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ORIGINAL ARTICLE

Comparison of Adverse Maternal Outcome in Early Onset Versus Delayed Onset Preeclampsia

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ABSTRACT

Aim: To determine the association of adverse maternal outcome with early onset preeclampsia than delayed onset preeclampsia.
Study Design: Cohort study.
Place and Duration of Study: Department of Obstetrics & Gynecology Unit-II, DHQ Hospital, Mirpur, AJK from 30th December 2020 to 29th June 2021.
Methodology: A total of 60 (30 early onset pre-eclampsia and 30 delayed onset pre-eclampsia) females of age 18-40 years with parity <5, presenting at gestational age >24 weeks were included. Patients with chronic or gestational diabetes (BSR>186mg/dl), chronic hypertension (BP≥140/90mmHg), cardiac disease (on medical record), multiple gestation (on ultrasound), abnormal placenta (abruption, previa, accrete, increta, percreta on ultrasound) were excluded. Females were evaluated for eclampsia (BP>160/100mmHg along with convulsions), DIC, HELLP syndrome and avascular tubular necrosis by using blood samples and ultrasound findings.
Results: The adverse maternal outcome i.e. eclampsia was recorded in 11 (36.67%) in exposed group (early onset pre-eclampsia) versus 04 (13.33%) in unexposed group (delayed onset pre-eclampsia) (p= 0.053; relative risk = 2.75), acute tubular necrosis was recorded in 05 (16.67%) in exposed group (early onset pre-eclampsia) while 00 (0.0%) in unexposed group (delayed onset pre-eclampsia) (p = 0.099; relative risk = 11.00), DIC was recorded in 03 (10.0%) versus 00 (0.0%) respectively (p= 0.192; relative risk = 7.00) and HELLP syndrome in 06 (20.0%) versus 00 (0.0%) respectively (p = 0.076; relative risk = 13.0).
Practical Implication: We recommend that a proper protocol should be designed in these high risk patients for antenatal monitoring and proper management plans in order to reduce the morbidity and mortality of the mother and fetus.
Conclusion: This study concluded that adverse maternal outcome is higher in early onset preeclampsia as compared to delayed onset preeclampsia.
Keywords: Preeclampsia, Adverse Maternal Outcome, Eclampsia.

INTRODUCTION

Hypertensive disorders of pregnancy are responsible for significant maternal and perinatal morbidity and are second leading cause after embolism of maternal mortality.[1] They complicate approximately 12-22% of all pregnancies.[2] Preeclampsia (PE) is a pregnancy specific syndrome and a leading cause of maternal and fetal morbidity and mortality[1]. Pre-eclampsia is estimated to affect 8 370 000 women every year in the world[3].

Preeclampsia is a multi-systemic disorder characterized by hypertension and new-onset proteinuria which develops after the 20th week of pregnancy[3,4]. Preeclampsia occurs in approximately 28% of all pregnancies and accounts for one of the major placental-related pregnancy complications. Preeclampsia is a complex disease characterized by increased maternal blood pressure and proteinuria during pregnancy and increased risks of fetal and maternal complications.[5]It is a global problem and complicates approximately 10-17% of pregnancies. The incidence of preeclampsia is 2 to 10% of all pregnancies in the world. According to WHO the incidence is 7 times greater in developing countries compared to developed countries[6].

Currently there has been a change in the definition and understanding of preeclampsia, known as Early Onset Preeclampsia and late onset preeclampsia. Early onset where preeclampsia occurs at <34 weeks of gestation. Even though the presenting features overlap, there are differences in maternal and perinatal outcome, prognosis and complications. Early and late onset preeclampsia have different etiologies and should be considered as different disease.[7] Although it is known that defective trophoblast invasion could result in reduced placental growth, it is unclear when placental growth is adversely affected during pregnancy and how the pathological mechanisms differ between early-onset preeclampsia and late-onset preeclampsia[8].

Early onset preeclampsia is the most severe clinical variant of disease occurring in 5-20% of all cases of preeclampsia and is associated with neonatal morbidity and mortality. Late onset preeclampsia occurring in about 75-80% of all cases of preeclampsia; which are associated with maternal morbidity, normal birth weight and normal placental volume[9]. It has been reported that the frequency of eclampsia was high (30.4%) in early onset preeclampsia than 3.0% in late onset preeclampsia (RR=4.5, 95% CI; 3.0-6.8, p<0.0001)[10].

