

Research Article

ESTIMATION OF BLOOD UREA (BUN) AND SERUM CREATININE LEVEL IN PATIENTS OF RENAL DISORDER

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ABSTRACT

There is substantially higher prevalence of the earlier stages of chronic kidney disease with adverse outcomes, including loss of kidney function, cardiovascular disease and pre-mature death. Blood tests for Blood urea nitrogen (BUN) and creatinine are the simplest way to monitor kidney function. These substances are normal metabolic waste products that are excreted by the kidneys. BUN is an indirect & rough measurement of renal & live function measuring the amount of urea nitrogen in blood. BUN is directly related to the excretory function of the kidney. Creatinine tests diagnose impaired renal function & measure the amount of creatinine phosphate in the blood. The present study have been made on twenty(20) normal individuals as control and twenty(20) patients of renal disorder (study group) to investigated the association between blood urea nitrogen (BUN) and serum creatinine levels with the presence of renal disorder. Comparing the values of study group with the control group, the blood urea nitrogen (BUN) and serum creatinine values (mean as well as range) were found to be much higher than the control. When gender wise compared, a significant sex wise variation found only in serum creatinine level but not in urea level. High serum creatinine levels were seen in males than females which could be because of storage of creatinine as a waste product in muscle mass and the presence of high muscle mass in males. In conclusion, a strong correlation has been found between Blood urea nitrogen (BUN) and serum creatinine level with renal disorder. Blood urea nitrogen (BUN) and serum creatinine are widely accepted parameters to assess the renal functions. Kidney failure is a worldwide public health problem, with increasing incidence and prevalence, high costs, and poor outcomes. Strategies to improve outcomes will re-quire a global effort directed at the earlier stages of chronic kidney disease.

Keywords: *Renal Function, Glomerular Filtration Rate, Blood Urea, Serum Creatinine*

INTRODUCTION

Chronic kidney disease has recently been recognized as a public health problem. Clinical assessment of kidney function is part of routine medical care for adults (Stevens *et al.*, 2006). Acute renal failure (ARF) is abrupt deterioration of renal function sufficient to result in failure of urinary elimination of nitrogenous waste products (urea nitrogen and creatinine). This deterioration of renal function results in elevations of blood urea nitrogen and serum creatinine concentrations (Dwinnell and Anderson, 2012). Causes of acute renal failure can be broadly divided into three categories. In the prerenal form there is a reversible increase in serum creatinine and blood urea concentrations; it results from decreased renal perfusion, which leads to a reduction in glomerular filtration rate (GFR). Postrenal acute renal failure is due to obstruction of the urinary collection system by either intrinsic or extrinsic masses. The remaining patients have the renal form, in which structures of the nephron, such as the glomeruli, tubules, vessels, or interstitium, are affected (Lameire *et al.*, 2005). Chronic kidney disease (CKD) be diagnosed, classified, and staged by GFR (KDIGO, 2012). Accurate assessment of GFR is essential for interpreting the symptoms, signs, and laboratory abnormalities that may indicate kidney disease; for drug dosing; and for detecting and managing chronic kidney disease and assessing the prognosis (Inker *et al.*, 2012). Urea nitrogen is a small, uncharged molecule that is not protein bound, and as such, it is readily filtered at the renal glomerulus. Urea nitrogen undergoes renal tubular reabsorption by specific transporters. This tubular reabsorption limits the value of BUN as a marker for glomerular filtration. However, the BUN usually correlates with the symptoms of uremia. By contrast, the production of creatinine is usually more

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constant unless there has been a marked reduction of skeletal muscle mass (eg, loss of a limb, prolonged starvation) or diffuse muscle injury. Although creatinine undergoes secretion into renal tubular fluid, this is very modest in degree. A steady-stable serum creatinine concentration is usually a relatively good marker of glomerular filtration rate (Dwinnell and Anderson, 2012). So the study aimed to investigate the association between blood urea nitrogen (BUN) and serum creatinine levels with the presence of renal disorder.

MATERIALS AND METHODS

This study was conducted in Department of Clinical Pathology, Patna Medical College and Hospital, Patna. 20 blood samples of renal disorder patients and 20 normal controls were analyzed. The main variables under study were urea and creatinine levels. Estimation of blood urea nitrogen (BUN) was done by Berthelot’s method (Berthelot, 1859) while serum creatinine was estimated by alkaline Jaffe’s Picrate method (Owen *et al.*, 1954).

RESULTS AND DISCUSSION

Result

The present study have been made on twenty(20) normal individuals as control and twenty(20) patients of renal disorder (study group). The values of blood urea nitrogen (BUN) and serum creatinine were comparing with study group and control group. Comparing the values of study group with the control group, the blood urea nitrogen(BUN) range of study group 18.1- 31.7 mg/ dl was much higher than the normal range of BUN 6-20 mg/ dl and control group blood urea nitrogen(BUN) range 6.4-15.0 mg/dl. The mean value of study group was 25.1 mg /dl which was also higher than control group mean value 9.8 mg/ dl. In the context of serum creatinine the range of study group were 0.8-2.2 mg/ dl which were higher than the normal range of serum creatinine 0.6 - 1.3 mg/ dl and control group serum creatinine range 0.5-1.0 mg /dl .The mean value of patients 1.3 mg /dl which was also much higher than mean value of control group 0.7 mg/ dl.

