

# Evaluation of Laboratory Disorders in Admitted Neonates in NICU Who Were Born to Preeclamptic Mothers

Ziba Mosayebi<sup>1,2</sup>, Shahin Nariman<sup>2</sup>, Ladan Hosseini<sup>2</sup>, Amir Hossein Movahedian<sup>3,\*</sup>

<sup>1</sup> Department of Pediatrics, Arash Women's Hospital, Tehran University of Medical Sciences, Tehran, IR Iran

<sup>2</sup> Research Development Center, Arash Women's Hospital, Tehran University of Medical Sciences, Tehran, IR Iran

<sup>3</sup> Department of Pediatrics, Kashan University of Medical Sciences, Kashan, IR Iran

\*Corresponding author: Amir Hossein Movahedian, Department of Pediatrics, Kashan University of Medical Sciences, Kashan, IR Iran. Tel: +98-3615550026, Fax: +98-3615550089, E-mail: movahedian.ah@gmail.com.

**Received:** Jun 08, 2013; **Revised:** July 31, 2013; **Accepted:** August 28, 2013

**Background:** Preeclampsia is a major cause of maternal, fetal and neonatal morbidity and mortality.

**Objectives:** The aim of this study was to determine the laboratory disorders in neonates born to preeclamptic mothers.

**Patients and Methods:** This descriptive-retrospective study was conducted in the neonatal units of Shahid Beheshti and Shabihkhani Hospitals on neonates born to preeclamptic mothers during 2009 - 2012. Neonatal data including gestational age, gender, mode of delivery, birth weight and laboratory reports were extracted from their medical records and analyzed.

**Results:** A total of 2600 newborns were admitted during the study period, of which 84 infants were born to mothers with preeclampsia. The laboratory disorders were neutropenia in 32 cases (37%), anemia in 33 (39.2%), leucopenia in 24 (28.5%), thrombocytopenia in 23 (27.3%), hypoglycemia in 28 (33.3%), hypocalcemia in 22 (26.1%) and hyperbilirubinemia in 33 (39.2%) patients. Thirty one infants (35.6%) had positive cultures. Twenty nine patients (35%) developed coagulative disorders with prolonged PT and PTT. Evaluation of ABG showed 16 infants (19%) with metabolic acidosis, 23 (27.3%) hypoxia and 24 (28.5%) respiratory acidosis cases. All the laboratory abnormalities were more common in preterm and low birth-weight infants.

**Conclusions:** Early detection and timely management of laboratory disorders in preeclamptic infants may lead to improvement of the neonatal outcomes.

**Keywords:** Laboratories; Disorders; Pregnancy; Newborn

## 1. Background

Hypertensive disorders are the most medical complications in pregnancy and leading causes of maternal, fetal and neonatal morbidity and mortality (1, 2). National high blood pressure education working group has classified hypertensive pregnancy disorders into five categories including: A) preeclampsia (blood pressure equal to or higher than 140/90 in association with proteinuria equal to or greater than 300 mg in 24-hour urine), B) transient hypertension of pregnancy (blood pressure equal to or higher than 140/90 without proteinuria), C) eclampsia (occurrence of seizures in women with preeclampsia), D) chronic hypertension (blood pressure equal to or higher than 140/90 before pregnancy or diagnosed before 20<sup>th</sup> week of pregnancy) and E) preeclampsia superimposed on chronic hypertension (establishment of proteinuria equal to or greater than 300 mg in 24-hour urine in women who have had blood pressure equal to or higher than 140/90 before pregnancy or have been diagnosed before 20<sup>th</sup> week of pregnancy but have not

had proteinuria). In order to determine the best management courses, differentiation of these disorders is mandatory. The gestational and neonatal outcomes for each of these categories differ; with the worst outcome found in severe preeclampsia and less severe found in chronic hypertension. Approximately 30% of hypertensive disorders were due to chronic hypertension whereas 70% were diagnosed as preeclampsia (1, 3).

