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Original Research Article

COMPARATIVE STUDY OF CORD BLOOD HAEMATOLOGICAL PROFILE OF NEONATES BORN TO HYPERTENSIVE MOTHERS WITH NORMOTENSIVE MOTHERS

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ABSTRACT

Background: Hypertensive disorders of pregnancy (HDP) are associated with a wide range of adverse maternal and neonatal outcomes, including alterations in fetal hematological parameters. Cord blood analysis offers insight into the hematopoietic and physiological status of neonates at birth, particularly in high-risk pregnancies such as those complicated by HDP.

Aim: To compare the cord blood hematological profile of neonates born to hypertensive mothers with those born to normotensive mothers and evaluate the implications on neonatal outcomes.

Materials and Methods: This case-control study was conducted at the Department of Obstetrics and Neonatal Intensive Care Unit (NICU), Sri Guru Ram Das Institute of Health Sciences and Research, Amritsar, between February 2020 and June 2021. A total of 90 neonates were divided equally into two groups: cases (born to mothers with HDP) and controls (born to normotensive mothers without comorbidities). Cord blood and day-3 peripheral venous blood samples were collected and analyzed using an automated hematology analyzer. Hematological parameters including hemoglobin (Hb), packed cell volume (PCV), red cell indices, total leucocyte count (TLC), absolute neutrophil count (ANC), absolute lymphocyte count (ALC), platelet count, and nucleated red blood cells (nRBCs) were compared. Data were analyzed using SPSS v26, with p-values <0.05 considered significant.

Results: Significant differences were observed in several hematological parameters between the two groups. Neonates born to hypertensive mothers had significantly lower cord blood TLC ($p < 0.001$), ANC ($p < 0.001$), and ALC ($p < 0.001$), and higher nRBC count ($p = 0.016$). Day-3 peripheral blood continued to show lower TLC ($p = 0.036$) and ANC ($p = 0.046$) in the hypertensive group. Thrombocytopenia on day 3 was significantly more common among neonates of hypertensive mothers ($p = 0.001$). Additionally, higher rates of small for gestational age ($p = 0.025$), asymmetrical IUGR ($p = 0.038$), and NICU admissions were observed in the hypertensive group, although mortality differences were not statistically significant.

Conclusion: Neonates born to hypertensive mothers show significant hematological alterations, particularly in leukocyte and platelet counts, indicating a higher risk of early neonatal complications. Early identification through cord blood profiling can guide prompt intervention and management. The study emphasizes the importance of close monitoring of neonates born to mothers with hypertensive disorders of pregnancy.

Keywords: Cord blood, Hematological profile, Hypertensive pregnancy, Neonatal outcome, Thrombocytopenia.

INTRODUCTION

Normal pregnancy is associated with wide range of blood changes like neutrophilic leucocytosis, hyperlipidaemia and procoagulant, hypofibrinolytic changes.[1] Maternal physiology endures several alterations in haematological parameters with an average rise of 40-50%in blood and plasma volume during pregnancy.[2] During pregnancy, fluid retention occurs because of sodium and water retention under estrogen and progesterone's hormonal effects, leading to haemodilution. This haemodilution produces pronounced effect in haematocrit values. Pregnancy-induced hypertension is currently believed to be a two stage disease with the shallow trophoblastic invasion of maternal spiral arterioles initially resulting in placental insufficiency.[3,4] Acute or chronic intrapartum insufficiency results in antepartum or intrapartum anoxia that may lead to fetal death, IUGR and or preterm delivery.[5] Neonatal complications occurring in these babies is closely related to severity of hypertension and proteinuria and duration of the disease. Pregnancy induced hypertension is associated with adaptive changes in fetal circulation. Placentally derived factors implicated in the pathogenesis of the maternal manifestations of the disease are known to contribute to the development of neonatal thrombocytopenia and growth restriction. Severe preeclampsia may negatively affect the foetus. This is due to decreased uteroplacental perfusion resulting in increased incidence of IUGR, foetal hypoxia, meconium aspiration syndrome and perinatal death. These babies are frequently delivered prematurely, may tolerate labour poorly, and require resuscitation. PIH is often associated with encephalopathy. A systemic inflammatory response in foetus could explain link between maternal PIH and encephalopathy. While most of the effects of PIH put the foetus and new born at higher risk of complications, preeclampsia in mother may also lead to acceleration of maturation of brain and lungs as an adaptation response to foetal stress. Early initiation of parturition may also be associated. These adaptive changes may represent a lifesaving earlier birth of more mature new born and increased survival as long as the unfavourable foetal environment is not too early or too severe.[5-7] Preeclampsia that requires preterm delivery is associated with adverse maternal and perinatal outcomes in subsequent pregnancies, even if they don't develop preeclampsia in a subsequent pregnancy.8 Foetal mortality markedly increases with rising maternal diastolic blood pressure and proteinuria. Diastolic blood pressure of more than 95 mm hg is associated with a threefold rise in the foetal death rate. Foetal morbidity may include IUGR, foetal acidaemia and complications from prematurity.

