

Platelet Count and Complications in Pre-Eclampsia and Eclampsia Patients in Pregnancy: An Observational Study in a Tertiary Care Hospital in Bangladesh

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Abstract

Original Research Article

Objective: To measure the platelet count and complications in pre-eclampsia and eclampsia patients in pregnancy. **Methods and Materials:** This was a cross sectional observational study and conducted from 1st January 2018 to 30th June 2018 in Sheikh Sayera Khatun Medical College Hospital, Gopalganj. Total 150 samples were taken from indoor of Sheikh Sayera Khatun Medical College Hospital, Gopalganj. Purposive sampling technique was followed in this study. Patients who clinically present with features of pre-eclampsia and eclampsia without any chemical evidence of haemorrhagic manifestations were included in this study. Any maternal abnormalities in pregnancies (such as multiple pregnancy, pregnancy with heart diseases, GDM etc.) except pre-eclampsia and eclampsia and patients who were suffering from other coagulation disorder before pregnancy were excluded from the study. **Results:** average mean maternal age for the eclampsia group was 23.12±4.20 years and mean maternal age for the pre-eclampsia group was 28.24±5.86 years. Average mean gestational age for the eclampsia group was 33.50±2.58 weeks and average mean gestational age for the pre-eclampsia group was 37.50±3.89 weeks. 58 (70.73%) eclamptic patients and 18 (39.13%) pre-eclamptic patients were primigravida. 24 (29.27%) eclamptic patients and 28 (60.89%) pre-eclamptic patients were multigravida. p value of Mean maternal age, gestational age and gravida is < 0.001, which is significant. platelet count in > 200,000 / cu mm in eclampsia is (4.87%) and PET is 02 (4.35%), platelet count between 150,000 – 200,000 / cu mm in eclampsia is (13.41%) and PET is (30.43%). platelet count between 100,000 -150,000 / cu mm in eclampsia is 66 (80.49%) and PET is 30 (65.22%). Platelet count between 50,000 - 100,000/ cu mm in eclampsia is 01 (1.21%). Platelet count > 200,000 / cu mm present in 4 eclampsia is and 2 PET patients, out of which no patient developed any abnormal coagulation or any complications. Platelet count between 150,000 -200,000 / cu mm present in 11 eclampsia and 14 PET patients, out of which no patient shows any abnormal coagulation but 6 (4.69%) cases show adverse maternal outcome and 3 (2.34%) patients need blood transfusion. Platelet count between 100,000 - 150,000 / cu mm present in 66 eclampsia and 30 PET cases, out of which 4 (3.12%) developed abnormal coagulation and 23 (17.96%) cases shows adverse maternal outcome and 16 (12.5%) cases needs blood transfusion platelet count between 50,000 - 100,000 / cu mm present in 1 eclampsia and 0 PET patient, out of which 1 (0.78%) patient shows abnormal coagulation, and also the same patient developed adverse maternal outcome and needs blood transfusion and the same patient dies due to HELLP Syndrome. **Conclusion:** A platelet count < 100 × 10⁹/L should alert caregivers to the real possibility of generalized coagulopathy. It is thought that thrombocytopenia is the early manifestation of generalized coagulopathy in pre-eclampsia and eclampsia and the main risk of thrombocytopenia is excessive blood loss during and after delivery.

Keywords: Pre-eclampsia, Eclampsia, Maternal complication, Fatal complication.

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INTRODUCTION

Hypertension is a frequent pregnancy medical issue that affects 6–8% of all pregnancies. Preeclampsia and eclampsia are two hypertension illnesses that can wreak havoc on a pregnant woman's life. This causes

maternal and newborn morbidity and mortality in roughly 5% to 10% of hypertensive pregnancies [1]. Preeclampsia is estimated to affect 5-14 percent of all pregnancies worldwide, with 10% of all preeclampsia instances occurring in pregnancies of less than 34 weeks' gestation. Preeclampsia is mild in 75% of cases

