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Clinical and Biochemical Indicators Associated with Impaired Kidney Function in Children with Type 1 Diabetes: A Case-Control Study

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Abstract **Background:** Kidney failure is the leading cause of early death for diabetic patients and is regarded as the most dangerous consequence of the disease. This can only be reversed through early detection and intervention. The incidence of type 1 diabetes, which comprises 5-10 per cent of all diabetic diseases, is rising across the globe with a rate of 15 per 100,000 people per year. **Methods:** 90 samples were collected and used in this study. Thirty healthy children served as controls (aged 5-15 years), 30 patients with Type 1 Diabetes Mellitus (T1DM, aged 5-15 years) and 30 patients with type 1 diabetic nephropathy (DN), aged 5-20 years). **Results:** highly significant increase in Urea and Creatinine levels when compared control with DN also between T1DM with DN. Duration Showing highly substantial ($p<0.01$) when compared T1DM with DN, fasting serum glucose and HbA1c highly significantly increased when comparing the Controls group with T1DM and DN also between T1DM with DN in Fasting blood sugar Showing significant ($p<0.05$) as well as with HbA1c Showing no significant ($p>0.05$), as well as the age showing substantial increase when compared control with DN and T1DM with DN ($p<0.05$), **Conclusion:** People with type 1 diabetes have an alarming risk of developing end-stage renal disease, Kidney disease is an important clinical issue among type 1 diabetes patients. This is because early intervention and detection are vital to preventing kidney failure.

Key Words Type 1 Diabetes Mellitus, Diabetic Nephropathy, Chronic Kidney Disease

INTRODUCTION

Kidney involvement is more likely to occur in children with Type 1 Diabetes Mellitus (T1DM). At the onset of type 1 diabetes, a portion of the risk is established. Indeed, up to 65% of kids with type 1 diabetes may suffer from Acute Kidney Injury (AKI), which increases the risk of developing Chronic Kidney Disease (CKD) later. The other part of the risk, which arises throughout the following course of type 1 diabetes, may be associated with poor glycemic control and the subsequent development of diabetic kidney disease [1]. AKI, tubular damage and DN are among the acute and chronic kidney involvement symptoms that children with Type 1 Diabetes Mellitus (T1DM) may experience [2-7]. In cases of recurrent diabetic ketoacidosis DKA episodes, the prevalence of AKI has been found to increase further, reaching 81% [8,9]. Additionally, 15-20% of T1DM patients may eventually develop DN, which, in susceptible patients, typically begins shortly after disease onset and may worsen during adolescence [5]. For these reasons, to prevent the development of CKD and the potential progression towards kidney failure in the future, kidney health for children with type 1 diabetes should not be neglected at any stage of the illness. DN, which has a high risk of dialysis and death, is one of the leading causes of CKD [10]. Due to the rising prevalence of type 1 and type 2 diabetes in children and adolescents, DN is a significant public health concern [11,12]. The thickening of the glomerular basement membrane is one of the first structural changes in the kidney to become noticeable within 1.5 to 2 years of diagnosis with type 1 diabetes. After a diagnosis of diabetes, mesangial volume expansion can be seen 5-7 years later and increased albuminuria may follow [13]. Tubular-interstitial injury may be the initial change in DN, despite DN being classified as a glomerular disease [14]. Blood urea, creatinine, HbA1c and Random Blood Sugar (RBS) levels were measured.

Pathophysiology Type 1 Diabetes

The destruction of the pancreatic islet cells that produce insulin is a hallmark of type 1 diabetes, a chronic autoimmune disease. The ensuing insulin inadequacy interferes with the homeostasis of glucose and instigates the clinical syndrome of diabetes. The most common endocrine and metabolic disease in childhood and adolescence is type 1 diabetes [15]. It has close relations with an increased risk of late-onset chronic complications that cause high morbidity and mortality [16]. One of the leading causes of morbidity and death for people with Type 1 Diabetes Mellitus (T1DM), a chronic autoimmune disease characterized by an absolute lack of insulin, is kidney failure. It is caused by observations of T-cell-mediated selective killing of insulin-producing pancreatic beta cells in predisposed individuals. Despite decades of research aimed at delaying or preventing the development of beta-cell autoimmunity, T1DM continues to increase worldwide, with a current annual rate of 3-4%. Around the world, reported prevalence varies, from 0.1 per 1,000 in China and Venezuela to 12-40 per 1,000 in the United States and Finland. What is of interest to public health is that, in the absence of comorbid hypertension, the natural course of diabetic kidney disease is often associated with kidney failure, leading to a higher risk of cardiovascular mortality and low quality of life [17].

