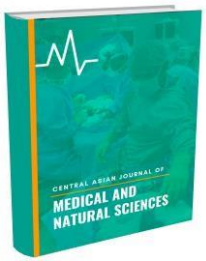

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Article

Type 2 Diabetes is a Common Condition that Causes High Blood Sugar (Glucose) Levels in Iraqi Patients with Some Parameters Measured

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Abstract: Diabetes mellitus (T2DM) is a multifactorial disorder that is quickly expanding in all landmasses of the world, causing high glucose levels in patients. An example of 60 Iraqi patients with type 2 diabetes was explored in view of a few standards. The glycemic control standards included fasting blood glucose (FBG), glycated hemoglobin (HbA1C), and insulin levels. The serum uric analysis was incorporated. The consequences of the current review showed a huge increment (P>0.05) in FBG, HbA1c, and insulin levels in type 2 diabetes patients contrasted with controls. Insulin responsiveness showed a huge decline (P>0.05) in patients contrasted with controls. Uric corrosive levels expanded in type 2 diabetes patients, yet the distinctions were non-huge. TAC level had a huge reverse relationship (P>0.05). HbA1c levels showed a critical opposite relationship (P>0.05) with uric corrosive.

Keywords: diabetes, glucose, insulin

1. Introduction

High glucose, brought about by deficient insulin combination, is the sign of diabetes, a metabolic problem. Normal microvascular messes influencing the skin, kidneys, and nerves are related with high glucose in individuals with persistent diabetes, as well as an expanded gamble of cardiovascular illness. Retinopathy, a confusion of diabetes, is one of the microvascular complexities featured in the symptomatic rules for the illness [1]. Type 2 diabetes, which isn't insulin-subordinate, represents 90-95 percent of all instances of the illness. It normally influences individuals who are insulin safe because of an imperfection in insulin receptors on cells or a lack in insulin discharge to some extent that doesn't make them need insulin treatment since the beta cells in the pancreas actually emit insulin [2]. Patients with type 2 diabetes frequently have insulin-safe fat cells, liver cells, and muscle cells.

Type 2 diabetes patients will quite often be overweight, with a weight record (BMI) more prominent than 20. Because of insulin opposition, the pancreas needs to work harder to create sufficient insulin for hefty individuals. Be that as it may, the insulin created won't be sufficient to keep glucose levels stable [3].

Type 2 diabetes can be constrained by controlling the sort and measure of food and exercise and hence controlling weight. As the sickness advances, diabetes prescriptions are

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required. To guarantee ideal eating routine and exercise control, notwithstanding adherence to prescription, it is prescribed to play out the HbA1c test a few times during the year as this test gives the typical glucose level over the existence of red platelets [3, 4, 5].

At the point when glucose levels ascend to where they show up in the pee, it causes unreasonable pee, thirst, craving, and aggravations in protein and fat digestion [6]. Both arising and created nations face a serious test with the spread of type 2 diabetes. It is a significant executioner illness, positioning seventh in the rundown of executioner sicknesses [6]. The variety of diets and ways of life that contrast in various nations and societies plays a significant part in the expansion in the quantity of diabetics during the year and is supposed to reach around 300 million of every 2025 [7].

The entanglements coming about because of insulin lack are metabolic problems, which make high blood glucose what's more elevated degrees of creatinine, cholesterol and transaminase compounds joined by a lessening in body proteins [8]. Optional confusions in diabetic patients have been seen to be changes in the piece of the vascular cellar film notwithstanding the amassing of glucose and response items that lead to expanded glucose usage in non-insulin-subordinate tissues [9]. Expansions in serum glucose proteins and different changes in atherosclerotic gamble factors have been approved by far most of examinations. This study remembered an assessment of a few biochemical variables for type 2 diabetic patients and contrasted them and the outcomes got in solid people [9].

Mechanisms of obesity-induced insulin resistance Obesity:

Fuels type 2 diabetes by inciting insulin obstruction. Treatment of type 2 diabetes has been restricted by the unfortunate comprehension of insulin opposition. Be that as it may, a few investigations have portrayed the relationship between mitochondrial brokenness, irritation, hyperinsulinemia, lipotoxicity, and insulin opposition [10]. Endoplasmic reticulum stress, oxidative pressure, hereditary foundation, maturing, hypoxia, and lipoatrophy have additionally been ensnared in the pathogenesis of type 2 diabetes by actuating insulin opposition. Notwithstanding, none of these ideas have prompted the revelation of successful medications for type 2 diabetes.

The explanation might be the absence of settlement on the interconnected systems of insulin opposition in type 2 diabetes. Raised degrees of supportive of fiery cytokines or an expanded number of white platelets in the blood or tissue are portrayed by aggravation. Overstimulation of provocative interaction habitually prompts different irregularities like organ brokenness and tissue injury. Corpulence could cause persistent and second rate aggravation that is engaged with T2DM. Moreover, fat explicit cytokines (leptin, adiponectin, and provocative cytokines (growth necrotic variable α (TNF-α)) and interleukin-6 (IL-6)) are emitted by instinctive adipocytes. A raised measure of fat tissue depleting into the entry vein, chemokines, and IL-6 creation can actuate liver and fundamental insulin opposition [11]. Besides, instinctive fat terminal and adipocyte size in people are additionally connected with insulin obstruction. The inclination of instinctive fat tissue for raised aggravation and the ensuing emission of cytokines that adjust insulin flagging may extensively add to insulin obstruction in stoutness.

