New Diagnostic Score for Hepatic Steatosis

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Abstract: Background: Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease in Western countries that is predicted to become also the most frequent indication for liver transplantation by 2030. Over the last decade, it has been shown that the clinical burden of NAFLD is not only confined to liver-related morbidity and mortality, but there is now growing evidence that NAFLD is a multisystem disease, affecting extra-hepatic organs and regulatory pathways. For example, NAFLD increases risk of type 2 diabetes mellitus (T2DM), cardiovascular (CVD) and cardiac diseases, and chronic kidney disease (CKD). **Aim of the work:** This work is aimed to correlate between fatty liver index, high sensitive CRP, lipid profile, and anthropometric measures as a new diagnostic score for hepatic steatosis. **Subjects and methods:** This study included 50 patients with hepatic steatosis and 20 normal persons as a control group, all patients and controls were subjected to: **1**-full medical history, thorough clinical examination, assessment of BMI, measurement of waist hip ratio.**2**-Laboratory assessment of: I- lipid profile, high sensitivity CRP. II-AST, ALT, alkaline phosphatase, HCV –RNA by PCR, HBsAg. III Calculation of fatty liver index. **3**-Abdominal sonar for diagnosis of fatty liver. **Result**: There is statistically significant different between study groups with high mean in case group as regards to anthropometric measures (WC- hip circumference - W/H ratio). There is positive correlation between fatty liver index and lipid profile (TG, TG and LDL), and there is a negative correlation between fatty liver index and HDL. There is non significant correlation between fatty liver index and CRP. **Conclusion**: Fatty Liver Index is a simple and accurate measure for diagnosis of hepatic steatosis.

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1-Introduction

Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease in Western countries that is predicted to become also the most frequent indication for liver transplantation by 2030. Over the last decade, it has been shown that the clinical burden of NAFLD is not only confined to liver-related morbidity and mortality, but there is now growing evidence that NAFLD is a multisystem disease, affecting extra-hepatic organs and regulatory pathways. For example, NAFLD increases risk of type 2 diabetes mellitus (T2DM), cardiovascular (CVD) and cardiac diseases, and chronic kidney disease (CKD [1]. Biological markers have been studied for evaluation of steatosis, the presence of necroinflammation and the development of fibrosis to avoid performing liver biopsy. The most important parameter to be identified through non-invasive methods is inflammation, as it plays a central role in NAFLD progression. The C-reactive protein (CRP) is an acute-phase reactant produced by the liver and has an increased serum concentration in a variety of inflammatory conditions. The assessment of plasma levels of CRP proved to be useful in differentiating between simple steatosis and NASH. Moreover, it seems that high concentrations of high-sensitivity CRP are associated with extensive liver fibrosis in NASH [2].

Aim of the work

This work aims to correlate between fatty liver index, high sensitive CRP, lipid profile, and anthropometric measures as a new diagnostic score for hepatic steatosis. So we added new parameters to fatty liver index to increase its sensitivity.

2-Subjects and method

This study include 50male and female patients with hepatic steatosis and 20 normal persons as a control group, all study groups enrolled in the study were subjected to: 1- Full history taking.2-Thorough examination. 3-Anthropometric measurement in the form of weight, height, waist circumference, hip circumference, assessment of BMI, measurement of waist hip ratio. 4-Laboratory investigations in the form of blood triglycerides, total cholesterol, HDL-cholesterol and LDL- cholesterol, blood aspartate aminotransferase (AST), alanineaminotransferase (ALT), gamma-glutamyl transpeptidase (GGT),

alkaline phosphatase, highly sensitive CRP, hepatitis B virus surface antigen and hepatitis Cvirus RNA.

Statistical analysis:

Data was collected and coded to facilitate data calculation and double entered into Microsoft Access and data analysis was performed using SPSS software version 18 under windows 7.

Simple descriptive analysis in the form of numbers and percentages for qualitative data, and arithmetic means as central tendency measurement, standard deviations as measure of dispersion for quantitative parametric data, and inferential statistic test:

3. Result:

This work was conducted in Fayum University hospital. 50 patients were embeded, 38 female, 12 males and 20 persons as a control group, 11 females and 9 males. Patients were chosen according to ultrasound. Results showed:

Table (1): Comparisons of lipid profile in different study groups.

