World Journal of Pharmaceutical Sciences
ISSN (Print): 2321-3310; ISSN (Online): 2321-3086
Available online at: http://www.wjpsonline.org/
Original Article

Role of blood urea, creatinine and uric acid as a marker of renal function tests in hypertensive patients

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Received: 12-02-2018 / Revised Accepted: 11-03-2018 / Published:12-03-2018

ABSTRACT

Hypertension is both a cause and consequence of renal disease, but the prevalence of kidney disease throughout the diagnostic spectrum of blood pressure has not been established. The presence of hypertension with renal dysfunction has the strongest relationship; hence the study to examine the status of blood urea, creatinine and uric acid as the possible marker for renal function in hypertensive patients. 300 patients (188 males and 112 females) clinically diagnosed to be hypertensive, under regular visit to OPD of GMC Jammu were recruited to participate in this study as cases. All patients, except with secondary hypertension, gout, significant history of alcohol abuse were excluded from our study. 100 age and gender-matched healthy individuals were considered as the controls. The systolic and diastolic blood pressure was significantly higher in all the cases as compared to healthy controls. Patients in the hypertension stage II had higher values of blood urea, creatinine and uric acid as compared to patients in other sub groups, indicating clear association with RFT. The significance of the data was further revealed with the comparative elevation of hypertension, or duration thereof, leading to the higher levels of Blood urea, creatinine and uric acid.

Key words: Hypertension, Renal function markers

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How to Cite this Article: Sanjeev Kumar and Ashima Badyal. Role of blood urea, creatinine and uric acid as a marker of renal function tests in hypertensive patients. World J Pharm Sci 2018; 6(3): 131-135.

INTRODUCTION

Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive heart failure (CHF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease. No single or specific cause is known for most cases of hypertension, however, prolonged hypertensive patients face a greater risk of end-stage renal diseases. Persistent hypertension can develop in response to an increase in cardiac output or a rise in peripheral resistance and the interplay of various derangements factors affecting cardiac output and peripheral resistance may aggravate the disease.[1] From an epidemiologic perspective, there is no obvious level of blood pressure that defines hypertension. The Multiple Risk Factor Intervention Trial (MRFIT), which included >350,000 male participants, demonstrated a continuous and graded influence of both systolic and diastolic blood pressure on CHD mortality, extending down to systolic blood pressures of 120 mm Hg.[2] Clinically, hypertension may be defined as that level of blood pressure at which the institution of therapy reduces blood pressure–related morbidity and mortality. Current clinical criteria for defining hypertension generally are based on the average of two or more seated blood pressure readings during each of two or more outpatient visits.[3] Based on the seventh report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC VII report) BP is classified into the following stages.

Table 1: Blood Pressure Classification

Blood pressure classification	Systolic, mm Hg	Diastolic, mm Hg
Normal	<120	And <80
Pre hypertension	120-149	Or 80-90
Stage 1 hypertension	140-159	Or 90-99
Stage 2 hypertension	>160	Or >100

It is a well-known fact that many conditions affect the ability of the kidneys to carry out their vital functions. Initially this study was conducted to assess the renal functions in patients with hypertension by measuring the serum urea and creatinine but we further included the measurement of uric acid in our study owing to the frequent presence of hyperuricemia in hypertensive patients thereby reflecting underlying renal dysfunction or reduced renal perfusion.[4]

The aims and objectives of the present study were:

1. To determine the status of blood urea, creatinine and uric acid in the hypertensive patients.
2. To compare and correlate these parameters with those of healthy controls.
3. To derive necessary inferences from the results thus obtained.

The purpose of the study was to evaluate the effect of severity and duration of hypertension on our study parameters.

