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Assessment of hepatic enzyme activity in relation to body mass indices in adults

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Abstract

Background: Altered body mass index can disturb the metabolism of body which can be a risk factor for metabolic syndrome, may lead to chronic diseases such as diabetes, cardiovascular disease and non-alcoholic fatty liver disease (NAFLD). Obesity may be associated with progression of hepatic dysfunction through several mechanisms. High levels of cytokines including interleukin-6 (IL-6) and C-reactive protein (CRP) may disrupt the production of hepcidin leading to some types of liver diseases such as non-alcoholic fatty liver disease (NAFLD) and liver cancer.

Objectives: The purpose of this study was to investigate the correlation between varying body mass indices BMI and hepatic enzyme activity in both adult males and females.

Materials and Methods: A total of 220 study participants of both genders males and females were analysed for the activities of enzymes AST, ALT, ALP, GGT and LDH and were correlated with their respective body mass indices, which were categorized as per WHO guidelines.

Results: Significantly raised levels of ALT, AST, and ALP in both males and females had shown a positive correlation with BMI and were commonly observed in overweight and individuals, indicating potential liver dysfunction and metabolic disturbances. GGT and LDH activities were found positively correlated in obese males having BMI greater than 30 kg/m^2 as compared to females in age group of 40-60 years.

Conclusion: Excessive weight is independently associated with increased levels of liver enzymes in both the genders. Body Mass Index appeared as good predictor of elevated hepatic enzymes. It can be helpful in clinical settings to identify patients at risk of liver dysfunction, which is closely related to metabolic syndrome.

Keywords: Body mass index; Metabolic syndrome; Hepatic enzymes; Obesity; Non-alcoholic fatty liver disease; Liver cancer

1. Introduction

With rapid economic growth, changes in lifestyle and prevalence of obesity, liver ailments are increasing all over the world. It is estimated that about ¼ of adults have suffered from non-alcoholic fatty liver disease (NAFLD). It is expected to being a huge public health problem and economic burden to society [1]. The prevalence of general and abdominal obesity has been increased in past few decades due to unhealthy dietary habits, less physical activity and increased sedentary lifestyles. Obese, diabetic, hypertensive and married individuals are at higher risk of having non- alcoholic fatty liver disease (NAFLD) than others. Increased levels of WC (waist circumference) and BMI have been found to be independently related to NAFLD and people with abdominal obesity have an increased risk of NAFLD than subjects with

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general obesity [2]. Some epidemiological studies and meta-analysis demonstrated that the abdominal obesity can be an important predictor of metabolic disorder along with general obesity [3].

The serum level of five enzymes including aspartate aminotransferase (AST), alkaline phosphatase (ALT), and alkaline phosphatase (ALP) and γ-glutamyl transferase (GGT) are generally used in the assessing liver function. Lactate dehydrogenase (LDH) is mostly found in tissues, blood and other body fluids. ALT and AST are found mostly in the liver, and serum levels of AST and ALT are considered as specific markers for hepatic dysfunction. GGT is present in cell membrane of many tissues with greatest activity in biliary epithelial cells, pancreatic acinar cells and renal tubular epithelial cells. ALP is an enzyme that is primarily present in liver, bones, intestine and kidneys [4].

Obesity is a disorder involving excessive body fat when a person's Body Mass Index (BMI) is 25 or greater [5]. When a person gains too much weight due any metabolic disorder or any other physical condition, they don't just accumulate on the outside of the body but also inside the body that involves internal organs such as Liver. This condition is called fatty liver disease. Liver cells, or hepatocytes, fill with large fat droplets that can become stressed, damaged or scarred sometime fetal [6].

The association between Body Mass Index (BMI) and serum biomarkers of liver function, independent of dietary intake and physical activity, is not clear yet. Due to the role of liver enzymes in the body's metabolism, if the link between BMI and serum enzyme is proven, this finding may be a clue to possible mechanism by which BMI plays a role in risk of diseases such as fatty liver, diabetes and cancer. The World Health Organization (WHO) classifies weight in relation to the risk of other health conditions and death due to obesity, putting adults into different categories from underweight to very severe obesity, with a BMI of <18.5 to ≥30.0, respectively [7]**.** This has been of clinical use to counsel adults regarding their desired weight goals [8]**.**

The measurement of enzymes is not always specific for damage to a particular organ but it is most helpful when used with other tests, clinical symptoms and patient history [9]. The objective of the study was to assess the body mass indices of study population and to categorize into distinct BMI groups, such as normal weight, overweight, and obese, according to established BMI criteria and to assess the baseline levels of hepatic enzymes in participants with varying body–mass indices (BMIs). The study aimed to investigate the correlation between varying body mass indices BMI and hepatic enzyme activity, in both genders of study population.

