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Systemic Inflammation and Oxidative Stress Biomarkers Response to Life Style Modification among Obese Patients with Non-Alcoholic Steatohepatitis

Essam H Jiffri*

Department of Medical Laboratory Technology, Faculty of Applied Medical Sciences, King Abdulaziz University, Saudi Arabia

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***Corresponding author:** Essam H Jiffri, Department of Medical Laboratory Technology, Faculty of Applied Medical Sciences, King Abdulaziz University, P.O. Box 80324, Jeddah, 21589, Saudi Arabia, Email: ejiff@hotmail.com

Abstract

Background: Oxidative status may be an influential factor for increasing the progress and decreasing the effectiveness of nonalcoholic steatohepatitis treatment.

Objective: This study aimed to examine effects of weight reducing program on inflammatory cytokines and oxidative stress markers among obese Saudi patients with non-alcoholic steatohepatitis.

Material and Methods: Eighty obese patients with non-alcoholic steatohepatitis participated in this study, mean age was 44.27±3.46 year and body mass index was 32.68±2.95kg/m^2. All Subjects were included in two groups: The first group received life style modification in the form of treadmill aerobic exercises in addition to diet control where, the second group received no therapeutic intervention. Parameters of body mass index (BMI), tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), conjugated dienes (CD), malondialdehyde (MDA), glutathione peroxidase (GPx), superoxide dismutase (SOD) and glutathione (GSH) were measured before and after 6 months at the end of the study.

Results: The mean values of BMI, TNF-α, IL-6, CD and MDA were significantly decreased, while the mean values of GPx, SOD and GSH were significantly increased in patients of group (A) as a result of weight loss, while changes were not significant in group (B). Also, there were significant differences between mean levels of the investigated parameters in group (A) and group (B) at the end of the study.

Conclusion: Within the limit of this study, life style modification modulates inflammatory cytokines and oxidative stress markers among obese Saudi patients with non-alcoholic steatohepatitis.

Keywords: Obesity; Non-alcoholic steatohepatitis; Oxidative stress; Cytokines; Life style modification

Introduction

Non-alcoholic steatohepatitis (NASH) has emerged as a serious public health burden, where the estimated worldwide pre-valences of NASH range from 3%-5% [1,2]. Whereas non-alcoholic fatty liver disease (NAFLD) can progress to cirrhosis in 2% to 3%, NASH has an increased risk for the progression to cirrhosis at 15% to 20% and predisposes patients to the development of hepatocellular carcinoma and increased mortality [3,4]. Non-alcoholicsteatohepatitis (NASH) is a chronic progressive liver disease characterized by accumulation of fat in the liver accompanied by necroinflammation and hepatocellular injury [5]. In all probability NASH prevalence figures will rise in the future as NASH is considered the hepatic manifestation of the metabolic syndrome and the number of overweighed individuals is growing [6,7]. The prevalence of NASH increased dramatically in the last few years as a consequence of excessive consumption of high-caloric food and/or sedentary life style [8,9]. NASH is characterized, among other factors, by aberrant hepatic lipid droplet accumulation, pro-inflammatory cellular environment and insulin resistance [10,11].

The incidence of hepatic steatosis and NASH has increased dramatically in parallel with the underway obesity epidemic that currently afflicts two thirds of Americans [12]. Approximately 40 million American adults are estimated to have hepatic steatosis or NASH with as many as 58-74% of obese individuals being afflicted [13]. At present, there are no validated treatments for these diseases beyond co-morbidity management. Dietary modification and weight loss are first lines of treatment [13], but poor compliance limits their effectiveness [14]. Oxidative

stress has been identified as a central mechanism contributing to hepatic damage in NASH [13,15].

Oxidative stress is also suggested to play a significant role in progression of steatosis to NASH, and the pathogenesis of NASH includes insulin resistance, increased inflammation, tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6) and increased oxidative damage [16,17]. Moreover, inflammatory cytokines, including TNF-α, also contribute to mitochondrial dysfunction by interfering with the mitochondrial respiratory chain and by forming superoxide anion [18].

Lifestyle intervention with diet and exercise is still the mainstay in the management of patient with NAFLD [19]. Commonly individuals are recommended to restrict caloric intake by approximately 500-1000kcal/day in conjunction with regular interactions with a dietician [20,21]. Several studies indicate that a reduction of between 7 and 10% of body weight is associated with a reduction in inflammation in the setting of NAFLD, and thus is set as a target [22,23]. While exercise in isolation has not been proven to be effective, as part of dietary changes moderate intensity exercise such as brisk walking of 30-45 minutes per day can improve biochemical and histological aspects of NAFLD [23]. Moreover, lifestyle modification study consisting of dietary restriction plus aerobic exercise led to significant decrease in adipose tissue, lipid peroxidation and a significant increase in adiponectin level [24]. As there is limitation in studies reporting the benefits of lifestyle modification on oxidative stress markers among obese patients with NASH. This study aimed to examine effects of weight reducing program on inflammatory cytokines and oxidative stress markers among obese Saudi patients with NASH.

