

Original Research Paper

Comparison of Haematological Parameters in Normal and Pregnancy Induced Hypertension in a Tertiary Care Teaching Hospital

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ABSTRACT:

Background: Women undergo a physiological process called pregnancy, which comes with some health complications for both the mother and the unborn child. ^[1] Hypertension is one significant problem affecting 7% - 10% of pregnancies and causes maternal and neonatal death and morbidity. ^[1] Approximately 5% – 7% of pregnancies are affected with PIH, while the incidence ranges from 5% to 15% in India. ^[2]

Objectives: To study and compare the haematological parameters in PIH patients to normotensive pregnant women.

Material & Methods: Study Design: Hospital based observational study. **Study area:** Department of Physiology and Obstetrics and Gynaecology, Government medical college. Kadapa, Andhra Pradesh. **Study Period:** 1 year. **Study population:** Pregnancy induced hypertensive pregnant women and normotensive pregnant women visiting the Obstetrics and Gynaecology department. **Study tools and Data collection procedure:** Cases comprised of Primi-gravida at 3rd trimester, with known cases of Pregnancy Induced Hypertension admitted in hospital for safe institutional delivery. The control group are the women primi-gravida who have attended the regular O.P. and I.P. Five ml of blood anti-coagulated with EDTA was collected and various haematological parameters were studied. These included Hemoglobin, total and differential counts, Platelet count, red cell indices like PCV, MCV, MCH, MCHC and BT, CT, Prothrombin and APTT.

Results: The mean for platelet count in control group was 2.21 and study group was 1.67. The estimated P value for platelet count in control group and study group was <0.001. The mean for APTT in control group was 25.33 and study group was 27.2. The estimated P value for APTT in control group and study group was <0.001. Thus there was significant decrease in platelet count in study group compared to control group.

Conclusion: Using the absolute platelet count as a gauge, we can evaluate pregnancy-induced hypertension. Therefore, it is important to detect the illness early, stop it from becoming worse and developing into eclampsia, and lower foetal morbidity and mortality.

Keywords: Pregnancy-induced hypertension, Platelet count, Prothrombin Time

INTRODUCTION:

Women undergo a physiological process called pregnancy, which comes with some health complications for both the mother and the unborn child. ^[1] Hypertension is one significant problem affecting 7% - 10% of pregnancies and causes maternal and neonatal death and morbidity. ^[1] Approximately 5%–7% of pregnancies are affected with PIH, while the incidence ranges from 5% to 15% in India. ^[2] Along with sepsis and hemorrhage, the hypertensive diseases that complicate pregnancy make up one of the fatal causes of maternal death.

One of the major illnesses related to high blood pressure is pregnancy-induced hypertension (PIH), which emerges for the first time after 20 weeks of gestation and goes away after birth ^[3] Compared to multiparous women, primigravida women are more likely to experience it. ^[4] Eclampsia is a severe form of pregnancy-induced hypertension accompanied by seizures ^[5].

The National High Blood Pressure Education Programme Working Group on High Blood Pressure in Pregnancy recommends categorising PIH into four groups. ^[6] Preeclampsia, Eclampsia, Chronic Hypertension of Any Aetiology, and Gestational Hypertension are a few examples of hypertension. After 20 weeks of pregnancy, preeclampsia is defined as having a blood pressure (BP) reading of 140/90 or above, recorded at least twice, 2 hours apart, and having proteinuria (defined as 300 mg or more of urinary protein in 24 hours or 100 mg/dl or more in at least two random urine samples obtained at least 6 hours apart). ^[7] Due to increased consumption during low-grade intravascular coagulation, thrombocytopenia is the most prevalent aberration seen in PIH among the range of haematological alterations ^[8,9].

Severe preeclampsia is defined as a BP of 160/110 or higher measured on two separate occasions at least 6 hours apart, proteinuria of at least 5 g in 24 hours, oliguria of less than 400 ml in 24 hours, or cerebral or visual disturbances. Preeclampsia conventional clinical symptoms include hypertension, proteinuria, excessive weight gain, and oedema. ^[10]

Thrombocytopenia, anaemia, hemoconcentration, hyperuricemia, and abnormal liver function tests are further characteristics.^{10,11} PIH may exhibit a variety of haematological symptoms, from normal laboratory results to severe anaemia and thrombocytopenia (caused by platelet activation and consumption). Preeclampsia is characterised by elevated hematocrit levels and hemoconcentration. A decrease in the recurring hematocrit levels could signify clinical advancement.¹²

Hence the present study was undertaken to study and compare the hemoglobin, hematocrit, WBC (white blood cell), lymphocytes and platelet counts in PIH patients to normal patients.

