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Original Article

Association between dietary glycemic index and liver enzymes level among apparently healthy adults

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ABSTRACT

Objective: The previous studies have revealed that there is a link between dietary glycemic index and lipid profile in overweight and obesity. The aim of study was to investigate whether the glycemic index is associated with liver enzymes.

Method: Anthropometric and biochemical parameters were measured in 265 participants. Dietary glycemic index (GI) was assessed by using a validated food-frequency questionnaire. With adjusting confounder variable, Binary logistic regression was also used to predict the relationship between liver enzymes and quartile of intake.

Results: There was a significant difference between low and high GI diet for BMR (P = 0.01), FFM (P = 0.03), TG (P = 0.02), HDL (P = 0.002). The association between HDL and glycemic index remained significant after adjustment of sex and age (P = 0.03). Using the regression model following adjustment revealed that for each 1% increase in the degree of the GI, there was 11% elevation in liver enzyme abnormalities. In both groups of men and women, enzyme abnormalities positively correlated with GI, while only men showed remarkable correlation in all models (crude model: β = 0.07, OR = 1.07, CI = 0.98to 1.16). Additionally, an increase in the degree of GI caused an elevation in enzyme abnormalities by 7%. With adjusting sex, age, BMI, and Physical activity, a significance correlation was found between GI and Enzyme abnormalities (p-value = 0.03, OR = 1.115).

Conclusion: Our study indicated that high glycemic index diet led to the elevated levels of the liver enzymes, while being significant only in men.

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1. Introduction

Liver is one of the main organs of the body with a wide spectrum of functions which can be tracted via biochemical tests. Non-alcoholic fatty liver disease (NAFLD) is a disorder which has been reported to affect 9%–37% of the general population [1]. This organ has a wide spectrum of independent vital functions that unlike kidney and heart is difficult to measure. The main goal for clinical investigation of liver function is to diagnose possible diseases or their complications and to discern disease progression and thereby enhance response to therapy. The diseases of liver range from steatosis (fat infiltration into the liver) and steatohepatitis (hepatocellular inflammation and injury) to fibrosis, cirrhosis, and ultimately cancer [2,3]. When serum concentrations of alanine transaminase (ALT) exceed 30 IU and 19 IU in men and women, the likelihood of NASH disease increases, while an aspartate transaminase (AST)-to-ALT ratio of >0.9 may be associated with progressive fibrosis [2,4]. On the other hand, Nutritional abnormalities

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to some extent related to the metabolic disturbances of severe chronic liver disease [5].

Nutritional status tremendously affects liver as does other vital organs. The nutrient homeostasis is shown to be governed in liver, where dietary nutrient metabolism occurs. Besides, the liver function is associated with obesity and metabolic syndrome [6]. After a meal, liver takes up carbohydrate to restore glycogen stores and excess dietary carbohydrate is converted to lipids in de novo pathway [7]. The increased dietary fat and carbohydrate lead to the fat accumulation in the liver. The resulted liver disease elevates insulin levels which in turn give rise to the insulin resistance [6,7]. Weight loss and supplementation with dietary antioxidants have been suggested for to be used for the control of (nonalcoholic fatty liver) NAFLD and (nonalcoholic steady hepatitis) NASH [8–11], While change in lifestyle aimed at improving weight loss has been proposed in a number of studies as the principal therapy [12,13], there still exists degree of uncertainty about whether weight loss and/or change in the quality of food improve liver function, IR, dyslipidemia, and body composition [12,13].

Prevalence of high-grade fatty liver increases significantly throughout high dietary glycemic index: inasmuch as in a research the prevalence in patients with high GI was two times greater than that in those with low dietary GI [4]. The increased liver enzymes are a regarded as an alarm for fatty liver disease. According to epidemiological data, weight loss and lower food intake may lower dietary glycemic index. Also, dietary GI appears to be associated with suitable lipid profile and lower concentrations of C-reactive protein in people with overweight and obesity. It has been proposed that the metabolic effects of dietary carbohydrates may be of particular importance in people suffering from insulin resistance [8]. However, consuming a diet with high glycemic-index in insulin-sensitive subjects has showed no impact on hepatic steatosis [4]. With this in mind, we aimed to examine the association of glycemic index diets with liver enzymes and other variables.

