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The Effect of Pregnancy Induced Hypertension on Complete Blood Count of Newborn

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Abstract

Background: Pregnancy induced hypertension is one of the most common causes of maternal and neonatal mortality and morbidity. New born delivered to mothers with hypertension are more liable for intrauterine growth retardation and may be delivered prematurely. We aimed to determine the effect of pregnancy-induced hypertension on complete blood count, gestational age and birth weight of newborn.

Materials and Methods: This study includes 200 neonates, 100 neonates born to mothers diagnosed as having pregnancy induced hypertension this is considered as the study group and 100 neonates born in the same period to healthy mothers considered as the control group, the study conducted in the neonatal unit of the department of pediatrics and gynecological ward in AL-Imamein Al-Kadhimein Medical city, Iraq, from 1st August 2015 to 30th May 2016. Details of the baby including gender, Apgar score, birth weight and gestational age were recorded and hematological parameters (complete blood count) of both babies and mothers have been studied.

Results: The study showed the significant difference between the two groups regarding mode of delivery, 94% of mothers in study group delivered by cesarean section while 60% of control group, delivered by cesarean section. There was the significant effect of gestational hypertension on gestational age, birth weight, need for resuscitation, Apgar score, mean WBC count and mean platelet count since ($P< 0.05$). There were no significant differences between two groups regarding another element of complete blood count.

Conclusion: At current study, gestational hypertension affects adversely growth parameter, wellbeing and both WBC and platelet count of a newborn.

Key Words: Apgar score, Complete blood count, Gestational hypertension, Hematology, Newborn.

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1- INTRODUCTION

Hypertensive disorders complicate 5 - 10% of all pregnancies, and together they are one member of the deadly triad—along with hemorrhage and infection—that contributes greatly to maternal morbidity and mortality.

Of these disorders, the preeclampsia syndrome, either alone or superimposed on chronic hypertension, is the most dangerous.

The basic classification was retained, as it describes four types of hypertensive disease:

1. Gestational hypertension.
2. Preeclampsia and eclampsia syndrome.
3. Chronic hypertension of any etiology.
4. Preeclampsia superimposed on chronic hypertension.

Importantly, this classification differentiates the preeclampsia syndrome from other hypertensive disorders because it is potentially more ominous (1).

Gestational hypertension is defined as new hypertension presenting after 20 weeks without significant proteinuria, this diagnosis is made in women whose blood pressures reach 140/90 mm Hg or greater for the first time after 20 weeks of gestation, but in whom proteinuria is not identified (2).

Preeclampsia changes the intrauterine environment of the fetus, and the fetus has to adapt to living in the unfavorable environment. These effects include an increased neonatal mortality and morbidity; intrauterine growth restriction; premature birth; hematological abnormalities, such as thrombocytopenia, polycythemia, and neutropenia; necrotizing enterocolitis; bronchopulmonary dysplasia (3).

Several hematological abnormalities are associated with the preeclampsia syndrome. Among those commonly identified is thrombocytopenia, which at times may become severe enough to be life-threatening (1).

Neonatal thrombocytopenia defined as a platelet count less than 150,000/μL based upon the definition used in adults. The degree of severity of thrombocytopenia can be further subcategorized according to platelet count in the affected individuals:

- Mild thrombocytopenia—platelets count 100,000 to 150,000/μL,
- Moderate thrombocytopenia—platelets count 50,000 to 99,000/μL,
- Severe thrombocytopenia—platelets count < 50,000/μL .

The pathogenesis of thrombocytopenia among infants born to mothers with preeclampsia is presently unknown. The principal mechanism postulated is that preeclampsia and the resultant fetal hypoxia have a direct depressant effect on fetal megakaryocytopoiesis and platelet production the combined effect of impaired megakaryocyte formation and increased platelet activation mediated through cytokines, thrombopoietin, and interleukin-6 are said to be the most likely causative mechanisms (4).

Neutropenia is a common hematologic disorder in the newborn intensive care unit, particularly in preterm neonates. Although its cause varies, a significant proportion of the episodes are associated with the pregnancy complicated by preeclampsia (5).

Neonates delivered to women with preeclampsia have a 50% incidence of neutropenia (defined as absolute neutrophil count less than 500 μL). Neutropenia has a variable course, typically lasting days to weeks in affected infants. The biological

mechanism for preeclampsia resulting in neonatal neutropenia has not been fully elucidated (6). Intrauterine Growth Restriction (IUGR) is defined as birth weight less than 10th centile for gestational age. Infants with IUGR or Small for Gestational Age (SGA) are at increased risk of perinatal morbidity and mortality. They also have higher rates of physical, neurological and mental impairment than babies with appropriate intrauterine growth (7).

