The Association Between Fatty Liver on Ultrasound and Liver Fnuction Testes

Sadiq Jabbar Al-muhanna[1] Sabah Ali J. Al-helu[2] Abdulrazzq Hassan Alkaaby[3]
Jabbar Abd- Alnabi
[1] Collage of Medicine, Kufa University
[2] Alsader Medical City, Najaf
[3] Alsader Medical City , Najaf

Received 24 September 2014 Accepted 9 July 2014

Abstract

Background: The presence of non-alcoholic fatty liver disease (NAFLD) on ultrasound study is a common finding in apparently healthy individuals. It may be associated with liver damage and may predispose to chronic liver disease .

Aim of the study: To show if there is any relation between fatty liver on ultrasound and hepatic damage.

Patients and methods:60 patients with fatty liver on ultrasound and 65 persons are control were enrolled in this study.72 Of them were males and 53 were females, their ages ranged from (30 – 68) years .

All patients underwent abdominal ultrasound and blood sample where taken and sent for liver function testes to detect any changes in liver enzyme and hepatic damage

Results: The result of this study consist of 60 patients with liver changes on ultrasound and 65 healthy controls, 72(57.6%) were males while 53(42.4%) were females .

In this study , 53(88.3%) of the patients had AST/ALT < 1, while only 7(11.7%) had AST / ALT >1 and all controls 65(100%) had AST/ALT < 1 .

32 of male and 21 of female patients (total 53) had AST/ALT < 1 while 4 male and 3 female (total 7) had AST / ALT >1 .

Conclusion: NAFL is not a benign condition and may lead to abnormal elevation in liver enzymes.

تدهن الكبد غير الكحولي بفحص الأمواج فوق الصوتية وعلاقته باختبارات وظائف الكبد

الخلاصة

خلفية البحث:

وجود تدهن الكبد غير الكحولي في فحص الأمواج فوق ألصوتيه غالبا ما يوجد في الأشخاص الأصحاء ظاهريا .ومن الممكن أن يكون له علاقة بضرر الكبد أو إصابته بمرض مزمن.

الهدف من الدراسة

هدف الدراسة لإثبات هل هناك علاقة بين تدهن الكبد غير الكحولي عند بعض الأشخاص الذي يظهر في فحص الأمواج فوق الصوتية وبين وجود ضرر في الكبد.

المرضى والطرق :

تتضمن الدراسة ستون مريضا يظهرون تدهن الكبد في فحص الأمواج فوق الصوتية وخمسة وستون آخرين لا يظهرون ذلك اخذوا كمقياس.

اثنان وسبعون منهم كانوا رجالا وثلاث وخمسون منهم كانوا نساء،تنحصر أعمارهم بين الثلاثين و ألثمان وستون سنة، خضعوا جميعهم لفحص البطن بالأمواج فوق الصوتية وأُخذت منهم عينات دم أرسلت لغرض الفحص ألمختبري لوظائف الكبد ومعرفة هل هناك تغير غير طبيعي في مستويات إنزيمات الكبد أو أي ضرر ممكن له.

النتائج :

تتكون نتائج هذه الدراسة من (٦٠) مريضا يظهرون تدهن الكبد بفحص الامواج فوق الصوتية و (٦٥) أصحاء .(٧٢) منهم رجالا و(٥٣) نساء.

(٥٣) من المرضى لديهمAST/ALT<1 بينما (٧) لديهم AST/ALT>1

اثنان وثلاثون من الرجال و ٢١ من النساء من المرضى لديهمAST/ALT<1

بينما ٤ من الرجال و ٣ من النساء لديهم AST/ALT > 1

الاستنتاج:

إن تدهن الكبد غير الكحولي قد يؤدي إلى تغير غير طبيعي في مستوى إنزيمات الكبد قد يعكس ضرره.

التوصيات :

يحتاج المرضى المصابون بتدهن الكبد غير الكحولي إلى المتابعة المستمرة .

Introduction

Nonalcoholic fatty liver disease (NAFLD) is the most common form of chronic liver disease in both children and adults and threatens to become a serious public health problem[1,2] .

NAFLD refers to a wide spectrum of diseases ranging from simple fatty liver to non alcoholic steatohepatitis to cirrhosis[3] .

