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Research Article

Biochemical parameters and quality of life response to life-style modification in obese with type 2 diabetes mellitus

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Abstract: Globally, type 2 diabetes mellitus (T2DM) is an epidemic medical problem that adversely affect the patient's functional capacity and quality of life (QoL). This study designed to examine the effects of weight reduction program on biochemical parameters and quality of life for obese T2DM patients. Eighty obese subjects with T2DM (48 males and 32 females), the range of their body mass index (BMI) was 30 to 34 Kg/m^2. Smoking, pregnancy, heart, kidney, hepatic and respiratory failure were the main exclusion criteria. Participants randomly assigned into two study groups; group (A) enrolled in weight-reducing program composed of aerobic exercise and diet regimen. However, group (B) received no clinical intervention. All participants signed the informed consent. Weight reducing program of group (A) resulted in significant increase in mean values of SF-36 subscale scores, HDL cholesterol and QUICKI. In addition to significant increase in mean values of LDL cholesterol, TC, TG, HOMA-IR and BMI. Changes of group (B) were not significant. Moreover, comparison between both groups revealed significant differences at the end of the study. Weight reducing program improves quality of life and selected biochemical parameters of obese type 2 diabetics.

Keywords: Weight reduction; Biochemical parameters; Life response quality; Life style intervention; Type 2 diabetes; Obesity

1. Introduction

Globally, diabetes mellitus is a common health problem, as about 365 millions of subjects will have diabetes by 2030 (WHO: https://www.who.int/). However, type 2 diabetes (T2DM) is an important risk factor for cardiovascular disorders[1]. A chronic metabolic disease called type 2 diabetes is typified by persistently high blood sugar levels. Resistance to the peripheral effects of insulin, decreased insulin production, or both could be the cause. When combined with other metabolic abnormalities in individuals with diabetes mellitus, chronic hyperglycemia can harm multiple organ systems and result in devastating and potentially fatal health complications. The most common of these are microvascular (retinopathy, nephropathy, and neuropathy) and macrovascular (an increased risk of cardiovascular diseases by two to four times). Defective insulin secretion by pancreatic β-cells and the incapacity of insulin-sensitive tissues to react to insulin adequately are the two main causes of T2DM, one of the most prevalent metabolic diseases. The molecular mechanisms involved in the synthesis, release, and detection of insulin are closely regulated since these actions are necessary for maintaining glucose homeostasis. A metabolic imbalance that causes the disease can result from flaws in any of the pathways underlying these activities[2-4].

Both genetics and the environment have an impact on the epidemiology of T2DM. After being exposed to an environment that promotes sedentary behavior and high calorie intake, genetic variables come into play. Although genome-wide association studies have discovered common glycaemic genetic variations for T2DM, these only explain 10% of the variance in the trait, indicating the importance of uncommon variants. Individuals from diverse ethnic backgrounds may exhibit distinct phenotypes that heighten susceptibility to clusters of cardiovascular disease risk factors, such as insulin resistance, dyslipidemia, and hypertension.

Factors that contribute to the prevalence of T2DM include a complex interplay of metabolic, genetic, and environmental factors. Evidence from epidemiological studies indicates that many cases of T2DM can be avoided by improving the primary modifiable risk factors (obesity, low physical activity, and an unhealthy diet), even though individual

predisposition to the disease due to non-modifiable risk factors (ethnicity and family history/genetic predisposition) has a strong genetic basis.

Obesity is an excessive adipose tissue mass due to adipocytes increased number and size[5, 6]. High mortality rate among diabetic patients usually due to due to associated cardiovascular disorders, dyslipidemia and hypertension[1, 6-9]. The greatest risk factor for T2DM is obesity (body-mass index [BMI]≥30 kg/m2), which is linked to metabolic abnormalities that cause insulin resistance. The age at which T2DM is diagnosed is inversely correlated with BMI. Although the precise processes by which obesity causes T2DM and IR are still unknown, a number of components have been implicated in the development of this pathological process, which includes both inter-organ interactions and cell-autonomous pathways. Another risk factor for T2DM is a sedentary lifestyle, as demonstrated by the Women's Health Study and the Kuipio Ischemic Heart Disease Risk Factor Study, which found that people who walked for two to three hours per week or for at least forty minutes per week had a 34% and 56% lower chance of getting T2DM, respectively. Physical activity has three main advantages for delaying the onset of T2DM. First, as skeletal muscle cells contract, more blood flows into the muscle, which improves the muscle's ability to absorb glucose from plasma. Second, the well-known intra-abdominal fat, a known risk factor that encourages IR, is decreased by physical exercise. Lastly, it has been demonstrated that moderate-intensity exercise increases glucose absorption by 40%. In addition to improving insulin sensitivity and glucose absorption, physical activity can also reduce or even reverse oxidative stress and inflammation, two variables that predispose people to T2DM[10, 11].