2. Material and methods

This study was cross-sectional conducted on 220 study participants with 50% male population and 50% female population. The clinical samples of patients and controls were collected independently from Baba Deep Singh Nursing Home, Attari, Amritsar and Khalsa Diagnostic Lab, Amritsar, and biochemical analysis was performed in Khalsa Diagnostic Lab, Amritsar. Informed consents were taken from the patients and study protocol was approved by the Institutional Ethical Committee. The study participants were categorized into three groups as per WHO classification of BMI viz.

- **Group-I** healthy subjects with BMI in the range of 18.5 to 24.9 kg/m^2 **,**
- **Group-II** overweight subjects with BMI in the range of 25 to 29.9 kg/m^2 and
- **Group-III** obese subjects with BMI above 30.0 kg/m^2.

The healthy male and female subjects of age groups 20-40 yrs and 40-60 yrs were included in the study. The individuals who were clinically diagnosed with liver diseases, patients suffering with renal failure, cardiac diseases, Type-II diabetes, hypertension and respiratory diseases, alcoholic, people with routine medication, pregnant and lactating women were excluded from the study. Data collection procedure included fulfilling a brief questionnaire on anthropometric and demographic measurements, smoking status and physical activity. In brief anthropometric parameters such as weight (in cm) were measured with participants wearing no shoes and light clothes using a modern electronic digital weighing scale, height (in meter) while the participants stood in erect posture and BMI were observed. Data was recorded in pre-structured Performa. After an overnight fast, three milliliters (ml) of blood were collected using a vein puncture method. All the collected blood specimens were left undisturbed at room temperature for 30 min by keeping the gel clot activator vial in upright position in tube rack. All the specimens were observed clotted after 30 min. Then, the tubes were gently tapped in order to detach the clot from bottom of the tube. Then the clotted specimens were centrifuged at 2500 rpm for 10-15 minutes using Remi R 8C BL Centrifuge. After centrifugation, tubes were carefully observed for hemolysis before serum was separated. Finally, the supernatant was separated for the further analysis. Serum levels of AST, ALT, ALP, GGT and LDH were determined using a kinetic method using an ERBA-Chem 5

plus semi-auto biochemistry analyzer. Before the serum specimen analysis, the analyzer was calibrated for the serum liver enzyme using the human multi calibrator from ERBA, and Calibration was done according to the manufacturer's instructions. Following the calibration, two quality control (QC) samples (Human Assayed Control from ERBA), one with a known normal value (normal level control) and other with a known abnormal value (pathological level control) for each serum liver enzymes, were run in order to verify the performance of measurement procedure. The kits used in the study for investigating enzyme activities were from ERBA Diagnostics and Coral Clinical System. Descriptive statistical analysis was done using ORIGIN Lab (Version-9.1). Data was represented as mean ±S.D. One–way ANOVA was used to compare the statistical significance of difference in mean levels of enzymes activities between groups. Correlation analysis was done to evaluate the relationship between BMI and enzyme activity.

3. Results

Out of 220 study participants, 110 were males and 110 were females. In the study population, 45% subjects had BMI ≥25.0-29.9 Kg/m^2 i.e. overweight whereas 27% population fall in obese category. It was observed that 72% subjects had BMI higher than the normal range.

Table 1 Mean age of male and female subjects under study

S. No.	Groups	Age Groups (yrs)	Mean age (yrs)	Mean age (yrs)
			Males	Females
1	CONTROL (18.5-24.9 Kg/m^2)	20-40	24.47±3.42	25.93±4.85
		40-60	50.67±4.69	46.13±4.79
2	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	26.76±5.04	27.72±4.65
		40-60	50.88±5.32	47.08±5.11
3	OBESE (≥30.0 Kg/m^2)	20-40	28.20±6.35	31.93±4.06
		40-60	49.60±7.55	52.60±4.31

*Data represented as mean ±S.D.

