Aerobic exercises versus low-caloric diet for mild fatty liver patients with central obesity
Hany F.E.M. Elsisia, Fatma M.M. Abdeen Sallamaa, Awny F. Rahmya, Mohammed A. Sweilamb

Background and aims
Exercise and weight loss are the first-line therapy for patients with mild fatty liver with central obesity. We aimed to (i) summarize a moderate aerobic exercise program and low-caloric diet program and (ii) to compare the effect of moderate aerobic exercise program versus low-caloric diet program on liver biochemistry. The purpose of this study was to determine the response of liver enzymes to aerobic exercise versus low-caloric diet in mild fatty liver patients with central obesity.

Patients and methods
The study was carried out on 40 patients with mild fatty liver, central obesity, and elevated liver enzymes. Their ages ranged between 30 and 45 years; the patients were assigned to two groups equal in number. Group 1 received a program of moderate intensity aerobic exercise on electronic treadmill (60–70% of maximum heart rate) for 50 min three times a week for 8 weeks. Group 2 followed a program of low-caloric diet for 8 weeks. Liver enzymes, BMI, waist circumference, and fat content were measured for both groups before and after program.

Results
The results of this study showed that there were no significant (P>0.05) differences between the findings of both groups. However, there were significant (P<0.05) decreases in all outcome measures after treatment compared with that before treatment in both groups except for the BMI.

Conclusion
Moderate intensity aerobic exercise and low-caloric diet are effective in decreasing liver enzymes, waist circumference, and fat content in patients with mild fatty liver.

Keywords:
aerobic exercise, fatty liver, liver enzymes, low caloric diet

Introduction
The liver is the largest internal and most metabolically complex organ in humans. It performs over 500 different functions, including fighting of infection, neutralizing toxins, manufacturing proteins and hormones, controlling blood sugar, and helping to clot the blood [1].

Mild fatty liver or simple hepatic steatosis is a condition of excessive triglyceride (TG) accumulation in the liver. Fatty acids stored in adipose tissue and de novo lipogenesis of fatty acids within the liver are the major source of TGs. Increased influx of free fatty acids to the liver stimulates the hepatic glucogenesis and synthesis of TG, impairs the ability of insulin to suppress hepatic glucose output, and affects the other metabolic insulin effects [2].

The increased prevalence of obesity has heralded a rise in nonalcoholic fatty liver disease (NAFLD). It is estimated that 20–30% of adult populations in developed countries have NAFLDs, and, although high-quality data are currently lacking, the condition is clearly increasing in children also. NAFLD should be suspected in those with commonly available simple clinical signs and biochemistry consistent with insulin resistance [3], and the estimated prevalence of NAFLD is 38.5% in obese Egyptian children and adolescents [4].

Liver enzyme elevation can only be used as a crude estimate of the presence of fatty liver disease. In hepatic steatosis or nonalcoholic steatohepatitis, most patients have no signs of liver disease at the time of diagnosis. If present, the symptoms are nonspecific and do not correlate well with the severity of the disease. Most commonly, fatigue or malaise and a right upper quadrant pain or sensation of fullness are reported.

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In addition, hepatomegaly can be found on physical examination [5].

A 4-week aerobic exercise program results in a significant reduction in visceral adipose tissue, thus positively affecting the levels of circulating free fatty acids and hepatic lipid accumulation, but appears to be too short a time frame to reduce insulin resistance. Unfortunately, the disruption of inflammatory biomarkers has been not addressed by Johnson et al. [6]. Promrat et al. [7] were able to demonstrate this, providing evidence that patients undergoing consistent abdominal adipose tissue loss have improved lobular inflammation and also reduced insulin resistance [8].

Obese patients are advised to achieve a gradual and sustained weight loss through proper nutrition and exercise. For patients with nonalcoholic steatohepatitis who are not overweight and not diabetic, a low-fat diet is often recommended. It is also recommended that people avoid drinking alcohol as it can cause and contribute to fatty liver disease. Patients with fatty liver disease should see their primary healthcare providers on a regular basis [9].

