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HEMATOLOGICAL PARAMETERS IN THE ASSESSMENT OF PREGNANCY INDUCED HYPERTENSION

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ABSTRACT

Pregnancy induced hypertension (PIH) is one of the most common causes of both maternal and neonatal morbidity. The hypertensive disorders account for thrombocytopenia in pregnancy and the risk of anemia may also increase with the severity of hypertensive disorders. Hyperuricemia in hypertensive pregnancy is an important finding because it identifies women at increased risk of adverse fetal outcome even women with gestational hypertension without any other features of pre-eclampsia. There is a constant ongoing research on hematological parameters for better predictors and prognostic factors to assess the progress and severity of the disease. The present study was undertaken to evaluate hematological parameters in assessment of pregnancy induced hypertension.

KEYWORDS: **P.I.H, hematological parameters, thrombocytopenia, serum uric acid**

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INTRODUCTION

Pregnancy induced hypertension (PIH) is one of the commonest outcomes with unknown etiology that causes the most maternal and perinatal morbidity and mortality [1]. It is associated with HELLP syndrome and pre-eclamptic liver dysfunction. HELLP syndrome stands for hemolysis, elevated liver enzyme AST (>70IU/L) and low platelet count (<100,000/μL.) [2]. Pre-eclampsia is a multiorgan disease process of unknown etiology characterized by increased blood pressure and proteinuria after 20 weeks of gestation[3].The disorder affects approximately 5 to 7 percent of pregnancies and is a significant cause of maternal and fetal morbidity and mortality [4]. Its incidence is 23.6 cases per 1,000 deliveries in the United States[5]. Pre eclampsia accounts for about one-fifth of antenatal admissions, two-thirds of referrals to day assessment units and a quarter of obstetric admissions to intensive care units [6].It may present diverse hematological features, varying from normal laboratory tests to severe thrombocytopenia (due to platelet activation and consumption), and/or anemia. Many clinical and biochemical parameters have been used to detect pregnancy induced hypertension (PIH) and to assess its severity .But currently there are no individual screening tests that are reliable, valid, and economical. The present study was undertaken to evaluate the relevance of routinely done hematological parameters in assessment of pregnancy induced hypertension.

MATERIALS AND METHODS

The study was conducted in the Department of Physiology in collaboration with Department of Biochemistry and O&G of IMS & SUM Hospital, Bhubaneswar.

Selection of cases

Selection of pregnancy induced hypertension cases were made from patients attending both OPD and indoor of Department of Obstetrics and Gynaecology with the criteria of pregnant women with blood pressure over the baseline ≥140/90 mmHg with or without proteinuria after 20th weeks of gestation till 2nd day post partum period .The subjects under the study group & control group were divided into five group taking their blood pressure as criteria. Group I comprised of 20 normotensive pregnant women taken as control. Group II- V included 80 subjects taken in the study groups and were divided based on their Diastolic Blood Pressure. Thus Group II – mild P.I.H (DBP 90-100mmHg), Group III- moderate P.I.H (DBP100-110mmHg), Group IV- severe P.I.H (DBP≥110 mmHg) and Group V included patients with eclampsia.

Exclusion Criteria

The cases with previous history of essential hypertension, renal disease, diabetes, ITP, previous hepatic disease, pheochromocytoma etc during non-pregnant state were excluded.

The data were analyzed for statistical significance using one way ANOVA. P value < 0.05 was considered as statistically significant.

OBSERVATION TABLE

Table 1
Distribution of cases

GROUP OF CASES	NO.OF CASES	PERCENTAGE (%)
Group I (Control group)	20	-
Group II (Mild PIH)	4	5
Group III (Moderate PIH)	18	22.5
Group IV (Severe PIH)	26	32.2
Group V (Eclampsia)	32	40

Table 2
Uric acid level in study group

GROUP	<4mg%		4-6mg%		>6mg%		
	Cases	%	Cases	%	Cases	%	Total
Mild PIH	1	25	3	75	-	-	4(100%)
Moderate PIH	6	33.3	12	66.7	-	-	18 (100%)
Severe PIH	4	15.4	12	46.2	10	38.4	26(100%)
Eclampsia	2	6.25	10	31.25	20	62.25	32(100%)

Table 3
Mean haemoglobin & platelet count level.

