

# Measurements of Kidney Function Tests in Diabetic Patients Type 2

**Hassan AB\*, MerzaFA, AL-MsaidHLF and Mushattat SJ**

Department of Biology, Faculty of Science, University of Kufa, Iraq

## Abstract

This study was performed within the Diabetes Mellitus (DM) center in Al-Sadder Teaching Hospital in Al-Najaf province to work out the impact of DM for males & females on the excretory organ operate tests. This study includes (21), and (10) that considered the management cluster. The results show an important increase ( $P \leq 0.05$ ) in organic compounds and Creatinine in examination with the management cluster and an important increase in males than females in organic compounds and Creatinine.

**Keywords:** Diabetes Mellitus; Urea; Creatinine

\***Correspondence to:** Adhraa Baqir Hassan, Department of Biology, Faculty of Science, University of Kufa, Al-Najaf, Iraq, E-mail: [adhraa.alshabawy@uokufa.edu.iq](mailto:adhraa.alshabawy@uokufa.edu.iq)

**Citation:** Hassan AB, MerzaFA, AL-MsaidHLF, et al. (2019) Measurements of Kidney Function Tests in Diabetic Patients Type 2. *Prensa Med Argent*, Volume 105:6. 167. DOI: <https://doi.org/10.47275/0032-745X-167>.

**Received:** November 20, 2019; **Accepted:** December 02, 2019; **Published:** December 05, 2019

## Introduction

Diabetes Mellitus may be a cluster of metabolic diseases characterized by hyperglycemia ensuing from defects in endocrine secretion, endocrine action, or both. Chronic hyperglycemia and alternative metabolic disturbances of DM cause long-run tissue and organ harm yet as dysfunction involving the eyes, kidneys, nerves, and tube systems [1-4]. the foremost common type of damage genetic is Genetic disorder is sorted from. concerning 90 to 95 p.c of the type of genetic disorders is related to old age, obesity, history of genetic disorder status and previous history of physiological state genetic disorder, physical inactivity and quality [5,6]. Concerning 80 p.c of individuals with sort a pair of polygenic disorder square measure overweight. sort a pair of polygenic disorder is more and more being diagnosed in youngsters and adolescents. once sort of a pair of polygenic disorder is diagnosed, the duct gland is sometimes manufacturing enough endocrine, except for unknown cause, cannot the body use the endocrine effective way, a condition referred to as endocrine resistance. when many years, endocrine production decreases. The result's like for sort one accumulates sugar with diabetes within the blood and therefore the body cannot create economical use of its main supply of fuel [7]. The symptoms of the sort of a pair of polygenic disorder develop bit by bit. Their onset isn't as sharp as in sort one polygenic disorder [8].

A natural compound by and large known as an enormous sub-atomic natural compound is a synthetic compound related to a compound grade with a natural compound containing 2 NH<sub>2</sub> gatherings connected to a purposive gathering of carbonyl [9]. The chemical compound serves a significant that have an impact in the metabolism of N-containing compounds by animals and is contain nitrogen substances within the piddle of mammals. it is a colorless,

scentless solid, copious water soluble, and additionally non-toxic. fuzzed in water, it's neither acidic nor alkalescent. The body uses it in many processes, most notably N excretion [10]. Urea was containing Contains substances used as a topical medication product to drive dry skin. matter four-hundredth is indicated for a skin condition, skin condition, corns, and calluses. 400th compound preparations might boot be used for the nonsurgical surgical process of nails. compound 400th "dissolves the physical object matrix" of the nail. solely unhealthy nails square measure which removes, as there's no impact on healthy parts of the nail. This drug is additionally used as AN cerumen removal aid [11]. The organic compound was accustomed to treat euvoletic hyponatremia and was found safe, cheap, and easy [12,13]. Injections of organic compounds have been used to perform an abortion [14]. The blood organic compound N (BUN) check may be alive of the many nitrogens within the blood that comes from organic compounds. it's utilized as a marker of renal work, even though it's second rate compared to elective markers like creatinine because blood natural compound levels square measure affected by elective factors, for example, diet and lack of hydration [15].

Creatinine is also a necessary Indicator of healthy kidney function for being as a final production for metabolism by muscular [16]. The Creatinine made in the liver as the first and transport by the blood to body organisms which become molecular by the phosphorylation convert the Creatinine to become high energy [17,18]. Creatinine enters the blood by the muscular then spreads in all parts of the body and is an indicator of the kidney healthy through the nomination by the kidneys and is considered an increasing indicator of the deterioration of the condition of the kidneys [19]. Ketoacids, cimetidine, and trimethoprim reduce Creatinine by many mechanisms which that happening in severe



nephritic dysfunction every day the muscular return the Creatinine to the blood by the metabolism when the increase intake food that has met that lead to increase too much Creatinine in blood [20-22].

## Materials and Methods

The study was conducted on indiscriminately elite (21) patients of DM within the Al-Sadder Teaching. A group of (10) apparently management subjects were enclosed as a healthy group. The average age of patients was varying 35-65y. Patient data consisted of a name, age, weight and vital sign. The ELISA kits used in this study were Determination of Urea (UA) (ab83362) and Determination of Creatinine (ab65340) ABCAM Company the USA in Origin.

