

## Fibroscan for Assessment of Non Alcoholic Fatty Liver Disease in Type 2 Diabetic Patients in Tanta University Hospital

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### Abstract

**Background:** Fibroscan has recently been investigated as a new technique in the assessment of fibrosis of the liver in many diseases.

**Aim of Study:** Was to evaluate the role of fibroscan in assessment of liver steatosis and fibrosis in patients with type 2 diabetes and NAFLD.

**Patients and Methods:** The study population consisted of 40 patients with type 2 diabetes and 20 patients with obesity as controls. Patients with type 2 diabetes were divided into two groups: Group IA patients with type 2 diabetes and obesity and group IB patients with type 2 diabetes without obesity. Correlation between steatosis and fibrosis and disease was analyzed.

**Results:** Steatosis of patients with type 2 diabetes was significantly higher compared to those of controls ( $p=0.042$ ) further more patients with type 2 diabetes and obesity had higher level of steatosis than those without obesity. Also, patients with type 2 DM and obesity had higher levels of fibrosis than controls ( $p=0.023$ ).

**Conclusion:** Diabetic patients with insulin resistance (IR) and obesity have high prevalence of NAFLD and advanced liver fibrosis and we can use fibroscan for assessment of fibrosis and steatosis in those patients.

**Key Words:** Non alcoholic fatty liver disease (NAFLD) – Diabetic – Tanta University.

### Introduction

**DIABETES** is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels [1].

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Non-alcoholic Fatty Liver Diseases (NAFLD) and its subtype, Non-Alcoholic Steatohepatitis, or (NASH), are usually seen in individuals with metabolic syndrome (MS) or its components such as obesity, type-2 diabetes (DM), dyslipidemia, and insulin resistance [2].

Type 2 diabetes is a major risk factor for NAFLD. Both feature insulin resistance as a core component of their pathophysiology. As such, up to 90% of diabetic patients in some populations also have NAFLD [3].

Key issues in the diagnosis of patients with NAFLD are the differentiation of NASH from simple steatosis and staging of fibrosis [4].

The diagnosis of NASH and staging of fibrosis are essentially based on histological examination of a tissue specimen obtained by liver biopsy. However, liver biopsy has well-known limitations (invasiveness and sampling variability) and cannot be proposed for all patients, especially given the high prevalence of NAFLD worldwide. Over the past decade, there has been a growing interest in alternative novel noninvasive strategies for the evaluation of NAFLD [5].

Transient Ultrasound Elastography (FibroScan) is an ultrasound-based technology for quantitatively assessing hepatic stiffness. It has been introduced in the last several years both in Europe and other

### Abbreviations:

NAFLD : Non alcoholic fatty liver disease.  
DM : Diabetes mellitus.  
IR : Insulin resistance.  
MS : Metabolic syndrome.  
NASH : Non alcoholic steatohepatitis.  
FBG : Fasting blood glucose.  
HDL : High density lipoprotein.  
LDL : Low density lipoprotein.

parts of the world and is consistently gaining traction [6].

### Patients and Methods

This study was conducted on 60 patients, 40 patients with type 2 DM admitted to Inpatient wards and outpatient clinic of Internal Medicine Department of Tanta University Hospital between January 2017 and June 2017 and 20 patients with obesity and without diabetes mellitus who underwent routine physical examination in the same hospital during the same period were enrolled as the control group.

#### Inclusion criteria:

Patients aged >18 years with type 2 diabetes and suffer from the disease for more than 10 years.

Waist circumference more than 88cm for women and more than 102cm for men.

#### Exclusion criteria:

- Active malignancy.
- Positive hepatitis B surface Antigen or antibody against hepatitis C virus.
- Secondary causes of fatty liver (e.g: Consumption of amiodarone and tamoxifen).
- Alcohol consumption.

#### Consent:

An informed written consent was taken from every patient included in this study.

