ISSN 0974-3618 (Print) 0974-360X (Online) www.rjptonline.org



RESEARCH ARTICLE

The effect of Morbid Obesity on the Liver Function Enzymes

Amera Kamal Mohammed^{1,2}, Thanaa Abdulmahdi Mokif³, Mahmoud Hussein Hadwan^{4*}

¹Environmental Research and Studies Centers, Babylon University -51002
 ²Kirkuk University, College of Pharmacy
 ³Babylon Technical Institute, Al-Furat Al-Awsat Technical University, 51015, Hilla-Iraq.
 ⁴Chemistry Dept., College of Science, University of Babylon, Iraq, Hilla-Iraq
 *Corresponding Author E-mail: mahmoudhadwan@gmail.com

ABSTRACT:

Background: The liver is the major organ in the body and it is considered as an important organ for protein manufacture and detoxification. Liver health is evaluated by study liver function (LFTs) which include TSB, AST, ALT and ALK. The current study is an attempt to understand the correlation between morbid obesity and liver function enzymes. **Method**: The research population consisted of 67 subjects that divided into two groups; morbidly obese group (41) and the control group (healthy appearance subjects) (26). The levels of serum liver enzymes activities (ALT, AST, ALK) and TSB were determined by suitable kits. **Result:** In current study no statistically significant differences were observed in AST, ALT and ALK levels between the two types of procedures. The highest differences were noted for TSB levels between obese and individual healthy. While there was an association between the BMI and both AST and ALT; there was no relationship between the body mass index BMI and TSB and ALK. **Conclusion:** This study showed a correlation between the BMI with AST and ALT of morbidly obese.

KEYWORDS: Obesity, GPT, GOT, ALK, TSB.

INTRODUCTION:

The major organ in the body is the liver; it is considered as the most significant organ for detoxification and protein synthesis, both of which are facilitated by a number of enzymes. Therefore, analyze liver health is represented by estimating the enzymes that released from liver cells and proteins that created by the liver and released into the blood stream (1).

The most common tests of liver function (LFTs) include bilirubin, aspartate aminotransferase [AST or total glutamic oxaloacetic transaminase (SGOT)], alanine aminotransferase [ALT or glutamic pyruvic transaminase (SGPT)] and alkaline phosphatase (2) Whereas, bilirubin is the break down product of hemoglobin produced within the reticuloendothelial system, emitted in unconjugated form which enters into the liver, converted to conjugated forms bilirubin mono and diglucuronides by the enzyme UDP-glucuronyl transferase (3). While ALT is presence fundamentally in the liver, with clinically small quantities create in the skeletal muscle, kidneys and heart. AST is found, in tapering, order of concentration, in the liver, cardiac muscle, skeletal muscle, kidneys, pancreas, brain, leukocytes, lungs and erythrocytes (4).

As AST can be rise in diseases affecting other organs, involve acute hemolytic anemia pancreatitis, myocardial infarction, burns, muscular skeletal diseases and acute kidney injury. Thus, ALT is more specific for liver inflammation and disease than AST. In addition to that, clearance of serum aminotransferases involves catabolism by the reticuloendothelial system, and in that assessment, is comparable to the clearance of other serum proteins. AST is consumption more rapidly than ALT, and the major site of clearance is the hepatic sinusoidal cell (5). At the same time, alkaline phosphatase is important enzyme in the subject, which represents a group of isoenzymes created by liver, kidneys, bones, placenta and small intestine (6). The serum ALP level signifies the total quantities of isoenzymes that are released from the specialized tissues into the blood stream and its major function is thought facilitate transport across cell membranes and assistance in metabolism (7). A number of researchers including Meyer et al., and Donaldson et al. they suggested that liver enzymes abnormalities have relationship with obesity, hormone therapy or autoimmunity (8,9).

Obesity is one of the most significant public health in the worldwide. Furthermore, an extreme phenotype morbid obesity (MO) has damagingly become a global problem (10). Where through the course of the disease that adipocyte hypertrophy lead to hypoxia, inflammation and oxidative stress which increase morbidity and mortality of this complex endocrine and metabolic disease (11,12). The changes in plasma levels of various hormones and vitamins in obese patients were occurred because functioning of adipose tissue as an endocrine organ. These functions were produced metabolic changes in many organs (13).

