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ORIGINAL ARTICLE

Assessment of GFR among diabetic patients: A case control study

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ABSTRACT:
Background: The current research aimed for assessing GFR among diabetic patients. **Materials & methods:** The current research was conducted for evaluating GFR among diabetic patients. A total of 50 diabetic patients were enrolled. GFR analysis was done. Another set of 50 healthy subjects were enrolled as controls. All data were initially entered in Microsoft Excel and subsequently exported to SPSS for statistical evaluation. **Results:** The glomerular filtration rate (GFR) differed significantly between the diabetic and control groups. The mean GFR in the diabetic cohort was 143.8 ± 23.9 mL/min/1.73m², which was notably higher compared to 112.9 ± 5.9 mL/min/1.73m² observed in the control group. This difference was statistically significant, with a p-value of 0.002. **Conclusion:**Glomerular filtration rate (GFR) is significantly altered in individuals with diabetes mellitus, reflecting early renal hemodynamic changes and progressive diabetic nephropathy.
Key words: Glomerular filtration rate, diabetes

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INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder that significantly compromises quality of life due to its progressive nature and potential for severe systemic complications. The World Health Organization (WHO) has raised concern over the rapid global escalation in diabetes prevalence. According to estimates by Shaw et al., the global prevalence of diabetes among adults aged 20–79 years was 6.4% (285 million) in 2010, with projections indicating an increase to 7.7% (439 million) by 2030.[1-3]

The disease poses a critical public health challenge due to its high morbidity and mortality rates.In individuals with type 2 diabetes mellitus, insulin resistance in peripheral tissues leads to a compensatory increase in insulin demand. However, this heightened demand cannot be sustained over time due to progressive dysfunction of pancreatic β-cells, which exhibit impaired insulin secretion despite persistent hyperglycemia. As the disease advances, β-cell function deteriorates further, resulting in declining endogenous insulin production[57]. This progressive β-cell failure may eventually necessitate exogenous insulin therapy, causing a subset of previously non–insulin-dependent patients to become functionally insulin-dependent. Nonetheless, the majority of individuals with type 2 diabetes retain partial β-cell function, and complete insulin depletion is uncommon. This distinguishes type 2 diabetes from type 1 diabetes, wherein patients exhibit absolute insulin deficiency due to autoimmune-mediated β-cell destruction. Additionally, ketoacidosis, a hallmark of type 1 diabetes, is rarely observed in type 2 diabetes, further highlighting the pathophysiological divergence between the two forms of the disease.[4-6]Hence; the current research aimed for assessing GFR among diabetic patients.

MATERIALS & METHODS

The current research was conducted for evaluating GFR among diabetic patients. A total of 50diabetic patients were enrolled. Eligible participants exhibited hyperglycemia, defined as random blood glucose ≥300 mg/dL. Clinical data—including age, sex, height, and weight—were recorded using a standardized case report form. Random blood glucose was assessed using a glucometer, while blood and urine specimens were collected for glomerular filtration rate (GFR) analysis. In the laboratory, plasma creatinine (Pcr) and urine creatinine (Ucr) concentrations were measured using the spectrophotometric method. The estimated GFR (eGFR) was calculated via the creatinine clearance (Ccr) formula. Another set of 50 healthy subjects were enrolled as controls. All data were initially entered in Microsoft Excel and subsequently exported to SPSS for statistical evaluation. Paired sample t-tests were used to compare GFR and random blood glucose levels, with statistical significance set at $p < 0.05$ and a 95% confidence interval.

RESULTS

A total of 50 participants were included in each group, comprising diabetic patients and age-matched non-diabetic controls. In the diabetic cohort, 34% (n = 17) were aged below 40 years, while 66% (n = 33) were 40 years or older. Similarly, in the control group, 30% (n = 15) were under 40 years of age, and 70% (n = 35) were aged above 40. The difference in age distribution between the groups was not statistically significant (p = 0.225). Regarding sex distribution, 58% (n = 29) of the diabetic participants were male, and 42% (n = 21) were female, compared to 62% (n = 31) male and 38% (n = 19) female in the control group. This variation also lacked statistical significance (p = 0.162), indicating comparable demographic profiles between the diabetic and non-diabetic populations in terms of age and sex.The glomerular filtration rate (GFR) differed significantly between the diabetic and control groups. The mean GFR in the diabetic cohort was 143.8 ± 23.9 mL/min/1.73m², which was notably higher compared to 112.9 ± 5.9 mL/min/1.73m² observed in the control group. This difference was statistically significant, with a p-value of 0.002, indicating a potential state of hyperfiltration commonly observed in the early stages of diabetic nephropathy.