and severe in 25% of them [2]. There have been numerous reports of hemostatic abnormalities associated with hypertensive diseases during pregnancy. The most prevalent of these is thrombocytopenia, which affects 11 percent to 29 percent of individuals with pregnancy-induced hypertension [3]. These pregnancies are also associated to qualitative changes that indicate an increase in platelet turnover. In the peripheral blood smear, there are more megakaryocytes in the bone marrow, a shorter platelet life span, and a higher amount of immature platelets [4]. Many researchers believe that increased platelet usage is caused by disseminated intravascular coagulation, while others believe it is caused by an immunological response. The most common hematological condition seen in pregnancy-induced hypertension is thrombocytopenia [5]. The severity of thrombocytopenia rises with illness severity, and the occurrence of thrombocytopenia is dependent on the severity of disease progression. The lower the platelet count, the higher the maternal and fetal morbidity and death. Overt thrombocytopenia, defined as a platelet count of less than 1 lac/mm^3 , indicates the severity of the illness process, and delivery is usually recommended because platelet numbers continue to decline after that [6]. Platelet counts of less than 1 lac/mm^3 are associated with a poor neonatal outcome in HELLP syndrome [7]. It occurs in 2–12% of women who have severe pre-eclampsia or eclampsia. To avoid consequences like HELLP syndrome and increased maternal and fetal morbidity and death, early detection of the severity of PIH is critical. Platelet count by automated cell counter, in-direct and direct method, test for prothrombin time, partial thromboplastin time (PTT), decrease in 2 antitrypsin, increase fibronectin level/decrease antithrombin III level, increase in SF1f-1 (soluble Fmslike tyrosine kinase-1), decrease in circulating free placental growth factor (PGF) and vascular endothelial growth factor (VEGF) concentration. Platelet count (indirect and direct methods), prothrombin time, partial thromboplastin time (PTT), decrease in 2 antitrypsin, increase fibronectin level/decrease antithrombin III level, increase in SF1f-1 (soluble Fmslike tyrosine kinase-1), decrease in circulating free placental growth factor (PGF) and vascular endothelial growth factor (VEGF) concentration. Platelet estimation, on the other hand, is a quick, accurate, simple, and inexpensive procedure that does not require any expensive ingredients; thus, prognosis of diseases could be monitored using simple platelet count estimation.

Pre-eclampsia and eclampsia are linked to a number of problems, including preterm labor, IUGR, IUD, bleeding by accident, pulmonary oedema, and

heart failure. HELLP syndrome (3%), DIC (3%), renal failure (4%), adult respiratory syndrome (3%), and cerebral haemorrhage (3%). (1.2 percent) [8]. It continues to be one of the leading causes of maternal and fetal death and morbidity [9]. They are thought to be responsible for 14% of direct maternal deaths and 18% of fetus or newborn deaths [10, 11]. Preeclampsia and eclampsia are thought to be responsible for about 14% of maternal mortality per year (50,000-75,000) 19 across the world.

There is a decrease in platelet count in women with PE and/or IUGR [12]. Platelet function alterations are more complicated. Platelet aggregation is raised in the early stages of PE, but it is diminished in established severe illness [13]. Longitudinal studies imply that increased platelet aggregation may occur 2–5 weeks before the onset of PE [14].

The purpose of the study is to measure the platelet counts and to find out the complications of pre-eclampsia and eclampsia patients in pregnancy.

METHODS AND MATERIALS

This was a cross sectional observational study and conducted from 1st January 2018 to 30th June 2018 in the Sheikh Sayera Khatun Medical College Hospital, Gopalganj. Total 150 samples were taken from indoor of Sheikh Sayera Khatun Medical College Hospital, Gopalganj. Purposive sampling technique was followed in this study. Patients who clinically present with features of pre-eclampsia and eclampsia without any chemical evidence of haemorrhagic manifestations were included in this study. Any maternal abnormalities in pregnancies (such as multiple pregnancy, pregnancy with heart diseases, GDM etc.) except pre-eclampsia and eclampsia and patients who were suffering from other coagulation disorder before pregnancy were excluded from the study. Data were analyzed by using software SPSS version 23.

RESULTS

Table 1 shows that average mean maternal age for the eclampsia group was 23.12 ± 4.20 years and mean maternal age for the pre-eclampsia group was 28.24 ± 5.86 years. And average mean gestational age for the eclampsia group was 33.50 ± 2.58 weeks and average mean gestational age for the pre-eclampsia group was 37.50 ± 3.89 weeks. Table 1 also shows that maximum patients were primigravida. The results are statistically significant.