Age matched individuals were enrolled in the study. The mean age of both male and female participants in all three groups was comparable to each other. The mean age of control males was 24.47±3.42 yrs, comparable to overweight males (26.76±5.04 yrs) and obese males (28.20±6.35 yrs) in the age category of 20-40 yrs. Similarly, mean age of control males (50.67±4.69 yrs) was comparable to overweight males (50.88±5.32 yrs) and obese males (49.60±7.55 yrs).The mean age of control females was 25.93±4.85 yrs was comparable to overweight females (27.72±4.65 yrs) and obese females (31.93±4.06 yrs) in the age category of 20-40 yrs. Similarly, in the age group of 40-60 yrs age group, mean age of control females (46.13±4.79 yrs) was comparable to overweight (47.08±5.11 yrs) and obese females (52.60±4.31 yrs) (Table. 2).

Table 2 Mean BMI of male and female subjects under study

S. No:	Groups	Age groups (years)	BMI (Kg/m^2) Males	BMI (Kg/m^2) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	22.70±1.74	21.43±2.06
		40-60	23.18±1.97	24.00±0.76
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	27.16±1.43	27.08±1.45
		40-60	27.70±1.45	27.75±1.38
3.	OBESE (≥30.0 Kg/m^2)	20-40	33.14±2.08	31.71±1.12
		40-60	31.73±1.59	33.13±1.62

*Data represented as mean ±S.D.

The mean level of body mass index was 22.70±1.74 kg/m^2 in the males of 20-40 yrs age group whereas, body mass index was 23.18±1.97 kg/m^2 in 40-60 yrs age group. The mean level of BMI i.e. 27.16±1.43 Kg/m^2 for overweight males of 20-40 yrs age group were significantly similar to males of 40-60 yrs age group (27.70±1.45 kg/m^2). Obese males had BMI of 33.14±6.08 Kg/m^2 in age group of 20-40 yrs and 31.73±1.59 Kg/m^2 in 40-60 yrs age group.

In females, the mean BMI were 21.43±2.06 kg/m^2 and 24.00±0.76 kg/m^2 for the healthy control of 20-40 and 40-60 yrs age groups respectively. In the overweight females, it was seen that the BMI was 27.08±1.45 kg/m^2 for 20-40 yrs age group and 27.75±1.38 kg/m^2 in 40-60 yrs age group. In the obese females, the BMI seen was 31.71±1.12 kg/m^2 for the 20-40 yrs age group and 33.13±1.62 kg/m^2 in the 40-60 yrs age group. In the study population, the BMI of healthy control was between 21-24 kg/m^2. In overweight subjects BMI was 27 kg/m^2 and in obese subjects the BMI was between 31-34 kg/m^2.

Table 3 Aspartate aminotransferase (AST) activity of males and females of different age groups in relation to body mass

S. No:	Groups	Age groups (years)	AST (U/L) Males	AST (U/L) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	28.42±4.50	26.01±9.39
		40-60	28.10±4.36	27.08±3.07
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	56.12±10.42	41.61±12.06
		40-60	61.34±10.70	45.62±14.59
3.	OBESE (≥30.0 Kg/m^2)	20-40	67.98±19.11	76.94±20.18
		40-60	69.16±19.05	85.52±16.72

*Data represented as mean ±S.D.

The mean AST activity in healthy control males was 28.42±4.50 U/L in 20-40 yrs age group and 28.10±4.36 U/L in the 40-60 yrs age group. In the overweight male's enzyme AST activity was increased to 56.12±20.42 U/L ($p<0.05$) and 61.34±20.70 U/L ($p<0.05$) of 20-40 and 40-60 yrs age group. Whereas, it further increased in the obese males. AST activity in obese males was 67.98±39.11 ($p<0.05$) of age group of 20-40 yrs age groups and 69.16±19.05 ($p<0.05$) in 40-60 yrs age group. The mean AST activity in the healthy control females were 26.01±9.39 U/L and 27.08±3.07 U/L for the 20-40- and 40-60-years age group respectively. In the overweight females it was seen that the level of aspartate aminotransferase activity was 41.61±12.06 U/L ($p<0.05$) for 20-40 years age group and 45.62±14.59 U/L ($p<0.05$) for the 40-60 yrs age group. In the obese females, level of AST was observed with values of 76.94±20.18 U/L ($p<0.05$) and 85.52±16.72 U/L ($p<0.05$) for 20-40 and 40-60 years respectively. The mean values of aspartate aminotransferase levels were compared across the categories of BMI in male and females of study population. There was a significant difference in AST levels across BMI categories in both age groups under study ($p<0.05$). AST was found to have a positive correlation with BMI in both males ($r= 0.160$) and females ($r= 0.406$). There was significant difference in AST levels of overweight and obese males and females when compared with normal weight individuals. Maximum level of AST activity was 85.52±16.72 U/L ($p<0.05$) observed in obese females of 40-60 yrs age group.

