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Original Research Article

Correlation between serum uric acid levels and outcomes of pre-eclampsia in Abakaliki, South-east, Nigeria

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ABSTRACT

Background: Pre-eclampsia is a leading cause of fetomaternal and perinatal morbidity and mortality. The role of serum uric acid (SUA) in determining the complications of preeclampsia has been controversial. This study compared mean SUA levels between severe pre-eclamptics and normotensive women at term and ascertained its correlation with outcomes of preeclampsia; as well as determined if there is a threshold value of SUA level beyond which fetomaternal complications occur.
Methods: A case-controlled study where 80 severe pre-eclamptics at term and 80 normotensive women matched for gestational age were recruited. Blood samples were collected from them for assay of SUA levels and they were followed till delivery. The fetomaternal outcomes and the corresponding SUA levels at diagnosis were documented and variables statistically analyzed. A receiver operating characteristic curve was used to determine the cut-off value of SUA beyond which adverse fetomateral complications are likely to occur in pre-eclampsia.
Results: The mean SUA level in severe pre-eclamptics (0.283±0.09 mmol/l) was not significantly higher than that of normotensive women (0.263±0.09 mmol/l, p=0.13). There was a weak positive correlation between the SUA levels and fetomaternal outcomes [maternal (r=0.102, p=0.236) and fetal (r=0.096, p=0.226)]. The study was unable to identify the threshold SUA level at which adverse fetomaternal outcomes occur as the values of SUA were closely related.
Conclusions: SUA levels of pre-eclamptics and normotensive women did not show significant difference and correlated weakly with fetomaternal outcomes and are therefore poor predictor of fetomaternal outcomes in pre-eclampsia.

Keywords: Correlation, Serum uric acid, Pre-eclampsia, Outcomes

INTRODUCTION

Pre-eclampsia is a pregnancy specific multi-systemic disease.[1] It is characterized by onset of elevated blood pressure and proteinuria after 20 weeks of gestation.[2] This may be diagnosed during pregnancy, labour or puerperium in a previously normotensive and aproteinuric woman.[3] The disorder globally affects approximately 5-7% of pregnancies.[4] Worldwide, pre-eclampsia increases perinatal mortality by five folds and approximately 50,000 women die due to pre-eclampsia annually.[5] The World Health Organization (WHO) approximates that 60,000 maternal deaths occur yearly from hypertensive diseases in pregnancy and over 98% of these deaths occur in developing countries.[6] Also in these developing countries, a woman is seven times more likely to develop pre-eclampsia than in developed countries; and about 10-25% of these cases will result in maternal death.[7] The incidence of pre-eclampsia varies with the diagnostic criteria and the population being studied.[8] In Sokoto, Northern Nigeria, 6% prevalence of pre-eclampsia was obtained, while 40% of maternal death in Northern Nigeria was attributed to

pre-eclampsia/eclampsia.[4,9] Calabar and Bayelsa, in south-south Nigeria, recorded prevalence of 1.2% and 5.56% respectively.[10,11] In Abakaliki, southeast Nigeria, prevalence of 4.7% was obtained for hypertensive disorders and 0.99% for severe pre-eclampsia.[12,13]

The aetiology of pre-eclampsia is largely unknown.[14] Central to its pathology is the abnormal placentation.[15] There is a complete or partial failure of trophoblastic invasion of the myometrial and the spiral arteries resulting in muscular vasculature of the placental bed that is responsive to vasoactive substances.[14] This leads to the release of soluble factors from the ischemic placenta to the maternal plasma leading to endothelial dysfunction which characterizes this disease.[16] Risk factors include: primigravidity, extremes of maternal age, black race, chronic hypertension, renal disease, gestational diabetes among others. Management principle continuously balances the risk–benefit ratio of induced preterm delivery and fetomaternal complications.[3]