Fetal growth is a useful marker for fetal well-being. Pregnancies complicated by IUGR, defined as a pathological process of reduced fetal growth, have been associated with an increase in perinatal mortality. Preeclampsia is a significant risk factor in the development of IUGR and represents the most common cause of IUGR in the non-anomalous infant (8).

2- MATERIALS AND METHODS

2-1. Study design and setting

This case-control study was conducted in the neonatal unit of the department of pediatrics and gynecological ward in AL-Imamein Al-Kadhimein Medical city, Iraq, from 1st August 2015 to 30th of May 2016.

2-2. Participants and Data Collection

This study includes 200 neonates, 100 neonates born to mothers diagnosed as having pregnancy induced hypertension this is considered the study group and 100 neonates born in the same period to healthy mothers this is considered the control group.

The mothers in this study were diagnosed as having pregnancy induced hypertension when their blood pressure was equal or greater than 140 \ 90 mmHg, started after 20 weeks of gestation, if associated with proteinuria it was classified as preeclampsia and if not associated with proteinuria it was classified as gestational hypertension. Any babies with anemia at birth, ABO and Rh incompatibility, twins, severe birth asphyxia or congenital malformation were excluded from this study. Also babies born to mothers who have an acute illness like antepartum hemorrhage and chronic illness likely to cause changes in hematological profile like severe anemia, connective tissue disorders, diabetes mellitus and chronic hypertension were also excluded from the study.

2-3. Clinical Parameters

Maternal details including age, parity, time of delivery, and mode of delivery or any complications during labor were also recorded. Details of the baby including sex need for resuscitation; Apgar score, birth weight and gestational age (by modified bollards scoring) were recorded.

According to normal hematological values in infancy and childhood showed in **Figure.1**, two ml of EDTA venous blood was collected within 24 hours of delivery from both study and control groups and the following hematological parameters have been studied: these included hemoglobin, total and differential WBC, platelet count, red cell indices MCH, MCV, and MCHC. These parameters were also studied in the mothers of these babies. The blood film was difficult to be done for all the patients because of that not available in the lab over the all-time of the study.

2-4. Statistical Analysis

The statistical analysis was performed using Chi-square test. The lower level of accepted statistical significant difference is equal or below 0.05.

	Hemoglobin (g/dL)		Hematocrit (%)			Leukocytes (per mm^3)		Neutrophils (%)		Differential Counts			
Age	Mean	Range	Mean	Range	Reticulocytes (%) Mean	Mean	Range	Mean	Range	Lymphocytes (%) Mean	Eosinophils (%) Mean	Monocytes (%) Mean	Nucleated Red Cells/100 WBCS
Cord blood	16.8	13.7-20.1	55	45-65	5	18,000	9000-30,000	61	40-80	31	2	6	7
2 wk	16.5	13-20	50	42-66	1	12,000	5000-21,000	40		48	3	9	3-10
3 mo	12.0	9.5-14.5	36	31-41	1	12,000	6000-18,000	30		63	2	5	0
6 mo-6 yr	12.0	10.5-14	37	33-42	1	10,000	6000-15,000	45		48	2	5	0
7-12 yr	13.0	11-16	38	34-40	1	8000	4500-13,500	55		38	2	5	0
Adult													
Female	14.0	12-16	42	37-47	1.6	7500	5000-10,000	55	35-70	35	3	7	0
Male	16.0	14-18	47	42-52									

Fig.1: Normal hematological values in infancy and childhood.

3-RESULTS

Two hundred newborn babies and their mothers were included in this study, 100 newborns of mothers with gestational hypertension (study group) 42 male and 58 females. Of one hundred newborns of healthy mothers (control group), 56 were female and 44 were male. The male: female ratio of the study group 0.72:1 while it's 0.78:1 for the control group. Regarding age distribution, the mean age of the hypertensive mothers was (30.86±6.02) years while that of normotensive mothers was (26.08±3.71) years.

The majority of the mothers were between (20 – 30) years old (64% of hypertensive mothers and 84% of normotensive mothers), but 36% of hypertensive mothers were above 31 years, while only 16% of normotensive mothers were above 31 years (P<0.001). Cesarean section was the major mode of delivery in the hypertensive mothers (94%) compared with (60%) in the normotensive mothers (P<0.001). Preterm delivery in hypertensive mothers (64%) was significantly higher than in normotensive mothers (44%) (P < 0.005).

The percentage of primipara was higher in the hypertensive mothers than in the normotensive mothers (52% vs. 35%) (P=0.022). All these data are shown in **Table.1**. The mean gestational age of the study group was (36.68±1.81) weeks which is lower than the gestational age of the control group (37.40±1.02) weeks (P<0.001).

