p=0.279
Prolonged aPTT*	2.9% (n=2)	4.2% (n=9)	5.1% (n=13)	p=0.696

females especially at the time of delivery. These changes involve increase in majority of clotting factors, decrease in overall platelet count and serum fibrinogen levels and decrease in efficacy of natural anticoagulants. The reason for low platelet count during pregnancy can be explained by two possible condition namely, gestational thrombocytopenia and idiopathic thrombocytopenic

purpura (ITP). Normally despite the presence of low platelet count in gestational thrombocytopenia, haemostasis remains normal and also in most cases of idiopathic thrombocytopenic purpura^{16,17}.

Multiple studies have been done regarding frequency of low platelet count and low platelet volume in patients suffering from pregnancy induced hypertensive disorders of preeclampsia and eclampsia. Ratio between platelet count and platelet volume is also reported to be low in preeclampsia patients as compared to the normal patients¹⁸.

Conclusion

This study concludes that derangement in clotting profile occurred in certain percentage of patients suffering from preeclampsia and frequency of derangement was significant.

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