MATERIAL AND METHODS:

Samples were collected from 67 Individuals at the Marjan teaching hospital (Babylon province/Hilla city/ Iraq), in February 2018 for aged 17 to 70 years, they were divided into in two groups as morbidly obese groups (41) and the control as healthy appearance groups (26). The formula BMI= weight (kg)/ height (meter)² using for the calculated the body mass index (BMI). The serum liver enzymes levels of alanine amino transaminase (ALT) aspartate amino (GPT), transaminase (AST) (GOT), alkaline phosphatase (ALK) and total serum bilirubin (TSB) were determined by kits that purchased from randox and biomerieux company.

RESULT:

In the current study, there was a significant difference in the mean age between morbidly obese and normal individuals (32.00 ± 14.95 vs. 23.92 ± 9.78) (years). The TSB also had significant difference at a significant level (0.81 ± 0.39 vs 0.69 ± 0.18 µmol/L; P ≤ 0.5). There were significant differences in the rate of GPT, GOT, ALK between morbidly obese and healthy individuals (19.09 ± 7.30 vs 14.41 ± 6.86 U/L), (22.09 ± 9.67 vs. 24.35 \pm 11.36 U/L) and (53.00 + 20.74 vs 53.73 + 20.35 LU/L) respectively in table (1).

Table 1 The mean differences between morbidly obese patients and control groups in parameters study.

control groups in parameters study.				
Group	Ν	Mean	Std.	p value
			Deviation	
control	26	23.92	9.78	0.03*
obese	41	32.00	14.95	
control	26	21.19	1.78	0.05*
obese	41	38.58	2.63	
control	26	14.41	6.86	0.63
obese	41	19.09	7.30	
control	26	24.35	11.36	0.24
obese	41	22.09	9.67	
control	26	53.73	20.35	0.82
obese	41	53.00	20.74	
control	26	.69	.18	0.04*
obese	41	.81	.39	
	Group control obese control obese control obese control obese control	GroupNcontrol26obese41control26obese41control26obese41control26obese41control26obese41control26obese41control26obese41control26obese41control26	Group N Mean control 26 23.92 obese 41 32.00 control 26 21.19 obese 41 38.58 control 26 14.41 obese 41 19.09 control 26 24.35 obese 41 22.09 control 26 53.73 obese 41 53.00 control 26 .69	Group N Mean Std. Deviation control 26 23.92 9.78 obese 41 32.00 14.95 control 26 21.19 1.78 obese 41 38.58 2.63 control 26 14.41 6.86 obese 41 19.09 7.30 control 26 24.35 11.36 obese 41 22.09 9.67 control 26 53.73 20.35 obese 41 53.00 20.74 control 26 .69 .18

BMI: Body Mass Index, GPT: alanine amino transaminase, GOT: aspartate amino transaminase, ALK: alkaline phosphatase, TSB: total serum bilirubin.^{*} significant levels at p value ≤ 0.05 .

In Figure (1) there was a relationship between BMI (Kg/M^2) and GPT (U/L) of morbidly obese (p value level is significant ≤ 0.05). According to the equation of the straight line Y=2.5+0.75*X. There was also a relationship between the BMI (Kg/M^2) and GOT (U/L) of morbidly obese equation of the straight line Y=3.33+96.66 * X; as shown in figure (2).

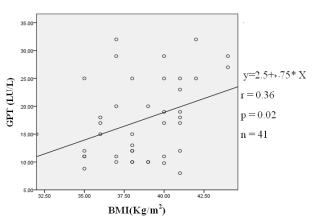


Figure (1): The correlation between BMI (Kg/M²) and alanine amino transaminase (ALT)[GPT] LU/L of morbidly obese group

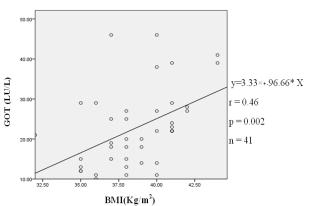


Figure (2): The correlation between BMI (Kg/M^2) and aspartate amino transaminase (AST, GOT) U/L of morbidly obese group

While there was no correlation between the BMI (Kg/m^2) and ALK (U/L) of morbidly obese as in figure (3). Also, in figure (4) there was no correlation between body mass index (Kg/m^2) and TSB µmol/L of morbidly obese according to the equation of the straight line Y=5.83+166.66*X and Y=0.16+5.33 *X respectively.

