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ORIGINAL RESEARCH

Correlation of Clinical, Hematological and Biochemical Parameters in Women with Severe Preeclampsia and Maternal Outcome: An Observational Study

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ABSTRACT

Background: To find correlation of clinical, hematological and biochemical parameters in women with severe preeclampsia and maternal outcome and to study the maternal and perinatal outcome in severe preeclampsia and.

Materials and Methods: The study was done in the Department of Obstetrics and Gynaecology, Raichur Institute of Medical Sciences, Raichur. 140 patients with blood pressure ≥ 160/110 mm of hg with proteinuria or Blood pressure ≥ 140/90 mm of hg with proteinuria of ≥ 2+ were involved in the study. Statistical analysis was done by applying chi-square.

Results: Out of 140 women with severe preeclampsia, 66.4% had no complications, 20.7% had eclampsia and 15% had other complications (19.1% renal failure, abruption 10.6%, 8.5% HELLP, one each of PPH, cardiomyopathy and cerebral hemorrhage). Mean arterial pressure of >127mm of Hg was found in 31.8% women with eclampsia and 18.2% with other complications. Urine protein >2+ 22.2% had eclampsia and 15.8% had other complications . Twenty-four-hour urine protein > 3g was observed in 6%. Hemoglobin > 13g% and total count > 11000 cells/cumm had no correlation with the maternal outcome related to severe preeclampsia. Platelets <1 lakh/ml were found to have correlation with other complications related to preeclampsia but not with eclampsia. Serum uric acid >7U/l was found to have significant correlation with eclampsia as well as other complications related to preeclampsia. Serum creatinine>1.2mg/dl 35.2% had eclampsia and 52.9% had other complications. Eighty one percent of the group had poor perinatal outcome(IUGR 30.7%, stillbirths 28.9%, neonatal deaths 7.01%); 77.1% neonates required NICU admission. Out of 140 delivered 83.5% were preterm and 16.4% were term. Stillbirth and neonatal deathswere more 58% and 41% among very low birth weight babies, 66% survived in this group. Umbilical artery doppler study was done in 70% of women, 25% detected to have IUGR, 45.7% had appropriate growth and 21.9% were abnormal.

Conclusion: Mean arterial pressure >126 mm of Hg, uric acid >7mg/dl, platelet count <1,00,000 cells/cc, serum creatinine>1.2 mg/dl were found correlate significantly with

poor maternal outcome in women with severe preeclampsia. Prematurity and fetal growth restrictions are the main factors causing perinatal morbidity/ mortality. Timely cesarean delivery seems to improve perinatal outcome in settings with facilities for newborn care.

Keywords: Preeclampsia, LDH, Uric acid, maternal outcome

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INTRODUCTION

Preeclampsia is the most challenging clinical entity affecting both mother and the fetus. It is one of the leading causes for maternal as well as perinatal morbidity/ mortality. Preeclampsia is the most challenging clinical entity affecting both mother and the fetus. Traditionally women with severe preeclampsia-eclampsia are delivered immediately regardless of the consequences of extreme fetal prematurity. Delaying this definitive management for severe preeclampsia to at least gain benefit of antenatal steroids and to organize resources for managing the anticipated complications is referred to as expectant management. The investigative work up for end organ dysfunction related to preeclampsia guides in timing the delivery. The present observational study is an attempt to analyse maternal and perinatal outcome in severe preeclampsia and to find the usefulness if the investigative work up as predictors of outcome. In developing nations, the incidence of the disease is reported to be 4-18% with hypertensive disorders being the second most common obstetric cause of stillbirths and early neonatal deaths. Approximately 1,00,000 women die worldwide per annum because of eclampsia. It is said that pre-eclampsia and eclampsia contribute to death of a women every 3 minutes worldwide. In India, incidence is 5-15% incidence being more in primigravida around 15% and multigravida around 10%.

