International Journal of Community Medicine and Public Health
Dan S et al. Int J Community Med Public Health. 2025 Oct;12(10):4366-4372
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20252919

Original Research Article

Association between obesity and liver enzyme levels: a cross-sectional observational study among individuals visiting a diagnostic laboratory in Burdwan, West Bengal

Subhasish Dan[1]*, Prithwijit Banerjee[2], Satrajit Dan[3], Sanjoy K. Sadhukhan[1]

[1]Department of Epidemiology, All India Institute of Hygiene and Public Health, Kolkata, West Bengal, India
[2]Department of Pharmacology, Prafulla Chandra Sen Government Medical College and Hospital, Arambagh, Hooghly, West Bengal, India
[3]Department of Pharmacology, R. G. Kar Medical College and Hospital, Kolkata, West Bengal, India

Received: 29 July 2025
Accepted: 10 September 2025

***Correspondence:**
Dr Subhasish Dan,
E-mail: subhasish.dan@gmail.com

ABSTRACT

Background: Obesity is a global health crisis, acting as a risk factor for various chronic diseases. Elevated levels of liver enzymes are important biomarkers of liver disease and may signal metabolic changes resulting from obesity. The aim of this study was to explore the association between obesity and liver enzyme levels, while also assessing the role of sociodemographic, behavioural, and dietary factors in obesity prevalence.
Methods: This was a cross-sectional observational study carried out among 390 individuals aged 18 to 59 years without any diagnosis of liver disease. Information regarding sociodemographic, behavioural, and dietary habits were collected using structured questionnaires. Anthropometric measures, blood pressure, fasting blood glucose, lipid profile, and liver enzyme levels were assessed using standard methods. Chi-square tests, t-tests, Pearson correlation, and univariate logistic regression were performed to evaluate the relationships between variables.
Results: Obesity prevalence among the study participants was 67.2% and a statistically significant relationship was found between obesity and elevated liver enzyme levels ($p<0.05$). No significant associations were found between obesity and sociodemographic or behavioural variables, but excess oil and sugar consumption were linked to obesity. Furthermore, logistic regression analysis revealed that sugar consumption, high systolic blood pressure, fasting plasma glucose, and liver enzyme levels were significantly associated with obesity.
Conclusion: A significant association was identified between obesity and elevated levels of liver enzymes, indicating that they could be used as non-invasive biomarkers for detecting liver disease in early stages among overweight and obese individuals.

Keywords: Alanine aminotransferase, Aspartate aminotransferase, Alkaline phosphatase, Metabolic risk markers, Non-alcoholic fatty liver disease

INTRODUCTION

According to the World Health Organization (WHO), 650 million people worldwide are obese and over 1.9 billion people are overweight. Over the past 40 years, there has been a considerable increase in overweight and obesity among individuals in the age range of 5 to 19 years.[1] Obesity is a risk factor for several chronic non-communicable diseases such as cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), musculoskeletal disorders, and cancers. Apart from this, it also has severe pathophysiological, psychological, social, and financial consequences for individuals and their families.[2] As a result, individuals with overweight and obesity have a lower quality of life (QoL).

Liver enzymes are considered to be important markers for liver dysfunction, and elevated levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) have been considered indicators of liver fibrosis, hepatocellular carcinoma, non-alcoholic steatohepatitis (NASH), and non-alcoholic fatty liver disease (NAFLD). The metabolic activities in the liver depend on the sources of dietary nutrition, and when there is excess energy consumption through food, it starts accumulating in various organs in the form of fat droplets.[3] Obesity also directly affects the liver by causing metabolic changes and leading to the development of NASH and NAFLD.[4]

Liver biopsy is an invasive, risky, and expensive procedure. On the other hand, liver function tests (LFTs), which measure the levels of liver enzymes, are cost-effective and non-invasive, and have potential in clinical settings. Therefore, the aim of this study was to identify the link between obesity and liver enzymes, specifically AST, ALT, and ALP, and to explore the associations between obesity and various sociodemographic, behavioural, and dietary parameters.

