Significance of Serum Uric Acid in Pregnancy Induced Hypertension

Apeksha Niraula, M.D., Madhab Lamsal, Ph.D., Shankar Majhi, M.D., Seraj Ahmed Khan, M.D., Pritha Basnet, M.D.

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Abstract: Background and objective: Hypertension is one of the common medical complications of pregnancy and contributes significantly to maternal and perinatal morbidity and mortality. Uric acid (UA) is filtered, reabsorbed and secreted by the kidney. Thus, this study was conducted to assess the serum UA levels in PIH and to evaluate the diagnostic value of serum UA level in PIH.

Materials and methods: This is a hospital based comparative cross-sectional study conducted in BPKIHS. Ninety study participants were included; forty five participants were diagnosed of PIH and forty five in control group after obtaining informed consent from study participants from August 2014 to May 2015. Serum UA was done by Uricase method and serum creatinine was done by Jaffe's alkaline picrate method in cobas c311 autoanalyser. Data were expressed in frequency, percentage, mean ± S.D., median (IQR), and Independent t-test, Mann–Whitney U test were applied. p Value <0.05 is considered to be significant.

Results: Mean serum UA levels was higher in PIH compared to control group (5.46 ± 1.51 vs 4.03 ± 0.69) respectively. ROC curve demonstrated that serum UA showed a superior diagnostic efficiency (Sensitivity – 79.07%, Specificity – 71.19%) compared to creatinine (Sensitivity – 62.75%, Specificity – 27.45%) in PIH respectively.

Conclusion: The present study shows that serum UA is significantly raised in PIH compared to the control group. Assessment of uric acid is a convenient and cost-effective method for determination of severity in PIH. Thus, serum uric acid can still be used as prevalent marker for risk assessment in PIH.

Keywords: Hypertension■Pregnancy induced■Uric acid

Author affiliations: Apeksha Niraula, Department of Biochemistry, Birat Medical College and Teaching Hospital, Tankisinwari-02, Morang, Nepal; Madhab Lamsal, Department of Biochemistry, Birat Medical College and Teaching Hospital, Tankisinwari-02, Morang, Nepal; Shankar Majhi, Department of Biochemistry, Birat Medical College and Teaching Hospital, Tankisinwari-02, Morang, Nepal; Seraj Ahmed Khan, Department of Biochemistry, Birat Medical College and Teaching Hospital, Tankisinwari-02, Morang, Nepal; Pritha Basnet, Department of Obstetrics and Gynecology, B.P. Koirala Institute of Health Sciences, Dharan, Nepal

Correspondence: Apeksha Niraula, M.D., Department of Biochemistry, Birat Medical College and Teaching Hospital, Tankisinwari-02, Morang, Nepal., email: apekshyaniraula@gmail.com

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INTRODUCTION

Hypertensive disorders of pregnancy include gestational hypertension and preeclampsia which possess a high obstetric risk.[1] Hypertensive disorders are the most common medical complication contributing to about 2–10% of all pregnancies.[2] It is still considered to be a major cause of maternal and perinatal morbidity and mortality.[3] Gestational hypertension is considered as a predictor for the subsequent development of cardiovascular disease and metabolic syndrome in future non-pregnant life.[4] Majority of patients with gestational hypertension share a common risk factors and pathophysiological abnormalities such as rise in maternal age, elevated body mass index (BMI), and hyperuricemia.[5]

Pregnancy may induce hypertension in women who are apparently normotensive before pregnancy.[6] Screening for preeclampsia at an early stage permits for a vigilant antenatal surveillance and an appropriate timing of fetal delivery in order to avoid serious sequel i.e.: Eclampsia. Hyperuricemia is an independent predictor of cardiovascular and renal disease in non-pregnant population and in subjects with chronic hypertension.[7] Women with Pregnancy Induced Hypertension (PIH) are frequently found with elevated uric acid level in maternal blood, presumably due to decreased renal urate excretion. Numerous studies have been conducted to find out the relationship between elevated uric acid level and preeclampsia.[8] Raised serum uric acid associated with preeclamptic pregnancies has been reported almost a century ago.[9] Reduced uric acid clearance secondary to reduced glomerular filtration rate, increased reabsorption, and decreased secretion may be at the origin of elevated serum levels in women with PIH.[10,11] Several studies have reported a positive correlation between elevated maternal serum uric acid and adverse maternal and fetal outcomes.[12–15] Studies from different part of the world[16,17] have evaluated several tests and parameters, including uric acid, during the first or second trimester of pregnancy, as potential predictors of preeclampsia, with mixed results, and generally with unsatisfactory sensitivity and/or specificity. The present study restricts the analysis to the pregnant women with gestational hypertension, to evaluate the prognostic value of serum uric acid for the subsequent development of preeclampsia, thereby helping to identify the patients with gestational hypertension at greater risk.