Predicting and monitoring pre-eclampsia has been of great concern and a lot of variables have been used without any consensus. Uric acid ($C_5H_4N_4O_3$) is one of such variables. It is a product of purine degradation catalyzed by the enzyme xanthine oxidase and is a normal component of urine.[4] It is one of the most sensitive indicators of disease severity in pregnancy induced hypertensive disorders and may be of great help in monitoring the course of disease process.[17] Elevated uric acid is thought to be associated with both cardiovascular disease events and risk factors such as hypertension, metabolic syndrome, chronic kidney disease, obesity and diabetes in non-pregnant adults.[18] Uric acid stimulates monocytes to produce pro-inflammatory cytokines IL1β, IL-6, and tumor necrosis factor- α (TNF-α).[4] It also promotes endothelial dysfunction which may lead to hypertension, vascular disease and renal disease.[4]

Pregnancy involves remarkable orchestration of physiologic changes.[19] In early pregnancy, SUA fall to <3 mg/dl due to the uricosonic effects of estrogen and increase in renal blood flow; it increases during the third trimester, reaching levels of 4 to 5 mg/dl by term.[20] Reference value of 3.4-7.0 mg/dl and 2.4-5.0 mg/dl are seen in males and females respectively.[21] Increased production of urate is primarily due to increased activity of enzymes involved in purine breakdown such as adenine phosphoriboxyltransferase. Secondary factors include myeloproliferative disease, lymphoproliferative disease, haemolytic anaemia, drugs such as salicylate, diuretics, pyrazinamide, ethambutol, nicotinamide, ethanol and cytotoxic drugs.

In pre-eclampsia, vasospasm and glomerular endotheliosis lead to reduction in renal blood flow. This first involves impairment of tubular function and reduction in uric acid clearance with development of hyperuricemia. Later glomerular filteration becomes impaired and proteinuria develops. Rise in plasma urate is therefore an early sign in the evolution of pre-eclampsia and is cited as a better predictor of fetal risk than blood pressure.[5] It identifies women in increased risk of adverse maternal and particularly fetal outcome.[22] The association between raised SUA and pre-eclampsia was reported in 1917 by Siemon.[23] He attributed elevated SUA to be due to reduced renal clearance following a reduced GFR. In 1990, Fay described that increased breakdown of cells in the placenta leads to over production of uric acid in pre-eclamptics and this elevated level reflects the degree of placenta cell destruction as well as severity of the disease.[24,25] On the other hand, decreased uric acid excretion may be secondary to decreased tubular secretion or enhanced tubular reabsorption.

Previous related studies have shown no consensus on a threshold value of SUA level above which adverse fetomaternal outcomes are likely to occur and the accuracy of the marker in predicting pregnancy outcomes. For some researchers there is a positive correlation between elevated maternal SUA and adverse fetomaternal outcomes with an identifiable threshold value for SUA above which adverse outcomes result.[15,26-28]

On the other hand, other studies have contradictory results on the accuracy of SUA with unsatisfactory sensitivity and/or specificity.[15,28] Uric acid threshold values for occurrence of complications were obtained in studies conducted in European countries.[15,26] No study has evaluated these thresholds among women of Igbo extraction in Abakaliki, Nigeria. A study of this group is necessary to validate the results of earlier studies.

METHODS

This was a case control study where consenting participants from two equal groups (severe pre-eclamptics and control) were recruited at term (37^{+0} – 41^{+6} weeks gestation) from the 01 May 2019 to 30 November 2019. The control group comprised of normotensive women at term and matched for gestational age. Blood samples were collected from each group for assay of the SUA levels and they were followed up till delivery. The accuracy of SUA levels in predicting pregnancy outcomes among the pre-eclamptic patients admitted and managed at the Alex-Ekwueme Federal University Teaching Hospital Abakaliki (AE-FUTHA) and St. Patrick's Mile 4 Hospital Abakaliki was determined.

