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ASSESMENT OF SERUM URIC ACID LEVEL AND FETOMATERNAL OUTCOME IN PRE-ECLAMPSIA

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ABSTRACT

Backround Preeclampsia complicates 5-7% of the pregnancies worldwide and is the major cause of maternal and perinatal morbidity and mortality. Serum uric acid is one of the most consistent changes in preeclampsia. An elevated level of uric acid reflects the degree of placental cell destruction as well as severity of preeclampsia. The objectives of our study was to estimate and compare serum UA levels in normal pregnant women and in women with preeclampsia, as a marker for early detection of severity of preeclampsia and to assess the perinatal outcome.1 **Methodology** 25 pregnant women with mild preeclampsia and 25 pregnant women with severe preeclampsia diagnosed as per National High Blood Pressure Education Program working group (NHBPEP) Classification admitted in Obstetrics and Gynecology department at tertiary health care centre **Results** We included total 50 cases fulfilling the eligibility criteria. Out of 50 cases, majority were from 21-30 years i.e. 78% followed by 18% from below 20 years and 4% from above 30 years age group. SUA was elevated in 12 cases i.e. 24%. Mean SUA in our study was 5.47±1.2. We observed statistically significant association of elevated SUA with the fetal outcome like Still birth-25%, Preterm-50%, SGA- 50%, NICU required -58.3%, Low APGAR score-50%, LBW-50% and RDS-41.7% **Conclusion** This study is Descriptive Observational type of study done at Tertiary health care center in the Department of Obstetrics and Gynaecology. The objective of our study was to estimate the serum uric acid levels in participant pregnant women with preeclampsia and also to evaluate the fetomaternal outcome in relation to the serum uric acid level in women presented with pre-eclampsia. We observed statistically significant association of elevated SUA with the maternal risk factors like Placental abruption-25%, thrombocytopenia-58.3%, Hepatic dysfunction-16.7%, ICU hospitalisation-33.3%, IUGR-66.7%, Oligohydramnios-50% and Preterm labour-50%.

KEYWORDS : Uric acid; Pre-eclampsia; Perinatal outcome.

INTRODUCTION

Preeclampsia is a serious pregnancy complication. It is a multisystem disorder characterized by hypertension (blood pressure ≥140/90 mm Hg), proteinuria (24 hr urinary protein ≥0.3 g) with or without pathological edema, beyond the 20th week of gestation in previously normotensive and nonproteinuric woman.[1]

Preeclampsia affects approximately 5–7% of all pregnancies and is associated with several complications. While maternal death due to preeclampsia is less common in developed countries, maternal morbidity is high and is a major contributor to intensive care unit admissions during pregnancy.[2]

Approximately 12 to 25% of fetal growth restriction and small for gestation age infants as well as 15 to 20% of all preterm births are attributable to preeclampsia; the associated complications of prematurity are substantial including neonatal deaths and serious long-term neonatal morbidity.

Thus, clinical prediction of disease complications may facilitate initiation of timely management to avert mortality and morbidity in the mother and baby. Although the etiology is still largely unknown, there are a few hypotheses regarding the pathophysiology and prediction of preeclampsia.[3]

Preeclampsia is associated with uricaemia. Nevertheless, some studies reported that uricaemia is not a consistent predictive factor of preeclampsia.

Uric acid is the end product of purine metabolism in humans and is generated by the action of the enzyme, xanthine oxidase, which catalyzes the last two steps of uric acid conversion: hypoxanthine to xanthine and from xanthine to uric acid.[4]

Uric acid is a potent mediator of inflammation. Uric acid stimulates monocytes to produce pro-inflammatory cytokines IL-1β, IL-6, TNF-α. Uric acid promotes endothelial dysfunction per se which could promote hypertension, vascular disease and renal disease. Since then, there have been a lot of conflicting reports in the literature as regards the association between maternal hyperuricaemia and severity of preeclampsia and pregnancy outcome.[5]

Hence the present study was designed to assess the association of uric acid levels with severity of pre-eclampsia and its relation to fetomaternal outcome.[6]

MATERIAL AND METHODS

Study Design

This study is Descriptive Observational type of study done at Tertiary health care center in the Department of Obstetrics and Gynecology. The study protocol is approved by ethical committee for research studies in a clinical setting at a medical intuition under supervision & guidance.