The levels of the macrophage-determined apoptosis inhibitor of macrophage protein that animates lipolysis in fat tissue and answerable for neighborhood enrollment of fat tissue macrophages are additionally expanded with obesity [12]. Moreover, immersed unsaturated fats (FAs), glucose, and changes in stomach microbiota have been considered as triggers of metabolic irritation through the excitement of example acknowledgment receptors (PRRs, for example, cost like receptors (TLR), nucleotide oligomerization space (Gesture), and inflammasome [13]. These in the long run increment supportive of fiery cytokines creation and safe cell enlistment like T lymphocytes and macrophages in metabolic tissues. These favorable to provocative cytokines enact various kinases that obstruct insulin flagging and insulin activity in adipocytes and hepatocyte [14]. Different concentrates

on revealed that medicates that smother aggravation further develop insulin awareness and upgrade glucose guideline in T2DM insulin-safe patients [15]. For example, salsalate, TNF-α inhibitors (etanercept, infliximab, adalimumab), IL-1β bad guys like canakinumab, thiazolidinediones, and metformin are viewed as against diabetes drugs with calming properties [16].

Insulin, glucagon and the natural history of type 2 diabetes:

Studies play broadly analyzed the part insulin plays in creating T2DM. Martin et al [17] followed 155 subjects from 86 families more than 25 years. Albeit the example size is somewhat little, the length of the follow-up reinforces the review's convention. The gathering utilized intravenous glucose resilience test with the base model appraisal to assess insulin awareness (Si). The creators detailed subjects who created T2DM had lower upsides of Si. This demonstrates that insulin obstruction is a significant element in the pathophysiology of T2DM. Besides, Godsland et al [18] investigated β-cell insulin discharge at expanding fasting plasma glucose (FPG) fixations. The review estimated intense insulin reaction to glucose (AIRg) levels in 466 nondiabetic men. The creators revealed a precarious decrease in AIRg levels after FPG levels of 5mmol/L. Taken together, these examinations show that both insulin opposition and loss of β-cell capability are ensnared in the normal history of T2DM.

The effect of glucagon has likewise been perceived in diabetic patients [19]. Typical reaction to a carb dinner includes an ascent in insulin levels and a complementary decline in glucagon concentrations [20]. Conversely, in patients with T2DM, postprandial insulin discharge is discouraged, while glucagon levels are expanded. Shah et al. [21] estimated that absence of α-cell concealment because of hindered insulin emission in T2DM brought about hyperglycemia. Their review assessed the impact of glucagon concealment on glucose focus in people. The creators implanted insulin and glucose into the fundamental course at a rate which emulated postprandial profiles in diabetic patients. The review revealed that absence of glucagon concealment brought about a fundamentally expanded top plasma glucose fixation. Contrasted with glucagon-smothered diabetic profile. From these outcomes, the creators closed disappointment of glucagon concealment in diabetic patients causes hyperglycemia. Be that as it may, the concentrate just evaluated one postprandial insulin profile. In T2DM, insulin emission fluctuates enormously; accordingly, the aftereffects of this study are restricted exclusively to this review. A new rat study has likewise proposed that β-cells dedifferentiate under pressure to forebear pluripotent cells which emit glucagon [22]. While this study may feature a further component of expanded glucagon emission, further examinations should explore whether this cycle happens in human β-cells and its contribution in the pathogenesis of T2DM [24].