MATERIALS AND METHODS

300 patients (188 males and 112 females), clinically diagnosed to be hypertensive, under regular visit to OPD of GMC Jammu, were recruited to participate in this study as cases. These patients were following up check-up from time to time. An inclusion criterion for them was adult males and females diagnosed as hypertensive according to JNC VII classification, with or without complications or any co-morbid conditions like diabetes mellitus type 2, coronary artery disease etc. However, patients with secondary hypertension, gout, significant history of alcohol abuse were excluded from our study. 100 age and gender matched normotensive healthy individuals who visited the hospital for medical check-ups in the general outpatients department were considered as the controls. All the subjects, including the controls, were fully informed about the study and their voluntary informed consents were taken.

Fasting venous blood was collected from the cases (hypertensive patients) as well as the controls (healthy subjects), it was centrifuged and the serum was separated for the analysis on the same day for serum urea, creatinine and uric acid as per the routine procedure which was followed in the biochemistry laboratory. The sample analysis for serum urea, creatinine and uric acid was carried out on a semiautomated analyser by using different reagent kits as per the procedure defined by the manufacturer as per their defined principles.

Urease method was evaluated by Talke H et al,[5] in 1965, defining the principle, briefly: Urease specifically hydrolyzes urea to form ammonia and carbon dioxide. The ammonia is used by the enzyme glutamate dehydrogenase to reductively amminate alpha keto glutarate, with simultaneous oxidation of NADH. The change in absorbance at 340 nm due to disappearance of NADH is directly

proportional to the BUN concentration in the sample and is measured by using a micromatic rate technique; giving the reference range of serum urea to be 7-18 mg/dl.

Jaffes method was evaluated by Knapp ML, and Mayne PD,[6] in 1987, defining the principle, briefly as: Creatinine in the presence of alkaline medium reacts with picric acid to form reddish yellow colour complex called as creatine picrate, and measured at 520nm; giving the reference range of serum creatinine: Males: 0.7-1.3 mg/dl; Females 0.6-1.02mg/dl.

Uricase method was evaluated by Clinical and laboratory standards institute in 2003,[7] defining the principle as: Uric acid, which absorbs light at 293nm is converted by uricase to allantoin, which is non-absorbing at 293nm. The change in absorbance at 293nm due to the appearance of uric acid is directly proportional to the concentration of uric acid in the sample and is measured using a bichromatic 293, 700nm; giving the reference range of uric acid: Females 2.6-6.0mg/dl; Males 3.5-7.2mg/dl.

The results were analysed statistically using student's "t" test on the suitable software. P values of <0.05 were considered as statistically significant.

RESULTS

There seems to be a strong relationship between hypertension and chronic kidney diseases. It is known to be an important cause of end stage renal disease (ESRD). The present study was conducted keeping Blood Urea, Creatinine and Uric Acid as representative markers for kidney functions and they were found to be significantly higher in hypertensive cases irrespective of age and gender. The systolic and diastolic blood pressure was significantly higher in all the cases as compared to healthy controls. (p <0.001) (Table 2).

All the cases of hypertension were included in the study, irrespective of their co-morbidities as it was quite prevalent along hypertensive cases, while a minor population (32.7%) presented only hypertension as the ailment. (Table 3) However Diabetes Mellitus was found to be the most prevalent amongst all co-morbidities (39.3%) with hypertension. Patients in the hypertension stage II had significantly higher values of blood urea, creatinine and uric acid as compared to patients in other sub groups, indicating clear association with kidney function tests.

On further comparison, the significance of the data was revealed as the comparative elevation of hypertension, or duration thereof, clearly lead to the higher levels of Blood urea, creatinine and uric acid. (Table 4 and Table 5).

Table 2: Comparative study of HT with kidney functions

	Hypertensive cases (n=300)	**Controls (n=100)**	**p value**
Age (years)	50.74±9.05	48.53±6.21	Not significant
Gender (males: females)	188:112	59:41	Not significant
Systolic blood pressure (mm of Hg)	148.66±14.45	122.74±8.32	<0.001 (significant)
Diastolic blood pressure (mm of Hg)	92.02±8.54	79.44±4.62	<0.001 (significant)
Blood urea (mg/dl)	31.92±11.85	20.15±4.73	<0.001 (significant)
Serum creatinine (mg/dl)	0.89±0.31	0.51±0.22	<0.001 (significant)
Serum uric acid (mg/dl)	5.68±1.04	4.08±0.86	<0.001 (significant)

