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Estimating liver function changes in obese adults

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Abstract

Background: Chronic low-grade systemic inflammation, a hallmark of obesity, may independently contribute to hepatic impairment via metabolic and inflammatory pathways. A thorough evaluation of hepatic function is crucial for the early detection and tracking of obesity-related liver problems because obese people are more vulnerable to liver impairment. **Aim of the study:** By contrasting obese adults with normal-weight controls, this study examined the relationship between obesity and serum levels of important liver biomarkers, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). **Materials and Methods:** Eighty participants with obesity (BMI: 35–39.5 kg/m²) and twenty age-matched, healthy, normal-weight controls (BMI: 18–24.5 kg/m²) made up a total of one hundred persons between the ages of twenty and forty. As biochemical markers of hepatic function, serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were measured. This allowed for the evaluation of changes in liver enzyme profiles associated with obesity. **Results:** The majority of obese groups had significantly higher serum levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) than normal-weight controls ($P < 0.05$). Additionally, across all age groups, obese subjects had significantly higher levels of alkaline phosphatase (ALP) ($P < 0.05$). Significant positive correlations between obesity and serum hepatic enzyme concentrations were also found by correlation analysis, suggesting that changes in biochemical indicators of hepatic function are linked to increasing adiposity. **Conclusion:** Elevated blood levels of ALT, AST, and ALP were substantially correlated with obesity, suggesting changes in hepatic function. These biomarkers might be helpful markers for the early identification and evaluation of liver damage associated with obesity.

Key Words: Obesity, Liver function, (ALT), (AST), and (ALP).

Introduction

One of the most intricate and metabolically active organs in the human body, the liver is vital for detoxification, the creation of bile, the metabolism of proteins, fats, and carbohydrates, and the preservation of systemic homeostasis. It contributes to gluconeogenesis and controls glucose homeostasis through the production, storage, and breakdown of glycogen. Amino acid deamination in protein metabolism is mediated by the liver, which then uses the

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urea cycle to transform the resultant ammonia into urea. Additionally, the liver is essential for lipid metabolism, which includes the production and control of phospholipids, lipoproteins, triglycerides, and cholesterol. Additionally, it contributes to the metabolism of bilirubin and the excretion of metabolic waste products through bile, as well as the synthesis of albumin and the majority of plasma proteins, including a number of coagulation factors (1,2). The liver is also responsible for supporting immunity, digestion, and vitamin storage (3).

Excessive triacylglycerol buildup in hepatocytes is closely linked to obesity. This can result in hepatic steatosis and, in certain cases, the development of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH). There is growing evidence that obesity is a significant risk factor for the onset and advancement of liver disease, with a higher body mass index (BMI) gradually increasing the risk of nonalcoholic fatty liver disease (NAFLD) (4). Obesity is often associated with changes in blood liver enzymes, especially gamma-glutamyl transferase (GGT), aspartate aminotransferase (AST), and alanine aminotransferase (ALT), which may indicate underlying hepatic problems. However, liver enzyme levels do not always accurately reflect the severity of hepatic steatosis or steatohepatitis (5).

A cytosolic enzyme mostly found in the liver, alanine aminotransferase (ALT) catalyzes the reversible transamination of alanine and α-ketoglutarate to produce pyruvate and glutamate. A common biochemical indicator of hepatocellular damage is the human ALT enzyme, which has 496 amino acids. Sex, body mass index (BMI), and lipid metabolism are among the demographic and metabolic characteristics that affect serum ALT concentrations. Men typically have greater ALT levels than women, and these levels tend to rise with rising BMI and metabolic disorders, such as elevated triglyceride concentrations (6).

Aspartate aminotransferase (AST) exists in the cytosolic and mitochondrial isoenzymes and is found in the liver, kidneys, brain, pancreas, lung, leukocytes, cardiac and skeletal muscles, and also in the red blood cells, while the maximum level of ALT was found in the liver, and the level of this enzyme may be a more specific indicator of liver injury (7).

