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Neonatal complications and preeclampsia: Descriptive study at a tertiary care hospital

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Abstract
Risk factors consistently shown to be associated with increased rate of PIH include chronic hypertension, high prepregnancy BMI, advanced maternal age, family history of preeclampsia or cardiovascular disease, preexisting medical conditions such as hypertension, renal disease, diabetes with vasculopathy and obstetrics indications such as multiple gestation and hydrops foetalis, nulliparity, history of previous preeclamptic pregnancy. All the pregnant women with ≥ 20 weeks of gestation who fulfilled the selection criteria registered in the Department of Obstetrics and Gynaecology, enrolled in this study. In this study the frequency of preterm neonates was noted in 18.52% of the mothers with APE compared to 27.66% of the mothers with SPE and 11.11% of the mothers with MPE while none of the mother had preterm neonate (0%) in women with normal pregnancy and this difference was statistically significant ($p<0.001$). In this study maximum number of babies who were born to the women with APE developed complications (22.22) compared to 10.64% who were born to the mothers with SPE, 7.41% of the neonates who were born to women with MPE and 10% of the neonates who were born to the mothers with normal pregnancy. But this difference was statistically not significant ($p=0.271$).

Keywords: Neonatal complications, preeclampsia, HELLP syndrome

Introduction
PE is characterized by an increased BP equal to or above 140/90 mmHg in the presence of proteinuria developed after 20 weeks of gestational age. PE can result in eclampsia when convulsion develops or manifests as hemolysis, elevated liver enzymes and low platelet count (HELLP) syndrome. Eclampsia and HELLP syndrome are known to be associated with severe complication such as cerebral hemorrhage, renal insufficiency, lung edema and liver hemorrhage. The current hypothesis regarding the etiology of PE focuses on mal-adaptation of the immune responses and defective trophoblast invasion. Thus, an excessive maternal inflammatory response, perhaps directed against foreign fetal antigens, results in a chain of events including shallow trophoblast invasion, defective spiral artery remodeling, placental infarction and release of pro-inflammatory cytokines in the systemic circulation [1, 2].
The incidence of preeclampsia varies widely from 5-15%. In India the incidence of preeclampsia is reported to be 8-10% of all the pregnancies.
Risk factors consistently shown to be associated with increased rate of PIH include chronic hypertension, high prepregnancy BMI, advanced maternal age, family history of preeclampsia or cardiovascular disease, preexisting medical conditions such as hypertension, renal disease, diabetes with vasculopathy and obstetrics indications such as multiple gestation and hydrops foetalis, nulliparity, history of previous preeclamptic pregnancy [3].
PIH can be a major contributor to maternal morbidity and mortality as a immediate consequence of progression to eclampsia. The most common major obstetric complications include acute renal failure, disseminated intravascular coagulation, HELLP syndrome and pulmonary edema. Pre-eclampsia/eclampsia probably accounts for more than 50,000 maternal deaths worldwide each year [4].
Although the cause of PIH remains unknown, evidence for its manifestation begin early in pregnancy with overt pathophysiological changes that gain momentum across gestation and eventually become clinically apparent. Unless delivery supervenes these changes ultimately result in multiorgan involvement. These are thought to be consequence of endothelial dysfunction, vasospasm and ischemia. While the myriad of maternal consequences of PIH are described in terms of individual organ systems, they frequently are multiple and overlap clinically [5, 6].

Methodology

Study design
The study design was a prospective comparative study.

Study period
This study was conducted for a period of 18 months.

Source of data
All the pregnant women with ≥ 20 weeks of gestation who fulfilled the selection criteria registered in the Department of Obstetrics and Gynaecology, enrolled in this study.

Sample size
A total of 151 women registered in the Department of Obstetrics and Gynaecology, during the study period were studied.

Sample size calculation
The sample size was calculated considering following formula.

$$n = \frac{2(Z_\alpha + Z_\beta)^2 \, p \, q}{(P_0 - P_1)^2}$$

Based on the above formula the minimum effect size required in each group was 19. However, during the study period a total of 151 women were eligible and provided written informed consent. Hence a total of 151 women were studied.

Sampling technique
These women were divided into four cohorts as below.

- **Normal pregnancy (NP): N**ormotensive (Normal) pregnant women
- **Mild preeclampsia (MPE):** Women with mild preeclampsia**.**
- **Severe preeclampsia (SPE):** Women with severe preeclampsia**.**
- **Antepartum eclampsia (APE):** Women with anterpartum eclampsia**.**

Selection Criteria
Inclusion criteria
Pregnant women with gestational age of ≥20 weeks of pregnancy (according to a reliable last menstrual period and ultrasound confirmation) with either mild, severe or antepartum preeclampsia.