Long-Term Complications

DN is a severe microvascular complication of type 1 diabetes with the potential of causing eventual end-stage renal disease and hemodialysis or transplantation [18]. Patients with DN and type 1 diabetes are at high risk for cardiovascular and renal complications, including morbidity and death. In the last ten years, alternative interventions have been introduced with a view to reversing the progressive deterioration of renal function and avoiding hemodialysis or renal transplantation in DN patients. Insulin-dependent diabetes mellitus is associated with the development and progression of diabetic microvascular complications, which are prevented by intensive insulin therapy [19]. The Diabetes Control and Complications Trial (DCCT) is well known for its reduced risk of retinopathy and nephropathy in patients treated with intensive insulin therapy. Intensive glucose control leads to better microvascular outcomes but severe hypoglycemia and the risk of vascular events also occur. Thorough care can dramatically reduce the incidence of retinopathy, nephropathy and neuropathy, according to long-term follow-up studies. The main markers of cardiovascular and renal outcomes in individuals with diabetes mellitus are albuminuria and kidney function [20].

Objectives

This study assessed the relationship between T1DM and DN by analysing biochemical data from Roche Laboratories diagnostic kits using multivariate methods. People with type 1 diabetes have an alarming risk of developing end-stage renal disease. Kidney disease is an important clinical issue among type 1 diabetes patients. This is because early intervention and detection play a vital part in kidney failure prevention.

METHODS

Study Design

From November 2024 to February 2025, a cross-sectional study was conducted at the Department of Biochemistry at AL-Karama Education Hospital, Baghdad, Iraq. A total of 90 samples were collected and used in this investigation. Thirty healthy children served as controls (C, aged 5-15 years), 30 patients with T1DM (aged 5-15 years) and 30 patients with DN (aged 5-20 years). All study participants underwent an annual medical examination at Al-Karama Teaching Hospital. Those enrolled were those diagnosed with type 1 diabetes and its complications as defined by the World Health Organisation diagnostic criteria (DN).

Sample Collection and Biochemical Analysis

The study included children aged 5-20 years from AL-Karama Educational Hospital in Baghdad. Blood samples were collected from 30 children with T1DM, 30 children with DN and 30 healthy children as control samples. To measure RBS, blood urea, creatinine and HbA1c levels, 5 mL of blood was drawn from the brachial vein and placed into two tubes. The first tube contained the anticoagulant Ethylenediaminetetraacetic acid to maintain sample quality and prevent clotting. Approximately 2 mL of blood was collected in EDTA tubes for direct HbA1c measurement. After collection, the specimens were gently inverted at least five times to ensure proper mixing. The measurement was conducted immediately or the sample was stored in a refrigerator at 2-8°C for 3-7 days. The leftover blood was transferred to serum gel separator tubes and left to clot for 30 minutes at 18-25 degrees Celsius. Tubes were centrifuged for 10 minutes at 18-25°C at 1500 g to calculate the other parameters, including RBS, serum blood urea and serum creatinine (Cr). After sample collection, they were immediately transported to the laboratory for analysis using spectrophotometric techniques to obtain accurate measurements. Advanced hematological equipment was used to determine HbA1c. Additionally, personal and medical data of the participating children, including medical history and overall health status, were recorded to ensure accurate and comprehensive results.

Statistical Analysis

Data were analysed using SPSS software (SPSS 26.0; IBM Inc., Chicago, Illinois, USA). The Kolmogorov-Smirnov test was used to divide variables between research groups. One-way analysis of variance (ANOVA) was used to calculate and compare means and standard deviations

(Mean±Standard deviation) for homogeneously distributed variables. The p-values less than 0.05 are used to determine statistical significance. Statistical results were expressed as Highly Significant (HS), Not Significant (NS) and Standard Deviation (SD).

RESULTS

Table 1 shows that fasting serum glucose and HbA1c levels were significantly higher in the C group compared with T1DM and DN and between DM and DN in FBS, with fasting serum glucose ($p<0.05$) and HbA1c ($p>0.05$) showing no significant difference. BMI showed no significant difference across all groups and age increased significantly compared with the control group with DN and T1DM with DN ($p<0.05$) but not compared with the control group with DM ($p = 0.05$).

Table 2 shows that the levels of Urea and Creatinine are not significantly different between the C group and T1DM but show a highly significant increase between Control and DN, as well as between T1DM and DN. Duration showed a highly significant difference ($p<0.01$) compared with T1DM with DN.

Table 3 shows a strong relationship between HbA1c and fasting blood sugar and urea, as well as a strong relationship with creatinine and no significant increase with age. Also, no relationship with BMI was observed; HbA1c increased with increasing age and other parameters, except for BMI (Figure 1). Table 4 shows a strong relationship between urea and fasting blood sugar and HbA1c, as well as with creatinine and age; no relationship was observed with BMI, as urea increased with all parameters except BMI (Figure 2).

Table 5 shows a strong relationship between creatinine and fasting blood sugar and HbA1c, as well as with urea and age; no relationship was demonstrated with BMI.