## Statistical Analysis

The results are expressed as (mean±Standard error). analysis of variance check has been used to compare patients and management groups, while pooled t was used to verify comparison between groups divided at measured parameter intervals. Pearson correlation coefficients (r) were calculated to estimate the relationship between parameters. The distinction between groups is fully statistically complete (p < 0.05). All analyzes were performed using math statistics, SPSS version19, while EXCELL generated numbers were exploited for Microsoft Point 2007.

## Results

The table shows the results of the increase (P≤0.05) in UA and Creatinine levels in males of DM patients (71.34±4.19, 1.5±0.02) severally in examination with management groups (32.33±1.02, 0.80±0.02) severally (Table 1).

**Table 1:** Body fluid levels of Organic compound (Urea) and Creatinine in males and management teams.

Male	Urea	Creatinine
	Mean ± Standard error	Mean ± Standard error
Control	32.33 ± 1.02	0.80 ± 0.02
vPatient	71.34 ± 4.19	1.5 ± 0.02

The table shows the results of the increase (P≤0.05) in UA and Creatinine levels in females of DM patients (61.06±2.59, 1.49±0.02) severally in examination with management groups (32.75±3.44, 0.82±0.08) severally (Table 2).

**Table 2:** Body fluid levels of Organic compound and Creatinine in females and management teams.

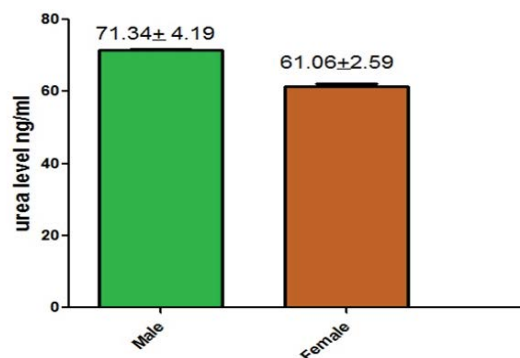
Male	Urea	Creatinine
	Mean ± Standard error	Mean ± Standard error
Control	32.75 ± 3.44	0.82 ± 0.08
Patient	61.06 ± 2.59	1.49 ± 0.02

The results in figures show an increase (P≤ 0.05) in organic compound levels in males of DM patients (71.34±4.19) in examination with females (61.06±2.59) (Figure 1).

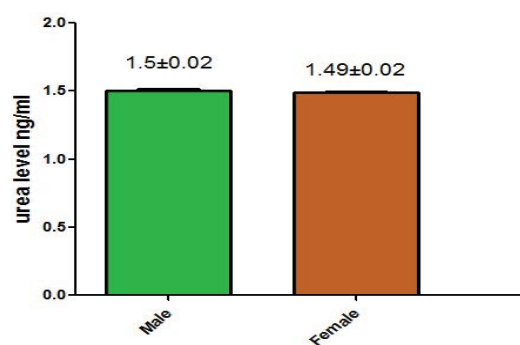
The results in figures show a big increase (P≤ 0.05) in Creatinine levels in males of DM patients (1.5±0.02) in examination with females (1.49±0.02) (Figure 2).

## Discussion

DM may be a major reason for morbidity and mortality. Diabetic uropathy is that the uropathy that happens because of polygenic disorder. a global study has according to that sickness management worsened with a longer period of the disease, with pathology because



**Figure 1:**



**Figure 2:**

the commonest complication followed by vas complications, nephritic complications, retinopathy, and foot ulcers [23]. Different levels of Creatinine in diabetes mellitus patient but in the male, the level of Creatinine is less than because the more activity that may Possible to say the Creatinine Remains in body, this residual weakens the kidneys [24]. Also the hyperuricemia in men is more than in female in diabetes patient that lead to High blood pressure through excessive drinking water the age and sex on the muscular component which that become more of Creatinine were is formed the salt cleavage of amino alkanolic acid, the men are more affected than women by a large amount of the condition could be responsible [25-27]. The male diabetic subjects had considerably (p ≤ 0.05) higher glucose levels compared to feminine diabetic subjects. Similarly, each body fluid creatinine and blood organic compound were considerably (p ≤ 0.05) higher in males over females. the upper values of abstinence and glucose levels in males than the females during this study indicate poor glycemic management in males that is an indicator of Diabetic uropathy (DN). this will be explained that strict glycemic management lowers the danger of uropathy and alternative diabetic complications [28,29]. High body fluid creatinine levels were seen in males than females, that might be owing to the presence of high muscle mass in males compared to females as according earlier [5].

## Conclusion

The study indicates that the upper levels of organic compound and Creatinine square measure joined with a rise of DM risk factors, so organic compound and Creatinine square measure higher in males in compare with females.