OBJECTIVES: To study and compare the haematological parameters in PIH patients to normotensive pregnant women.

MATERIAL & METHODS:

Study Design: Hospital based observational study.

Study area: Department of Physiology and Obstetrics and Gynaecology, Government medical college, Kadapa, Andhra Pradesh.

Study Period: 1 year.

Study population: Pregnancy induced hypertensive pregnant women and normotensive pregnant women visiting the Obstetrics and Gynaecology department.

Sample size: study consisted a total of 60 subjects. (30 cases and 30 controls)

Sampling method: Simple random method

Inclusion criteria:

Control Group: -

1. In the age group of 20 -25 years.
2. All are primi -gravida.

For study group: -

1. In the age group of 20 -25 years.
2. All are primi -gravida with known case of pregnancy induced hypertension.

Exclusion criteria:

1. Known case of Hypertension.
2. Known case of Diabetes.
3. Known case of Asthma
4. Known case of Epilepsy

Ethical consideration: Institutional Ethical committee permission was taken prior to the commencement of the study.

Study tools and Data collection procedure:

Cases comprised of Primi-gravida at 3 rd trimester, with known cases of Pregnancy Induced Hypertension admitted in hospital for safe institutional delivery. The control group are the women primi-gravida who have attended the regular O.P. and I.P. Five ml of blood anti-coagulated with EDTA was collected and various haematological parameters were studied. These included Hemoglobin, total and differential counts, Platelet count, red cell indices like PCV, MCV, MCH, MCHC and BT, CT, Prothrombin and APTT. Maternal details like age, parity, immunization status gestation age, onset of symptoms, blood pressure recordings and history of any seizures were noted. Before taking the sample, the women were spoken to about the procedure and given the opportunity to give their verbal informed permission. Every patient's pertinent clinical information, including a thorough medical history, general, systemic, and obstetric examinations, baseline investigations, and ultrasound, was gathered in order to evaluate the study's findings.

The statistics used to analyze the data was mean, standard deviation and student t- test. For all analytical tests the level of significance was $p < 0.05$.

OBSERVATIONS & RESULTS:

In our study a 60 cases were included, where 30 cases were study group and 30 were control group.

Table 1: Comparison of Hb in control group and study group

Hb	CONTROL GROUP	STUDY GROUP -2
Mean	10.6	10.89
SD	1.09	1.35
t-Value	0.71	

p-Value	>0.05	
Result	Not Significant	

There is no significance for Hb% in our study.

Table 2: Comparison of Platelet counts in control group and study group

PT	CONTROL GROUP	STUDY GROUP
Mean	2.21	1.67
SD	0.13	0.32
t-Value	7.85	
p-Value	<0.001	
Result	Significant	

The mean for platelet count in control group was 2.21 and study group was 1.67. The estimated P value for platelet count in control group and study group was <0.001.

Table 3: Comparison of BT in control group and study group

BT	CONTROL GROUP	STUDY GROUP
Mean	1.41	1.39
SD	0.28	0.19
t-Value	0.23	
p-Value	>0.05	
Result	Not Significant	

There is no significance for BT in our study.

Table 4: Comparison of CT in control group and study group

CT	CONTROL GROUP	STUDY GROUP
Mean	3.46	3.47
SD	0.36	0.25
t-Value	0.43	
p-Value	>0.05	
Result	Not Significant	

There is no significance CT in our study.

Table 5: Comparison of Prothrombin Time in control group and study group

PT	CONTROL GROUP	STUDY GROUP
Mean	14.7	15.86
SD	0.65	1.12
t-Value	3.71	
p-Value	<0.001	
Result	Significant	

The mean for Prothrombin time in control group was 14.7 and study group was 15.86. The estimated P value for Prothrombin time in control group and study group was <0.001. Thus there was significant decrease in platelet count in study group compared to control group.

Table 6: Comparison of APTT in control group and study group

APTT	CONTROL GROUP	STUDY GROUP
Mean	25.33	27.2
SD	0.55	1.57
t-Value	5.94	
p-Value	<0.001	
Result	Significant	

The mean for APTT in control group was 25.33 and study group was 27.2. The estimated P value for APTT in control group and study group was <0.001. Thus there was significant decrease in platelet count in study group compared to control group.

Table 7: Comparison of INR OF PT in control group and study group

INR OF PT	CONTROL GROUP	STUDY GROUP
MEAN	1.0744	1.266
SD	0.083912	0.152703

t-Value	-4.26	
p-Value	<0.001	
Result	Significant	

The mean for INR of PT in control group was 1.0744 and study group was 1.266. The estimated P value for Prothrombin time in control group and study group was <0.001. Thus there is significant decrease in platelet count in study group compared to control group.