Variables	Case (n=50)		Control (n=20)		p-value	Sig.
	Mean	SD	Mean	SD		
Lipid profile						
Cholesterol	**186.2**	49.7	166.1	23.4	**0.03**	**S**
Triglycerides	**187.5**	87.1	142.3	30.6	**0.03**	**S**
HDL	**34.9**	6.4	38.1	4.5	**0.03**	**S**
LDL	**180.1**	71	146.7	22.8	**0.04**	**S**

Table (1) illustrates that there is statistically significance difference (p-value <0.05) between study groups as regards to triglycerides, cholesterol and LDL level with **high** mean among cases. On the other hand there is statistically significance difference (p-value <0.05) between study groups as regards to HDL level with **low** mean among cases.

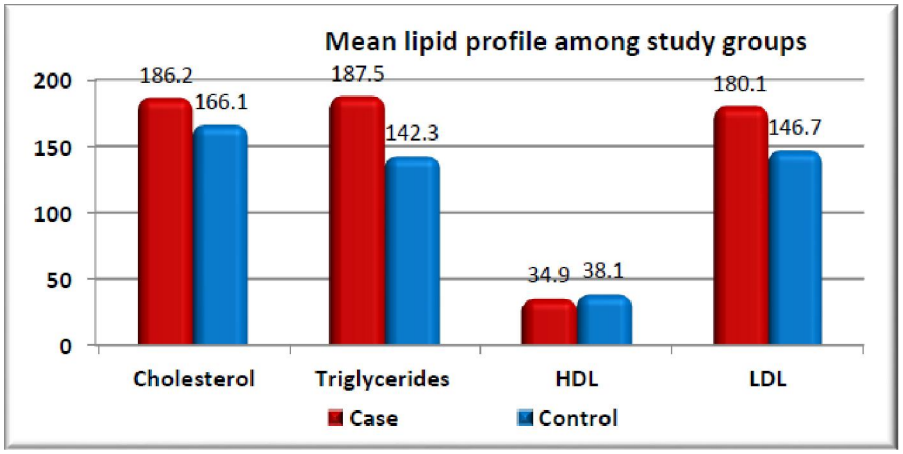

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Figure (1) illustrates that there is significance difference (p-value <0.05) between study groups as regards to triglycerides, cholesterol and LDL level with high mean among cases. On the other hand there is statistically significance difference (p-value <0.05) between study groups as regards to HDL level with low mean among case.

Table (2): Comparisons of CRP and Fatty Liver Index in different study groups.

Variables	Case (n=50)		Control (n=20)		p-value	Sig.
	Mean	SD	Mean	SD		
CRP	**7.6**	4.5	6	0	**0.1**	**NS**
Fatty liver index	**74.7**	20.2	30.5	6.9	**<0.001**	**HS**

Table (2) illustrates that there is statistically significance difference (p-value <0.001) between study groups as regards to fatty liver index with high mean among cases.

On the other hand there is no statistically significance difference (p-value 0.1) between study groups as regards to CRP.

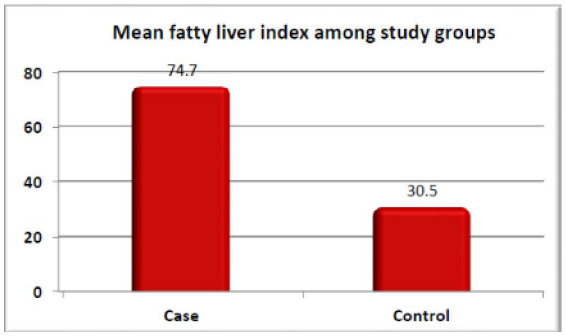

Figure (2)

Figure (2) illustrates that there is statistically significance difference (p-value <0.001) between study groups as regards fatty liver index with **high** mean among cases.

Table (3): Correlation between Fatty liver index with anthropometric measurements among cases.

