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ADULTS WITH TYPE 2 DIABETES MELLITUS AND RENAL INSUFFICIENCY IN ABSENCE OF ALBUMINURIA AND RETINOPATHY

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Abstract

Background:

The kidney damage caused by type 2 diabetes mellitus (T2DM) is more varied than that of type 1 diabetic mellitus (T1DM). People with type 2 diabetes don't always get classical diabetic glomerulosclerosis, which is marked by a drop in the glomerular filtration rate (GFR), albuminuria, and retinopathy. If the estimated glomerular filtration rate (eGFR) is less than 60 mL/min per 1.73 m^2 of body surface area (BSA), then the person has chronic renal insufficiency.

Patients and Methods: Based on the glomerular filtration rate, 22 people had both chronic renal insufficiency and type 2 diabetes mellitus, whereas 78 subjects did not have either condition. People ranging in age from 45 to 65 had their BMI, blood pressure, and medical history recorded. Excluding hypertension was the aim. We wanted to make sure there weren't any underlying issues, like heart failure or a drug past. A slit lamp was used to look at urine samples from people with type 2 diabetes mellitus and chronic renal insufficiency for retinopathy and the albumin/creatinine ratio.

Results: While 22% of the individuals without type 2 diabetes mellitus did not have CRI, 100% of those with the disease had it (n=100). Albuminuria affected 18.18% of CRI patients, and diabetic retinopathy affected 13.6 percent of patients (n=3). forty-two individuals. Not only did 22.72% (n=S) of patients with type 2 DM and CRI not have retinopathy, but they also did not have microalbuminuria or macroalbuminuria. Aim of the study How can we determine the CRI if albuminuria and retinopathy are not present? The study involves 100 individuals with type 2 diabetes mellitus.

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Keywords: albuminuria, retinopathy, type 2 diabetes mellitus, Renal Insufficiency.

INTRODUCTION

The percentage among patients with type 2 diabetes, chronic renal disease (CRI), diabetic retinopathy, and albuminuria was 45.45% (N=10). Excuse me! Type 2 diabetes is characterized by persistent high blood sugar and problems with the metabolism of carbohydrates, lipids, and proteins. Type 2 diabetes is one of many metabolic disorders that affect insulin secretion or action, or both. There is a wide range of immediate, intermediate, and late repercussions that affect every system and organ in the body caused by fasting and postprandial hyperglycemia. The following symptoms are associated with type I diabetes, which is more prevalent in adults: 1. Being diagnosed with diabetes before the age of 30; 2. The patient must have a lean body mass index (BMI) below 30. To initiate therapy, the patient needs to take insulin. An increased risk of further autoimmune disorders is present. On the flip side, these symptoms are more common in people who have type 2 diabetes: The average age at which type 2 diabetes begins to manifest in a person is thirty. Native Americans. East Asian American and Pacific Islander communities categorize Native Americans as 'FG' or 'GT.'. The patient has a family history of diabetes while pregnant, having children, or gaining more than 39 pounds (ca. 18 kg). The patient's blood pressure measurement, 2140/90 mm Hg, indicates a slight elevation. The patient's triglyceride level is >250 mg/dL (2.82 mmol/L), while their Joint International Normalized Level of Cholesterol is 05 mg/dL (0.90 mmol/L). Polycystic ovarian syndrome and acanthosis nigricans are conditions that can occur in people with a history of vascular disease.

Chronic renal disease: Diabetic nephropathy causes about 30% of instances of end-stage renal disease (ESRD) in developed countries. After 20–30% of individuals with type 2 diabetes and type 21 (M) already have it, the likelihood of developing long-term diabetes, often known as nephropathy, increases. Diabetic nephropathy (2c) can occur in people with hypertension, dyslipidemia, hyperglycemia, or who smoke. When diabetes is first diagnosed, microalbuminuria (20-200 mcg/min, 30-300 mg/24 h) often begins around ten to fifteen years later. Microalbuminuria tests should be performed on people with Type I diabetes five years after diagnosis and then annually thereafter. It is not always apparent how Type 2 diabetes develops; thus, testing is done for individuals with the diagnosis at first and then once a year thereafter (section 2d). If the macroalbuminuria level is >200 mcg/min or >300 mg/24 hours, a dipstick will typically provide a positive result. It is not likely that strict control of blood sugar will stop diabetic nephropathy from progressing to end-stage renal disease (ESRD). However, keeping blood pressure in a healthy range and eating protein in the recommended range of 0.6 to 0.8 g/kg per day will help. Once a patient receives a type 2 diabetes diagnosis, they should begin annual proteinuria screening. Microalbuminuria is as simple as collecting a urine sample at random and analyzing the albumin to creatinine ratio. This measurement strongly correlates with estimates of 24-hour urine protein levels.

