

Consumption Coagulopathy in Neonates Born to Mothers with Pregnancy Induced Hypertension

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Pregnancy induces complex changes concerned with hemostasis. Normal pregnancy is associated with hypercoagulable state which becomes more pronounced at term while in pre-eclampsia or pregnancy-induced hypertension (PIH), localized coagulation fibrinolytic imbalance has been reported(1,2). Various coagulation factors are decreased in concentration in PIH, especially fibrinogen, factor V, VII, VIII. Thrombocytopenia and increased serum FDP levels are also observed(3). In term and preterm neonates of these mothers, a variety of coagulation alterations with significant thrombocytopenia have been reported(4,5).

This study was conducted to determine the nature and extent of coagulation defects in neonates of mothers with PIH as there is a paucity of such a study in Indian literature.

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Material and Methods

Coagulation profile of 20 neonates born to pregnancy-induced hypertensive mothers was compared with 20 age-matched control neonates born to normotensive mothers. No aspirin therapy was given to PIH members.

The neonates of PIH mothers were subdivided as follows: (i) Neonates born to mothers with mild pre-eclampsia; and (ii) Neonates born to mothers with severe pre-eclampsia.

Pre-eclampsia was defined as severe, if any one of the following signs or symptoms were present(6):

1. Systolic blood pressure 160 mm Hg or more, diastolic 110 mm Hg or more on at least two occasions 6 hours apart.
2. Proteinuria 3+ or 4+ (5 g or more in 24 hours).
3. Oliguria (passage of 400 ml or less urine in 24 hours).
4. Cerebral or visual disturbances.
5. Pulmonary edema or cyanosis.

Coagulation tests included prothrombin time (PT)(7), activated partial thromboplastin time (APTT)(7), thrombin time (TT)(7), platelet count(7) and serum fibrin/fibrinogen degradation products (FDP) assay(7).

Oral vitamin K as 1 mg dose to term babies and 0.5 mg to preterm babies was given to all neonates within an hour of birth.

Results

Of the 20 PIH mothers, 5 had mild, while 20 had severe PIH. In severe PIH group, the incidence of preterm neonates was more (46.7%) as compared to that in mild PIH group (20%).

Results of coagulation profile (*Table I*)

showed significant prolongation of PT in study group (27.3 ± 14.0 sec) as compared to control group (20.0 ± 5.2 sec) ($p < 0.01$). However, because of high value of SD in the study group, it is necessary to evaluate this test in a larger group. differences in APTT and TT were highly significant ($p < 0.001$) between study/ and control groups. Study group showed low platelet count (mean 1.60 ± 0.42 lacs/cu mm) as compared to control group (mean 2.06 ± 0.18 lacs/cu mm). Serum FDP assay could be done in 10 cases of study group, out of which 60% showed increased levels (10-320 Hg/ml).

Neonatal outcome in mothers of mild PIH showed no significant clinical manifestation, whereas neonates of severe PIH mothers had DIC (6 cases 30%). Of these, two neonates had aspiration pneumonia, two developed septicemia and two developed DIC with cerebral and gastrointestinal hemorrhage. These neonates were discharged after treatment.

Discussion

Pregnancy induced hypertension causes increased fetal and neonatal morbidity and mortality. The presence of severe hypertension causes a marked imbalance in the hemostatic system of the mother and neonate.

The present study showed statistically significant prolongation of PT, APTT and TT in neonates born to PIH mothers as compared to those born to normotensive mothers, the derangements were more pronounced in neonates of severe PIH as compared to neonates of mild PIH mothers, especially in preterm babies. Preterm babies had an increased abnormality of PT as compared to term babies which indicates that deficiency of vitamin K dependent factors are more marked in preterm babies. Prolongation of PT, APTT and TT is consistent with the findings of Lox *et al.*(5) who also showed abnormal values.

The finding of thrombocytopenia in study group as compared to control group confirms the earlier report by Brazzy *et*

TABLE I-Results of Coagulation Studies: Mean (\pm SD)

Tests	Control group (n=20)	Study group (n=20)
Prothrombin time (sec)	20.0 (\pm 5.19)	@ 27.26(\pm 13.96)
Activated partial thromboplastin time (sec)	46.31 (\pm 10.08)	*64.10 (\pm 19.82)
Thrombin time (sec)	14.46 (\pm 1.5)	*18.00 (\pm 3.11)
Platelet count (Lacs/cu mm)	2.06 (\pm 0.18)	@ 1.60 (\pm 0.425)
Serum fibrinogen degradation product assay (n =10)	-	Increased in 6 cases (Range 10-320 g/ml)

@ p value < 0.01 ;

* P value < 0.001 .

al.(4). The findings of thrombocytopenia can be explained by DIC in neonates (as seen in 30% of neonates) or it might result from platelet adhesion at the site of endothelial damage in uteroplacental circulation(2), or decreased prostacyclin activity in the fetal venous blood as observed by Thiagrajan *et al.*(8).

Serum FDP levels were raised in 60% cases (6 out of 10) in study group. Derangements were more pronounced in neonates born to severe PIH mothers than in mild PIH especially in preterm babies.

The low platelet count with prolongation of PT, APTT, IT and raised serum FDP levels in neonates could be explained by DIC. In spite of vitamin K administration, the derangements of coagulation profile were statistically significant between study and control groups, more so in preterm babies which suggest that liver immaturity or some other factors are involved. Disturbances of balance between coagulation and fibrinolysis in localized area of vascular compartment particularly uteroplacental circulation could be a reasonable explanation for deranged coagulation profile in these neonates. Hence, neonates of PIH mothers who may be at an increased risk for bleeding disorder require close follow-up and further studies are suggested to see the modalities of management of these babies.

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