Table 1: Demographic data

Demographic data	Diabetic group		Control group		p-value
	Number	Percentage	Number	Percentage	
Age of less than 40 years	17	34	15	30	0.225
Age of more 40 years	33	66	35	70	
Males	29	58	31	62	0.162
Females	21	42	19	38	

Table 2: Comparison of GFR

GFR (mL/min/1.73m^2)	Diabetic group	Control group
Mean	143.8	112.9
SD	23.9	5.9
p-value	0.002 (Significant)	

DISCUSSION

Diabetic nephropathy is the leading cause of kidney disease in patients starting renal replacement therapy and affects approximately 40% of type 1 and type 2 diabetic patients. It increases the risk of death, mainly from cardiovascular causes, and is defined by increased urinary albumin excretion (UAE) in the absence of other renal diseases. Diabetic nephropathy is categorized into stages: microalbuminuria (UAE >20 microg/min and < or =199 microg/min) and macroalbuminuria (UAE > or =200 microg/min). Hyperglycemia, increased blood pressure levels, and genetic predisposition are the main risk factors for the development of diabetic nephropathy.[6- 10]Hence; the current research aimed for assessing GFR among diabetic patients.

A total of 50 participants were enrolled in both the diabetic and control groups. Age and sex distributions were comparable between groups, with no statistically significant differences (p = 0.225 and p = 0.162, respectively). However, the mean glomerular filtration rate (GFR) was significantly elevated in the diabetic group (143.8 ± 23.9 mL/min/1.73m²) compared to controls (112.9 ± 5.9 mL/min/1.73m²; p = 0.002), suggesting early glomerular hyperfiltration associated with diabetic nephropathy.Meeme AK et al assessed the effect of glucose lowering on GFR in diabetic patients admitted for short term therapy at Mulago hospital. Glomerular filtration rate was measured using creatinine clearance in 40 type I and II diabetes mellitus patients admitted on the basis of a glucometer reading of RBS ≥ 300mg/dl both at admission and discharge. Data was analyzed using a paired sample t-test and p-value set at 0.05 (95% CI). A total of 40 patients were assessed. Their mean age was 49.8years. 82.5% of them had type II DM while only 17.5% had type I DM. The average duration of disease was 7years. GFR was found to decrease from 157.4ml/min on admission to 86.4ml/min at discharge (p=0.001). Random blood sugar levels decreased from 332.2mg/dl on admission to 119.8mg/dl at discharge (p=0.000). Results imply that glycaemic control lowers the GFR in diabetic patients admitted for short term treatment. A reduction in GFR reflects reduction of hyperfiltration, a process that starts diabetic nephropathy.[10]Hovind P et al measured GFR with 51Cr-EDTA plasma clearance technique, blood pressure, albuminuria, glycosylated hemoglobinA1c, and serum cholesterol every year for seven years (range 3 to 14 years) in 301 consecutive type 1 diabetic patients with diabetic nephropathy recruited consecutively during 1983 through 1997. Diabetic nephropathy was diagnosed clinically if the following criteria were fulfilled: persistent albuminuria> 200 μg/min, presence of diabetic retinopathy, and no evidence of other kidney or renal tract disease. In total, 271 patients received antihypertensive treatment at the end of the observation period. Mean arterial blood pressure was 102 ± 0.4 (SE)mm Hg. The average decline in GFR was 4.0 ± 0.2 mL/min/year and even lower (1.9 ± 0.5 mL/min/year) in the 30 persistently normotensive patients, none of whom had ever received antihypertensive treatment (P < 0.01). A multiple linear regression analysis revealed a significant positive correlation between the decline in GFR and mean arterial blood pressure, albuminuria, glycosylated hemoglobin A1c, and serum cholesterol during follow-up = 0.29, P ≤ 0.001). No threshold level for blood pressure, glycosylated hemoglobinA1c, or serum cholesterol was demonstrated. A two-hit model with mean arterial blood pressure and glycosylated hemoglobin A1c below and above the median values (102mm Hg and 9.2%, respectively) revealed a rate of decline in GFR of only 1.5 mL/min/year in the lowest stratum compared with 6.1 mL/min/year in the highest stratum (P < 0.001).The prognosis of diabetic nephropathy has improved during the past decades, predominantly because of effective antihypertensive treatment. Genuine normotensive patients have a slow progression of nephropathy.[11]

CONCLUSION

Glomerular filtration rate (GFR) is significantly altered in individuals with diabetes mellitus, reflecting early renal hemodynamic changes and progressive diabetic nephropathy.

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