#### All patients in the study were subjected to:

- A- Full history taking regarding age, sex and any other disease. History of diet was also taken from patients.
- B- Full clinical examination particularly for presence of acanthosis nigricans, psychological disturbances, neuropathy and hypertension.
- C- Laboratory investigations including:
  - Fasting blood glucose (FBG) and 2 hours post prandial blood glucose.
  - Complete lipid profile: Serum triglycerides, serum total cholesterol. High density lipoprotein (HDL) and low density lipoprotein (LDL).
  - Liver function tests.
  - Kidney function tests.
  - Full blood count: (CBC), C-Reactive protein (CRP), Erythrocyte sedimentation Rate (ESR).
- D- Radiological investigations:
  - Pelviabdominal ultrasonography.
  - Fibroscan.

According to clinical examination and investigations the patients were classified into two groups:

- Group I: 40 patients with type 2 diabetes mellitus (for more than 10 years), who were subdivided into:
  - Group IA: 20 diabetic patients with obesity with BMI >30kg/m<sup>2</sup>.
  - Group IB: 20 diabetic patients without obesity.
- Group II: 20 persons with obesity and without diabetes mellitus as a control group.

#### Statistical analysis:

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using number and percent. The Kolmogorov-Smirnov test was used to verify the normality of distribution Quantitative data were described using range (minimum and maximum), mean, standard deviation and median. Significance of the obtained results was judged at the 5% level

*Outcome parameters:* The presence or absence of Steatosis and Fibrosis in the studied patients and their grades.

## Results

### 1- Demographic data:

Study population were compared according to age and sex (Table 1) with no statistically significant difference as *p*-value was 0.053 and 0.233 (>0.05) according to age and sex respectively.

### 2- Duration of DM:

As for the duration of diabetes: In group IA the duration range was 10-25 years, while in group IB the duration range was 10-28 years.

There was no significant difference between group IA and group IB as regard the duration of diabetes mellitus (*p*=0.765) as shown in Table (2).

### 3- Body measures:

- Regarding to body weight:

There was a significant increase in B.W in group IA in comparison to group IB&II (*p*<0.001) as shown in Table (3).

- Regarding to BMI:

There was a significant increase in BMI in group IA in comparison to group IB&II (*p*<0.001) as shown in Table (3).

- Regarding to waist circumference:

There was a significant increase in W.C in group II in comparison to group IA&IB ( $p<0.001$ ) as shown in Table (3).

4- Clinical data:

As regard total cholesterol:

There was a significant increase in total cholesterol in group II in comparison to groups IA and IB ( $p<0.001$ ) as shown in Table (4).

As regard HDL:

There was a significant increase in HDL in groups IA and IB in comparison to group II ( $p<0.001$ ) as shown in Table (4).

As regard LDL:

There was a significant increase in LDL in group II in comparison to groups IA & IB ( $p<0.001$ ) as shown in Table (4).

As regard serum triglycerides:

There was a significant increase in T.G in group IA in comparison to groups IB & II ( $p=0.014$ ) as shown in Table (4).

As regard FBG:

There was a significant increase in FBG in group IA in comparison to group IB & II ( $p<0.001$ ) as shown in Table (5).

As regard ALT:

There was a significant increase in ALT in group IA & IB in comparison to group II ( $p=0.027$ ) as shown in Table (6).

Table (1): Comparison between groups according to demographic data.

	Group IA (n=20)		Group IB (n=20)		Group II (n=20)		Test of Sig.	p
	No.	%	No.	%	No.	%		
<b>Sex:</b>								
Male	7	35.0	11	55.0	6	30.0	$\chi^2 =$	0.233
Female	13	65.0	9	45.0	14	70.0	2.917	
<b>Age (years):</b>								
Min. - Max.	40.0-65.0		38.0-65.0		38.0-60.0		F =	0.053
Mean ± SD.	52.30±6.29		47.70±8.30		47.10±6.93		3.102	
Median	52.0		45.50		45.50			

Table (2): Comparison between group IA & group IB according to duration of DM.