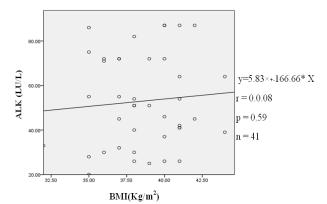


Figure (3): The correlation between BMI (Kg/m²) and alkaline phosphatase (ALK) U/L of morbidly obese group.

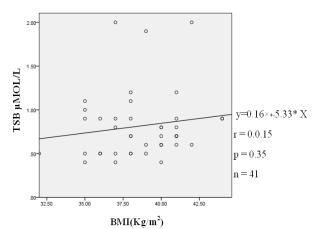


Figure (4): The correlation between BMI (Kg/m^2) and total serum bilirubin (TSB) μ mol/L of morbidly obese group.

DISCUSSION:

Currently, the number of obese individuals has reached the dimensions of frightful. The alteration in dietary type of the world population via its components from sugars and fats, attached with lowering physical inactivity, and this leads to largest increased prevalence of obesity not only in advanced but also in developing countries (14). The results of the current study showed significant differences between patients with excessive obesity and healthy in age and this corresponds to the study of Skinner et al., (15) where they observed increment in the levels of severe obesity in all ages in the past 18 years (15). Another study was showed that about 20–30% of the adult population is affected by "non-alcoholic fatty liver disease (NAFLD)" (16). On the other hand, the current study showed a relationship between the BMI with ALT and AST. The results have good agreement with the earlier study that demonstrate a relation between BMI and serum liver enzymes activity (ALT and AST)"(17). In addition, some published data were indicated that a significant combination between elevation ALT levels, metabolic syndrome, diabetes mellitus type-2, and insulin resistance (18). The study indicates severe cases of nonalcoholic fatty liver disease that causes abnormalities in serum AST and ALP levels (19). Nonalcoholic fatty liver disease has been associated with metabolic syndrome. It is concomitant with a decrease in beta oxidation and rises in free fatty acids, and as a result, increment the susceptibility to hepatic fibrosis (19,20). Also they reported that metabolism; age and BMI factors have great effects on ALT levels (21). Generality, various studies have demonstrated that ALT levels correlate with increasing BMI (22-24). Previous study that done on the Mexican population was proved that weight gain will increase ALT activity by more than 3 times (25). Another study completed in the United States of America was showed that the high prevalence of obesity will led to high incidence of "chronic liver disease and nonalcoholic fatty liver disease" (26). Javanta et al., (27) were introduced an evidence to clarify the association of NAFLD in developing countries like India with the increment of obesity (27). Several studies have good agreement with our study; the results were indicated no relationship with obesity and liver enzyme levels (28). On the other hand, earlier studies in this field have contrary results to our study (29,30,31). Perhaps there are other reasons for the rise of some liver enzymes such as exercise (32), Based on that; both AST and ALT may increase with vigorous exercise (32,33). Other reasons may be related to immunogenic factors or as a result to certain treatments.

Liver enzymes abnormalities have been suggested to have relationships with therapy and/or autoimmunity (34,35). There may be some other points to be noted which may be the cause of changes in liver enzymes. In the case of older people, demolitions are more than construction, and sugars are converted into fat due to calmness, relaxation, slow movement, lack of physical exertion and lack of exercise. At the stage of youth, the body is in constant activity and due to excessive eating and increasing the calorie in the youth stage, which leads to increased production of fat cells with age, gender difference, different genotypes and nature of food and the environment surrounding the people (14).

The most important point is the method of measuring lipid since measuring the body mass index is the first method and depends on the accuracy of this method to repeat the measurement process several times during specific periods. The layers under the skin determines the amount of body fat above the muscle where there are other methods more accurate are dual-energy x-ray absorptiometry (DAX). skinfold thicknesses (anthropometry), ultrasound, radiography and bioelectrical impedance analysis (BIA) to measure the proportion of body fat (36,37). Finally; the limitation of the present study was small sample size study. Further large sample size studies are required and include sex and genes to obtain results more comprehensive.