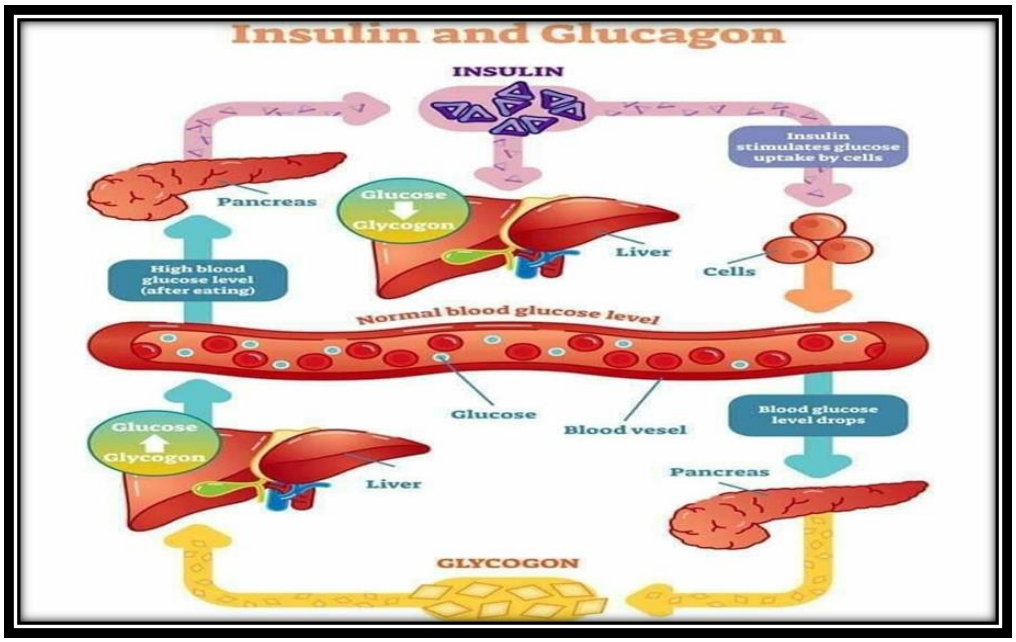

Figure 1. The metabolic processes of glucose [23]

2. Materials and Methods

Study population:

This study was a cross-sectional study conducted on 60 individuals aged 35-50 years, consisting of 20 healthy individuals who do not take nutritional supplements and vitamins, and 40 individuals with chronic diabetes. Samples were collected from Abu Ghraib General Hospital in Baghdad city in 2024 in Abu Ghraib district, Iraq, for both patients and healthy individuals. Blood samples of 5 ml were drawn from the patient intravenously using a plastic syringe and collected in a regular tube. Then the serum was separated using a centrifuge at 3000 rpm for 10 minutes and stored at -20 °C until testing.

Data collection and insulin:

The test uses a sandwich immunodetection method; the detector antibodies in the buffer bind to the antigen in the sample, form antigen-antibody complexes, and migrate to a nitrocellulose matrix to be captured by other antibodies immobilized on the test strip. More antigens in the sample will form more antigen-antibody complexes, resulting in a stronger fluorescence signal from the detector antibodies, which is processed by the Instrument for ichromaTM tests to show the concentration of ferritin in the sample as (ng/ml).

3. Results

Table 1. Includes control and patients

			HbA1C	FBS	U.A	INSULIN
Control	B1	MEAN	5.1	100.1	4.8	8.6
		SD	0.4	12.6	0.4	1.8
	B2	MEAN	4.9	101.4	4.8	8.4
		SD	0.4	11.1	0.6	0.7
	P value		>0.05	>0.05	>0.05	>0.05
Patients	B1	MEAN	8.73	178.86	6.35	3.19
		SD	1.74	53.86	0.90	1.06
	B2	MEAN	8.46	200.14	6.06	4.19
		SD	2.72	79.62	0.72	3.13
	B3	MEAN	8.7	191.0	6.3	2.5
		SD	1.1	38.2	0.7	0.3
	P value ANOVA		>0.05	>0.05	>0.05	<0.05

4. Discussion

In two groups: diabetic and normal. The data regarding age (range 35 to 55 years) and healthy subjects group were two groups for each group B1 which are non-obese subjects and the mean result of HbA1C .FBS .UA INSULIN was (5.1, 100.1, 4.8, 8.6) respectively while the mean (SD) for the same group was (0.4, 12.6, 0.4, 1.8) respectively while the mean (SD) for the B2 control group was (4.9, 4.101, 4.8, 8.4) respectively while the standard deviation (SD) was (0.4, 11.1, 0.6, 0.7) respectively while there were three groups for patients according to weight (B1, B2, B3) and the measurements of HbA1C .FBS .UA INSULIN were as follows for group B1 the mean was (8.73, 178.86, 6.35, 3.19, while (SD) was (1.74 53.86, 0.90, 1.06) respectively.

While group B2 for mean was (8.46, 200.14, 6.06, 4.19) respectively between them (SD) was (2.72, 79.62, 0.72, 3.13) respectively while group B3 for mean was (8.7, 191.0, 6.3, 2.5) respectively while (SD) was (1.1, 38.2 0.7, 0.3) respectively.

5. Conclusion

Type 2 diabetes is high blood sugar levels due to a lack of a hormone called insulin. Either your body doesn't produce enough or the insulin it produces doesn't work properly. This is sometimes called insulin resistance. The lack of insulin causes glucose from what you eat or drink to build up in your blood. That's why you may have symptoms of type 2 diabetes. If you have enough insulin or it's working properly, glucose in your blood should be released into your cells to give you energy. People with type 2 diabetes have poor blood sugar control. Overnutrition, combined with a sedentary lifestyle, leads to an abundant

buildup of glucose within muscle, fat tissue, and pancreatic cells. Fluctuating blood glucose concentrations may contribute significantly to oxidative stress, perhaps more than chronic hyperglycemia. Fasting blood sugar levels were significantly positively correlated with the main parameters studied (HbA1C . FBS . UA INSULIN). The significant association observed between FBS and other parameters tested revealed that hyperglycemia is a clear independent risk factor for the progression of type 2 diabetes.

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