

RESEARCH TITLE

UREA AND CREATININE IN RENAL FAILURE PATIENTS WITH VIRAL HEPATITIS.

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Abstract

Hepatitis is a description of a medical illness as liver inflammation and is characterized by the presence of inflammatory cells in the liver. The aim of this study was to detect the frequency of HBV and HCV infection, and the alteration in the activity of renal function represented by blood urea and S. creatinine. The study revealed that viral hepatitis caused by HBV, HCV was found in 41% of patients. The HBsAg was found in 18.52 % and anti-HCV in 22.22%. Non-significant statistical difference was found in comparison between chronic renal failure patients with hepatitis antibodies and those with no hepatitis antibodies in their sera regarding blood urea, the mean concentration of both groups were higher than the normal value, a significant statistical difference ($P < 0.05$) was found between both groups regarding S. creatinine and both groups were with rang above the normal value.

Key Words: Viral hepatitis, Renal failure, Urea, Creatinine.

1. Introduction

Hepatitis is a term used to describe a medical illness that is classified as liver inflammation and is characterized by the presence of inflammatory cells in the tissue of the organ. When it lasts less than six months, hepatitis is referred to as acute hepatitis, and when it lasts longer than that, it is referred to as chronic hepatitis. Hepatitis frequently causes jaundice, low appetite, and malaise, but it might occasionally have no symptoms. Most occurrences of hepatitis in the globe are caused by a family of viruses known as hepatitis viruses, while it can also be brought on by other conditions[1]. Renal Function Tests (urea, creatinine): urea is the main byproduct of protein catabolism. It is created in the liver, put into the bloodstream, and eliminated by the kidneys. A helpful biomarker of renal and hepatic integrity is the measurement of urea in the blood[2]. The body typically produces creatinine at a very steady pace, a little quantity of creatine is actively secreted but the kidneys filter the majority of it. If the kidney's filtering is insufficient, there is little to no tubular reabsorption of creatinine, which causes blood levels to increase[3]. Because of this, measuring creatinine acts as a diagnostic for healthy glomerular filtration, elevated levels are linked to urinary tract blockage and acute and chronic renal failure. Levels less than 0.6 mg/dL have little relevance [4].

2. Material and methods

2.1. one hundred and eight person were included in this work; 81 males and 27 females, blood samples were collected from them, their ages was between 20-80 year who had chronic renal failure undergoing hemodialysis (HD).

2.2. Determination of urea and creatinin in serum concentration in serum done by the Abbott Architect c4000. The appropriated

sample 1-35 μ l (average 7 μ l) of serum, maximum throughput 800 test/hour and it displays analysis results automatically according to manufacturer's instruction (Diamond Diagnostics - USA).

2.3. The statistical analysis was obtained using the statistical package for Social Science (SPSS) version (18) and Microsoft Excel (2003) software's.

3. Results and Discussion

As shown in table (1) there was no statistically significant difference ($P > 0.05$) between CRF patients with hepatitis antibodies and those without hepatitis antibodies in their sera with regard to blood urea in the current investigation. Though there was a significant statistical difference ($P 0.05$) in S.creatinine between the two groups. as indicated in table (2). The normal kidney can excrete huge amounts of urea; nevertheless, creatinine is mostly formed from endogenous sources through tissue creatine breakdown. Urea is derived from amino acids and so from protein. To evaluate renal function, many laboratories prefer to analyze plasma creatinin. Impaired glumerular function is probable if the plasma content of either urea or creatinine is noticeably increased [5]. Our study research found no significant (P - value 0.427) relation of seropositive hepatitis virus with B. urea where the mean value in CRF patients with hepatitis 99.04 mg/dl and for CRF patients without hepatitis 74.48 mg/dl . Six of 20 patients (30%) with HBV had raised levels were the highest concentration was 152 mg/dl, 22/24 (91.6%) patients with HCV had high level, the highest concentration was 171 mg/dl, while in CRF patients without hepatitis 15/64 (23.4%) the highest concentration was 963 mg/dl. In general B. urea concentration was high in 39.8% and the mean of both groups were above the normal rang 15-48 mg/dl. According to s.creatinine there was highly significant

difference in both study groups (P- value 0.0013) CRF patients with hepatitis had mean 7.42 mg/dl, CRF patients without hepatitis had mean 3.12 mg/dl. Six patients from 20 (30%) who had HBV had high level were the highest concentration was 11.08 mg/dl, 22/24 (91.6%) patients with HCV had high level were the highest concentration was 11.86 mg/dl, while in CRF patients without hepatitis 16/64 (25%) the highest concentration was 25 mg/dl. In general s. creatinine concentration was high in 40.7% and the mean of both groups were above the normal rang 0.57-1.25 mg/dl. This result is explained by the fact that patients with chronic renal failure had elevated B. urea and s. This conclusion is supported by other studies like Grunfeld et al [6]. A rise in s. creatinine level is observed only with marked damage to functioning nephrons fluid and waste products from the blood, such as urea and creatinine[11], and is an alternative to renal transplantation in end-stage kidney failure. [12]