Maternal preeclampsia can result in neonatal thrombocytopenia typically defined as platelet count below 1.5 lakhs. Approximately 1/3rd of the infants born to mothers with PIH have decreased platelet count at birth or within the first 2-3 days following delivery, with resolution by 10 days of life in most cases.[8] Severity of thrombocytopenia related to pregnancy induced hypertension is highly variable, with a small percentage of infants developing severe or clinically significant thrombocytopenia (<50000). Haematological abnormalities like neutropenia and thrombocytopenia can lead to serious neonatal complications like sepsis, increased predisposition to infections and disseminated intravascular coagulation (DIC). Its incidence is higher in preterm neonates. Bleeding manifestations including intracranial haemorrhage may result from platelet deficiency due to any cause.[8]

Neonatal outcome is influenced by gestational age and the severity of hypertension as expressed by the need for antihypertensive treatment. The major impact on the foetus is under nutrition because of uteroplacental insufficiency, which leads to growth retardation. The short-term effect observed is restricted foetal growth resulting in greater foetal liability. The foetal health as well as its weight is highly compromised, leading to various degrees of foetal morbidity and foetal damage.

MATERIALS AND METHODS

This case-control study was conducted in the Department of Obstetrics and Neonatal Intensive Care Unit (NICU) of Sri Guru Ram Das Institute of Health Sciences and Research, Amritsar, over a period from February 2020 to June 2021. A total of 90 neonates were enrolled and divided into two groups: the case group included neonates born to mothers diagnosed with hypertensive disorders of pregnancy (HDP), such as gestational hypertension, preeclampsia, eclampsia, chronic hypertension, and superimposed preeclampsia, while the control group comprised neonates born to normotensive mothers with no underlying medical comorbidities. Neonates born to mothers with clinically diagnosed hypertensive disorders of pregnancy were included in the study. Neonates were excluded if they were born to mothers with diabetes mellitus, renal disease, severe anemia, heart disease, or those receiving medications such as aspirin. Additionally, neonates with congenital malformations were excluded from the study.

Methodology

Immediately after delivery, the umbilical cord was clamped and wiped with an antiseptic solution. Using a sterile 23G or 26G needle and syringe, 0.5 mL of umbilical venous blood was drawn and transferred into an EDTA microtainer. Samples were analyzed within one hour using a Beckman Coulter automated hematology analyzer with a 5-part differential cell counter for estimation of

hemoglobin (Hb), packed cell volume (PCV), and red cell indices. Nucleated red blood cells (nRBCs) were assessed through peripheral blood smear stained with Leishman's stain.

On the third postnatal day, 0.5 mL of peripheral venous blood was collected from each neonate for follow-up testing. The venipuncture site was prepared by cleaning with alcohol, followed by application of 0.5% chlorhexidine in alcohol using a back-and-forth motion for 30 seconds. After allowing the site to dry, venipuncture was performed using a sterile needle, and pressure was applied post-procedure to ensure hemostasis. The collected samples were analyzed using the same hematology system to assess serial changes in hematological parameters.

Statistical Analysis

All collected data were systematically compiled in Microsoft Excel and analyzed using IBM SPSS version 26. Quantitative data were summarized as median and interquartile range (IQR), while qualitative data were expressed as frequency and percentage. Comparisons between case and control groups were performed using the Mann-Whitney U test for continuous variables and the Chi-square test for categorical variables. A p-value of <0.05 was considered statistically significant, while $p < 0.001$ was considered highly significant.