Table 1: Mean maternal age, gestational age on admission (n=150)

Factors	Pre-eclampsia (n=50) Mean±SD	Eclampsia (n=100) Mean±SD	P value
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Maternal age (years)	28.24±5.86	23.12±4.20	<0.001**
Gestational age (weeks)	37.50±3.89	33.50±2.58	<0.001**

Figure 1 shows that the majority 70.735% pre-eclampsia patients and 30.13% eclampsia patients were 1st gravid. Followed by 60.89 % pre-eclampsia

patients and 29.27% eclampsia patients were multi gravid.

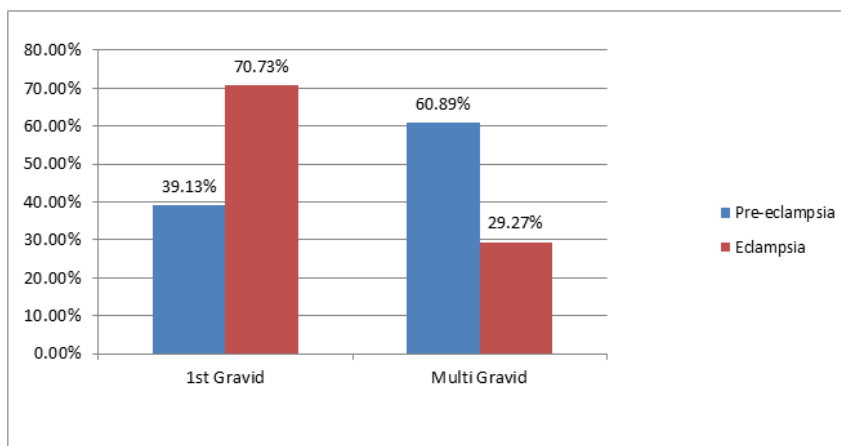


Figure 1: Types of gravid on admission

Table 2 shows, the mean baseline antenatal systolic BP in PET cases was 157.65±21.75 and in eclampsia cases was 157.58±19.03. Mean diastolic

blood pressure in PET cases was 106.75±11.23 and in eclampsia cases was 109.12±10.26 and there had no significant changes.

Table 2: Mean value of blood pressure (n=150) on admission

Blood pressure	Pre-eclampsia (n=50) Mean±SD	Eclampsia (n=100) Mean±SD	P value
Systolic BP	157.65±21.75	157.58±19.03	0.984
Diastolic BP	109.12±10.26	106.75±11.23	0.239

Figure 2 shows that maximum 59.37% of the patients had LUCS as a mode of delivery whereas the 40.13% had vaginal delivery.

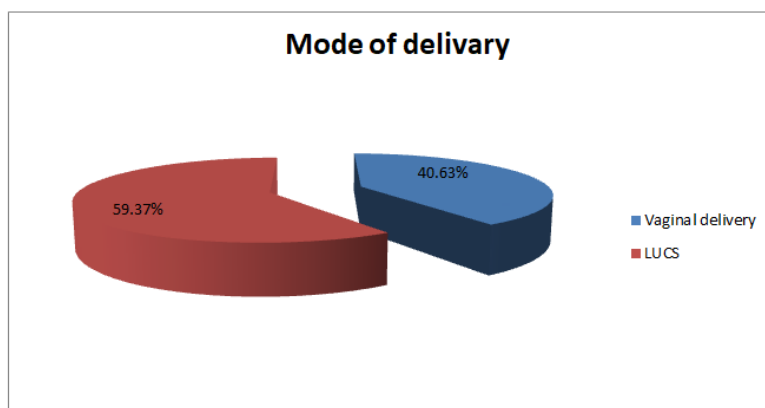


Figure 2: Mode of Delivery

Figure 3 shows that the maximum patients 76.56% had no complications followed by 14.84% had

postpartum hemorrhage and 6.25% had pulmonary edema.