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Retinal pathology: The age of the patient at diagnosis and the length of their diabetes are factors that are linked to the occurrence and severity of diabetic retinopathy. Approximately a quarter of those with type 2 diabetes may already be suffering from retinopathy. All diabetic patients should undergo an annual dilated funduscopic examination by an ophthalmologist upon diagnosis of type 2 diabetes mellitus. Although better blood sugar control will eventually improve retinopathy, it may temporarily worsen the condition. Diabetic retinopathy distinguishes between proliferative and non-proliferative stages. Minor bulges of blood vessels, blot hemorrhages, and cotton-wool patches can be seen in the retina in cases of diabetes that do not lead to the development of new blood vessels. Typically, it manifests itself sometime between the middle of the first and early second decades following a diagnosis. Low oxygen levels in the blood induce new blood vessels to form in the retina, a symptom known as proliferative diabetic retinopathy. Near the optic nerve and macula, new veins form; these veins are prone to bursting, which causes vitreous hemorrhage, fibrosis, and, in the long run, retinal detachment.

Diabetes-Related Kidney Disease Pathogenesis

Multiple pathogenic processes inside the kidney are driven by diabetes-induced hyperglycemia, which contributes to the complex pathophysiology of diabetic kidney disease (DKD). identified as oxidative stress, inflammation, hemodynamic stress, metabolic stress, and fibrotic stress as the primary factors contributing to this disease. Pathophysiology of DKD is controlled by pathways that happen in different parts of the kidney, such as the glomerular, vascular, tubular, and interstitium, and these parts constantly interact with each other. The renal glomeruli and tubules undergo changes in structure and function as a result of diabetes, which causes glomerulosclerosis, albuminuria, tubulointerstitial fibrosis, mesangial expansion, homogeneous thickening of the glomerular basement membrane (GBM), and a gradual decrease in renal function (Figure 1). 2.1. Alterations in Hemodynamics It's because of glomerular hyperfiltration that the glomerular filtration rate is higher. This is because diabetes makes the osmolarity inside the glomerular capillaries rise, the glomerular pressure rises, and the afferent arterioles get bigger [8,9]. These changes in blood flow speed up the progression of chronic kidney disease (CKD) and also start and activate many vasoactive systems in the kidneys [10, 11]. There are several of these pathways, such as the AGE-dependent polyol and renin-angiotensin-aldosterone system (RAAS), the PKC, and the pro-oxidant enzyme NADPH oxidases [12]. In particular, hyperfiltration happens when the pressure inside the glomeruli increases and angiotensin II, a key effector hormone of the RAAS [17–19], causes the efferent arteriole to narrow. Injury to the glomeruli and tubules occurs because of the rise in glomerular capillary pressure and glomerular filtration rate. Angiotensin II also plays a role in the main diseases of inflammation and fibrosis in DKD. It does this by increasing the expression of many mediators that cause inflammation and fibrosis. This can happen directly in cells or indirectly through barotrauma in glomerular capillaries [2]. Reabsorption of filtered glucose in the proximal tubules is another way the kidney maintains glucose homeostasis under physiological settings [13]. A protein called SGLT2 is involved in about 90% of glucose reabsorption. It is found on the inner membrane of cells in the proximal tubular epithelial system [1]. However, glycosuria is a common sign of diabetes due to increased glucose filtration through the glomeruli. In this case, the body has changed to replace the glucose that has been filtered out

