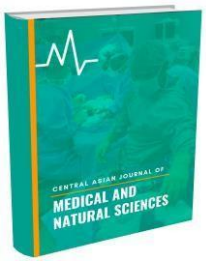

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Article

Metformin Versus Empagliflozin Monotherapy on Short-Term Glycemic Dairy: A true Experimental Study

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Abstract: Background: Glycemic control is still the most important aspect of T2DM treatment sodium-glucose cotransporter-2 (SGLT2) inhibitor, empagliflozin, and metformin therapy has been shown to be effective and well tolerated in patients with T2DM and is first of the several recommended treatment options. Since empagliflozin is poorly studied solely or in comparison with metformin, the current study aims at comparing the influence of these drugs on glycemic level individually in patients with type 2 diabetes mellitus (T2DM). Method: The study was a quantitative experimental investigation (cross-sectional study) which contrasted the influence of metformin and empagliflozin on the short-term glycemic level, the metformin group included 30 newly diagnosed type 2 diabetic patients are treated with 1500mg of metformin divided 3 times daily and empagliflozin group includes 30 newly diagnosed type 2 diabetic patients are treated with 10mg of Empagliflozin once time daily). A total of fifty male patients aged (30-70) years were enrolled in this study. This study was performed from may 2022 to August 2022. All patients were already a newly diagnosed as diabetic patients with type 2 according to their medical history of diabetes and the ADA and WHO diagnostic criteria. Blood samples were analyzed for basic and advanced investigations. A complete blood cell count (CBC) was assessed via the URIT-3000Plus automated analyzer electrochemiluminescence (E.C.L.) technology for immunoassay analysis was used for measurement of Lipid profile, Liver function test, Renal function tests, Fasting blood glucose level, Hormones (insulin, TSH) and Hemoglobin A1C (HbA1c) Test. Also SPA antigen Sandwich immunoluminometric assay was used for Measurement of the immunological parameter glutamic acid decarboxylase antibody (GAD65). Results: the results of human samples were indicated that the metformin-treated group was shown a significant difference in the mean level of pre-post glucose test when compared to the empagliflozin-consuming group. The level of glucose was decreased significantly in the group treated with metformin more than in the group treated with empagliflozin, P values were <0.001. Interestingly, the repeated measurements of glucose level also indicated a significant difference, P values were <0.001. In conclusion: Metformin and empagliflozin have been shown through clinical studies an improved in the lowering blood glucose. Both medications were significantly improved glycemic control.

Keywords: Type 2 Diabetes Mellitus, Glycemic Control, Metformin, Empagliflozin, Fasting Blood Glucose.

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1. Introduction

Diabetes Mellitus (DM) is a metabolic endocrine disorder that is characterized by persistent hyperglycemia with impaired carbohydrate, protein, and lipid metabolism due to defects in insulin secretion, insulin action, or both [1].

There is a need to gradually intensify therapy to achieve and maintain glycemic control [2]. Simplification of complex treatment plans is now recommended to reduce hypoglycemia and polypharmacy risk and decrease disease burden if it can be achieved within the individualized HbA1c target [3].

Glycemic control is still the most important aspect of T2DM treatment. Lifestyle changes and pharmacologic treatment are often used to treat T2DM [4]. The majority of people with T2DM are treated with oral medications rather than injectable medications, and most diabetes medicines were equally successful in lowering HbA1c levels [5].

Type 2 diabetes mellitus is considered an XXI century epidemic because it is the main cause of death in developed countries [6]. It is estimated that diabetes mellitus has been imposed on 463 million people in 2019 [7]. T2DM accounts for around 90 % of all cases of diabetes, which imposes 416.7 million for the same year. It is the most prevalent metabolic disorder with disastrous multi-systemic complications. According to the International Diabetes Federation (IDF), the prevalence of T2DM globally in 2019 was 9.3%, and it's going to rise to10.9% by 2045, affecting 700.2 million people globally [8].