Alkaline phosphatase (ASP) originates mainly from two sources: liver and bone; it is also found in many other tissues such as intestine, leukocytes, kidney, and placenta. The rise of this enzyme may be physiological or pathological; the production can be increased in tissues undergoing metabolic stimulation (8). Many studies indicate evidence of hepatocellular injury with elevated APT, which signals damage to the lining of the biliary tract (9).

The liver is influenced by obesity, which may be associated with hepatomegaly, rising liver biochemistry values, and changes in the liver histology like macrovesicular steatosis, steatohepatitis, fibrosis, and cirrhosis (10). Obesity was correlated with a rise in liver enzyme levels; several researchers have reported many changes in liver function and morphology in morbidly obese persons. Recent Japanese studies have found adverse effects on liver function in individuals who were only moderately obese (30 -50 % overweight). The influence of obesity was important in the case of ALT; the prevalence of elevated ALT values in obese subjects may be more than eight times that in those with normal weight (11).

Data from the Third National Health and Nutrition Examination Survey (NHANES III) demonstrated that elevated serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were associated with higher body mass index (BMI) and metabolic abnormalities characteristic of metabolic syndrome. These findings further support the important role of obesity and its associated metabolic disturbances in the pathogenesis of obesity-related liver disease (12). Recent evidence indicates that the distribution of adipose tissue, particularly visceral and abdominal adiposity, may be a more important determinant of metabolic and hepatic risk than total body fat alone, as estimated by body mass index (BMI) (13). Central adiposity is strongly associated with hepatic steatosis, steatohepatitis, and progression of liver disease, independent of BMI. Furthermore, increasing severity and duration of obesity are associated with a greater risk of hepatic dysfunction and progression of metabolic dysfunction-associated steatotic liver disease (MASLD) (14).

The liver functions are involved in the pathogenesis of obesity and obesity related diseases; we tried to assess liver function status by estimating serum levels of ALT, AST, and ALP in obese adults compared to the control group.

Materials and Methods

Study Design and Subjects

At Al-Diwaniyah Teaching Hospital, this study was carried out between July 2022 and September 2024. A normal-weight control group (n = 20; BMI: 18–24.5 kg/m²) and an obese group (n = 80; BMI: 35–39.5 kg/m²) were formed from a total of 100 male participants, aged 20–40, based on their body mass index (BMI). Four age groups—20–25, 26–30, 31–35, and 36–40 years—were used to further stratify the individuals. The study excluded participants with a history of drug use, chronic illnesses, long-term medication use, or other pertinent conditions. To assess changes in hepatic enzyme profiles related to obesity, serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were assessed in the obese and control groups.

Ethical Considerations

The relevant hospital ethics committee examined and approved the study protocol. The study was carried out in compliance with relevant ethical guidelines, and all participants were made aware of its goals, methods, and importance. Before the sample was collected, individuals provided written informed consent.

Blood Collection and Serum Preparation

After an overnight fast, venous blood samples were taken between 8:00 and 10:00 AM. Five milliliters of blood were drawn and placed in gel-filled serum-separation tubes. After letting the samples coagulate, serum was extracted by centrifuging them. Prior to biochemical analysis, the serum samples were aliquoted and kept in triplicate at -20°C. Only when necessary for analysis were samples thawed.

Assessment of Liver Enzymes

A Reflotron Plus automated biochemical analyzer (Roche Diagnostics, Germany) and the accompanying commercially available reagent cassettes were used to measure serum ALT, AST, and ALP activity in accordance with the manufacturer's instructions. Enzyme activity was measured in units per liter (U/L) for each test after 200 µL of serum was added to the relevant reagent cassette.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS), version 21.0 (IBM SPSS Statistics, Chicago, IL, USA), was used to conduct statistical analyzes. Mean ± standard deviation (SD) was used to express the data. The Mann-Whitney U test was used to evaluate group differences. Spearman's rank correlation coefficient was used to assess correlations between variables. Statistical significance was defined as a two-tailed P value of less than 0.05.