The mean birth weight was lower in the study group (2.56 kg) than in the control group (2.96 kg). P value was highly significant. The percentage of babies need resuscitation is higher in the study group than in the control group (16% vs. 6%). P value was statistically significant (P=0.006).

In this study, 40% of the study group was small for the gestational age while only 20% of the control group was small for gestational age. P value was statistically significant (P= 0.002). The Apgar score in the study group at 5 minutes was significantly lower than that of the control group (8.62±1.7 vs. 9.44±0.7) (P<0.001). The Apgar score at 1 and 10 minutes of both study and control group showed no significant difference. The neonatal profile is shown in **Table.2**.

There was no difference in platelets count of hypertensive and normotensive mothers (240.12X109/L vs. 241.72X109/L), but the mean platelets count in the study group was significantly lower than that of the control group (246.98±81.11) X109/L vs. (281.08±92.66)X109/L. P value was significant (P=0.007).

There was no significant difference in the WBC count of the mothers while the mean WBC count in the study group was 16.14±5.13) x 109/L compared with the control group (20.58±13.12) x 109/L. P value was significant (P=0.002). Regarding other parameters (Hb, MCV, MCH, MCHC, and neutrophil) no significant difference between study and control groups. The above data are shown in **Table.3**. The percentages of thrombocytopenia in newborns are shown in **Figure.2**. In this study 16% of the hypertensive mothers have preeclampsia and 84% have Gestational hypertension thrombocytopenia was found in 50% of neonates of preeclamptic mothers while it's found only in 9.5% of neonates of mothers with gestational hypertension, the P value is highly significant.

The Hemoglobin and WBC count showed no statistically significant difference between mothers with preeclampsia and those with gestational hypertension and their babies. All these parameters are shown in **Table.4**.

Table-1: Profile of the hypertensive and normotensive mothers

Variables		Hypertensive mothers	Normotensive mothers	P- value
Age (year)		30.86±6.02	26.08±3.71	<0.001
Age groups	20-30 years	64 (64%)	84(84%)	0.001
	31-40 years	26 (26%)	16(16%)	
	>40 years	10 (10%)	0	
Mode of Delivery	Normal	6 (6%)	40(40%)	<0.001
	Caesarians section	94 (94%)	60(60%)	
Time of Delivery	Pre-term	64 (64%)	44(44%)	0.005
	Term	36 (36%)	56(56%)	
Primipara	Yes	52 (52%)	35 (35%)	0.022

NS: non statistical significance (P>0.05); Statistical significance (P≤0.05); Highly statistical significant difference (P≤0.001).

Table-2: Comparison between the mean neonatal profile of the study and control groups

Variables		Study group	Control group	P-value
Mean Gestational age (week)		36.68±1.81	37.40±1.02	<0.001
Birth weight (kg)		2.56±0.64	2.96±0.48	<0.001
Gender	Female	58 (58%)	56 (56%)	0.775
	Male	42 (42%)	44 (44%)	
Need for resuscitation	Yes	16 (16%)	6 (6%)	0.006
	No	84 (84%)	94 (94%)	

Small for gestational age	Yes	40 (40%)	20 (20%)	0.002
	No	60 (60%)	80 (80%)	
Mean gestational age (weeks)	Pre-term	35.81±1.72	36.45±0.79	0.023
	Term	38.22±0.42	38.14±0.35	0.332
APGAR score	1 minute	6.8±1.54	7.2±1.66	0.079
	5 minutes	8.62±1.7	9.44±0.7	<0.001
	10 minutes	9.9±1.49	9.88±0.59	0.901

NS: Non statistical significance (P>0.05); Statistical significance (P≤0.05); Highly statistical significant difference (P≤0.001).

Table-3: Complete blood count of the study and control groups and their mothers

Variables	Mother			Newborn		
	Hypertensive Mean (± SD)	Normotensive Mean (± SD)	P-value	Study Group Mean (± SD)	Control group Mean (± SD)	P-value
Hb (g/dl)	11.62±1.53	11.85±1.23	0.244	17.27±2.29	16.86±1.33	0.132
WBC (x10^9/L)	12.80±4.20	12.69±4.28	0.859	16.14±5.13	20.58±13.12	0.002
Neutrophil count (x10^9/L)	9.76±3.65	10.25±4.07	0.371	8.49±3.76	8.70±3.92	0.709
Platelets count (x10^9/L)	240.12±77.93	241.72±72.04	0.880	246.98±81.11	281.08±92.66	0.007
MCV (fL)	86.18±6.49	87.21±6.07	0.246	106.16±4.74	103.54±14.43	0.087
MCH (pg)	28.67±3.17	29.18±3.44	0.277	35.64±2.31	35.45±1.03	0.480
MCHC (g/l)	32.97±1.83	32.81±1.27	0.480	34.16±1.49	33.71±1.33	0.029
Thrombocytopenia				16%	4%	0.008

NS: Non statistical significance (P>0.05); Statistical significance (P≤0.05), SD: Standard deviation.