Exclusion criteria
- Pregnant women with history of;
- Liver disease
- Diabetes mellitus
- Renal failure
- Hemolytic anemias
- Stroke
- Coronary artery disease
- Chronic lung diseases
- Connective tissue disorder
- DIC
- Seizures
- Chronic hypertension
- Gestational diabetes mellitus (GDM)
- Multiple pregnancy
- Hepatotoxic drugs.

Informed Consent
All the women fulfilling the selection criteria were explained about the nature of the study and a written informed consent was obtained before enrollment.

Data collection
After obtaining written informed consent, demographic data such as age, detailed history (including obstetric history, family history and other comorbid conditions) was obtained through an interview. Also presenting complaints were noted. Further these women were subjected to complete examination (general and systemic examination). Blood pressure was recorded by Residents using a mercury sphygmomanometer and stethoscope from the upper arm after the subjects had been sitting for more than 5 minutes according to the guidelines of the American Heart Association. Three readings were recorded after 5 minutes rest interval between the measurements and the average value was recorded. These findings were noted on a predesigned and pretested proforma.

Investigations
All the pregnant women underwent routine hematological test including haemoglobin, blood grouping, human immunodeficiency (HIV)/ surface antigen of the hepatitis B virus (HBsAg), ultrasound examination special investigations were done which included blood urea, serum creatinine, serum uric acid, serum electrolytes, blood sugar level, liver function tests, fundoscopy, bleeding time, clotting time, coagulation profile, urine routine and urine culture. Further all these women were subjected to Serum LDH investigation.

Results

Table 1: Comparison of age with preeclampsia

Age group (Years)	Groups								Total	
	NP		MPE		SPE		APE			
	No.	%	No.	%	No.	%	No.	%	No.	%
≤ 20	13	26.00	1	3.70	5	10.64	12	44.44	31	20.53
21 to 25	28	56.00	12	44.44	15	31.91	11	40.74	66	43.71
26 to 30	9	18.00	11	40.74	22	46.81	4	14.81	46	30.46
> 30	0	0.00	3	11.11	5	10.64	0	0.00	8	5.30
Total	50	100.00	27	100.00	47	100.00	27	100.00	151	100.00

In the present study 56% of the women with normal pregnancy were aged between 21 to 25 years, 44.44 % of the women with MPE were aged between 21 to 25 years, 46.81% of the women were aged between 26 to 30 years while 44.44% of the women with APE were aged ≤ 20 years.

Table 2: Comparison of gestational age with preeclampsia

Gestational age	Groups								Total	
	NP		MPE		SPE		APE			
	No.	%	No.	%	No.	%	No.	%	No.	%
Preterm	0	0.00	3	11.11	13	27.66	5	18.52	21	14.89
Term	50	100.00	24	88.89	27	57.45	19	70.37	120	85.11
Total	50	100.00	27	100.00	40	85.11	24	88.89	141	100.00

$p<0.001$

In this study the frequency of preterm neonates was noted in 18.52% of the mothers with APE compared to 27.66% of the mothers with SPE and 11.11% of the mothers with MPE while none of the mother had preterm neonate (0%) in women with normal pregnancy and this difference was statistically significant ($p<0.001$).

Table 3: Comparison of birth weight with preeclampsia

Birth weight of the baby (kg)	Groups								Total	
	NP		MPE		SPE		APE			
	No.	%	No.	%	No.	%	No.	%	No.	%
< 2.5	9	18.00	11	40.74	26	55.32	14	51.85	60	42.55
≥ 2.5	41	82.00	16	59.26	14	29.79	10	37.04	81	57.45
Total	50	100.00	27	100.00	40	85.11	24	88.89	141	100.00

$p<0.001$

In this study 55.32% of the women with SPE and 51.85% of the women with APE gave birth to the neonates who weighed < 2.5 kg ($p<0.001$).

Table 4: Comparison of mean birth weight of the baby

Groups	Number of patients (n)	Birth weight (kg)	
		Mean	SD
NP	50	2.68	0.31
MPE	27	2.49	0.46
SPE	40	2.32	0.46
APE	24	2.27	0.42
Overall	141	2.47	0.44
F value	8.227		
p value	**<0.001**		

In the present study the mean birth weight born to the women with SPE (2.32±0.46 Kg) and APE (2.27±0.42) was significantly low (p<0.001).

Table 5: Comparison of neonatal complications with preeclampsia

Neonatal complications	Groups								Total	
	NP		MPE		SPE		APE			
	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	5	10.00	2	7.41	5	10.64	6	22.22	18	12.77
No	45	90.00	25	92.59	35	74.47	18	66.67	123	87.23
Total	50	100.00	27	100.00	40	85.11	24	88.89	141	100.00

p=0.271

In this study maximum number of babies who were born to the women with APE developed complications (22.22) compared to 10.64% who were born to the mothers with SPE, 7.41% of the neonates who were born to women with MPE and 10% of the neonates who were born to the mothers with normal pregnancy. But this difference was statistically not significant (p=0.271).