Table 1: Comparison of Biochemical Parameters between Control group and Study group (Patients)

Parameters	No. of individuals	Blood Urea Nitrogen(BUN)	Serum Creatinine(mg/dl)	
		(mg/dl)	Mean	Range
Control group	20	9.8	6.4-15.0	0.7
Study group	20	25.1	18.1-31.7	1.3

Table 2: Comparison between Biochemical Parameters of Study group of male and female with the Biochemical Parameters of Control group of male and female respectively

Parameters	Sex distribution	For Male			
		Blood Urea Nitrogen(BUN)	Serum Creatinine(mg/dl)		
		(mg/dl)	Mean	Range	
Control group	8	10.3	7.4-15.0	0.7	0.6-1.0
Study group	13	25.5	19.1-31.7	1.4	1.0-2.2
Parameters	Sex distribution	For Female			
		Blood Urea Nitrogen(BUN)	Serum Creatinine(mg/dl)		
		(mg/dl)	Mean	Range	
Control group	12	9.4	6.4-14.0	0.6	0.5-0.9
Study group	7	24.3	18.1-28.9	1.1	0.8-1.4

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When gender wise compared, in study group the range of blood urea nitrogen (BUN) in males were found to be 19.1-31.7mg/ dl and mean 25.5mg/dl while in females the range of blood urea nitrogen (BUN) were 18.1-28.9mg/ dl and its mean value was 24.3mg/dl. This showed that there was not significant increase in blood urea nitrogen (BUN) level in males than females whereas, the range of serum creatinine in males were 1.0 -2.2 mg /dl and it's mean was 1.4 mg/ dl while in females the range were 0.8- 1.4 mg/ dl and mean value was 1.1 mg/ dl. This showed high levels of serum creatinine in males than females. Thus, a significant sex wise variation found only in serum creatinine level but not in urea level.

Discussion

Urea and creatinine are good indicators of a normal functioning of the kidney and increase of the substances in the serum are indications kidney dysfunction, although several factors such as excessive protein intake, shock, gastrointestinal hemorrhage etc. could also contribute to this (Anderson, 1996). By comparing the values of study group with the control group it was found that blood urea nitrogen (BUN) and serum creatinine values (mean as well as range) were found to be much higher than the control. This increased mean value said that there may be slight obstruction in kidney disease patients in excreting urea and also showed that there was an impairment of renal function either due to reduction of GFR or obstruction that interferes with urinary excretion.

This corroborates with the findings of Schutte *et al.*, (1981) that serum creatinine and urea concentrations change inversely with changes in GFR and are therefore useful in gauging the degree of renal dysfunction. Urea excretion also depends upon hydration status and the extent of water re-absorption as well as upon GFR. Blood urea levels are quite sensitive indicators of renal disease, becoming elevated when renal function drops to around 25-50% of normal (Sharma *et al.*, 2011). Creatinine levels slightly increased due to damage to the kidney and with this; there was reduced glomerular filtration rate due to inflammation of the kidney (Moses and Johnkennedy, 2013). When gender wise compared for both male and female, male showed slightly higher creatinine level than the female. This result is supported by various researchers who showed that high serum creatinine level was seen in males than females, which could be because of storage of creatinine as a waste product in muscle mass and the presence of high muscle mass in males (Ashavaid *et al.*, 2005).

Females usually have a lower creatinine than males, because they usually have less muscle mass (Molitoris, 2007). The amount of creatine per unit of skeletal muscle mass is consistent and the breakdown rate of creatine is also consistent. Thus, plasma creatinine concentration is very stable and a direct reflection of skeletal muscle mass (Martin, 2003). So, Blood tests for Blood urea nitrogen (BUN) and creatinine are the simplest way to monitor kidney function.

Conclusion

In conclusion, a strong correlation has been found between blood urea nitrogen (BUN) and serum creatinine level with renal disorder. It provides supplemental information in regard to renal function. Blood urea nitrogen (BUN) and serum creatinine are widely accepted parameters to assess the renal functions. Kidney failure is a worldwide public health problem, with increasing incidence and prevalence, high costs, and poor outcomes. Strategies to improve outcomes will re-quire a global effort directed at the earlier stages of chronic kidney disease.

ACKNOWLEDGEMENT

I am very thankful to Dr. Manorma Mishra, H.O.D Clinical Pathology, Patna Medical College and Hospital and Dr. R.V. N Singh, H.O.D, Pathology Patna Medical College, Patna, Dr. S.R Padmadeo , HOD Dept. Of Biochemistry, Patna Science College, Patna University, Patna and Dr. A.K Ghosh, HOD Dept. of Chemistry, Patna Science College, Patna University, Patna for their valuable guidance, constant inspiration, encouragement and providing facilities for carrying out this work.

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