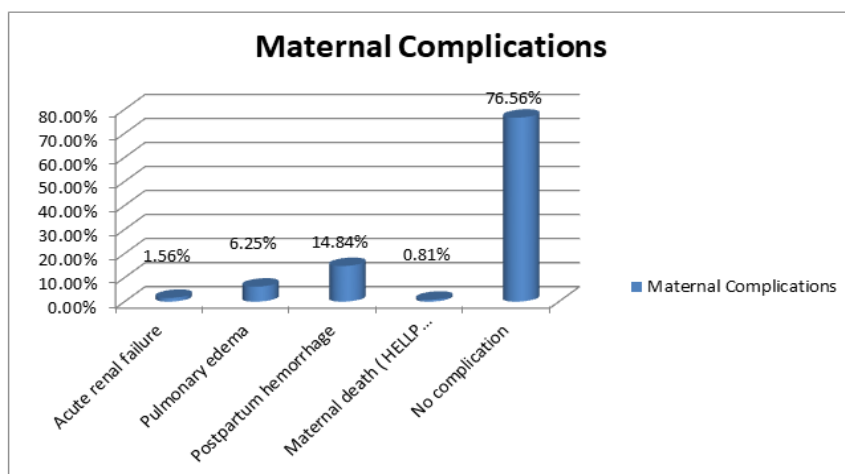


Figure 3: Maternal complications

Figure 4 shows , among the 19 PPH patients, maximum that is 15 (78.94%) patients had platelet count between 100,000 – 150,000 / cu mm.

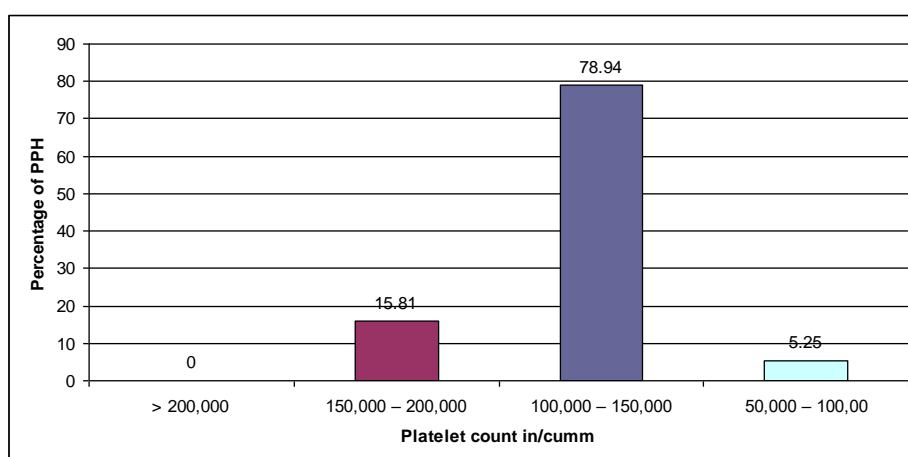


Figure 4: Platelet count in post partum hemorrhage cases (n=19)

Figure 4 shows that the maximum 49.21% fatal were preterm or had low birth Wight followed by 34.38% were healthy baby, 8.63% were still birth and 7.81% were prenatal death.

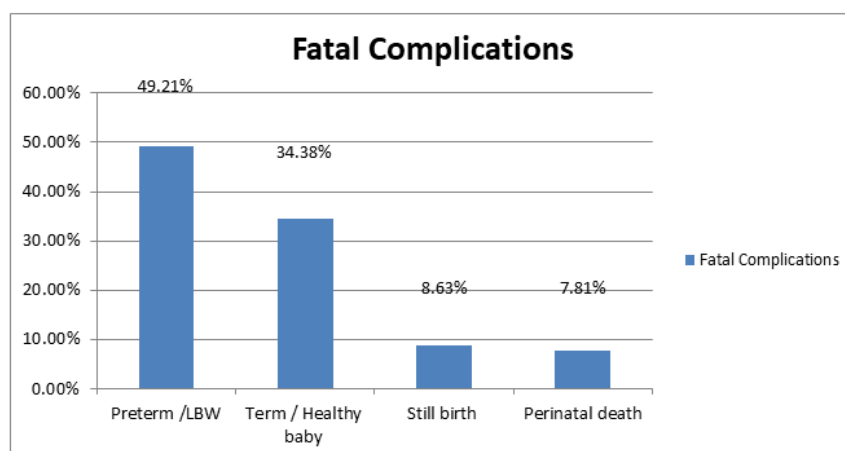


Figure 5: Fatal complications

Table 3 shows, that out of 128 babies, 82 babies had born with LBW.