Results

One-way ANOVA Statistical analysis demonstrated no significant increases (P> 0.05) in ALT levels between control and obesity groups in age (20-25 years), with a (mean ± SD) of (18.66 ± 7.40) in control groups vs. (21.42 ± 7.87). While a significant increase in ALT levels (p< 0.05) in obesity groups in the age (26- 30 years), (31-35 years) and (36-40 years) in comparison with control group, the (mean ± SD) in obesity groups (24.31 ± 5.98), (27.28 ± 7.86), and (29.36 ± 6.22) (Table1).

Statistical analysis also demonstrated a significant increase (p< 0.05) in the AST serum level in the obesity group compared with the control group in age groups (26-30 years), (31-35 years), and (36-40 years), with (mean±SD) (28.28± 7.2), (29.98±8.40), and (29.84 ±7.50), respectively. While no significant increase in the AST serum level in the obesity group (20-25 year) the (mean± SD) (21.43 ± 4.87) compared with the control (Table 2).

Results showed significant increased the serum level **ALP** (p<0.05) in all obesity age groups, with (mean ± SD) (217.34 ± 68.34), (237.36 ± 98.11), (239.33 ± 111.22) and (253.13 ± 126.27) respectively comparison with control groups (Table3)

Table1: Comparison of mean ± SD liver enzyme (ALT) serum levels in obesity and control in different age groups.

Parameter	Mean ± SD (age groups)				p-value
ALT U/L (Obesity)	20 - 25 Year N=20	26 - 30 Year N=20	31 - 35 Year N=20	36 - 40 Year N=20	
	21.43 ± 4.87	24.31 ± 5.98*	27.28 ±7.86 *	29.36 ± 6.22 *	0.006
ALT U/L (Control)	20-25 Year N=5	20-30 year N=5	31-35 Year N=5	36-40 Year N=5	
	18.66 ± 7.40	20.43 ± 7.87	22.92 ± 5.87	22.20 ± 5.36	P<0.05

*Significant (p>0.05)

Table2: Comparison of mean ±SD liver enzyme (AST) serum levels in obesity and control in different age groups.

Subject	Parameter	Mean ± SD (age groups)				p-value
Obesity BMI 35-39.9 kg/m^2 N=80	AST U/L	20-25 Year N=20	26-30 Year N=20	31-35 Year N=20	36-40 Year N=20	
		21.41 ± 5 .72	28.28 ± .20 *	29.98 ± 6.48 *	29.84 ± 7.50 *	0.004
Control BMI 18-24.9 Kg/ m^2 N=20	AST U/L	20-25 Year N=5	20 -30 year N=5	31-35 Year N=5	36-40 Year N=5	
		21.26 ± 5.97	23.80 ± 11.15	25.92 ± 5.87	24.27 ± 4.46	P<0.05

*Significant(p>0.05)

Table3: Comparison of mean ±SD liver enzyme (AST) serum levels in obesity and control in different age groups.

Subject	Parameter	Mean ± SD(age groups)				p-value
Obesity BMI 35-39.9 Kg/m^2 N=80	ALP U/L	20 - 25 Year N=20	26 - 30 Year N=20	31 - 35 Year N=20	36 - 40 Year N=20	
		217.34 ± 64.34*	237.36 ± 98.11*	239.33 ± 91.86*	253.1 ± 111.22 *	0.006
Control BMI 18-24.9 Kg/ m^2 N=20	ALP U/L	20 - 25 Year N=5	20 - 30 Year N=5	31 - 35 Year N=5	36 - 40 Year N=5	
		71.74 ± 12.52	79.12 ± 17.33	80.50 ± 18.57	89.86 ± 12.18	P<0.05

*Significant(p>0.05)

Studying the correlation between elevated liver enzyme levels in obesity and age reveals a positive, significant association between increased age and elevated liver enzyme levels in obesity (p=0.757, r=0.040).

