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Original Research Article

Hepatic Physiological Consequences of Diet-Induced Obesity in Male Rats

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Abstract: The present research was performed using animals at the College of Veterinary Medicine, Tikrit University during the period between September 1, 2025, and December 1, 2025, in the animal house of the college under standard laboratory conditions of rearing animals. This study used male laboratory rats. The males were selected because they are larger and having a more stable weight than the females hence reducing hormonal interference that would have influenced the outcome of the study. At the beginning of the experiment, the weight of the rats was 250 to 300 grams. Pre-weighing of the animals was done prior to feeding to find out the initial weight of each rat. Monitoring was done over the 60 day period of rearing whereby under appropriate environment they received free access to water and feed. ELISA was used to measure liver enzymes (ALT, AST, and ALP), total cholesterol and triglycerides in serum, according to the laboratory standards. The levels of GOT (AST) and GPT (ALT) were significantly raised at the end of the feeding period, which means that the cells of the liver were damaged by the fat accumulation inside of the cells and increased the cell membrane permeability. Conversely, the level of the enzyme alkaline phosphatase (ALP) did not show any significant change, which means that the pathological effect of obesity was not serious at the initial stages and did not spread to the bile ducts. The findings also presented the fact that the total cholesterol and triglyceride levels had been significantly increased. It may be concluded that dietary induced obesity is one of the key causes of lipid imbalance and hepatocellular damages, and can be one of the initial phases in the evolution of metabolic liver diseases in the event that exposure to causative factors is sustained.

Keywords: Obesity, High-Fat Diet, Liver Enzymes, Lipid Imbalance, Rats.

INTRODUCTION

In terms of body mass and glucose metabolism, obesity is a severe metabolic disorder characterized by increases in energy consumption and decreases in energy expenditure [1]. Diabetes and coronary heart disease [2], sleep apnea and pulmonary dysfunction, stroke, gallbladder, liver, and musculoskeletal system diseases, reproductive dysfunction, venous insufficiency, deep vein thrombosis, poor wound healing, etc. are some of the major issues linked to obesity [3]. Obesity must be considered a low-grade inflammatory disease because the majority of obese patients showed high levels of the inflammatory markers interleukin-6 and tumor necrosis factor-alpha [4]. Cancer, endometrial, colon, and prostate can all be brought on by obesity. The fast-paced lifestyle that results in an excessive intake of fats and carbohydrates and a decrease in energy expenditure is the cause of the obesity trend in contemporary society [5]. Like in most other nations, more than 50% of people are overweight or obese. Hyperlipidemia and insulin resistance are linked to obesity [6]. Obese rats have been shown to have hyperinsulinemia and hepatic glucose production, and an increase in visceral adipose tissue is frequently associated with insulin resistance [7]. It has long been known that the consequences of insulin resistance are closely linked to nonalcoholic fatty liver disease. Obese individuals with type 2 diabetes mellitus and hyperlipidemia account for the majority of NAFLD cases [8]. NAFLD is a highly common cause of chronic liver disease. Obesity, a risk factor for liver cell malignancy and cirrhosis-related mortality, is another important risk factor in the pathophysiology of NAFLD [9]. NAFLD and alcoholic liver disease share several histological similarities. Cirrhosis and its consequences may be present in a considerable proportion of NAFLD patients with liver fibrosis. Nevertheless, it is unclear how NAFLD

develops histologically [10]. According to the same study, NAFLD patients show signs of severe lobular inflammation, hepatocellular necrosis, hepatocellular ballooning, portal tract inflammation, pericellular and portal fibrosis, enlargement of microvilli, lysosome emergence, and cytoplasmic swelling when examined under an electron microscope [11]. Although the histology damage procession is unknown, the structure of the liver was examined under a microscope using four different chemical assessments. Rats' livers were also removed at different stages of the experiment. A high-fat diet may cause obesity, according to several previous studies on rats and mice [12]. In the current study, a high-fat diet is proposed for adult male rats, and the livers of the obese rats are analyzed as part of an obesity-induced dietary model. We tried to understand the effects of obesity on male rats in this manner.

Study Objectives:

Using weight measurements and other biochemical markers, the study aims to evaluate the physiological effects of obesity brought on by a high-fat diet in male rats.

METHODS

The present experimental was carried out in the animal house of the college of veterinary medicine, Tikrit University, between the dates September 1, 2025, to December 1, 2025, with the laboratory rearing conditions being in standard conditions. This study was conducted using male laboratory rats. The males were also selected because they are larger in size and more stable in weight than females therefore, they would not interfere with the hormones and alter the outcome of the study. he rats used weighed between 250 and 300 grams at the beginning of the experiment. Feed Preparation: The fattening of the rats in the animal house was prepared using a special feed. It was made of some simple plant-based products, such as: Soy milk, Corn, Wheat, in amounts that were dictated by the accepted source. The 1.5 kg of animal fat (sheep tail fat) and another protein source were introduced to the diet. The ingredients were mixed well to form a homogenous feed mixture that was used in causing obesity in rats.