Table 2: Birth weight in gm in relation to platelet count (n=128)

Platelet count	< 2500 gm (n=66) No. (%)	2500 gm and above (n=62) No. (%)	Total (n=128) No. (%)
> 200,000	4 (6.06%)	06 (9.68%)	10 (7.81%)
150,000-200,000	24 (36.37%)	12 (19.35%)	36 (28.13%)
100,00-150,000	38 (57.58%)	44 (70.97%)	82 (64.06%)
Total	100 (100.0%)	50(100.0%)	128 (100.0%)

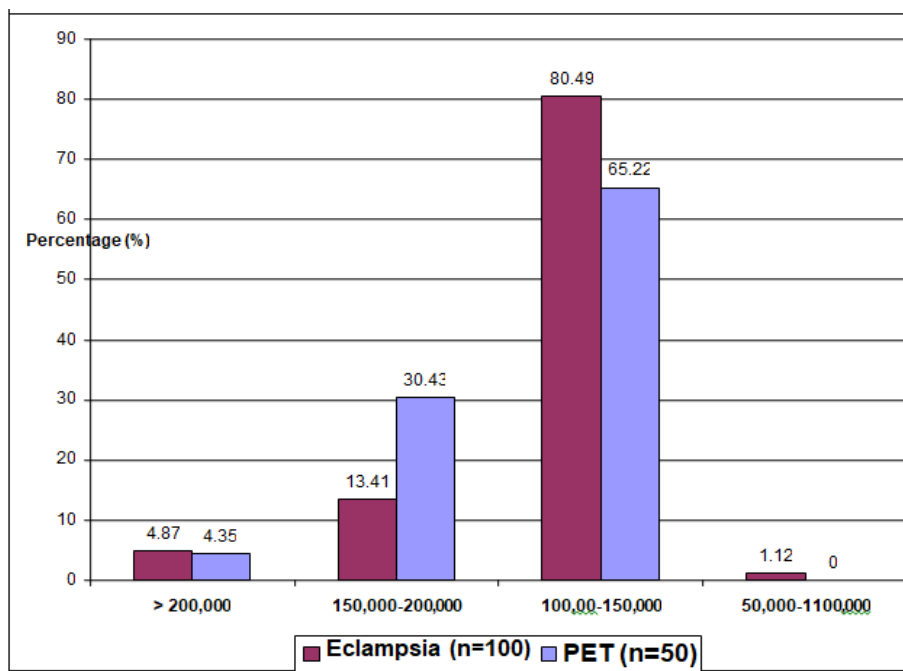


Figure 6: Percent distribution of platelet count in pre-eclampsia and eclampsia (n=150)

Figure 6 shows that, among 150 patients 97 patients had platelet count < 150X10⁹ /L.

Table 3: Relation of platelet count among women with preeclampsia and eclampsia and abnormal coagulation or adverse maternal outcome (n = 128)

	50-100 n=1	100-150 n=96	150-200 n=25	>200 n=26
Outcome				
Abnormal coagulation*	1	4	0	0
N(%)	.78%	3.12%	0%	0%
Adverse maternal outcome	1	23	6	0
N(%)	.78%	17.96%	4.69%	0%
Blood transfusion	1	16	3	0
N(%)	.78%	12.5%	2.34%	0%

Table 3 shows that, out of 128 cases, 4 cases shows abnormal coagulation and 30 cases developed complications (such as PPH, pulmonary edema, acute renal failure, HELLP syndrome), out of which 19 patients required blood transfusion. 23 cases who developed complications and 16 cases who required blood transfusion had platelet count between 100 – 150 X 10⁹ /L.

DISCUSSION

A cross sectional observational study was done among 150 patients of eclampsia and pre-eclampsia, of

which 100 were eclamptic patients and 50 were pre-eclamptic patients.

In current study, average mean maternal age for the eclampsia group was 23.12±4.20 years and mean maternal age for the pre-eclampsia group was 28.24±5.86 years. Average mean gestational age for the eclampsia group was 33.50±2.58 weeks and average mean gestational age for the pre-eclampsia group was 37.50±3.89 weeks. 58 (70.73%) eclamptic patients and 18 (39.13%) pre-eclamptic patients were primigravida. 24 (29.27%) eclamptic patients and 28 (60.89%) pre-eclamptic patients were multigravida. p value of Mean maternal age, gestational age and gravida is < 0.001,

which is significant. Vitthal G Kuchake *et al.*, (2010) conducted a prospective observational study on a total of 971 pregnant women between 24 weeks to 40 weeks gestation, they reported that a total of 73 preeclamptic (7.5%) were observed in between 18-22(61.64%) years of age and in primigravida (n=43) and most of them belong to middle class [16].