Study background

Ebonyi state, located in the South Eastern Nigeria, was created on 01 October 1996. Abakaliki is the state capital and its population is about 438,700 and consists of different ethnic groups, predominantly Igbo. The climate is characterized by high uniform temperature, moderate to heavy seasonal rainfall and high relative humidity. Habitants are mainly farmers, traders and civil servants. They are predominantly Christians.

AE-FUTHA was established in December 2011 by upgrading the then Federal Medical Centre to a teaching hospital and merging it with the then Ebonyi State University Teaching Hospital. The department of Obstetrics and Gynaecology of the hospital runs antenatal clinics managed by consultants and resident doctors with trained nurses/midwives. The hospital serves as a major referral center for Ebonyi and surrounding states. St. Patrick's Mile 4 Hospital is one of the mission hospitals in the state. It was established in 1964 and it currently provides maternal and child health services. It is managed by Consultant Obstetricians, resident doctors who are routinely posted from AE-FUTHA, medical officers and nurses.

Study population

This included severe pre-eclamptic patients at gestational ages of ≥37 weeks who were stabilized for immediate delivery and normotensive women with normal pregnancies at term and matched for gestational age. Women with the following conditions were excluded: current and previous history of renal disease, chronic hypertension, diabetes mellitus in pregnancy, multiple pregnancy, patients with mal-presenting fetuses and eclamptic patients, patients who had premature rupture of membranes (PROM) or chorioamnionitis, with history of recent use of salicylates or anti-seizure medications, history of medical/systemic conditions that affect pregnancy outcome such as sickle cell disease, systemic lupus erythematosus (SLE), tuberculosis, human immune deficiency virus (HIV), pneumonia, sarcoidosis, thyroid disease of pregnancy and administration of corticosteroids within the last 7 days.

Sample size determination

Sample size was calculated using formula for case control study with variable outcomes as documented by a Jaykaran et al.[29]

$$N = \frac{r + 1(SD)^2(Z_\beta + Z\alpha_{/2})^2}{rd^2}$$

Where N is the minimum sample size at 95% confidence level, r is the ratio of case to control=1:1, SD is the standard deviation from previous study by Nwankwo et al=1.1, d is the expected mean difference between case and control from related study=0.514, Z_β is the standard normal variant for power of 90% set at 0.84, and $Z_{\alpha/2}$ is the standard normal variant for level of significance set at 1.96.[30]

$$N = \frac{1 + 1(1.1)^2(0.84 + 1.96)^2}{1 \times (0.514)^2}$$

$$N = \frac{2 \times [1.21(2.8)^2]}{0.2642}$$

$$N = \frac{2 \times (1.21 \times 7.84)}{0.2642}$$

$$N = \frac{2 \times 9.4864}{0.2642}$$

$$N = 2 \times 35.91 = 71.8 \sim 72$$

The power of the study was increased by adding 10% attrition rate and this resulted to a final minimum sample size of 80 per arm. Thus, a total of 160 participants were involved.

Diagnosis

Diagnosis of severe pre-eclampsia was made following a manual blood pressure measurement (using stethoscope, blood pressure cuff and mercury column sphygmomanometer [accosure®]) and a dipstick urinalysis (medi-test®).[31] Repeat measurements were done immediately to ensure accuracy using Korokoff 5. Severe pre-eclamptics were selected using a systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg on a single reading with proteinuria ≥2+.

Study procedure/participant recruitment

Once a woman met the criteria for inclusion into the study, the objective and methodology were explained to her by the researcher. She was made to appreciate her autonomy throughout the course of the study. She then signed a consent form to partake in the study. The recruitments were done in antenatal clinics, antenatal wards, labour wards and accident/emergency units of the hospitals. Blood samples were collected from all patients by venopuncture observing aseptic protocols. These were done before the administration of medications as the patients were managed according to the departmental protocol.[32]