Experimental Design and Division of Groups

The present work had been categorized into two major groups in regard to the dosing phase and diet as follows: Weight Measurement and Follow-up; the animals were first weighed to ascertain the weight of each rat at the start of the diet. They were also observed during the rearing process which lasted 60 days, and free access to water and feed was controlled.

Blood and serum: Specimens of blood and serum were collected and prepared according to the following routines:

The Rats were Twice Drawn of Blood:

- First time: prior to the commencement of the experiment.
- Second occasion: at 60 days of dosage.

The blood samples were then put into special tube and the serum was separated using centrifuge so as to get the serum required to make laboratory tests. Using ELISA by the standard laboratory procedures, liver enzymes (ALT, AST, ALP), and total cholesterol and triglycerides in the blood serum were determined. Manufacturer: BioAssay Technology Laboratory; Country of Origin: China.

RESULTS

Table 1 provides an overview of the General characteristics of the research male rats groups for participants falling Body wt, g range means ± SD of Control groups 543.3 ± 32.15 , Before Treatment 541.5 ± 29.12 as well as After Treatment 591.5 ± 43.54 , while Abdominal circumference, cm recorded that of Control groups 26.32 ± 0.87, Before Treatment 25.46 ± 0.85 as well as After Treatment 27.13 ± 0.91, according to Blood pressure, mmHg recorded that of Control groups 128.2 ± 4.12, Before Treatment 147.9 ± 20.76 as well as After Treatment 172.8 ± 19.87 which were significantly higher $p \leq 0.01$ as shown in Table 1.

Table 1: General characteristics of the research male rats groups

General characteristics	means ± SD			
	Control	Before Treatment	After Treatment	P-value
Body wt, g	543.3 ± 32.15	541.5 ± 29.12	591.5 ± 43.54	0.01
Abdominal circumference, cm	26.32 ± 0.87	25.46 ± 0.85	27.13 ± 0.91	0.01
Blood pressure, mmHg	128.2 ± 4.12	147.9 ± 20.76	172.8 ± 19.87	0.01

The means and SD are presented in table 2. The mean levels of GOT (AST), in male rats groups Before Treatment recorded that 18.76 ±3.32, After Treatment36.12±6.32 compared to Control groups recorded that 22.35 ±5.18, results showed a highly significant difference ($P < 0.01$).

The mean levels of GPT (ALT), in male rats groups Before Treatment recorded that 14.99±3.21, After Treatment 31.43±3.92 compared to Control groups recorded that 18.65±2.42, results showed a significant difference ($P < 0.05$).