2. Methods and materials

2.1. Population study

The current cross-sectional study was conducted in Central and West regions of Iran based on cluster sampling. The subjects included 126 males and 139 females whose ages ranged between 18 and 55 years.

Inclusion criteria were an age of 18–55 years, no alcohol or drug abuse, absence of any acute or chronic inflammatory disease, no history of hypertension, and not being pregnant. Exclusion criteria, on the other hand, were subjects with alcohol or drug abuse, history of hypertension, being pregnancy, current smoking, having thyroid, hepatic, renal or cardiovascular diseases, heart failure, malignancies, diabetes mellitus, being in any acute or chronic inflammatory state that affects inflammatory markers and any kind of infection. An informed written consent of participation in the study was signed by the subjects.

This study was approved by local Ethics Committee of the Endocrinology and Metabolism Research Center of University of Medical Sciences (ethic number: 93-04-159-28031-144521).

2.2. Anthropometric assessments

Height was measured in subjects with shoes off and they were weighed in light clothings. Waist circumference was measured in the slimmest area when subjects at the first of a normal respiration to the nearest 0.1 cm. Hip circumference was also measured in the largest part of the hip when subjects had worn light clothes.

2.3. Complete body composition analysis

BIA was measured using a TANITA BC-418 MA instrument. The main outputs of device are BMI, FM, FFM, TBW and visceral fat levels using manufacturers equations. For this equipment, impedance is measured with the participant standing and holding hand grips. This instrument works by dispatching a very weak electric current for measuring the impedance (electrical resistance) of the body. The subject's age, gender, and HT data were entered into the machine, and a standard of 2 kg was entered as an adjustment of clothing weight in all subjects. Subjects were then asked to stand barefoot on the machine while holding the handles for approximately 1 min. Measurements were conducted as the subject sufficiently rested, owing to prevent a possible disagreement in measured values. The body composition analyses were executed during the morning in a fasting status (always urinating before taking measurements, etc.). Trunk fat was reported along with other important body composition components including fat percent, fat mass, free fat mass, and visceral fat.

2.4. Dietary intake assessment

The dietitian taught participant to fill 147 items of food frequency questionnaire, validity and reliability of which has already been approved [14]. In the presence of a trained dietitian, the subjects were asked to fill in the questionnaires according to their usual diet. Data were recorded in household measures servings and finally converted into grams and milliliters. Also, Dietary intake data were analyzed using the NUTRITIONIST 4 (First Data Bank, San Bruno, CA) food analyzer.

2.5. Glycemic index calculation

After each food test, the Area under the glycemic response curve (AUC) for each subject was shown as a percentage of the mean AUC drawn by the reference food in the same subject. The mean of these values for all the subjects was determined as the food GI. In case of using white bread as the reference food, the GI values were multiplied by 0.71 to be transformed to the glucose scale (i.e. the GI of glucose = 100) [15].

Total dietary GI was estimated using the following formula: $\sum$ (GIa X available carbohydrate$_a$)/total available carbohydrate. To calculate available carbohydrate, fiber$_a$ was subtracted from total carbohydrate$_a$, derived from the USDA food composition table [16].

2.6. Abnormality definition

AAR: In this study, enzyme abnormalities AST/ALT ratio above 1 considered as abnormal.

2.7. Blood pressure measurement

Blood pressure for all participants was measured following 15-min rest and on the chair-by Automatic Inflate Blood Pressure Monitor (Samsung BA507S automatic digital blood pressure monitor, Samsung America, Inc.).