DISCUSSION

The results in Table 1 align with research indicating that DN is a multifaceted condition involving interconnected mechanisms, primarily influenced by hyperglycemia [21]. The following are the most common primary diseases that lead to end-Stage Renal Disease (ESRD) and CKD:

Table 1: The Mean±SD for FBG, HbA1c, Age and Body Mass Index, Among Different Groups (n = 90)

Parameters	C (30) (Mean±SD)	DM (30) (Mean±SD)	DN (30) (Mean±SD)	Groups	p-value
FBG (mg/dl)	97.06±12.86	250.63±104.90	306.34±90.64	C*T1DM	0.000**
				C*DN	0.000**
				DM*DN	0.027
HbA1c %	5.17±0.47	11.04±1.77	10.90±2.181	C*DM	0.000**
				C*DN	0.000**
				DM*DN	0.981
BMI (Kg/m^2)	17.95±4.54	17.43±2.709	18.41±1.55	C*DM	0.896
				C*DN	0.925
				DM*DN	0.562
Age	11.93±4.891	12.21±4.66	15.17±4.11	C*DM	0.993
				C*DN	0.023
				DM*DN	0.044

**p<0.01 is extremely significant, *p<0.05 is significant and p>0.05 is no significant, C stands for control, T1DM for Type 1 diabetic mellitus and DN for diabetic nephropathy

Table 2: The Mean±SD for Urea, Creatinine and Duration Among Different Groups

Parameters	C (30) (Mean±SD)	DM (30) (Mean±SD)	DN (30) (Mean±SD)	Groups	p-value
Urea	23.76±6.36	28.66±7.28	131.34±41.04	C*T1DM	0.815
				C*DN	0.000
				T1DM*DN	0.000
Creatinine	0.77±0.206	0.83±0.26	6.492±1.60	C*T1DM	0.991
				C*DN	0.000
				T1DM*DN	0.000
Duration		7.23±3.45	6.82±2.67	T1DM*DN	0.000

**p<0.01 is extremely significant, *p<0.05 is significant and p>0.05 is no significant, C stands for control, T1DM for Type 1 diabetic mellitus and DN for diabetic nephropathy

Table 3: The Pearson Correlation of HbA1c Activity with Other Studied Parameters

	HbA1C		
	DM group (30)		
Parameters	R	P	Sig.
FBG (mg/dl)	0.880**	0	HS
HbA1c (%)	1	-	HS
BMI (kg/m^2)	-0.149	0.164	NS
Urea	0.471**	0.006	HS
Creatinine	0.420**	0	HS
Age	-0.021	0.847	NS

**p<0.01 is highly significant (HS), *p<0.05 is significant (S) and p>0.05 is NS, C stands for control, T1DM for Type 1 diabetic mellitus and DN for diabetic nephropathy

Table 4: The Pearson Correlation of Urea with Other Studied Parameters

Parameters	Urea		
	DM group (30)		
	R	P	Sig.
FBG (mg/dl)	0.444**	0.000	HS
HbA1c (%)	0.395**	0.000	HS
BMI (kg/m2)	0.108	0.315	NS
Urea	1	-	NS
Creatinine	0.959**	0.000	HS
Age	0.291**	0.006	HS

**p<0.01 is highly significant (HS), *p<0.05 is significant (S) and p>0.05 is NS, C stands for control, T1DM for Type 1 diabetic mellitus and DN for diabetic nephropathy

Table 5: The Pearson Correlation of Creatinine with Other Studied Parameters

Parameters	Creatinine		
	DM group (n = 32)		
	R	P	Sig.
FBG (mg/dl)	0.494**	0.000	HS
HbA1c (%)	0.420**	0.000	HS
BMI (kg/m2)	0.100	0 .353	NS
Urea	0.959**	0.000	HS
Creatinine	1	-	HS
Age, year	0.299**	0.000	HS

**p<0.01 is highly significant (HS), *p<0.05 is significant (S) and p>0.05 is NS, C stands for control, T1DM for Type 1 diabetic mellitus and DN for diabetic nephropathy