Collection of blood samples

The researchers observed asepsis by wearing sterile gloves and covering their mouths and noses with disposable face masks. New sterile disposable 10ml syringe, needle and lithium heparinized bottle were used for each pregnant woman. For each patient, a spotted area on the cubital fossa or the dorsum of the hand for the veno-puncture was located and swabbed using cotton wool soaked in 70% alcohol (methylated spirit) after applying tourniquet above the site. With the syringe, 8 ml of blood was collected and emptied into the lithium heparin bottle. The tourniquet was released and pressure applied at the puncture site with dry cotton wool to achieve homeostasis. The bottle was well covered and then gently rocked for about 3 seconds to ensure proper mix up of the blood in the tube with the anticoagulant. The specimens were taken immediately to the laboratory for assay of the serum uric acid levels. Medications were administered to the patients and the

eventual mode of deliveries were documented in a proforma and used for follow up.

Processing

Samples from the two study centers were analyzed in the same laboratory. All the samples arrived at the laboratory within 1 hour of its collection. In the laboratory, the blood specimens were spun in a centrifuge at 3500 rpm for 10 minutes to separate the serum from the blood cells. The serum components were decanted into a new fresh bottle without any anticoagulant and refrigerated under -20^{0}C for preservation. The first batch of specimen analysis was done 4 months into the study with the already collected specimens. The specimen collection continued and was completed in the next 3 months during which the second batch of specimen analysis was done.

Estimation/analysis of serum uric acid levels

This was done by enzymatic colorimetric method using 721 visible spectrophotometer at the wave length of 520 nm. Here, uricase was added to the specimen. This transformed the uric acid in the specimen into allantion and hydrogen peroxide. Then 4-aminoantipyrine and 3-hydroxyl- 2, 4, 6- triiodobenzoic acid were added to the mixture. These two compounds in the presence of peroxidase reacted with hydrogen peroxide to produce a coloured complex whose intensity, measured in optical density (OD), was directly proportional to the uric acid concentration in the sample. This was determined with the use of a spectrophotometer.

Follow-up

All the patients were followed up. The pre-eclamptics were admitted into the wards and plans on the best mode of delivery were individualized. Decisions on use of anti-hypertensives and magnesium sulphate were taken according to departmental protocols. Fetomateral vital signs were monitored hourly till delivery.

For parturients allowed for vaginal deliveries, continuous electronic fetal heart monitoring was used in labour while the maternal vital signs were monitored every 30 minutes. The labour events were managed actively with partograph and deliveries were conducted by resident doctors on duty. For the participants (case or control) delivered by cesarean sections, surgeries were done by consultants and senior registrars and the indications for the surgeries were well documented. Neonatologists were present during the deliveries irrespective of the route of delivery for prompt neonatal assessment/ resuscitation.

The APGAR scores at the 1st and 5th minutes of delivery were noted. Records on neonatal intensive care unit admissions, the birth weight, seizures, stillbirths and neonatal deaths were taken and documented by the researcher.

Outcome measures

Various parameters for maternal and fetal outcomes were documented. The primary outcome measure was the mean serum uric acid level in the patients while the secondary outcome measures were as outlined below.

Fetal

APGAR scores at 1st and 5th minutes, birth weights, neonatal intensive care unit (NICU) admissions, stillbirths, seizures and neonatal death.

Maternal

Blood pressure values, maternal complications such as abruptio placentae, eclampsia, postpartum hemorrhage, disseminated intravascular coagulation, hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome, oligouria, intensive care admissions and maternal death.

Statistical analysis

The generated data were analyzed using the IBM statistical package for the social sciences (SPSS) software (version 20, Chicago II, USA). Categorical variables were represented as frequencies and percentages while continuous variables were represented with mean±SD. Comparison between categorical variables were evaluated using Chi-square/Fisher's exact test while comparison between continuous variables were done using students t-test.

Binary logistic regression was used to test for association between SUA levels and adverse pregnancy outcomes. Adjusted odds ratio at 95% confidence interval was calculated for other confounding variables. A p value of <0.05 was considered significant. A receiver operating characteristic (ROC) curve was used to determine the cut-off value (threshold) of uric acid using the best sensitivity and specificity values beyond which adverse fetomateral complications are likely to occur in pre-eclamptics.