2.8. Blood sampling and biochemical parameters

The participants admitted in the Shariati Hospitalans blood samples were taken in the fasting state. All baseline blood samples were obtained between 8:00 a.m. and 10:00 a.m. After

centrifugation, serum was isolated and stored at 80 °C. IR was assessed using the homeostasis model assessment (HOMA) calculator software. Serum levels of insulin were measured by enzyme-linked immunosorbent assay (ELISA) kit: DRG International-U.S.A, EIA-2935. Moreover, serum concentration of 1,25 (OH)2D3 was determined using an enzyme-linked immunosorbent (RIA) assay (ELISA) kit)Bioactive Diagnostic-U.S.A origin, BD-200BA (based on the manufacturer's instructions. Aspartate aminotransferase (AST) and Serum alanine aminotransferase (ALT) were specified by an automatic analysis system (Autoanalyzer; Hitachi Ltd, Tokyo, Japan) with a Randox laboratories kit.

2.9. Assessments were as follows

Triglyceride measurements were performed by the enzyme glycerol-3-phosphate oxidase Phenol 4-Aminoantipyrine Peroxidase (GPO-PAP) method. **Serum high-sensitivity C-reactive protein** (hs-CRP), a pro inflammatory marker, was evaluated using a high-sensitivity Immunoturbidimetric assay (Hitachi 902 analyzer; Hitachi Ltd., Tokyo, Japan) and **Fasting Blood Sugar** (FBS) levels were measured by glucose oxidase phenol 4-Aminoantipyrine Peroxidase (GOD/PAP) method. Also, Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were determined by automatic analysis system (Autoanalyzer; Hitachi Ltd, Tokyo, Japan) with Randox laboratories kit. **Total cholesterol** (TC) levels, direct **low-density lipoprotein** (LDL) and **high-density lipoprotein** (HDL) cholesterol were measured via the enzymatic endpoint method and enzymatic clearance assay, respectively. 7 Randox Laboratories kits (Random Laboratories Ltd., Ardmore, UK) were used for all evaluations.

2.10. Statistical analysis

All statistical analyses were performed using SPSS 16 (SPSS Inc., Chicago, IL) software. The values were expressed as mean ± standard deviation. Besides, the Kolmogorov-Smirnov test was used to verify normal distributions.

Correlation between normal enzymes and abnormal enzymes was tested using independent sample T-test (as illustrated in Table 3), with the same approach being used to analyze the association between high glycemic index diet and low glycemic index diet (the results are presented in Table 2). Total diet intakes were adjusted for total calorie intakes through residual methods in linear regression. In addition, binary logistic regression was used to predict the relationship between liver enzymes and dietary intakes after adjusting the confounder variables. The level of significance was considered as p value ≤ 0.05 for all analyses.

3. Results

3.1. Descriptive statistics

A total of 265 patients, 126 men and 139 women, with a mean age of 35.08 (8.78) years, weight of 73.51 (15.66), height of 168.23 (9.43), BMI of 25.93 (4.89) were participated in this study (Table 1).

3.2. Association of GI and studied variables

An independent sample T-test was employed to assess the difference between low glycemic and high glycemic group in studied variables. As shown in Table 2, significance of independent T-test demonstrated the meaningful differences in BMR (P = 0.01), FFM (P = 0.03), TG (P = 0.02), and HDL (P = 0.002). The association between HDL and glycemic index was remained significant even following adjustment of sex and age (P = 0.03). However, this difference was not markedly found for age, weight, height, PA, waist circumference, hip circumference, WHR, SBP, DBP, BMI, Visceral fat, FBS, Insulin, HOMA-IR, cholesterol level, LDL, VIT D, HS-CRP, and AST (P > 0.05).(Table 2).

Table 1
Descriptive statistics.