RESULTS

In this comparative study of neonates born to hypertensive versus normotensive mothers, maternal age distribution was broadly similar across both groups, with the majority of mothers in both cohorts falling within the 21–30 years age bracket—77.8% in the hypertensive group and 60% in the normotensive group. The mean maternal age was 25.96 ± 4.64 years in the hypertensive group and 26.56 ± 5.86 years in the normotensive group. Parity distribution showed a predominance of primigravida mothers in both groups, comprising 57.8% and 55.6% in the hypertensive and normotensive groups respectively.

Regarding mode of delivery, 82.2% of hypertensive mothers underwent lower segment cesarean section (LSCS) compared to 66.7% in the normotensive group, whereas normal vaginal delivery (NVD) was higher in normotensive mothers (33.3% vs. 17.8%), though this difference was not statistically significant ($p = 0.091$). Neonatal gender distribution showed a predominance of male neonates in both groups—68.9% in the hypertensive group and 60% in the normotensive group ($p = 0.378$).

In terms of birth weight, a higher incidence of low birth weight (LBW) and very low birth weight (VLBW) was observed in the hypertensive group. Notably, 20% of neonates born to hypertensive mothers were VLBW compared to 6.7% in the normotensive group, and 4.4% were extremely low birth weight (ELBW), though the differences did not reach statistical significance ($p = 0.262$). A greater proportion of normotensive neonates (53.3%) had normal birth weight compared to hypertensive neonates (42.2%).

Gestational age analysis revealed that preterm births (before 37 weeks) were more common in the hypertensive group (55.6%) than the normotensive group (35.6%), particularly in the moderate-to-late preterm category (46.7% vs. 28.9%). However, this difference was not statistically significant ($p = 0.294$). Evaluation of intrauterine growth restriction (IUGR) using Fenton's chart showed a significantly higher frequency of SGA/IUGR neonates in the hypertensive group (44.4%) compared to 22.2% in the normotensive group ($p = 0.025$). When analyzed further based on the Ponderal Index, asymmetrical IUGR was significantly more frequent in hypertensive neonates (60%) versus normotensive neonates (20%), indicating uteroplacental insufficiency associated with hypertensive disorders of pregnancy ($p = 0.038$).

The APGAR score distribution at both 1 and 5 minutes post-delivery showed no statistically significant difference between the two groups. At 1 minute, most neonates had scores of 8 or more (75.6% in the hypertensive group vs. 84.4% in the normotensive group, $p = 0.831$). At 5 minutes, APGAR scores of 9 were seen in 84.4% of hypertensive group neonates and 88.9% of normotensive group neonates ($p = 0.639$), indicating overall comparable immediate neonatal outcomes.

Hematological parameters showed several significant differences between the two groups. At day 0 (cord blood), neonates born to normotensive mothers had higher median hemoglobin values [17.2 (15.35–18.55) vs. 16 (14.5–17.65), $p = 0.050$]. Although the difference in hemoglobin levels on day 3 was not significant ($p = 0.766$), total leucocyte count (TLC), absolute neutrophil count (ANC), and absolute lymphocyte count (ALC) were consistently lower in hypertensive group neonates across both time points.

TLC on day 0 was significantly reduced in hypertensive neonates [9,500 (4,600–11,350)] compared to normotensive neonates [14,400 (12,050–22,000), $p < 0.001$]. A similar trend persisted on day 3 ($p = 0.036$). ANC was markedly lower in hypertensive neonates on day 0 [4,940 (1,240–6,804.9) vs. 9,490 (6,533–13,558), $p < 0.001$] and remained significantly reduced on day 3 ($p = 0.046$). ALC was also significantly lower at birth ($p < 0.001$), though it did not reach statistical significance on day 3 ($p = 0.065$).

Nucleated RBCs, a marker of fetal hypoxia or stress, were significantly higher in neonates born to hypertensive mothers [median 9.5 (7–45.5)] than those born to normotensive mothers [median 5 (2.5–11.5), $p = 0.016$]. This indicates increased hematopoietic response due to intrauterine stress in the hypertensive group.

When analyzed categorically, 14 out of 45 neonates in the hypertensive group had hemoglobin <13.5 g/dL on day 0 compared to only 3 in the

normotensive group (p = 0.107). Thrombocytopenia was more evident in the hypertensive group on day 3, with 20 neonates showing platelet counts <1.5 lakhs/mm³ compared to only 7 in the normotensive group—a statistically significant difference (p = 0.001). Furthermore, leukopenia (TLC <9,000/mm³) and neutropenia (ANC <1,500/mm³) were significantly more common in the hypertensive group on day 0 (p < 0.001). Lymphopenia (ALC <3,000/mm³) also occurred more frequently in the hypertensive group (p = 0.003).