Table 6: Comparison of neonatal mortality with preeclampsia

Neonatal mortality	Groups								Total	
	NP		MPE		SPE		APE			
	No.	%	No.	%	No.	%	No.	%	No.	%
Yes	0	0.00	5	18.52	3	6.38	2	7.41	10	7.09
No	50	100.00	22	81.48	37	78.72	22	81.48	131	92.91
Total	50	100.00	27	100.00	40	85.11	24	88.89	141	100.00

p=0.010

In the present study neonatal mortality was noted in 18.52% of the mothers with MPE compared to 6.38% of the mothers with SPE and 7.41% of the mothers with APE and this difference was statistically significant (p=0.010).

Discussion

In this study significantly higher number of preterm neonates were delivered by mothers with SPE (27.66%), APE (18.52%), MPE (11.11% compared to the mothers with normal pregnancy where no preterm delivery was noted (0%) (p<0.001). These findings suggest that, preeclamptic women are at high risk of delivering preterm neonates. In this study significantly higher number of neonates were preterm who were born to the mothers with LDH levels of >800 IU/L (41.94%) compared to those who were born to the women with LDH levels between 600 to 800 IU/L (14.89%) and those with LDH levels < 600 IU/L (1.59%) (p<0.001). In a study by Hemalatha CR and Kittur S [7]. (2018) from our hospital, mean gestational age at delivery in patients with LDH>800IU/L was 35.0+/- 4.02 weeks. Similar findings have been observed in a study by Jaiswar SP *et al*. (2011) [8] in whom mean gestational age at delivery in patients with LDH>800IU/L was 35.25 +/-3.23 weeks in.2 in another study by Umasatyasri Y. et.al. (2015) [9] study the mean gestational age during delivery was 37.60±2.76 weeks in LDH levels <+600 IU/l, 36.71±2.96 weeks when LDH levels between 600-800IU/l and 36.27±2.69wks in LDH >800 IU/l.15[D1] Despite the methodological differences, the finding of the present study are in agreement with the studies by Hemalatha CR and Kittur S [7]. (2018) Jaiswar SP *et al*. (2011) [8] and Umasatyasri Y. et.al. (2015) [9]. Preterm newborns are more likely to develop unfavorable fetal outcome as compared with term ones. APGAR score has significant statistical association with fetal management outcome. Newborns with low Apgar score are more likely to develop unfavorable fetal outcome as compared with newborns with good Apgar score.

In this study more than half of the women (55.32%) with SPE and APE (51.85%) gave birth to the neonates who had low birth weight (< 2.5 kg) compared to 18% of the mothers who had normal pregnancy (p<0.001). Also the mean birth weight among the neonates who were born to the women with SPE (2.32±0.46 Kg) and APE (2.27±0.42) was significantly low compared to the women who had normal pregnancy (2.68±0.31) (p<0.001). These findings hypothesize that, women with preeclampsia are likely to deliver babies with low birth weight. Furthermore, significantly higher number of neonates who were born to the mothers with LDH levels of >800 IU/L had birth weight of <2.5 kg (67.74%) compared to those who were born to the women with LDH levels between 600 to 800 IU/L (53.19%) and those with LDH levels < 600 IU/L (22.22%) (p<0.001). Hence it may be postulated that, neonates delivered by the women with raised LDH levels who develop preeclampsia are likely to have low birth weight. This indicates increase in preterm deliveries in patients with higher LDH levels in the present study. Some studies showed association of low birth weight of infants with increase in serum LDH levels. This is in accordance with the study done by Umasatyasri Y. et.al. (2015) [9]. Jaiswar SP *et al*. (2011) [8] noted with LDH levels <600 IU/l, the mean baby weight was 2.426±0.791 kg, LDH levels 600-800 IU/l, the mean baby weight was 1.992±0.618 kg while with LDH levels >800 IU/l it was 1.979±0.787 kg (P=0.019). This observation was similar to present study indicating that there is reduction in the average weight of babies with higher level of LDH. On the contrary, Qublan HS et.al. (2005) [10] did not find any significant association.

Conclusion

In this study maximum number of babies who were born to the women with APE developed complications (22.22) compared to 10.64% who were born to the mothers with SPE, 7.41% of the neonates who were born to women with MPE and 10% of the neonates who were born to the mothers with normal pregnancy. But this difference was statistically not significant (p=0.271).

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