Correlation of Maternal Serum Uric Acid and Perinatal Outcome in Hypertensive Disorders of Pregnancy

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ABSTRACT

Background: Hypertension is a common medical disorder seen in pregnancy and it is responsible for significant maternal and perinatal morbidity and mortality, especially in developing countries. A direct correlation has been observed between maternal serum uric acid levels and adverse fetal outcomes. An assay of uric acid may influence the foetus's delivery timing to avoid further intrauterine complications. **Objectives:** To determine the relationship between maternal serum uric acid level and perinatal outcomes in women with hypertensive disorders in pregnancy and normotensive pregnant women and to determine the critical value of serum uric acid for the occurrence of adverse perinatal outcomes in women with hypertensive disorders in pregnancy. **Method:** This was a hospital-based cross-sectional study in the Department of Obstetrics and Gynaecology, University of Maiduguri Teaching Hospital (UMTH), Maiduguri, Nigeria, from 1st December 2010 to 30th April 2011. Patients were recruited consecutively as they were admitted into the labour ward with a hypertensive disorder in pregnancy. Socio-demographic variables, clinical characteristics, and perinatal outcomes were obtained. An assay of maternal serum uric acid and degree of proteinuria were carried out and recorded on a predesigned proforma. **Results:** The majority of the hypertensive group were unbooked 119(74.4%) and had no formal education 85(53.1%). Twenty-two-point-five percent 36(22.5%) of the hypertensive group were delivered by caesarean section compared to 33(10.3%) of the normotensive group, P 0.008. The hypertensive group delivered earlier (37.1±2.2weeks vs 38.6±0.9) and their babies weighed less (2.45±0.7 vs 3.15±0.5kg) (P <0.001). Hyperuricaemia was significantly associated with adverse perinatal outcomes such as prematurity (P <0.001), low birth weight (P <0.001), intrauterine growth restriction (P <0.001) foetal distress (P <0.001), and early neonatal death (P <0.001). The critical value of maternal serum uric acid was 455umol/l and it was significantly associated with low birth weight (P <0.001), intrauterine growth restriction (P <0.001), intrauterine fetal death (P 0.001) and admission to special care baby unit (P 0.017), among the hypertensive group after regression analysis for confounders. **Conclusion:** Elevated maternal serum uric acid level is significantly associated with poor perinatal outcomes in women with hypertensive disorders in pregnancy and a critical value of 455umol/l can be used to time delivery and avert adverse perinatal outcomes.

Keywords: Correlation, Hypertensive disorders in pregnancy, maternal serum uric acid, perinatal outcome.

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Introduction

Hypertensive disorders of pregnancy complicate 5-10% of all pregnancies[1] and it accounts for about 15% of maternal deaths, 18% of perinatal deaths, and 46% of infants born small for gestational age.[2] In developed countries where prenatal care is routine, hypertensive disorders account for 15% of preterm deliveries in a year.[3]

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The clinical course of hypertension in pregnancy is progressive and characterised by continuous deterioration that is ultimately stopped by the delivery of the fetus and placenta.[4,5] Although early detection and appropriate management of pregnancy are associated with improved outcomes for both the mother and the foetus, this is not always obtainable in developing countries where women fail to access antenatal care and present with severe disease or eclamptic fits when the lives of both mother and fetus are in danger.[1,4,5] However, the 2018 demographic survey reported that 67% of Nigerian women attended antenatal clinics at least once giving room for early detection of hypertension.[6] The obstetrician is then faced with balancing the appropriate timing of fetal delivery to avoid serious perinatal sequelae against the severity of the maternal disease.

Hyperuricaemia, characterised by high levels of uric acid in the blood, is common in patients with hypertensive disorders in pregnancy. It is one of the earliest and most consistent laboratory findings. Uric acid is the end product of nitrogen metabolism and is produced when the liver enzyme uricase is unable to convert uric acid to allantoin in humans. This leads to significantly higher levels of uric acid in humans compared to most other mammals.[7]

Uric acid is produced from amino acid precursors, purines in the diet, and the breakdown of DNA, RNA, and ATP. Two-thirds of it is excreted by the kidneys, and one-third by the gut.[7-9]