Table 8: Comparison of INR OF APTT in control group and study group

INR OF APTT	CONTROL GROUP	STUDY GROUP
MEAN	1.056467	1.191733
SD	0.087562	0.0012924
t-Value	3.6	
p-Value	0.001	
Result	Significant	

The mean for INR of APTT in control group was 1.0564 and study group was 1.191. The estimated P value for Prothrombin time in control group and study group was 0.001. Thus there was significant decrease in platelet count in study group compared to control group.

DISCUSSION:

One of the main causes of maternal and foetal morbidity and mortality is pre-eclampsia. Even after decades of ongoing research, the cause of PIH is still unknown. Recent research indicates that there may be a number of underlying factors or predispositions that contribute to endothelial dysfunction and generate the indications of hypertension, proteinuria, and edema that are all used to diagnose the pre-eclampsia syndrome.¹⁴ The goal of the current study was to assess haematological variables in preeclampsia.

Thrombocytopenia is the most frequent anomaly seen in patients with pregnancy-induced hypertension out of all the haematological alterations that might occur. Due to hemodilution, increased platelet consumption, and elevated levels of thromboxane A2, thrombocytopenia is frequently seen in the third trimester. Endothelial dysfunction in pre-eclampsia causes changes in fibrinogen levels, the activated partial thromboplastin time, the prothrombin time, fibrin degradation products, and D-dimers. According to the current study, blood coagulopathy is linked to several complex changes during pregnancy that are not fully understood.^[1]

In our study, the pre-eclampsia group's platelet counts significantly decreased when compared to the control group. Our research shows that a low platelet count is particularly noticeable during the third trimester of pregnancy. Pregnant women with PIH may have a change in platelet count because of increased consumption from a shorter life expectancy and increased aggregation from higher amounts of thromboxane A2 in placental circulation.¹⁵

In a study conducted in 2014 by Subbalakshmi N.K. *et al.*, it was found that preeclampsia patients had considerably lower PCV, haemoglobin, and platelet concentrations than women who were pregnant normally.¹⁶ This retrospective analysis of 75 singleton women who had PIH included singleton mothers. Our study also shows low haemoglobin and platelets level in PIH patient.

In their study, Sharma *et al.* found that the mean platelet count fell from 2.05 lakhs/cumm in instances of moderate preeclampsia to 1.32 lakhs/cumm in situations of severe preeclampsia and to 1.03 lakhs/cumm in cases of eclampsia. As preeclampsia severity increased, the platelet counts sharply decreased^[17]. In a research by Sultan R *et al.*, the average platelet count in patients and controls was 1.44 lakhs per cubic millimetre and 1.98 lakhs per cubic millimetre, respectively. According to the study, pre-eclampsia and low platelet counts are related^[18].

The study showed increased prothrombin time (sec) in the cases group is 15.9(sec) as compared to the 14.8(sec) control group. Also there was an increase in Activated partial thromboplastin time (sec) in cases group is 27.2 (sec) as compared to 25.3(sec) control group. The difference was statistically significant.

The results were consistent with a research by Lakshmi *et al.*, which found that severe preeclampsia and eclampsia are accompanied by a rise in prothrombin time (PT) and activated partial thromboplastin time (APTT)^[19]. The study conducted by Joshi SR *et al.* reveals thrombocytopenia and anomalies in the coagulation process, specifically a rise in APTT^[20]. In light of this, the current investigation showed that coagulation parameters were predictive markers for estimating preeclampsia severity. According to Swetha *et al.*²¹, PIH is markedly different from a typical pregnancy in terms of total platelet count (TPC), prothrombin time, activated partial thromboplastin time, bleeding time, and clotting time.

The severity of PIH is inversely correlated with platelet count, and thrombocytopenia-related coagulopathy risk rises as PIH gets worse. There are no instances of prolonged PT or aPTT in patients with normal platelet counts. In their comparative analysis of the coagulation profiles in preeclamptic, eclamptic, and normotensive patients, Chauhan *et al.* discovered that the PT was 13.78 seconds in moderate cases, 14.1 seconds for normal and 13.8 seconds for severe preeclampsia. However, the prothrombin time increase in his study was not statistically significant^[21].