Study Setting

The study will be conducted in a Tertiary health care center in the department of Obstetrics and Gynecology. The study will be conducted following ethical principles and good clinical practice guidelines as per declaration by World Medical Association's declaration of Helsinki as revised in 2013 and National Ethical Guidelines for Biomedical and Health Research involving Human Participants by Indian Council of Medical Research 2017.

Study Population

25 pregnant women with mild preeclampsia and 25 pregnant women with severe preeclampsia diagnosed as per National High Blood Pressure Education Program working group (NHBPEP) Classification admitted in Obstetrics and Gynecology department at tertiary health care centre

Study Duration

The study was conducted during the period over January 2021 to December 2022.

RESULTS

In this study 25 pregnant women with mild preeclampsia and 25 pregnant women with severe preeclampsia diagnosed as per National High Blood Pressure Education Program working group (NHBPEP) Classification admitted in Obstetrics and Gynecology department at tertiary health care centre were studied. These results are as follows

Table 1: Distribution according to severity of preeclampsia

		Frequency	Percent
Severity of PE	Mild	39	78.0
	Severe	11	22.0
	Total	50	100.0

Mild preeclampsia was seen in 78% and severe in 22% cases.

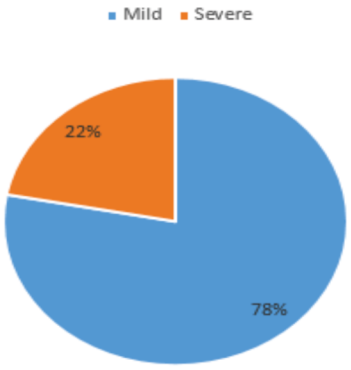

Figure2: Pie diagram showingDistribution according to severity of preeclampsia

Out of 12 cases with elevated SUA, majority i.e. 91.7% had severe preeclampsia and 8.3% had mild PE. Out of 38 cases with normal SUA, all 100% had mild preeclampsia. We observed statistically significant association of SUA with severity of PE ($p<0.05$).

Table 2: Distribution according to fetal outcome

		Frequency	Percent
Fetal outcome	Preterm	7	14.0
	SGA	7	14.0
	NICU admission	7	14.0
	Low APGAR score	9	18.0
	LBW	7	14.0
	RDS	5	10.0

Fetal outcome in our study was as follows: Low APGAR score -18%, Preterm-14%,SGA-14%,NICU admission-14%,LBW-14% and RDS-10%.

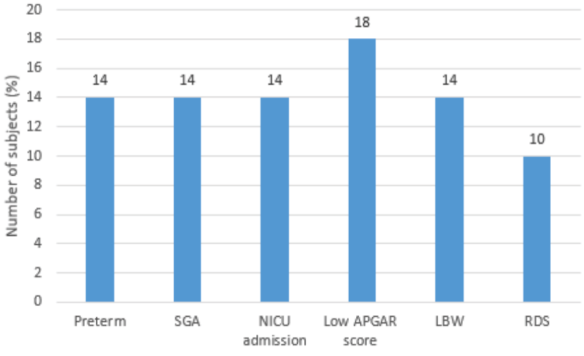

Figure2: Bar diagram showing Distribution according to fetal outcome

We observed statistically significant association of elevated SUA with the maternal risk factors like Placental abruption-25%, thrombocytopenia-58.3%, Hepatic dysfunction-16.7%, ICU hospitalisation-33.3%, IUGR- 66.7%, Oligohydramnios-50% and Preterm labour-50%.

We observed statistically significant association of elevated SUA with the fetal outcome like Still birth-25%, Preterm-50%, SGA- 50%, NICU required -58.3%, Low APGAR score-50%, LBW-50% and RDS-41.7%.