No more studies have been conducted on variables as we are going to observe in our study. So this study would help us to determine the association of adverse maternal outcome with early onset preeclampsia than delayed onset preeclampsia.

MATERIALS AND METHODS

Sample size of 60 females; 30 in each group is calculated with 80% power of study, 5% level of significance and taking expected percentage of eclampsia i.e. 30.4% in early onset preeclampsia and 3.0% in late onset preeclampsia. After ethical committee permission, sixty females who fulfilled our selection criteria were enrolled in the study from OPD of Department of Obstetrics & Gynaecology, DHQ Hospital, Mirpur, AJK. Informed consent was obtained from all patients. Demographic details (name, age, parity, BMI, gestational age) were noted. Then females were divided in two groups i.e. exposed group with early onset preeclampsia and

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unexposed group with late onset preeclampsia. Females were evaluated for eclampsia (BP>160/100mmHg along with convulsions), DIC, HELLP syndrome and avascular tubular necrosis by using blood samples and ultrasound findings (as per operational definition). Females with complications were managed as per standard protocol. All this information was recorded.Data was stratified for age, gestational age, BMI and parity. Poststratification, 2x2 contingency tables was generated to calculate relative risk between adverse outcome and early onset preeclampsia for each strata. Relative risk >1 was considered as significant.

RESULTS

Age range in this study was from 18 to 40 years with mean age of 27.72±5.32 years. The mean age of women in group A was 27.70±5.03 years and in group B was 27.97±5.55 years. Majority of the patients 73.33% were between 18 to 30 years (Table 1). The mean gestational age in group A was 25.35±2.64 weeks and in group B was 34.50 ± 2.76 weeks. The mean parity was 2.51±1.17 (Table 2). Mean BMI was 29.86 ± 3.64 kg/m2 (Table 3). The mean height was 1.52±12.92 m and weight was 74.92±12.85 kg. The adverse maternal outcome i.e. eclampsia was recorded in 11 (36.67%) in exposed group (early onset pre-eclampsia) versus 04 (13.33%) in unexposed group (delayed onset pre-eclampsia) (p= 0.053; relative risk = 2.75), acute tubular necrosis was recorded in 05 (16.67%) in exposed group (early onset pre-eclampsia) while 00 (0.0%) in unexposed group (delayed onset pre-eclampsia) (p0.099; relative risk = 11.00), DIC was recorded in 03 (10.0%) versus 00 (0.0%) respectively (p0.192; relative risk = 7.00) and HELLP syndrome in 06 (20.0%) versus 00 (0.0%) respectively (p0.076; relative risk = 13.0) (Table 4). Stratification of eclampsia with respect to age, parity and BMI (Table 5).Stratification of ATN with respect to age, parity and BMI (Table 6).Stratification of DIC with respect to age, parity and BMI (Table 7). Stratification of HELLP syndrome with respect to age, parity and BMI (Table 8).

Table 1: Age distribution of both groups (n=60)

Age (years)	Exposed (n=30)		Unexposed (n=30)		Total (n=60)	
	No.	%	No.	%	No.	%
18-30	23	76.67	21	70.0	44	73.33
31-40	07	23.33	09	30.0	16	26.67
Mean±SD	27.70±5.03		27.97±5.55		27.72±5.32	

Table 2: Distribution of patients according to parity in both groups

Parity	Exposed (n=30)		Unexposed (n=30)		Total (n=60)	
	No.	%	No.	%	No.	%
Para ≤2	15	50.0	14	46.67	29	48.33
Para >2	15	50.0	16	53.33	31	51.67
Mean±SD	2.50±1.17		2.53±1.17		2.51±1.17	

Table 3: Distribution of patients according to BMI in both groups

BMI (kg/m^2)	Exposed (n=30)		Unexposed (n=30)		Total (n=60)	
	No.	%	No.	%	No.	%
≤30	16	53.33	14	46.67	30	50.0
>30	14	46.67	16	53.33	30	50.0
Mean±SD	29.53±3.40		30.07±3.67		29.86±3.64	

Table 4: Association of adverse maternal outcome with early onset pre-eclampsia than delayed onset pre-eclampsia (n=60).