RESULTS

Over the study duration of 8 months, 160 patients were recruited into the study. Eighty participants with severe pre-eclampsia at term were the case, and were matched for gestational age with 80 normotensive women also at term as controls.

Table 1 shows the frequency distributions of the socio-demograhic variables in the study groups. P values for maternal age, parity and gestational age are 0.985, 0.221 and 0.09 respectively. The mean age of participants was 29.1±5.47 years.

Table 2 shows the frequency distribution of various ranges of SUA levels for the study groups. The most frequent

range seen amongst the pre-eclamptic group was 0.20 mmol/l–0.24 mmol/l and this occurred in 22 cases. For the normotensive group, the most frequent range occurring in 24 cases was 0.25 mmol/l–0.29 mmol/l. The p value was 0.385 which was statistically insignificant. The mean SUA level for the pre-eclamptics (cases) 0.283±0.09 mmol/l was higher than that for the normotensives (controls) 0.263±0.09 mmol/l but it was not significantly higher (p=0.13).

Table 3 shows the comparison of the various maternal outcome measures for both arms of the study. With regards to route of delivery, 57 participants had vaginal delivery in the case group while 62 achieved vaginal delivery in the control arm. The difference in route of delivery was not statistically significant for both groups [p=0.24, OR=O.72, 95% CI (0.35-1.46)]. Abruptio-placentae were recorded in seven participants in the case group while only one participant in the control group had this complication. This difference was statistically significant (p=0.032, OR=7.58, 95% CI (1.12-174.0). Similarly, eclampsia was recorded in 8 participants in the case group while none was seen in the normotensives. This was also statistically significant (p=0.003, OR=2.11, 95% CI [1.79-2.49]).

Table 4 shows the comparison of the various fetal/ neonatal outcome measures for both arms of the study. For the cases and controls, 1st minute APGAR scores of <7 were recorded in 31 and 11 participants respectively (p=0.0002, OR=3.97, 95% CI [1.82-8.65]). This was statistically significant. Similarly, the 1st minute APGAR scores ≥7 showed a significant difference between the two study groups as this was recorded in 49 cases and 68 controls (p=0.001, OR=0.28, 95% CI [0.13-0.60]). The difference in the new born special care unit (NBSCU) admissions (p=0.004, OR=2.98[1.43-6.20]) and the occurrence of low birth weight (p=0.00007, OR=24.6 [3.20-118.9]) between the study groups were statistically significant; with the mean birth weight for the cases 2.94±0.72 kg being significantly lower than that for control 3.35±0.41 kg (p=0.0002).

Table 5 shows the binary logistic regression analysis for both maternal and fetal complications. The t-statistic, correlation coefficient and p values for the maternal complications were 1.19, 0.102 and 0.236 respectively while those for fetal complications were 1.22, 0.096 and 0.226 respectively. These were not statistically significant.

Figure 1 suggests that the sensitivity of SUA level in determining maternal complications is 67% (95% CI 38.6-68.0%). This suggests that SUA is not a very good predictor of maternal outcomes judging by its sensitivity of 67%.

Figure 2 suggests that the sensitivity of SUA level in determining fetal complications is 33% (95% CI 43.2-69.6%).

Table 1: Frequency distribution of participants' ages, parities and gestational ages.

Parameters	Case n=80 (%)	Control n=80 (%)	P value
Age (years)			
15–19	2 (2.5)	2 (2.5)	0.985
20–24	17 (21.3)	14 (17.5)	
25–29	26 (32.5)	25 (31.3)	
30–34	22 (27.5)	9 (11.3)	
35–39	11 (13.8)	15 (18.8)	
40–44	2 (2.5)	15 (18.8)	
Parity			
0	42 (52.5)	50 (62.5)	0.221
1–4	34 (42.5)	27 (33.8)	
≥5	4 (5.0)	3 (3.8)	
Gestational age (weeks)			
$37–38^{+6}$	51 (63.8)	40 (50.0)	0.09
$39–40^{+6}$	18 (22.5)	22 (27.5)	
$41–41^{+6}$	10 (12.5)	18 (22.5)	
≥42	1 (1.3)	0 (0)	

Table 2: Comparison of uric acid levels for the study groups.