Preeclampsia is mild in 75% of cases and severe in 25% of them.6 In its extreme, the disease may lead to liver and renal failure, disseminated intravascular coagulopathy (DIC), and central nervous system (CNS) abnormalities. Besides placental insufficiency may cause fetal growth restriction and related perinatal complications; premature birth either spontaneous or induced for indications, is an important cause for perinatal morbidity/ mortality. Hemorrhage and hypertensive disorders together account for the largest proportion of maternal deaths in developing countries.

Contradicting reports are found in the literature regarding the role of investigative workups as indicators of severe disease that will direct to terminate pregnancy; also there are conflicting reports regarding the predictive role of these investigative parameters.

The present observational study is an attempt to analyze maternal and perinatal outcome in severe preeclampsia and to find the usefulness of the clinical and investigative work up as predictors of outcome.

MATERIALS & METHODS

An observational study was conducted in the Department of Obstetrics and Gynecology, Raichur Institute of Medical Sciences, Raichur. 140 patients were included in the study. Women with severe preeclampsia- eclampsia was evaluated and managed as per the hospital protocol. Blood pressure of patient was measured in supine position after 10 minutes of rest using mercury sphygmomanometer with appropriate cuff size tied at heart level. The women were monitored clinically as well as by investigative work up. Proteinuria was estimated daily by dip stick method. Serum analysis of uric acid was done by urease method and serum creatinine was done by Jaffe rate blanked method to check renal function, liver function

tests(AST, ALT, LDH, bilirubin etc.) were done by using automated analyser. Platelet count and protein estimates in 24-hour urine (biuret method) were done at admission and as indicated by the clinical disease behavior. Peripheral smear was done to look for spherocytosis, schizocytosis, reticulocytosis, anisocytosis, triangular cells, helmet cells and burr cells for screening for HELLP syndrome. Fundus oculi was examined using ophthalmoscope for hypertension induced changes and the findings was graded as per Keith, Wagner and Barker (1939) classification. Sonographic estimation of fetal growth, weight and amount of liquor was carried out. Fetal condition was monitored by fetal heart rate auscultation and fetal heart rate tracings, Doppler analysis and modified biophysical profile.

Women were put on antihypertensive drugs- Alpha methyl dopa (maximum dose up to 2g/day)/ labetalol (800mg/day)/ amlodepine and/ or nifedipine(up to 30mg); dose adjustments or treating with additional drugs was done as per individual requirement. Two doses of intramuscular dexamethasone 12 mg, 12 hours apart was given for preterm salvageable pregnancies.

Women on expectant management was asked to report if they had headache or epigastric pain or vomiting or visual disturbances, those with eclampsia received magnesium sulfate(Pritchard regime) with standard monitoring for magnesium toxicity

Pregnancy was terminated for eclampsia, uncontrolled hypertension in spite of being on maximum dose of antihypertensives, persisting/ progressively deteriorating clinical symptoms or the biochemical markers, occurrence of complications such as placental abruption, eclampsia, renal failure and indication of non-reassuring fetal status. The decision regarding the route of delivery was based on estimated fetal weight, salvageability, gestational age, amniotic fluid index, fetal status and cervical score.

All investigative parameters were compared with the maternal outcome. Clinical, hematological and investigative parameters considered and the cut off are as follows:

Clinical parameters: Mean arterial pressure was calculated using the formula: MAP = [(2 x diastolic)+systolic] / 3= [(2X 110)+ 160] / 3= 127 mm of Hg. It was calculated using blood pressure at the time of admission. Fundoscopy- Hypertensive retinopathy was graded according to Keith-Wagener-Barker classification of hypertensive retinopathy. Patients who had blood pressure ≥ 160/110 mm of hg with proteinuria of any degree or Blood pressure ≥ 140/90 mm of hg with proteinuria of ≥ 2+ were included in the study. Institutional ethics committee approval was taken before starting the study.

Hematological and biochemical parameters are as in [Table1].