## References

1. Adler AI, Stevens RJ, Manley SE, Bilous RW, Cull CA, et al. (2003) Development and progression of nephropathy in type 2 diabetes: The United Kingdom Prospective Diabetes Study (UKPDS 64). *Kidney Int* 63: 225-232.
2. Allen PJ (2012) Creatine metabolism and psychiatric disorders: does creatine supplementation have therapeutic value? *Neurosci Biobehav Rev* 36: 1442-1462.
3. Al-Rawi K (2000) Entrance to the Statistics. Faculty of Agriculture and Forestry, University of Mosul, Iraq.
4. American Diabetes Association (2013) Diagnosis and classification of diabetes mellitus. *Diabetes care* 36: S67-S74.
5. Ashavaid TF, Todur SP and Dherai AJ (2005) Establishment of reference intervals in Indians population. *Ind J of Clin Biochem* 20: 110-118.
6. Bruce DG, Davis WA and Davis TME (2005) Longitudinal predictors of reduced mobility and physical disability in patients with type 2 diabetes: the Fremantle Diabetes study. *Diabetes Care* 28: 2441-2447.
7. Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hamalainen H, et al. (2001) Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344: 1343-1350.
8. Klein S, Sheard NF, Pi-Sunyer X, Daly A, Wylie-Rosett J, et al. (2004) Weight management through lifestyle modification for the prevention and management of type 2 diabetes: rationale and strategies. A statement of the American Diabetes Association, the North American Association for the Study of Obesity, and the American Society for Clinical Nutrition. *Am J Clin Nutr* 80: 257-263.
9. Favre HA, Powell WH (2013) Nomenclature of organic chemistry: IUPAC recommendations and preferred names. International Union of Pure and Applied Chemistry, The Royal Society of Chemistry, United Kingdom.
10. Marsh KL, Sims GK and Mulvaney RL (2005) Availability of urea to autotrophic ammonia-oxidizing bacteria as related to the fate of <sup>14</sup>C- and <sup>15</sup>N-labeled urea added to soil. *Biol Fert Soils* 42: 137.
11. Hama H, Kurokawa H, Kawano H, Ando R, Shimogori T, et al. (2011) Scale: a chemical approach for fluorescence imaging and reconstruction of transparent mouse brain. *Nat Neurosci* 14: 1481-1488.
12. Traynor J, Mactier R, Geddes CC, Fox JG (2006) How to measure renal function in clinical practice. *BMJ* 333: 733-737.
13. Gibb BC (2009) Teetering towards chaos and complexity. *Nature Chemistry* 1: 17-18.
14. Klein J, Blount MA and Sands JM (2011) Urea Transport in the Kidney. *Compr Physiol* 1: 699-729.
15. Welch I (2007) Urea vs UAN: the relative merits of choosing solid urea or urea ammonium nitrate (UAN) solutions. *Nitrogen+Syngas* 289: 26.
16. McDonald T, Drescher KM, Weber A, Tracy S (2012) Creatinine inhibits bacterial replication. *J Antibiot* 65: 153-156.
17. Smithee S, Tracy S, Drescher KM, Pitz LA, McDonald T (2014) A novel, broadly applicable approach to isolation of fungi in diverse growth media. *J Microbiol Methods* 105: 155-161.
18. Leland KM, McDonald TL, Drescher KM (2011) Effect of creatine, creatinine, and creatine ethyl ester on TLR expression in macrophages. *Int Immunopharmacol* 11: 1341-1347.
19. Samra M, Abcar AC (2012) False estimates of elevated creatinine. *Perm J* 16: 51-52.
20. Gross JL, de Azevedo MJ, Silveiro SP, Canani LH, Caramori ML, et al. (2005) Diabetic nephropathy: diagnosis, prevention, and treatment. *Diabetes Care* 28: 164-176.
21. Faul R (2007) Prescribing in renal disease. *Australian Prescriber* 30: 17-20.
22. Harita N, Hayashi T, Sato KK, Nakamura Y, Yoneda T, et al. (2009) Lower serum creatinine is a new risk factor of type 2 diabetes: the Kansai healthcare study. *Diabetes Care* 32: 424-426.
23. Kaveeshwar SA, Cornwall J (2014). The current state of diabetes mellitus in India. *Australas Med J* 7: 45-48.
24. Cholongitas E, Shusang V, Marelli L, Nair D, Thomas M, et al. (2007) Review article: renal function assessment in cirrhosis - difficulties and alternative measurements. *Aliment Pharmacol Ther* 26: 969-978.
25. Wallace KI, Riedel AA, Joseph-Ridge N, Wortman R (2004) Increasing prevalence of gout and hyperuricemia over 10 years among older adults in a managed care population. *J Rheumatol* 31: 1582-1587.
26. Choi HK, Atkinson K, Karlson EW, Carham G (2005) Obesity, weight change, hyperuricemia, diuretic use and risk of gout in men. *Arch Intern Med* 165: 742-748.
27. Griffin KA, Kramer H and Bidani AK (2008) Adverse renal consequences of obesity. *Am J Physiol Renal Physiol* 294: F685-F96.
28. Young DS (2000) Effects of drugs on clinical laboratory tests. (5<sup>th</sup> edition), AACC Press, USA.
29. Young DS (2001) Effects of disease on Clinical laboratory tests. (4<sup>th</sup> edition), AACC Press, USA.