Uric serum acid levels (mmol/l)	Case (n=80)	Control (n=80)	P value
<0.05	0	0	0.385
0.05–0.09	0	1	
0.10–0.14	2	2	
0.15–0.19	12	5	
0.20–0.24	22	20	

Continued.

Uric serum acid levels (mmol/l)	Case (n=80)	Control (n=80)	P value
0.25–0.29	21	24	
0.30–0.34	15	8	
0.35–0.39	1	13	
0.40–0.44	1	3	
0.45–0.49	2	3	
0.50–0.54	2	1	
0.55–0.59	0	0	
0.60–0.64	2	0	
Serum uric acid level (mmol/l) mean±SD	0.283±0.09	0.263±0.09	0.13

Table 3: Comparison of maternal outcomes.

Parameters	Case (n=80)	Control (n=80)	P value	AOR (95% C.I.)
Route of delivery				
Vaginal	57	62		
C-section	23	18	0.24	0.72(0.35 -1.46)
Abruptio -placentae	7	1	0.032	7.58(1.12–174.0)
Eclampsia	8	0	0.003	2.11 (1.79–2.49)
PPH	3	1	0.31	0.4 (0.31–30.24)
HELLP syndrome	2	0	0.25	0.22 (0.15–2.37)
ICU admission	15	1	0.0001	18.2(2.35–141.71)
Death	1	0	0.5	0.89 (0.70–2.01)

Table 4: Comparison of fetal/neonatal outcomes.

Parameters	Case (n=80)	Control (n=80)	P value	AOR (95% C.I)
APGAR score (1st minute)				
<7	31	11	0.0002	3.92 (1.82-8.65)
≥7	49	68	0.001	0.28 (0.13-0.60)
APGAR score (5th minute)				
<7	23	3	0.00006	10.35(2.97-36.18)
≥7	57	76	0.0002	0.13 (0.03-0.42)
NBSCU admission	31	14	0.004	2.98 (1.43-6.20)
LBW	19	1	0.00007	24.6 (3.20-118.9)
Seizures	5	3	0.72	1.71 (0.39-7.41)
Stillbirth	8	2	0.04	1.67 (1.17-2.37)
Neonatal death	2	0	0.49	0.24 (0.13-2.37)
Birth weight (kg) mean±SD	2.94±0.72	3.35±0.41	0.0002	N/A

Table 5: Binary logistic regression/correlation analysis.

Parameter	R (correlation coefficient)	T statistics	P value
Maternal complications			
Acute pulmonary edema			0.129
PPH			0.502
Eclampsia			0.634
ICU admission			0.596
Maternal death			0.939
Abruptio placentae			0.582
Fetal complications			
Stillbirth		2.1834	0.141
Neonatal seizures		0.524	0.470
Neonatal death		1.3199	0.252
NBSCU admission		0.0118	0.913
LBW		1.496	0.223

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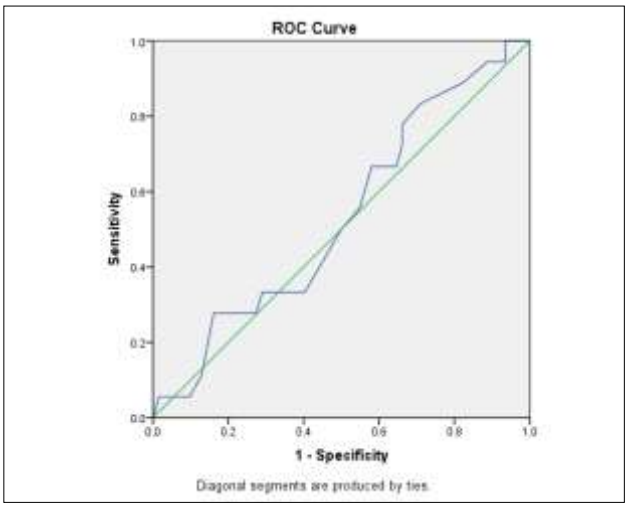