Uric acid acts as an antioxidant, but at higher levels or under hypoxic conditions, it can cause gout, kidney stones, metabolic disorders, cardiovascular issues, and hypertension.[7]

In populations with established normal uric acid values, adolescents and adults with new-onset hypertension often have serum uric acid levels above the age-specific cutoff values.[9]

In pregnancy, serum uric acid levels decrease in the early weeks, then increase due to fetal and placental uric acid production, and decrease again towards the end of pregnancy. However, in pregnancies complicated by hypertension, elevated uric acid levels are commonly found. [7,8]

The kidney is the major player in uric acid homeostasis. Uric acid induces oxidative stress, reduces endothelial nitric oxide availability, and activates plasma renin and intra-kidney angiotensin activity, leading to kidney vasoconstriction, ischaemia, and immune system activation causing persistent kidney vasoconstriction and salt-sensitive hypertension.[7]

Initial studies suggested that hyperuricaemia in hypertensive disorders of pregnancy may be due to inflammatory processes and altered renal clearance. However, several studies have shown links between disease severity, uric acid concentration, and foetal outcome.[7,8,10] There is also a growing body of evidence, that uric acid plays a role in the pathogenesis of hypertension in pregnancy, especially pre-eclampsia.[7-10] Furthermore, there is a suggestion that hyperuricaemia is at least as important as proteinuria for assessing fetal and maternal risks. However, its role as a biochemical marker remains disputed.[7-9]

Degree of proteinuria, blood pressure elevation, and gestational age at onset of hypertension have been used as markers of maternal disease severity associated with poor foetomaternal outcome.[11-13] Hypertensive disorders in pregnancy are associated with adverse maternal outcomes, including abruptio placentae, pulmonary edema, heart failure, renal failure, cerebrovascular accidents, disseminated intravascular coagulation, and death.[14,15] Adverse perinatal outcomes associated with hypertensive disorders in pregnancy include intrauterine growth restriction, prematurity, low birth weight, small for gestational age, stillbirth, and low Apgar scores.[6,14,15] The risk of these adverse outcomes is increased in all women with hypertension in the presence of elevated serum uric acid with/without proteinuria.[11,13,16-18] However, serious perinatal morbidity and mortality have also been observed in non-proteinuric gestational hypertension.[11,13,16,19] This suggests that the risk of adverse perinatal outcomes may be beyond elevated blood pressure and proteinuria. Several studies suggest that elevated maternal serum uric acid levels correlate with perinatal morbidity and serial estimation of uric acid may also allow appropriate timing of delivery of a fetus with an improved chance of survival.[2-5,11,14] There are, however, still conflicting reports about the relationship between maternal serum uric acid levels and perinatal outcomes and the critical value of uric acid associated with adverse perinatal outcomes.[20,21] In UMTH, there is no established critical value for maternal serum uric acid associated with these adverse perinatal outcomes in women with HDP. Since the determination of serum uric acid can be reliably done in our facility, this study will assess the association of maternal serum uric acid level with perinatal outcome in women with

hypertensive disorders of pregnancy who presented to the Labour ward in the University Maiduguri Teaching Hospital and determine the critical value of serum uric acid associated with the adverse perinatal outcomes observed.

Methods:

We conducted a hospital - based cross - sectional study to determine the relationship between maternal serum uric acid level and perinatal outcome among pregnant women with hypertensive disorders in pregnancy who delivered at UMTH and compare it with normotensive women who delivered at UMTH from 1st December 2010 to 30th April 2011.

The subjects of the study were recruited from the population of pregnant women with any hypertensive disorder in pregnancy who delivered in the Department of Obstetrics and Gynaecology of UMTH. The next two normotensive, non-proteinuric pregnant women matched for age group and parity admitted into the labour ward were recruited for comparison. Patients were recruited consecutively as they were admitted into the labour ward. Samples were taken at delivery. Mothers were observed until discharge from the hospital, and babies were observed for the first week of life and their outcomes were documented. No mother was lost to follow-up but 5 babies who were discharged from the SCBU on the 5th day of life were lost to follow-up.

All booked and unbooked pregnant women at a gestational age of 28 -40 weeks with any hypertensive disorder in pregnancy and who gave consent were included in the study.