According to the disease's etiology, unusually high blood glucose levels are caused by a breakdown in the feedback loops between insulin action and secretion. Insulin production is decreased in β-cell malfunction, which restricts the body's ability to sustain physiological glucose levels. Conversely, IR leads to a decrease in glucose absorption in muscle, liver, and adipose tissue as well as an increase in glucose synthesis in the liver. β-cell dysfunction is typically more severe than IR, even though both processes occur early in the pathophysiology and contribute to the development of the disease. However, hyperglycemia is exacerbated and T2DM progresses when both IR and β-cell dysfunction are present[1, 10].

Adipose tissue involved in secretion of some chemical materials that locally and systemically have roles in multiple metabolic and inflammatory processes. Moreover, adipose tissue endocrine dysfunction induces atherosclerosis, insulin resistance and T2DM. Long-term morbidity usually associated with T2DM as stroke, heart disease, blindness, lower extremity amputation and renal failure[12, 13]. In the other hand, obesity is commonly associated with T2DM, cancer, musculoskeletal disease and infertility[14].However, T2DM adversely affects quality of life (QoL)[15-18]. In addition, obese subjects had lower QoL than non-obese subjects[16, 19]. Obesity and its associated medical co-morbidities in addition to psychological disorders adversely affect their quality of life (QOL)[20, 21]. However, obesity usually accompanied with poor mood and emotional well-being[21-24]. Moreover, obese women usually have more deterioration in quality of life, low self-esteem and depression than obese men. However, exercise training was proved to modulate depression and deficit of both quality of life and mood in obese women[25-30].

The aim of the present study is to measure the effects of weight reduction program on QoL in obese Saudi patients with T2DM.

2. Methods

2.1. Subjects: Eighty obese subjects with T2DM (48 males and 32 females), the range of their body mass index (BMI) was 30 to 34 Kg/m^2. Smoking, pregnancy, heart, kidney, hepatic and respiratory failure were the main exclusion criteria. Participants randomly assigned into two study groups; group (A) enrolled in weight-reducing program composed of aerobic exercise and diet regimen. However, group (B) received no clinical intervention. All participants signed the informed consent.

2.2. Measurements:

1. Health-related quality of life (SF-36 HRQL): SF-36 used as a measure of quality of life by detecting the degree of change in subject's general health that involve eight subscales: General Health, Vitality, Physical Functioning, Bodily Pain, Social Functioning, Emotional Role Functioning, Physical Role Functioning and Mental Health[31].

2. Biochemical analyses: Blood lipids profile included HDL cholesterol, LDL cholesterol, Plasma total cholesterol (TC), triglycerides (TG) determined by chromatography method (Boeringher Mannheim kit). Insulin kit (Roche Diagnostics, Indianapolis, IN, USA) with cobas immunoassay analyzer (Roche Diagnostics) used to measure serum insulin. However, While, insulin resistance evaluated with homeostasis model assessment (HOMA-IR). HOMA-IR = [fasting blood glucose (mmol/l) _ fasting insulin (mIU/ml)]/22.5. Moreover, The quantitative insulin-sensitivity check index (QUICKI) using the formula: QUICKI=1/[log(insulin) + log(glucose)] was used to measure insulin sensitivity[11, 32].

2.3. Procedure: Participants were randomly assigned into groups:

Group (A): Treadmill aerobic exercise training was the training program that included three session every week for six months and composed of warming up for 5 minutes, aerobic exercise for 30 minutes with an intensity was 60-70% of each subject individual maximum heart rate and ended with 10 minutes cooling down for 10 minutes. Moreover, a diet

control program under supervision of a dietician was conducted to provide each individual with only 1200 Kilocalories/day for 6 months[32, 33].

Group (B): Participants of this group received no clinical intervention.

3. Results

3.1. Fundamental parameter analysis: Here, we collected the parameters values for the two groups and also predicted the significance or P-values (Table 1). Here, all the 12 predicted parameters (baseline criteria) do not show any significance as we can see in Table 1.