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of the urine by making the proximal tubules more active and expressing SGLT2 [11]. However, increased glucose reabsorption exacerbates hyperglycemia. When SGLT2 activity goes up, proximal sodium reabsorption goes up. This means that less sodium gets delivered to the macula densa cells through the tubule-glomerular feedback mechanism. This, in turn, reduces adenosine production, a potent vasoconstrictor [11], which causes the afferent arteriole to vasodilate, worsening hyperfiltration. Modification of the Glomerular Filtration Barrier and Albuminuria One of the first signs of DKD development is albuminuria. Damage to the tightly controlled glomerular filtration barrier can make the filtration process less effective. This damage can be caused by oxidative stress, renal inflammation, fibrosis, and changes in blood flow, among other unhealthy conditions. An early sign of DKD is damage to the glomerular basement membrane, specifically to the podocytes that keep the structure of the filtration barrier strong [4]. Damage to podocytes can cause foot process effacement, podocyte detachment, and podocyte depletion, all of which can result in malfunctioning glomerular endothelial cells [5]. In cases where the glomeruli, which remove extra protein and albumin, get damaged, the protein leaks into the urine. This is known as proteinuria or albuminuria [3]. From 30 to 299 mg/g creatinine, microalbuminuria is initially observed in DKD, but as the disease advances, it will rise to 300 mg/g creatinine or more, known as macroalbuminuria [8]. This phenomenon is a characteristic of diabetic ketoacidosis, and studies in mice have demonstrated that diabetic mice had far more albuminuria than control mice that do not have diabetes [3].

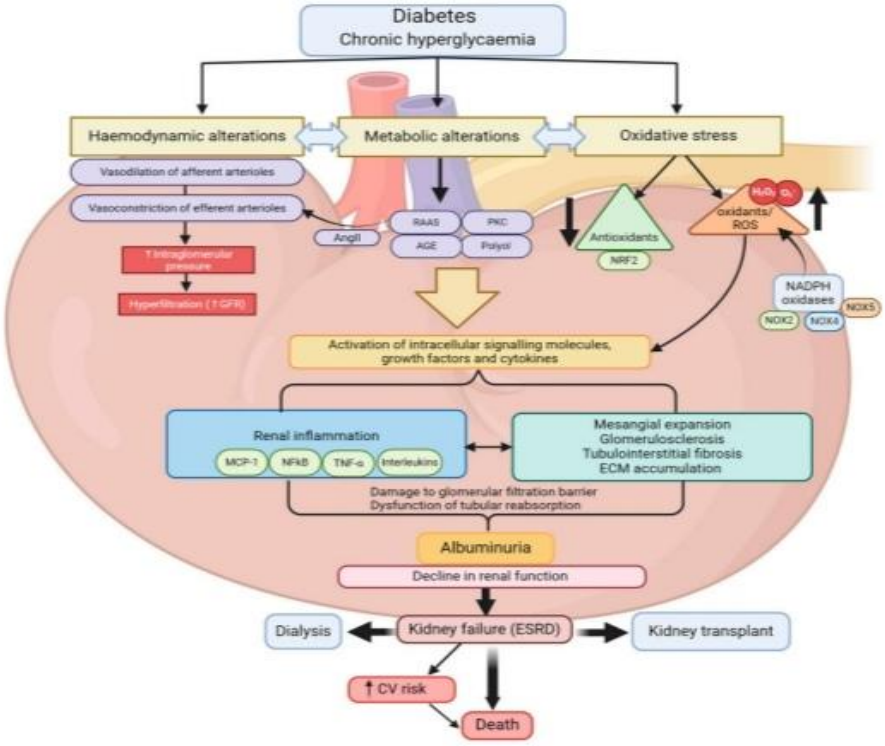

Figure 1. Schematic outlining the major pathways involved in the pathogenesis of DKD.

Methods:

The research period for this study was from October 1, 2012, to April 30, 2013, and one hundred people were eligible to participate. Twelve females out of twenty-two participants met the criteria set by the World Health Organization for type 2 diabetes. They had one of three blood sugar problems: (a) it was too high, causing symptoms like lack of hunger, genital itching, and overall weakness; (b) it was too high, with either a fasting level of 7 mmol/l or an unpredictable level of 11.1 mmol/l; or (c) it was different on two occasions, with either a fasting level of 7 mmol/l or an unpredictable level of 11.1 mmol/l, and the 2-hour value was equal to or higher than 11.1 mmol/l. 3) The glomerular filtration rate indicated the presence of chronic renal insufficiency. There was no chronic renal insufficiency in the 78 type 2 diabetic patients (46 male and 32 female). All parties involved consulted the endocrine clinic at Alkadimya Teaching Hospital for their assessment. For each patient, we estimated their body mass index (BMI) using the formula {weight (kg)/(length)^2 (m^2)} and the glomerular filtration rate (GFR) using the Cockcroft-Gault equation (2e). We determined their albumin to creatinine ratio (ACR) to be normal (4c) from the urine samples they provided by 2-Lab. A slit-lamp test confirmed diabetic retinopathy, a condition that primarily affects adults between the ages of 30 and 65 and is the leading cause of blindness in industrialized nations. One effective treatment option is retinal photocoagulation, which is most effective when done while the patient is usually asymptomatic. This finding shows how important it is to check the back of all diabetic patients' eyes regularly with their pupils dilated. Diabetic retinopathy has three symptoms in real life: it stops cells from dividing, it causes blood clots and microaneurysms, and it causes the hypercoagulation phase one to include the growth of new blood vessels. To be sure the patient didn't have hypertension, we looked at their medical history. Blood pressure readings above 140/90 are considered hypertension according to the British Hypertension Society. There are also things like the patient's medical history, other health problems they may have, and the medicines they take, like angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.