The pathological picture resembles that of alcohol induced liver disease but occurs in those who do not abuse alcohol[4] .

NAFLD was initially believed to be a benign condition, Recent studies have shown it to be associated with obesity, type II diabetes mellitus, dyslipidemia and hypertension with insulin resistance being a common factor.

These conditions cluster to form the metabolic syndrome, which carries a high risk for cardiovascular disease[5] .

Although NAFL, which is the most common form of NAFLD, appears to follow a benign non progressive clinical course, NASH is a potentially serious condition because as many as 25% of these patients may progress to cirrhosis and experience complications of portal hypertension, liver failure, and hepatocellular carcinoma[6,7,8,9] .

Although some studies suggest the use of liver enzymes as a marker of NAFLD underestimates its prevalence[10] . mild to moderately elevated serum levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) or both are the most common findings, In which the elevation of liver enzymes by 1.5 to 5 times is corresponding with (NASH) and the ratio of AST/ALT of >1 in patient with NAFLD indicate liver damage[11] .

Palekar et al generated a clinical model to distinguish NASH from simple steatosis by combining 6 different variables including age (>50 years), female gender, AST (>45 U/L), body mass index(BMI >30 kg/m2), and AST/ALT ratio (>1), and serum hyaluronic acid (HA; >55 mg/L).

The presence of 3 or more of these factors had a sensitivity and specificity for NASH diagnosis of 74% and 66%, respectively.[12]

Ultrasound has a sensitivity of 89% and a specificity of 93% in detecting steatosis and a sensitivity and specificity of 77% and 89% in detecting increased fibrosis[13] .

CT and MRI are more sensitive for both, but ultrasound is more available, however it is operator dependable.

At present, the available noninvasive markers for NAFLD include a set of clinical signs and symptoms, easily available laboratory and radiological imaging tests, and combinations of clinical and blood test results.

Although several of these markers are in general useful for the diagnostic evaluation of a patient with suspected NAFLD, they lack specificity and sensitivity to distinguish NAFL from NASH and determine the presence and stage of fibrosis[14] , this represents a key clinical problem because patients with NASH and fibrosis are probably those who need close monitoring and follow up as well as the potential targets for therapeutic intervention when specific treatments for this condition become available[15] .

Patients and Methods

Sixty patients with non alcoholic liver disease and 65 healthy control were attended the AL- Sadder Medical City in AL-Najaf during the period from February 2012 to October 2012. (72) of them were males and (53) were females, were enrolled in this study, their ages ranged from (30 – 68) years with a mean age of 49.1±9.8 years .

Detailed history was taken including the age, sex, job, weight, height, body mass index (weight (kg) / square height in meters) and drugs history.

Blood samples were drawn and sent for liver enzymes level (alanine aminotransferase, aspartate aminotransferase,) by using Abbott Architect plus and viral serology using ELISA method by using Monobind co. Germany instrument .

All patients were examined by ultrasound for abdomen by using (Medison machine) .The following changes were taken into consideration for the proposal of analysis: The level of ALT, AST, AST/ALT ratio , BMI and ultrasound changes of fatty liver, fibrosis and cirrhosis.

In this study, we exclude patients with:

- Rapid weight loss.
- Wilson's disease.
- Hemochromatosis.
- Inflammatory bowel disease.
- Hepatitis B and C.
- Gastrointestinal surgery for obesity.
- Total parenteral nutrition.
- Protein caloric malnutrition.
- Human Immune deficiency virus infection.
- Pregnancy.
- Drugs.

Statistical analysis

This study is a cross sectional; statistics was performed using SPSS system, and p value of less than 0.05 was considered statistically significant.

Results:

The result of this study consist of 60 patients with fatty changes presentations on ultrasound , 37 patients were males and 23 patients were females with mean age of (49.10 ±9.80 year) and 65 healthy controls, 29 were females and 36 were males with mean age of (43.37±9.3 year) .

In this study, 14 patients had ALT > 50 and 46 of them had ALT < 50 while all healthy controls had ALT < 50 which is statistically significant (p value 0.0007) as shown in table (1).

Twenty-eight patients had BMI > 30 , of them (16) had ALT> 50 and (12) of them had ALT< 50 which is statistically significant (p value 0.0007) as shown in table (2).