GROUP	Mean Hb%(gm/dl)±SD	Mean platelet count Lakhs/cumm blood(venous)±SD
Control	9.85±1.47	2.29±0.38
Mild PIH	9.82±1.53	2.05±0.24
Moderate PIH	9.47±1.27	1.9±0.38
Severe PIH	8.67±0.96	1.75±0.5
Eclampsia	8.32±1.12	1.61±0.46
Statistical analysis by Anova	Df =99, f=91.76, p < 0.001	Df =99, f= 89.22,p < 0.001

DISCUSSION

PIH accounts for one of the top five causes of maternal death in the world[7]. It is associated with greater increases of IUGR & IUD of fetus. But currently there are no suitable indicators in monitoring the progression of PIH leading to IUGR and IUD of fetus[8].They may also suffer from the consequences of high rates of operative deliveries and the adverse effects of maternal drugs. The neonates may also have a spectrum of hematological changes which may add to the existing morbidity in them[9] Table 1 shows the distribution of cases in the study and control group and control. Table 2 is a comparison of serum uric acid level between different study groups. This table shows the serum uric acid level increases with the increase in the severity of PIH. The patho-genesis of hyperuricemia in PIH have not yet been determined. Chesely and Williams[10] stated that in PIH there was impaired glome-rular filtration rate and an increased tubular reabsorption of uric acid, leading to impaired uric acid clearance, but Pollak and Nettles[11] reported that decreased uric acid clearance was the result of enhanced tubular reabsorption or inhibited tubular secretion or both. Slemons and Bogert [12] first observed an association between SUA concentration and the presence of PIH. Stander and Cadden [13] were first to demonstrate a high correlation between the severity of PIH and concentration of SUA level. In the study of Mustaphi R et al, high positive correlation was observed between levels of serum uric acid and severity

of PIH in relation to hypertension and proteinuria[14]. Table 3 shows the mean haemoglobin level (gm/dl) and the mean platelet count in lakhs/cumm of blood in the control group and study group There was a significant decrease in the haemoglobin level with the increase in severity of the disease .There was a significant reduction in the platelet count in the study group as compared to control group moreover the women with eclampsia had very low platelet count as compared to mild and severe preeclampsia. It has also been reported that the mean value of platelet counts was significantly decreased in the pre-eclamptic patients than normotensive pregnant women. A study conducted in India showed that platelet count in pre-eclampsic group and eclampsic group showed significantly decreased platelet count when compared with the normotensive[15] control group. Another study conducted at Turkey also showed lower platelet count in severe[16] pre-eclampsic women ($p<0.05$).Moreover hypertensive disorders accounts for 21% of cases of thrombocytopenia in pregnancy Nazli R et al too have reported high frequency of thrombocytopenia in PIH mothers[17]. It has been shown by earlier researchers that Hematological abnormalities such as thrombocytopenia and decrease in some plasma clotting factors may develop in pre-eclamptic[18] women. The degree of thrombocytopenia increases with severity of disease and the incidence of thrombocytopenia depend on the severity of the disease process. Lower the platelet count, greater are the maternal and fetal morbidity[19] and mortality .Some other researchers also reported that during pregnancy, the commonest cause of thrombocytopenia are gestational thrombocytopenia[20-21], preeclampsia and eclampsia . It has been observed and reported that about 6.6-11.6% [22] of pregnancies results in thrombocytopenia some researcher reported that thrombocytopenia is caused due to the involvement of endothelial damage and peripheral consumption [23]. It is also found that in pregnancies complicated with preeclampsia, the life span of platelet is reduced from 3 to 5 days and the altered platelet membrane accelerates its aggregation[24] and destruction. The hematological changes that appear in pre-eclamptic pregnancy are divided into:

A)Numerical and functional platelet anomalies

The major role played by the thrombocyte in the pathophysiology of preeclampsia is related to the release of thromboxane A2, with subsequent increase of thromboxane/prostacyclin ratio [25, 26]. Thromboxane A2 promotes vasospasm, induces supplementary platelet aggregation and endothelial damage, which add an important contribution to maintaining platelet dysfunction and promoting platelet consumption (activation, aggregation, microangiopathic hemolysis induced by severe vasospasm), resulting in thrombocytopenia, which is an important sign of severe/aggravating preeclampsia.