DISCUSSION

Maternal outcome

Maternal outcome in our study was observed as follows: Oligohydramnios-22%, IUGR-18%, Thrombocytopenia-14%, Preterm labour-12%, PROM-10%, ICU hospitalisation-8%, Placental abruption-6%, Hepatic dysfunction-4% and HELLP syndrome-2%.

Meena R. et al[7]reported thatmaternal outcome in both groups, 18% patient had good outcome and discharged on time, 58% had average outcome with different minor ante, intra and postpartum complications, 6% had abruptio placentae, 12% APE, 02% ARF and 04% had PPH in study group as compare to control group all had good outcome which was statistically significant ($p<0.001$)

Fetal outcome

In our study, incidence of still birth in our study was 6%. In our study, fetal outcome in our study was as follows: Low APGAR score -18%, Preterm-14%, SGA-14%, NICU admission-14%, LBW-14% and RDS-10%.

Meena R. et al[7]reported thatfetal outcome in both groups, in study group perinatal morbidity was very high as compare to control, 58% babies were normal, 34% babies were admitted in NICU and 08% were IUFD ,while in control group 92% babies were normal and only 08% babies were admitted in NICU.

Study done by **Magnnan et al**[8] found that there is a positive correlation between increasing/raised SUA level and increased incidence of perinatal mortality, stillborn.

SUA and PE

Out of 12 cases with elevated SUA, majority i.e. 91.7% had severe preeclampsia and 8.3% had mild PE. Out of 38 cases with normal SUA, all 100% had mild preeclampsia. We observed statistically significant association of SUA with severity of PE ($p<0.05$).

Asgharnia M. et al[9]reported thatmean level of uric acid in women with severe pre- eclampsia (5.66 ± 1.46) was significantly higher than non-severe preeclampsia (5.12 ± 1.29) ($p=0.031$).

Maternal and fetal outcome and SUA

We observed statistically significant association of elevated SUA with the maternal risk factors like Placental abruption-25%, thrombocytopenia-58.3%, Hepatic dysfunction-16.7%, ICU hospitalisation-33.3%, IUGR- 66.7%, Oligohydramnios-50% and Preterm labour-50%.

We observed statistically significant association of elevated SUA with the fetal outcome like Still birth-25%, Preterm-50%, SGA- 50%, NICU required -58.3%, Low APGAR score-50%, LBW-50% and RDS-41.7%.

Zangana JM et al[10]reported thatsignificant association between the two groups of level of serum uric acid and different perinataloutcome with highest percentage of (low birth weight 63.77%, admission to NCU 57.48%, caesarean section delivery65.35%, preterm delivery 75.59) were found from 127 mothers with serum uric acid ≥ 6 mg/dL.

Mean SUA in live births was 5.30 ± 1.02 and that of still births was 8.17 ± 0.50. We observed statistically significant difference in the mean SUA between two groups ($p<0.05$). It means SUA was found higher in still births as compared to live

births. Mean SUA in live births was 4.89±0.40 and that of still births was 7.55±0.72. We observed statistically significant difference in the mean SUA between two groups ($p<0.05$). It means SUA was found higher in still births as compared to live births.

Deshpande HG et al[11] observed statistically significant negative correlation between SUA and birth weight, SUA and APGAR at 0,1 and 5 minutes. We observed statistically non-significant negative correlation between SUA and age group.

CONCLUSION

- SUA was elevated in 12 cases i.e. 24%.
- Mean SUA in our study was 5.47±1.2.
- We observed statistically significant association of elevated SUA with the maternal risk factors like Placental abruption-25%, thrombocytopenia-58.3%, Hepatic dysfunction-16.7%, ICU hospitalisation-33.3%, IUGR-66.7%, Oligohydramnios-50% and Preterm labour-50%.
- We observed statistically significant association of elevated SUA with the fetal outcome like Still birth-25%, Preterm-50%, SGA- 50%, NICU required -58.3%, Low APGAR score-50%, LBW-50% and RDS-41.7%
- We observed statistically significant negative correlation between SUA and birth weight, SUA and APGAR at 0,1 and 5 minutes. We observed statistically non-significant negative correlation between SUA and age group.

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