Women with multiple pregnancies, fetal congenital malformations, postdate, HIV infection, diabetes mellitus, sickle cell disease, known history of renal disease, and non-consenting women were excluded from the study.

The sample size was obtained using the $n = z^2pq/d^2$ [22] using a prevalence of 9.8% (≈10%) from a similar study done in Ibadan, Nigeria giving a minimum sample size of 145 patients.[23] The standard normal deviation was set at a 1.96 confidence level, which corresponds to a 95% confidence level. The P-value was set at 0.05. Therefore 160 patients with a hypertensive disorder in pregnancy and 320 normotensive and nonproteinuric pregnant women who satisfied the inclusion criteria and gave their consent were recruited for the study.

All aspects of the study were reviewed by the Research and Ethics Committee of UMTH and approval was granted (ADM/TH/497/VOL 2). Informed consent was obtained from all subjects after the study was thoroughly explained to them, stating clearly that they could withdraw at will at any time without any consequences.

For the diagnosis and classification of hypertensive disorders in pregnancy, the Davey and MacGillivray classification adopted by the ISSHP was used.[24] Samples from 2 patients in the hypertensive group were lost to a laboratory accident and 1 sample haemolysed before separation. Therefore, samples from 157 hypertensive women and 320 normotensive women were analysed.

Socio-demographic variables and clinical characteristics such as age, parity, admitting blood pressure, degree of proteinuria, mode of delivery, gestational age at delivery, birth weight, Apgar score, and admission to special care baby unit were noted by the researchers and trained assistants on a proforma. The babies admitted into the special care baby unit were followed up until discharge and the outcomes were recorded. Preterm birth was defined as birth before 37 completed weeks of gestation, low birth weight was defined as a birth weight of less than 2500g, birth asphyxia was defined as an APGAR score of <7 at the 5th minute of life, and fetal distress as fetal heart rate >160 or <110 beats per minute. [25] Intrauterine Growth Restriction (IUGR) was fetal weight that is below the 10th percentile for gestational age.[26]

The assay of maternal serum uric acid was done in the chemical pathology laboratory of U.M.T.H. using the uricase colorimetric method and a serum uric acid level of ≤312umol/l was considered normal. [27] Qualitative urinary protein estimation was done using a dipstick.

Data obtained were recorded on a proforma and analysed using the statistical package for social science (SPSS.16 Inc, Illinois.). Percentage, means, standard deviation, and critical value of maternal serum uric acid were calculated. The two groups were compared and risk was estimated using an odds ratio, Chi-square, and t-test where appropriate. The level of significance was set at a 95% confidence interval and the P value was set at <0.05. Tables and Figures were used to illustrate patterns in the variables. The critical value of serum uric acid was determined and receiver-operating characteristic (ROC) analyses for predicting adverse fetal outcomes using the critical maternal serum uric acid level were done after adjusting for confounding variables such as abruptio placentae, HELLP syndrome, and gestational age at delivery.

Results

The mean age of patients in the hypertensive and normotensive groups were similar (24.3±7.2 vs 24.9±4.7 years, χ^2 =1.164, P 0.245), as shown in Table I. While the majority 119 (74.4%) of the hypertensive women were unbooked, a majority 235 (73.4%) of the normotensive women were booked (χ^2 =1.113, P <0.0001). More than half 85(53.1%) of the hypertensive group had no formal education compared with 93 (29.1%) of the normotensive group. While 97(30.3%) of the normotensive group had tertiary education, only 18(11.2%) of the hypertensive group attained that level of education (χ^2 =35.396, P <0.0001). This is illustrated in Table 1.

Table 2 shows the mode of delivery of the study population. Spontaneous vaginal delivery was achieved in 274(85.6%) of the normotensive group and 45(28.1%) of hypertensive group. Two-fifth 36(22.5%) of the hypertensive group were delivered by caesarean section compared to one out of ten 33(10.3%) of the normotensive group, P 0.08. Instrumental delivery was done in 11 (6.9%) and 8 (2.5%) of hypertensive and normotensive groups respectively. The hypertensive group delivered earlier (37.1 vs 38.6 weeks; P <0.001) and their babies weighed less (2.45 vs 3.15kg; P <0.001).