The prevalence of diabetes mellitus increased rapidly in low-medium income society more than in high income once. The Global Burden of Disease Study 2013 identified all forms of diabetes mellitus as the 9th major cause of reduced life expectancy. The mortality rate of diabetes in 2017 was 10.7% among adults (20-70 years).in the Middle East and North Africa region, diabetes accounts for 373557 deaths (including Iraq) [9].

Metformin is the recommended first-line pharmacological treatment for T2DM by the American Diabetes Association [10]. Low cost, no adverse effect on cholesterol level, and also have cardiovascular beneficial effect. In medically reviewed studies, metformin is a common medication used by healthcare providers for type 2 diabetes, insulin resistance, PCOS, and prediabetes [11]. Metformin acts mainly by reducing hepatic glucose production via inhibition of gluconeogenesis and also increases glucose uptake in peripheral tissue. Metformin is associated with a low risk of hypoglycemia and is weight-neutral or can lead to weight loss [12]. It also improves insulin sensitivity, decreases insulin levels, and controls hyperglycemia. In addition, improves lipid profiles and lowers blood pressure in both patients and animal models with impaired glucose tolerance and type 2 diabetes mellitus [13]. On the other hand, Empagliflozin is a novel third-generation antidiabetic class of drugs known as a sodium-glucose co-transporter 2 (SGLT2) inhibitor and works by prompting the kidneys to eliminate extra glucose via the bladder and urine. It is a potent and selective sodium glucose co- transporter 2 (SGLT2) inhibitor in development for the treatment of T2DM [14].

Knowledge gap: Since metformin is a well-studied drug that has been shown to improve hyperglycemia, the study hypothesizes that metformin will have a superior effect to empagliflozin therapy when compared on a short-term level.

While empagliflozin is poorly studied solely or in comparison with metformin, the current study aims to compare the influence of these drugs on glycemic levels individually in patients with type 2 diabetes mellitus (T2DM).

2. Materials and Methods

The study was a quantitative experimental investigation (cross-sectional study) that contrasted the influence of metformin and empagliflozin on the short-term glycemic level. A total of sixty male patients aged 30-70 years were enrolled in this study. This study was

performed from May 2022 to August 2022. All patients were newly diagnosed as diabetic patients with type 2 according to their medical history of diabetes and the ADA and WHO diagnostic criteria. Every patient, after diagnosis and informed consent documentation, Demographic data which include age, gender, co-existent diseases, drug use, height/weight/BMI, pulse rate, blood pressure, and oxygen saturation (pulse oximeter reading in the index finger) and other vital signs were be questioned and examined clinically: Blood and urine samples were then be analysed for basic and advanced investigations.

Subsequently, patients were allocated into one of two groups: the metformin-taking group and the empagliflozin-consuming group. They were informed about the medication's dose, probable expected adverse effects, anticipated effects, and what they have to do in relation to their lifestyle and diabetic diary. In essence, patients were instructed to use their home glucose monitor (capillary blood) and to document their glucose readings four times per day. The timings were in accordance with the American Diabetes Association and included a fasting glucose reading and 1-2 hours post-meal readings

The patients will then be assigned randomly to one of two groups:

Metformin-treated group:- includes 30 newly diagnosed type 2 diabetic patients who are treated with 1500mg of metformin divided 3 times daily.

Empagliflozin-treated group:- includes 30 newly diagnosed type 2 diabetic patients who are treated with 10mg of Empagliflozin once daily.

Most of the patients are either overweight, having a BMI ranging from 25-29.9 kg/m^2, or obese, having a BMI≥30 kg/m^2, and all of them are newly diagnosed with no history of other associated illnesses or infections at the time of the study.