Table 3: Prevalence of hypertension along with co-morbidities

	Only HT	**DM with HT**	**CAD with HT**	**DM + CAD with HT**	**CVA with HT**
Prevalence (%)	32.7	39.3	17.3	7.4	3.3
Prevalence in figures (n=300)	98	118	52	22	10

Table 4: Comparison of the parameters between different sub-groups of hypertensive patients

Parameters	Pre-hypertension stage (n=75)	Stage I hypertension (n=131)	Stage II hypertension (n=94)	Healthy controls (n=100)
SBP (mm Hg)	135.19±4.23	146.18±5.16	173.35±12.06	121.17±6.33
DBP (mm Hg)	86.28±7.90	88.11±11.86	96.51±7.26	80.20±4.43
Blood Urea (mg/dl)	27.10±9.18	33.26±8.07	35.13±10.81	21.08±4.00
Serum creatinine (mg/Dl)	0.88±0.26	0.88±0.21	0.97±0.38	0.48±0.20
Serum uric acid (mg/dl)	5.18±1.16	5.72±1.03	6.10±1.42	4.23±0.98

Table 5: Comparative elevation of hypertension with duration

	Duration of hypertension: <5 years (n=243)	Duration of hypertension: 5-10 years (n=46)	Duration of hypertension: >10 years (n=11)
Blood urea (mg/dl)	32.10±10.18	34.56±11.07	35.13±5.81
Serum creatinine (mg/dl)	0.92±0.32	1.14±0.41	1.17±0.24
Serum uric acid (mg/dl)	5.88±1.06	6.35±1.29	6.26±0.78

DISCUSSION

The present study found RFT significantly higher in hypertensive cases as compared to the controls, irrespective of age and gender. Majority of cases in our study, i.e. 81%, pertained to duration of less than 5 years. Serum urea levels increased from 32.10±10.18 mg/dl for the duration of <5 years to 34.56±11.07 mg/dl and 35.13±5.81 mg/dl for the duration of 5-10 year and >10 years respectively. Similar increase was noticed for other renal function tests like serum creatinine and serum uric acid. The comparative analysis of duration of hypertension thus showed significant elevation of derailment of RFT results with increasing duration of HT. Co-morbidities are quite prevalent with hypertension with the majority (67.3%) showing the presence of one or more serious ailment along with HT and the same was studied by Humes et al.[8] Serum creatinine and uric acid was most pronounced in the patients with stage II hypertension as compared to others, further substantiating the results achieved by Appel et al.[9] With hypertension induced nephrosclerosis, the plasma creatinine levels begin to rise and the results showing significant derailment of mean creatinine levels to 0.89±0.31 mg/dl in comparison to 0.51±0.22 mg/dl of the controls, similarly shown by Matts et al.[10] The results also showed significant rise of mean uric acid levels to 5.68±1.04 mg/dl in comparison to 4.08±0.86 mg/dl of the controls, similarly shown by Friedl et al.[11] Kidney is both the target and a cause of hypertension. Primary renal disease is the most common etiology of secondary hypertension. The results of the present study are, quite invariably, in agreement with the findings of several parallel researches.

CONCLUSION AND FUTURE PROSPECTS:

The routine tests conducted in the clinical biochemistry laboratories i.e. serum urea, creatinine and uric acid are giving the valuable insight to the renal status in hypertension and thereby coming as useful markers in the renal status of these patients, it becomes important to monitor the renal status of the patients with prolonged history of hypertension. The test results are conclusively showing the prevalence of co-morbidities with hypertension, therefore mandating urgent screening of RFT for all patients with HT with a duration >5 years, in order to prevent the occurrence of end-stage renal diseases. Although there are more biochemical markers that improve the clinical outcome, like Cystatin C, Beta trace proteins, etc, in terms of usefulness in assessing the renal function in hypertensive patients, they are still not in the list of essential tests and must be included in the routine sampling for better management of the disease.

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