Figure 1: The receiver operating curve for maternal complications.
AUC: Asytomatic sig—0.67 (95% CI 0.386–0.680)

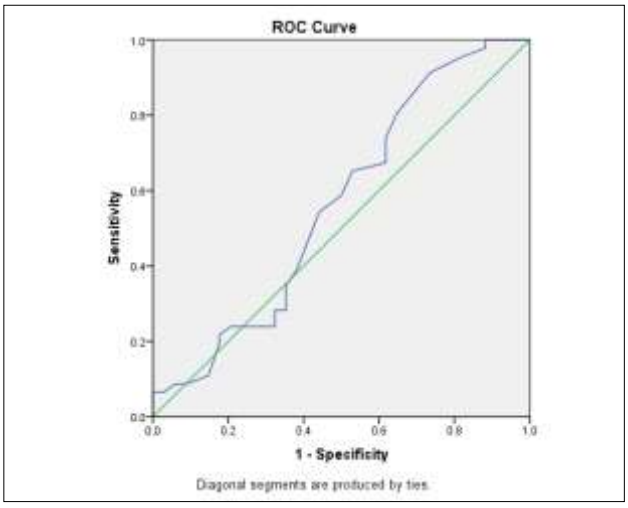

Figure 2: The receiver operating curve for fetal/neonatal complications.
AUC: Asytomatic sig—0.330 (95% CI 0.432 – 0.696)

DISCUSSION

The sociodemographic parameters in both arms of this study were similar giving credence to the methodology. This also gives credence to the results of the study as there were unlikely to be affected by confounded variables. The above result also validates the matching process for the two arms of the study.

The mean SUA level for the pre-eclamptic group was 0.28±0.09 mmol/l (approximately 5.09 mg/dl) compared to 0.26±0.09 mmol/l (approximately 4.73 mg/dl) in the normotensive group. This difference was not statistically significant. The two means approximate each other and as such it was not surprising that any difference noted was not statistically significant. This might imply that elevated SUA may not be a good marker for the severity of pre-eclampsia. The finding above was similar with those of the studies done by Mangareeka et al and Weerasekera et al which also found no significant difference in the mean SUA levels of the pre-eclamptic and normotensive women.[33,34] The mean SUA levels obtained by Manjareeka et al for pre-eclamptics and controls were 5.29±0.89 mg/dl and 3.86±0.9 mg/dl respectively.[33] These values were comparable to those obtained in this study. As was done in this study, Manjareeka et al also used the enzymatic colometeric method of uric acid estimation and participants were recruited at the third trimester only.[33] These similarities in methodology may have led to the identical results obtained.

The route of delivery was comparable for both arms of the study. It can be deduced from the foregoing that the diagnosis of pre-eclampsia on its own may not be an indication for caesarean delivery unless the chances of vaginal delivery are remote. Moreover pre-eclamptics/eclamptic patients are known to progress quickly in labour. This differs from that obtained by Tejal et al where a 3.4 fold risk of caesarean delivery was recorded among the pre-eclamptics.[35] This contrasting view may be due to difference in the population studied. Tejal's study evaluated only pre-eclamptics divided into 2 separate arms by a chosen level of SUA. The above observation was made on pre-eclamptics with SUA >6 mg/dl alone.