Patients and Methods

Subjects

This study was carried out on a sample of eighty consecutive obese patients with NASH, were selected from gastroenterology outpatient clinic, King Abdulaziz University Hospital, Jeddah, Saudi Arabia, mean age was 44.27±3.46 year and body mass index was 32.68±2.95kg/m2. Exclusion criteria were: (1) advanced liver cirrhosis; (2) hepatocellular carcinoma; (3) other causes of liver disease or mixed etiologies (excessive alcohol consumption, hepatitis B, hepatitis C, autoimmune liver disease, Wilson's disease, hemochromatosis, or alpha1-antitrypsin deficiency); (4) human immunodeficiency virus infection; (5) previous treatment with antiviral therapy, immunosuppressive drugs, and/or regular use of drugs influencing lipid metabolism and/or oxidative stress; (6) active intravenous drug addiction. This study was approved by the Ethics Committee of King Abdulaziz University Hospital, Jeddah, Saudi Arabia and all patients gave informed consent for participation in this study. All subjects underwent a routine clinical examination, including physical examination, biochemical tests, and liver ultrasonography.

All participants will be free to withdraw from the study at any time. All participants were divided in to two equal groups: Group (A): received weight reduction program in the form of treadmill aerobic exercises in addition to diet control, where group (B): received no therapeutic intervention.

Measurements

The following measurements were taken before the study and after 6 months at the end of the study.

Measurement of oxidative stress markers and anti-oxidant status: For all participants serum (from 10ml blood in plain vial) and plasma (from 5ml blood in EDTA vial) were separated from the sample within 30min of collection and was stored in pyrogen free polypropylene cryo-tubes at (-80ºC) until analysis. Assessment of lipid markers for peroxidation such as malondialdehyde (MDA) and conjugated dienes (CD) were determined according to Buege & Aust [25]. However, Anti-oxidant status, glutathione (GSH) that was determined by the method of Beutlerand colleagues [26], in the other hand, glutathione peroxidase (GPx) and superoxide dismutase (SOD) were measured by the method of Nishikimi et al. [27].

Measurement of inflammatory cytokines: Venous blood samples after a 12-hours fasting were centrifuged at 4 °C (1000 X g for 10 min). Interleukin-6 (IL-6) levels were analyzed by "Immulite 2000" immune-assay analyzer (Siemens Healthcare Diagnostics, Deerfield, USA). However, tumor necrosis factor-alpha (TNF-α) levels was measured by ELISA kits (R&D, USA) by using ELISA technique (ELX 808; Bio-Tek Instruments, USA).

Body mass index (BMI): The participants were measured whilst wearing their undergarments and hospital gowns. Height was measured with a digital stadiometer to the nearest 0.1cm (JENIX DS 102, Dongsang, South Korea). Body weight was measured on a calibrated balance scale to the nearest 0.1kg (HC4211, Cas Korea, South Korea), and BMI was calculated as BMI =Body weight / (Height) [2].

Procedure

Following the previous evaluation, all patients will be divided randomly into the following groups: The training group (Group A) received aerobic exercise training for 6 months on the treadmill (EnrafNonium, Model display panel Standard, NR 1475.801, Holand) which was conducted according to recommendation of aerobic exercise application approved by the American College of Sports Medicine [28]. Training program will include 5 minutes for warming–up in the form of range motion and stretching exercises , 30 minutes of aerobic exercise training with intensity equal 60-70% of the individual maximum heart rate followed by cooling down for 10 minutes (on treadmill with low speed and without inclination). Participants had 3 sessions /week for 6 months with close supervision of physical therapist. Also, a dietician performed an interview-based food survey for all participants of group (A) for detection of feeding habits, abnormal dietary behavior and to prescribe the balanced low caloric diet [29] that provided 1200Kilocalories/day for 6 months. The same dietitian continuously monitored

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all participant caloric intakes through reviewing the detailed record of food intake every 2 weeks [30,31]. The control group (Group B) received no exercise intervention or diet regimen.

Statistical Analysis

The mean values of the investigated parameters obtained before and after three months in both groups were compared using paired "t" test. Independent "t" test was used for the comparison between the two groups (P<0.05).