4. Conclusion and Recommendations

Regarding blood urea there was no statistical difference ($P > 0.05$) when comparing between CRF patients with hepatitis antibodies and those with no hepatitis antibodies in their sera, the mean concentration of both groups were higher than the normal value. While significant statistical difference ($P < 0.05$) was found between both groups regarding S.

creatinin and both groups were with rang above the normal value. Therefore, we advise using liver biopsies, hematological testing, immunological tests, and renal function tests to assess the severity of liver damage following hepatitis infection. HBV and HCV infection should be detectable in patients with liver disease using more sophisticated techniques, including PCR.

so this test is appropriate for noticing early stage of kidney disease[7]. That its agreed with NAS AL-jeboury[8] who referred that there was a raise in urea concentration in urine of renal failure patients in 73% of them, resulting in weakened kidney function with ongoing age where leading to decrease glumerular filtration after the 30 years old, this raise was due to increase in breakdown of protein materials, or due to chronic renal failure[9], NAS AL-jeboury [8] found an elevation in creatinine concentration in the urine of in renal failure patients in 35% of them. In CRF the increase of serum urea is relative to the advance of the disease but it is highly influenced by a catabolic state or an excessive protein ingestion, leading to a higher production of other waste substances of protein catabolism[10]. Hemodialysis is used to eliminate extra

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Table (1): Comparison between B.urea Results in Both Study Groups in Current Study.

<i>Study group</i>	<i>Mean B. urea ± SD</i>	<i>T- test</i>	<i>Degree of Freedom (df)</i>	<i>Standard Error</i>	<i>P- value</i>
CRF patients with Hepatitis	99.04 ± 51.5	0.79	86	30.78	0.427
CRF patients without Hepatitis	74.48 ± 197.6				

*The concentration unit of B. urea is mg/dl.

Table (2): Comparison between S. creatinine Results in Both Study Groups in Current Study.

<i>Study group</i>	<i>Mean creatinine ± SD</i>	<i>T- test</i>	<i>Degree of Freedom (df)</i>	<i>Standard Error</i>	<i>P- value</i>
CRF patients with Hepatitis	7.42 ± 4.88	3.32	86	1.24	0.0013*
CRF patients without Hepatitis	3.12 ± 6.6				

* *Highly Significant*, the concentration unit of S. creatinine is mg/dl.

References

1. J Fonseca. History of viral hepatitis. *Rev Soc Bras Med Trop*, vol.43, no. 3, pp 322-30, 2010.
2. A S Kanagasabapathy, S Kumari. Guidelines on Standard Operating Procedures for Clinical Chemistry, SEA-HLM-328, 2000.
3. Diabetes care, vol. 28, no.1, pp. 146-176. PMID 15616252. (2005).
4. Biochemistry Profile in Refrigerated Serum NHANES 1999–2000
5. A Martin. Crock. Clinical chemistry and metabolic medicine. *The Kidney* 2006; 7: 50.
6. Grunfeld, *et al.* Familial C3 glomerulopathy associated with CFHR5 mutations: Clinical characteristics of 91 pateints in 16 pedigrees *CJASN*, vol. 6, no.6, pp.1436-1446, 2011.
7. JL Gross, *et al.* Diabetic nephropathy: diagnosis, prevention, and treatment. *Diabetes Care* accessioned, vol. 28, no. 1, pp.164-76, Oct. 22, 2010
8. NAS AL-Jebouri . Biological study of renal failure patients urine in Salahalddin Governorate. M sc thesis. Education College / Tikrit University, 2005.
9. TE Willnow and GA Coles. Proteinuria: a direct cause of renal morbidity. *Kidney Intern.*, vol.45, pp.443-450, 1994.
10. I Jumaa. A study of some biochemical parameters in blood serum of patients with chronic renal failure. *Journal of Basrah researchers (sciences)*, vol. 39, no. 4, 2013.
11. M Schoorl. Universiteit Van Amsterdam Digital Academic Respiratory (UVA DARE), vol. 176, 2014.
12. NS Nagane and JV Ganu . Lipid profile and serum paraoxonase 1 activity in CRF patients pre and post hemodialysis 2011.