Table-4: Hematological profile of mothers with gestational hypertension, preeclampsia and their babies

Variables	Baby		P-value	Mother		P-value
	Gestational hypertension (n=84)	Preeclampsia (n=16)		Gestational hypertension (n=84)	Preeclampsia (n=16)	
Hb (g/dL)	17.34±1.96	16.93±3.65	0.667	11.52±1.56	12.16±1.31	0.123
WBC (X10^9 /L)	16.33±4.25	15.19±8.51	0.608	12.94±4.18	12.04±4.38	0.432
Platelets (X10^9 /L)	247.74±73.88	243±114.88	0.876	238.71±78.59	247.5±76.41	0.682
Thrombocytopenia	8/84(9.5%)	8/16(50%)	<0.001	10/84(12%)	4/16(25%)	0.231

NS: Non statistical significance (P>0.05); Statistical significance (P≤0.05); Highly statistical significant difference (P≤0.001).

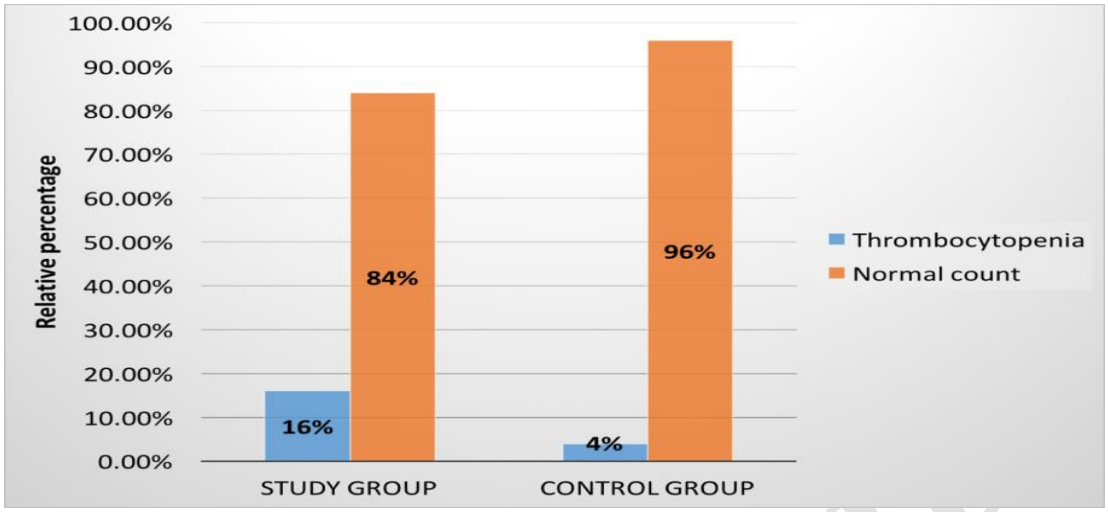

Fig.2: Percentage of thrombocytopenia in study and control group.

4- DISCUSSION

Pregnancy induced hypertension is one of the most common causes of both maternal and neonatal morbidity. A newborn delivered to mothers with hypertension are more liable for intrauterine growth restriction and may be delivered prematurely. In this study, the mean age of Hypertensive mothers group was (30.86±6.02) years and the normotensive mother's groups (26.08±3.71) years. The similar result was shown by Onisai et al. (2009) who observed that the mean age of PIH was 29.8 years old (9). This was disagreed by Annam et al., (2011) who stated that hypertensive mothers are younger, mean age is (24.57±3.46) years (10).

We noticed that the age group of hypertensive mothers greater than 31 years (36%) compared with (16%) in the normotensive group with the statistically significant P value. This result agreed with Xiong et al. (2002) stated that (37.4%) of hypertensive mothers were over 30 years old (11). Cesarean section was the major mode of delivery in hypertensive mothers (94%) compared with 60% in the normotensive mothers. Similar data were observed by Coelho et al. (2004), where the global occurrence of cesarean was 73.3% (12). On the other hand, Buga et al. (1999) stated that the prevalence of cesareans among hypertensive women was 30.2 % (13); this can be explained by the fact that at the time of the last study there were limitations in diagnostic tools to assess fetal distress which is a major cause of cesarean. The rate of pre-term delivery in hypertensive mothers was 64% and it was significantly higher than normotensive mothers 44% with a statistical significant association.

Also, the mean birth weight was statistically significantly lower among infants of hypertensive mothers 2.56 kg when compared to those belongs to normotensive mothers 2.96 kg. Xiong et al. (2000) observed a significantly decreased mean birth weight among babies born to mothers with preeclampsia (2.9 kg) as compared with those born to normotensive mothers (3.3kg), the proportion of preterm births among preeclamptic patients was high (27.5%) (14). Also Xiong, et al. 2002 reported that there may be an increasing fetal growth restriction among hypertensive mothers.

These mothers often deliver prior to term with low birth weight (11). This difference could be related to the better regular antenatal care. The percentage of primiparity is higher in hypertensive mothers 52% than normotensive mothers 35%. This finding has been agreed with Xiong et al 2000 (14), Jahromi et al 2009 (15), who reported that the percentage of primigravidas in induced hypertension (PIH) was 60% and 56%, respectively. In this study, Apgar score was lower in the study group at the 5 minutes (8.62±1.7) compared with the control group (9.44±0.7) which is statistically significant. Oliveira et al. (2006) showed that most newborns (99.2%) had Apgar score ranging from (7-8.5) in the fifth minutes of life (16). In this study, 40% of babies born to hypertensive mothers were small for the gestational age while only 20% of babies born to normotensive mothers were small for gestational age which is statistically significant. Studies were done by Xiong et al. (2000) (14). Eskenazi et al. (1993) stated that PIH, especially preeclampsia, was associated with an increased incidence of intrauterine growth restriction and low birth weight (38%, vs. 11%) respectively (17).

The mean platelets count in infants born to hypertensive mothers was significantly lower than of the normotensive group (246.98±81.11) vs. (281.08±92.66). We reported that 16% of babies born to hypertensive mothers have been developed thrombocytopenia compared with only 4% of those of normotensive mothers (P= 0.008). The rate of occurrence of thrombocytopenia in this study was less than other studies like in the study done by Xiong et al., (2000) who reported thrombocytopenia in 22% (14), and Brazy et al., who reported thrombocytopenia in 34% of the newborn of hypertensive mothers (18). This differs from the study done by Burrows et al. (1990), who reported that there is no difference in the incidence of thrombocytopenia in the newborn of pregnancy induced hypertension and those of normotensive mothers (19). The total WBCs count showed significant difference between the mean WBC count in the study and control groups (16.14±5.13 and 20.58±13.12), P-value significant (0.002), done by other studies done by Moallem and Koenig (2009) (5) and Backes et al. (2011)(6), showed decrease in neutrophil count in newborn of hypertensive mothers rather than total WBC count, other study by Xiong et al., (2000) that showed no statistical significant of the mean WBC count between the study and control groups (14).

In preeclamptic mothers 50% of their babies developed thrombocytopenia while in gestational hypertension only 9.5% have thrombocytopenia which is highly significant statistically and this result is similar to a study done by Bhat and Cherian (2008) who reported that in neonates of mothers with preeclampsia, the percentage of neonatal thrombocytopenia was 36% and it is more likely to occur in preterm and low birth weight infants (20). Other hematological indices in both study and control groups showed no significant difference.

4-1. Limitations of the study

In this study the data collection, it was reflecting percentage of our province and not- all cities in our country. The children including in our study composed about all cases attend to hospital. We need more information and studies for covering such subjects.

5- CONCLUSION

It is concluded that mothers with pregnancy induced hypertension are more liable to be delivered by cesarean section, their babies are more liable to be premature and to have lower body weight for gestational age, low Apgar score and

neonatal resuscitation is more frequent in babies of hypertensive mothers than those of normotensive mothers. Neonates of hypertensive mothers are more liable to have lower WBC and thrombocytopenia and this risk increase when the mothers develop preeclampsia. Hemoglobin means corpuscular volume, mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration was not affected by pregnancy-induced hypertension in the mothers.

5-1. RECOMMENDATIONS

We recommend encouraging regular antenatal care for more effective preventive and therapeutic measures of pregnancy induced hypertension. And for further research, large population based studies are recommended in order to determine the scope of this problem nationwide and include more associated factors.

6- ABBREVIATION

- WBC: white blood cell,
- MCH*:* mean corpuscular hemoglobin,
- MCHC: mean corpuscular, hemoglobin concentration,
- Hb: hemoglobin,
- MCV: Mean corpuscular volume.

7- CONFLICT OF INTEREST: None.

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