B)*Alterations of hemoglobin and erythrocytic parameters*

Most frequently – hemoconcentration manifested with increased hematocrit[27] due to increased endothelial permeability; anemia may also be present in rare cases. The anemia is most frequently associated with HELLP syndrome and it is due to microangiopathic intravascular hemolysis – physical destruction of erythrocytes in the microcirculation affected by disseminated microthrombosis; the anemia will be slight/ medium, normochromic, normocytic, with a hemolytic pattern (increased bilirubin – unconjugated fraction, increased LDH, increased reticulocyte count), fragmented erythrocytes and microspherocytes or peripheral blood smear, and, in severe forms, hemoglobinuria and hemoglobinemia.

C)Considering the coagulation changes, it is known that normal pregnancy is a procoagulant status and that this tendency is increasing during the development of the pregnancy with the end-point of minimizing the blood loss intrapartum. In pre-eclamptic pregnancies, the coagulation cascade is generally activated [28-29] preeclampsia being by itself a highly thrombotic and procoagulant state, with platelet activation and consumption, promoting of thrombin formation, promoting of

fibrin formation and destruction. About 20% of patients have altered coagulation [30]

CONCLUSION

Preeclampsia (pregnancy induced hypertension) represents an important pathology in pregnancy, which may present vital prognosis and which may be complicated with prematurity, dismaturity – small newborns for gestational, intrauterine growth restriction, or even fetal death. Regarding platelet count which is the main tool of early detection of thrombocytopenia in women with pregnancy-induced hypertension .Therefore present study is carried out to provide the information and suggestion to the patient and clinician for the early detection of thrombocytopenia in women presenting with varying degree of pregnancy induced hypertension (PIH) in preventing the life threatening complications like HELLP syndrome.

REFERENCES

1. Fatemeh T, Marziyeh G, Nayereh G, Anahita G,Samira T, Maternal and perinatal outcome in nulliparious women complicated with pregnancy hypertension**.** J Pak Med Assoc, 60(9): 707-710, (2010).
2. Padden MO, HELLP syndrome; recognition and perinatal management. Am Fam Physician; 60(3): 827-836, (1999).
3. Rahimi Z, Mozafari H, Parsian A, Preeclampsia and angiotensin converting enzyme (ACE) I/D and angiotensin II type-1 receptor (AT1R) A1166C polymorphisms: Association with ACE I/D polymorphism J**.** Renin-Angiotensin-Aldosterone System,14 (2): 174-180, (2013).
4. Witlin AG, Sibai BM, Magnesium sulfate therapy in preeclampsia and eclampsia. Obstet Gynecol, (92):883–889, (1998).
5. Samadi AR, Mayberry RM, Zaidi AA, Pleasant JC, McGhee N Jr, Rice RJ,Maternal hypertension and associated pregnancy complications among African-American and other women in the United States. Obstet Gynecol,(87):557–563,(1996).
6. Duley L. Preeclampsia and the hypertensive disorders of pregnancy. Br Med Bull, (67)161–76, (2003).
7. T. Sunitha1, K.Sameera and G.Umaramani, Int J Pharm Bio Sci, Oct 3(4): (B) 840 – 844, (2012).
8. Gearaldine Monteiro , Subbalakshmi N.K. & Sheila R. Pai ,Relevance of measurement of hematological parameters in subjects with pregnancy induced hypertension, NUJHS, 4(1):15-20, (2014)**.**
9. Sandhya Sivakumar, B. Vishnu Bhat and Bhawana Ashok Badhe **,** Effect of Pregnancy Induced Hypertension on Mothers and their Babies, Indian Journal of Pediatrics(74)July,623-625, (2007).
10. Chesley LC and Williams LO,Renal glomerular and tubular function in relation to hyperuricemia of preeclampsia and eclampsia. Am J. Obstet Gynecol(50):367,(1945).
11. Pollak VE and Nettles JB. The kidney in toxoemia of pregnacny: A clinical and pathological study based on renal biopsies. Medicine, (39):469, (1960).
12. Slemons JM and Bogert LJ. The uric acid content of maternal and foetal blood. J. Biol. Chem, (32):63, (1934).
13. Takiar tS, Deshmukh MB. Platelet dysfunction in toxamia of pregnancy. J. Obstet. Gynae. India, (38): 670, (1988).
14. Mustaphi R, Gopalan S, Dhaliwal L, Sarkar AK,Hyperuricemia and pregnancy induced hypertension—reappraisal.Indian J Med Sci, 50 (3):68-71,(1996).
15. Annam V, Srinivasa K , Santhosh KY , Suresh DR. Evaluation of platelet indices and platelet counts and their significance in Pre eclampsia and eclampsia**.** Int J Biol Med Res, 2(1):425-428,(2011).
16. YaprakEU, KezbanD, LlgnT, Yusuf U, Mehmet MM, AyfleK et al, Evaluation of Hemoglobin and Platelet Levels in Mild,

