MPV/Platelet Ratio is a Significant Predictor for Mortality in ELBW Newborns of Preeclamptic Mothers

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Background: Mean platelet volume (MPV) is a measurement that describes the average size of platelets in the blood. MPV is associated with RDS, NEC, BPD, IVH and sepsis in newborns.

Aim: To assess the relationship between MPV in first 6 hours of life and overall mortality, mortality in first 7 days, APGAR Scores, CRIB score and extremely low birth weight newborns (1000 gr) of preeclamptic mothers.

Subjects: We evaluated the complete blood counts parameters of 169 extremely low birth weight infants (ELBW) (Birth weight 1,000 g) at sixth hour of life. Forty five of the newborns were preeclamptic mother’s infant.

Results: Twelve of 45 died in first 7 days of life. MPV value of the infants of preeclamptic mother was 7,6 fL. MPV value of the infants who has not preeclamptic mother was 7,7 fL. There was not significantly difference between two groups (p=0,88). MPV was significantly correlated with CRIB score, APGAR 1 scores in infants of preeclamptic mother (p= 0,015, p=0,045, respectively). MPV was slightly correlated with APGAR 5 score (p= 0,06). But MPV was not correlated with overall mortality and mortality in first 7 day of life in infants of preeclamptic mother (p= 0,491, p=0,395, respectively). In this group, MPV/Platelet count was significantly correlated with overall mortality and mortality in first 7 days of life (p=0,05, r=0,319; p=0,04, r=0,336, respectively).

Conclusions: Our results suspect that MPV is not but MPV/ Platelet count is a significant predictor of mortality in ELBW newborns of preeclamptic mothers. MPV, is a simple and easy method of assessing platelet function and/or platelet reactivity. The role of MPV as a novel prognostic marker in different diseases has emerged, but in newborns there is not enough data and also the underlying mechanism of MPV as a prognostic factor has not been fully elucidated. There are much data on the importance of MPV predicting preeclampsia but no data in infants of preeclamptic mothers. Further studies are needed in this area.
Therapeutic Hypothermia for Neonatal Encephalopathy: Experiences of a Medical Center in Northern Taiwan

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Background: Therapeutic hypothermia (TH) should be offered for newborn infants ≥36 weeks of gestations with stage II or III perinatal hypoxic ischemic encephalopathy (HIE). However, some “grey case” situations exist in the practice of TH.

Objective: To describe our experiences in performing TH in the NICU.

Methods: Newborn infants admitted to the NICU at Chang Gung Children’s Hospital between April 2011 and December 2012 and underwent TH were enrolled. Whole body cooling therapy was used in our NICU. Eligible infants were kept at 33-34°C for 72 hours and re-warmed for at least 6 hours. Clinical details were documented and analyzed.

Results: Twenty-two patients received TH. Seventeen full-term neonates with perinatal HIE (8 with stage II and 9 with stage III) were treated with TH within 6 hours of life. Among them, 59% (10/17) were male and 76% (13/17) were out-born patients. Three patients were moribund and treatment was terminated. Thus, the survival rate was 82% (14/17). Two neonates were treated with TH beyond 6 hours of life. One out of them received dialysis therapy and died due to sepsis. Among the remainder three patients, one with 33 weeks of gestations had stage II HIE and received TH. Two preterm infants, 32 and 25 weeks of gestations, had postnatal collapse at PMA 34 weeks and received TH. The temperature was kept stably in the target range for the later three patients. However, one patient developed infantile spasm at 6 months of corrected age.

Conclusion: The survival rate of infants with stage II HIE was higher if TH was started within 6 hours of life compared to that after 6 hours of life in the current study. Infants 35 weeks of gestations could tolerate TH; however, our data did not show benefits of TH for infants with postnatal collapse.
Background: The principal sites of neonatal phototherapy (NNPT) action may localize not only in the skin but also in capillary circulation under the skin. Thus, the safety of phototherapy particularly in relation to hemodynamic control must be investigated.

Aim of the work: This study aimed to evaluate the effect of phototherapy on blood ET and NO in hyperbilirubinemic newborn infants of different gestational ages with stable vital signs and to observe changes in vital signs in those newborn infants after 24 hours of phototherapy.

Patients and Methods: This study included 50 newborn infants (29 preterm ≥37 weeks and 21 full term ≥37 weeks) with stable vital signs who received phototherapy for high unconjugated bilirubinemia. Heart rate (HR), respiration rate (RR), mean arterial blood pressure (MABP), Serum endothelin-1 and nitric oxide level were detected before phototherapy subsequently monitored after 24 hours after phototherapy. Results: The present study revealed that after 24 hours of phototherapy there is a significant increase in NO and ET serum level, NO:ET ratio, HR and a significant decrease in MABP in both full term and preterm newborns. There is no significant change in RR in both preterm and full term newborns.

Conclusions: Phototherapy can disturb the dynamic balance between NO and ET, leading to a more prominent effect of NO. This effect may be not harmful in newborns with stable vital signs, but harmful effect may occur in newborns with sepsicaemia or decompensated vital signs. Careful observation of vital signs should be done in all newborns under the effect of Phototherapy. Also the safety of continuous phototherapy for more than 24 hours in newborn infants with different gestational ages and different birth weights must be verified.
Blood Glucose Homeostasis in the First 24 Hours of Life

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Background: Soon after birth, healthy term babies mobilise glucose from stores to meet metabolic demands. This process is compromised in sick and/or preterm babies.

Few studies describe changes in early blood glucose concentration (BGC) in these babies. No data exist that define BGC that causes neurological damage and there is considerable debate about when intervention is necessary to treat hypoglycaemia.