Preeclampsia is an unpredictable multisystemic disorder with unknown etiology unique to human pregnancy which complicates about 10% of all pregnancies and even higher in developing countries because of poor antenatal care, lack of health awareness and low socioeconomic status. Preeclampsia is influenced by nulliparity, age and race (1, 4). This disease remains a great challenge even to the most experienced obstetricians because there are no effective interventions to treat or prevent it and antenatal care involves a difficult balance between the risks for mother to continue the pregnancy and the risks for the baby's preterm birth. Primiparity, multiple pregnancies, previous preeclampsia, autoimmune disease, diabetes,

### Implication for health policy/practice/research/medical education:

Evaluation of the laboratory disorders in neonates born to preeclamptic mothers in order to prevent hematological and metabolic disorders.

Copyright © 2013, Iranian Society of Pediatrics. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

renal disease, chronic hypertension and obesity are risk factors for preeclampsia. A family history of preeclampsia increases the women's risk, suggesting a possible genetic predisposition (3).

A number of different complex mechanisms including lipid and protein oxidation, altered nitric oxide production and adhesion molecules and placental glycoproteins playing a role in trophoblastic-endothelial dysfunction, may be suggested as the etiopathogenesis of preeclampsia. A recent study revealed that excessive placental secretion of soluble fms-like tyrosine kinase-1 may contribute to endothelial dysfunction, hypertension and proteinuria in preeclampsia (5). Preeclampsia can lead to acute maternal complications such as progression to eclampsia, acute renal or hepatic failure, pulmonary edema, cardiovascular disorders, intracranial hemorrhage, disseminated intravascular coagulation and HELLP syndrome (hemolysis-elevated liver enzymes and low platelet count) (1, 2). Furthermore, there is an increased risk of long-term cardiovascular disease. Hypertensive disorders account for 16% of maternal deaths in developed countries, 25% in Latin America and 9% in Asia and Africa (6).

Fetal and neonatal complications associated with preeclampsia are fetal distress, fetal death, growth restriction, oligohydramnios, low APGAR scores and preterm delivery (2). The rate of fetal complications depends mostly on gestational age at the time of delivery. Prematurity a common complication of preeclampsia either due to spontaneous labor or conduct of interrupting the pregnancy due to compromised maternal-fetal health is the most important factor for increased morbidity and mortality including more neonatal intensive care unit admissions and rehospitalization (6). Neonates delivered to preeclamptic mothers may have spectrum of laboratory disorders which may be added to their existing morbidity. Hence, early detection of these disorders may lead to improvement of the perinatal outcomes.

## 2. Objectives

To our knowledge, very few studies have been conducted on laboratory findings of neonates born to preeclamptic mothers in Iran. The aim of this study was to determine the laboratory parameters in neonates born to preeclamptic mothers.

## 3. Patients and Methods

This descriptive-retrospective study was conducted on symptomatic neonates born to preeclamptic mothers who required admission in neonatal unit of Shahid Beheshti and Shabihkhani Hospitals affiliated to Kashan University of Medical Sciences (in a city of Iran) from 2009 to 2012. This study was approved by medical ethics

committee. Preeclampsia was defined as blood pressure  $> 140/90$  mmHg with proteinuria  $> 300$  mg in a 24-hour period, 20 weeks after gestation. In these two hospitals of Kashan University of Medical Sciences, management of preeclampsia was based on Williams Obstetric Textbook. Babies with congenital anomalies, who died immediately after birth, together with any illness observed in the mothers likely to increase neonatal morbidity and mortality (such as severe anemia, Rh incompatibility, diabetes mellitus, chronic diseases and aspirin consumption) were excluded from the study. Laboratory examinations including CBC, biochemistry analysis, evaluation of coagulation profile, blood gas analysis and cultures were performed on all neonates with clinical symptoms of poor feeding, hyporeflexia, lethargy, respiratory distress, hypoglycemia and etc.

In the present study, leukocyte count of less than  $5000/\text{mm}^3$  has been considered as leukopenia. Infants were considered to have neutropenia when absolute neutrophil count was less than  $1500/\text{mm}^3$  in the first 72 hours of life. Anemia is defined by hemoglobin or hematocrit value more than two standard deviations below the mean for age and thrombocytopenia was defined as less than  $150000/\text{mm}^3$  platelet count. Although there are controversies about the definition of neonatal hypoglycemia, blood glucose of less than  $40$  mg/dL is considered as the operational threshold to treat hypoglycemia in all neonates in the first day of life and less than  $50$  mg/dL thereafter. Hypocalcemia is defined as total serum calcium of less than  $7$  mg/dL in preterm infants and less than  $8$  mg/dL in term neonates.