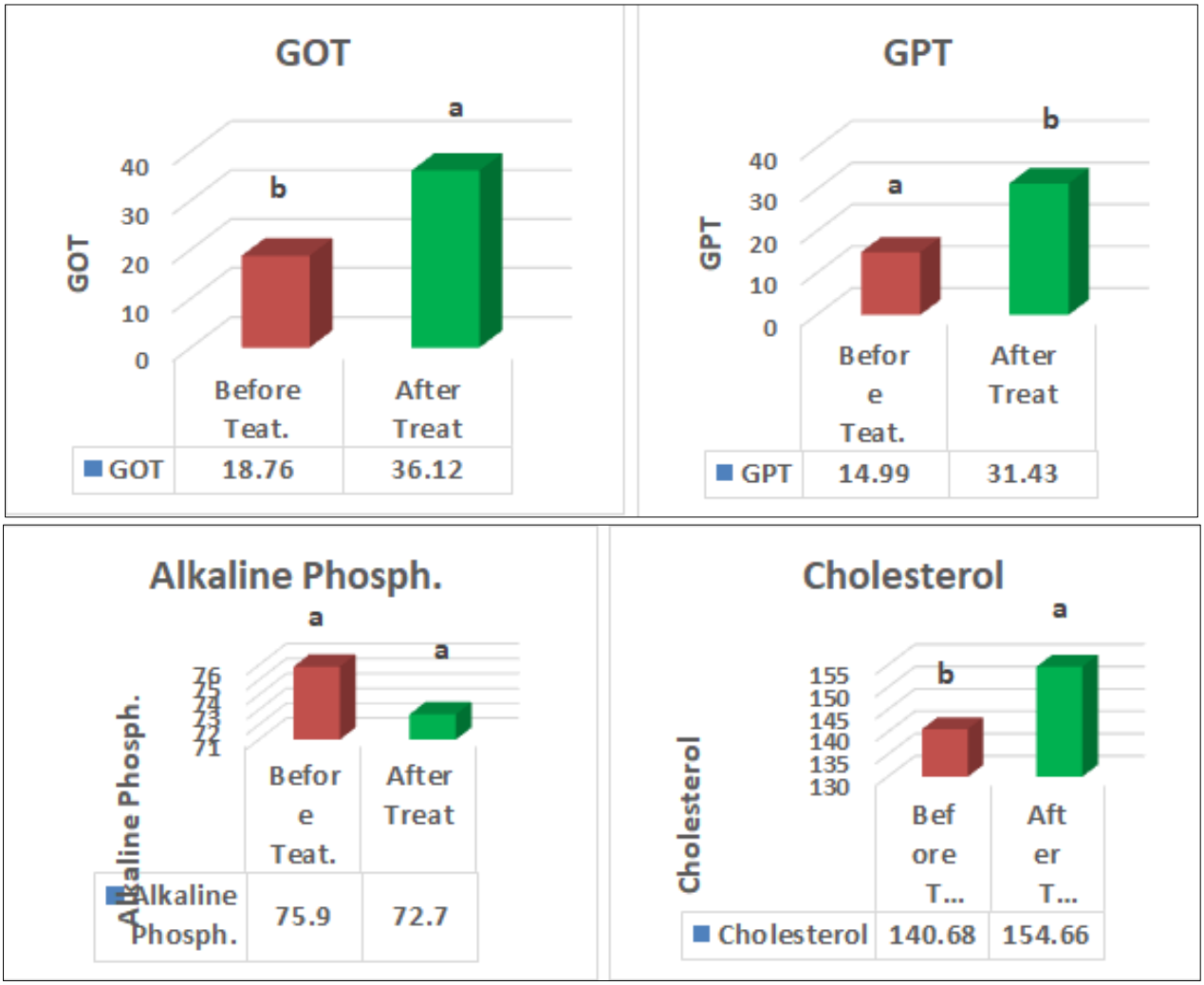
The mean levels of ALP, in male rats groups before treatment recorded that 75.9±12.5, After Treatment 72.7±14.0 compared to Control groups recorded that 67.9±11.2, results showed a non-significant difference ($P < 0.75$).

As well as the mean levels of Cho, in male rats groups before Treatment recorded that 140.68±8.71, after Treatment 154.66±7.00 compared to Control groups recorded that 132.12±6.65, results showed a highly significant difference ($P < 0.01$).

Additional as the mean levels of TG, in male rats groups before Treatment recorded that 115.80±6.57, after Treatment 127.08±5.59 compared to Control groups recorded that 113.60±5.34, results showed a highly significant difference ($P < 0.01$) was presented in Figure (1) and Table (2).

Table 2: Biochemical parameters of the research male rats groups

Biochemical parameters	means ± SD			
	Control	Before Treatment	After Treatment	P-value
GOT (AST)	22.35 ±5.18	18.76 ±3.32	36.12±6.32	0.01
GPT (ALT)	18.65±2.42	14.99±3.21	31.43 ±3.92	0.05
ALP	67.9±11.2	75.9±12.5	72.7±14.0	0.75
Cho, mg/dL	132.12±6.65	140.68±8.71	154.66±7.00	0.01
TG mg/dL	113.60±5.34	115.80±6.57	127.08±5.59	0.01