Regarding the gender, only 5 of (37) of male patients had ALT > 50 while only 9 of 23 of female patients had ALT > 50 which is statistically significant (p value 0.022) as shown in table (3).

In this study, seven of patients and none of control had ALT/AST < 1 which is significant (p value 0.005) as shown in table (4).

Twenty-eight patients had age > 50, of them (15) had ALT> 50 and (13) of them had ALT< 50 which is statistically significant (p value 0.0007) as shown in table (5).

In this study, 10 of patients and none of control had AST > 45U/L which is significant (p value 0.0006) as shown in table (6).

Table (1) relation between level of ALT in study groups :

	ALT > 50	ALT < 50	Total
Patients	14 (23.3%)	46(76.7%)	60(100%)
Control	2(3%)	63(97%)	65(100%)
Total	16(12.8%)	109(87%.2)	125 (100%)

P value: 0.0007

Table (2) Relationship between BMI and ALT level :

BMI>30	ALT > 50	ALT < 50	Total
Patients	16 (57%)	12(43%)	28(100%)
Control	0 (0%)	12(100%)	12(100%)
Total	16(40%)	24(60%)	40(100%)

P value: 0.0007

Table (3) relationship between gender and level of ALT:

Gender	ALT > 50	ALT < 50	Total
Males	5 (13.5%)	32(86.5%)	37
Females	9(39.1%)	14(60.9%)	23
Total	14(23.3%)	46(76.7%)	60

P value: 0.022

Table (4) relationship between study group and AST/ALT ratio :

	AST / ALT < 1	AST /ALT > 1	Total
Patients	53(88.3%)	7(11.7%)	60(100%)
Control	65(100%)	0 (0%)	65 (100%)
Total	118(94.4%)	7(5.6%)	125 (100%)

P value: 0.005

Table (5) relationship between age and ALT level:

	ALT > 50	ALT < 50	Total
Patients Age > 50	15(53.6%)	13(46.4%)	28(100%)
Control Age > 50	0 (0%)	15 (100%)	15 (100%)
Total	15 (35%)	28(65%)	43(100%)

P value: 0.0004

Table (6) relation between level of AST in study groups:

	AST > 45	AST < 45	Total
Patients	10 (16.7%)	50 (83.3%)	60 (100%)
Control	0(0%)	65(100%)	65 (100%)
Total	10 (8%)	115 (92%)	125 (100%)

P value: 0.0006

Discussion

This study consist of 60 patients with fatty changes on ultrasound and 65 healthy controls, we found that patients with ALT > 50 had significant relation to NASH (P value 0.001) ,this result correlates with Dixon *et al*[16] study that designed (ALT > 40 U/L) as parameter for prediction of NASH, when 105 patients enrolled in his study and found that 25% samples diagnosed with NASH.

In this study , patients with BMI > 30 had significant relation to NASH (P value 0.0007) ,this result correlate with Zein *et al*[17] study who take 177 patients and correlate with Marchesani et al study that showed that 80% of patients with NAFLD were obese[18]

The AST / ALT ratio of > 1 occur in 7 of patients out of 60 which is significant (p value 0.005), this result correlate with Palekar *et al*[12] who designed AST / ALT > 1 as variable for detection of NAFLD when 80 patients included in his study were 39 had simple steatosis and 41 had NASH).

Regarding the gender, 14 patients (5 males, & 9 females) had ALT value > 50 U/L with (P value 0.022) which is significant and this result correlated with Zein *et al*[17] study.(It show the significant of female gender).

In this study, 28 of patients had age > 50 years, of them (15) had ALT > 50 which is significant relation with NAFLD (P value 0.005), this result correlated with Zein *et al*[17] and Palekar *et al*[12] studies .

In this study, 10 of patients out of 60 had AST > 45 U/L which is statically significant (P value 0.0006), with NAFLD, this result correlate with Gholam *et al*[20] study that included 97 patients in regarding to NAFLD .(which show the significance of AST>45 U/L in patient with NAFLD.)

Conclusion

NAFL **is** not a benign condition and may lead to abnormal elevation in liver enzymes.

Recommendation

Patients with fatty liver on ultrasound need close monitoring and follow up as well as the potential targets for therapeutic intervention when specific treatments for this condition become available.