Hyperbilirubinemia is defined as deviation of bilirubin level from the normal range and was managed based on baby's gestational age/birth weight, hours of age and presence or absence of risk factors. The equipment were Sysmex Kx21 (Japan), BT2000 (Italy) and GEM premier 3000 (USA) for evaluating CBC, biochemistry and ABG respectively. PT and PTT were measured by manual method with Fisher Kit. Culture media was TSB for blood culture, EMB and blood Agar for urine culture and Chocolate Agar, blood Agar and EMB for CSF culture. Neonatal data including gestational age, gender, mode of delivery, birth weight and laboratory reports (including hematologic, metabolic, blood gases, cultures, etc.) were extracted from their medical records and analyzed. Statistical analysis was performed using SPSS version 16. Frequency used for describing data.

## 4. Results

A total of 2600 newborns were admitted during the study period, of which 125 infants born to mothers with preeclampsia, while only 84 of them had the inclusion criteria to be involved in the study and 41 were excluded. Basic demographic data of the patients is given in Table 1.

**Table 1.** Demographic Characteristics of Patients

| Characteristics        | No. (%)   |
|------------------------|-----------|
| <b>Gestational age</b> |           |
| < 32 wk                | 36 (42.8) |
| 32 - 37 wk             | 36 (42.8) |
| > 37 wk                | 12 (14.3) |
| <b>Weight</b>          |           |
| < 1500 g               | 29 (34.6) |
| 1500 - 2500 g          | 35 (41.6) |
| > 2500 g               | 20 (23.8) |
| <b>Gender</b>          |           |
| Boy                    | 46 (54.8) |
| Girl                   | 38 (45.2) |
| <b>Delivery</b>        |           |
| Vaginal delivery       | 33 (39.3) |
| Cesarean               | 51 (60.7) |

Hematological disorders in these infants were neutropenia 32/84 (37%), anemia 34/84 (39.2%), leucopenia 24/84

(28.5%) and thrombocytopenia 26/84 (27.3%). Metabolic disorders were hypoglycemia 28/84 (33.3%), hypocalcemia 22/84 (26.1%) and hyperbilirubinemia 33/84 (39.2%). About 35% of infants had positive cultures including blood 15/84 (17.8%), urine 10/84 (11.9%) and CSF 6/84 (7.1%). Positive urine cultures were mainly obtained during the hospital stay. Thirty five percent of infants developed coagulative disorders with prolonged PT (23/84) and PTT (6/84). Evaluation of ABG showed 16/84 (19%) of infants having metabolic acidosis, 23/84 (27.3%) hypoxia and 24/84 (28.5%) respiratory acidosis.

All the laboratory abnormalities were more common in preterm and low-birth-weight infants (Table 2). Therapeutic intervention were needed for 16 infants (48.4%) with anemia (packed red cell transfusion), 17 neonates (73.9%) with thrombocytopenia (platelet transfusion), 21 cases (75%) of hypoglycemia (glucose infusion), 11 babies (47.8%) with coagulation disorders (Vitamin K and/or fresh frozen plasma) and 12 infants (52.1%) with hypoxia and acid base imbalance (Oxygen administration, mechanical ventilation, bicarbonate infusion). Sixteen (50%) hyperbilirubinemic infants needed exchange transfusion in addition to phototherapy for their jaundice.