REFERENCE:

- Kasarala, G.; and Tillmann, H. L. Standard Liver Tests Clinical Liver Disease 2016; 8(1):13-18.
- Maan W, Warsi AQ, Rashed A, Karjoo M. Unexplained High Activity of Aspartate Aminotransferase in an Asymptomatic Pediatric Patient. Int J Pediatr 2017; 5(10): 5805-5808
- Mauro P, Renze B, Wouter W. Enzymes. In: Tietz text book of clinical chemistry and molecular diagnostics. Carl AB, Edward R, David EB. 4th edition, Elsevier 2006, 604-616.
- Giannini EG, Testa R, Savarino V. Liver Enzyme Alteration: A Guide for Clinicians. CMAJ. 2005;172(3):367–79.
- Lord R, Fahie-Wilson M, Suri S.A Paediatric Case of Macro Aspartate Aminotransferase. Ann Clin Biochem 2008; 45:323-4.
- Yulia. A, Wijesiriwardana. A. Extremely High Isolated Maternal Alkaline Phosphatase Serum Concentration -2 Case Reports. Int J Pregn& Chi Birth 2017; 2(4): 00031
- Salerno M, Di Maio S, Gasparini N, Rizzo M, Ferri P, Vajro P. Liver abnormalities in Turner syndrome. Eur J Pediatr. 1999;158(8):618-623.
- Donaldson MD, Gault EJ, Tan KW, Dunger DB. Optimising management in Turner syndrome: from infancy to adult transfer. Arch Dis Child. 2006;91(6):513-520.
- Meyer, R. E., Thompson, S.J., Addy, C.L., Garrison, C. Z., Best, R.G. Maternal serum placental alkaline phosphatase level and risk forpreterm delivery. Am J ObstetGynecol 1995;173(1): 181-186
- Chang H-C, Yang H-C, Chang H-Y, Yeh C-J, Chen H-H, Huang K-C, et al. Morbid obesity in Taiwan: Prevalence, trends, associated social demographics, and lifestyle factors. PLoS ONE 2017; 12(2): e0169577. doi:10.1371/journal. pone.0169577
- Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. Annu Rev Immunol 2011;29: 415-45.
- Swinburn BA, Caterson I, Seidell JC, James WP. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr 2004;7(1):123-46.
- Álvarez-Castro P, Sangiao-Alvarellos S, Brandón-Sandá I, Cordido F. EndocrinaFuncion in obesity. EndocrinolNutr 2011;58(8):422-32.
- World Health Organization. 2016. Obesity and overweight. Fact sheet No 311. Availableat http:// www.who.int/ mediacentre/ factsheets/ fs311/ en/ (accessed on September 2016).
- Skinner, A. C; Ravanbakht, S. N; Skelton J. A; Perrin E. M and Armstrong, S. C. Prevalence of Obesity and Severe Obesity in US Children, 1999–2016. Pediatrics. 2018;141(3): e20173459
- Lonardo A, Bellentani S, Ratziu V et al. Insulin resistance in nonalcoholic steatohepatitis: necessary but not sufficient— death of a dogma from analysis of therapeutic studies? Exp Rev GastroenterolHepatol 2011; 5:279–89.
- Salvaggio₃A.; Periti, M.; Miano, L.; Tavanelli, M. and Marzorati,
 D. Body Mass Index and Liver Enzyme Activity in Serum CLINICALCHEMISTRY,1991. 37 (5): 720-723
- Gómez, Z. S; Fernández, Q. A.; Lasa, A.; Hijona, E.; Bujanda, L. and Portillo M.P. Effects of resveratrol on obesity-related inflammation markers in adipose tissue of genetically obese rats. Nutrition. 2013; 29(11-12):1374-80.
- Marchesini, G.; Brizi, M.; Bianchi, G.; Tomassetti, S.; Bugianesi, E.; Lenzi, M.; Mccullough, A.J.;Natale, S.; Forlani, G. and Melchionda N. Nonalcoholic fatty liver disease: a feature of metabolic syndrome. Diabetes. 2001;50(8):1884-1850.
- 20. Alta'ee AH, Hadwan MH, Almashhedy LA. The balance between superoxide dismutase and catalase activi-ties in sera of obese Iraqi

men. Euro Mediterranean Biomedical Journal. 2015;10(17):197-202.