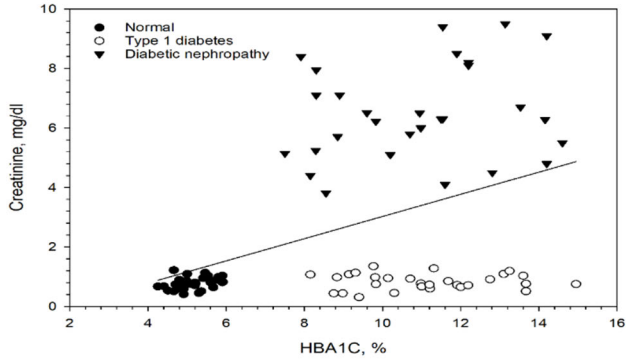

Figure 1: Correlation between HbA1c and Creatinine

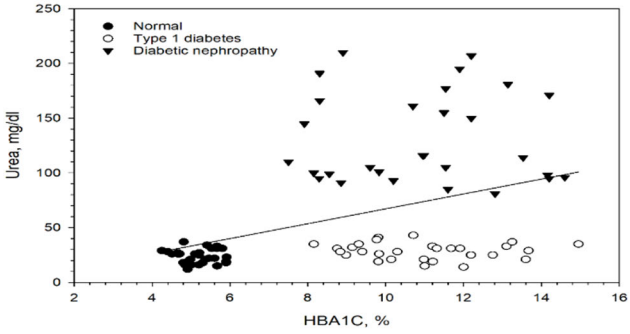

Figure 2 Correlation between HbA1c and Urea

- Diabetes type 2 (30-50%)
- Diabetes type 1 (3.9%)
- High blood pressure (27.2%)
- 8.2% have primary glomerulonephritis
- 3.6% of patients have chronic tubulointerstitial nephritis
- Cystic or hereditary diseases (3.1%)
- Vasculitis or secondary glomerulonephritis (2.1%)
- Neoplasms or dyscrasias of plasma cells (2.1%)

In the US, less than 1% of patients with ESRD have sickle cell nephropathy [22]. Another reason could be that, like the general population, the number of obese T1DM patients is rising [23,24]. In addition to having a higher central fat distribution, people with T1DM who gain more weight also exhibit changes in blood pressure and lipid levels that resemble those observed in insulin resistance syndrome [25,26]. This could increase the risk of kidney disease and retinopathy [27]. The results in Table 2 are supported by research showing that hyperglycemia results in glomerular vascular permeability and abnormal blood flow homeostasis. The increased blood flow and intracapillary pressure eventually cause the efferent side of the glomerular capillaries to produce less nitric oxide, which has profibrotic effects and makes the capillaries more sensitive to angiotensin II. When hyperglycemia persists, the lesions become irreversible, even though the increased permeability may initially be reversible [4]. DN is schematically separated into five stages. The final stage of kidney failure, which is characterized by uremia and usually appears ten years after proteinuria, can occur in up to 40% of individuals with type 1 diabetes [2]. The greatest serum creatinine at T1DM onset/basal creatinine (HC/BC) ratio, which suggests a more severe AKI, was significantly associated with acute tubular necrosis [28].

Table 3 results are supported by the study that found that the risk of retinopathy was best explained by the estimated explained relative risk (R±SD) for each variable in the eGDR formula for HbA1c (0.0242±0.0049), followed by BMI (0.0012±0.0011) and hypertension (0.0006±0.0010). The corresponding explained relative risk for kidney disease was 0.0067±0.0059 for hypertension, 0.0389±0.0103 for HbA1c and 0.0163±0.0064 for BMI. A stand-in for insulin resistance is eGDR [29]. This nationwide observational study demonstrates that among young people with T1DM,

eGDR, a proxy for insulin resistance, is linked to an increased risk of kidney disease and retinopathy. Lower eGDR was associated with a higher risk of retinopathy. Lower eGDR also increased the risk of kidney disease but not for the lowest eGDR category (<4 mg/kg/min). eGDR has been validated as a tool for assessing insulin resistance in individuals with type 1 diabetes [30]. By evaluating the individual contributions of each variable in the model using Heller's formula, we found that HbA1c was most strongly linked to both kidney disease and retinopathy [31].

The results in Table 5 showed that creatinine increases with increasing values of the parameters, except for BMI. This supports research showing that elevated levels of calcium, phosphorus, ammonia, creatinine, decreased water reabsorption and elevated urate and oxalate salts in urine are among the substances associated with diabetic nephropathies. This thus worsens the disease's symptoms and increases its rate [32].

CONCLUSIONS

Numerous topics in the literature on kidney damage in type 1 diabetes reflect issues that both patients and medical professionals deal with. Epidemiological studies show that between 2005 and 2008, the number of kidney problems fell alarmingly but it has since plateaued. Patients frequently exhibit high rates of proteinuria and microalbuminuria, indicating the existence of early indicators of kidney impairment that cannot be disregarded. The results of early intervention strategies have shown that the prevalence and advancement rates of kidney disease in diabetics can be considerably reduced when combined with early therapy, eventually increasing the patients' quality of life and life expectancy.

Acknowledgement

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Ethical Considerations

Ethical Considerations This study received ethical approval from the Ethics Review Committee (ERC) at the University of Sumer, College of Medicine (Approval No. 2/8/2400) dated 18/11/2024. Informed consent was obtained from the legal guardians of all participating children and the study was conducted in accordance with the Declaration of Helsinki.

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