

Assessment of Kidney Function Tests in Diabetes Mellitus Patients under Insulin Doses in Arar Region, Kingdom of Saudi Arabia

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*Article History:

Received: 02/03/2018

Revised: 19/03/2018

Accepted: 19/03/2018

DOI: <https://doi.org/10.7439/ijbar.v9i4.4674>

Abstract

Objectives: The aim of this work was to assess the kidney function tests in Diabetes Mellitus patients under insulin doses in Arar region

Introduction: Diabetes Mellitus (D.M) is a prevalent endocrinal disorder in Kingdom of Saudi Arabia. Present study is to highlight the effect of long lasting cases of controlled D.M on Kidney Functions in a local region. Diabetic renal disease in human often requires the better part of two decades to develop the signs and symptoms of advanced/clinical diabetic nephropathy with underlying extensive damage to the various segments of the nephron, thereby causing albuminuria, rising blood pressure, and a progressive decline of glomerular filtration.

Materials and Methods: The serum biochemistry investigation for Glucose, Urea and Creatinine.

1-Glucose: The fasting serum glucose level was obtained from the laboratory of Clinical Biochemistry using department Autoanalyser (Dimension model RXL MAX) of the Arar Central Hospital, Arar.

2-Urea: Urea Kit (Randox) and a spectrophotometer were used to determine the serum urea concentration in the samples.

3-Creatinine: Serum creatinine was measured by a Kit (Randox), using the compensated kinetic Jaffe assay.

Results & Discussions: It is clear from the result that in almost all patients blood glucose is within normal range but their kidney function test i.e. urea and creatinine lies in high range. Measurements of Blood pressure (SBP & DBP) also found to be in normal range for all patients. So obtained results could be interpreted as: The patients are controlled diabetics under insulin therapy but with very high serum creatinine level indicating severely affected glomerular filtration rate.

Conclusion: Based on this study it could be concluded that long duration of diabetes mellitus can affect renal function through destructive effects on vascular bed of renal tissues as a part of general vascular and nervous pathological effects of diabetes mellitus. These patients are advised to go for dialysis regularly and should be under observation of well qualified Nephrologists.

Keywords: SBD: Systolic Blood Pressure, DBD: Diastolic Blood Pressure, FBS: Fasting Blood Sugar, YOD: Years of Diabetes.

1. Purpose of the study

Diabetes Mellitus (D.M) is a prevalent endocrinal disorder in Kingdom of Saudi Arabia. Present study was to highlight the effect of long lasting cases of controlled D.M on Kidney functions in a local region of Arar. Diabetes mellitus now is one of the major health problems in the kingdom. Changes in the life styles and eating habits of the population are important factor in the increase of its prevalence which exceeded 23%. [1]

2. Introduction

The prevalence of diabetes mellitus (DM) in the Saudi population is high and 90% of diabetics suffer from Type II DM [2]. An epidemiological study of Saudi subjects aged 15 years or older, from different regions of the kingdom found that the age-adjusted prevalence of DM (using WHO criteria) was higher in urban areas (males 12%, females 14%) than rural areas (males 7%, females

7.7%) [2]. The highest prevalence was in urban females aged 51–60 years (49%).

Al-Nozha *et al* [1] found that the overall prevalence of DM in KSA was 23.7%. The prevalence in males and females were 26.2% and 21.5% ($p < 0.00001$). The calculated age-adjusted prevalence for Saudi population for the year 2000 is 21.9%. Diabetes mellitus was more prevalent among Saudis, living in urban areas of 25.5%

In the late 1970s both World Health Organization WHO [3] and the National Diabetes Data Group [4]; produced new diagnostic criteria and a new classification system for diabetes mellitus. This brought order to a chaotic situation in which nomenclature varied and diagnostic criteria showed enormous variations using different oral glucose loads. In 1985 WHO slightly modified their criteria to coincide more closely with the NDDG values [3]. There are now many data available, and also much more etiological information has appeared. It seemed timely to re-examine the issues and to update and redefine both the classification and the criteria, and to include a definition of the "Metabolic Syndrome".

Diabetes Mellitus describes a metabolic disorder of multiple etiology characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The effects of diabetes mellitus include long-term damage, dysfunction and failure [5]. It is recommended that the terms "insulin-dependent diabetes mellitus" and "non-insulin-dependent diabetes mellitus" and their acronyms "IDDM" and "NIDDM" no longer be used. These terms have been confusing and frequently resulted in patients being classified based on treatment rather than on pathogenesis [6].