AIMS AND OBJECTIVES

1. To assess and compare serum uric acid, creatinine and urea in pregnancy induced hypertension and normal pregnant women.
2. To determine the diagnostic efficiency of uric acid as a predictive marker of severity in PIH.

MATERIAL AND METHODS

The present study was conducted at the Department of Biochemistry in collaboration with Department of Obstetrics and Gynaecology, BPKIHS, Dharan, Nepal after being approved by the Institutional Ethical Review Board (IERB), BPKIHS. An informed consent was obtained from all the study participants. Renal function was investigated in two groups of pregnant women: one with PIH (n = 45) and the other of healthy pregnant women (n = 45).

Blood pressure measurement and urine analysis was performed at the beginning of the pregnancy to exclude pre-existing proteinuria or renal disease. Maternal conditions potentially affecting GFR during the study (Pre-gestational Hypertension, Diabetes, Other concomitant Renal disease), if present, were not included.

The common inclusion criteria for both groups were: normal fetal morphology and the absence of concomitant disease and gestation between ≥24 and 36 gestational weeks. Additional inclusion criteria for PIH were a systolic blood pressure level of 140 mmHg or higher or a diastolic blood pressure level of 90 mmHg or higher that occurred after 20 weeks of gestation without proteinuria in a 24 h urine specimen. Other parameters included in the study were gestational age, parity, body mass index (BMI). Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were also noted in both the study groups.

The results from PIH group were compared with that of the healthy age and gestational week matched control group.

Serum uric acid levels were measured by standardized enzymatic PAP method with Uricase and peroxidase, in cobas c311 autoanalyser with intra- and interassay %CV less than 2.44%, and according to the procedure recommended by the manufacturer. Serum creatinine levels were measured by a Jaffe's method, in cobas C311 Autoanalyser (Roche Diagnostics) with intra- and interassay %CV less than 2.45%, and according to the procedure recommended by manufacturer. Serum Urea was estimated by standard urease method in cobas c311 auto-analyzer with intra- and interassay %CV less than 2.44%, and according to the procedure recommended by the manufacturer.

In patients with PIH, total urinary protein levels were measured by modification of the dye binding method used by Fujita et al and with commercial Uric 3V SGO 3100.

Statistical analysis

Data were first checked for normality using Kolmogorov–Smirnov test. Student's t-test was used for variables with normal distribution and Mann–Whitney U test for the non-normally distributed variables. The Chi square test was used for categorical variables, and correlations among the parameters were examined by Pearson's and Spearman's rho correlation according to the nature of the data. The calculations were performed using the statistical program SPSS for Windows Version 11.5 (Chicago Inc), with a p-value of <0.05 considered to be statistically significant. Receiver operating characteristic (ROC) plots and cut-of levels for maximal sensitivity and specificity were produced using the program MedCalc for PC (MedCalc Software, Belgium).

Table 1. Baseline and clinical characteristics of the study population.

General characteristics	PE (n = 51)	Control (n = 51)	p Value
Age (years)	26.84 ± 5.20	25.84 ± 4.54	0.095[a]
POG (weeks)	35.02 ± 4.59	29.35 ± 3.35	0.001[a]
BMI (kg/m^2)	28.71 ± 4.30	24.47 ± 2.86	0.001[a]
SBP (mmHg)	136.86 ± 10.67	104.31 ± 10.24	0.001[a]
DBP (mmHg)	95.69 ± 8.77	71.76 ± 7.67	0.001[a]
MAP	109.41 ± 8.21	82.61 ± 7.86	0.001[a]

[a] = *Independent t-test.*

**p Value < 0.05 is considered to be significant.*

RESULTS

Descriptive characteristics of the study participants are illustrated in Table 1. Overall, most of the study participants in both the study groups were Multigravida and Nulliparous. Our study shows that the female developed PIH in late gestational period (35.02 ± 4.59 weeks). The details of the baseline and clinical parameters have been summarized in Table 1.

Serum uric acid was significantly higher in the PIH group compared to the control women. While, the serum creatinine and urea were approximately similar in between the groups as depicted in Table 2 and Figs. 1 and 2 respectively.

Table 2. Biochemical parameters of the study population.

Parameters	PIH (n = 45)	Control (n = 45)	p Value
Uric acid	5.46 ± 1.51	4.03 ± 0.69	0.001[a,*]
Creatinine	0.50 ± 0.36	0.49 ± 0.14	0.777[a]
Urea	15.77 ± 6.35	15.28 ± 4.70	0.674[a]

[a] = *Independent Student's t-test.*

**p Value < 0.05 is considered to be statistically significant.*

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