

Some Biochemical Assays in Hemodialysis Patients with Chronic Hepatitis C

Arwa F. Jameel¹, Eqbal N. Tawfeeq²

¹ Department of Biology, College of Education, Ibn al - Haytham Pure Sciences, Baghdad University, Baghdad, Iraq.

² Department of Biology, College of Science, Baghdad University, Baghdad, Iraq

Abstract: *The present study aimed to evaluate calcium, potassium, albumin, protein, creatinine, urea, uric acid levels, and the level of total sialic acid in the sera of patients with chronic renal failure who had been infected with Hepatitis C virus and in the sera of patients with chronic renal failure, and compare them with healthy volunteers. A total of 90 subjects with age 25-55 years, were divided into three groups. G1 represents 30 patients with chronic renal failure who had treated by dialysis and infected with chronic Hepatitis C virus (positive group). G2 represents 30 patients with chronic failure who had been treated by dialysis (negative group), while G3 represents 30 healthy volunteers (control group). The results showed that the calcium, albumin, protein, and uric acid levels were decreased significantly ($P \leq 0.05$) in serum of G1 and G2 as compared with healthy group (G3). While levels of Potassium, creatinine, urea, and total sialic acid were significantly ($P \leq 0.05$) increased in serum of G1 and G2 as compared to G3. In conclusion patients with renal failure whom suffering from chronic hepatitis C, which they are serious diseases in Iraq; and although hemodialysis is an attempt to keep homeostasis in the patients, but it is not easy to retain them from the risk of mortality.*

Keywords: hemodialysis, hepatitis C, total sialic acid

1. Introduction

Kidneys are the chief organs for excretion wastes and other products from blood, and therefore, deserve special attention. Besides its function, kidneys function in a significant manner in the maintenance of internal environment of the body. If the kidneys stop working effectively, kidneys failure means kidneys fail to adequately filter toxins and waste products from the blood, so blood will need to undergo dialysis treatments [1].

Hemodialysis is a method for extracorporeal removing waste products such as creatinine and urea, as well as free water from blood when the kidneys in renal failure. In addition to hemodialysis there were two another therapies renal transplant and peritoneal dialysis [2]. Since hemodialysis requires access to the circulatory system, patients undergoing hemodialysis may expose their circulatory system to infections, which can lead to complications [3]. The risk factors associated with hepatitis C virus (HCV) infection among hemodialysis patients include history of blood transfusions, the volume of blood transfused, and years on dialysis [4],[5]. Number of years on dialysis is the major risk factor independently associated with higher rates of HCV infection. Hepatitis C has frightening tendency to result in chronic hepatitis and the sequel of cirrhosis and hepatocellular carcinoma [6], [7].

The present study aimed to evaluate calcium, potassium, albumin, protein, creatinine, urea, uric acid levels, and the level of sialic acid in the sera of patients with chronic renal failure who had been infected with Hepatitis C virus and in the sera of patients with chronic renal failure, and the effect of hemodialysis on them.

2. Subjects, Material & Methods

2.1 Subjects

Total serum sialic acid level (TSA) and other biochemical tests were studied in the blood samples of forty patients with end-stage renal disease attending Al-Kindy Teaching Hospital / Baghdad during the period May - October 2017. Their age range was 30-50 years. The patients were divided into three groups: 30 patients of chronic renal failure who had been treated by dialysis and had been infected with chronic Hepatitis C virus (positive group) (G1), 30 patients with chronic renal failure who had been treated by dialysis (negative group) (G2), and 30 serum samples from healthy volunteers (G3).

2.2 Biochemical Tests

Determination of calcium:

Calcium in serum was determined by referee method of Paul, et al. [8].

Determination of potassium:

Potassium was measured by enzymatic determination spectrophotometric method [9].

Determination of albumin:

Quantitative determination of albumin was done based on the method of (Rutstein, et al., 1953) (10).

Determination of protein:

Total protein in samples was measured by a commercially kit (Randox) with Biuret method [11].

Determination of creatinine:

The concentration of creatinine was measured by a commercially kit (Biomerieux) with enzymatic method [12].

Determination of urea:

The concentration of urea was measured by a commercially kit (Biomereux) with enzymatic method [13].