Moderate and Severe Preeclampsia. Perinatal Journal, 15(3): 93-98, (2007).
17. Nazli R, Khan MA, Akhtar T, Mohammad NS, Aslam H, Haider J,Frequency of thrombocytopenia in pregnancy related hypertensive disorders in patients presenting at tertiary care hospitals of Peshawar.Khyber Med Univ J, 4 (3):101-105,(2012).
18. Namavar BJ, Rafiee SH,Coagulation Factors in Severe Preeclampsia. IRCMJ, 11(3): 321-324, (2009).
19. Cunningham FG, Norman FG, Kerneth JL, Lary CG, Hauth JC, Wenstom KD, et al, Hypertensive disorders in pregnancy. Williams Obstetrics New York: McGraw Hill; International 21st Edt: 567–618, (2001).
20. Parnas M, Sheiner E, Shoham-Vardi I, Burstein E, Yermiahu T, Levi I, et al, Moderate to severe thrombocytopenia during pregnancy. Eur J Obstet Gynecol Reprod Bio, (128): 163–168,(2006).
21. Burrows RF, Kelton JG,Fetal thrombocytopenia and its relation to maternal thrombocytopenia.N Eng J. Med(329): 1463-1466, (1993).
22. Boehlen F, Hohlfeld P, Extermann P, Perneger TV,de Moerloose P, Platelet count at term pregnancy: a reappraisal of the threshold. Obstet Gynecol (95): 29-33, (2000).
23. Asghar Khan , Amin Fahim , Aneela Qureshi , Ghulam Shah Nizamani , Mohammad Ahmed Azmi, Pregnancy Induced Hypertension;Assessment of prognostic value of platelet count in women with varying degree.Professional Med J,21(3): 436-440,(2014).
24. Magann EF, Martin JN Jr. Twelve steps to optimal management of HELLP syndrome. ClinObstet Gynecol, (42): 532-550, (1999).
25. VanWijk MJ, Kublickiene K, Boer Ket al, Vascular function in preeclampsia.Cardiovasc Res Jul; 47(1):38-48,(2000).
26. Mills JL, DerSimonian R, Raymond E et al – Prostacyclin and thromboxane changes predating clinical onset of preeclampsia: a multicenter prospective study. JAMA, (282):356-362, (1999).
27. Hayman R, Warren A, Brockelsby J et al, Plasma from women with pre-eclampsia induces an in vitro alteration in the endothelium-dependent behavior of myometrial resistance arteries. BJOG 107(1):108-115, (2000).
28. Heilmann L, Rath W, Pollow K, Hemostatic abnormalities in patients with severe preeclampsia. Clin Appl Thromb Hemost, 13(3):285-291,(2007).
29. Weiner CP, Preeclampsia-eclampsia syndrome and coagulation. Clin Perinatol, 18(4):713-726, (1991).
30. Ana-Maria Vladareanu, Horia Bumbea, Mihai Ciorascu, Ciprian Pop, Cristian Andrei, Maedica, et al .A study of the hematological picture and of platelet function in preeclampsia ,report of a series of cases, A Journal of Clinical Medicine, Volume 4 (4) 326-337(2009).