It was reported that short-term calorie restriction decreases intrahepatic TG before significant weight loss. It is clear that weight loss from reduced calorie intake is effective in reducing intrahepatic TG. However, similar to the overfeeding studies, it is not clear whether this benefit is due to weight loss or ongoing negative caloric balance [10].

It was hypothesized that there may be no significant difference between the effects of aerobic exercises and low-caloric diet on fatty liver patients with central obesity.

**Outcome measures**
Both groups underwent an identical battery of tests: baseline (before training) and after 8 weeks (after training). Initially, data on the participants’ characteristics were collected in the first session, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST) using 3 cm of blood for blood analysis. Weight was measured using a standard weight scale. Height was measured with the participant standing in a vertical scale. BMI and fat content were measured using the Body Stat device (Bodystat Ltd. P.O. Box 50 Douglas isle of Man IM99 1DQ British, Isles). Waist circumference (WC) was measured using simple tape measurement from the level of umbilicus. The primary outcome measures in this study were ALT and AST, and the secondary outcome measures were BMI, fat content, and WC.

**Sample size calculation**
A preliminary power analysis [power (1 α error P) = 0.85, α=0.01, effect size=0.5] determined a sample size of 40 for this study. This effect size was chosen because it yielded a realistic sample size.

**Procedures of the study**
Initial medical screening was performed for every patient by the physician, diagnosed as mild fatty liver by means of abdominal ultrasound by the radiologist and liver enzymes were measured in the laboratory.

Initially, 74 patients were selected from the outpatient clinic of internal medicine of El-Santa Hospital from March 2015 to June 2016 and identified as potentially eligible for the study as mild fatty liver patients with central obesity. On blood analysis for measuring liver enzymes for each patient, 34 patients were found to be ineligible according to inclusion and exclusion criteria and therefore excluded. Forty patients were found to be eligible and already participated in the study. All 40 participants had completed the 8 weeks’ program to the end. The study was conducted at Abed Physical Therapy Center in El-Santa, Gharbia governorate, Egypt.

Eligibility criteria: all participants had mild fatty liver with liver enzymes above accepted cutoffs with central obesity, free from any immunodeficiency disorders or diseases that can affect the treatment process. Criteria of fatty liver: patients were diagnosed with mild fatty liver using abdominal ultrasound when more than 5–10% of liver cells were filled with fat droplets with mild elevated liver enzymes with cutoffs of 20 U/l for female and 30 U/l for male patients.

**Patients and methods**
Forty patients with mild fatty liver and central obesity between 30 and 45 years of age were screened and selected randomly to be enrolled into this 8-week blinded randomized comparative clinical trial. They were recruited from outpatient clinics of internal medicine of El Santa Hospital to participate in this study. This study was approved by the Ethics Committee for Scientific Research of the Faculty of Physical Therapy, Cairo University No: P.T.REC/012/00818. Instructions and details of the study were explained for each patient. Patients who accepted to participate in the study were asked to sign a consent form Appendix 1.
The 40 eligible participants were randomly assigned to two groups equal in number, groups 1 and 2. It was a randomized trial using sealed envelope allocation sequence method. Group 1 received a program of moderate intensity aerobic exercise. Group 2 followed a program of low-caloric diet program. Each patient had been re-evaluated after 8 weeks of their treatment program for BMI, body fat content, WC, ALT, and AST.

**Equipment**

(1) Assessment equipment:
- (a) Height and weight scale: RGZ 220 (China).
- (b) Tape measurement: 150 cm.
- (c) Body Stat 1500: SN 213056 (UK).
- (d) Liver enzyme measuring machine: Microlab 300 SN 11-5046 (France).
- (e) Liver enzyme measuring kits: BioSystems (Spain).
- (f) Ultrasonography: SonoScope SN 3322415 made in China.

(2) Therapeutic equipment:
- Electronic treadmill: Grand Fit AC 999 (China).

Aerobic exercise is defined as any activity that uses large muscle groups working continuously for a long duration and rhythmic in nature with moderate intensity. Aerobic means ‘with oxygen’, involves or improves oxygen consumption by the body, and refers to the use of oxygen in a muscle’s energy-generating process. Many types of exercises are aerobic and by definition are performed at moderate levels of intensity for extended periods of time [11].