Results

Sixty obese patients with NASH completed the screening evaluation and underwent randomization. The baseline characteristics of the patients who underwent randomization, none of the baseline characteristics differed significantly between the two groups as listed in Table 1.

The mean values of body mass index (BMI), TNF-α, IL-6, conjugated dienes (CD) and malondialdehyde (MDA) were significantly decreased, while the mean values of glutathione peroxidase (GPx), superoxide dismutase (SOD) and glutathione (GSH) were significantly increased in patients of group (A) (Table 2), while changes were not significant in group (B) (Table 3). Also, there were significant differences between mean levels of the investigated parameters in group (A) and group (B) at the end of the study (Table 4).

Table 1: Baseline clinical participants' characteristics in both groups.

	Mean±SD		Significance
	Intervention group	Control group	
Age (year)	45.27±6.81	43.67±5.93	P >0.05
Gender (male/female)	24/16	23/17	P >0.05
BMI (kg/m^2)	34.81±3.24	33.52±2.65	P >0.05
SBP (mm Hg)	145.14±10.12	142.75±8.83	P >0.05
DBP (mm Hg)	88.43±5.22	86.24±5.61	P >0.05
Total cholesterol (mg/dl)	195.15±13.11	193.48±12.32	P >0.05
HDL-C (mg/dl)	34.27±4.18	36.51±4.77	P >0.05
LDL-C (mg/dl)	120.12±9.61	117.93±8.25	P >0.05
Triglycerides (mg/dl)	156.43±10.24	154.72±11.13	P >0.05
AST (IU)	64.21±5.75	63.58±4.92	P >0.05
ALT (IU)	51.73±4.82	48.64±4.18	P >0.05

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance Index; HDL-c: High Density Lipoprotein Cholesterol; LDL-c: Low Density Lipoprotein Cholesterol; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase

Table 2: Mean value and significance of BMI, IL-6, TNF-α, GPx, SOD, GSH, MDA and CD in-group (A) before and at the end of the study.

	Mean ±SD		Significance
	Before	After	
BMI (kg/m^2)	34.81±3.24	25.21±2.93*	P<0.05
TNF- α(pg/mL)	15.36±2.51	12.75±2.18*	P<0.05
IL-6 (pg/mL)	4.52±1.24	3.19±1.16*	P<0.05
CD (mmol/L)	23.48±4.63	18.15±3.27*	P<0.05
MDA (mmol/L)	24.11±5.12	19.43±4.15*	P<0.05
GPx(units/gHb)	20.26±3.94	25.80±4.13*	P<0.05
SOD (units/mL)	42.35±6.22	51.27±6.51*	P<0.05
GSH (mmol/gHb)	2128.43±151.25	2561.31±182.72*	

BMI: Body Mass Index; TNF-α: Tumor Necrosis Factor -alpha; IL-6: Interleukin-6; CD: Conjugated Dienes; MDA: Malondialdehyde; GPx: Glutathione Peroxidase; SOD: Superoxide Dismutase; GSH: Glutathione;

*Significant level (p<0.05).

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Table 3: Mean value and significance of BMI, IL-6, TNF-α, GPx, SOD, GSH, MDA and CD in-group (B) before and at the end of the study.

	Mean+SD		Significance
	Before	After	
BMI (kg/m^2)	33.52±2.65	34.84 ± 3.19	P >0.05
TNF- α(pg/mL)	14.17±2.43	15.14 ± 2.52	P >0.05
IL-6 (pg/mL)	4.26±1.45	4.91 ± 1.63	P >0.05
CD (mmol/L)	23.12±4.37	23.84 ± 4.50	P >0.05
MDA (mmol/L)	23.79±5.38	24.25 ± 5.42	P >0.05
GPx(units/gHb)	20.87±3.72	20.19 ± 3.58	P >0.05
SOD (units/mL)	43.21±5.83	42.65 ± 5.75	P >0.05
GSH (mmol/gHb)	2158.16±160.27	2137.22 ± 157.11	P >0.05

BMI: Body Mass Index; TNF- α: Tumor Necrosis Factor -Alpha; IL-6: Interleukin-6; CD: Conjugated Dienes; MDA: Malondialdehyde; GPx: Glutathione Peroxidase; SOD: Superoxide Dismutase; GSH: Glutathione.

Table 4: Mean value and significance of BMI, IL-6, TNF-α, GPx, SOD, GSH, MDA and CD in group (A) and group (B) at the end of the study.