Because our study concerned on the relationship between fatty liver on

ultrasound with liver function test. We suggest further studies to show the relations between fatty liver on ultrasound with other markers as:
Lipid profile, type of diet, waist circumference and blood pressure.

References

1. Angulo P. Nonalcoholic fatty liver disease. N Engl J Med 2002;346:1221–1231.
2. Wieckowska A, Feldstein AE. Nonalcoholic fatty liver disease in the pediatric population: a review. Curr Opin Pediatr 2005;17:636–641 .
3. Jeanne M Clark, Federick L, Brancati and Mae Dhiel ,et al.Non Alcoholic Fatty Liver Disease. Gastoenterology 2002; 122. : 1649 -1669.
4. Scaffner F, Thaler H,et al. Non Alcoholic Fatty Liver Disease - A progressive liver disease. Scandinavian Journal of Gastroenterology 1986; 8: 283 – 93.
5. Cortez , Pinto H , Camilu M, Bapista,et al. NAFLD –Another feature of metabolic syndrome. Diabetes 1996; 36: 185 - 92.
6. Adams LA, Lymp JF, St Sauver J, et al. The natural history of nonalcoholic fatty liver disease: a population-based cohort study. Gastroenterology 2005;129:113–121 .
7. Matteoni CA, Younossi ZM, Gramlich T, Boparai N, Liu YC, McCullough AJ. Nonalcoholic fatty liver disease: a spectrum of clinical and pathological severity. Gastroenterology 1999;116:1413–1419 .
8. Ekstedt M, Franzen LE, Mathiesen UL, et al. Long-term follow-up of patients with NAFLD and elevated liver enzymes. Hepatology 2006;44:865–873 .
9. Dam-Larsen S, Franzmann M, Andersen IB, et al. Long term prognosis of fatty liver: risk of chronic liver disease and death. Gut 2004;53:750–755 .
10. Zelber –Sagi, S Nitzan, Kauski D, Halpern ,et al. Prevalennce of NAFLD in population based study and its association with anthropometric and biochemical measures. Liver Int 2006; 7: 856 –863 .
11. Pratt DS, Kaplan MM,et al.Evaluation of abnormal liver enzymes results in asymptomatic patients. New England Journal of Medicine 2000; 342: 1266-1271.
12. Palekar NA, Naus R, Larson SP, Ward J, Harrison SA.Clinical model for distinguishing nonalcoholic steatohepatitisfrom simple steatosis in patientswith nonalcoholic fatty liver disease. Liver Int 2006;26:151–156.
13. Joseph A .E , Saverymuttu S H , Al-Sams , Cook M G ,et al.Comparison of liver histology with USG in assessing diffuse parenchymal liver disease. Radiology 1991;43:26-31.
14. Wieckowska A, McCullough AJ, Feldstein AE. Noninvasive diagnosis and monitoring of nonalcoholic steatohepatitis: present and future. Hepatology 2007;46(2):582–589 .
15. Ramesh S, Sanyal AJ. Hepatitis C and nonalcoholic fatty liver disease. Semin Liver Dis 2004;24:399–413 .
16. Younossi ZM, Diehl AM, Ong J P, et al. Non Alcoholic Fatty Liver Disease- an agenda for clinical research. Hepatology 2002; 35: 746-742.
204 © JAPI MARCH 2009 VOL. 57 .
17. Dixon JB, Bhathal PS, O'Brien PE. Nonalcoholic fatty liver disease: predictors of nonalcoholic steatohepatitis and liver fibrosis in the severely obese. Gastroenterology 2001;121:91–100.
18. Zein CO, Edmison J, Schluchter M, Feldstein AE, ZeinNN, McCullough AJA. NASH predictive index (NPI) foruse in patients with nonalcoholic fatty liver disease. Hepatology 2007;46:747A.
19. Marchesini, Brizi M, Bianchi et al. NAFLD - A feature of metabolic

syndrome. Diabetes 2001; 50: 1844 - 1850.
20. Gholam PM, Flancbaum L, Machan JT, Charney DA, Kotler DP. Nonalcoholic fatty liver disease in severely obese subjects. Am J Gastroenterol 2007;102:399–408 .