Table 1. participants' baseline criteria.

	Group (A)	Group (B)	Significance
Age (year)	48.73 ± 7.26	50.12 ± 6.91	P>0.05
Gender (male/female)	21/19	22/18	P>0.05
BMI (kg/m2)	32.32 ± 4.94	32.21 ± 4.55	P>0.05
Duration of diabetes (years)	12.19 ± 2.15	11.78 ± 2.16	P>0.05
Waist circumference (cm)	105.11 ± 7.27	103.96 ± 7.35	P>0.05
Waist-hip ratio	0.94 ± 0.07	0.93 ± 0.05	P>0.05
Body fat (%)	34.52 ± 6.73	33.71 ± 6.45	P>0.05
SBP (mmHg)	141.31 ± 14.86	140.53 ± 15.27	P>0.05
DBP (mmHg)	85.29 ± 7.63	83.12 ± 7.28	P>0.05
HBA1c (%)	8.23 ± 1.94	8.14± 1.81	P>0.05
Glucose (mmol/L)	6.13 ± 0.98	6.05 ± 0.87	P>0.05
Insulin (pmol/L)	19.92 ± 3.27	18.76 ± 3.12	P>0.05

BMI: Body Mass Index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HDL-C: High density lipoprotein cholesterol; HBA1c: glycosylated hemoglobin.

3.2. Weight reducing program: Weight reducing program of group (A) resulted in significant increase in mean values of SF-36 subscale scores, HDL cholesterol and QUICKI. In addition to significant increase in mean values of LDL cholesterol, TC, TG, HOMA-IR and BMI (Table 2 and 3).

Table 2. Mean value and significance of measured variables of group (A) before and at the end of the study.

	Mean +SD		T- value	significance
	Before	After		
BMI (kg/m2)	32.32 ± 4.94	27.45 ± 3.57*	5.21	P<0.05
TC (mg/dl)	199.17 ± 18.36	168.95 ± 15.12*	7.96	P<0.05
HDL-c (mg/dl)	35.15 ± 4.22	40.26 ± 4.75*	5.65	P<0.05
LDL-c (mg/dl)	138.32 ± 14.14	122.47 ± 11.29*	6.68	P<0.05
TG (mg/dl)	159.24 ± 18.28	127.19 ± 16.43*	5.91	P<0.05
QUICKI	0.121 ± 0.016	0.146 ± 0.015*	5.83	P<0.05
HOMA-IR	5.38 ± 0.93	4.11 ± 0.86*	4.94	P<0.05

TC: Total cholesterol; HDL-c: High density lipoprotein cholesterol; LDL-c: Low density lipoprotein cholesterol; TG: Triglyceride; (*) indicates a significant difference between the two groups; P < 0.05.

Table 3. Mean value and significance of SF-36 subscale scores in group (A) before and at the end of the study.

SF-36 subscale variables	Mean +SD		T- value	Significance
	Before	After		
SF-36: Health transition	2.87 ± 0.72	1.76 ± 0.65*	4.88	P<0.05
SF-36: Physical functioning	72.34 ± 7.62	80.27 ± 8.36*	6.37	P<0.05
SF-36: Role functioning: Physical	79.11 ± 8.12	86.91 ± 9.21*	5.84	P<0.05
SF-36: Bodily pain	74.22 ± 8.15	69.73 ± 7.19*	6.23	P<0.05
SF-36: General health	74.18 ± 7.27	78.45 ± 6.93*	5.75	P<0.05

SF-36: Vitality	56.92 ± 5.26	65.14 ± 6.24*	5.68	P<0.05
SF-36: Social functioning	86.35 ± 8.12	93.65 ± 9.11*	6.21	P<0.05
SF-36: Role functioning: Emotional	89.43 ± 9.15	84.97 ± 8.28*	5.62	P<0.05
SF-36: Mental health	85.18 ± 7.23	79.31 ± 6.64*	6.26	P<0.05

(*) indicates a significant difference between the two groups; P < 0.05.

Changes of group (B) were not significant (Table 4 and 5). Moreover, comparison between both groups revealed significant differences at the end of the study (Table 6 and 7).

Table 4. Mean value and significance of Mean value and significance of measured variables of group (B) before and at the end of the study.