Women with Hypertensive disorders in pregnancy had more adverse perinatal outcomes and all were statistically significant. Prematurity (χ^2 =47.000, P<0.001), low birth weight (χ^2 =131.589, P<0.001), intrauterine growth restriction (χ^2=103.965, P<0.001), birth asphyxia(χ^2 =50.581, P<0.001), fetal distress in labour (χ^2 =47.548, P<0.001), admission to special baby care unit (χ^2 =49.567, P<0.001), intrauterine fetal death (χ^2 =26.097, P <0.001) and early neonatal death (χ^2 =57.272, P<0.001). This is shown in Table 3.

Table 4 shows the relationship between hyperuricaemia and perinatal outcome in women with hypertensive disorders in pregnancy (n=157). Hyperuricaemia was significantly associated with the following adverse perinatal outcomes; Low birth weight; OR (95%CI) 20.937(11.014-39.800), P <0.0001), prematurity OR (95%CI) 4.675(2.987-7.623), P <0.0001, Birth asphyxia; OR (95%CI) 18.057 (6.316-51.625), P <0.001), Fetal distress; OR (95%CI) 30.334 (7.170-128.328), P <0.001), Intrauterine growth restriction; OR (95%CI) 4.765 (2.978-7.623), P <0.001), Intrauterine fetal death; OR (95%CI); 6.093 (2.817-13.176), P <0.001), Admission to special care baby unit; OR (95%CI) 11.651 (5.102-26.609), P <0.001) and early neonatal death: OR (95%CI);14.706(3.135-71.429), P <0.001). The mean serum uric acid value was 487.96±148.48mmol/l and the critical value of maternal serum uric acid was found to be 455umol/l. The critical value of maternal serum uric acid of 455umol/l was significantly associated with low birth weight (OR (95%CI) 4.556(2.322-8.940), P <0.001), intrauterine growth restriction (OR (95%CI) 5.067(2.298-11.172), P <0.001), intrauterine fetal death (OR (95%CI) 1.836(1.361-2.937), P 0.001) and admission to special care baby unit (OR (95%CI) 2.637(1.133-6.138), P 0.017), among the hypertensive group after regression analysis for confounding variables like antepartum haemorrhage, HELLP (haemolysis, elevated liver enzymes, low platelet) syndrome, and Pulmonary oedema. This is shown in table 5. Receiver-operating characteristic (ROC) analyses for predicting these adverse fetal outcomes using maternal serum uric acid levels are as shown in Figure 1 and Table 6. The area under the receiver operating characteristics curve (AUC) was significant for low birth weight (0.700, P <0.001) and intrauterine growth restriction (0.706, P <0.001) with a sensitivity of 67.6% and 70.8% respectively.

Table 1: Characteristics of the study population

	Hypertensive group	Normotensive group	χ^2 /t-test *	P value
Age (mean±SD).	24.3±7.2	24.9±4.7	1.164*	0.245
Parity(mean±SD).	2.1 ± 2.8	2.1±2.4	0.123*	0.902
Booking status:	N (%)	N(%)		
Booked	36(22.5)	235(73.4)		
Unbooked	119(74.4)	81(25.3)		
Referred	5(3.1)	4(1.3)	1.113	<0.001
Total	160(100)	320(100)		
Educational status				
None	85(53.1)	93(29.1)		
Primary	19(11.9)	40(12.5)		
Secondary	38(23.8)	90(28.1)		
Tertiary	18(11.2)	97(30.3)	35.396	<0.001
Total	160(100)	320(100)		

Table 2: Regression analysis for Mode of delivery of the study population

	Hypertensive N (%)	Normotensive N (%)	OR (95CI)/ t-test*	P-value
Induction of labour and Vaginal delivery	67(41.9)	5(1.6)	0.022(0.008-0.055)	<0.001
Spontaneous vaginal delivery	45(28.1)	274(85.6)	12.468(7.942-19.573)	<0.001
Caesarean section	36(22.5)	33(10.3)	0.489(0.288-0.831)	0.008
Instrumental delivery	11(6.9)	8 (2.5)	0.153(0.049 0.483)	0.001
Destructive operation	1(0.6)	-	22.40(2.055-244.217)	0.011
Total	160(100)	320(100)		
Gestational age delivery(weeks).	37.1±2.2	38.6±0.9	9.794 *	<0.001
Birth weight(kg).	2.45±0.7	3.15±0.5	12.517*	<0.001