**Training program**

The purpose of training program was explained to each patient. Each patient participated in the exercise training program on electronic treadmill with moderate intensity for 30–50 min three times a week day after day for 8 weeks.

**Phases of exercise**

(1) Warming up phase: 5–10 min with mild intensity [30–40% maximum heart rate (HRmax)].

\[ HR_{max} = 211 - 0.64 \text{ age} \ [12] \]

(2) Training phase: each patient trained for 20–30 min with moderate intensity (60–70% HRmax).

(3) Cooling down phase: 5–10 min with mild intensity (30–40% HRmax).

**Low-caloric diet program**

In a professional review, a low-caloric diet of about 1200–1500 kcal/day is recommended for patients with Nonalcoholic Steatohepatitis (NASH) [13]. A diet of 1400 kcal/day could be effective in improving histology in patients with biopsy-proven NASH [14].

In this study, four different graduated low-caloric diet programs were established. Every 2 weeks the diet program had been changed gradually from 1800 kcal/day to 1500 to 1200 to 1000 kcal.

Following is an example for 1800 kcal/day diet program:

**Breakfast:** One leaf of dark bread+three spoonfuls of beans with olive oil+light cheese+two vegetables.
**Snack:** One fruit+small cup of coffee or tea with a teaspoonful of sugar.
**Lunch:** Nine spoonfuls of rice or macaroni+green salad +250 g of any type of skinned protein (fish, tuna, meat, chicken, and, liver).
**Snack:** One fruit+a cup of cinnamon with a teaspoonful of sugar.
**Dinner:** Half loaf of dark bread+boiled egg+grilled or boiled eggplant+two vegetables.
**Snack:** One fruit+a cup of green tea.

Calorie intake was gradually reduced every 2 weeks until reaching 1000 kcal/day at the last 2 weeks.

**Statistical analysis**

Results are expressed as mean±SD or number and percent. Comparison between categorical data \([n \%]\) was made using the \(\chi^2\)-test. According to test of normality, comparison between different variables in the two groups was performed using either the unpaired \(t\)-test or the Mann–Whitney test whenever it was appropriate. Within-group comparison (pretreatment vs. post-treatment) was performed using either the paired \(t\)-test or the Wilcoxon signed-ranks test whenever it was appropriate. Statistical package for the social sciences (SPSS) (IBM corporation in Armonk, New York, USA) computer program (version 19 Windows) was used for data analysis. A \(P\) value less than or equal to 0.05 was considered significant.

**Results**

There were no statistically significant differences between the two groups at baseline in the mean values of age, height, and weight.

**Comparison between the mean values of body mass index in the two studied groups measured before and after treatment**

*Within-group comparison*

In the exercise group, there was a statistically significant difference in the mean value of BMI measured before treatment (44.24±5.47 kg/cm²)
when compared with its corresponding value after treatment (43.56±5.46 kg/cm²) ($P=0.002$).

In the diet group, there was no statistically significant difference between the mean value of BMI measured before treatment (43.41±4.97 kg/cm²) and its corresponding value after treatment (42.24±5.17 kg/cm²) ($P=0.436$).

The percentage of decrease in BMI in both the exercise and diet groups was 1.54 and 2.17%, respectively.

**Between-group comparison**
Before treatment, there was no statistically significant difference between the mean value of BMI in the exercise group (44.24±5.47 kg/cm²) and its corresponding value in the diet group (43.41±4.97 kg/cm²) ($P=0.615$). After treatment, there was no statistically significant difference between the mean value of BMI in the exercise group (43.56±5.46 kg/cm²) and its corresponding value in the diet group (42.24±5.17 kg/cm²) ($P=0.436$) (Fig. 1).

**Comparison between mean values of waist circumference in the two studied groups measured before and after treatment**

**Within-group comparison**
In the exercise group, there was a statistically significant difference in the mean value of WC measured before treatment (124.50±10.50 cm) when compared with its corresponding value after treatment (120.30±10.29 cm) ($P=0.001$).