	Mean +SD		T- value	Significance
	Before	After		
BMI (kg/m2)	32.21 ± 4.55	32.42 ± 3.61	0.91	P>0.05
TC (mg/dl)	197.96 ± 17.87	198.43 ± 17.92	0.86	P > 0.05
HDL-c (mg/dl)	33.67 ± 3.54	33.12 ± 3.51	0.96	P > 0.05
LDL-c (mg/dl)	136.74 ± 15.21	139.28 ± 15.44	1.15	P>0.05
TG (mg/dl)	156.55 ± 15.87	158.35 ± 15.91	1.23	P <0.05
QUICKI	0.116 ± 0.021	0.113 ± 0.019	0.97	P > 0.05
HOMA-IR	4.95 ± 0.95	4.98 ± 0.97	0.85	P>0.05

TC: Total cholesterol; HDL-c: High density lipoprotein cholesterol; LDL-c: Low density lipoprotein cholesterol; TG: Triglyceride; (*) indicates a significant difference between the two groups; P < 0.05.

Table 5. Mean value and significance of SF-36 subscale scores in group (B) before and at the end of the study.

SF-36 subscale variables	Mean +SD		T- value	Significance
	Before	After		
SF-36: Health transition	2.86 ± 0.83	2.91 ± 0.85	0.93	P>0.05
SF-36: Physical functioning	70.93 ± 7.21	70.29 ±7.32	0.87	P>0.05
SF-36: Role functioning: Physical	79.87 ± 7.95	79.11 ± 7.86	0.85	P>0.05
SF-36: Bodily pain	74.16 ± 8.24	74.45 ± 8.29	0.92	P>0.05
SF-36: General health	73.48 ± 7.53	73.13 ±7.66	0.78	P>0.05
SF-36: Vitality	56.24 ± 5.94	55.97 ± 5.83	1.15	P>0.05
SF-36: Social functioning	85.93 ± 9.21	85.16 ± 9.14	0.69	P>0.05
SF-36: Role functioning: Emotional	89.25 ± 7.73	89.64 ± 7.75	0.91	P>0.05
SF-36: Mental health	85.33 ± 7.92	85.76 ± 8.04	0.75	P>0.05

(*) indicates a significant difference between the two groups; P < 0.05.

Table 6. Mean value and significance of Mean value and significance of measured variables of group (A) and group (B) at the end of the study.

	Mean +SD		T- value	significance
	Group (A)	Group (B)		
BMI (kg/m2)	27.45 ± 3.57*	32.42 ± 3.61	4.32	P<0.05
TC (mg/dl)	168.95 ± 15.12*	198.43 ± 17.92	6.11	P<0.05
HDL-c (mg/dl)	40.26 ± 4.75*	33.12 ± 3.51	4.43	P<0.05
LDL-c (mg/dl)	122.47 ± 11.29*	139.28 ± 15.44	5.27	P<0.05
TG (mg/dl)	127.19 ± 16.43*	158.35 ± 15.91	4.36	P<0.05
QUICKI	0.146 ± 0.015*	0.113 ± 0.019	4.51	P<0.05
HOMA-IR	4.11 ± 0.86*	4.98 ± 0.97	3.29	P<0.05

TC: Total cholesterol; HDL-c: High density lipoprotein cholesterol; LDL-c: Low density lipoprotein cholesterol; TG: Triglyceride; (*) indicates a significant difference between the two groups; P < 0.05.

Table 7. Mean value and significance of SF-36 subscale scores in group (A) and group (B) at the end of the study.

SF-36 subscale variables	Mean +SD		T- value	Significance
	Group (A)	Group (B)		
SF-36: Health transition	1.76 ± 0.65*	2.91 ± 0.85	4.13	P<0.05
SF-36: Physical functioning	80.27 ± 8.36*	70.29 ±7.32	5.22	P<0.05
SF-36: Role functioning: Physical	86.91 ± 9.21*	79.11 ± 7.86	5.24	P<0.05
SF-36: Bodily pain	69.73 ± 7.19*	74.45 ± 8.29	5.18	P<0.05
SF-36: General health	78.45 ± 6.93*	73.13 ±7.66	5.34	P<0.05
SF-36: Vitality	65.14 ± 6.24*	55.97 ± 5.83	5.27	P<0.05
SF-36: Social functioning	93.65 ± 9.11*	85.16 ± 9.14	5.38	P<0.05
SF-36: Role functioning: Emotional	84.97 ± 8.28*	89.64 ± 7.75	5.21	P<0.05
SF-36: Mental health	79.31 ± 6.64*	85.76 ± 8.04	5.14	P<0.05

(*) indicates a significant difference between the two groups; P < 0.05.