	Min	Max	Mean	Std. Deviation
Age by year	18	55	35.08	8.78
Weight by (kg)	36.50	142.00	73.51	15.66
Height by centimeter	148.00	193.50	168.23	9.43
Waist by centimeter	58.00	130.00	88.80	12.50
Hip by centimeter	68.00	144.00	102.62	9.57
Systolic blood pressure	9.00	17.30	11.94	1.28
Diastolic blood pressure	5.00	10.90	7.73	.910
Fat percent	2.40	48.20	25.59	9.37
Visceral fat rating	1.00	17.00	5.52	3.40
FBS (mmol/L)	73.00	292.00	94.10	18.51
TG (mmol/L)	32.00	726.00	126.04	96.01
Cholesterol (mmol/L)	109.00	433.00	184.58	40.33
HDL (mg/dl)	20.00	84.00	48.78	11.68
LDL (mg/dl)	44.00	282.00	101.28	27.22
AST	5.00	70.00	20.46	6.82
ALT	4.00	121.00	17.05	10.97
CRP (mg/L)	.10	20.00	2.33	3.34
BMI	14.62	46.22	25.93	4.89
GIADJ	48.12	66.93	61.50	3.50
Glycemic index	42.67	67.60	59.31	6.70
Valid N (list wise)				

FBS fast blood sugar, TG triglyceride, HDL high-density lipoprotein, LDL low density protein, AST Aspartate Aminotransferase, ALT Alanine Aminotransferase, CRP C. Reactive protein, BMI Body mass.

3.3. Association of studied variable and abnormality in liver enzymes

We categorized participants based on having normal or abnormal levels of enzymes into two groups. At first, we used binary regression models to explore the association of enzyme abnormalities and studied variables, revealing that there was statistically significant differences in height (CI = 3.48 to 9.10), weight (CI = 4.67 to 14.06), BMR (CI = −325.80 to −130.83), waist circumference (CI = 1.97 to 9.72), WHR (CI = −0.6 to −0.1), TG (CI = 17.50 to 91.07), HDL (CI = −9.84 to −2.82), AST (CI = 2.66 to 8.23) and ALT (CI = 13.56 to 22.65) between two groups. With adjusting for sex and age, a significant higher BMI, age, weight, waist circumference, HIP, WHR were found in abnormal participants (Table 3).

3.4. Association of GI and enzyme abnormalities

In this study, it was evident from the binary regression that there was correlation between enzyme abnormalities level and GI. Enzyme abnormalities positively correlated with increased level of glycemic index. Among the studied patients, 51 showed no evidence of enzyme abnormalities while 213 subjects represented otherwise. In crude model: (beta = 0.07, odds ratio = 1.073), (CI = 0.987to 1.167), an increase in the degree of glycemic index led to 7% elevation in enzyme abnormalities. In the third model after adjustment of sex, age, BMI and physical activity, a significant correlation was found to be remained between GI and Enzyme abnormalities (p-value = 0.03, odds ratio = 1.115, CI = 1.010–1.231 (Table 4) (see Table 5).

Table 2
Association of GI and studied variables.