Objective: To document changes in blood glucose concentration in sick newborn babies in the first 24 hours of postnatal life.

Methods: Babies admitted to St George’s Hospital Neonatal Unit over a one month period were included in this study and the following data were collected:

- All available BGC in the first 24 hours of life from routine samples
- Glucose infusion rate
- Additional nutrient intake
- Dextrose bolus administration in response to low BGC
- Neurological signs associated with hypoglycaemia

Results: 55 cases were reviewed, including 36 preterm and 19 term babies. BGC was lowest from 0 to 4 hours after birth. In babies 32 weeks gestation, BGC increased after the initial 4 hours, reaching up to 10mmol/L. None of the babies documented had neurological signs of hypoglycaemia. 7 babies received dextrose boluses and these were administered in response to BGC between 0.8mmol/L and 2.7mmol/L. No consistent BGC threshold was applied for administering dextrose boluses. The mean BGC was 3.2mmol/L in those who received a bolus and 4.8mmol/L in those who did not.

Conclusion: The need for administering a dextrose bolus in response to low BGC alone, particularly in the first 2 hours, is questionable. No neurological signs of hypoglycaemia were observed and many babies that received boluses had similar BGC to those babies that did not receive boluses yet recovered spontaneously. Furthermore, bolus administration can stimulate insulin secretion, posing the risk of “rebound” hypoglycaemia.
Background: Inborn errors of metabolism (IEM) are hereditary affections resulting from incompetence in enzymatic reactions of intermediary metabolism, and commonly present around the time of birth. Although most affected newborns born healthy, they subsequently deteriorate, some may present at (or shortly after) birth.

Objective: To determine admission complaints, clinical and laboratory findings, the ratio and outcomes of the neonates diagnosed IEM at a level III tertiary care hospital.

Methods: We determined the neonates diagnosed IEM in the neonatal intensive care unit (NICU) of our hospital. The patients were regularly followed-up at divisions of neonatology, developmental pediatrics, endocrine-metabolism-nutrition outpatient clinics prospectively.

Results: Among hospitalized 2600, 45 neonates diagnosed IEM between 2010/2012 with the ratio of 1.7% in our NICU. Their birth weight’s was 2986.9±588 g (1400-4300), gestational age’s was 38.4±2.5 week (31-41), consanguinity ratio was 68.9%, history of sibling was 33.3%. Admission complaints were poor feeding, decreased activity, jaundice, seizure, and respiratory problems. Distribution of IEM was as follows; phenyl ketonuria (8), propionic acidemia (PA) (5), maple syrup urine disease (5), citrullinemia (4), galactosemia (4), non-ketotic hyperglycinemia (4), tyrosinemia (2), Zellweger syndrome (1), wolman disease (1), pyruvat carboxylase deficiency (1), N-Acetylglutamate synthetase deficiency (1).

Mortality was 26.7% (12), 3 patients were lost to follow-up, 1 of them referred to local hospital after discharge. The follow-up period was 2-42 months. Among followed 29 neonates, 16 had normal neurodevelopment, 9 had developmental delay, 4 had cerebral palsy by the Quide for Monitoring Child Development. Gestational age, peritoneal dialysis, and delivery route had significant effect on outcome.

Conclusion: We want to emphasize the importance of early neonatal metabolic screening, especially in the countries with high ratios of consanguinity that IEM is still common occurrence. Early diagnosis by expanded newborn screening and treatment is essential in order to prevent neurological sequelae and good prognosis.
Increased Asymmetric Dimethylarginine Levels in Severe Transient Tachypnea of the Newborn

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Introduction: Nitric oxide (NO) which is synthesized from arginine by the NO-synthase in the lung is an important mediator of normal development and vascular smooth muscle relaxation. Asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of NO synthase. NO has been suggested to play a key role in the pathogenesis of pulmonary hypertension. Transient tachypnea of the newborn (TTN) is a consequence of inadequate neonatal lung fluid clearance. Although TTN usually has a self-limiting benign course, some cases experience substantial morbidity such as pulmonary hypertension which results in prolonged intensive care.

We aimed to investigate serum ADMA levels in infants with TTN and its relation to systolic pulmonary artery pressure (sPAP) and disease severity.

Method: A prospective controlled study involving infants born at ≥34 gestational age with TTN and controls was conducted. Study and control groups were compared for serum ADMA levels measured at the 6-24th (first samples) and 48-72th hours (second samples) of life. Patients were divided into 2 groups according to duration of tachypnea as prolonged (72 hours) and mild tachypnea (≤ 72 hours). sPAP were evaluated by echocardiography in the study group on the second day of life.

Results: Thirty-eight infants with TTN and 41 controls were enrolled. In the first samples ADMA levels were significantly higher in infants with TTN compared to controls (1.06 ±0.26 vs 0.83 ±0.18 p=0.001). In the second samples ADMA levels were significantly higher in infants with prolonged tachypnea compared with to mild tachypnea (0.91 ± 0.18 vs 0.79 ± 0.15 p=0.025) and controls (0.91 ± 0.18 vs 0.81 ± 0.1, p=0.014). Mean sPAP levels was significantly higher in prolonged tachypnea group than mild tachypnea group (30.8 ±10.5 vs 23.6 ± 7.4 mm Hg, p=0.042). Second samples’ ADMA levels were correlated with tachypnea duration (p=0.033; r= 0.346).

Conclusion: Infants with prolonged tachypnea had increased serum ADMA and mean sPAP levels. An increased ADMA concentration could reduce NO synthesis, which could lead to increase pulmonary artery pressure and, consequently a longer duration of tachypnea.