CONCLUSION:

According to our research, the platelet counts decreased in relation to an increase in PIH blood pressure. When compared to pregnant women who were not abnormal, the PT and APTT were prolonged. When the platelet function activity was compromised and the platelet count drops below 1 lakh/cmm (requiring intervention, alarming as count 60,000/cmm. disseminated intravascular coagulation), the thrombocytopenia (physiological in gestation), becomes critical and coagulopathy was seen in immediate danger. Using the absolute platelet count as a gauge, we can evaluate pregnancy-induced hypertension. Therefore, it is important to detect the illness early, stop it from becoming worse and developing into eclampsia, and lower foetal morbidity and mortality.

REFERENCES:

1. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. *Lancet*. 2010 Aug 21;376(9741):631-44.
2. Sultana F, Parthiban R, Shariff S. Thrombocytopenia in pregnancy induced hypertension. *J Med Sci Health*. 2015;1(2):19-24.
3. Duley L. The global impact of pre-eclampsia and eclampsia. *Semin Perinatol*. 2009 Jun;33(3):130-7.
4. Fatma T, Azmi S, Chaudary A. Coagulation profile as important markers in preeclampsia and eclampsia. *Int J Adv Res*. 2017;5(5):2211-18.
5. Mohapatra S, Pradhan BB, Satpathy UK, Mohanty A, Pattnaik JR. Platelet estimation- its prognostic value in pregnancy induced hypertension. *Indian J Physiol Pharmacol*. 2007 Apr-Jun;51(2):160-4.
6. Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. *Am J Obstet Gynecol*. 2000 Jul;183(1): S1-S22.
7. Cunningham FG, MacDonald PC, Gant NF: *Williams Obstetrics*, ed 18. Norwak, Appleton & Lange, 1989, 653-694.
8. Rosevear SK, Liggins GC. Platelet dimensions in pregnancy-induced hypertension. *N Z Med J*. 1986 May 28;99(802):356-7.
9. Annam, Vamseedhar, *et al.* "Evaluation of platelet indices and platelet counts and their significance in preeclampsia and eclampsia". *Int J Biol Med Res*. 2011;2(1):425-428.
10. Luft FC: Hypertensive nephrosclerosis: update. *Curr Opin Nephrol Hypertension*: 2004 Mar 1;13(2):147-54.
11. Persu A, De Plaen JF. Recent insights in the development of organ damage caused by hypertension. *Acta cardiologica*. 2004 Aug 1;59(4):369-81.
12. Mushambi MC, Halligan AW, Williamson K. Recent developments in the pathophysiology and management of preeclampsia. *Br J Anaesthesia*. 1996; 76:133-48.
13. Ratnam SS, Rao KB, Arul kumaran S. *Obstetrics and Gynecology for postgraduates*, Vol 1. 2nd Edition. Orient Longman, Hyderabad. 1999:60.
14. Schlembach D. Pre-eclampsia. Still a disease of theories. *Fukushima J Med. Sci*. 2003;49(2):69-115.
15. Lescale KB, Eddlenmank KE, Cine DB. Ant platelet antibody testing in thrombocytopenic pregnant women. *Am J Obs Gynecol*. 1996;174(3):1014-8.
16. Monteiro G, Subbalakshmi NK, Pai SR. Relevance of measurement of hematological parameters in subjects with pregnancy induced hypertension. *Nitte University Journal of Health Science*. 2014 Mar 1;4(1):15.
17. Sharma UP, Kouli R, Sonowal R, Saikia P. Coagulation Parameters in Pre- eclamptic and Eclamptic Patients- A Comparative Study of 90 Cases. *Intern J Contem Med Res*. 2016;3(8):2235-8.
18. Sultana R, Karim SF, Atia F, Ferdousi S, Ahmed S. Platelet count in preeclampsia. *Journal of Dhaka National Medical College & Hospital*. 2012;18(2):24-26.
19. Lakshmi C Vijaya. "Comparative Study of Coagulation Profile in Mild Pre-eclampsia, Severe Pre-eclampsia, and Eclampsia". *International journal of scientific study*. 2016;4(4):180-183.
20. Kumar P L, Nirmala T, Vani B R, *et al.* Study of coagulation profile in pregnancy induced hypertension (PIH). *Indian J Pathol Oncol*. 2015;2(1):1-6.
21. Swetha A G, Nagaraja Puranik, K F Kammar. "A comparative study on coagulation profile and neutrophil-lymphocyte ratio in pregnancy-induced hypertension". *National Journal of Physiology, Pharmacy and Pharmacology*. 2018;8(3):400-405.
22. Chauhan P, Rawat U, Bisht V, Purohit R C. Comparison of coagulation profile in pre eclamptic and eclamptic patients with normotensive pregnant patients. *Journal of Evolution of Medical and Dental Sciences*. 2014;3(12):3208-3216.