Demography	Lower GI Mean ± SD (n = …)	Highest GI* Mean ± SD (n = 156)	0.95 CI of the difference	P value	P value*
Age (years)	35.18 ± 8.97	34.20 ± 8.40	−1.53 to 3.48	0.446	0.6
Height (cm)	167.67 ± 9.68	170.36 ± 8.68	−5.40 to .01	0.051	0.30
Weight (kg)	73.28 ± 16.83	73.78 ± 12.09	−4.35 to 3.36	0.80	0.15
BMR	1573.74 ± 326.19	1686.83 ± 34.92	−206.38 to −19.78	0.01	0.68
PAIPAC	17.09 ± 17.94	21.20 ± 21.47	9.61 to 1.39	0.14	0.23
WAIST (cm)	88.52 ± 13.22	88.61 ± 10.12	−3.28 to 3.10	0.95	0.64
HIP(cm)	102.65 ± 10.32	101.79 ± 6.55	−1.36 to 9.09	0.44	0.63
WHRn	.86 ± .07	.869 ± .07	−0.02 to .01	0.39	
WHR	.86 ± .07	.869 ± .07	−0.02 to .01	0.39	0.45
SBP	11.88 ± 1.28	12.09 ± 1.34	−0.58 to .17	0.28	0.66
DBP	7.67 ± .90	7.87 ± .94	−0.46 to .06	0.13	0.11
Body Composition					
BMI(kg/m2)	26.003 ± 5.13	25.40 ± 3.91	−0.80 to 2.004	.39	0.43
FFM	49.71 ± 5.65	48.59 ± 3.69	−2.18 to −0.05	0.03	0.26
Visceral Fat	5.44 ± 3.44	5.50 ± 3.15	−1.04 to 0.90	0.89	0.52
Blood Parameters					
FBS(mmol/L)	94.05 ± 20.95	93.96 ± 9.75	−5.26 to 5.43	0.97	0.69
TG (mmol/L)	117.70 ± 85.64	147.01 ± 104.04	−55.07 to −3.54	0.02	0.10
Insulin (IU/mL)	12.99 ± 7.19	12.76 ± 6.17	2.36 to 2.83	0.85	0.81
HOMA_IR	2.92 ± 1.62	2.92 ± 1.45	59.19 to 0.58	0.99	0.92
CHOLESTROL	182.84 ± 38.43	187.59 ± 40.83	−15.85 to 6.36	0.40	0.54
HDL	49.98 ± 11.95	44.85 ± 10.03	1.85 to 8.39	0.002	0.03
LDL	99.64 ± 25.77	104.70 ± 28.30	−12.57 to 2.46	0.18	0.37
VITD (NG/mL)	23.58 ± 22.82	20.22 ± 12.63	−4.47 to 11.20	0.39	0.29
Inflammatory Cytokine					
HS-CRP (mg/L)	2.42 ± 3.39	1.92 ± 3.18	−0.44 to 1.45	0.29	0.55
Enzymes					
AST	20.01 ± 6.21	21.20 ± 7.81	−3.08 to 0.69	0.21	
ALT	16.33 ± 8.97	19.15 ± 15.72	−5.96 to 0.32	0.07	

BMI, Body mass index; FFM, Fat free mass; TBW, Total body water; FBS, Fasting blood sugar; TG, triglyceride; Tchol, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; LDL-ox, Oxidized low-density lipoprotein; VITD, Vitamin D; HS-CRP, High-sensitivity C-reactive Protein .

Table 3
Association of studied variable and abnormality in liver enzymes.