	Group IA (n=20)	Group IB (n=20)	U	p
<b>Duration (years):</b>				
Min. - Max.	10.0-25.0	10.0-28.0		
Mean ± SD.	15.10±4.68	14.75±5.16	189.00	0.765
Median	14.0	13.0		

Table (3): Comparison between the three studied groups according to measures.

Measures	Group IA (n=20)	Group IB (n=20)	Group II (n=20)	F	p
<b>Weight (kg):</b>					
Min. - Max.	81.0-174.0	67.0-86.0	82.0-150.0	17.701 *	<0.001*
Mean ± SD.	107.10±23.78	76.50±5.80	104.35±19.30		
Median	103.50	79.0	101.0		
Sig. bet. grps.	$p_1<0.001$ *, $p_2=0.631$ , $p_3<0.001$ *				
<b>BMI (kg/m<sup>2</sup>):</b>					
Min. - Max.	32.56-53.70	26.50-29.40	31.20-55.30	28.085*	<0.001*
Mean ± SD.	38.88±6.07	28.02±0.84	37.65±6.17		
Median	37.64	28.10	36.73		
Sig. bet. grps.	$p_1<0.001$ *, $p_2=0.440$ , $p_3<0.001$ *				
<b>Waist circumference (cm):</b>					
Min. - Max.	99.0-152.0	73.0-100.0	93.0-158.0	37.208*	<0.001*
Mean ± SD.	118.05±15.60	87.05±9.02	120.90±15.61		
Median	112.50	84.0	118.50		
Sig. bet. grps.	$p_1<0.001$ *, $p_2=0.515$ , $p_3<0.001$ *				

Table (4): Comparison between the three studied groups according to lipid profile.

Lipid profile	Group IA (n=20)	Group IB (n=20)	Group II (n=20)	Test of Sig.	p
<i>Total cholesterol (mg/dl):</i>					
Min. - Max.	103.0-274.0	125.0-178.0	70.0-300.0	F =	<0.001*
Mean ± SD.	201.75±41.02	149.15±14.31	218.75±51.95	17.227*	
Median	199.50	152.50	220.0		
Sig. bet. grps.	<i>p</i> 1<0.001*, <i>p</i> 2=0.174, <i>p</i> 3<0.001*				
<i>HDL (mg/dl):</i>					
Min. - Max.	40.0-60.0	39.0-58.0	29.0-56.0	F =	0.006*
Mean ± SD.	47.55±5.98	44.58±5.75	41.25±6.05	5.655*	
Median	47.50	42.0	40.0		
Sig. bet. grps.	<i>p</i> 1=0.119, <i>p</i> 2=0.001*, <i>p</i> 3=0.081				
<i>LDL (mg/dl):</i>					
Min. - Max.	45.0-190.0	70.0-115.0	82.0-163.0	F =	<0.001*
Mean ± SD.	133.50±39.04	90.09±13.01	133.35±22.41	17.106*	
Median	132.0	92.50	139.50		
Sig. bet. grps.	<i>p</i> 1<0.001*, <i>p</i> 2=0.989, <i>p</i> 3<0.001*				
<i>Triglycerides "TG" (mg/dl):</i>					
Min. - Max.	75.0-267.0	82.0-189.0	76.0-346.0	H =	0.014*
Mean ± SD.	149.68±51.68	110.74±30.25	150.40±65.74	8.520*	
Median	147.50	98.0	142.50		
Sig. bet. grps.	<i>p</i> 1=0.008*, <i>p</i> 2=0.751, <i>p</i> 3=0.019*				

Table (5): Comparison between the three studied groups according to FBG.

FBG (mg/dl)	Group IA (n=20)	Group IB (n=20)	Group II (n=20)	H	p
Min. - Max.	135.0-300.0	140.0-190.0	70.0-101.0	40.568**	<0.001*
Mean ± SD.	188.40±49.39	158.75±14.60	81.80±9.02		
Median	177.0	160.0	80.0		
Sig. bet. grps.	<i>p</i> 1=0.285, <i>p</i> 2<0.001*, <i>p</i> 3<0.001*				

Table (6): Comparison between the three studied groups according to ALT.