Table 1: Hematological and biochemical parameters in severe preeclampsia

Parameters	**Cut off values**
Mean arterial pressure	≥127 mm of hg
Urine protein random	≥2+
24hr urine protein	≥3g
Hemoglobin	≥11g%
Total count	≥11000 cu/mm
Platelets	<1 lakh/ml
Serum uric acid	≥7 mg/dl
Serum creatinine	≥1.2 mg/dl

These were compared with various adverse maternal outcome. The maternal outcome variables were divided in to three categories: 1. Normal outcome, 2. Eclampsia, 3. Other complications (Abruprtion, HELLP, renal failure, pulmonary edema, cardiomyopathy, cerebral hemorrhage).

Perinatal outcome measures studied were live births, fetal growth restriction (IUGR), still births, neonatal complications (sepsis, intraventricular hemorrhage, hyperbilirubinemia, necrotizing enterocolitis) and neonatal deaths. Perinatal outcome was studied against different gestational age and birth weight categories and also depending on fetal Doppler abnormalities. Statistical analysis was done by applying chi-square.

RESULTS

During the period of study 150 patients who satisfied the inclusion criteria were included in the study. [Table 2] showing the mean age of women with severe preeclampsia was 27.2 years. Eleven were above 35 years and 6 were teenage pregnancies. Two third of the group (61.4%) was primigravidae. Previous history of preeclampsia, bad obstetric history and chronic hypertension was seen in 25 women. Associated medical disorder systemic lupus erythematosus and pheochromocytoma was seen in 4 women. outside (85%) and only 15% were booked with us. Due to maternal or fetal indications emergency cesarean delivery was done in 105(75%) and 35(25%) had vaginal delivery. Three women had cesarean delivery outside before referral here. Among vaginal deliveries 4 had spontaneous onset of labor and 25 had induced preterm delivery. It was observed that mean gestational age at diagnosis of severe preeclampsia was 30 weeks and gestational age at delivery was 33 weeks. Most of them were referred from outside.

Table 2: Patient profile

Patient characteristics		**Observation**
Mean age (years)		27.2 (18-38)
Parity (Number)	Primigravida Multigravida	86 (61.4%) 54(38.5%)
Previous history [number (%)]	Preeclampsia BOH Chronic hypertension	12(8.5%) 5(3.5%) 6(4.2%)
Associated medical disorder	SLE Pheochromocytoma	2 2
Gestational age (weeks)	At diagnosis At delivery	30(20-39.3) 33(20-40)
Antenatal Booking [Number (%)]	Booked Referred	21(15%) 119(85%)
Vaginal delivery Caesarean delivery		35(25%) 105(75%)

[Table 3] shows that systolic blood pressure was >160 mm of Hg only in 42(30%) of the group. The remaining 70% of them though the systolic BP was not in the range of severe PE, had other criteria of severe preeclampsia. Similarly only 22 women(15%) had diastolic BP more than 110 mm of Hg. Blood pressure (both systolic and diastolic) ≥ 160/110 mm of Hg was seen in 44(31.4%).

Signs of impending eclampsia were seen in 26 women with severe preeclampsia. Twenty-nine (20%) women had eclampsia. Four women required ICU admission: Two had renal failure and one each had HELLP and disseminated intravascular coagulation.

Table 3: Disease profile

Clinical parameter		**Number(%)**
Systolic BP(mm of Hg)	≤ 140	35(25)

n = 140	140 - 160	63(45)
	>160	42(30)
Diastolic BP(mm of Hg) n = 140	90 – 110	118(84.3)
	>110	22(15.7)
Impending eclampsia (n=26)	Severe headache	10(38.4)
	Epigastric or upper right quadrant pain	5(19.2)
	Persistent visual symptoms	6(23.07)
	Oliguria	5(19.2)
Eclampsia (n = 140)		29(20.1)
ICU admission		4(2.85)

[Table 4] shows that abnormal value above cutoff to define severe PE was found commonly for proteinuria (90%). Other investigations had values in the severe PE range only in 12-34% of the group.