METHODS

The study was an institution-based cross-sectional observational study conducted at a diagnostic laboratory located in Burdwan city, Purba Bardhaman district, West Bengal, India from December 2023 to November 2024. All participants belonged to the age range of 18 to 59 years and were residents of Burdwan for more than one year. People who were already diagnosed with chronic liver diseases including hepatitis B, hepatitis C, alcoholic liver disease, and cirrhosis were excluded from the study. Additionally, people who had had active infection within the last month, were taking drugs known to alter liver enzyme levels, and had a history of psychiatric illness were excluded. Sample size was calculated using the formula of Charan and Biswas (2013), and based on this, 390 individuals were recruited for the study out of around 2500 eligible individuals using a systematic random sampling method. Informed consent was obtained from the participants before the study.[5]

Structured schedules were prepared to collect information from the participants regarding sociodemographic variables, behavioural variables, and health-related information. Questions were available in both Bengali and English. The sociodemographic variables comprised of age, gender, marital status, education level and type of occupation as per the Kuppuswamy scale, socioeconomic status, employment status, and family income.[6] Also, the socioeconomic status of the participants was calculated using the modified B. G. Prasad's socioeconomic status (SES) scale.[7] Behavioural information comprised of alcohol and tobacco use, physical activity status, and diet. For the anthropometric measurements, a flexible non-stretchable measuring tape was used to measure the height and a calibrated portable weighing machine was used to measure the weight. Basal mass index (BMI) was calculated using the values for height and weight, and this was used to categorize the participants based on their obesity status. Lastly, blood samples were collected from the participants and fasting blood glucose, serum lipid profile, and levels of AST, ALT, and ALP were determined using standard procedures.

Microsoft Excel was used to collect and organize participant data, and for carrying out preliminary calculations and generating graphs. Data analysis was performed using Jamovi software, v2.3.28, which was used to test relationships between variables, assess the significance of the relationships, and evaluate differences in the data.

Descriptive statistics were computed and chi-square tests were performed for categorical variables. Students' t-tests were used to compare continuous variables across the BMI categories and Pearson correlation was done to assess the relationships between continuous variables. The significance level was set at $p<0.05$. For multiple binary logistic regression analyses, variables with $p<0.1$ in bivariate analysis were considered.

RESULTS

Sociodemographic variables

Out of a total of 390 participants, 261 were male (66.9%) and 129 were female (33.1%). With regards to age, 44 individuals (11.3%) belonged to the age range of 18-29, 65 (16.7%) belonged to the age range of 30-39, 73 (18.7%) belonged to the age range of 40-49, and 79 (20.3%) belonged to the age range of 50-59. Around 57.7% of males and 29% of females had completed graduation, while 58.5% of males and females were currently married. With respect to occupation, 4.9% of males and 2.6% of females were professionals, 22.6% of males and 10% of females owned businesses, and 19.7% of females were homemakers.

Behavioural variables

A total of 115 (29.5%) participants were found to smoke tobacco, while 68 (17.4%) used smokeless tobacco and 158 (40.5%) were regular alcohol users. In contrast, 67.4% of participants had never smoked, 76.2% had never used smokeless tobacco, and 53.3% had never consumed alcohol.

Considering physical activity related to work, 161 (41.3%) were sedentary workers and 152 (39%) were moderate intensity workers. Most participants spent an average of four to eight hours on work and more than 60 minutes travelling every day to and from work. Apart from this, 124 (31.8%) participants performed high-intensity exercises, 130 (33.3%) performed medium-intensity exercises, and 136 (34.9%) performed low-intensity exercises.

Dietary characteristics

The average consumption of cooking oil for the participants was 32.1 ml/day and that of ghee and butter was 13.2 gm/day. The average sugar and salt consumptions were 21.2 gm and 6.3 gm respectively. A total of 221 participants consumed fast foods on four or more days per week.

Relationship between obesity and sociodemographic, behavioural, and dietary variables

Chi-square tests were performed to understand the association between obesity and sociodemographic variables, behavioural variables, and dietary variables (Tables 1-3).

Table 1: Association between obesity and sociodemographic variables (n=390).