Determination of uric acid:

The concentration of uric acid was measured by a commercially kit (Biomeghreb) with enzymatic method [13].

Measurement of Total Serum Sialic Acid (TSA):

Determination of total serum sialic acid for control, patients and calibration samples was performed by the resorcinol method [14].

Statistical analysis with analysis of variance (ANOVA) test and least significant difference (LSD) by probability of less than 0.05(p<0.05), were used.

3. Results

Table [1] refers to the mean of level of Ca²⁺, K⁺, Albumin, Protein, Urea, Uric acid, and Total sialic acid in serum of G1, G2, and G3; and revealed that there was a significant

(P≤ 0.05) decrease in level of Ca²⁺ in each of G1 and G2 in comparison to control group (G3). Also the results showed that there was significant (P≤ 0.05) increase in the level of K⁺ in groups of patients of positive and negative (G1 and G2) respectively as compared to control group (G3). In serum albumin results G1 and G2 showed a significant (P≤ 0.05) decrease in level of it in comparison to G3 (control). Serum protein level was also significantly (P≤ 0.05) decreased in positive group (5.5±0.2g) and negative group (5.1±0.2g) as compared with healthy group (7.4±0.2g). While the mean of creatinine levels in G1 was (10.8±0.4mg/dl) and in G2 was (11±0.3mg/dl), they were significantly (P≤ 0.05) increased when compared with healthy group (G3) (1±0.07mg/dl). Serum urea level as the results suggested, showed highly significant (P≤ 0.05) variation between positive group (229±12.8mg/dl), negative group (212±13.5mg/dl) and healthy group (32.7±1.6mg/dl). In relation to serum uric acid levels, results showed a significant (P≤ 0.05) decrease in G1 and G2 when compared with G3 (control). The serum total sialic acid (TSA) levels of G2 were (110.7±7.5mg/dl), while TSA level of serum in G1 were (68±2.9mg/dl), they were significantly higher than those in healthy group (58.3±1.7mg/dl) (G3).

Table 1: Levels of serum calcium, potassium, albumin, protein, creatinine, urea, uric acid and total sialic acid in the patients and control groups

Group	Ca ²⁺ mg /dl	K ⁺ mEq / l	Albumin U/L	Protein gm	Creatinine mg/dl	Urea mg /dl	Uric acid mg /dl	Total sialic acid mg/dl
G1(positive)	7.3±0.3 c	5.2±0.1 c	3.4±0.2c	5.5±0.2 c	10.8±0.4c	230±12.8 c	3.8±0.1c	66±4.8c
G2(negative)	7.8±0.2 b	4.9±0.2 b	3.3±0.1b	5.1±0.2 b	11±0.3b	212±13.5 b	3.5±0.07 b	108±10.5 b
G3 (control)	9.4±0.2 a	4.4±0.2 a	4.2±0.1a	7.4±0.2 a	1±0.07a	32.7±1.6 a	4.8±0.3 a	56±1.4 c

Values are means± SE, Different letters refer to significant differences (p<0.05)

4. Discussion

Kidneys in general, organs have three functions. First functions as filters, removing toxins and metabolic products from the blood then excreting them with urine. Second play an important role in haemostatic by balancing electrolytes and acid-base in body fluids. Third they produce hormones, and regulating blood flow and blood pressure [15].

The goal of dialysis is to keep different materials, such as calcium at normal levels. Calcium helps to build bones and move muscles [16]. If calcium levels get disturbance, it can affect of work of nervous system and muscles, including heart problems [17]. Both of chronic kidney disease and Hepatitis C are harmful and truly medical problems in the entire world [18]. Results in present study showed decrease of Ca²⁺ in positive and negative patients from healthy subjects, and importantly must know that there is calcium exchangeable pool in bone, which is working as a buffer inside the body[19], [20]. That buffer is depending on the movement of Ca²⁺ into and out from the blood of patients within membrane during dialysis [21], and it's clear the patient groups has the ability to lose calcium ions and not to retain them because of failure of kidney. In relation to balancing the level of K⁺ in kidney failure patients, which is a serious problem in the world because malfunction of kidney and overload of K⁺ level may led to stop working of heart [21], and our results agreed with this opinion. The