Table 4: Investigation profile

Investigations	**Cut off values**	**Number (%)**
Urine protein	1+	14 (10)
	≥2+	126 (90)
24 hr urine protein (n=42)	<3 g	33(23.5)
	3-5 g	9(6.4)
Serum creatinine(mg/dl)	≤1.2	123 (87.8)
	>1.2	17 (12.1)
Serum uric acid(mg/dl)	≤7	106 (75.7)
	>7	34 (24.2)
AST(I/U)	≤70	119 (85)
	>70	17 (12.1)
ALT(I/U)	≤70	123 (87.8)
	>70	17 (12.1)
LDH(I/U)	≤600	96 (68.5)
	>600	44 (31.4)
Platelets (lakh/ml)	≤1	25 (17.8)
	>1	115 (82.1)

[Table 5] shows that out of 140 women studied 20.7%(29) had eclampsia, abruption 3.5%(5), HELLP 4.28%(6), renal failure 6.42%(9), pulmonary edema 2.85%(4), cardiomyopathy 0.71%(1), cerebral hemorrhage 0.71%(1). 17 women had more than 1 complication (HELLP, eclampsia, pulmonary edema, cerebral infarct, renal failure). Two women were discharged against medical advice one with renal failure with pulmonary edema and the other with HELLP. There was no maternal mortality among 140 women studied.

Among 29 women with eclampsia, 25 women had antepartum eclampsia at the time of arrival and 2 each had antepartum and postpartum eclampsia after admission.

Table 5: Maternal complications.

Complication	**Frequency (Number)**	**%**
Eclampsia	29	20.7
Abruption	5	3.57
HELLP	6	4.28

Renal failure	9	6.42
Pulmonary edema	4	2.85
Cardiomyopathy	1	0.71
Cerebral haemorrhage	1	0.71
Discharged against medical advice	2	1.4
Maternal mortality	Nil	

[Table 6] shows that among the 140 women 114 had perinatal complications [stillbirth 33, neonatal morbidity 81(neonatal deaths 8)]. Of the 114 with perinatal complications 106 were preterm and 8 were term babies. In the preterm group 81(69%) babies survived; Of the 88 live newborns the survival was 92%. Among term babies 4(50%) were still births(1 due to abruption, 2 were due to severe IUGR and oligoamnios and 1 due to severe oligoamnios). there was one neonatal death due to severe respiratory distress syndrome.

Table 6: Perinatal outcome

	Preterm (n=117)		**Term(n=23)**	
Perinatal outcome	n	%	n	%
Stillbirth	29	27.4	4	50
Live birth	88	75	19	82.6
Neonatal death	7	6.6	1	12.5
NICU admissions	64	60.3	4	50
Respiratory distress syndrome	23	21.7	3	37.5
Sepsis	2	1.9	0	
IVH	1	0.9	0	
Hyperbilirubinemia	9	8.5	5	62.5
NEC	2	1.8	0	
Survivors	81	69	18	78

[Table 7] shows that of the 44 women who had mean arterial pressure >127 mm of Hg 31.8% women had eclampsia and this had significant correlation.

Table 7: Correlation of mean arterial pressure and maternal outcome

Outcome	**<127 mm of Hg (n=96)**	**>127 mm of Hg (n=44)**	**Total (n=140)**	**P value**
Normal	70(72.9%)	23(52.3%)	93(66.4%)	
Eclampsia	15(15.6%)	14(31.8%)	29(20.7%)	0.042
Other complications	13(13.5%)	8(18.2%)	21(15%)	0.646

[Table 8] shows that out of 140 women who had severe preeclampsia 20 had serum creatinine more than 1.2 mg/dl. Among them 5 had normal outcome, 6 had eclampsia and 9 had other complications. Statistically significant ($p > 0.05$) correlation was seen between serum creatinine and maternal complications.

Table 8: Correlation of Serum creatinine and maternal outcome

Outcome	**<1.2mg/dl (n=123)**	**>1.2mg/dl (n=20)**	**Total (n=140)**	**P value**
Normal	88(71.5%)	5(29.4%)	93(66.4%)	
Eclampsia	23(18.6%)	6(35.2%)	29(20.7%)	0.819
Others	12(9.7%)	9(52.9%)	21(15%)	0.025

[Table 9] shows that 37 women who had severe preeclampsia had serum uric acid more than 7 mg/dl. Among them 11 had normal outcome, 16 had eclamspia and 10 had other complications. There was statistically significant correlation between serum uric acid >7mg/dl and both eclampsia and other maternal complications.