Sociodemographic characteristics	N	Obese; N (%)	Statistical significance
Age (years)			
18-29	63	43 (68.3)	X^2=4.76 df=3 p=0.19
30-39	99	58 (58.6)	
40-49	107	74 (69.2)	
50-59	121	87 (71.9)	
Sex			
Male	261	176 (67.4)	X^2=0.023 df=1 p=0.88
Female	129	86 (66.7)	
Religion			
Hindu	345	231 (67.0)	Fisher Exact test p=0.64
Muslim	38	25 (65.8)	
Others	7	6 (85.7)	
Caste			
General	124	86 (69.4)	X^2=1.15 df=3 p=0.77
OBC	95	66 (69.5)	
SC	80	52 (65.0)	
ST	91	58 (63.7)	
Level of education			
Higher secondary	52	36 (69.2)	X^2=0.115 df=1 p=0.73
Graduate and above	338	226 (66.9)	
Type of occupation			
Professional	127	91 (71.7)	X^2=1.72 df=2 p=0.42
Semi-professional	231	150 (64.9)	
Others	32	21 (65.6)	
Marital status			
Never married	41	29 (70.7)	X^2=0.262 df=1 p=0.6
Ever married	349	233 (66.8)	
SES			
Upper	293	195 (66.6)	X^2=0.21 df=1 p=0.65
Upper middle	97	67 (69.1)	

Table 2: Association between obesity and behavioural variables (n=390).

Variables	N	Obese; N (%)	Statistical significance
Use of addictive substances			
Tobacco smoking only (bidi/cigarette)			
Never	263	178 (67.7)	X^2=4.01 df=2 p=0.135
Past	12	11 (91.7)	
Current	115	73 (63.5)	

Continued.

Variables	N	Obese; N (%)	Statistical significance
Smokeless tobacco only (Khaini/Gutka/Snuff)			
Never	297	193 (65.0)	X^2=4.34 df=2 p=0.114
Past	25	16 (64.0)	
Current	68	53 (77.9)	
Alcohol only			
Never	208	144 (69.2)	X^2=1.30 df=2 p=0.522
Past	24	17 (70.8)	
Current	158	101 (63.9)	
Physical activity related to occupation			
Nature of work			
Sedentary	161	108 (67.1)	X^2=2.03 df=2 p=0.362
Light	77	47 (61.0)	
Moderate	152	107 (70.40)	
Average time spent at work (hours / day)			
4–8	176	121 (68.8)	X^2=0.359 df=1 p=0.549
>8	214	141 (65.9)	
Time spent on travelling to and from work (mins/day)			
<30	57	40 (70.2)	X^2=0.279 df=2 p=0.87
30–60	92	61 (66.3)	
>60	241	161 (66.8)	
Physical activity not related to occupation			
Time spent on household chores (mins/day)			
<30	119	72 (60.5)	X^2=4.54 df=2 p=0.103
30–60	206	141 (68.4)	
>60	65	49 (75.4)	
Time spent on physical exercise (mins/day)			
<30	207	143, (69.1)	X^2=0.724 df=1 p=0.395
30–60	183	119, (65.0)	
Type of exercise based on intensity (self-reported)			
Low	136	91 (66.9)	X^2=0.172 df=2 p=0.918
Medium	130	86 (66.2)	
High	124	85 (68.5)	

Table 3: Association between obesity and dietary variables (n=390).

Dietary consumption (g/day/person)	N	Obese; N (%)	Statistical significance
Cooking oil (ml)			
<50	349	228 (65.3)	X^2=5.15, df=1, p=0.023
50-80	41	34 (82.9)	
Butter/ghee (g)			
<20	309	205 (66.3)	X^2=0.472, df=1, p=0.492
20-40	81	57 (70.4)	
Sugar			
Low (<15)	90	42 (46.7)	X^2=31.4, df=2, p=0.001
Medium (15-25)	172	114 (66.3)	
High (>25)	128	106 (82.8)	
Salt			
Low (<5)	125	90 (72.0)	X^2=1.94, df=1, p=0.164
Medium (5-10)	265	172 (64.9)	
Fast food (frequency/week)			
≤3	169	106 (62.7)	X^2=2.69, df=1, p=0.1
4-7	221	156 (70.6)	

Table 4: Pearson's correlation matrix using selected continuous variables (n=390).

Variables	Body mass index (kg/m^2)	SBP (mmHg)	DBP (mmHg)	Fasting plasma glucose (mg/dl)	Fasting serum cholesterol (mg/dl)	Fasting serum triglyceride (mg/dl)	Serum SGOT (u/l)	Serum SGPT (u/l)	Serum ALP (u/l)
Body mass index (kg/m^2)	1.00								
SBP (mmHg)	0.85#	1.00							
DBP (mmHg)	0.72#	0.72#	1.00						
Fasting plasma glucose (mg/dl)	0.61#	0.60#	0.53*	1.00					
Fasting serum cholesterol (mg/dl)	0.44*	0.43*	0.43*	0.30	1.00				
Fasting serum triglyceride (mg/dl)	0.48*	0.48*	0.41*	0.37	0.23	1.00			
Serum SGOT (u/l)	0.55*	0.46*	0.42*	0.32	0.30	0.33	1.00		
Serum SGPT (u/l)	0.47*	0.43*	0.41*	0.29	0.28	0.27	0.80#	1.00	
Serum ALP (u/l)	0.25	0.29	0.26	0.22	0.17	0.21	0.09	0.12	1.00