- Lee, J.K.; Shim, J.H. and Lee, H.C et al. Estimation of the healthy upper limits forserum alanine aminotransferase in Asian populations with normal liver histology. Hepatology2010; 51: 1577 – 83.
- Nomura, F.; Ohnishi, K. and Satomura, Y. et al. Liver function in moderate obesity--study in 534 moderately obese subjects among 4613 male company employees. Int J Obes1986; 10: 349 – 54.
- Wejstal, R.; Hansson, G. and Lindholm, A. et al. Persistent alanine amino transferase elevation in healthy Swedish blood donors--mainly caused by obesity. Vox Sang 1988; 55: 152 – 6.
- 24. Piton, A.; Poynard, T .and Imbert-Bismut, F. et al. Factors associated with serum alanine transaminase activity in healthy subjects: consequences for the definition of normal values, for selection of blood donors, and for patients with chronic hepatitis C. MULTIVIRC Group. Hepatology1998; 27: 1213 – 9
- 25. Flores, Y.N.; Auslander, A.; Crespi, M.C.; Rodriguez, M.; Zhang, Z. F. and Durazo, F. et al.. Longitudinal association of obesity, metabolic syndrome and diabetes with risk of elevated aminotransferase levels in a cohort of Mexican health workers. Journal of digestive diseases. 2016; 17:304-12
- 26. Younossi, Z. M.; Stepanova, M.; Afendy, M.; Fang, Y.; Younossi, Y.; and Mir, H. et al. Changes in the prevalence of the most common causes of chronic liver diseases in the United States from 1988 to 2008. Clinical gastroenterology and hepatology: the official clinical practice journal of the American Gastroenterological Association. 2011; 9:524-30.
- Jayanta, P.; Raj Vigna, V.; Lorance, P.;, Shihaz, H.; Kula Naresh, K.S. and Mohit P. Shetti .Effects of lifestyle modification on liver enzyme and Fibroscan in Indian patients with non-alcoholic fatty liver.2018. disease Gastroenterology Report, 6(1):49–53
- Rohania, F.; Golgiric, F.; Alaeid M. R., Karimie, M.; Nikraftarf, P. and Bozorgmehrf, R. Relationship Between Obesity and Liver Enzymes Levels in Turner's Syndrome. Gastroenterol Res. 2017;10(1):28-32
- Salerno M, Di Maio S, Gasparini N, Rizzo M, Ferri P, Vajro P. Liver abnormalities in Turner syndrome. Eur J Pediatr. 1999;158(8):618-623.
- El-Mansoury, M.; Berntorp, K.; Bryman,I.; Hanson, C.; Innala, E.; Karlsson, A. and Landin-Wilhelmsen, K. Elevated liver enzymes in Turner syndrome during a 5-year follow-up study. ClinEndocrinol (Oxf). 2008;68(3):485-490.
- Wasniewska, M.; Bergamaschi, R.; Matarazzo, P.; Predieri, B.; Bertelloni, S.; Petri, A.; Sposito, M. et al. Increased liver enzymes and hormonal therapies in girls and adolescents with Turner syndrome. J Endocrinol Invest. 2005;28(8):720-726.
- Dufour, D.R.; Lott, J.A.; Nolte, F.S.; Gretch, D.R.; Koff, R.S. and Seeff, L.B. Diagnosis and monitoring of hepatic injury. II. Recommendations for use of laboratory tests in screening, diagnosis, and monitoring. Clin Chem 2000; 46:2050–2068.
- Dufour, D. R. Effects of habitual exercise on routine laboratory tests. Clin Chem 1998;44: A136.
- Salerno, M.; Di Maio, S.; Gasparini, N.; Rizzo, M.; F erri, P. and Vajro, P. Liver abnormalities in Turner syndrome. Eur J Pediatr. 1999;158(8):618-623.
- Donaldson, M.D.; Gault, E.J.; Tan, K. W. and Dunger, D.B.Optimising management in Turner syndrome: from infancy to adult transfer. Arch Dis Child. 2006;91(6):513-520.
- Willems, A.; Paulson, TA.W.; Keil, M.; Brooke-Wavell, K .and Goosey-Tolfrey, V.L. Dual-Energy X-Ray Absorptiometry, Skinfold Thickness, and Waist Circumferencefor Assessing Body Composition in Ambulant and Non-Ambulant Wheelchair Games Players. Front. Physiol. (2015) 6:356
- 37. Demmer, D.L.; Beilin, L.J; Hands, B. Burrows, S.; Pennell, C.E; Lye, S.J.and et al. Dual Energy XRay Absorptiometry Compared with Anthropometry in Relation to Cardio-Metabolic Risk Factors in a Young Adult Population: Is the 'Gold Standard' Tarnished? PLoS ONE. (2016) .11(9): e0162164.