Table 3: The Perinatal outcome of the study population

Perinatal Outcome		Hypertensive	Normotensive	Chi-Square	P - Value
Prematurity					
	Yes	60	32		
	No	100	288	47.000	<0.001
Total		160	320		
Low birth weight					
	Yes	77	11		
	No	83	309	131.589	<0.001
Total		160	320		
Birth Asphyxia					
	Yes	36	4		
	No	124	316	50.581	<0.001
Total		160	320		
Fetal Distress					
	Yes	31	2		
	No	129	318	47.548	<0.001
Total		160	320		
Intrauterine growth restriction					
	Yes	57	31		
	No	103	289	47.000	<0.001
Total		160	320		
Special care baby unit admission					
	Yes	40	7		
	No	120	313	49.567	<0.001
Total		160	320		
Intrauterine fetal death					
	Yes	29	8		
	No	131	312	26.097	<0.001
Total		160	320		
Early neonatal death					
	Yes	51	11		
	No	109	309	57.272	<0.001
Total		160	320		

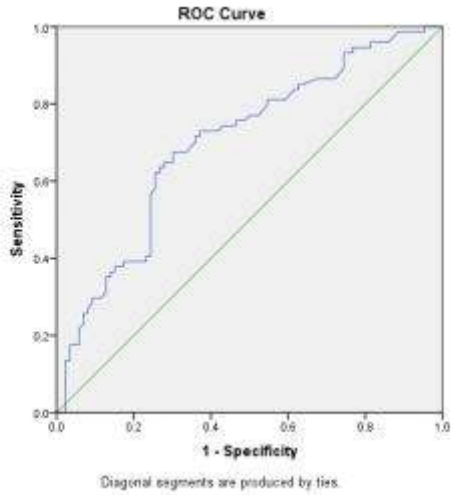
Table 4: The relationship between maternal serum uric acid level(>312umol/l) and perinatal outcome.

Perinatal Outcome	Odd ratio (OR)	95% Confidence interval (95CI)	P – Value
Prematurity	4.675	2.987-7.623	<0.001
Low Birth Weight	20.937	11.014-39.800	<0.001
Birth Asphyxia	18.057	6.316-51.625	<0.001
Foetal Distress	30.334	7.170-128.328	<0.001
Intrauterine growth restriction	4.765	2.978-7.623	<0.001
Admission to special care baby unit	11.651	5.102-26.609	<0.001
Intrauterine fetal death	6.093	2.817-13.176	<0.001
Early neonatal death	14.706	3.135-71.429	<0.001

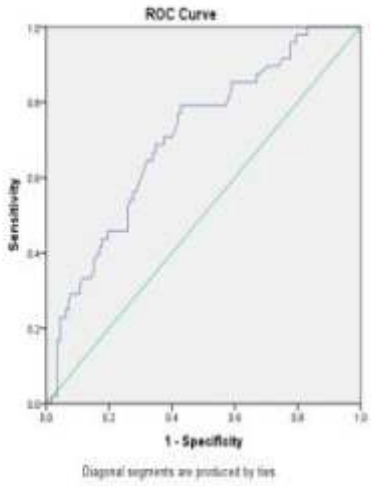
Table 5: Regression analysis for the relationship between the uric acid critical value of 455umol/l and perinatal outcome (n=157).

Perinatal Outcome	Odd ratio (OR)	95% Confidence interval	P – Value
Prematurity	0.642	0.309- 1.512	0.229
Low Birth Weight	4.556	2.322-8.940	<0.001*
Birth Asphyxia	1.694	0.728-3.944	0.153
Foetal Distress	1.156	0.519-2.573	0.441
Intrauterine growth restriction	5.067	2.298-11.172	<0.001*
Admission to special care baby unit	2.637	1.133-6.138	0.017*
Intrauterine foetal death	1.836	1.361-2.937	0.001*
Early neonatal death	0.868	0.420- 1.794	0.422