Regarding NICU admissions, 68.9% of neonates born to hypertensive mothers required NICU care compared to 60% in the normotensive group, though the difference was not statistically significant (p = 0.397). Mortality was higher among neonates in the hypertensive group, with 5 deaths (11.1%) compared to only 1 (2.2%) in the normotensive group, but this difference also did not achieve statistical significance (p = 0.091).

Table 1: Maternal and Neonatal Characteristics in Hypertensive vs. Normotensive Groups

Parameter	Category	Hypertensive (n, %)	Normotensive (n, %)	*p*-value
Maternal Age (years)	≤20	5 (11.1%)	8 (17.8%)	—
	21–30	35 (77.8%)	27 (60.0%)	—
	31–40	5 (11.1%)	10 (22.2%)	—
	Mean ± SD	25.96 ± 4.64	26.56 ± 5.86	—
Parity	Primigravida	26 (57.8%)	25 (55.6%)	—
	Multigravida	19 (42.2%)	20 (44.4%)	—
Mode of Delivery	LSCS	37 (82.2%)	30 (66.7%)	0.091
	NVD	8 (17.8%)	15 (33.3%)	
Gender of Neonates	Male	31 (68.9%)	27 (60.0%)	0.378
	Female	14 (31.1%)	18 (40.0%)	
Birth Weight	ELBW (<1 kg)	2 (4.4%)	0 (0.0%)	0.262
	VLBW (1–1.5 kg)	9 (20.0%)	3 (6.7%)	
	LBW (1.6–2.5 kg)	15 (33.3%)	18 (40.0%)	
	Normal (2.6–4 kg)	19 (42.2%)	24 (53.3%)	
Gestational Age	Extremely preterm (<28 wks)	0 (0.0%)	0 (0.0%)	0.294
	Early preterm (28–31.6 wks)	4 (8.9%)	3 (6.7%)	
	Mod-to-late preterm (32–36.6 wks)	21 (46.7%)	13 (28.9%)	
	Early term (37–38.6 wks)	14 (31.1%)	21 (46.7%)	
	Full term (39–40.6 wks)	6 (13.3%)	8 (17.8%)	
SGA/IUGR (Fenton's Chart)	AGA	25 (55.6%)	35 (77.8%)	**0.025**
	SGA/IUGR	20 (44.4%)	10 (22.2%)	
Type of IUGR (Ponderal Index)	Symmetrical	8 (40.0%)	8 (80.0%)	**0.038**
	Asymmetrical	12 (60.0%)	2 (20.0%)	

Table 2: Frequency and Percentage Wise Distribution of Neonates on the Basis of Apgar Scores

APGAR Score		Hypertensive		Normotensive		p value
		N	%	n	%	
at 1min	3	1	2.2%	0	0.0%	0.831
	4	1	2.2%	0	0.0%	
	6	4	8.9%	3	6.7%	
	7	5	11.1%	4	8.9%	
	8	34	75.6%	38	84.4%	
at 5min	7	1	2.2%	0	0.0%	0.639
	8	6	13.3%	5	11.1%	
	9	38	84.4%	40	88.9%	

Table 3: Comparison of Hematological Parameters in Neonates Born to Hypertensive vs. Normotensive Mothers

Parameter	Time Point	Range	Hypertensive (n)	Normotensive (n)	*p*-value
Hemoglobin (g/dL)	Day 0	<13.5	8	3	0.107
		≥13.5	37	42	
	Day 3	<13.5	7	6	0.624
		≥13.5	33	36	
MCV (fL)	Day 0	<95 or >125	4	2	0.291
		95–125	41	43	
	Day 3	<95 or >125	2	5	0.263
		95–125	38	37	
MCH (pg)	Day 0	<31 or >37	12	13	0.813
		31–37	33	32	
	Day 3	<31 or >37	8	11	0.506
		31–37	32	31	
MCHC (g/dL)	Day 0	<30 or >35	3	2	0.645
		30–35	42	43	
	Day 3	<30 or >35	5	9	0.282