concentration of albumin in serum is maintained by its rate of synthesis, catabolism, and distribution between vascular and extravascular compartments, and in well dialyzed patients, inflammation is the principle cause of a decrease in serum albumin while protein intake plays an insignificant role [22]. Our results agreed with these findings. Protein was also significantly decreased in G1 and G2 as compared to control group (G3), hemodialysis treatment causes protein losses of about 20g to 30g per 24 hours. Malfunction of the kidney may lead to hematuria (blood loss in the urine) and proteinuria (protein loss in the urine) [23]. Our results agreed with it. Biochemically, renal failure is typically detected by an elevated serum creatinine level [22]. These finding together with present results, that there was relation between renal failure and chronic HCV patients [24], and it is clearly about HCV increases serum creatinine level in the suffering patients. Urea is the major end product in the liver in the urea cycle. It is easily diffusible and exists in all body fluid in practically the same concentration. It is filtered freely by the glumeruli and reabsorbed by the proximal and distal tubules. Most clinicians agree that creatinine is a more specific indicator of glomerular function than blood urea nitrogen (BUN). However the BUN to creatinine ratio may be used as an indirect estimate of renal function and in summary, increased blood urea nitrogen associated with renal failure [24]. This agrees with our results. The means of uric acid level in the sera, showed significant difference between the three investigated groups (G1, G2, and G3) in Table (1). Low serum uric acid is a mortality risk factor in incident hemodialysis patients with a high comorbidity burden and hypoalbuminemia [25]. Serum uric acid can be

elevated due to reduced exertion by the kidneys, and elevated serum level of uric acid are common in patients with kidney disease or in those receiving maintenance dialysis therapy [22]. These findings together with the present results, suggest the role of dialysis in reducing uric acid excretion. Total sialic acids level in our results indicated there was increase in patients groups as compared to healthy group and although hemodialysis is not responsible for putting sialic acid off the body, but sialic acids production happens in glomerular basement membrane (GBM) in cells of visceral layer of Bowman's capsule, and it is composed of sialoglycoproteins, glycosaminoglycan, collagens [26]. When GBM is lost its function because of chronic kidney diseases, it may be expected that production of sialic acids will not stop so their levels will arise. In conclusion patients with renal failure whom suffering from chronic hepatitis C, which they are serious diseases in Iraq; and although hemodialysis is an attempt to keep homeostasis in the patients, but it is not easy to retain them from the risk of mortality.

References

- [1] Rastogi, S.C.(2008). Essentials of animal physiology, 4th Edition, chapter 14 excretion pp. 285-310.
- [2] Dhondup, T. and Qian, Q. (2017). Electrolyte and Acid–Base Disorders in Chronic Kidney Disease and End-Stage Kidney Failure. *Blood purification*, 43: 179-188.
- [3] Thomas, R., Kanso, A., and Sedor, J.R.(2008). Chronic Kidney Disease and Its Complications. *Prim. Care Clin. Office Pract.*, 35: 329–344.
- [4] Meyers, C.M., Seeff, L.B., Stehman-Breen, C.O., and Hoofnagle, J.H. (2003). Hepatitis C and Renal Disease: An Update. *Am. J. Kidney Dis.*, 42:631-657.
- [5] Azmi, A. N., Tan, S.S., and Mohamed, R.(2015). Hepatitis C and kidney disease: An overview and approach to management. *World J. Hepatol.*, 7(1): 78-92.
- [6] Carvalho-Filho, R.J., Feldner, A.C.C.A, Silva, A.E.B, and Ferraz, M.L.G.(2015). Management of hepatitis C in patients with chronic kidney disease. *World J. Gastroenterol.*, 21(2): 408-422.
- [7] Cholongitas, E., Pipili, C., and Papatheodoridis, G. (2015). Interferon-free regimens for the treatment of hepatitis C virus in liver transplant candidates or recipients.
- [8] Paul, J., Bowers, G.N., and Young, D.S.(1973). A referee method for the determination of total calcium in serum. *Clinical Chemistry*, 19 (10) : 1208-1213.
- [9] Mazzachi, R.D., Mazzachi, B.C., and Berry, M.N. (1994). A manual spectrophotometric method for the measurement of serum sodium. *Eur. J. Clin. Chem. Clin. Biochem.*, 32 : 709-717.
- [10] Rutstein, D.D., Ingenito, E.F., Reynolds, W.E., and Burke, J.M. (1954). The determination of albumin in human blood plasma and serum. A method based on the interaction of albumin with an anionic dye-2-(4'-Hydroxybenzeneazo) benzoic acid. *J. Clin. Invest.*, 33(2): 211-221.
- [11] Henry, R.J., Canon, D.C., and Winkelman, J.W.(1974). *Clinical Chemistry "Principles and Techniques"*, 2nd ed., Harper and Row.
- [12] Oliver, I.T. (1955). A spectrophotometric method for the determination of creatine phosphokinase and myokinase. *Biochem. J.*, 61 (1): 116-122.
- [13] Fawcett, J.K., and Scott, J.F.(1960). A rapid and precise method for the determination of urea. *J.Clin. Pathol.*, 13 (2): 156-159.
- [14] Svennerholm, L. (1957). Quantitative estimation of sialic acids II. Colorimetric resorcinol - hydrochloric method. *Biochemica. biophysic. acta.*, Vol. 24.
- [15] Giebisch, G., and Windhager, E.(2005). Organization of the urinary system, p. 737-876. In: W.F., Boron and E.L., Boulpaep (ed.), *Medical physiology*, updated edition, Elsevier Saunders, USA.
- [16] Guyton, A.; and Hall, J. (2006). *Urine Formation by the Kidneys: I. Glomerular Filtration, Renal Blood Flow, and Their Control*", p. 308–325. In: Grulicow, R. (ed.). *Textbook of Medical Physiology* (11th ed.). Philadelphia, Pennsylvania: Elsevier Inc. ISBN 0-7216-0240-1.
- [17] Jimbo, R., and Shimosawa, T. (2014). Cardiovascular risk factors and chronic kidney disease—FGF23: A Key molecule in the cardiovascular disease. *International Journal of Hypertension*, 2014,9.
- [18] Meyers, C., Seeff, L., Stehman-Breen, C.O., and Hoofnagle, J.H.(2003). Hepatitis C and Renal Disease: An Update. *Am. J. Kidney Dis.*, 42:631-657.
- [19] Kay, M.(1995). Hypocalcemia after an acute phosphate load is secondary to reduced calcium efflux from bone: studies in patients with minimal renal function and varying parathyroid activity. *J. Am. Soc. Nephrol.*, 6: 273–280.
- [20] Pirklbauer, M., and Mayer, G.(2011). The exchangeable calcium pool: physiology and pathophysiology in chronic kidney disease. *Nephrol. Dial. Transplant*, 26: 2438–2444.
- [21] Basile, C., and Lomonte, C. (2015). A neglected issue in dialysis practice: haemodialysis. *Clinical Kidney Journal*, 8 (4): 393-399.
- [22] Perico, N., Cattaneo, D., Bikbov, B., and Remuzzi, G. (2009). Hepatitis C infection and chronic renal diseases. *Clin. J. Am. Soc. Nephrol.*, 4(1):207-220.
- [23] Abel, J.J., Rowntree, L.G., and Turner, B.B.(1990). On the removal of diffusible substances from the circulating blood by means of dialysis. *Transactions of the Association of American Physicians*, 1913. *Transfusion Science*, 11 (2): 164-165.
- [24] Fabrizi, F., Messa, P., and Martin, P.(2014). The unravelled link between Chronic Kidney Disease and Hepatitis C Infection. *New Journal of Science*, Volume 2014, pages 9.
- [25] Kaysen, G.A., Dubin, J.A., Müller, H-G., Rosales, L., Levin, N.W., Mitch, W.E., and the hemo study group(2004). Inflammation and reduced albumin synthesis associated with stable decline in serum albumin in hemodialysis patients. *Kidney International*, Vol. 65: 1408–1415.
- [26] Ross, M., and Pawlina, W.(2011). *Urinary system*, chapter 20, p. 698-739. In: Ross, M., and Pawlina, W.(eds.). *Histology A text and atlas with correlated cell and molecular biology*, 6th ed., Wolters Kluwer/Lippincott Williams & Wilkins, Philadelphia.