Variables	Fatty liver index		
	R	p-value	Sig.
Weight (kg)	**0.56**	**<0.001**	**HS**
Waist circumference (cm)	**0.78**	**<0.001**	**HS**
Hip circumference (cm)	**0.74**	**<0.001**	**HS**
Waist /hip ratio	**0.53**	**<0.001**	**HS**
BMI	**0.58**	**<0.001**	**HS**

Table (3) illustrates that there is statistically significance **positive** correlation (p-value 0.001) between Fatty liver index and anthropometric measurements (WT, WC, HC, W/H ratio, and BMI) which indicates increase anthropometric measurements had positive impact on increasing fatty liver index.

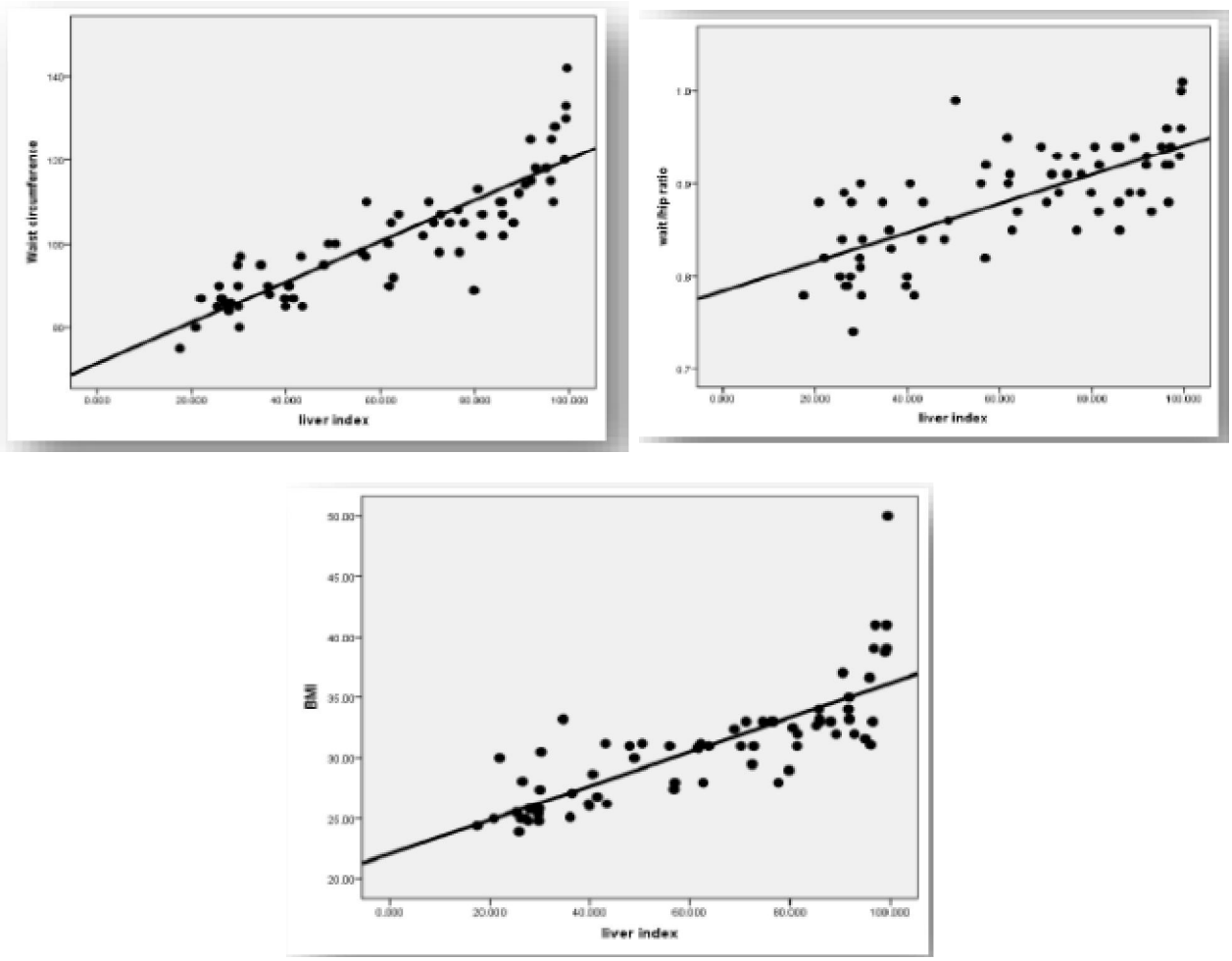

Figure (3): Correlation between Fatty liver index and anthropometric measurements among cases.