Table 9: Correlation of serum uric acid and maternal outcome

Outcome	<7mg/dl (n=106)	>7mg/dl (n=37)	Total (n=140)	P value
Normal	82(77.3%)	11(32.3%)	93(66.4%)	
Eclampsia	13(12.2%)	16(47.1%)	29(20.7%)	0.001
Others	11(10.3%)	10(29.4%)	21(15%)	0.010

[Table 10] shows that out of 140 women with severe preeclampsia 28 had platelets less than one lakh. Among them 10 women had normal outcome, 9 had eclampsia and 9 had other complications. There was correlation between low platelets and maternal complications other than eclampsia.

Table 10: Correlation of Platelet count and maternal outcome

Outcome	<1 lakh/ml (n=28)	≥ 1 lakh/ml (n=115)	Total (n=140)	P value
Normal	10(40%)	83(72.1%)	93(66.4%)	
Eclampsia	9(36%)	20(17.3%)	29(20.7%)	0.626
Others	9(36%)	12(10.4%)	21(15%)	0.003

DISCUSSION

The maternal and perinatal morbidity and mortality due to preeclampsia has come down dramatically in developed countries. This has been achieved by improvements in antenatal care and early hospitalization and proper maternal and fetal survelliance. In developing countries preeclampsia-eclampsia still stands as one of the major complications of pregnancy.

It has been observed that preeclampsia is more common in young or elderly primigravidas and it is reported that maternal age > 35 years was significantly associated with preeclampsia and attributed this to the fact that progressive vascular endothelial damage occurs with aging.[1] In a study by Abuheja AT had a still higher age preponderance (45 – 46 years) of the preeclampsia.[2] However in our study the mean age of the women with preeclampsia was 26 years probably because in India the age of primigravida is mostly between 20-30 years.

It is shown that nulliparous women are at an increased risk which is related to maternal first exposure to chorionic villi.[3] The majority of women in this study were also were primigravida (61.4%).

In the present study, 15% were booked case and 85% were referred cases. All with eclampsia were unbooked in our study similar to 80-90% of the eclampsia being unbooked cases in the studies Zuspan.[3] However Sibai BM,[4] reported that one – third of women with regular prenatal care had abrupt onset of severe preeclampsia/eclampsia without prior positive clinical or laboratory findings.

In this study, the most common maternal complication was eclampsia 20.7%, followed by renal failure (6.42%), HELLP syndrome (4.28%), placental abruption (3.5%) and pulmonary edema (2.8%). Also, there were preeclampsia associated rare complications such as cerebral haemorrhage and cardiomyopathy. This was comparable with the various studies.[5]

In present study 4(2.8%) women required ICU admission because of renal failure, HELLP and pulmonary edema. There was no maternal death in this study. The most common cause

of death are intracranial bleeding and acute renal failure secondary to abruption placentae. It was comparable with the other studies where the ICU admissions in their study were due to HELLP, renal failure, abruption and DIC.[6] The greatest risk of maternal death is when eclampsia develops before 28 weeks of gestation.[4] In this study there were no early onset eclampsia case seen.

Babies born to severe preeclampsia and eclampsia mothers are mainly low birth weight (<2.5kg) which may be due to preterm delivery or intrauterine growth restriction. In present study, of 107 (93%) live new-borns were low birth weight (<2.5 kg). In this group 93 babies survived; and among the very low birth weight (<1000g) new-borns the survival rate was 66%.

In our group there were 75.5% of preterm babies and 5.7% were term babies. Sixty percent of preterm new-borns and 50% of term new-borns required NICU admission, the common neonatal complication in preterm new-borns was respiratory distress syndrome 23 (21.7%) and in term new-borns was hyperbilirubinemia 5 (62.5%). Survival among preterm and term new-borns was 92% and 95% respectively. It has been estimated that of all pregnancies among women suffering from severe preeclampsia and eclampsia, 11.5% to 30% end up as still births or perinatal deaths.[7] In the present study stillbirths were 23.5% and perinatal deaths were 29%. All still births were referred cases.