Demography	Normal Enzyme Mean ± SD (n = …)	Abnormal Enzyme Mean ± SD (n = 156)	0.95 CI of the difference	P value	P value*
Age (years)	35.25 ± 8.69	34.39 ± 9.30	−3.56 to 1.84	0.53	0.04
Height (cm)	166.99 ± 9.05	173.29 ± 9.45	3.48 to 9.10	0.0001>	0.6
Weight (kg)	71.69 ± 14.82	81.06 ± 16.99	4.67 to 14.06	0.0001>	0.01
BMR	1556.90 ± 314.78	1785.22 ± 301.82	−325.80 to −130.83	0.0001>	0.89
PAIPAC	17.78 ± 18.12	17.95 ± 21.16	5.94 to 5.78	0.97	0.69
WAIST (cm)	87.66 ± 12.40	93.51 ± 12.05	1.97 to 9.72	0.03	0.01
HIP(cm)	102.26 ± 9.47	104.28 ± 9.89	−0.98 to 5.01	0.18	0.04
WHR	0.85 ± 0.07	0.89 ± 0.06	−0.6 to −0.1	0.0001>	0.03
SBP	11.88 ± 1.32	12.19 ± 1.11	−0.08 to 0.71	0.12	0.6
DBP	7.70 ± 0.91	7.86 ± 0.88	−0.12 to 0.44	0.28	0.71
Body Composition					
BMI(kg/m2)	25.69 ± 4.82	26.98 ± 5.12	−0.22 to 2.80	0.09	0.01
Visceral Fat	5.37 ± 3.40	6.10 ± 3.41	−1.78 to 0.33	0.18	0.12
Blood Parameters					
FBS(mmol/)	94.22 ± 19.54	93.60 ± 13.54	−6.30 to 5.08	0.83	0.24
TG (mmol/L)	115.55 ± 84.85	169.84 ± 124.6	17.50 to 91.07	0.004	0.07
Insulin (IU/mL)	12.47 ± 6.25	14.85 ± 8.94	4.98 to 0.22	0.07	0.31
HOMA_IR	2.82 ± 1.44	3.34 ± 1.97	1.11 to 0.06	0.08	0.57
CHOLESTROL	184.34 ± 42.04	185.58 ± 32.57	−11.16 to 13.64	0.84	0.86
HDL	50.00 ± 11.71	43.66 ± 10.14	−9.84 to −2.82	0.0001>	0.42
LDL	100.99 ± 28.65	102.49 ± 20.32	−6.87 to 9.86	0.72	0.73
VITD (NG/mL)	22.79 ± 22.49	23.56 ± 11.80	−8.75 to 7.22	0.85	0.53
Inflammatory Cytokine					
HS-CRP (mg/L)	2.20 ± 3.15	2.90 ± 4.08	−0.32 to 1.72	0.18	0.55
Enzymes					
AST	19.41 ± 5.52	24.86 ± 9.55	2.66 to −8.23	0.0001>	
ALT	13.55 ± 5.02	31.66 ± 15.99	13.56 to 22.65	0.0001>	

BMI, Body mass index; FFM, Fat free mass; TBW, Total body water; FBS, Fasting blood sugar; TG, triglyceride; Tchol, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; LDL-ox, Oxidized low-density lipoprotein; VITD, Vitamin D; HS-CRP, High-sensitivity C-reactive Protein.

Table 4
Odds Ratio of Abnormality's liver enzymes test risk by Glycemic Index in the binary model.

	β (OR)	95% CI	P-value
Crude Model	0.07 (1.073)	0.987to 1.167	0.10
Model 1[a]	0.127 (1.135)	1.035to 1.244	0.007
Model 2[b]	0.118 (1.126)	1.024 to 1.237	0.014
Model 3[c]	0.109 (1.115)	1.010 to 1.231	0.031

Over weight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health.
[a] Adjusted for sex.
[b] Adjusted for sex, age and BMI.
[c] Adjusted for sex, age, BMI and Physical activity.

3.5. Association of GI and abnormality in liver enzyme levels assessed by binary regression model for different genders

Gender was the main factor in this regard. We separately analyzed data for male and female subgroups. In men, association was significant for all models and enzyme abnormalities showed positive correlation with GI, with being stronger in the crude model than that in other models. After adjustment of age, BMI and physical activity, a constant correlation was found between GI and enzyme abnormalities (P = 0.046) (beta = 0.125), (OR = 1.133), (CI = 1.002 to 1.281). For women, enzyme abnormalities levels positively correlated with GI, while not being meaningful for all models.

4. Discussion

The glycemic index term is derived from the fiber hypothesis [17]. The current study was the first investigation on the relation of dietary glycemic index with the liver enzymes and other studied variables in an adult population, demonstrating a remarkable association in men. Glycemic index prior to adjustment was found to be markedly linked with basal metabolic rate, fat free mass, triglyceride, HDL. after adjustment, the association was only seen for the HDL level. We also compared abnormality status in liver enzymes with other studied variables, indicating a significant correlation with HDL, Weight, WAIST circumference, HIP, WHR, Body mass index, and liver enzymes in men. Our findings demonstrated that abnormal liver enzymes was not significantly associated with height, basal metabolic rate, physical activity, systolic blood pressure, diastolic blood pressure, visceral fat, fast blood sugar, insulin, HOMA-insulin resistance, cholesterol, HDL, LDL, VIT-D, and hs-CRP.