In current study, about 66 (51.56 %) babies had born with birth weight < 2500 gm, out of which 38 (57.58%) babies belongs to patients having platelet count 100,00-150,000 / cu mm. About 62 (48.44%) babies had born with birth weight 2500 gm and above, out of which 44 (70.91%) babies belongs to patients having platelet count 100,00-150,000 / cu mm. p value of birth weight in gm in relation to platelet count is p = 0.09, which is not significant. Romero R *et al.*, (1989), reported that, thrombocytopenia was linked with a higher occurrence of intrauterine growth retardation and preterm delivery [17].

Current study shows, platelet count in > 200,000 / cu mm in eclampsia is 4 (4.87%) and PET is 02 (4.35%), platelet count between 150,000 – 200,000 / cu mm in eclampsia is 11 (13.41%) and PET is 14 (30.43%). platelet count between 100,000 -150,000 / cu mm in eclampsia is 66 (80.49%) and PET is 30 (65.22%). Platelet count between 50,000 - 100,000 / cu mm in eclampsia is 01 (1.21%) and PET is 0 (0%). p value of platelet count (n=128) in pre-eclampsia and eclampsia is p = 0.21 which is not significant. Burrows *et al.*, (1987) reported incidence of thrombocytopenia to be about 17%, Kelton *et al.*, (1987) reported that 20% of patients with PET develop consumptive thrombocytopenia and Gibson *et al.*, (1982) showed that thrombocytopenia is such a common finding in eclampsia and PET, occurring in about 15% of the patients [18].

Current study shows, platelet count among women with preeclampsia and eclampsia and Relation of platelet count with abnormal coagulation and adverse maternal outcome. Platelet count > 200,000 / cu mm present in 4 eclampsia and 2 PET patients, out of which no patient developed any abnormal coagulation or any complications. Platelet count between 150,000 - 200,000 / cu mm present in 11 eclampsia and 14 PET patients, out of which no patient shows any abnormal coagulation but 6 (4.69%) cases shows adverse maternal outcome and 3 (2.34%) patients need blood transfusion. Platelet count between 100,000 -150,000 / cu mm present in 66 eclampsia and 30 PET cases, out of which 4 (3.12%) developed abnormal coagulation and 23 (17.96%) cases shows adverse maternal outcome and 16 (12.5%) cases need blood transfusion. Platelet count between 50,000 - 100,000 / cu mm present in 1 eclampsia and 0 PET patient, out of which 1 (0.78%) patient shows abnormal coagulation, and also the same patient developed adverse maternal outcome and needs

blood transfusion and the same patient dies due to HELLP Syndrome.

A cohort study by PIERS in September 2011 shows that, out of 1405 pre-eclamptic and eclamptic patients, platelet count < 150 × 10⁹/L occurred in 395 women (28.1%) and a total of 122 (8.7%) women had a platelet count < 100 × 10⁹/L and 105 women (7.5%) had abnormal coagulation as defined by an abnormal INR, serum fibrinogen, or both, on the same blood sample as the worst platelet count 84. This is consistent with previous studies and reports [85]. In total, 152 (10.8%) women had one or more adverse maternal outcomes during their hospital stay. The most common adverse outcomes were a need for blood transfusion, which occurred in 60 women (4.3%) and pulmonary edema, which occurred in 35 women (2.5%). Abnormal coagulation occurred in 105 of 1405 eligible women (7.5%). These results do not fully support the findings of Leduc *et al.*, [7] who studied 100 women with severe preeclampsia and concluded that abnormal coagulation (defined by low serum fibrinogen or elevated aPTT) was always associated with thrombocytopenia. Other similar studies resulted in the same conclusion for women with severe preeclampsia [19].

CONCLUSION

This study shows that, thrombocytopenia occurs commonly among women admitted to hospital with pre-eclampsia and eclampsia, and there was a significant association between both a platelet count between 100 - 150 × 10⁹/L and abnormal coagulation test results and an increased risk of adverse maternal outcome, or blood transfusion specifically. A platelet count < 100 × 10⁹/L should alert caregivers to the real possibility of generalized coagulopathy.