In the diet group, there was a statistically significant difference between the mean value of WC measured before treatment (124.00±6.81 cm) and its corresponding value after treatment (119.55±7.90 cm) ($P=0.001$).

The percentage of decrease in WC in both the exercise and diet groups was 3.37 and 3.59%, respectively.

**Between-groups comparison**
Before treatment, there was no statistically significant difference between the mean value of WC in the exercise group (124.50±10.50 cm) and its corresponding value in the diet group (124.00±6.81 cm) ($P=0.859$). After treatment, there was no statistically significant difference between the mean value of WC in the exercise group (120.30±10.29 cm) and its corresponding value in the diet group (119.55±7.90 cm) ($P=0.797$) (Fig. 2 and Table 1).

**Comparison between mean values of fat content in the two studied groups measured before and after treatment**

**Within-group comparison**
In the exercise group, there was a statistically significant difference in the mean value of fat measured before treatment (56.82±12.42 kg) when compared with its corresponding value after treatment (54.17±12.26 kg) ($P=0.001$).

In the diet group, there was a statistically significant difference between the mean value of fat measured before and after treatment (43.45±12.68 kg) when compared with its corresponding value after treatment (41.57±12.62 kg) ($P=0.001$).

The percentage of decrease in fat in both the exercise and diet groups was 3.76 and 3.84%, respectively.

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**Figure 1**
Mean values of BMI in both groups measured pre- and post-treatment

**Figure 2**
Mean values of WC in both groups measured pre- and post-treatment

**Table 1** Comparison between mean values of body mass index and waist circumference in the two studied groups measured before and after treatment

<table>
<thead>
<tr>
<th></th>
<th>Exercise (n=20)</th>
<th>Diet (n=20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>44.24±5.47</td>
<td>43.41±4.97</td>
<td>0.615 (NS)</td>
</tr>
<tr>
<td>Post-treatment</td>
<td>43.56±5.46</td>
<td>42.24±5.17</td>
<td>0.436 (NS)</td>
</tr>
<tr>
<td>Mean difference</td>
<td>0.68</td>
<td>1.17</td>
<td></td>
</tr>
<tr>
<td>% Change</td>
<td>1.54↓↓</td>
<td>2.7↓↓</td>
<td></td>
</tr>
<tr>
<td>t-Value</td>
<td>3.624</td>
<td>1.624</td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td>0.002 (S)</td>
<td>0.121 (NS)</td>
<td></td>
</tr>
<tr>
<td><strong>WC</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>124.50±10.50</td>
<td>124.00±6.81</td>
<td>0.859 (NS)</td>
</tr>
<tr>
<td>Post-treatment</td>
<td>120.30±10.29</td>
<td>119.55±7.90</td>
<td>0.797 (NS)</td>
</tr>
<tr>
<td>Mean difference</td>
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<tr>
<td>% Change</td>
<td>3.37↓↓</td>
<td>3.59↓↓</td>
<td></td>
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<tr>
<td>t-Value</td>
<td>12.178</td>
<td>10.156</td>
<td></td>
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<tr>
<td>P value</td>
<td>0.001 (S)</td>
<td>0.001 (S)</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as means±SD. WC, waist circumference. $P<0.05$, significant (S). $P>0.05$, NS. ↓↓Decrease.
before treatment (53.79±11.77 kg) and its corresponding value after treatment (49.27±12.00 kg) 
\( (P=0.001)\).

The percentage of decrease in fat in both the exercise and diet groups was 4.66 and 8.40%, respectively.

**Between-group comparison**
Before treatment, there was no statistically significant difference between the mean value in the exercise group (56.82±12.42 kg) and its corresponding value in the diet group (53.79±11.77 kg) \( (P=0.433)\). After treatment, there was no statistically significant difference between the mean value of the exercise group (54.17±12.26 kg) and its corresponding value in the diet group (49.27±12 kg) \( (P=0.210)\) (Fig. 3 and Table 2).