RESULTS:

Table 2 shows that a comparison was made between 78 persons without CRI and 22 adults with type 2 DM and CRI. Both the mean and standard deviation of systolic blood pressure were higher in the former group of individuals with type 2 DM and CRI compared to those without CRI. However, except for that, there was no difference between the two groups. Patients with type 2 diabetes mellitus have a mean disease duration of 6 years, 25% of whom use insulin, 50% of whom use diabetic oral hypoglycemic medicines, and 22% of whom have CRI. Albuminuria without retinopathy was found in 18.18% of cases (n=4), while diabetic retinopathy was present in 13.6% of cases with CRI. In a study of 15 people (22.72%), none of the patients with type 2 diabetes and chronic renal disease (CRI) exhibited retinopathy or albuminuria. Ten adults (45.8% of the total) with type 2 diabetes and chronic renal disease (CRI) exhibited albuminuria and diabetic retinopathy.

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Table :1

Quantification of proteinuria:(4c)

24-hr urine Protein	Alb./Creat. Ratio	Prot./creat. Ratio	Significance
<0.03 g	<3.5(female) <2.5(male)	_	Normal
0.03-0.3 g	~3.5 – 15	_	Microalbuminuria
0.3-0.5 g	~ 15- 50	~ 15-50	Dipsticks positive
>2.5 g	5- 200	>250	Glomerular disease More likely
>3 g	> 200	> 300	Nephrotic range-always glomerular disease

Estimation of GFR(Glomerular filtration rate):(2e)

$$\text{Creatinine clearance (mL/min)} = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{plasma creatinine (mg/dL)} \times 72}$$

(this value should be multiplied by 0.85 for women, since a lower fraction of the body weight is composed of muscle):We defined CRI as{ a GFR less than 60 mL/min per 1.73 m2 BSA}.

parameters	Subjects with Type 2DM with CRI 22. patients
With retinopathy and albuminuria	10 (45.5%)
Microalbuminuria Without retinopathy	3 (13.6%)
Macroalbuminuria Without retinopathy	1 (4.5%)
Retinopathy without albuminuria	3 (13.6%)
No retinopathy and albuminuria	5 (22.72%)

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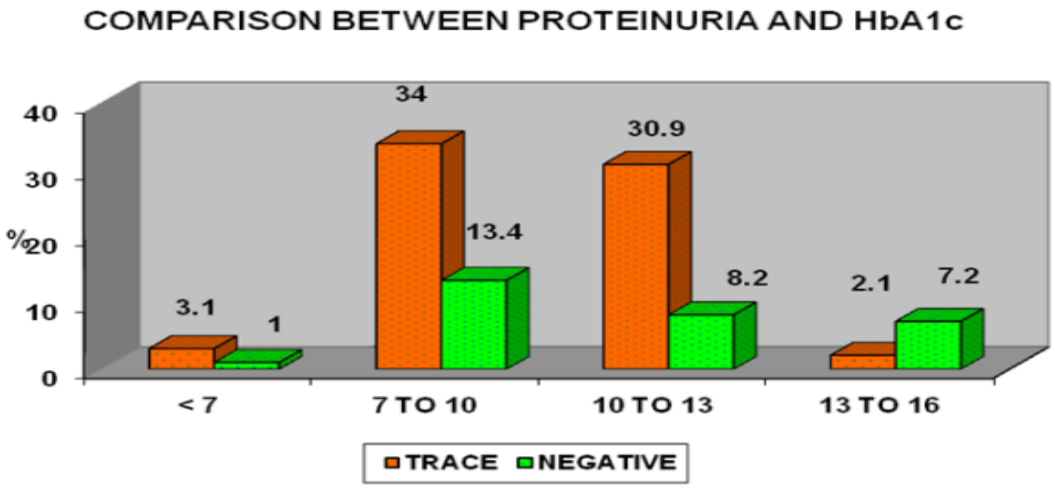