ALT (mg/dl)	Group IA (n=20)	Group IB (n=20)	Group II (n=20)	H	p
Min. - Max.	15.0-104.0	28.0-95.0	11.0-116.0	7.256	0.027*
Mean ± SD.	49.95±22.51	63.58±21.61	43.63±29.17		
Median	49.50	55.0	40.0		
Sig. bet. grps.	<i>p</i> 1=0.119, <i>p</i> 2=0.261, <i>p</i> 3=0.007*				

As regards to steatosis there was a significant difference between the groups ( $p=0.042$ ) as shown in Table (7):

Patients in group IA there had the highest measures with mean of ( $322.35 \pm 64.36$ ). We found that among the 20 patients of the group, there were 13 patients S3, 3 patients S2, 2 patients S1 and only 2 patients S0.

In group IB we found 7 patients S3, 5 patients were S2, 8 patients S1 and no patients were S0 with mean of ( $285.05 \pm 57.96$ ).

In group II there were 6 patients S3, 3 patients S2, 2 patients S1 and 9 patients S0 with mean of ( $269.05 \pm 76.71$ ).

(N.B: S0: No steatosis, S1 : Mild steatosis, S2: Moderate steatosis, S3: Marked steatosis).

According to Table (8) there was a significant difference as regard to fibrosis ( $p=0.023$ ) as following:

- In group IA, highest levels of fibrosis were found with mean of ( $10.23 \pm 6.96$ ). Among the 20 patients of the group there were 5 patients F4, 4 patients F3, 1 patient F2-F3, 3 patients F2, 5 patients F1 and 2 patients F0.
- In group IB we found that no patients were F4, 5 patients F3, 2 patients F2-F3, 1 patient F2, no patients F1 and 12 patients F0 with mean of ( $6.56 \pm 2.13$ ).
- In group II there was only 1 patient F4, 4 patients F3, 2 patients F2-F3, 2 patients F2, 3 patients F1 and 8 patients F0 with mean of ( $7.01 \pm 3.66$ ).

(N.B: F0: No fibrosis, F1: Mild fibrosis, F2: Moderate fibrosis, F3: Marked fibrosis, F4: Cirrhosis).

Table (7): Comparison between the three studied groups according to steatosis.

	Group IA (n=20)		Group IB (n=20)		Group II (n=20)		Test of Sig.	p
	No.	%	No.	%	No.	%		
<i>Steatosis:</i>							$\chi^2 = 19.734^*$	$MC_p = 0.001^*$
S0	2	10.0	0	0.0	9	45.0		
S1	2	10.0	8	40.0	2	10.0		
S2	3	15.0	5	25.0	3	15.0		
S3	13	65.0	7	35.0	6	30.0	F = 3.353*	0.042*
Min. - Max.	202.0-400.0		228.0-375.0		124.0-400.0			
Mean $\pm$ SD.	322.35 $\pm$ 64.36		285.05 $\pm$ 57.96		269.05 $\pm$ 76.71			
Median	341.50		272.0		244.0			
Sig. bet. grps.	p1=0.083, p2=0.014*, p3=0.452							

Table (8): Comparison between the three studied groups according to fibrosis.

	Group IA (n=20)		Group IB (n=20)		Group II (n=20)		Test of Sig.	p
	No.	%	No.	%	No.	%		
<i>Fibrosis</i>							$\chi^2 = 19.860^*$	$MC_p = 0.012^*$
F0	2	10.0	12	60.0	8	40.0		
F1	5	25.0	0	0.0	3	15.0		
F2	3	15.0	1	5.0	2	10.0		
F3	4	20.0	5	25.0	4	20.0		
F2 - F3	1	5.0	2	10.0	2	10.0		
F4	5	25.0	0	0.0	1	5.0		
Min. - Max.	4.50-36.30		4.20-10.0		3.0-18.40		H = 7.540*	0.023*
Mean $\pm$ SD.	10.23 $\pm$ 6.96		6.56 $\pm$ 2.13		7.01 $\pm$ 3.66			
Median	8.10		5.20		6.0			
Sig. bet. grps.	p1=0.016*, p2=0.019*, p3=0.942							

Table (9): Correlation between steatosis and different parameters in group IA & IB.