		30–35	35	33	
PCV (%)	Day 0	>65	1	2	0.557
		≤65	44	43	
	Day 3	>65	1	0	0.613
		≤65	39	42	
Platelet Count (lakhs/mm³)	Day 0	<1.5	10	8	0.598
		1.5–3.5	35	37	
	Day 3	<1.5	20	7	**0.001**
		1.5–3.5	20	35	
TLC (×10³/mm³)	Day 0	<9	19	4	**<0.001**
		9–30	26	41	
	Day 3	<9	22	16	0.124
		9–30	18	26	
ANC (/mm³)	Day 0	<1500	14	1	**<0.001**
		≥1500	31	44	
	Day 3	<1500	5	0	0.062
		≥1500	35	42	
ALC (/mm³)	Day 0	<3000	17	3	**0.003**
		≥3000	28	42	
	Day 3	<3000	22	16	0.124
		≥3000	18	26	
Nucleated RBCs (peripheral smear)	Day 0	Present	24	15	0.055
		Absent	21	30	

Table 4: Comparison of Hematological Profile of Neonates Born to Hypertensive vs. Normotensive Mothers (Day 0 and Day 3)

Parameter	Day	Hypertensive *(Median, IQR)*	Normotensive *(Median, IQR)*	*p*-value
Hemoglobin (g/dL)	Day 0	16 (14.5–17.65)	17.2 (15.35–18.55)	0.050
	Day 3	17.25 (14.8–18.075)	16.7 (15.4–19.025)	0.766
Platelet Count (/mm³)	Day 0	170,000 (150,000–247,000)	209,000 (163,500–270,500)	0.200
	Day 3	173,500 (78,500–250,000)	193,500 (159,500–225,500)	0.489
Total Leucocyte Count (/mm³)	Day 0	9,500 (4,600–11,350)	14,400 (12,050–22,000)	**<0.001**
	Day 3	8,200 (5,600–11,275)	10,800 (7,575–13,400)	**0.036**
Absolute Neutrophil Count (/mm³)	Day 0	4,940 (1,240–6,804.9)	9,490 (6,533–13,558)	**<0.001**
	Day 3	4,465.5 (2,920–6,897.75)	6,289 (4,038–8,369)	**0.046**
Absolute Lymphocyte Count (/mm³)	Day 0	3,332 (2,685–4,595)	4,960.8 (3,908.5–6,430)	**<0.001**
	Day 3	2,937.5 (1,725–3,978)	3,423 (2,700–4,667.5)	0.065
Nucleated RBCs (/100 WBCs)	Day 0	9.5 (7–45.5)	5 (2.5–11.5)	**0.016**
	Day 3	—	—	—

Table 5: Distribution of Neonates Who Required Admission to NICU

NICU Admission Needed or Not	Hypertensive		Normotensive		p value
	n	%	n	%	
No	14	31.1%	18	40.0%	0.397
Yes	31	68.9%	27	60.0%	

Table 6: Frequency and Percentage Wise Distribution of Neonatal Deaths

Mortality	Hypertensive		Normotensive		p value
	n	%	n	%	
No	40	88.9%	44	97.8%	0.091
Yes	5	11.1%	1	2.2%	

DISCUSSION

Majority of neonates in our study were born to mothers in the age group of 21 to 30 years (n = 35, 77.8% in hypertensive and n = 27, 60% in normotensive), consistent with the findings of Annam et al,[9] who stated that hypertensive mothers are generally younger, with mean age ranging from 21–30 years. Neonates born to primigravida mothers were more frequent in both groups (57.8% in hypertensive and 55.6% in normotensive), suggesting a higher prevalence of PIH in first pregnancies, in agreement with Kaul et al.[10]

In our study, 82.2% of neonates born to PIH mothers were delivered via caesarean section, compared to 66.7% in the normotensive group. Though not statistically significant, this aligns with findings by Sikha Maria Siromani et al,[11] where 70.67% of hypertensive mothers underwent LSCS. Thus, mode of delivery had no significant association in relation to hypertensive status.

Prematurity was more common among hypertensive mothers (55.6%) than normotensive (35.6%), particularly in the late preterm range (46.7%). While this difference was statistically insignificant (p = 0.294), it is in agreement with Carl H Backes et al,[12] who highlighted a higher proportion of late preterm births in preeclampsia. Our findings contrast with Sikha Maria Siromani et al,[11] who reported 63% preterm births with statistical

significance, and Solange Regina et al,[13] who noted a 10.9% preterm birth rate.

Low birth weight (LBW) was observed in 57.7% of PIH neonates, with 20% being VLBW and 4.4% ELBW. In normotensive mothers, 46.7% of neonates were LBW or VLBW. This difference was not statistically significant (p = 0.262), though comparable to Shweta Anand et al,[14] who reported 60% prevalence of LBW in PIH babies.