The present study demonstrated significantly higher serum levels of the hepatic enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) in participants with obesity compared with normal-weight controls across most age groups ($P < 0.05$; Tables 1–3). Furthermore, significant positive correlations were observed between obesity and serum concentrations of ALT, AST, and ALP, with correlation coefficients of $r = 0.651$ ($P = 0.008$), $r = 0.816$ ($P = 0.004$), and $r = 0.726$ ($P = 0.006$), respectively. These findings indicate a significant association between obesity and alterations in serum hepatic enzyme profiles.

Discussion

In comparison to normal-weight controls, our results showed that serum hepatic enzyme levels were higher in obese people in the majority of age groups. Serum ALT and AST values, in particular, tended to rise as obesity severity increased, indicating a possible link between adiposity and hepatocellular damage. According to earlier research, a significant percentage of obese people have liver enzyme values above recognized reference ranges, and higher liver enzyme levels are increasingly common as BMI rises. Several hepatic and metabolic problems have been linked to elevated serum levels of ALT, AST, ALP, and GGT, which are commonly seen in obese people (15). All of these results lend credence to the link between elevated body fat and changes in biochemical indicators of liver function (16, 17).

Elevated AST levels in healthy individuals were linked to muscular damage, which was linked to aberrant blood creatine kinase levels and essential physical activity (18). Additionally, Lin et al. (2010) demonstrated that AST and ALT levels were significantly correlated with uric acid, cholesterol, and gender; gender was thought to be a good significant predictor of ALT levels; rising ALT levels were correlated with male sex, BMI > 25 kg/m2, and triglyceridemia > 150 mg/dl in subjects without known liver disease etiologies (19).

Chronic low-grade inflammation brought on by obesity may negatively impact vital organ function, especially the liver. A growing body of research indicates that hepatic impairment is linked to obesity through a variety of metabolic and inflammatory pathways. Elevated levels of pro-inflammatory mediators, such as C-reactive protein (CRP) and interleukin-6 (IL-6), may be linked to altered regulation of hepcidin, a crucial hormone involved in systemic iron homeostasis, and hepatic metabolic disorders in obese persons (20,21). In vulnerable people, dysregulation of hepcidin signaling may lead to anomalies in iron metabolism and the emergence of iron deficiency or anemia. Additionally, obesity-related chronic inflammation and metabolic dysfunction may accelerate the onset and progression of metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as nonalcoholic fatty liver disease (NAFLD), and raise the risk of advanced hepatic disease, such as hepatocellular carcinoma (22,23).

Liver functions are typically evaluated using the serum levels of liver enzymes, such as ALT, AST, and ALP, in obesity (24). Serum levels of ALT and AST, which are primarily present in the liver, are thought to be

particular indicators of hepatic dysfunction (25). The percentage of people with increased ALT who are overweight or obese (26).

This study showed a significant correlation between liver enzymes and BMI without any symptoms or prior history of liver disease. Our analysis revealed significant differences in ALT and AST in the 20–25 age group compared to older age groups in obesity, which may be related to physical activity or muscle mass in the 20–25 age group.

Our results are supported by research by Strauss et al. (2000) that suggested elevated liver enzymes are associated with adolescent overweight and obesity, which is caused by hyperlipidemia, hyperinsulinism, and lowered antioxidant levels (27).

According to the current results, liver enzymes can be regarded as a useful marker parameter for estimating liver function because control groups did not appear to have higher liver enzymes at different age groups.

Conclusion

In most age categories, the serum levels of hepatic enzymes were considerably higher in obese adults than in normal-weight controls. These results reveal a higher risk of obesity-related liver impairment by showing a strong correlation between obesity and changes in biochemical indicators of hepatic function.

Conflict of interest: NIL

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