4. Dicussion: Diabetes is the seventh leading cause of death worldwide[34]. About 600 million subject will have diabetes by year 2035. Globally, type 2 diabetes constitute up to 95% of all diabetes patients and about 8% of all population that is raise in parallel with obesity. This study designed to examine the influence of weight reduction program on some biochemical parameters and quality of life for obese T2DM patients[35].

Results of the present study proved that blood lipids profile and glucose control modulated with weight loss. Similarly, several studies reported **Shephard and Balady** noticed that exercise training reduced triglycerides, cholesterol and LDL cholesterol and improved HDL levels[20, 36, 37]. However, **Tran and Weltman** proved an association modulation of lipids profile and weight loss[38]. In addition, **Franz et al.** and **Huang et al.** mentioned an association between HbA1c, blood lipids and weight loss in report of their meta-analysis and systematic review on obese type 2 diabetics[39, 40]. Similarly, **the Look AHEAD study** stated that long-term weight reducing program improved glucose control, blood lipids and cardiovascular disorders risk factors[41]. Moreover, many previous studies confirmed that weight loss improve glucose control and modulated insulin resistance[42-44]. Improved glucose transporter protein and mRNA, post-receptor insulin signaling, clearance of free fatty acids, activity of glycogen syntheses and hexokinase in addition to better muscle glucose delivery and therefore, changes in muscle composition[45].

The results of our study regarding the QOL proved that combined dietary weight loss and exercise group improved aspects of QOL in obese patients with T2DM. Many previous studies reported that HRQOL parameters improved significantly following weight loss[30, 46, 47]. While, **Blissmer et al.** and **Ross et al.** stated that a six months weight reducing program significantly improved SF-36 subscales in obese subjects[29, 48]. In addition, **Thomson and colleagues** mentioned that obese polycystic ovary syndrome (PCOS) women experienced improved depression and quality of life parameters because of weight reducing program[49]. Moreover, **Yackobovitch-Gavan et al.** conducted a study on 162 obese children who had improved their QOL because of weight reducing program[50]. Similarly, **Ades et al.** and **Williamson et al.** reported that weight loss associated with improved aerobic fitness of obese type 2 diabetic subjects[51, 52].

The World Health Organization (WHO) defines diabetes mellitus as a long-term metabolic condition marked by high blood glucose levels that eventually damages the heart, blood vessels, eyes, kidneys, and nerves. T2DM, which accounts for more than 90% of cases of diabetes mellitus, is characterized by tissue insulin resistance (IR), insufficient compensatory insulin secretory response, and insufficient insulin secretion by pancreatic islet β-cells. Hyperglycemia results from insulin secretion's inability to maintain glucose homeostasis as the disease progresses. Obesity or a higher body fat percentage, primarily in the abdominal area, are the main characteristics of patients with type 2 diabetes. Through a number of inflammatory processes, such as elevated production of free fatty acids (FFA) and dysregulation of adipokines, adipose tissue in this situation stimulates IR. The global increase in obesity, sedentary lifestyles, high-calorie meals, and population aging are the primary causes of the type 2 diabetes epidemic, which has doubled the disease's incidence and prevalence. The liver, skeletal muscle, kidneys, brain, small intestine, adipose tissue, and the pancreas (β-cells and α-cells) are among the organs implicated in the development of type 2 diabetes. Adipokine

dysregulation, inflammation, and changes in gut microbiota have been identified as significant pathophysiological variables based on evolving data[1, 10, 53-56].

Cardiovascular disease (CVD) is the leading cause of morbidity and death in those with type 2 diabetes (T2DM), and patients with this condition have a 15% higher risk of dying from all causes than those without diabetes. A meta-analysis found that diabetes is linked to a higher risk of coronary heart disease (hazard ratio [HR] 2.00; 95% CI 1.83–2.19), ischemic stroke (HR 2.27; 1.95–2.65), and other deaths from vascular disease (HR 1.73; 1.51–1.98).

5. Conclusions: Based on our entire study, we conclude that the weight reducing program improves the quality of life and selected biochemical parameters of obese type 2 diabetics.

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