Intrauterine growth restriction (IUGR) was significantly higher in the hypertensive group (44.4% vs. 22.2%, p = 0.025). Asymmetrical IUGR was predominant among PIH babies (60% vs. 20%, p = 0.038), suggestive of uteroplacental insufficiency. These findings were not in accordance with Shweta Anand et al,[14] who reported 71% asymmetrical IUGR.

Regarding Apgar scores, 6 PIH neonates (12%) had scores <7 at 1 minute, and only 1 neonate had persistently low score at 5 minutes. In the normotensive group, 3 neonates had low Apgar scores initially, all of whom improved. These findings contrast with Sulaeman A Susilo et al,[15] who reported 19% and 5.4% low Apgar scores at 1 and 5 minutes respectively.

As per Avery et al,[16] hemoglobin <13.5 g/dL is considered low for neonates weighing 1.2–2.5 kg. In our study, 8 PIH neonates had Hb <13.5 at birth, and 7 at day 3, compared to 3 and 6 in normotensive. These differences were statistically insignificant (p = 0.107 and 0.624). Similarly, PCV >65% was seen in 1 PIH neonate at birth and day 3, and 2 in normotensive at birth (p > 0.05). Kurlat et al,[17] showed increased polycythemia in PIH neonates, likely due to placental hypoperfusion.

Red cell indices (MCV, MCH, MCHC) were mostly within normal limits in both groups. No significant differences were found for MCV (p = 0.291 and 0.263), MCH (p = 0.813 and 0.506), and MCHC (p = 0.645 and 0.282), indicating that erythrocyte morphology is not significantly affected by maternal hypertension.

As per Avery et al,[16] normal TLC in neonates ranges from 9,000–30,000/mm³. In our study, 19 PIH neonates had leucopenia on day 0 and 22 on day 3. In normotensive group, the numbers were 4 and 16 respectively. Leucopenia in PIH neonates was statistically significant (p < 0.001). This supports the hypothesis by Koenig and Christensen, who linked neutropenia to decreased granulopoiesis due to growth factor imbalance and hypoxia-induced shift towards erythropoiesis as noted by Mollaem and Koenig.[18]

Thrombocytopenia (<1.5 lakh/mm³) was significantly more common in PIH neonates (p = 0.001). Twenty PIH neonates had low platelet counts on day 3 compared to 7 normotensive neonates. The 22.2% incidence of thrombocytopenia in PIH neonates aligns with Sandhya Sivakumar et al,[19] Kleckner et al,[20] suggested that placental endothelial pathology may result in thrombocyte destruction, resolving after birth.

ANC <1500/mm³ was noted in 14 PIH neonates at birth and 5 on day 3, while only 1 normotensive neonate showed low ANC at birth. This difference was statistically significant (p < 0.001). Similarly, ALC <3000/mm³ was observed in 17 PIH neonates at birth and 22 on day 3, compared to 3 and 16 in normotensive (p = 0.003 at birth).

Nucleated RBCs were present in 53.3% of PIH neonates and 33.3% of normotensive neonates (p = 0.016), consistent with findings from Sandhya Sivakumar et al,[19] suggesting chronic fetal hypoxia in hypertensive pregnancies.

NICU admission was slightly higher in PIH neonates (68.9%) compared to normotensive (60%) though statistically insignificant (p = 0.397). Neonatal mortality was higher in the PIH group (11.1%) than normotensive (2.2%) but did not reach statistical significance (p = 0.091). Ravikant Patel et al,[21] and Bangal et al,[22] reported lower mortality rates in similar cohorts.

CONCLUSION

We concluded in our study that there are significant derangements in the cord blood hematological profile of neonates born to mothers with PIH as compared to normotensive mothers. There was increased need of NICU admission and increased risk of mortality in neonates born to mothers with hypertensive disorders of pregnancy. PIH is a maternal pathology involving placental modification which is associated with fatal complications. Babies born to hypertensive mothers are prone to develop several complications due to derangements in the cord blood haematological profile in the form of leukopenia, absolute neutropenia, and thrombocytopenia, as well as increased RBC count.