**Table 2.** The Prevalence of Laboratory Disorders Based on Gestational Age (Week)

| Type of Disorder             | < 32 wk, No. (%) | 32 - 37 wk, No. (%) | > 37 wk, No. (%) | P value           |
|------------------------------|------------------|---------------------|------------------|-------------------|
| <b>Hematologic disorders</b> |                  |                     |                  |                   |
| Anemia                       | 11 (33.33)       | 17 (51.51)          | 5 (15.15)        | 0.3               |
| Neutropenia                  | 15 (46.87)       | 15 (46.87)          | 2 (6.25)         | 0.2               |
| Leucopenia                   | 9 (37.5)         | 15 (62.5)           | 0                | 0.01 <sup>a</sup> |
| Thrombocytopenia             | 11 (47.82)       | 10 (43.47)          | 2 (8.69)         | 0.6               |
| <b>Coagulative disorders</b> |                  |                     |                  |                   |
| Prolonged PTT                | 7 (30.43)        | 9 (39.13)           | 7 (30.43)        | 0.03 <sup>a</sup> |
| Prolonged PT                 | 1 (16.66)        | 2 (33.33)           | 3 (50%)          | 0.03 <sup>a</sup> |
| <b>Positive cultures</b>     |                  |                     |                  |                   |
| Urine culture                | 3 (8.3)          | 6 (16.7)            | 1 (8.2)          | 0.5               |
| Blood culture                | 6 (16.6)         | 8 (22.2)            | 0                | 0.2               |
| CSF culture                  | 2 (5.5)          | 2 (5.5)             | 2 (16.7)         | 0.3               |
| <b>Metabolic disorders</b>   |                  |                     |                  |                   |
| Hyperbilirubinemia           | 8 (24.2)         | 17 (51.5)           | 8 (24.2)         | 0.01 <sup>a</sup> |
| Hypoglycemia                 | 17 (60.7)        | 8 (28.7)            | 3 (10.7)         | 0.06              |
| Hypocalcemia                 | 10 (45.4)        | 5 (22.7)            | 7 (38.1)         | 0.01 <sup>a</sup> |
| <b>ABG disorders</b>         |                  |                     |                  |                   |
| Metabolic acidosis           | 4 (25)           | 8 (50)              | 4 (25)           | 0.1               |
| Hypoxia                      | 9 (39.13)        | 12 (52.17)          | 2 (8.69)         | 0.4               |
| Respiratory acidosis         | 15 (62.5)        | 9 (37.5)            | 0                |                   |

<sup>a</sup> statistically significant

## 5. Discussion

Preeclampsia, as one of the most common and important hypertensive disorders of pregnancy, is a major health problem worldwide and a threat to mothers and infants' well-being (2). Based on geographic regions of the world, prevalence of hypertension-complicated pregnancies are different (1) and range from 1.5% in Sweden, 2.6% in southwest Saudi Arabia and 3% in northwest Saudi Arabia to 7.5% in Brazil (2). In the present study the prevalence of preeclampsia in the two hospitals of Kashan University of Medical Sciences was 5%. The exact incidence of preeclampsia is unknown but infants born to mothers with pregnancy-induced hypertension are susceptible to morbidity and mortality and have higher NICU admission rates than neonates born to healthy women. These babies may have a spectrum of laboratory disorders. In our study, hematologic abnormalities were the most common disorders among these infants.

A common and well-defined effect of preeclampsia on neutrophils of neonates is neutropenia with an incidence of 50% (7, 8). It is a transient hematologic alteration, lasting days to weeks, related to the severity of pregnancy-induced hypertension. Neutropenia mainly affects the smaller and younger neonates and may be associated with an increased risk of nosocomial infection. In our study prevalence of neutropenia was 38%, compatible with 33% neutropenia in Harms et al. study (9) and primarily among preterm and low-birth-weight infants. Neutropenia in preterm and low-birth-weight infants is supported by other studies (10).

The reason behind neutropenia is unknown, but it is claimed that the uteroplacental insufficiency inhibits the production of myeloid lineage by bone marrow. Reduced numbers of circulating colony forming unit granulocyte macrophage (CFU-GM) and decreased neutrophil storage pool is associated with neutropenia (4). In severe cases the use of granulocyte colony stimulating factor (G-CSF) can improve the absolute neutrophil count (11). There are some reports of an increased risk of neonatal infection, but other studies did not support these findings (12, 13).

Thrombocytopenia was present in 27.3% of our cases compared with the findings of Sivakumar et al. who have noted thrombocytopenia in 22% of infants born to preeclamptic mothers (12). Other studies showed variable incidences of thrombocytopenia as 33% by Harm et al. (9), 34% by Eltink et al. (14) and 36% by Bhat et al. (15). Occurrence of thrombocytopenia was found to be associated with decreased birth weight and gestational age. Raizada et al. showed that preterm infants born to preeclamptic mothers are at great risk of thrombocytopenia development (16). Patricia et al. demonstrated that infants with < 1200 g birth weight as well as < 32 week of gestational age born to mothers with gestational hypertension, preeclampsia or eclampsia syndrome, had leukopenia, neutropenia and thrombocytopenia (17).