Patients were invited to return to the clinic as soon as they feel with adverse effects or when no response was achieved. In an otherwise occasion, patients were informed to complete the dairy and revisit the clinic after 2-3 weeks from the initial diagnosis. The average readings of each group were contrasted with the counterpart reading of the other groups. Patients having any chronic disease of the liver, renal, thyroid, pulmonary, cardiovascular complications, T1DM, pregnancy and lactation, psychiatric, mental disorders, malignant diseases, and autoimmune diseases, female patients, and those with other types of diabetes were excluded.

Venous blood samples (10 ml) were drawn by venipuncture from each patient for both groups, separated into two parts: the first was held in an ethylene diamine tetraacetate (EDTA) tube for HbA1c percent and CBC, and the second was kept in a gel tube for serum isolation for biochemical, hormonal, and immunological tests.

Blood samples were divided immediately into 2 parts:

First parts: - (8ml) was put in the plain tube (gel tube containing a special gel that separates blood cells from serum to cause blood to clot quickly) and left to clot for (30 min) at room temperature, and then separated by centrifugation at (3000 rpm) for (15min), used for biochemical assays.

Second part: - which contains (2ml) blood was put in an EDTA tube, mixed gently, and used for HbA1c measurements and CBC assay. A complete blood cell count (CBC) was assessed via the URIT-3000Plus automated analyzer, and electrochemiluminescence (E.C.L.) technology for immunoassay analysis was used for measurement of Lipid profile, Liver function test, Renal function tests, fasting blood glucose level, Hormones (insulin, TSH), and Haemoglobin A1C (HbA1c) Test. Also, the SPA antigen Sandwich immunoluminometric assay was used for the measurement of the immunological parameter glutamic acid decarboxylase antibody (GAD65).

3. Results

A total of (60) participants were included in this study, patient groups were divided into subgroups based on Age, gender, and their medical history. The clinical demographic characteristics and laboratory parameters of the study groups were summarized in Table (1), Figure (1&2). The BMI mean was 29.8 while the mean age of the cases was 51.8 years. The age range of participants was (23%) (30-45) years old, (57%) of the patient were within (46-60) years, while (20%) of the patient were within the age range (61-75). Also, the analysis of data illustrated that most of the patients were overweight or obese and bout (22%) were having abnormal Blood pressure.

Table 1. Demographic and clinical characteristics of the study population.

Variables		Statistical values
Age (Years)		51.80±11.54
BMI (Kg/m^2)		29.76±5.36
Pulse rate		81.32±5.17
O_2 saturation		95.15±2.94
Age. Groups	30-45 Years	14
	46-60 Years	34
	61-75 Years	12

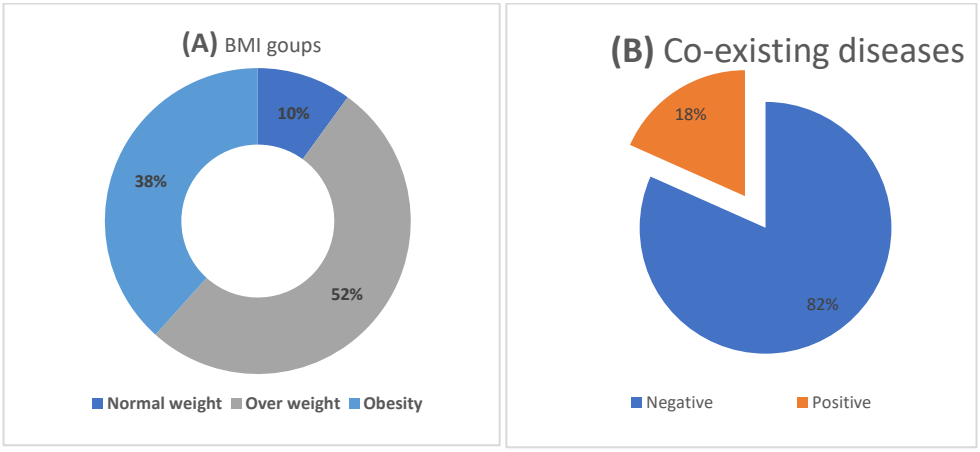

Figure 1. Demographic description of the study patients, (A) The BMI groups, (B) The Co-existing diseases.