REFERENCES

1. Roberts, J. M., & Cooper, D. W. (2001). Pathogenesis and genetics of pre-eclampsia. *The Lancet*, 357(9249), 53-56.
2. Khedun, S. M., Moodley, J., Naicker, T., & Maharaj, B. (1997). Drug management of hypertensive disorders of pregnancy. *Pharmacology & therapeutics*, 74(2), 221-258.
3. Gibson, G., Hunter, D., Neame, P. B., & Kelton, J. G. (1982). Thrombocytopenia in pre-eclampsia and eclampsia. *Semin Thromb Hemost*, 8, 234-247.
4. Burrows, R. F., Hunter, D. J., Andrew, M. A. U. R. E. E. N., & Kelton, J. G. (1987). A prospective study investigating the mechanism of thrombocytopenia in preeclampsia. *Obstetrics and gynecology*, 70(3 Pt 1), 334-338.
5. Saleh, A. A., Bottoms, S. F., Welch, R. A., Ali, A. M., Mariona, F. G., & Mammen, E. F. (1987). Preeclampsia, delivery, and the hemostatic system. *American journal of obstetrics and gynecology*, 157(2), 331-336.

6. Cunningham, F. G., Norman, F. G., Kerneth, J. L., Lary, C. G., Hauth, J. C., & Wenstom, K. D. (2001). Hypertensive disorders in pregnancy. In Seilis, A., Noujaim, S. R., Davis, K., editors. *Williams Obstetrics*. International Edn, 21st Edn, New York: McGraw Hill; p. 567–618.
7. Weinstein, L. (1982). Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. *American journal of obstetrics and gynecology*, 142(2), 159-167.
8. HMSO. (1997). Report on Confidential Enquiries into Maternal Deaths in the United Kingdom, 99.
9. Cunningham, F. G., Veno, K. J., & Bloom, S. L. (2010). Pregnancy Hypertension. In: *Williams Obstetrics*. 23e.
10. Mutter, W. P., & Karumanchi, S. A. (2008). Molecular mechanisms of preeclampsia. *Microvascular research*, 75(1), 1-8.
11. VanWijk, M. J., Kublickiene, K., Boer, K., & Vanbavel, E. D. (2000). Vascular function in preeclampsia. *Cardiovascular research*, 47(1), 38-48.
12. Hutt, R., Ogunniyi, S. O., Sullivan, M. H., & Elder, M. G. (1994). Increased platelet volume and aggregation precede the onset of preeclampsia. *Obstetrics and gynecology*, 83(1), 146-149.
13. Brantsæter, A. L., Myhre, R., Haugen, M., Myking, S., Sengpiel, V., Magnus, P., ... & Meltzer, H. M. (2011). Intake of probiotic food and risk of preeclampsia in primiparous women: the Norwegian Mother and Child Cohort Study. *American journal of epidemiology*, 174(7), 807-815.
14. Sibai, B. M., Mercer, B., & Sarinoglu, C. (1991). Severe preeclampsia in the second trimester: recurrence risk and long-term prognosis. *American journal of obstetrics and gynecology*, 165(5), 1408-1412.
15. Friedman, S. A., Lubarsky, S. L., & Lim, K. H. (2001). Mild Gestational Hypertension and Preeclampsia. In *Hypertensive Disorders in Women*. In: Sibai, B. M., ed. *Hypertensive Disorders in Women*. Philadelphia: W.B. Saunders; 9-23.
16. Kuchake, V. G., Kolhe, S. G., Dighore, P. N., & Patil, S. D. (2010). Maternal and neonatal outcomes in preeclampsia syndrome. *International Journal of Pharmaceutical Sciences and Research*, 1(11), 74-82.
17. Romero, R., Mazor, M., Lockwood, C. J., Emamian, M., Belanger, K. P., Hobbins, J. C., & Duffy, T. (1989). Clinical significance, prevalence, and natural history of thrombocytopenia in pregnancy-induced hypertension. *American journal of perinatology*, 6(1), 32-38.
18. Ketlon, J., Huner, J. D. S., & Naeme, P. B. (1987). A platelet function defect in PET and eclampsia. *Obstet Gynecol*, 65, 107.
19. Barron, W. M., Heckerling, P., Hibbard, J. U., & Fisher, S. (1999). Reducing unnecessary coagulation testing in hypertensive disorders of pregnancy. *Obstetrics & Gynecology*, 94(3), 364-370.