**Comparison between the mean values of alanine aminotransferase (serum glutamic-pyruvic transaminase) in the two studied groups measured before and after treatment**

**Within-group comparison**
In the exercise group, there was a statistically significant difference in the mean value of ALT measured before treatment (33.90±17.53 IU/l) when compared with its corresponding value after treatment (28.90±14.80 IU/l) \( (P=0.001)\).

In the diet group, there was a statistically significant difference between the mean value of ALT measured before treatment (33.80±11.47 IU/l) and its corresponding value after treatment (27.10±8.68 IU/l) \( (P=0.001)\).

The percentage of decrease in ALT in both the exercise and diet groups was 14.75 and 19.82%, respectively.

**Between-group comparison**
Before treatment, there was no statistically significant difference between the mean value of ALT in the exercise group (33.90±17.53 IU/l) and its corresponding value in the diet group (33.80±11.47 IU/l) \( (P=0.598)\). After treatment, there was no statistically significant difference between the mean value of ALT in the exercise group (28.90±14.8 IU/l) and its corresponding value in the diet group (27.10±8.68 IU/l) \( (P=0.882)\) (Fig. 4).

**Comparison between mean values of aspartate aminotransferase (serum glutamic oxaloacetic transaminase) in the two studied groups measured before and after treatment**

**Within-group comparison**
In the exercise group, there was a statistically significant difference in the mean value of AST measured at after treatment (40.00±13.97 IU/l) when compared with its corresponding value after treatment (32.50±13.37 IU/l) \( (P=0.001)\).

In the diet group, there was a statistically significant difference between the mean value of AST measured before treatment (38.85±15.73 IU/l) and its corresponding value after treatment (31.05±11.42 IU/l) \( (P=0.001)\).

The percentage of decrease in AST in both the exercise and diet groups was 18.75 and 20.08%, respectively.
Between-group comparison
Before treatment, there was no statistically significant difference between the mean value of AST in the exercise group (40.00±13.97 IU/l) and its corresponding value in the diet group (38.85±15.73 IU/l) \((P=0.957)\). After treatment, there was no statistically significant difference between the mean value of serum glutamic-pyruvic transaminase in the exercise group (32.50±13.37 IU/l) and its corresponding value in the diet group (31.05±11.42 IU/l) \((P=0.991)\) (Fig. 5 and Table 3).

Discussion
The results of this study showed that there was no significant difference between the effects of moderate intensity aerobic exercise versus low-caloric diet on liver enzymes in fatty liver patients.

All patients had mild fatty liver with liver enzymes above 20 U/l for female and 30 U/l for male with BMI greater than or equal to 30 kg/m². There were no other diseases or problems affecting the patient except fatty liver.

Sample size of the study was calculated by means of power analysis to avoid errors. Duration of the study was determined according the previous studies as a suitable time for the treatment program [15].

Rohollah et al. [15] in their experimental research reported that 8-week aerobic exercise showed a decrease in the levels of ALT and AST.

Rusu et al. [16] reviewed that there is no consensus as to what diet or lifestyle approach is the best for NAFLD patients. However, patients with NAFLD may benefit from a moderate-to-low carbohydrate (40–45% of total calories) diet, coupled with increased dietary Mono Unsaturated Fatty Acids (MUFA) and \(n\)-3 Poly Unsaturated Fatty Acid (PUFAs) and reduced Saturated Fatty Acid (SFAs).

More Clinical Randomized Trial (CRT) is needed to clarify the specific effects of different diets and dietary components on the health of NAFLD patients. A small clinical randomized trial showed that short-term carbohydrate restriction is more efficacious in reducing intrahepatic TG.

Petersen et al. [17] showed that a low-fat diet reduced calorie intake (daily intake 1200 kcal/day) and effectively reduced body weight and intrahepatic lipid content with improvement in insulin resistance in NAFLD patients.

Wang et al. [18] reported that central obesity is strongly associated with hepatic steatosis (HS). Modification of lifestyle including dietary restriction for weight loss and control of risk factors such as type 2 diabetes mellitus and dyslipidemia is recommended as the first and most important approach to manage people with NAFLD.