Adverse maternal outcome		Exposed (n=30)		Unexposed (n=30)		P value	RR
		No.	%age	No.	%age		
Eclampsia	Yes	11	36.67	04	13.33	0.053	2.75
	No	19	63.33	26	86.67		
Acute tubular necrosis	Yes	05	16.67	00	0.0	0.099	11.00
	No	25	83.33	30	100.0		
DIC	Yes	03	10.0	00	0.0	0.192	7.00
	No	27	90.0	30	100.0		
HELLP syndrome	Yes	06	20.0	00	0.0	0.076	13.0
	No	24	80.0	30	100.0		

Table 5: Stratification of eclampsia with respect to age, parity and BMI (n=60).

Effect modifiers		Exposed (n=30) Eclampsia		Unexposed (n=30) Eclampsia		P value	RR
		Yes	No	Yes	No		
Age (yrs)	18-30	08	15	02	19	**0.76**	3.65
	31-40	03	04	02	07	0.388	1.93
Parity	≤2	05	10	03	11	0.482	1.56
	>2	06	09	01	15	0.068	6.40
BMI (kg/m2)	≤30	08	08	02	12	0.074	3.50
	>30	03	11	02	14	0.519	1.71

Table 6: Stratification of acute tubular necrosis with respect to age, parity and BMI (n=60).

Effect modifiers		Exposed (n=30) Acute tubular necrosis		Unexposed (n=30) Acute tubular necrosis		P value	RR
		Yes	No	Yes	No		
Age (yrs)	18-30	03	20	00	21	**0.210**	6.42
	31-40	02	05	00	09	0.214	6.25
Parity	≤2	03	12	00	14	0.200	6.56
	>2	02	13	00	16	0.269	5.31
BMI (kg/m2)	≤30	04	12	00	14	0.152	7.94
	>30	01	11	00	16	0.390	3.92

Table 7: Stratification of DIC with respect to age, parity and BMI

Effect modifiers		Exposed (n=30) DIC		Unexposed (n=30) DIC		P value	RR
		Yes	No	Yes	No		
Age (yrs)	18-30	01	22	00	21	0.529	2.75
	31-40	02	05	00	09	0.214	6.25
Parity	≤2	02	13	00	14	0.305	4.69
	>2	01	14	00	16	0.468	3.19
BMI (kg/m2)	≤30	02	14	00	14	0.325	4.41
	>30	01	13	00	16	0.443	3.40

Table 8: Stratification of HELLP syndromewith respect to age, parity and BMI

Effect modifiers		Exposed (n=30) HELLP syndrome		Unexposed (n=30) HELLP syndrome		P value	RR
		Yes	No	Yes	No		
Age (yrs)	18-30	04	19	00	21	0.149	8.25
	31-40	02	05	00	09	0.214	6.25
Parity	≤2	05	10	00	14	0.103	10.31
	>2	01	14	00	16	0.467	3.19
BMI (kg/m2)	≤30	03	13	00	14	0.215	6.18
	>30	03	11	00	16	0.159	7.93

DISCUSSION

It has been pointed out that women with HDP may experience various complications, adverse outcomes to the fetus and mortality. Hypertensive disorders in pregnancies are associated with fetal growth restriction, perinatal asphyxia, iatrogenic prematurity, stillbirths, preterm delivery, perinatal death, neonatal mortality and affects vital maternal organ system such as renal, hepatic, cardiorespiratory, fetoplacental and hematologic[11]. Studies showed that about 30,000 maternal deaths105, 30% of maternal near-miss events[12], 16% of 2.6 million stillbirths[13], 10% perinatal deaths (8/1000live births) are associated with HDP[14].

The primary objective of HDP treatment is to prevent potential maternal complications and death whose importance to the fetus is dubious. Moreover the risk of perinatal death from hypertensive disorders in pregnancy is more daunting than the maternal death. For instance, the risk of perinatal death from severe preeclampsia is 13% whereas maternal death is less than 1% let-alone the short and long term consequences in surviving new-born[15]. Early onset preeclampsia is often defined as a syndrome of first pregnancies and its underline cause largely unknown[16]. It has been suggested that early onset and late onset preeclampsia should be regarded as different forms of disease[17]. Egbor and associates evaluated morphometric placental villous and vascular abnormalities in early and late onset preeclampsia. In contrast, early-onset preeclampsia was associated with placental dysfunction marked by a reduction in placental weights, volume or the intervillous space, terminal villous volumes and surface area[18]. Moldenhauser and colleagues studied placental lesions according to gestational age at delivery. The study found that the rate of placental lesions were higher the earlier the gestational age at the time of delivery, compared with normotensive control subjects[19]. It is not clear whether the implantation and placental abnormality recurs and affects fetal birth weight and length of gestation despite a normotensive second pregnancy[20].