Figure showing the comparison between HbA1c levels and proteinuria among the study participants

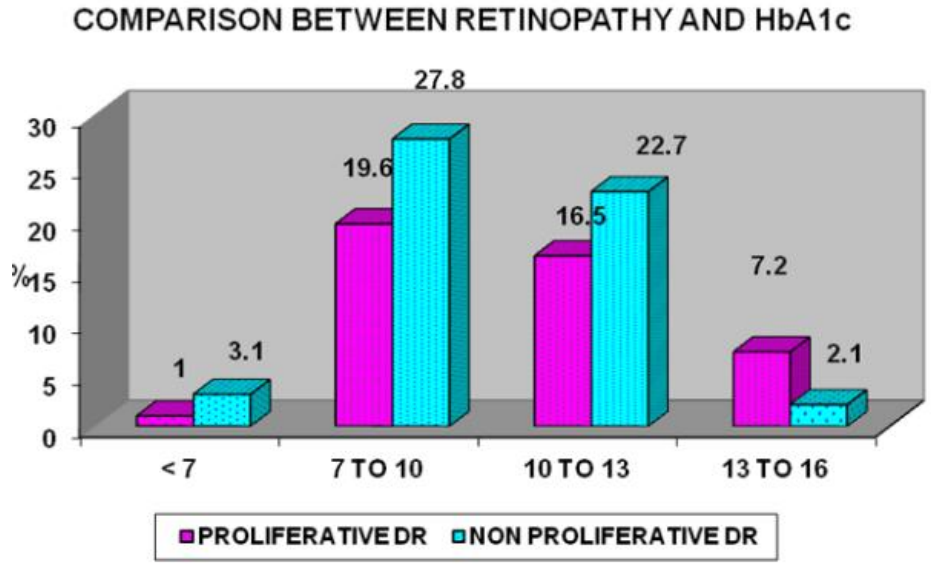

Figure showing the correlation between HbA1C and retinopathy

Discussion

The prevalence of CRI in this study's sample of people with type 2 diabetes is 22%. The current results are in agreement with the study's conclusions by Holly J. Kramer (14%). This group has a higher rate of CRI because they have a higher risk of kidney damage from having type 2 diabetes for a longer time, not being able to control their blood sugar as well, and starting DIM at a younger age. These results are in line with those of an earlier study by Holly J. Kramer that also discovered 64% retinopathy; in that study, 13% of individuals did not have albuminuria, and 45% showed it in conjunction with albuminuria. The failure to promptly diagnose diabetes and adhere to therapy are the main causes of this problem. Consistent with the results of the study by Holly J. Kramer,

the present investigation found that albuminuria and retinopathy were absent in 22% of the subjects. Thirdly, why do some people with type 2 diabetes have a diminished GFR even when they don't have retinopathy or albuminuria? Nephron loss can occur in vascular disorders when there is insufficient blood flow. The renal artery and smaller diameter arteries are affected by atherosclerosis, which is linked to interstitial fibrosis and "age-associated" factors. In addition, the impact of cholesterol emboli on CRI in type 2 diabetic patients is probably not as big as originally thought.

Conclusion and Recommendations

It is important to note that retinopathy and albuminuria are closely related, and individuals with type 2 diabetes are more likely to experience CRI. Only a third of patients with type 2 diabetes and chronic renal illness (CRI) exhibit albuminuria and diabetic retinopathy. Chronic renal injury is common in diabetic patients; however, classic diabetic glomerulosclerosis is not always the underlying renal disease. Researchers have found that keeping an eye on changes in funduscopic images, glomerular filtration rate (GFR), and urine albumin excretion can help find people with type 2 diabetes who have kidney damage. So, we need to focus on treating chronic renal damage (CRI) in type 2 diabetics who don't have diabetic glomerulosclerosis.

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