All Pearson's r values were statistically significant ($p<0.05$), *moderate correlation (Pearson's r=0.4 to <0.6), #high correlation (Pearson's r>0.6)

Table 5: Association between obesity and selected independent variables (n=390).

Explanatory variables	N	Obese, N (%)	OR (95% CI), p value	AOR (95% CI), p value
Cooking oil consumption (ml/day/person)				
50-80	41	34 (82.9)	2.58 (1.11-5.99), 0.028	2.32 (0.76-7.0), 0.137
<50	349	228 (65.3)	Ref	Ref
Sugar consumption (g/day/person)				
High (>25)	128	106 (82.8)	5.51 (2.97-10.22), 0.001	3.39 (1.38-8.31), 0.008*
Medium (15-25)	172	114 (66.3)	2.25 (1.33-3.78), 0.002	2.42 (1.07-5.48), 0.034*
Low (<15)	90	42 (46.7)	Ref	Ref
Fast food consumption (frequency/week)				
4-7	221	156 (70.6)	1.43 (0.93-2.18), 0.102	1.1 (0.57-2.13), 0.77
≤3	169	106 (62.7)	Ref	Ref
SBP (mmHg)			1.42 (1.32-1.52), 0.001	1.27 (1.16-1.39), 0.001*
DBP (mmHg)			1.41 (1.31-1.51), 0.001	1.04 (0.94-1.16), 0.461
Fasting plasma glucose (mg/dl)			1.14 (1.11-1.17), 0.001	1.07 (1.03-1.1), 0.001*
Fasting serum total cholesterol (mg/dl)			1.024 (1.02-1.03), 0.001	1.002 (0.99-1.01), 0.673
Fasting serum triglyceride (mg/dl)			1.035 (1.02-1.05), 0.001	0.99 (0.98-1.01), 0.671
SGOT (u/l)			1.11 (1.08-1.14), 0.001	1.1 (1.04-1.16), 0.001*
SGPT (u/l)			1.09 (1.06-1.11), 0.001	0.97 (0.92-1.02), 0.262
ALP (u/l)			1.014 (1.01-1.02), 0.001	0.99 (0.98-1.01), 0.611

*Statistically significant ($p<0.05$), model characteristics: R2 (McFadden)=0.507, AUC=0.926

The Pearson's correlation matrix showed that BMI had high correlation with SBP, DBP, and fasting plasma glucose (Table 4). SBP correlated strongly with DBP and fasting plasma glucose. Correlation between AST and ALT was also high among the study participants.

Univariate logistic regression analysis between various independent variables and obesity was carried out and it was seen that increase in sugar consumption, SBP, fasting plasma glucose levels and AST levels increased the likelihood of being obese (Table 5).

DISCUSSION

There has been a considerable increase in obesity globally, which has in turn increased the risk for many chronic diseases such as metabolic syndrome, diabetes, CVD, and NAFLD. Several studies have attempted to identify the links between obesity and different sociodemographic, behavioural, dietary, and health-related factors. One of these is high levels of liver enzymes namely ALT, AST, and ALP.[8] Changes in the levels of these enzymes have been linked to changes in liver function.[9] This study was carried out to explore the link between obesity prevalence and liver enzyme levels, so that liver enzymes can be used as biomarkers for liver dysfunction in people with overweight and obesity.