It has previously been postulated that thrombocytopenia is the result of placental insufficiency and decreased production (8); however Samuels et al. have demonstrated abnormal platelet antibodies in the infants of preeclamptic mothers and considered an immune theory. There are studies that support the role of mediators in developing thrombocytopenia. Normally, vascular endothelial growth factor (VEGF) and placental growth factor (PIGF) are responsible for maturation of megakaryocyte and participate in the regulation of megakaryocyte development. Low levels of PIGF and VEGF are shown in the cord blood of preeclamptic mothers' babies (18). Thrombocytopenia is a transitory alteration, more commonly found in the first 72 hours after birth, with resolution within 10 days and is not severe in most cases.

Leukopenia was seen in 28.5% of the patients of this study, more common in babies with gestational age of 32 - 37 weeks. Harms et al. demonstrated leukopenia in 21% of the affected infants (9). Hyperbilirubinemia was the most common metabolic disorder with an incidence of 39.2% in the current study, comparable to the one by Eltink et al. (14) in which 44.7% of cases had jaundice. Hypertensive disorder is a maternal risk factor for development of hypoglycemia in infants. In addition, vulnerability of premature and low-birth-weight infants to hypoglycemia is a well-recognized problem in neonatal medicine. The present study revealed hypoglycemia in 33.3% of patients, similar to Dhananjaya et al. study with a 40% incidence of hypoglycemia in preeclampsia/eclampsia (19) and comparable with Eltink et al. study (14).

Another metabolic derangement was hypocalcemia. Preeclampsia, prematurity, perinatal stress/asphyxia, intrauterine growth restriction, infant of diabetic mother, maternal hyperparathyroidism, iatrogenic (alkalosis and use of blood products, diuretic, phototherapy, lipid infusion), maternal intake of anticonvulsants (Phenobarbital, Phenytoin sodium) are all risk factors for hypocalcemia especially for the early-onset type which presents within 2 - 3 days of life (20).

Positive cultures including blood, urine and CSF were observed in 16.6%, 11.9% and 7.1% of infants respectively. They were almost all preterm neonates and had no evidence of neutropenia. There is concern about the possible relationship between preeclampsia and sepsis. Procianny et al. found early and late onset sepsis in 4.3% and 22.7% of study populations, respectively. They claimed of vaginal delivery and neutropenia as significant variables for early-onset sepsis as well as total parenteral nutrition, central catheters and mechanical ventilation for late-onset sepsis, regardless of the presence of neutropenia (21). Probably prematurity was the causative factor of infection in our cases. The risk of sepsis in infants born to preeclamptic mothers is still controversial, but those with prolonged neutropenia could be at a higher risk of infection because of impaired host immunity after birth.

In our study a variety of coagulation alterations was

seen as prolonged PT and PTT in 7.1% and 27.3% of patients respectively. Severe hypertension may result in a marked imbalance in the hemostatic system both in the mother and infant. Prolongation of PT, PTT and TT was demonstrated by Lox et al. and by Narayan et al. (21, 22). One reason for this derangement may be the lack of balance between coagulation and fibrinolysis in localized area of vascular compartment especially uteroplacental circulation (21). Neonates of preeclamptic mothers, who are at great risk for coagulation disorders and bleeding, require close follow up. In our patients, the acid base imbalance and hypoxia were mainly due to perinatal birth asphyxia and respiratory distress syndrome. Perinatal asphyxia experienced by these infants could be explained by the uteroplacental insufficiency and compromise of blood flow to the fetus which occurs in these pregnancies. Birth asphyxia and low APGAR score have been reported in many studies (1-3, 9).

There are conflicting results on the protective effects of hypertensive disorders for neonatal respiratory distress syndrome. Chang et al. claimed an increase in the incidence of hyaline membrane disease in the preeclamptic group especially in neonates with gestational age of < 32 weeks compared with non-preeclamptic control group (23). Also a study from Tunis showed the increased risk of hyaline membrane disease in premature infants of preeclamptic mothers weighing less than 2000 g (24). The confounding factors were controlled by these studies. Therefore, these findings support that fetal lung maturity is not accelerated in preeclampsia but in contrary it is delayed. Friedman et al. found no differences in the incidence of respiratory distress syndrome (25).