Figure (3) illustrates that there is statistically significance **positive** correlation (p-value 0.001) between Fatty liver index and anthropometric measurements (WC, W/H ratio, and BMI) which indicate increase anthropometric measurements had positive impact on increasing fatty liver index**.**

Table (4): Correlation between Fatty liver index and lipid profile among cases.

variables	Fatty liver index		
	R	p-value	Sig.
Lipid profile			
Cholesterol	**0.38**	**0.006**	**HS**
Triglycerides	**0.64**	**<0.001**	**HS**
HDL	**-0.19**	**0.04**	**S**
LDL	**0.56**	**<0.001**	**HS**

Table (4) illustrates that there is statistically significance **positive** correlation (p-value 0.006, 0.001, 0.001) between Fatty liver index and lipid profile (cholesterol, triglycerides and LDL level), which indicates increase lipid profile had positive impact on increasing fatty liver index, at the same time there is significant negative correlation (p-value 0.04) between fatty liver and HDLwhich indicate that decrease HDL had positive impact on fatty liver.

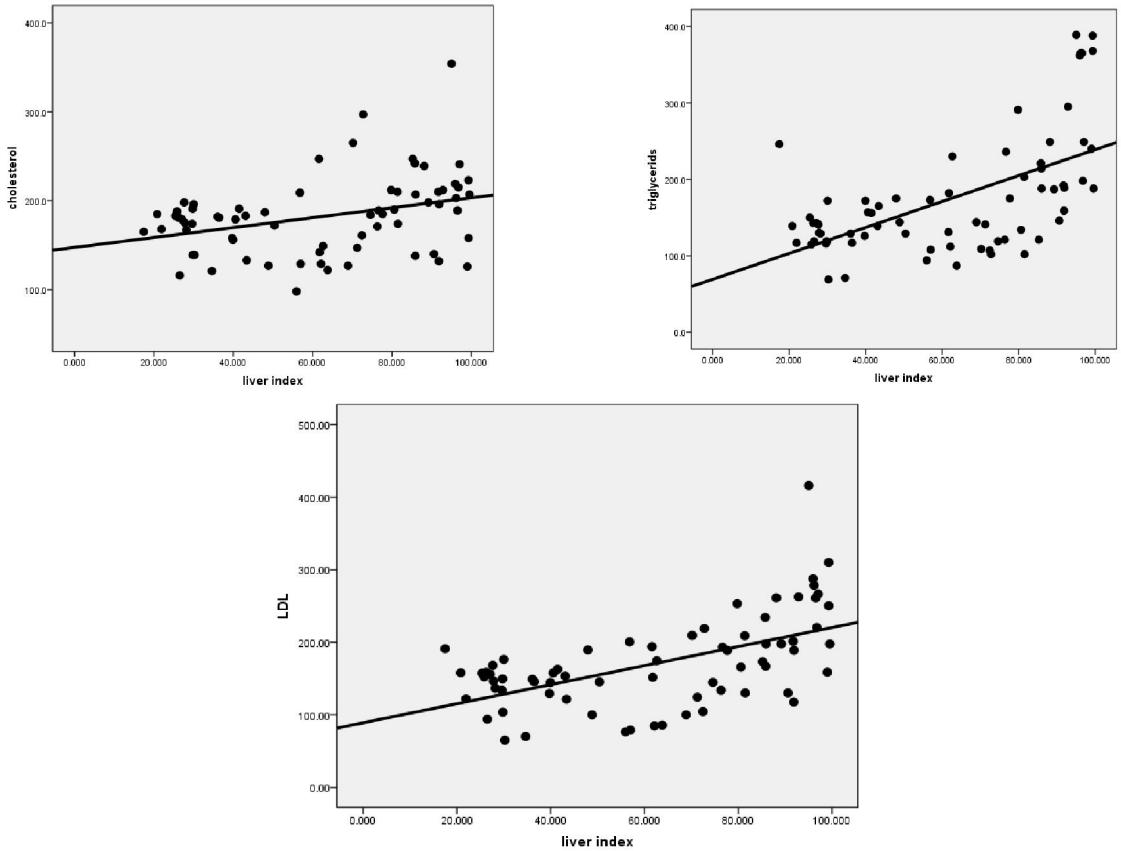

Figure (4): Correlation between Fatty liver index and lipid profile among cases.

Figure (4) illustrates that there is statistically significance **positive** correlation (p-value 0.006, 0.001, 0.001) between Fatty liver index and lipid profile (cholesterol, triglycerides, and LDL level), which indicates increase lipid profile had positive impact on increasing fatty liver index, and shows statistically **negative** correlation (p-value 0.04) between FLI and HDL level which indicates that decrease HDL level has a positive impact on increasing fatty liver.

4. Discussion

Hepatosteatosis, is triglyceride accumulation within the cytoplasm of hepatocytes [3], that is exceeding 5%–10% of its weight [4].

When hepatosteatosis is present in the absence of excessive alcohol consumption, it is termed non-alcoholic fatty liver disease, or NAFLD, which is considered to be the hepatic manifestation of the metabolic syndrome [5].

Our study showed statistically significance difference (p value <0.05) between study groups as regard to triglyceride with higher mean among cases. This work result is similar to the study of **Cheol and his colleagues in 2007**which revealed that triglyceride accumulation in hepatocytes was considered to be the major pathogenic trigger in the development of steatosis [6].