Huang et al. [14] conducted a study on reducing insulin resistance and included the following recommendations for caloric intake: 40–50% carbohydrates, with emphasis on complex carbohydrates with fibers, 35–40% from fat with emphasis on monounsaturated and polyunsaturated fats, and 15–20% from protein. After 12 months, there

| Table 3 Comparison between mean values of alanine aminotransferase (serum glutamic-pyruvic transaminase) and aspartate aminotransferase (serum glutamic oxaloacetic transaminase) in the two studied groups measured before and after treatment |
| Exercise (\(n=20\)) | Diet (\(n=20\)) | \(Z\) value\(^a\) | \(P\) value |
| ALT | Pretreatment | 33.90±17.53 | 33.80±11.47 | −0.528 | 0.598 (NS) |
| Post-treatment | 28.90±14.80 | 27.10±8.68 | −0.149 | 0.882 (NS) |
| Mean difference | 5.0 | 6.7 |
| % Change | 14.75↓ | 19.82↓ |
| \(Z\) value\(^b\) | −3.933 | −3.924 |
| \(P\) value | 0.001 (S) | 0.001 (S) |
| AST | Pretreatment | 40.00±13.97 | 38.85±15.73 | −0.054 | 0.957 (NS) |
| Post-treatment | 32.50±13.37 | 31.05±11.42 | 0.002 | 0.991 (NS) |
| Mean difference | 7.5 | 7.80 |
| % Change | 18.75↓ | 20.08↓ |
| \(Z\) value\(^b\) | −3.832 | −3.709 |
| \(P\) value | 0.001 (S) | 0.001 (S) |

Data are expressed as mean±SD. ALT, alanine aminotransferase; AST, aspartate aminotransferase. \(^a\)Mann–Whitney test. \(^b\)Wilcoxon’s signed-rank test. \(P<0.05\), significant (S). \(P>0.05\), NS. ↓↓Decrease.
was histological improvement in nine of 15 patients with biopsy-proven NASH, as measured by steatosis grade and NASH score.

Solga and Alkhuraishe [19] showed that the low caloric diet and diet low in saturated fats is often recommended. However, little is known about the effects of changes in dietary composition on liver histopathology in NAFLD.

In contrast, Capristo et al. [20] reported that recommendation of a low-fat diet could actually worsen NAFLD histopathology.

Johnson et al. [6] showed that aerobic exercise training reduces hepatic and visceral lipids in obese patients with sedentary lifestyle. Thus, regular exercise may mitigate the metabolic and cardiovascular consequences of obesity, including fatty liver, and this is not contingent upon weight loss.

Sreenivasa et al. [21] showed that moderate aerobic exercise helped normalize ALT in NASH but this normalization did not occur in the 15 patients who failed to comply with the exercise program.

Stewart et al. [22] stated that aerobic exercise increases insulin sensitivity independent of weight loss, and decreases peripheral lipolysis that probably interferes with the development of steatosis, inhibiting hepatic lipid synthesis and stimulating fatty acid oxidation. A number of studies suggested that NAFLD improves after weight loss. Improvements in liver biochemistry and ultrasonography have been found with modest weight reduction, but few studies have evaluated the effect of weight reduction on hepatic histology.

Suzuki et al. [23] reported body weight loss and lifestyle modifications with changes in serum ALT levels. They found that weight loss and regular exercise resulted in improvement in serum ALT. They suggested that reducing body weight by at least 5% with subsequent weight control and exercising regularly may be beneficial in treating fatty liver.

Adams et al. [24] reported that studies showed that a sedentary lifestyle with reduced physical activity, independent of diet, is another determinant of fatty liver. Although these risk factors may successfully be modified by moderate lifestyle intervention, the existence of other risk factors most probably may necessitate more intense treatment.

Keating et al. [25] showed clear evidence for the importance of exercise therapy on liver fat but not ALT levels. This benefit is apparent with minimal or no weight loss and at exercise levels below current exercise recommendations for obesity management.