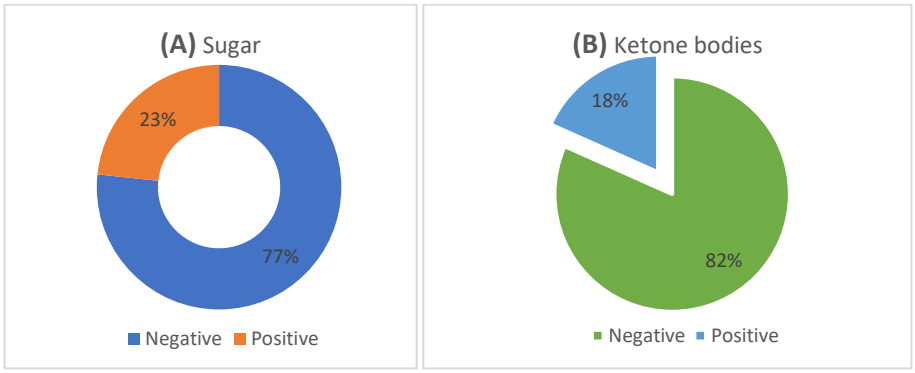

Figure 2. Demographic description of the study patients, General urine examination: (A) Sugar and (B) Ketone bodies.

Table (2) illustrated the mean difference of lipid penal, liver function tests, renal tests, thyroid tests, and the level of Immunology test (Anti GAD65) CLIA. Results indicated increased levels of TG and VLDL in the patient group compared to the normal range. All liver function tests were within the normal range, HbA1C, and the urea level and WBC were increased markedly.

Table 2. Baseline characteristics and mean difference of the measured biomarkers in DM patients, the number of participants (n= 60).

Variables	Patient		Normal value
	Median (Min-Max)	Mean±SD	
Lipid profile			
Cholesterol	199.65(114.6-305.4)	(196.36±46.44)	50-200 mg/dL
TG	227.5(83.7-580)	(231.98±98.03)	35-150 mg/dL
HDL	51.45(29.0-80.2)	(51.49±12.45)	35-65 mg/dL
LDL	118.1(51.8-213.8)	(115.78±35.78)	0-140 mg/dL
VLDL	44.45(16.3-146)	(51.93±30.65)	2-30 mg/dL
Liver Function			
ALT	25.85(6.6-70)	(29.53±15.84)	7-55 U/L
AST	18.1(6.6-50.5)	(19.53±8.34)	8-48 U/L
ALP	60(25.1-150.2)	(68.28±29.31)	30-120 U/L
TSB	0.58(0.2-1.5)	(0.56±0.23)	0.1-1.2
Kindy test			
Urea	30.9(17.1-75.5)	(34.01±13.02)	7-20 mg/dL
Creatinine	0.7(0.5-1.9)	(0.82±0.32)	0.5-1.1 mg/dL
	Thyroid function		
TSH	1.245(0.4-3.8)	(1.52±0.79)	0.4-4 mg/dL
	RBC		
HB	13.35(10.9-16.50)	13.46±1.27	11.5-16.5 g/dL
RBC	4.93(2.93-6.55)	4.97±0.65	3.50-5.50
WBC	8.7(4.6-20.60)	8.91±2.98	3.5-11
Glycated heamoglobin			
HbA1C	9(6.3-15.6)	9.44±2.03	< 6.5
	Immunity test		
Anti GAD65	0.8(0.4-280)	(9.11±38.20)	

Table (3) demonstrated the mean level of serum biomarkers in diabetic patients included (Lipid profile panel, liver function tests, renal function test and thyroid test) before divided them into two groups, this step was performed in order to choose the appropriate treatment for each one.

Table 3. The mean difference of pre-test of a biomarker in DM patients, the number of participants (n= 60).

Biomarkers	Groups	Mean	SD	P value
CHOL	Group 1	194.02	42.58	0.7
	Group 2	198.69	50.64	
TG	Group 1	225.94	112.03	0.637
	Group 2	238.02	83.22	
HDL	Group 1	49.41	13.19	0.02*
	Group 2	53.56	11.51	
LDL	Group 1	116.14	36.82	0.938
	Group 2	115.42	35.34	

VLDL	Group 1	50.79	32.93	0.776
	Group 2	53.07	28.70	
ALT	Group 1	30.29	14.93	0.712
	Group 2	28.76	16.91	
AST	Group 1	18.41	6.94	0.305
	Group 2	20.64	9.54	
ALP	Group 1	66.52	25.07	0.647
	Group 2	70.03	33.35	
TSB	Group 1	0.60	0.25	0.177
	Group 2	0.52	0.20	
Urea	Group 1	32.95	13.05	0.534
	Group 2	35.07	13.13	
Creatinine	Group 1	0.83	0.26	0.921
	Group 2	0.82	0.38	
TSH	Group 1	1.50	0.75	0.846
	Group 2	1.55	0.85	
(Anti GAD65) CLIA	Group 1	5.44	15.10	0.461
	Group 2	12.79	52.09	

In this study, patients were divided into two groups: a metformin-treated group and the empagliflozin-treated group, also all participants were instructed to use their home glucose monitor in order to document their glucose readings four times per day. The timings were in accordance with American Diabetic Association plus a fasting glucose reading and 1-2 hours post-meal reading.

In Table (4), the results indicated that the metformin-treated group was shown a significant difference in the mean level of pre-post glucose test when compared to the empagliflozin-consuming group. The level of glucose was decreased significantly in the group treated with metformin more than in the group treated with empagliflozin, P values were <0.001. Interestingly, the repeated measurements of glucose level also indicated a significant difference, P values were <0.001.

Table 4. Mean differences of the pre-post test of glucose in DM patients treated with either metformin or empagliflozin.

Biomarkers	Metformin N = (30)	Empagliflozin N = (30)	P value
Fasting Plasma Glucose	116.00±14.53	154.00±27.21	**<0.001[S]**
1-2 hours Post-Breakfast	150.70±22.57	187.83±27.75	**<0.001[S]**
1-2 hours post-Lunch	157.27±19.66	194.00±32.15	**<0.001[S]**
1-2 hours post-Dinar	160.67±20.33	199.33±37.39	**<0.001[S]**
T-test was *: significant at p ≤ 0.05			
N: number of cases; SD: standard deviation; S: significant; NS= Non significant			

4. Discussion

Type 2 diabetes (T2D) is a prevalent metabolic disorder. it is commonly asymptomatic and frequently recognized by the manifestation of excess body weight and elevation of random blood glucose [15]. Generally speaking, T2D starts with insulin resistance, which is a cumulative health consequence of obesity, dysfunctional adipose tissue, chronic inflammation, and decrease in pancreatic β-cell mass and consecutive failure in the production of insulin. In addition, prolonged uncontrolled blood glucose levels have harmful effects on multiple tissues [16].

Dyslipidaemia has been recognised as a risk factor for T2DM. A large prospective study among middle-aged adults conducted in the USA has shown the low levels of high-density lipoprotein cholesterol (HDL-C) and elevated triglyceride (TG) levels were significantly related to the development with T2DM, these results were completely agreed with the current study. It was also found that the elevated concentration of total cholesterol (TC), low- density lipoprotein cholesterol (LDL-C) and TG were independent risk factors for development of new-onset T2DM [17]. Moreover increasing number of studies recently demonstrated that combined lipid parameters such as TG/HDL-C were associated with T2DM [18].

There were several important findings which confirmed that participants with borderline high TG, hypercholesterolaemia, hypertriglyceridaemia and low HDL-C suffered a higher T2DM risks through 5-year national longitudinal study. Also, there is current evidence demonstrating that TC, TG and low HDL-C are risk factors for diabetes and emphasise that serum TG and combined lipid parameters were more efficient predictor factor for diabetes versus other lipid profiles [19].