In this study, high levels of AST were found in 67.44% of the participants, high levels of ALT were found in 42.56% of the participants, and high levels of ALP were found in 45.13% of the participants. In all three cases, the levels were higher in obese individuals than in non-obese individuals, with t-values ranging from -3.59 to -10.08. Obesity and liver enzyme levels have been demonstrated to have a significant relationship among Italians.[10] Another report demonstrated the link between increased ALT activity and high BMI and waist circumference.[11] A large cohort study of 9748 individuals also found a significant association between high levels of ALT and high BMI and waist circumference.[12]

The associations between obesity and sociodemographic variables, use of addictive substances, and levels of physical activity were not statistically significant. However, physical inactivity has been significantly associated with obesity in other studies.[13,14] Regarding smoking, there have been mixed reports with some studies indicating a negative relationship between obesity and smoking, while some studies indicating a positive relationship between obesity and smoking.[15-17] The reason for smokers to have a lower body weight may be attributed to the fact that cigarettes contain about 10-15 mg of nicotine, which lowers the appetite and increases the metabolism of individuals.[18,19] For the same reason, people who quit smoking tend to overeat to replace cigarettes with food resulting in increased body weight among past smokers compared to current smokers.[20,21] Alcohol use has also been linked to obesity in several studies.[22,23] With respect to dietary variables, cooking oil and sugar consumption were significantly associated with obesity, while butter/ghee, salt, and fast-food consumption had no significant association with obesity. High consumption of sunflower oil has particularly been linked to abdominal obesity.[24] While ghee consumption has been linked to increased serum triglyceride levels, it has been found to have no significant association with body weight.[25,26] Increased sugar consumption has been linked to liver fibrosis, liver disease, and NAFLD.[27,28] In contrast to findings of our study, high salt intake has been associated with increased risk of obesity, while high fast-food consumption is considered to be one of the most important causes of obesity worldwide.[29-33]

CONCLUSION

The findings of this study have highlighted significant correlations between obesity and elevated levels of liver enzymes, indicating that obesity may be a risk factor for liver disease. Also, the presence of high liver enzyme levels among obese individuals can serve as markers for metabolic liver disease. However, this study has certain limitations. This study is cross-sectional, and so, it was not possible to track changes in the relationship between obesity and liver enzyme levels over a period of time. The study was carried out in a single diagnostic laboratory setting, and hence, the results may not be generalizable to other demographic groups. Future research must aim to identify and target high-risk individuals and encourage them to follow a healthier lifestyle to avoid long-term health complications.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. World Health Organization. Noncommunicable diseases country profiles 2018. 2018. Available at: https://www.who.int/publications/i/item/ncd-country-profiles-2018. Accessed on 28 July 2025.
2. World Health Organization. Global strategy on diet, physical activity and health. 2003. Available at: https://www.who.int/publications/i/item/9241592222. Accessed on 28 July 2025.
3. Mundi MS, Velapati S, Patel J, Kellogg TA, Abu Dayyeh BK, Hurt RT. Evolution of NAFLD and its management. Nutr Clin Pract. 2020;35(1):72-84.
4. Younossi ZM, Golabi P, Price JK, Owrangi S, Gundu-Rao N, Satchi R, et al. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among patients with type 2 diabetes. Clin Gastroenterol Hepatol. 2024;22(10):1999-2010.
5. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? Indian J Psychol Med. 2013;35(2):121-6.
6. Majumder S. Socioeconomic status scales: Revised Kuppuswamy, BG Prasad, and Udai Pareekh's scale updated for 2021. J Fam Med Prim Care. 2021;10(11):3964-7.
7. Ghodke M. Updated BG Prasad's socioeconomic status classification for the year 2023. Indian J Community Med. 2023;48(6):934-6.
8. Van Beek JHDA, De Moor MHM, De Geus EJC, Lubke GH, Vink JM, Willemsen G, et al. The genetic architecture of liver enzyme levels: GGT, ALT and AST. Behav Genet. 2013;43(4):329-39.