In a local study[21] on 75 women with severe pre-eclampsia, 52 (69%) were between 20-30 years of age. About 35(46.6%) were primigravida. Majority 44(58.7%) were un-booked. 48(64%) women were presented at 31-37 weeks of gestation. Headache was most common complaint in 49(65.3%) followed by epigastric pain, blurring of vision and weight gain. 60(80%) women had blood pressure >160/110mmHg at the time of admission. Common mode of delivery was Caesarean section in 55(73.3%) women. Pre-eclampsia related complications were placental abruption occurred in 9(12%), eclampsia in 3(4%) and renal failure seen in 2(2.7%) women. There were 3(4%) maternal deaths. Perinatal outcome was poor due to severe pre-eclampsia. 14 were intrauterine deaths and 18 were neonatal deaths[21].

Aali[22] HELLP (5%), pulmonary edema (3.6%), ARF (5.4%), Abruption (5.6%) were noted in patients with complicated severe preeclampsia. A study by Yucesoyet al[23] noted eclampsia (11%), abruption (1.5%) and maternal mortality in 3% patients. Studies by Haddad[24] Wodeselsassie[25] support these results. In a study[27] frequency of occurrence of pre-eclampsia was found to be 1.55%. Most patients were between 20-25 (44%) years of age and primigravidas were most afflicted (48.8%). Eclampsia (2%) abruption (32%) acute renal failure (32%) HELLP Syndrome (8%) were the associated maternal complications. Maternal mortality was 4.0% and perinatal morality was 30%[27]. One of the predictor variables for perinatal outcomes of severe preeclampsia–eclampsia was parity. Newborns delivered from nully and primiparas women were more likely to develop unfavorable perinatal outcomes when compared with multi paras women with the odds ratio of (AOR = 8.3, 95% CI 6.27, 27.02) and (AOR = 5.2, 95% CI 4.15, 13.97) respectively.[28] It might be due to the fact that frequency and severity of preeclampsia–eclampsia is more common in nullyparas and primiparas which may possibly affect the perinatal outcomes.Gestational age was also associated with the outcome variable. Pregnancies interrupted from 20 to 27 and 28 to 36 completed weeks were with high unfavorable perinatal outcomes when compared with pregnancies terminated from 37–40 weeks with the odds ratio of (AOR = 9.6, 95% CI 2.00, 18.65) and (AOR = 5.4, 95% CI 1.98, 14.73) respectively[29].

Early onset preeclampsia (EOPE) is associated with increased risk of multi organ involvement including hepatic, hematologic, arterial, renal and adverse maternal and perinatal outcomes as compared with late onset preeclampsia (LOPE). There are limited number of studies and reviews that have compared characteristics of early onset and late onset pre-eclampsia. In a study conducted by Aziz, Mose, Indonesia shows maternal and perinatal morbidity and mortality is higher in EOPE group[30]. Valensis et al reported that patients who were diagnosed with EO-PE had higher total vascular resistance, while late-onset patients had low vascular resistance[31]. In a study conducted by Sreedeviatluri and Nandish in Mysore shows EOPE is more severe clinical entity than LOPE with high risk for life threatening maternal complications and fetal mortality[32]. The distinction between EO- and LO-PE is a relatively modern concept and is becoming widely accepted as a better indicator of disease significance than the classic “mild” vs “severe” terminology[33]. It was widely demonstrated that gestational age at birth had a major impact on perinatal outcome.Medical resources, diagnosis, and treatment must improve in developing countries. There are limited resources available: lack of access to medical and health resources to the patients about disease; limited knowledge and trainings, and awareness about disease. The trainings should be conducted to improve the health literacy and how to access the medical resources for patients in Pakistan[34-40].

CONCLUSION

This study concluded that adverse maternal outcome is higher in early onset preeclampsia as compared to delayed onset preeclampsia. So, we recommend that a proper protocol should be designed in these high risk patients for antenatal monitoring and proper management plans in order to reduce the morbidity and mortality of the mother and fetus.

Conflict of interest: Nil

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