Diabetes mellitus Type 1:

The etiological type named Type 1 encompasses the majority of cases which are primarily due to pancreatic islet beta-cell destruction and are prone to ketoacidosis. Type 1 includes those cases attributable to an autoimmune process, as well as those with beta-cell destruction and who are prone to ketoacidosis for which neither an etiology nor a pathogenesis is known (idiopathic). It does not include those forms of beta-cell destruction or failure to which specific causes can be assigned (e.g. cystic fibrosis, mitochondrial defects, etc.). Some subjects with this type can be identified at earlier clinical stages than "diabetes mellitus" [7].

Diabetes mellitus Type 2:

The type named Type 2 includes the common major form of diabetes which results from defect(s) in insulin secretion, almost always with a major contribution from insulin resistance. It has been argued that a lean phenotype of Type 2 diabetes mellitus in adults found in the Indian sub-continent may be very distinct from the more

characteristic form of Type 2 found in Caucasians. Not enough information is available, however, to characterize such subjects separately.

Signs, symptoms & Pathogenesis: The classical triad of diabetes symptoms is polyuria, polydipsia and polyphagia, which are respectively, frequent urination, increased thirst and consequent increased fluid intake and increased appetite. Symptoms may develop quite rapidly (weeks or months) in type 1 diabetes, particularly in children. However, in type 2 diabetes the symptoms develop much more slowly and may be subtle or completely absent. Type 1 diabetes may also cause a rapid yet significant weight loss (despite normal or even increased eating) and irreducible fatigue. All of these symptoms except weight loss can also manifest in type 2 diabetes in patients whose diabetes is poorly controlled [8].

When the glucose concentration in the blood is raised beyond the renal threshold, reabsorption of glucose in the proximal renal tubuli is incomplete, and part of the glucose remains in the urine (glycosuria). This increases the osmotic pressure of the urine and inhibits the reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume will be replaced osmotically from water held in body cells, causing dehydration and increased thirst. Prolonged high blood glucose causes glucose absorption, which leads to changes in the shape of the lenses of the eyes, resulting in vision changes. Blurred vision is a common complaint leading to a diabetes diagnosis. Type 1 should always be suspected in cases of rapid vision change whereas type 2 is generally more gradual but should still be suspected [8].

Diagnosis: The requirements for diagnostic confirmation for a person presenting with severe symptoms and gross hyperglycaemia differ from those for the asymptomatic person with blood glucose values found to be just above the diagnostic cut-off value. Severe hyperglycaemia detected under conditions of acute infective, traumatic, circulatory or other stress may be transitory and should not in itself be regarded as diagnostic of diabetes. The diagnosis of diabetes in an asymptomatic subject should never be made on the basis of a single abnormal blood glucose value. For the asymptomatic person, at least one additional plasma/blood glucose test result with a value in the diabetic range is essential, either fasting from a random (casual) sample or from the oral glucose tolerance test (OGTT).[9]

Effect of Diabetes Mellitus on Renal Function:

Diabetic renal disease in human often requires the better part of two decades to develop the signs and symptoms of advanced/clinical diabetic nephropathy with underlying extensive damage to the various segments of the nephron, thereby causing albuminuria, rising blood

pressure, and a progressive decline of glomerular filtration [10]

In the past, this stage has usually been classified as one of inexorable progression leading to renal failure, dialysis, transplantation, or death [10], with the patient more likely to die of macrovascular disease than renal disease [10], at the Steno Diabetes Centre in Gentofte, Denmark, contribute relevant observations to these issues with a summary of their long-term monitoring of the advanced stages of diabetic nephropathy, combined with their careful observational assessment of the efficacy of antihypertensive agents in altering its course [11].

3. Materials and Methods

3.1 Blood Samples were collected of Diabetes Mellitus patients in different age group from Arar Central Hospital -Centre for Diabetic Patients.

Adults Males: 8 (aged 19 – 74 yrs)

Adult Females: 7 (aged 21 – 72 yrs)

The serum biochemistry examination for Glucose, Urea and Creatinine.

Glucose: The fasting serum glucose level were obtained from the laboratory of Clinical Biochemistry using department Autoanalyser (Dimension model RXL MAX) of the Arar Central Hospital, Arar (Normal values : 70-110 mg/dl).

Urea: Urea Kit (Randox) and a spectrophotometer were used to determine the serum urea concentration in the samples (Normal values: Serum (urea) 10 – 50 mg / dl).

Creatinine: Serum creatinine was measured by a Kit (Randox), using the compensated kinetic Jaffe assay (Normal values: Men 0.6 – 1.1 mg/dl, Women 0.5 – 0.9 mg/dl).