9. Giannini EG. Liver enzyme alteration: a guide for clinicians. Can Med Assoc J. 2005;172(3):367-79.
10. Marchesini G, Avagnina S, Barantani EG, Ciccarone AM, Corica F, Dall'Aglio E, et al. Aminotransferase and gamma-glutamyl transpeptidase levels in obesity are associated with insulin resistance and the metabolic syndrome. J Endocrinol Invest. 2005;28(6):333-9.
11. Oh SY, Cho YK, Kang MS, Yoo TW, Park JH, Kim HJ, et al. The association between increased alanine aminotransferase activity and metabolic factors in nonalcoholic fatty liver disease. Metabolism. 2006;55(12):1604-9.
12. Xu L, Jiang CQ, Schooling CM, Zhang WS, Cheng KK, Lam TH. Liver enzymes as mediators of association between obesity and diabetes: the Guangzhou Biobank Cohort Study. Ann Epidemiol. 2017;27(3):204-7.
13. Hirani S, Kuril B, Lone D, Ankushe R, Doibale M. Obesity prevalence and its relation with some sociodemographic factors in bank employee of Aurangabad city, Maharashtra, India. Int J Community Med Public Health. 2016;1628-35.
14. Kadarkar K, Tiwari S, Velhal G, Giri P. Physical activity levels during work, leisure time and transport and its association with obesity in urban slum of Mumbai, India. Int J Community Med Public Health. 2016;715-20.
15. Patel M, Kaufman A, Hunt Y, Nebeling L. Understanding the relationship of cigarette smoking trajectories through adolescence and weight status in young adulthood in the United States. J Adolesc Health. 2017;61(2):163-70.
16. Plurphanswat N, Rodu B. The association of smoking and demographic characteristics on body mass index and obesity among adults in the U.S., 1999–2012. BMC Obes. 2014;1(1):18.
17. Veldheer S, Yingst J, Zhu J, Foulds J. Ten-year weight gain in smokers who quit, smokers who continued smoking and never smokers in the United States, NHANES 2003–2012. Int J Obes. 2015;39(12):1727-32.
18. Stedman RL. Chemical composition of tobacco and tobacco smoke. Chem Rev. 1968;68(2):153-207.
19. Schmidt HD, Rupprecht LE, Addy NA. Neurobiological and neurophysiological mechanisms underlying nicotine seeking and smoking relapse. Complex Psychiatry. 2018;4(4):169-89.
20. Moffatt RJ, Owens SG. Cessation from cigarette smoking: Changes in body weight, body composition, resting metabolism, and energy consumption. Metabolism.1991;40(5):465-70.
21. Sinha R, Jastreboff AM. Stress as a common risk factor for obesity and addiction. Biol Psychiatry. 2013;73(9):827-35.
22. AlKalbani SR, Murrin C. The association between alcohol intake and obesity in a sample of the Irish adult population, a cross-sectional study. BMC Public Health. 2023;23(1):2075.
23. Arif AA, Rohrer JE. Patterns of alcohol drinking and its association with obesity: data from the third national health and nutrition examination survey, 1988–1994. BMC Public Health. 2005;5(1):126.
24. Narasimhan S, Nagarajan L, Vaidya R, Gunasekaran G, Rajagopal G, Parthasarathy V, et al. Dietary fat intake and its association with risk of selected components of the metabolic syndrome among rural South Indians. Indian J Endocrinol Metab. 2016;20(1):47.
25. Sharma H, Zhang X, Dwivedi C. The effect of ghee (clarified butter) on serum lipid levels and microsomal lipid peroxidation. AYU Int Q J Res Ayurveda. 2010;31(2):134.
26. Mohammadi Hosseinabadi S, Nasrollahzadeh J. Effects of diets rich in ghee or olive oil on cardiometabolic risk factors in healthy adults: a two-period, crossover, randomised trial. Br J Nutr. 2022;128(9):1720-9.
27. Jensen T, Abdelmalek MF, Sullivan S, Nadeau KJ, Green M, Roncal C, et al. Fructose and sugar: A major mediator of non-alcoholic fatty liver disease. J Hepatol. 2018;68(5):1063-75.
28. Chen J, Sun M, Adeyemo A, Pirie F, Carstensen T, Pomilla C, et al. Genome-wide association study of type 2 diabetes in Africa. Diabetologia. 2019;62(7):1204-11.
29. Ma Y, He FJ, MacGregor GA. High salt intake: Independent risk factor for obesity? Hypertension. 2015;66(4):843-9.
30. MacGregor G. ME 03-2 High salt intake as a cause of obesity. J Hypertens. 2016;34(1):e379.
31. Allison SJ. High salt intake as a driver of obesity. Nat Rev Nephrol. 2018;14(5):285-285.
32. Musaiger A. Strategy to combat obesity and to promote physical activity in Arab countries. Diabetes Metab Syndr Obes Targets Ther. 2011;89.
33. Al-Mahroos F, Al-Roomi K. Overweight and obesity in the Arabian Peninsula: an overview. J R Soc Promot Health. 1999;119(4):251-3.

Cite this article as: Dan S, Banerjee P, Dan S, Sadhukhan KS. Association between obesity and liver enzyme levels: a cross-sectional observational study among individuals visiting a diagnostic laboratory in Burdwan, West Bengal. Int J Community Med Public Health 2025;12:4366-72.