Kakarla et al. [26] reported that the exercise training by inducing antioxidant capacity protects against oxidative stress. Similar changes were reported in male animals, which clearly envisage no sex difference in the antioxidant enzyme system with regard to age and exercise. In conclusion, it can be stated that 12 weeks of treadmill exercise training has beneficial effect in improving the antioxidant defense capacity of the liver tissue by augmenting superoxide dismutase, catalase, glutathione reductase activities, and glutathione levels in older rats, thereby preventing oxidative damage to the liver tissue.

Shelby [27] reported that some findings suggested that short-term high-caloric feeding increases intrahepatic triglyceride (IHTG), whereas short-term low-caloric feeding decreases IHTG despite little change in total body weight, suggesting that ongoing excess caloric delivery directly contributes to the development of NAFLD. Weight loss with low fat or low carbohydrate diets can improve IHTG; however, specific macronutrients such as fructose, trans-fatty acids, and saturated fat may contribute to increased IHTG independent of total calorie intake. N-3 polyunsaturated fatty acids and monounsaturated fatty acids may play a protective role in NAFLD. The mechanisms behind these effects are not fully understood. Diet has an important role in the pathophysiology of NAFLD. Patients with NAFLD must be advised to reduce calorie intake with either low fat or low carbohydrate diets as well as limit intakes of fructose, trans-fatty acids, and saturated fat.

Kirk et al. [10] reported that moderate calorie restriction causes temporal changes in liver and skeletal muscle metabolism; 48h of calorie restriction affects the liver (IHTG content, hepatic insulin sensitivity, and glucose production), whereas moderate weight loss affects muscle (insulin-mediated glucose uptake and insulin signaling).

Petersen et al. [17] stated that weight loss as well as exercise is associated with improvement in insulin sensitivity and logical treatment modalities for patients with NAFLD who are overweight or obese. Weight reduction is with caloric restriction and physical exercise.
Suzuki et al. [23] showed that weight reduction and lifestyle modification with diet changes and increased physical activity are recommended as the first step in the control of NAFLD. Achieving and maintaining weight reduction may improve NAFLD, but the result of several reports is inconsistent.

Palmer and Schaffner [28] showed that, in overweight adults free from primary liver disease, a weight reduction of greater than or equal to 10% corrected abnormal hepatic test results decreased hepatosplenomegaly, and resolved some stigmata of liver disease. In similarly studied overweight patients with primary liver disease, some findings improved, but the changes did not correlate with a greater than or equal to 10% weight loss. Increased ALT activity was the most frequent hepatic enzyme abnormality in this population. For every 1% reduction in body weight, ALT activity improved by 8.1%.

In contrast, Jian-Gao et al. [29] reported that a study indicated that it might be difficult to resolve steatohepatitis by merely short-term low caloric diet therapy, long-term appropriate diet control or concurrent administration of medications that can directly reduce the severity of liver damage may be reasonable alternatives for the treatment of NASH patients with obesity.

In contrast, Selezneva et al. [30] reported that low-caloric diet had poor compliance and little effect on ALT and AST during short-term diet intervention in NASH patients. Diet modifications should be reconsidered in NASH patients in favor of fewer calories restricted and easy to follow diet.

In rehabilitation clinics, doctors should advise obese mild fatty liver disease patients with mild elevated liver enzymes to follow regular moderate aerobic exercise or follow low-caloric diet according to general health status, lifestyle, co-operation, and psychological state of each patient. Both low-caloric diet and moderate aerobic exercise improve liver histology and help in normalizing liver biochemistry.

**Conclusion**

It was concluded that there was no significant difference between results of both groups. After the treatment both low caloric diet and moderate aerobic exercise showed a decrease in AST, ALT, WC, and fat content with no significant changes in the BMI compared with that before treatment.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

Appendix I

Consent form

I am freely and voluntarily consent to participate in this research study under the direction of the researcher/Fatma Mohammed Mohammed Abdeen Sallam.

Through description of the study procedure has been explained to me and I understand that I may withdraw my consent and discontinue participation in this research study at any time without prejudice to me.

Participant:

Date: