

Biochemical and Hormones Study on Diabetic Nephrotic Patients

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Abstract

Background: Diabetes mellitus is a growing epidemic and is the most common cause of chronic kidney disease (CKD) and kidney failure. Diabetic nephropathy affects approximately 20–40% of individuals who have diabetes. Diabetic nephropathy can be detected by the measurement of urine albumin or serum creatinine. Visceral adipose tissue-derived serine proteinase inhibitor (vaspin) was identified in the visceral adipose tissue of OLETF (Otsuka Long-Evans Tokushima Fatty) rats, an animal model of obesity and type 2 diabetes mellitus (T2DM).

Material and methods: In this study conducted on 70 individuals in the age group of 35-70 years, from Kirkuk General Hospital. study group consists 40 individuals with diabetic nephropathy and 30 ages and sex matched healthy individuals (control).

Results: Serum vaspin of diabetic nephropathy patients showed a high significant relationship ($p < 0.0001$), as compared with the controls. There is high significant decrease ($p < 0.001$) in the serum levels of T4 and T3. There is high significant increase ($p < 0.001$) in the serum levels of TSH as compared with the controls. There is high significant increase ($p < 0.001$) in the serum concentration of K^+ and uric acid and glucose as compared with the controls. There is high significant decrease ($p < 0.001$) in the serum levels of Ca^{++} and Na^+ as compared with the controls. There is high significant increase ($p < 0.001$) in the serum levels of Cholesterol, TG, LDL and VLDL as compared with the controls. There is significant decrease ($p < 0.05$) in the serum levels of HDL as compared with the controls.

Conclusion: The hormones (vaspin, TSH, T4, T3) have higher diagnostic validity values in the current study, which may be useful as a diagnostic tool to identify recurrence of the diabetic nephropathy syndromes.

Keywords: Vaspin; Diabetic nephrotic; Lipid profile serum T3; Serum T4; Serum TSH; Serum K^+ ; Na^+ ; Serum uric acid; Serum glucose

Introduction

Diabetes mellitus is a growing epidemic and is the most common cause of chronic kidney disease (CKD) and kidney failure. Diabetic nephropathy affects approximately 20–40% of individuals who have diabetes [1]. Diabetic nephropathy can be detected by the measurement of urine albumin or serum creatinine, and both tests should be performed at minimum annually [1]. Those with abnormal levels should have repeat tests done sooner. The first stage of nephropathy is usually the onset of elevated urine albumin which predicts the development of CKD and a gradual decline in glomerular filtration rate (GFR) [2].

Visceral adipose tissue-derived serine proteinase inhibitor (vaspin) was identified in the visceral adipose tissue of OLETF (Otsuka Long-Evans Tokushima Fatty) rats, an animal model of obesity and type 2 diabetes mellitus (T2DM) [3]. Vaspin is also expressed in the skin, hypothalamus, pancreatic islets, and stomach [4]. Circulating levels of adiponectin, a hormone produced predominantly by adipocytes, are highly heritable and are inversely associated with T2DM and other metabolic traits [5]. Both of the two adipocytokines are associated with the diabetes and other metabolic disorders. Some studies have shown that plasma vaspin concentrations are significantly higher in men with the metabolic syndrome compared with those without the metabolic syndrome [6]. That the serum vaspin levels are negatively correlated with the creatinine levels and were significantly reduced in the Japanese chronic hemodialysis (HD) patients [7].

The thyroid gland responds by producing and releasing the 2 thyroid hormones: Tri-iodothyronine (T3) and Thyroxine (T4) [8]. Two primary pathological conditions involving the thyroid gland are hyperthyroidism and hypothyroidism [9]. Hypothyroidism occurs when thyroid gland is not producing enough of thyroid hormones and is by far the most common thyroid disorder in the adult population. Hyperthyroidism is a condition in which thyroid gland is overactive and produces excessive amounts of thyroid hormones [10]. T4 and T3. Acting through nuclear receptors, these hormones play a critical role in

cell differentiation during development and help maintain thermogenic and metabolic homeostasis in the adult. Thyroid stimulating hormone (TSH), secreted by the anterior pituitary plays a pivotal role in control of the thyroid axis [11]. TSH production is suppressed when the T4 levels are high, and vice versa [12,13]. By a negative feedback mechanism, increased levels of free TH (T4 and T3) inhibit TSH secretion from the pituitary, whereas decreased levels of them cause an increase in TSH release from the pituitary. TSH secretion is also influenced by thyroxine releasing hormone (TRH) synthesized in the hypothalamus. TRH causes release of TSH [14,15].

Aim of the Study

Estimation of the levels of hormones (vaspin, TSH, T4, T3) and estimation of the concentrations of urea, creatinine and uric acid. Estimation of the levels of lipid profile. Estimation of the concentration of electrolytes. This author is not done in Iraq previously. To improve the effects of Iraqi environments on the parameters of the patient with diabetic nephropathy, because of the environment in our home is different from other country, socially, economically and also the presence of recurrent wars.

Materials and Methods

This study was conducted on 70 individuals in the age group of 35-70 years, from Kirkuk General Hospital (Iraq). Study group consists

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40 individuals with diabetic nephropathy all the individuals in the authors with diabetic nephropathy were diagnosed clinically and by laboratory findings by medical staff and 30 ages and sex matched healthy individuals (control). Five milliliter (5 ml) disposable plastic syringes were used to draw five ml of venous blood from each patient and control (healthy individuals). containing vacutainer tube and left for 20-30 minutes at 37°C. Blood samples were centrifuged at 1000 g for 10 minutes. Plasma specimens were then frozen and stored at -40°C until analysis. serum vaspin [16], serum T3 [17], Serum T4 [18], Serum TSH [19], were measured by ELISA. Serum potassium (K⁺) [20], Serum calcium (Ca⁺⁺) [21], Serum sodium (Na⁺) [22], Serum glucose [23], Serum urea [24], Serum creatinine [25], Serum uric acid [26], Serum lipid profile [27,28] were measured by spectrophotometer.

Statistical analysis was done using Microsoft office (SPSS version 14) which include the following: mean and standard deviation of variables. The significance of difference between mean values were estimated by student T-test. The probability P<0.0001=highly significant, P<0.05=significant, P>0.05=non-significant. Correlation regression, P value of less than 0.05 was considered significant, less than 0.01 and 0.001 were highly significant.

Results and Discussion

This study included 2 groups of patient, diabetic nephropathy syndrome group and control group. The mean serum vaspin level was 7.11 ± 2.11 ng/dL in controls, 5.21 ± 2.31 ng/dL in diabetic nephropathy. This difference is high significant in diabetic nephropathy subjects (P<0.0001). This study agreement with Yan et al. [29] (Table 1).

The mean serum TSH level was 1.025 ± 0.27 µIU/ml in controls, 3.169 ± 1.78 µIU/ml in diabetic nephropathy. This difference was high significant in diabetic nephropathy subjects (P<0.0001). The mean serum T4 level was 8.95 ± 1.33 µg/dl in controls, 6.96 ± 1.48 µg/dl in diabetic nephropathy. This difference was high significant in diabetic nephropathy subjects (P<0.0001). The mean serum T3 level was 1.36 ± 0.096 ng/ml in controls, 0.630 ± 0.144 ng/ml in diabetic nephropathy. This difference was high significant in diabetic nephropathy subjects (P<0.0001). this study agreement with Rai et al. [30] (Table 1).

The mean serum urea concentration was 35.65 ± 0.933 mg/dL in controls, 168.39 ± 44.4 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). The mean serum creatinine concentration was 0.64 ± 0.082 mg/dL in controls, 6.9 ± 1.27 mg/dL in diabetic nephropathy. This difference was

highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with Rai et al. [30] (Table 1).

The mean serum potassium concentration was 4.12 ± 0.309 mg/dL in controls, 6.01 ± 0.46 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with El-Zawhry et al. [31] (Table 1).

The mean serum calcium concentration was 8.83 ± 0.349 mg/dL in controls, 6.19 ± 0.69 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with Abdelgader et al. [32].

The mean serum sodium concentration was 141.6 ± 2.03 mg/dL in controls, 136.9 ± 4.2 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with csaba P [33] (Table 1).

The mean serum uric acid level was 5.8 ± 0.849 mg/dL in controls, 8.33 ± 0.781 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with Kuriyama et al. [34] (Table 1).

The mean serum glucose level was 95.9 ± 12.31 mg/dL in controls, 179.5 ± 28.7 mg/dL in diabetic nephropathy. This difference was highly significant in diabetic nephropathy subjects (P<0.0001). this study agreement with Rai et al. [30] (Table 1).

The mean serum (cholesterol, TG, LDL, VLDL) level was higher significant in diabetic nephropathy as compared with the control (P<0.0001). but HDL was low (p<0.05) in diabetic nephropathy as compared with the control. this study agreement with Tsimihodimos et al. [35] (Table 1).

Correlations of Study

The obtained results revealed significant relation between vaspin hormone and uric acid. There are stronger positive correlation between vaspin and uric acid. As shown in the Table 2.