In our study we considered mean arterial pressure (MAP) as one of the measures to study the correlation with the outcome. We noted that women had eclampsia or other preeclampsia related complications even at blood pressures- systolic or diastolic or both- not at a level beyond the cut off of 160/110 mm Hg for severe preeclampsia. When we considered MAP more than 127mm Hg, 31.8% had eclampsia, 18.2% had other maternal complications, 52.3% had normal outcome and one woman had cerebral haemorrhage for her MAP was > 127 mm of Hg. Studies by Menzies J and Zang J have reported that there is no relation between blood pressure and adverse maternal outcome. [8,9] However Martin Jr,[10] found correlation between systolic blood pressure more than 160 mm of hg and stroke. Systolic blood pressure also had a significant ($p < 0.05$) influence on perinatal deaths in the study by Dhananjay BS among women with eclampsia.[11]

Proteinuria was always thought to be a good indicator of the severity of preeclampsia. In present study random urine protein more than 2+ was seen in 129 patients; 22.2% had eclampsia and 15.8% had other complications. Twenty-four-hour urinary protein testing could be done only for 42 women. In other cases decision to terminate pregnancy had to be taken immediately, hence there was no time for 24 hour analysis. Seventy seven percent of women whose 24-hour urine protein was 3 to 5 grams had normal outcome. Chan P et al and Thangaratnam S et al have found that no level of proteinuria could be defined to predict outcomes and is a poor predictor of either maternal or fetal outcome. [12,13]

There is elevation of haemoglobin and haematocrit caused by decrease in plasma volume. It is known that haemoconcentration is a hallmark of eclampsia, because of haemoconcentration there is decreased regional perfusion and leads to altered cerebral autoregulatory function which can in turn lead to PRES8. In present study one women had haemoglobin more than 13g% and 21.4% had between 11 to 13 g% had eclampsia. HELLP syndrome was seen in women with haemoglobin 11 to 13 g% in 4 and more than 13 g% in two women. One study showed that increased haemoglobin/haematocrit reflects the severity of preeclampsia. So haemoglobin/haematocrit are not good predictors of eclampsia.[14]

Preeclampsia is a proinflammatory state caused by placental hypoperfusion. In this study 105 women had total count more than 11000/cc., 4 patients with HELLP had total count more than 11000/cc. A study by Magann EF,[15] found that there was significant elevation of total WBC count than platelet count. Platelet count varied inversely with WBC counts and the finding of an association between increasing leucocytosis and worsening thrombocytopenia

early in the course of HELLP syndrome supports the hypothesis that it may represent an inflammatory state. But it is not a good predictor for maternal outcome in sever preeclampsia. It is also seen that women with serum uric acid more than 7 mg/dl, 47.1% had eclampsia and 29.4% had other maternal complications. This was comparable with the studies by Parrish M et al,[16] and Menzies et al.[8] In present study Women who had uric acid less than 7 mg/dl 12.2% had eclampsia and 10.3% had other severe preeclampsia related complications.

In the present study it was found that women who had serum creatinine more than 1.2 mg/dl was found to have more other maternal complications like abruption, renal failure etc. Women who had serum creatinine less than 1.2mg/dl was found to have 18.6% eclampsia and 9.7% other maternal complications. Other studies have also found that serum creatinine >1.2 mg/dl is associated with adverse maternal outcome.[17,16]

Platelet count less than 100 X 109 was associated with maternal complications other than eclampsia. This was comparable with the studies Brown MA et al.[17]

CONCLUSION

Maternal and perinatal morbidity is still high among women with severe preeclampsia. Mean arterial pressure >126 mm of Hg, uric acid >7mg/dl, platelet count <1,00,000 cells/cc, serum creatinine >1.2 mg/dl were found correlate significantly with poor maternal outcome in women with severe preeclampsia. Prematurity and foetal growth restrictions are the main factors causing perinatal morbidity/ mortality. Timely caesarean delivery seems to improve perinatal outcome in settings with facilities for new-born care.

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