	Steatosis			
	Group IA		Group IB	
	r	p	r	p
Triglycerides "TG"	0.533*	0.016*	-0.122	0.607
Total cholesterol	0.703*	0.001*	0.305	0.190
HDL	0.265	0.260	0.377	0.101
LDL	0.187	0.430	-0.152	0.522
Duration (years)	-0.003	0.990	0.051	0.831
BMI (kg/m <sup>2</sup> )	0.469*	0.03*	-0.039	0.869
FBG	0.507*	0.02*	0.081	0.734

According to Table (9) and Fig. (1) there was a significant correlation between steatosis and triglycerides (TG) in group IA ( $p=0.016$ ).

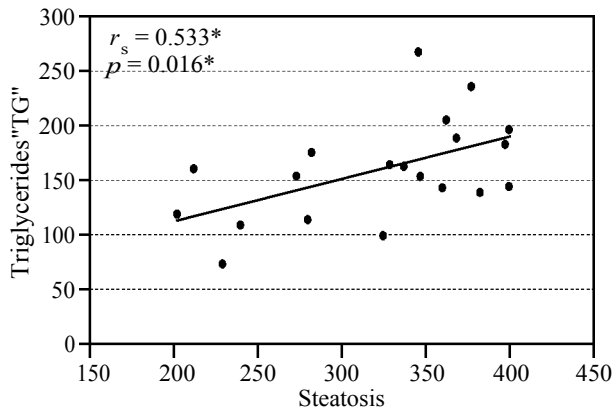


Fig. (1): Correlation between steatosis and Triglycerides "TG" in Group IA.

There was also a significant correlation between steatosis and total cholesterol in group IA ( $p=0.001$ ) according to Table (9) and Fig. (2).

Also according to Table (9) and Fig. (3) there was a significant correlation between steatosis and BMI in group IA ( $p=0.037$ ).

There was a significant correlation between steatosis and FBG ( $p=0.023$ ) in group IA as in Table (9) and Fig. (4).

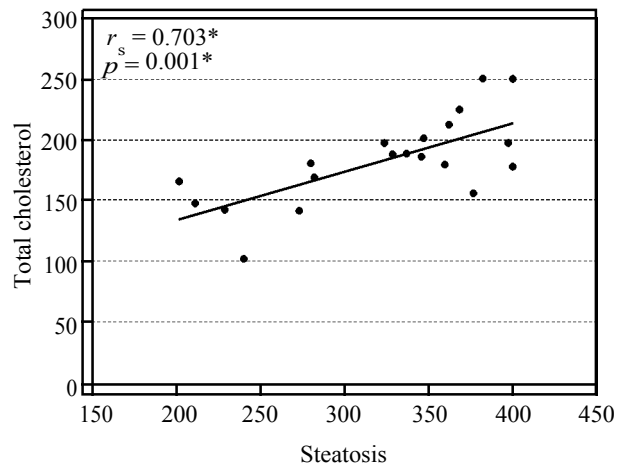


Fig. (2): Correlation between steatosis and Total cholesterol in Group IA.