The occurrence of abruption-placentae, eclampsia and need for intensive care unit (ICU) admission all showed a statistically significant increase among the pre-eclamptic parturients compared with their normotensive counterparts. These findings agree with similar hospital based studies conducted by Niraula et al and Tejal et al which showed significant increase of these outcomes for pre-eclamptic parturients.[35,36] However, unlike in the study by Tejal et al, this study showed a difference that was not statistically significant in the occurrence of postpartum hemorrhage, HELLP syndrome and maternal death.[35] These contrasting findings may have resulted because the aforementioned study recruited various forms of pregnancy induced hypertensive conditions without the normotensive parturients as control.

The mean birth weight for the pre-eclamptic group was significally higher than the control (p=0.0002) and this agreed with a similar study done by Akter et al who found a marked reduction in birth weight for pre-eclamptics compared with the normotensive parturients.[37] In this study, the occurrence of low birth weight (LBW) was observed in 19 pre-eclamptic participants while only one case of LBW was recorded for the normotensive group. This was also statistically significant (p=0.00007). This further supports the fact that pre-eclampsia has a significant effect on the placenta bed with reduced blood supply to the fetus resulting in low birth weight. This agreed with the study by Tejal et al which demonstrated a four-fold increased risk of LBW among pre-eclamptic parturients.[35] These similar findings may have occurred

because these studies recruited about the same number of participants in their third trimester. Since the occurrence of low birth weight has been shown to be dependent on the time of onset of preeclampsia one may expect similar outcomes for participants recruited at same trimester. First minute Apgar scores of <7 occurred significantly more in pre-ecalmptics (p=0.0002) and this agrees with the study done by Tejal et al were about 6 fold increased risk of APGAR <7 was observed among pre-eclamptics.[35] Both studies were hospital based. Tejal et al however used a chosen SUA cut off level of 6 mg/dl above which these adverse outcomes are expected.[35] This is contrary to the findings in this study as there was no correlation of the adverse outcomes to SUA levels. This contrasting finding may be due to the fact that Tejal et al study used only pre-eclamptics without normotensive participants as control.[35]

Newborn special care admissions were significantly higher in pre-eclamptics (p=0.004). This suggests that neonates born to pre-eclamptic mothers are more likely to develop conditions that will warrant hospital admissions. Other perinatal outcomes such as neonatal seizures and neonatal death showed no significant difference between the study groups. These findings differ from that obtained by Akter et al which showed significant stillbirth rate among the pre-eclamptics.[37] This diverse view may not be unconnected with the difference in the protocol. Akter et al study made its observation only in pre-eclamptics with SUA ≥6 mg/dl while this study recruited several pre-eclamptics at term, irrespective of the SUA levels.[37]

The binary logistic regression/correlation analysis shows that SUA had a weak positive correlation with fetomaternal outcomes which was not statistically significant. Values of correlation co-efficient closer to zero show a weak correlation between variables compared, while values closer to 1 suggest a stronger correlation. This suggests that SUA level is a poor predictor of fetomaternal outcomes. This result is similar with the findings obtained in study conducted by Lim et al which showed a weak correlation between SUA values and the several clinical outcome measures of pre-eclampsia.[30] They concluded that although mean SUA values are elevated in pre-eclamptics, its clinical utility is limited. Like in this study, Lim et al recruited only parturients at term with SUA assayed at the point of delivery.[38] Again, enzymatic colometeric method of uric acid estimation was used as in this study. This similar methodology may explain the similarity in the results obtained.

CONCLUSION

Mean SUA levels of severe pre-eclamptic mothers though elevated when compared with normotensive mothers in AE-FUTHA and Mile 4 Hospital Abakaliki showed no significant difference. There is also a poor positive correlation between SUA levels and fetomaternal outcomes in pre-eclampsia. This suggests that the clinical utility of SUA levels in determining/predicting fetomaternal outcomes in pre-eclampsia is limited and may add no added value in patient's management. This study failed to reject the null hypothesis.

Recommendations

The recommendation from this study is that determining SUA levels routinely in the management of pre-eclampsia is not recommended as this may not be of much benefit in determining their outcome. In developing countries where individual and institutional funding is highly limited, assaying of SUA levels for each patient may be an undue economic burden with little or no benefit.

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