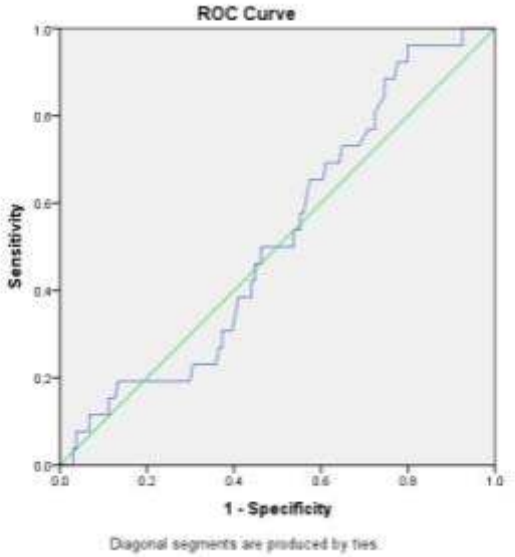
Figure 1: Receiver-operating characteristic (ROC) analyses for predicting adverse fetal outcomes using maternal serum uric acid level ≥455umol/l



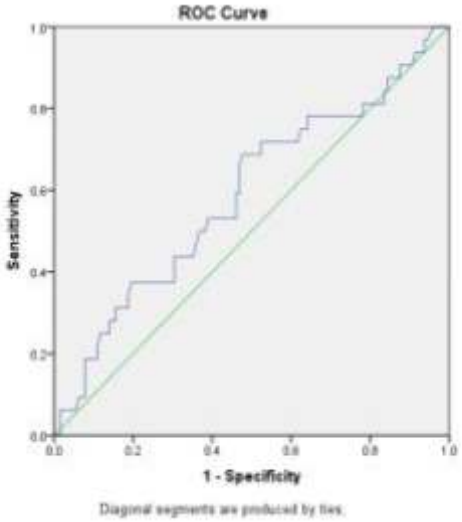
A. Lowbirth weight



B. Intrauterine growth restriction



A. Intrauterine fetal death



B. Admission to special baby care unit

Table 6: Area under the receiver operating characteristics curve (AUC), Sensitivity, and specificity for perinatal outcomes using Uric Acid Critical value of 455umol/l

	AUC	Sensitivity	Specificity	P value
Low birth weight	0.700	67.6%	33.7%	0.000
Intrauterine growth restriction	0.706	70.8%	40.2%	0.000
Intrauterine fetal death	0.521	50%	49.3%	0.732
Admission to special care baby unit	0.588	59.4%	46.9%	0.125

Discussion

The mean age of the hypertensive group in this study is 24.29 years, which is younger than the 27.3 years in a study in Sagamu, Nigeria[1,] and the 28.8 years reported in Benin, Nigeria.[10] This is likely because marriage is typically performed at a younger age in the country's northern region.[6]

Whereas 50.8 percent of the normotensive group had at least a secondary school education and 73.4% were booked, the majority of the hypertensive group were unbooked (74.4%) and had no formal education (53.1%). This is consistent with Sagamu's findings.[1] This result emphasizes the need for a redoubled effort to educate girls and highlights the connection between education and health resource use.

Uric acid-induced maternal oxidative stress, excitation of placental vascular endothelium with reduced endothelial nitric oxide availability, and upregulation of the inflammatory response lead to a dysfunctional placenta, which hinders fetal development. This typically presents as intrauterine growth restriction, oligohydramnios, intrauterine fetal death, and intrapartum fetal distress or demise.[28] The obstetrician usually resorts to Caesarean delivery to improve neonatal outcomes. The cesarean section rate of 22.5% in the hypertensive group in this study is therefore not surprising. Although several studies have shown an increase in the caesarean delivery rate in pregnant women with hypertensive disorders[1,2,10], there was no statistically significant difference in the Caesarean delivery rate in our study after ruling out confounding variables. This may be because the other studies did not rule out confounding variables as was done in our study.