Table 4 Alanine aminotransferase (ALT) activity of males and females of different age groups in relation to body mass

S. No:	Groups	Age groups (years)	ALT (U/L) Males	ALT (U/L) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	34.74±6.15	30.45±4.48
		40-60	36.45±5.86	33.66±3.66
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	66.32±13.98	48.49±13.19
		40-60	69.51±11.51	56.99±15.90
3.	OBESE (≥30.0 Kg/m^2)	20-40	85.80±16.69	83.67±15.36
		40-60	74.52±17.62	96.21±16.42

*Data represented as mean ±S.D.

Alanine aminotransferase activity in healthy control males, the value was 34.74±6.15 U/L for 20-40 yrs age group and 36.45±5.86 U/L in the 40-60 year of same BMI. In the overweight males the levels of ALT were 66.32±23.98 U/L and 69.51±21.51 U/L for the 20-40 and 40-60 yrs age groups respectively. In obese males the alanine aminotransferase activity was 85.80±56.69 U/L in 20-40 yrs age group and 74.52±17.62 U/L in 40-60 yrs age group.

The mean ALT activity in healthy control females, the value was 30.45±14.48 U/L for 20-40 years, and 33.66±3.66 U/L in the 40-60 yrs age group. In overweight females the level of ALT were 48.49±13.19 U/L ($p<0.05$) and 56.99±15.90 U/L ($p<0.05$) for the 20-40 and 40-60 yrs age groups respectively. The level of ALT activity in obese males was 83.67±20.36 U/L ($p<0.05$) of age 20-40 yrs age group and 96.21±18.42 U/L ($p<0.05$) of 40-60 yrs age group.

Table 5 Mean levels of serum alkaline phosphatase (ALP) activity in male and female of different age groups in relation to body mass

S. No:	Groups	Age groups (years)	ALP (U/L) Males	ALP (U/L) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	79.19±18.71	61.53±19.91
		40-60	66.13±18.11	58.30±19.00
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	81.90±25.71	73.48±18.83
		40-60	87.06±26.59	79.29±22.53
3.	OBESE (≥30.0 Kg/m^2)	20-40	109.85±23.22	102.87±5.55
		40-60	113.89±26.60	107.94±8.79

*Data represented as mean ±S.D.

The mean alkaline phosphatase activity in healthy control males was 79.19±18.71 U/L of 20-40 yrs age group and 66.13±18.11 U/L in the 40-60 yrs age group. In overweight males the level of ALP activity were 81.90±35.71 U/L ($p<0.05$) and 87.06±30.59 U/L ($p<0.05$) for the 20-40 and 40-60 yrs age groups respectively. In the obese males the level of ALP activity was 109.85±28.22 U/L ($p<0.05$) of 20-40 yrs age group and 113.89±32.60 U/L ($p<0.05$) of 40-60 yrs age group. The mean alkaline phosphatase activity in healthy control females, the level of ALP was 61.53±19.91 U/L and 58.30±19.00 U/L for 20-40 and 40-60 yrs age groups respectively. In the overweight females it was seen that the level of alkaline phosphatase was 73.48±18.83 U/L ($p<0.05$) for 20-40 yrs age group and 79.29±22.53 U/L ($p<0.05$) for the 40-60 yrs age group. In the obese females, it was observed that the level of ALP was 102.87±5.55 U/L ($p<0.05$) and 107.94±8.79 U/L ($p<0.05$) for 20-40 and 40-60 yrs age groups respectively.