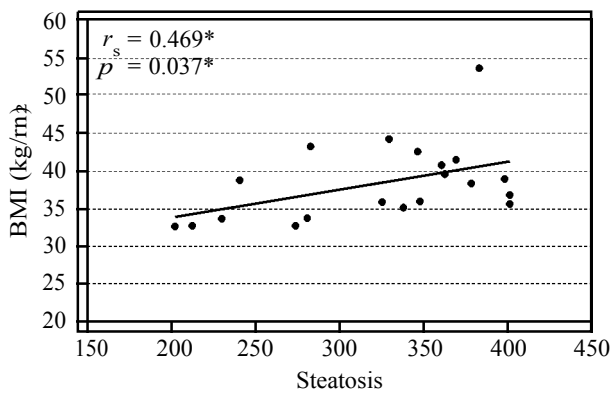


Fig. (3): Correlation between steatosis and BMI (kg/ml<sup>2</sup>) in Group IA.

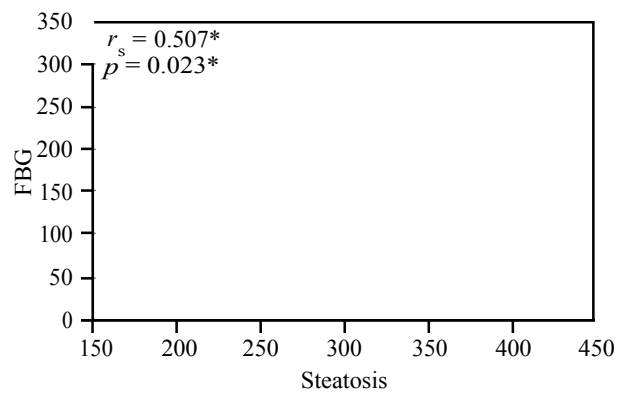


Fig. (4): Correlation between steatosis and FBG in Group IA.

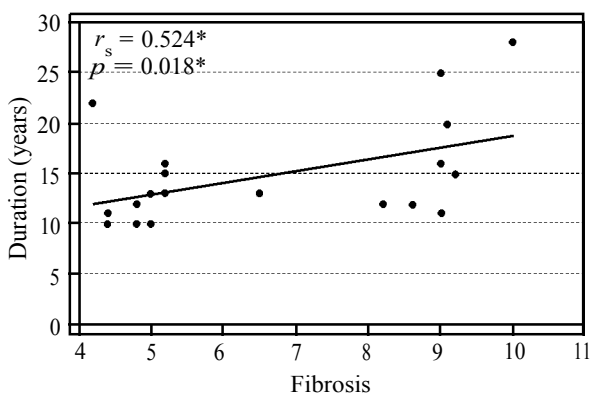


Fig. (5): Correlation between Fibrosis and duration (years) in group IB.

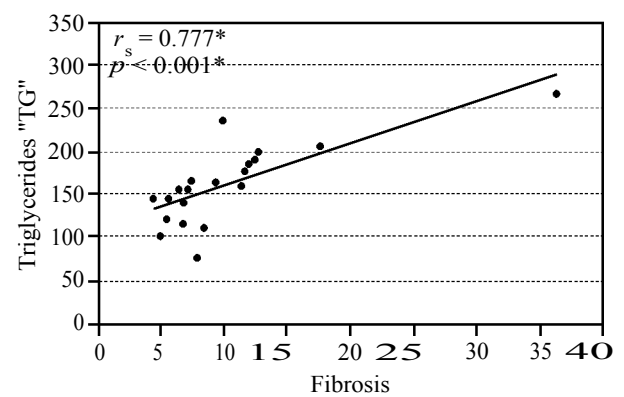


Fig. (6): Correlation between Fibrosis and Triglycerides "TG" in Group IA.

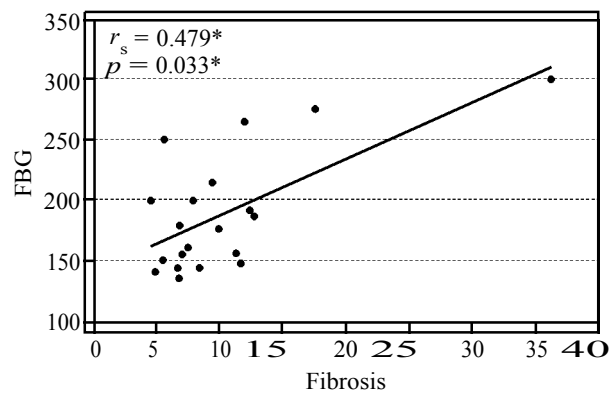


Fig. (7): Correlation between Fibrosis and FBG in Group IA.