4. Results & Discussions

Obtained results are expressed in Table 1 and Figures 1, 2, 3 & 4.

It is clear from the result that in almost all patients blood glucose is within normal range but their kidney function test i.e. urea and creatinine lies in high range (abnormal). Measurements of Blood pressure (SBP & DBP) also found to be in normal range for all patients.

So obtained results could be interpreted as:

- The patients are controlled diabetics under insulin therapy but with very high serum creatinine level indicating severely affected glomerular filtration rate.
- The patient's history revealed more than ten years duration of diabetes mellitus. They are diagnosed as diabetic patients and were under insulin therapy for controlling their blood sugar level during this period.
- From history of patients there is no other direct cause of renal problem.

Table 1: Results of Urea, Creatinine, Fasting Blood Sugar and Age of the Diabetes patients under Insulin doses versus Gender among study population

Parameter	Gender	Mean (SD)	SEM
Urea (mg/dl)	Male	80.9 (10.56)	3.70
	Female	81 (5.71)	2.20
Creatinine (mg/dl)	Male	8.5(0.85)	0.30
	Female	8.0(0.45)	0.17
FBS (mg/dl)	Male	111.0(8.04)	2.84
	Female	117.7(3.04)	1.15
Age (year)	Male	50.8 (3.9)	1.4
	Female	60.8 (1.8)	0.6

Key: SD: Standard Deviation; SEM: Standard error of the mean; FBS: Fasting Blood Sugar.

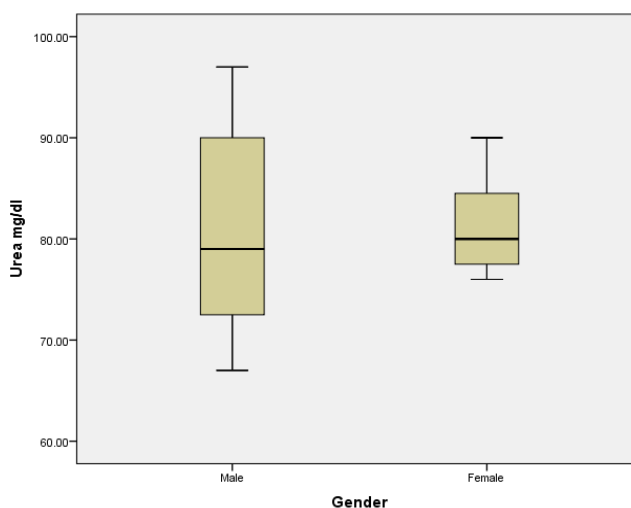


Figure 1: Results of Urea (mg/dl) Vs Gender in study population

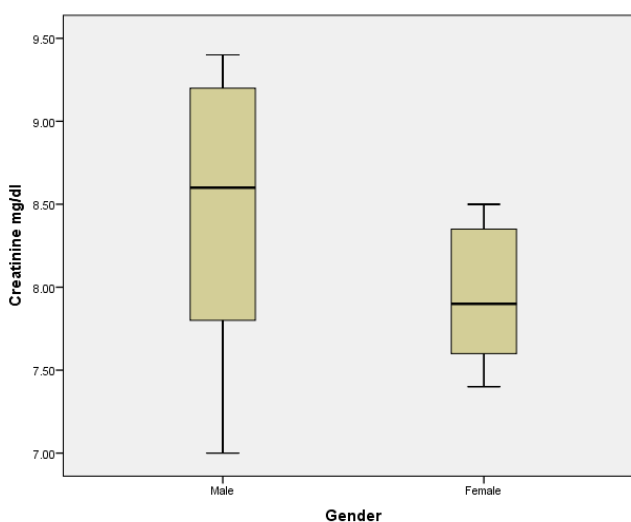


Figure 2: Results of Creatinine (mg/dl) Vs Gender in study population

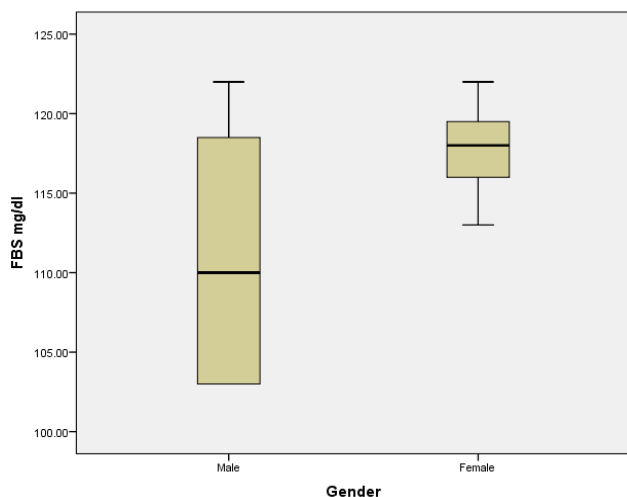


Figure 3: Results of Fasting Blood Sugar FBS (mg/dl) Vs Gender in study population