REFERENCES

1. Catrino C, Rebelo l, Belo L, Quintanilha A, Santos-Silva A. Umbilical cord blood changes in neonates from a preeclamptic pregnancy. From Preconception to postpartum. London: IntechOpen ; 2012: 269-87.
2. Timilsina S, Karki S, Gautam A, Bhusal P, Paudel G, Sharma D. Correlation between maternal and umbilical cord blood in pregnant women of Pokhara Valley: a cross sectional study. BMC Pregnancy Childbirth. 2018 Mar 21;18(1): 70.
3. Avery's disease of the newborn. H. William Tanesh, Roberts A. Ballard (eds) 10th ed. Elsevier, New Delhi; 2017: 99-105.
4. Steer P LMEO-N. Hypertensive disease in pregnancy. In: Rennie & Roberton's Textbook of Neonatology. 5th ed. Churchill Livingstone; 2012 :179–81.
5. Cunningham, Leveno, Bloom, Hauth, Rouse, Spong-Hypertensive disorders of pregnancy. William's Obstetrics 25th edition. McGraw Hill 2019: 268-274.
6. Yong HE, Melton PE, Johnson MP, Freed KA, Kalionis B, Murthi P, et al. Genome-wide transcriptome directed pathway analysis of maternal pre-eclampsia susceptibility genes. Plos ONE. 2015 ;10(5) :e0128230: 1-15.
7. Maher GM, O'Keeffe GW, Kearney PM, Kenny LC, Dinan TG, Mattsson M, et al. Association of hypertensive disorders of pregnancy with risk of neurodevelopmental disorders in offspring: a systematic review and meta-analysis. JAMA Psychiatry. 2018 ;75(8): 809-19.

8. Patil S, Mangshetty R, Patil B. Outcome of Neonates with Thrombocytopenia. J Evol Med Dent Sci. 2014; 3(17): 453-8.
9. Annam V, Srinivasa K, Yatnatti SK. Evaluation of platelet indices and platelet counts and their significance in pre-eclampsia and eclampsia. Int J Biol Med Res. 2011;2(1):425–8.
10. Kaul V, Bagga R, Jain V, Kalra J, Gopalan S. Pregnancy outcome in women with severe anemia. Int J Gynecol Obstet. 2002;77(1):47–9.
11. Siromani SM, Varahala AM, Gopu S, Chidugulla SK. Neonatal outcome in pregnancy induced hypertensive mothers – A tertiary care experience. IOSR J Dent Med Sci. 2015;14(11):23–7.
12. Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ. Maternal preeclampsia and neonatal outcomes. J Pregnancy. 2011; 2011:214365.
13. Chiam SRP, Oliveria SMJ, Kimura AF. Pregnancy-induced hypertension and the neonatal outcome. Acta Paul Enferm. 2008;21(1):53–8.
14. Shweta A, Kirshna A. Perinatal outcome in growth retarded babies born to normotensive and hypertensive mothers – A prospective study. Peoples J Sci Res. 2012;5(1):24–8.
15. Sulaeman A, Susilo KN, Adly NA, Fattah R, Irwinda N. Determinants of low APGAR score among preeclamptic deliveries in Cipto Mangunkusumo Hospital: A retrospective cohort study in 2014. Med J Indones. 2015;24(3):183–9.
16. Weindling M. Avery's diseases of the newborn. Semin Fetal Neonatal Med. 2013;18(1):65.
17. Kurlat I, Quijano G, Ramirez JA, Klein F, Grigera J, Colantonio SE. Polycythemia in newborns of mothers with hypertension. Arch Argent Pediatr. 2003;101(2):97–102.
18. Moallem M, Koenig JM. Preeclampsia and neonatal neutropenia. NeoReviews. 2009;10(9):e454–9.
19. Sivakumar S, Bhat BV, Badhe BA. Effect of pregnancy induced hypertension on mothers and their babies. Indian J Pediatr. 2007;74(7):623–5.
20. Kleckner HB, Giles HR, Corrigan JJ. The association of maternal and neonatal thrombocytopenia in high-risk pregnancies. Am J Obstet Gynecol. 1977;128(3):235–8.
21. Patel R, Patel SD, Shah D, Bala DV. A study on perinatal outcomes in eclampsia in a tertiary care hospital. NHL J Med Sci. 2014;3(1):26–9.
22. Bangal VB, Giri PA, Shinde KK, Gavhane SP. A study of maternal and fetal outcome in pregnancy induced hypertension in a tertiary care teaching hospital. Int J Biomed Adv Res. 2012;3(3):142–5.