In the present study there is statistically significance difference (p value <0.05) as regards to cholesterol level between cases and control groups with higher mean among cases, this is similar to the study of **Matsuzawa and his colleagues in 2007** which revealed that excess cholesterol intake contributes to the development of steatosis even in the absence of obesity [7].

This work showed statistically positive correlation (p value <0.05) between FLI and cholesterol level. This result is similar to the work result of **Simonen and his colleagues in 2011** which showed that cholesterol synthesis in steatosis patients is increased in contrast to diminished absorption of cholesterol, and also agree with the study result of Gylling and his colleagues 2010 which showed that Insulin resistance is associated with increased cholesterol synthesis [8].

The current study showed non significance correlation (p value >0.05) between CRP and fatty liver index [9], this result is similar to the study of Yoneda and his colleagues in 2007 in which There was no increase in CRP in steatosis patients in combared with NASH patients [10].

Steatosis defined as pure fatty liver or simple steatosis and it is characterized by simple fat infiltration with minimal inflammation, and minimal inflammatory markers, only one-third of patients in the spectrum of steatosis develop NASH, which is histologically defined by the presence of lobular inflammation, portal inflammation, cellular ballooning and fibrosis **[11].**

The assessment of plasma levels of CRP proved to be useful in differentiating between simple steatosis and NASH. Moreover, it seems that high concentrations of CRP are associated with extensive liver fibrosis in NASH **[12]**.

Our study showed statistically positive correlation (p value <0.05) between Fatty Liver Index and anthropometric measurements. This result is similar to the results of **W.S.Su in 2010** which revealed that increased WHR was the risk factor that could increase the risk for hepatic steatosis [13].

A Study of **Aekplakorn and his colleagues in 2007** revealed that high BMI and increased WHR have value in the prediction of steatosis, and are closely associated with the occurrence of steatosis [14]**.**

The study of **Eguchi and his colleagues in 2006** revealed that hepatic steatosis is strongly associated with obesity but body fat distribution appears to play a more important role in the pathogenesis of steatosis. Excess intraabdominal fat in particular may be a key determinant in the pathogenesis of steatosis, because of its strong association with insulin resistance and possibly as a source of FFAs [15]**.**

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