This study shows that adverse perinatal outcomes such as intrauterine growth restriction, low Apgar scores, prematurity, low birth weight, need for admission to neonatal intensive care unit, and neonatal death are significantly increased in women with hypertensive disorders in pregnancy compared with the normotensive women. This collaborates with findings from several studies done within and outside Nigeria.[6,12,13]

The association between elevated serum uric acid and hypertensive disorders of pregnancy has long been known. While several studies have correlated the rise in uric acid with the severity of perinatal morbidity and mortality[1,3,6,14,15] some reported poor correlation.[20,21,29] Uric acid has been shown to inhibit endothelial cell proliferation and it also passes freely into the placenta. A rise in uric acid could therefore lead to an inhibition of angiogenesis and kidney growth with a reduction in nephron number in the foetus.[17,18] It is therefore not surprising that adverse foetal manifestations in the form of oligohydramnios,

intrauterine growth restriction, intrauterine foetal death, intrapartum foetal distress/demise, and birth asphyxia have been observed in the presence of hyperuricaemia.[2,4,14,29] The positive correlation between hyperuricaemia and perinatal outcome such as low birth weight, prematurity, birth asphyxia, foetal distress, intrauterine growth restriction, admission into special care baby unit, intrauterine foetal demise, and neonatal death in this study further supports the finding in the aforementioned studies. This is also in keeping with findings in Dhaka, Bangladesh where the risk of intrauterine foetal death and prematurity is 5.3 times higher in the hyperuricaemic group and the risk of intrauterine growth restriction is 6 times higher in the hyperuricaemic group.[2] The mean gestational age at delivery of 37.1 weeks and 38.6 weeks in hypertensive and normotensive women respectively is similar to 36.6 weeks and 38.0 weeks in hypertensive and normotensive women respectively reported in Ibadan, Nigeria.[23] The mean birth weight of 2.45kg in the hypertensive group and 3.15kg in the normotensive group in this study is lower than 2.6kg and 3.4kg reported in Dhaka, Benin, and Ibadan [2,20,23.]

To further buttress the negative effect of hyperuricaemia on perinatal outcomes, some studies have reported poor perinatal outcomes in women with hyperuricaemia irrespective of the severity of hypertension and proteinuria.[2,3] Furthermore, hypertension without hyperuricaemia has been shown to have a good foetal prognosis.[2,3,11,13]

In this study, we determined the critical value of maternal serum uric acid associated with the adverse perinatal outcomes observed. The critical value was 455umol/l which is higher than the 393umol/l and 309umol/l observed in Vietnam and Italy respectively.[30,31] This critical value of 455umol/l was significantly associated with low birth weight, intrauterine growth restriction, intrauterine foetal death, and admission to special care baby unit among the hypertensive group after ruling out confounding variables like antepartum haemorrhage, HELLP (haemolysis, elevated liver enzymes, low platelet) syndrome, and Pulmonary oedema. The study done in Vietnam showed a correlation between the cutoff values with Prematurity, intrauterine growth restriction, birth asphyxia, and neonatal death.[30] Our study did not show a significant correlation between the cutoff value of uric acid with birth asphyxia and neonatal death. This may be due to the larger sample size (205) in the Vietnam study compared to the 160 used in our study. The study done in Italy also used a similar sample size(163) to our study and did not find a significant correlation with neonatal death at the cutoff value of 309umol/l.[31] The area under the receiver operating characteristics curve(AUC) was significant for low birth weight (0.700, P <0.001)and intrauterine growth restriction(0.706, P <0.001) with a sensitivity of 67.6% and 70.8% respectively which is similar to the report from Vietnam, however, our study showed lower specificities.[30]

Our report has some limitations. First, the current study is limited to a one-time estimation of serum uric acid levels in women admitted for delivery on account of a hypertensive disorder in pregnancy. Secondly, this study was done in a tertiary hospital that serves as a referral center. This was reflected by the majority of the hypertensive group being unbooked. Lack of antenatal care may have contributed to the adverse perinatal outcomes observed in the study.

We believe a multi-center prospective cohort study with serial measurements of serum uric acid levels across trimesters will fill in the gaps in our study.

Conclusion

The findings of this study suggest that hyperuricaemia at a critical value of 455umol/l correlates with low birth weight, intrauterine growth restriction, intrauterine foetal death, and admission to the special care baby unit. Elevated uric acid at a critical value of 455umol/l and above in women with hypertensive disorders in pregnancy can therefore be used to identify a group of women at increased risk of adverse perinatal outcomes, who will require more intense surveillance and time delivery to avert further perinatal morbidity and mortality. Therefore, the estimation of maternal serum uric acid should no longer be viewed as just part of the routine work-up of women with a hypertensive disorder in pregnancy.

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Conflict of interest: None

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