Table 6 Gamma-glutamyl transferase (GGT) activity in males and females of different age groups in relation to body mass

S. No:	Groups	Age groups (years)	GGT (U/L) Males	GGT (U/L) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	17.30±5.49	15.64±7.68
		40-60	16.39±3.73	18.92±8.72
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	26.38±4.44	20.23±9.67
		40-60	38.70±5.75	25.53±10.37
3.	OBESE (≥30.0 Kg/m^2)	20-40	34.02±8.74	23.32±9.36
		40-60	48.49±8.14	35.68±8.37

*Data represented as mean ±S.D.

The mean level of gamma-glutamyl transferase activity in healthy control male was 17.30±5.49 U/L in 20-40 yrs age group and 16.39±3.73 U/L in the 40-60 yrs age group. In the overweight males the level of GGT activity were 26.38±24.44 U/L ($p<0.05$) and 38.70±31.75 U/L ($p<0.05$) for the 20-40 and 40-60 yrs age groups respectively. In the obese males the level of GGT was 34.02±8.74 U/L ($p<0.05$) for 20-40 yrs age group and 48.49±38.14 U/L ($p<0.05$) for 20-40 yrs age group. The mean GGT activity of healthy control female subjects were 15.64±7.68 U/L in 20-40 yrs age group and 18.92±8.72 U/L in the 40-60 yrs age group. In the overweight females, the level of GGT were 20.23±9.67 U/L

(p<0.05) and 25.53±10.37 U/L (p<0.05) for 20-40 and 40-60 yrs age groups respectively. In the obese females, level of GGT was 23.32±9.36 U/L (p<0.05) for 20-40 yrs age group and 30.68±8.37 U/L (p<0.05) in 40-60 yrs age group. Maximum GGT activity was found 48.48±18.14U/L in obese males of 40-60 yrs age group and 30.68±8.37 U/L in females of same age group. The normal range of GGT is 0-30 U/L.

Table 7 Mean Level of LDH (lactate dehydrogenase) activity of male and female subjects under study in relation to body mass

S. No:	Groups	Age groups (years)	LDH (U/L) Males	LDH (U/L) Females
1.	CONTROL (18.5-24.9 Kg/m^2)	20-40	272.15±71.76	285.48±79.01
		40-60	319.09±60.82	337.58±45.38
2.	OVERWEIGHT (≥25.0-29.9 Kg/m^2)	20-40	258.40±75.37	340.47±70.47
		40-60	320.86±94.76	349.84±76.25
3.	OBESE (≥30.0 Kg/m^2)	20-40	481.20±106.66	472.45±50.89
		40-60	465.09±101.83	473.08±40.05

*Data represented as mean ±S.D.

The mean activity of lactate dehydrogenase in healthy control males was 272.15±71.76 U/L of 20-40 yrs age group and was 319.09±60.82 U/L in the 40-60 yrs age group of same BMI. In overweight males, the levels were 258.40±75.37 U/L (p<0.05) and 320.86±94.76 U/L (p<0.05) for 20-40 and 40-60 yrs age groups respectively. In obese males, LDH activity was 481.20±146.66 U/L (p<0.05) for 20-40 yrs age group and 465.09±101.83 U/L (p<0.05) in 40-60 yrs age group. The mean LDH activity in the healthy control females were 285.48±79.01 U/L and 337.58±45.38 U/L of 20-40 and 40-60 yrs age groups respectively. In the overweight females, it was seen that the level of LDH was 340.47±70.47 U/L (p<0.05) for 20-40 yrs age group and 349.84±76.25 U/L (p<0.05) for 40-60 yrs age group. In the obese females, it was observed that the level of LDH activity were 472.45±50.89 U/L (p<0.05) and 473.08±40.05 U/L (p<0.05) for 20-40 and 40-60 yrs age groups respectively.The reference range for LDH is 230-460 U/L. it can be inferred from the data that BMI had adverse effect in increasing in levels of LDH.

4. Discussion

As maximum activity of AST was observed in obese females of 40-60 years age group, this could be due to dietary habits like having junk in excess, irregular dietary habits like having high fat food in the obese female subjects. Obesity is associated with a spectrum of liver abnormalities, characterized by an increase in intra-hepatic triglyceride (IHTG) content. Reported abnormal serum enzyme activity in obese individuals can be due to NAFLD. During basal conditions, 1.5 L of blood is transported to the liver every minute, which deliver a large load of compounds that require metabolic processing. Excessive accumulation of IHTG is associated with alterations in glucose, fatty acid and lipoprotein metabolism leading towards metabolic syndrome [10].