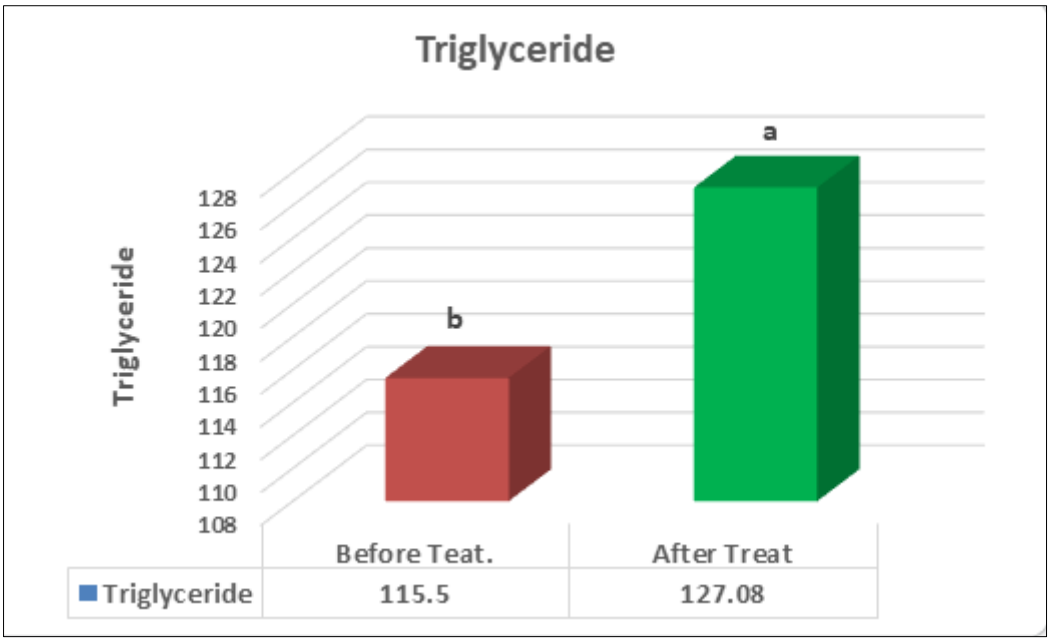


Figure 1: Boxplot of the distribution of serum level of liver function test and lipid profile in male rats groups before and after Treatment

DISCUSSION

The high-fat diet employed in the current study worked well in facilitating obesity because it was shown that the body weight has increased significantly. Animals that fed on high fat diet had a significant increase in fat deposits, adiposity index Body weight, abdominal circumference and Blood pressure [13]. Dietary obesity results in fat accumulation in the liver of male rats which causes liver fibrosis and non-toxic hepatitis (steatosis), and inflammation and increased oxidative stress. These alterations have adverse effects on the liver functioning and the evolution of non-alcoholic fatty liver syndrome (MASLD) [14]. Research has also indicated that fat in the liver can be guarded by the adipose tissue though the effect is sex-specific. The past results might be due to high calorie content. Previous results however show absolute fatty effect. Other notable histological processes we were able to identify were microvesicular steatosis, mononuclear cell infiltration and fibrosis as necrotic foci [15].

Liver enzyme plays an important role in the liver to mediate carbohydrate and lipid homeostasis by promoting synthesis of glycogen, lipogenesis, lipoprotein synthesis, inhibition of gluconeogenesis/glycogenolysis and cholesterol and triglyceride secretion in the fed state [16].

The results obtained indicated that the liver function test of after high fat fed in was highly differentiated when compared to control group this could be explained by the fact that General characteristics and biochemical parameters could change due to high fat diet which may require not short but long term exposure to the effect of high fat diet on blood picture, Abdominal circumference and Blood pressure [17].

The elevated values of ALT and AST on the serum of the high-fat fed rat than the control groups have been seen in the current investigation showing the detrimental impact of high-fat diet on the liver. One of the most sensitive index of the hepatic damage is usually considered to be the increase in the concentration of the serum marker enzymes [18]. ALT and AST levels in the serum of high fat diet rat are found to be high, which means that the increase could be as a result of hepatocellular damage secondary to the toxicity of high fat diet. The results of the current research coincide with the finding of the previous study that HFD led to the serious rise in serum concentration of AST, ALT and ALP (Baz, Lina, *et al*., [19]. Likewise, Sabir, Usman, *et al*., [20], found that the hepatic damage caused in the animal fed on high fat diet led to the increased concentration of AST and ALT in blood. Moreover, serum ALP levels were not significant difference in the rats in HFD fed in the present study. The localized destruction of the endoplasmic reticulum or hazardous effect of energy that librate in the metabolism of HFD may be the cause of the depletion in the ALP levels [21].

The results of the obtained data indicated that higher serum total cholesterol, triglyceride, in high fat diet group. These findings agreed with those of Azemi, Nurul Adila, *et al*., [22], who mentioned that high fat diets raise the concentration of cholesterol. The level of cholesterol increase could be attributed to the excessive loads of cholesterol on the liver leading to down regulation of LDL receptors that take cholesterol leading to the re-absorption of cholesterol in the blood. The mice that are fed on HFD accumulate high triglycerides, oxidized low density lipoproteins, free fatty acids

and VLDL-cholesterol [23]. The mice which are fed on HFD are subjected to the build up of triglycerides, oxidized low density lipoproteins, free fatty acids and VLDL-cholesterol [24]. Male infertility is among the health outcomes that are triggered by obesity [25].

CONCLUSION

In comparison to the control group, male mice given a high-fat diet (HFD) for eight weeks had a tendency toward higher lipid profile and liver function test values. Perhaps because the obesity was not as severe as that of rats subjected to a relatively short-term high-fat diet, the adiposity gain observed in the male mice in this study was sufficient to create a significant decrease in the lipid profile and liver function test values.

Conflict of Interest: None declared.

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