However, there are studies which have reported the protective effects of hypertensive disorders on neonatal respiratory morbidities including respiratory distress syndrome (26). Conditions that cause chronic stress may be associated with accelerated fetal pulmonary maturation due to increased cortisol production by the fetus. Neonatal morbidity in newborns of preeclamptic mothers is considerable and the need for medical intervention should be evaluated. Our findings showed that at least 50% of babies need some therapeutic measures.

Our study had some limitations. It had a retrospective design so it could not record all the variables completely. The small sample sizes besides the lack of a control group were other limitations of this study. Furthermore, we reported laboratory disorders in the babies born to preeclamptic mothers regardless of the severity of preeclampsia.

Despite all the obstetric attempts made to improve assessment and surveillance of women who have had hypertension in pregnancy, preeclampsia continues to be a common and serious complication of pregnancy and a cause of great concern because of the high rates of adverse maternal, fetal and neonatal outcomes. The present study demonstrates a spectrum of laboratory abnor-

malities especially hematological ones in babies born to preeclamptic mothers. These abnormalities need early detection, timely referral to tertiary medical centers and prompt management in order to improve perinatal and neonatal conditions in these critical groups of patients.

## Acknowledgements

We would like to express our gratitude to Dr. S. Madhkhani and all the staffs of neonatal units of Shahid Beheshti and Shabihkhani Maternity Hospitals (affiliated to Kashan University of Medical Sciences) who helped a lot for data acquisition as well as Research Development Center of Arash Women's Hospital (affiliated to Tehran University Of Medical Sciences) that helped for writing this paper.

## Authors' Contribution

Ziba Mosayebi: Designing the study, Shahin nariman: writing the manuscript, Ladan Hosseini: writing the manuscript, Amir Hossein Movahedian: Patients examination.

## Financial Disclosure

The authors declare that they have no conflicts of interest in the research.

## Funding Support

The present article was financially supported by Kashan University of Medical Sciences.

## References

1. Tavassoli F, Ghasemi M, Ghomian N, Ghorbani A, Tavassoli S. Maternal and perinatal outcome in nulliparous women complicated with pregnancy hypertension. *J Pak Med Assoc.* 2010;**60**(9):707-10.
2. Yucesoy G, Ozkan S, Bodur H, Tan T, Caliskan E, Vural B, et al. Maternal and perinatal outcome in pregnancies complicated with hypertensive disorder of pregnancy: a seven year experience of a tertiary care center. *Arch Gynecol Obstet.* 2005;**273**(1):43-9.
3. Sangkomkamhang UM, Laopaiboon M, Lumbiganon P. Maternal and Neonatal Outcomes in Pre-eclampsia and Normotensive Pregnancies. *Thai J Obstet Gynaecol.* 2012;**18**(3):106-113.
4. Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ. Maternal preeclampsia and neonatal outcomes. *J Pregnancy.* 2011;**2011**:214365.
5. Sachan R, Patel ML, Sachan P, Gaurav A, Singh M, Bansal B. Outcomes in hypertensive disorders of pregnancy in the North Indian population. *Int J Womens Health.* 2013;**5**:101-8.
6. Habli M, Levine RJ, Qian C, Sibai B. Neonatal outcomes in pregnancies with preeclampsia or gestational hypertension and in normotensive pregnancies that delivered at 35, 36, or 37 weeks of gestation. *Am J Obstet Gynecol.* 2007;**197**(4):406 e1-7.
7. Mouzinho A, Rosenfeld CR, Sanchez PJ, Risser R. Effect of maternal hypertension on neonatal neutropenia and risk of nosocomial infection. *Pediatrics.* 1992;**90**(3):430-5.
8. Steer P, Lupon M, Oteng-Ntim E. Hypertensive Diseases in Pregnancy. *Rennie and Robertson's textbook of Neonatology*. Churchill Livingstone: 2008. p. 179-181.
9. Harms K, Rath W, Herting E, Kuhn W. Maternal hemolysis, elevated liver enzymes, low platelet count, and neonatal outcome. *Am*