Conclusion

The hormones (Vaspin, TSH, T4, T3) have higher diagnostic validity values in the current study, which may be useful as a diagnostic tool to identify recurrence of the diabetic nephropathy syndromes. Hormones levels measurement showed: Valuable information for diagnosis, Good monitoring disease status, Progression of the disease. Serum level of urea, creatinine, uric acid, lipids profile electrolytes and

Parameters	Control	Diabetic nephropathy	P value
Vaspin (ng/dl)	7.11 ± 2.11	5.21 ± 2.31	0.04
TSH (µIU/ml)	1.025 ± 0.27	3.169 ± 1.78	0.001
T4 (µg/dl)	8.95 ± 1.33	6.96 ± 1.48	0.001
T3 (ng/ml)	1.36 ± 0.096	0.630 ± 0.144	0.000
Urea (mg/dl)	35.65 ± 0.933	168.39 ± 44.4	0.000
Creatinine (mg/dl)	0.64 ± 0.082	6.9 ± 1.27	0.000
Potassium (mg/dl)	4.12 ± 0.309	6.01 ± 0.46	0.000
Calcium (mg/dl)	8.83 ± 0.349	6.19 ± 0.69	0.000
Sodium (mg/dl)	141.6 ± 2.03	136.9 ± 4.2	0.000
Glucose (mg/dl)	95.9 ± 12.31	179.5 ± 28.7	0.000
Uric acid (mg/dl)	5.8 ± 0.849	8.33 ± 0.781	0.000
Cholesterol (mg/dl)	213.7 ± 22.6	226.8 ± 20.8	0.04
TG (mg/dl)	164.9 ± 17.4	178.466 ± 29.28	0.04
HDL (mg/dl)	42.9 ± 3.36	41.78 ± 7.1	0.5
VLDL (mg/dl)	33.05 ± 1.73	35.16 ± 4.45	0.04
LDL (mg/dl)	135.6 ± 21.3	148.5 ± 19.8	0.03

Table 1: Mean Serum parameters of the groups studied.

Parameters	r	P value
TSH	0.236	0.236
T4	-0.042	0.835
T3	0.116	0.563
Calcium	0.180	0.370
Potassium	-0.141	0.484
Sodium	0.118	0.559
Cholesterol	0.053	0.793
Triglyceride	0.240	0.227
HDL	0.094	0.642
LDL	-0.077	0.703
VLDL	0.367	0.060
Urea	0.341	0.081
Creatinine	0.186	0.354
Uric acid	0.454	0.017
Sugar	-0.267	0.179

Table 2: Correlations between vaspin and other parameters.

glucose have affected by disease which may be changed dramatically and have no useful role in the diagnosis of patients with renal failure. They may be used as additive factor to detect activity and extent of disease. The results of this study suggested a high correlations between vaspin level and others parameters levels.