Aspartate aminotransferase and alanine aminotransferase are found mostly in liver and serum levels of AST and ALT are considered as specific markers for hepatic dysfunction. Maximum ALT activity with value of 96.21±18.42 U/L (p<0.05) was seen in obese females of 40-60 yrs age groups. Significant differences in enzyme activity were encountered in healthy controls, overweight and obese individuals (p<0.05). Obesity may be associated with hepatic dysfunction, by a variety of mechanisms. High levels of cytokines including interleukin-6 and C-reactive protein may disrupt LFT. Positive correlation between BMI and specific markers of hepatic dysfunction has been observed in the study and this association is more pronounced in women than men. Elevated liver enzymes in women with overweight and obesity can be associated with hormonal disorder such as polycystic ovary syndrome and higher levels of free androgen and total testosterone which are prevalent in women with obesity. Various studies for increased levels of ALT have been frequently reported in PCOS women [11].

Alkaline phosphatase is an enzyme that is primarily present in liver, bones, intestine and kidneys. Serum ALP levels were highest among males and females with higher BMI ≥ 30 kg/m^2. Comparing with the healthy control, overweight and obese adults had significant higher serum ALP levels. Our study showed that serum ALP correlated positively with body mass index (P<0.05). ALP is a marker of visceral obesity and hepatic steatosis (fatty liver) and marker of subclinical inflammation. Obesity is associated with hyperparathyroidism and increased bone turn over [12]. Significant variation

was observed in female subjects as compared to male subjects. Obese adults especially women sight have low vitamin D levels, and higher parathyroid levels which leads to elevation of serum ALP activity. ALP is involved in controlling of intracellular lipid accumulation in human pre-adipocytes disproportion, intracellular fat depots. In return, ALP is released from adipose tissues into blood circulation in excessive amounts [13]. Gender based differences can be explained by gender disparities in gonadocorticoids, body fat distribution and lifestyle.

Significantly increased GGT activity was observed in males as compared to their female counterparts. Moreover, GGT was found positively correlated with BMI in obese males ($r = 0.145$). Although obesity may elevate serum GGT activities, the effects of overweight on the interpretation of GGT testing have remained poorly defined in various studies [14].

There was a significant association between study variables and LDH activity. Gender had no significant inputs on the levels of LDH. In the present study, significantly increased LDH activity was observed in obese individuals with BMI ≥ 30 kg/m^2, as compared to healthy controls and overweight individuals. Maximum activity was observed in obese males of 20-40 yrs age group. This is in consistent with other studies that showed the differences of levels of LDH between males and females were not statistically significant. The reason behind this could be irregular lifestyle, dietary habits, and lack in physical activity and in certain kind of medication such as aspirin. Their high BMI could also be the one of the main reasons of increased LDH levels. LDH acts through oxidation process as Pyruvate to be converted into lactate. It is mainly localized in cytoplasm of cells and become extracellular when cell dies.

5. Conclusion

An association between obesity and raised liver enzymes activity has been reported in the present study. Elevated levels of ALT, AST, and ALP in both males and females had shown a positive correlation with BMI and were commonly observed in individuals with obesity, indicating potential liver dysfunction and metabolic disturbances. GGT and LDH activities were found positively correlated in obese males having BMI greater than 30 kg/m^2 as compared to females in age group of 40-60 years. Obesity may generate oxidative stress and increase DNA (deoxyribonucleic acid) methylation in liver tissues which ultimately led to liver tissue destruction. This observational study provided evidence that high BMI is associated with modestly increased likelihood of having a diagnosis of an abnormal liver function test. The hypothesis of this study was that excessive weight is independently associated with increased levels of liver enzymes in both the genders. Body Mass Index appeared as good predictor of elevated hepatic enzymes. It can be helpful in clinical settings to identify patients at rise of NAFLD, which is closely related to metabolic syndrome. Metabolic disorders can potentially facilitate type-II DM, chronic kidney disease and cardiovascular disease and finally lead to increased mortality risk. Obesity induced inflammation may independently disturbs the function of this vital organ. Further research is needed to elucidate the underlying mechanisms and clinical implications of this relationship for the management of liver diseases.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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