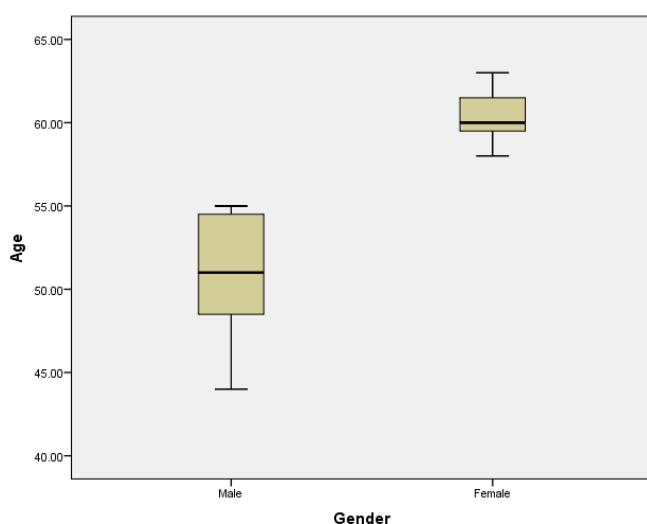


Figure 4: Results of Age Vs Gender in study population

5. Conclusion

On the basis of finding in this study it could be concluded that:

- Long duration of diabetes mellitus can affect renal function through destructive effects on vascular bed of renal tissues as a part of general vascular and nervous pathological effects of diabetes mellitus.
- These patients are advised to go for dialysis regularly and should be under observation of a well qualified Nephrologist.

References

- [1]. Al-Nozha MM, Al-Maatouq MA, Al-Mazrou YY, Al-Harhi SS, Arafah MR, Khalil MZ, Khan NB, Al-Khadra A, Al-Marzouki K, Nouh MS, Abdullah M. Diabetes mellitus in Saudi Arabia. *Saudi Medical Journal*. 2004; 25(11):1603-10.
- [2]. Al-Khader, A.A. (2001) Impact of Diabetes in Renal Diseases in Saudi Arabia. *Nephrology Dialysis Transplantation*, 16, 2132-2135. <http://dx.doi.org/10.1093/ndt/16.11.2132>.
- [3]. WHO. Expert Committee on Diabetes Mellitus. *Second Report*. Geneva: WHO, WHO Expert Committee on Diabetes Mellitus. *Technical Report Series* 1970; 646.
- [4]. Chronic kidney disease, NICE Clinical Guideline (September 2008); Early identification and management of chronic kidney disease in adults in The Renal Association (UK) website primary and secondary care.
- [5]. Alberti KGMM, Zimmet PZ for the WHO Consultation. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO Consultation. *Diabetic Medicine* 1998; 15: 539-553.
- [6]. Melbak AG, Christau B, Marner B, Borch-Johnsen K, Nerup J. Incidence of insulin-dependent diabetes mellitus in age groups over 30 years in Denmark. *Diabetes Med* 1994; 11: 650.
- [7]. Betterle C, Zanette F, Pedini B, Presotto F, Rapp LB, Monsciotti CM *et al*. Clinical and subclinical organ-specific autoimmune manifestations in type 1 (insulin-dependent) diabetic patients and their first-degree relatives. *Diabetologia* 1983; 26: 431.
- [8]. Levey AS, Coresh J, Balk E, *et al*. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Ann Intern Med* 2003; 139:137-47
- [9]. McCance DR, Hanson RL, Pettitt DJ, Bennett PH, Hadden DR, Knowler WC. Diagnosing diabetes mellitus - do we need new criteria? *Diabetologia* 1997; 40: 247-55.
- [10]. Kusmann MJ, Goldstein HH & Gleason RE. The clinical course of diabetic nephropathy. *JAMA* 1976; 238: 1861-1863.
- [11]. Hovind P, Rossing P & Tarnow L *et al*. Remission and regression in the nephropathy of type 1 diabetes when blood pressure is controlled aggressively. *Kidney Int* 2001; 60: 277-283.