References

1. ADA (2015) Microvascular complications and foot care. Section 9. In standards of medical care in diabetes. *Diabetes Care* 38: S58-66.
2. Hahr AJ, Molitch ME (2015) Management of diabetes mellitus in patients with chronic kidney disease. *Clinical Diabetes and Endocrinology* 1: 2.
3. Hida K, Wada J, Eguchi J, Zhang H, Baba M, et al. (2005) Visceral adipose tissue- derived serine protease inhibitor: a unique insulin-sensitizing adipocytokine in obesity. *Proc Natl Acad Sci USA* 102: 10610-10615.
4. Blüher M (2012) Vaspin in obesity and diabetes: pathophysiological and clinical significance. *Endocrine* 41: 176-182.
5. Dastani Z, Hivert MF, Timpson N, Perry JR, Yuan X, et al. (2012) Novel loci for adiponectin levels and their influence on type 2 diabetes and metabolic traits: a multi-ethnic meta-analysis of 45,891 individuals. *PLoS Genet* 8: e1002607.
6. Choi SH, Kwak SH, Lee Y, Moon MK, Lim S, et al. (2011) Plasma vaspin concentrations are elevated in metabolic syndrome in men and are correlated with coronary atherosclerosis in women. *Clin Endocrinol (Oxf)* 75: 628-635.
7. Inoue J, Wada J, Teshigawara S, Hida K, Nakatsuka A, et al. (2012) The serum vaspin levels are reduced in Japanese chronic hemodialysis patients. *BMC Nephrol* 13: 163.
8. Rajender S, Alaa JH, Ashok A (2011) Thyroid Hormones in Male Reproduction and Fertility. *The Open Reproductive Science Journal* 3: 98-104.
9. Shekhar CY, Alwin S, Biswajit M (2012) Status of Thyroid Profile in Type-2 Diabetes Mellitus. *Journal of Nobel Medical College* 1: 64-71.
10. Sachin B, Mahesh M, Sachin S, Vaishali G (2013) Evaluation of Thyroid Hormones in Patients with Type II Diabetes Mellitus. *Journal of Medical Education & Research* 3: 33-39.
11. Kopp P (2005) Thyroid hormone synthesis. In *The Thyroid: Fundamental and Clinical Text*, 9th edn, Braverman LE and Utiger RD (eds.), Lippincott Williams and Wilkins, Philadelphia, USA, p: 52.
12. Lechan RM, Fekete C (2006) The TRH neuron: a hypothalamic integrator of energy metabolism. *Prog Brain Res* 153: 209-235.
13. Zoeller RT, Tan SW, Tyl RW (2007) General background on the hypothalamic-pituitary-thyroid (HPT) axis. *Crit Rev Toxicol* 37: 11-53.
14. Mourouzis I, Politi E, Pantos C (2013) Thyroid hormone and tissue repair: new tricks for an old hormone? *J Thyroid Res* 2013: 312104.
15. Dumont JE, Lamy F, Roger P, Maenhaut C (1992) Physiological and pathological regulation of thyroid cell proliferation and differentiation by thyrotropin and other factors. *Physiol Rev* 72: 667-697.
16. Human Vaspin ELISA Kit, Shanghai Yehua Biotech Co. Ltd., Cat No: YHB3194Hu.
17. Braverman LE (1996) Evaluation of thyroid status in patients with thyrotoxicosis. *Clin Chem* 42: 174-178.
18. Muzaffari EL, Ghari H (1998) Thyroxine suppressive therapy in patients with nodular thyroid disease. *Ann intern med* 128: 386-394.
19. Spencer CA, Takeuchi M, Kazarosyan M, MacKenzie F, Beckett GJ, et al. (1995) Interlaboratory differences in functional sensitivity of immunometric assays of thyrotropin (TSH) and Impact on reliability of measurement of subnormal concentrations of TSH. *Clin Chem* 41: 367-374.
20. Hillmann G, Beyer G (1967) Rapid determination of serum potassium by turbidity measurement with kalignost after protein precipitation. *Z Klin Chem Klin Biochem* 5: 93-94.
21. Sarkar BC, Chauhan UP (1967) A new method for determining micro quantities of calcium in biological materials. *Anal Biochem* 20: 155-166.
22. Young DS (2001) Effects of disease on Clinical Lab. Tests. 4th edn. AACC.
23. Trinder P (1969) Determination of Glucose in Blood Using Glucose Oxidase with an Alternative Oxygen Receptor. *Ann Clin Biochem* 6: 24-33
24. Searcy RL, Reardon JE, Foreman JA (1967) A new photometric method for serum urea nitrogen determination. *Am J Med Technol* 33: 15-20.
25. Labbé D, Vassault A, Cherruau B, Baltassat P, Bonète R, et al. (1996) Method selected for the determination of creatinine in plasma or serum. Choice of optimal conditions of measurement. *Ann Biol Clin (Paris)* 54: 285-298.
26. Schultz A (1984) Uric acid. *Clin Chem. The CV Mosby Co., St Louis, Toronto, Princeton*, pp: 1261-1266.
27. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC (1974) Enzymatic determination of total serum cholesterol. *Clin Chem* 20: 470-475.
28. Fossati P, Prencipe L (1982) Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. *Clin Chem* 28: 2077-2080.
29. Yan M, Su B, Peng W, Li L, Li H, et al. (2014) Association of serum vaspin and adiponectin levels with renal function in patients with or without type 2 diabetes mellitus. *J Diabetes Res* 2014: 868732.
30. Rai S, Kumar JA, Krishan P, Shetty SK, Rai T, et al. (2013) Thyroid function in type 2 diabetes mellitus and in diabetic nephropathy. *J Clin Diagn Res* 7: 1583-1585.
31. Yigit IP, Dogukan A, Keskin L, Taskapan H (2015) Management of Hyperglycemia in Patients with Chronic Kidney Disease and Dialysis. *Austin Journal of Nephrology and Hypertension*.
32. Abdelgader NI, Abdrabo AA (2013) Serum Calcium, Phosphorous, and Parathyroid Hormone in Sudanese Patients under Regular Haemodialysis. *American Journal of Research Communication*.
33. Csaba P (2012) Kovesdy, Significance of hypo- and hypernatremia in chronic kidney disease. *Nephrol Dial Transplant* 27: 891-898.
34. Kuriyama S, Maruyama Y, Nishio SH (2015) Serum uric acid and the incidence of CKD and hypertension. *Clin Exp Nephrol* 19: 1127-1134.
35. Tsimihodimos V, Mitrogianni Z, Elisaf M (2011) Dyslipidemia associated with chronic kidney disease. *Open Cardiovasc Med J* 5: 41-48.

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