- J Perinatol.* 1995;**12**(1):1-6.
10. Zook KJ, Mackley AB, Kern J, Paul DA. Hematologic effects of placental pathology on very low birthweight infants born to mothers with preeclampsia. *J Perinatol.* 2009;**29**(1):8-12.
11. Kocherlakota P, La Gamma EF. Preliminary report: rhG-CSF may reduce the incidence of neonatal sepsis in prolonged pre-eclampsia-associated neutropenia. *Pediatrics.* 1998;**102**(5):1107-11.
12. Sivakumar S, Bhat BV, Badhe BA. Effect of pregnancy induced hypertension on mothers and their babies. *Indian J Pediatr.* 2007;**74**(7):623-5.
13. Bhaumik S, Ghosh S, Haldar KK, Mitra PK, Manna B. Risk of early onset neonatal septicemia in babies born to mothers with pre-eclampsia. *Indian Pediatr.* 2000;**37**(7):775-9.
14. Eeltink CM, van Lingen RA, Aarnoudse JG, Derks JB, Okken A. Maternal haemolysis, elevated liver enzymes and low platelets syndrome: specific problems in the newborn. *Eur J Pediatr.* 1993;**152**(2):160-3.
15. Bhat YR, Cherian CS. Neonatal thrombocytopenia associated with maternal pregnancy induced hypertension. *Indian J Pediatr.* 2008;**75**(6):571-3.
16. Raizada N, Lal A, Bhatia RC, Jain BK, Chander K, Goyal A. Neonatal thrombocytopenia due to pregnancy induced hypertension. *Indian J Pediatr.* 1996;**63**(2):226-8.
17. Nash PL, Gillespie K, Devaskar UP. Effect of Early Onset Bacterial Sepsis or Pregnancy Induced Hypertension (PIH) on Neonatal White Blood Cell and Platelet Counts in Infants Less Than 1,200 Grams. *J Matern-Fetal Neo M.* 1993;**2**(1):1-4.
18. Akcan AB, Oygucu SE, Ozel D, Oygur N. Mean platelet volumes in babies of preeclamptic mothers. *Blood Coagul Fibrinolysis.* 2011;**22**(4):285-7.
19. Dhananjaya CD, Kiran B. Clinical profile of hypoglycemia in newborn babies in a rural hospital setting. *Int J Biol Med Res.* 2011;**2**(4):1110-1114.
20. Jain A, Agarwal R, Sankar MJ, Deorari A, Paul VK. Hypocalcemia in the newborn. *Indian J Pediatr.* 2010;**77**(10):1123-8.
21. Narayan S, Kumari S, Mangwana S, Logani KB, Kabra M. Consumption coagulopathy in neonates born to mothers with pregnancy induced hypertension. *Indian Pediatr.* 1994;**31**(7):840-2.
22. Lox CD, Word RA, Corrigan JJ. Effects of preeclampsia on maternal and cord blood clotting activity. *Am J Perinatol.* 1985;**2**(4):279-82.
23. Chang EY, Menard MK, Vermillion ST, Hulsey T, Ebeling M. The association between hyaline membrane disease and preeclampsia. *Am J Obstet Gynecol.* 2004;**191**(4):1414-7.
24. Cherif A, Ben jemaa W, Kacem S, Guellouze N, Jebnoun S, Khrouf N. [Preeclampsia increases the risk of hyaline membrane disease in premature infant: a retrospective controlled study]. *J Gynecol Obstet Biol Reprod (Paris).* 2008;**37**(6):597-601.
25. Friedman SA, Schiff E, Kao L, Sibai BM. Neonatal outcome after preterm delivery for preeclampsia. *Am J Obstet Gynecol.* 1995;**172**(6):1785-8.
26. Langenveld J, Ravelli AC, van Kaam AH, van der Ham DP, van Pampus MG, Porath M, et al. Neonatal outcome of pregnancies complicated by hypertensive disorders between 34 and 37 weeks of gestation: a 7 year retrospective analysis of a national registry. *Am J Obstet Gynecol.* 2011;**205**(6):540 e1-7.