	Mean +SD		Significance
	Group (A)	Group (B)	
BMI (kg/m2)	25.21±2.93*	34.84±3.19	P<0.05
TNF- α(pg/mL)	12.75±2.18*	15.14±2.52	P<0.05
IL-6 (pg/mL)	3.19±1.16*	4.91±1.63	P<0.05
CD (mmol/L)	18.15±3.27*	23.84±4.50	P<0.05
MDA (mmol/L)	19.43±4.15*	24.25±5.42	P<0.05
GPx(units/gHb)	25.80±4.13*	20.19±3.58	P<0.05
SOD (units/mL)	51.27±6.51*	42.65±5.75	P<0.05
GSH (mmol/gHb)	2561.31±182.72*	2137.22±157.11	P<0.05

BMI: Body Mass Index; TNF- α: Tumor Necrosis Factor -Alpha; IL-6: Interleukin-6; CD: Conjugated Dienes; MDA: Malondialdehyde; GPx: Glutathione Peroxidase; SOD: Superoxide Dismutase; GSH: Glutathione;

*Significant level (p<0.05).

Discussion

Non-alcoholic steatohepatitis (NASH) can lead to advanced fibrosis, hepatocellular carcinoma, and end-stage liver disease requiring liver transplantation [32]. Oxidative stress (OS) caused by reactive oxygen species is, however, known to be of major importance in the progression of this disease [33]. As oxidative stress (OS) seems to be the major factor in the disease, it is clear that by blocking OS, the disease could be halted. Therefore this study aimed to examine effects of 6 months weight reducing program on inflammatory cytokines and oxidative stress markers among obese Saudi patients with non-alcoholic steatohepatitis. The main finding of the present study was that weight reducing program ameliorated inflammatory cytokines (TNF-α and IL-6) and markers of oxidative and anti-oxidative stress (MDA, CD, CPX, GSH and SOD) in obese patients with NASH as a result of weight loss, these results are in line with many previous studies.

Results of our study agreed with Dandona et al. [34] who reported that weight loss reduces TNF-α in obese. Also, Sandoval and Davis approved that patients who had bariatric surgery gained reduction in IL-6 concentration and improved insulin sensitivity in parallel to weight loss [35]. Also, Balagopal et al. [36] reported that obese adolescents who underwent a 3-month lifestyle intervention of enhanced physical activity and nutrition habits had decreased body fat percentage, insulin resistance and IL-6. Likewise, Sheu et al. [37] reported that 5% of body weight loss obtained after 12 weeks of caloric restriction and exercises resulted in significant reduction in TNF-α and IL-6 of obese women. Selvin et al. [38] clearly stated that in their systemic review that weight loss was associated with a decrease in sCRP in these subjects and related to the amount of weight loss. Moreover, You and Nicklas &Nicklas and colleagues stated that weight loss leads to reductions in circulating IL-6, TNF-α and CRP levels regardless of the way in which the weight loss was achieved, including hypocaloric dietary intake, exercise, or liposuction [39,40]. Lang et al. [41] established that a weight-reducing program had anti-atherogenic and inflammatory effects in their study on three obese men and eleven obese women for eight weeks. The three possible mecha¬nisms of exercise anti-inflammatory effects include reduction in visceral fat mass [42]; reduction in the circu¬lating numbers of pro-inflammatory monocytes [43] and an increase in the circulating numbers of regulatory T cells [44].

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Concerning the markers of oxidative and anti-oxidative stress, the observation in this study indicated a significant reduction in MDA& CD and increased in CPX, GSH and SOD as a result of weight loss at the end of the study. Nevertheless, the current data are in line with one previous study by Roberts et al. [45] Proved that after three weeks of combination between diet and exercise there was a significant reduction in BMI, lipid profile, fasting blood sugar, C-reactive protein and insulin homeostasis which ameliorates oxidative stress, inflammation and monocytes-endothelial interaction among diabetic patients. However, retrospective data from 169 obese, middle-aged men who were enrolled in a 12-week weight reduction program through lifestyle modification consisting of dietary restriction plus aerobic exercise showed significant decrease in lipid peroxidation and a significant increase in adiponectin level [24].

The possible mechanism for modulation of the oxidative stress markers induced by weight reduction could be due to reverse the mechanism by which obesity produces oxidative stress, which include reverse of mitochondrial and peroxisomal oxidation of fatty acids along with reduction of over-consumption of oxygen that generates free radicals in the mitochondrial respiratory chain that is found coupled with oxidative phosphorylation in mitochondria [46].

Conclusion

Weight loss ameliorates inflammatory cytokines and oxidative stress markers in obese patients with NASH.

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