This cross-sectional study was consistent with the our finding on the relation of High-GI diet with serum HDL-CHOLESTEROL concentration [18]. In a study by Jenkins and et al. (2002), having diets with low-glycemic index was associated with higher HDL-cholesterol concentrations. Furthermore in large cohort studies, this index demonstrated association with decreased risk of developing diabetes and cardiovascular diseases [17]. Amano and et al. (2004), highlighted the relationship between high dietary GI relationship and the low concentration of HDL-cholesterol, along with association between high triacylglycerol concentration and reduced immunoreactive insulin (Po0.01) [19]. Pelkman and et al. (2001). Moreover, several epidemiologic evidence have shown that the lower dietary GI was associated with lower serum triglycerides and higher HDL cholesterol [20]. Eral S et al. recently reported that the glycemic index and HDL-C concentration showed inverse association among 13907 participants aged ≥20 years in the third national health and nutrition examination survey (1988–1994) [21].

The current study revealed a positive correlation between the glycemic index and the extent of abnormality in liver enzymes, which was significant only in men. This may be related to sex hormones (Testosterone) affecting the glycemic index metabolism. Kapoor showed that the testosterone replacement therapy in hypogonadal men with Type 2 Diabetes reduced insulin resistance and improved glycaemic control [22]. Valtuena et al. showed that the high glycemic index diet led to the high grade of liver steatosis [8]; while in goletzke et al. study, carbohygrate quality showed no relationship with liver enzymes and serum lipids in older Australian population [23]. Interestingly in a study by S Kechagias et al. it was indicated that the hyper-alimentation and intake of simple sugars increased the level of ALT [24]. Another recently performed study showed that the elevated serum Aminotransferases among children with type 2 diabetes was not related to age, body mass index, glycemic control, blood lipids, or diabetic therapy [25].

High glycemic index diet lead to hyperinsulinemia which in turn results in the accumulation of fat in hepatocytes through two mechanisms [26]: a) increaseduptake of fatty acids in the hepatocytes which may cause hepatocellular injury, a consequence accompanied by increased serum level of ALT in the subjects, and b) raised entrance of different nutrients to liver and their convertion to fatty acid, which brings about the same consequence as the first mechanism. The main limitation of the present study was the relatively small sample size as well as failure in definitive diagnosis of liver diseases via liver function test. The strength of this study was being the pioneer to evaluate the association between GI and liver enzymes. The future studies are recommended to be carried out with experimental or interventional modalities in order to test our findings.

5. Conclusion

In summary, we found that the high glycemic index diet arisen from hypersecretion of insulin was associated with fat infiltration into the liver and elevated liver enzymes.

Table 5
Odd of liver enzymes abnormality by Glycemic index of dietary intakes among different gender.

Women			Men			
P-value	95% CI	β (OR)	P-value	95% CI	β (OR)	
0.166	0.952 to 1.336	0.12 (1.127)	0.021	1.020 to 1.270	0.130 (1.138)	Crude Model
0.157	0.954 to 1.341	0.123 (1.131)	0.02	1.021 to 1.272	0.131 (1.140)	Model 1[a]
0.321	0.920 to1.292	0.086 (1.090)	0.025	1.016 to 1.275	0.130 (1.138)	Model 2[b]
0.334	0918 to 1.287	0.083 (1.087)	0.046	1.002 to 1.281	0.125 (1.133)	Model 3[c]

[a] Adjusted for sex.
[b] Adjusted for sex, age and BMI.
[c] Adjusted for sex, age, BMI and Physical activity.

Conflicts of interest

The author declares no conflicts of interest.

Transparency declaration

The lead author affirms that this manuscript is an honest, accurate and transparent account of the study being reported, that no important aspects of the study have been omitted and that any discrepancies from the study as lanned (and registered with) have been explained.

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Appendix A. Supplementary data

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