Some studies have shown that lipoproteins will have some effects on B-cell insulin secretion and glucose metabolism, which indirectly indicated that dyslipidaemia plays a role in the development of diabetes. A cohort study stated that the decrease of insulin secretory capacity may due to increased serum TC level [20].

A systematic review concluded hypercholesterolaemia and low HDL-C levels may accelerate the development of beta-cell dysfunction under available evidence. This review proposed that cholesterol efflux obstructed by defective HDL could bring about accumulation of cholesterol in beta-cells, induce hyperglycaemic, damaged insulin secretion and b-cell apoptosis [21].

Diabetes is associated with quantitative changes in the amount of circulating lipids notably a reduction in HDL. Like other lipoproteins, HDL also undergoes significant qualitative changes in diabetes, in both structure and function. However, since dyslipidemia may be present several years before the onset of diabetes, it is hard to determine which of these changes are related to the pathognomonic features of the disease, and which precede and accelerate its progression [22]. Many factors contribute to the HDL dysfunction that is witnessed in T2DM. Oxidation and glycation of HDL-associated proteins were shown to render them inactive [23]. In fact, the role of cholesterol metabolism and HDL function in the pathogenesis of Type 2 diabetes mellitus (T2DM) has recently gained a lot of attention. Mice studies suggest that cholesterol accumulation in islet β-cells is the reason for DM pathology. HDL also appears to protect β-cells from the toxic effects of glucose and IL-1β, and to enhance insulin secretion. In skeletal muscles, HDL was demonstrated to increase insulin activity in the form of glucose uptake [24]. In this study, patients who had a high level of lipid profile were treated with empagliflozin.

As shown in Table 3.4, Metformin was demonstrated to be a good reducer of fasting plasma glucose concentrations by reducing rates of hepatic glucose production [25]. The current study shows that the metformin-treated group showed a significant difference in the mean level of pre-post glucose test when compared to the empagliflozin-consuming group; these results were completely consistent with other studies [26].

The mechanism by which metformin lowers endogenous glucose production has been reported before. different finding have previously examined the effect of metformin on the rates of net hepatic glycogenolysis and gluconeogenesis in patients with type 2 diabetes, with conflicting results, which may be due to the variability in doses and route of administration. McCreight et al. [27]. Studied diabetic individuals before and after treatment with metformin to estimate the rates of gluconeogenesis. It was found that metformin decreased endogenous glucose production through a 37% reduction in rates of gluconeogenesis.

Madiraju et al. [28] were performed an assessment rate of gluconeogenesis in diabetic subjects before and after metformin treatment. These investigators reported that metformin treatment significantly decreased glucose production, but they found no change in the contribution of gluconeogenesis from lactate. They therefore concluded that metformin decreased glucose production by inhibiting hepatic glycogenolysis. More research, Duca et al. examined the mechanism of metformin's action in diabetic subjects . it was also found that metformin treatment led to reduction in the rate of glucose production, with no change in the absolute rate of gluconeogenesis and concluded that this occurred through reduction in hepatic glycogenolysis [29].

5. Conclusion

The single-treatment of empagliflozin/or metformin may present a useful treatment option for patients with T2DM who are inadequately controlled with metformin. This option may be particularly suitable for patients who would benefit from the additional benefits of weight reduction, as well as individuals with risk factors for dyslipidemia or declining renal function. The empagliflozin could simplify therapy and potentially improve clinical outcomes compared with metformin.

Additional research is needed to further identify the advantages of early combination therapy in T2DM and to provide guidance on the selection of specific combination therapies to meet the individual needs of patients with T2DM. It might be a good idea to study a different range of the concentration individually or as a combination of the empagliflozin and metformin to assess their role on the glycemic control in T2DM

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