Table (10): Correlation between Fibrosis and different parameters in group IA & IB.

	Fibrosis			
	Group IA		Group IB	
	$r_s$	$p$	$r_s$	$p$
Triglycerides "TG"	0.777*	<0.001*	0.017	0.942
Total cholesterol	-0.042	0.860	-0.030	0.899
HDL	0.124	0.603	-0.237	0.314
LDL	-0.024	0.920	-0.146	0.540
Duration (years)	0.317	0.173	0.524*	0.018*
BMI (kg/m <sup>2</sup> )	0.395	0.084	-0.342	0.140
FBG	0.479*	0.033*	0.257	0.273

According to Table (10) and Fig. (5) there was a significant correlation between fibrosis and duration in group IB ( $p=0.018$ ).

There was a significant correlation between fibrosis and triglycerides in group IA ( $p<0.001$ ) as shown in Table (10) and Fig. (6).

According to Table (10) and Fig. (7) there was a significant correlation between fibrosis and FBG ( $p=0.033$ ) in group IA.

### Discussion

Nonalcoholic fatty liver disease is the most frequent form of chronic liver disease and one of the leading causes of end-stage liver disease in Western countries. It encompasses a broad spectrum of chronic liver conditions ranging from simple hepatic steatosis (nonalcoholic fatty liver) to non-alcoholic steato-hepatitis, with increased risk of progression to cirrhosis and hepatic cancer [7]. Moreover, excess mortality from chronic liver

disease in type 2 diabetes ranks with excess risk of death from cardiovascular complications [8], suggesting that end-stage liver disease should be added to the list of known complications of diabetes [9].

Knowledge on the epidemiology of NAFLD is incomplete because of the limitations of various diagnostic modalities. Liver biopsy is considered the reference standard, but is impractical to apply to a large study population. Abdominal ultrasonography is easily accessible but is only qualitative, poor in detecting minor steatosis and suffers from intra-observer and inter-observer variability. Furthermore, it cannot assess disease severity [10]. Transient elastography is a non-invasive test of liver fibrosis that is quick and easy to perform and has a high degree of patient acceptance [11]. It has high accuracy and reproducibility when used to detect advanced fibrosis and cirrhosis. In addition, the latest model measures a novel physical parameter called the controlled attenuation parameter (CAP). Since fat affects ultrasound propagation, CAP measurement has been shown to be accurate in estimating the amount of liver fat [12]. Using this non-invasive technique, it is now possible to measure liver fat and fibrosis in a large number of patients. In this study, we aimed to test the strategy of NAFLD and fibrosis screening in patients with type 2 diabetes & obesity Kowk et al., [10].

In this study we assessed fibrosis in patients with type 2 diabetes by using fibroscan in 40 Egyptian patients with type 2 diabetes divided into two subgroups; group IA: 20 diabetic patients with obesity, group IB: 20 diabetic patients with no obesity who were investigated and compared to fibroscan in 20 non diabetic patients with obesity only (group II).

In our study, there were 24 male patients who constituted about 40% of studied groups and 36 females who constituted about 60% of studied groups.

On the other hand Giorda et al., [9] about 56.6% of the studied groups were males. While Dobrin et al., [13] investigated 62 patient 20 females & 42 males.

In our study there was significant difference between studied groups according to waist circumference. It was significantly higher in groups II & IA in comparison to group IB. This agreed with Kowk et al., [10].

In our study the mean of waist circumference was about  $118.05 \pm 15.6$ cm for diabetic obese pa-

tients &  $87.05 \pm 9.02$ cm in diabetic non obese. While in the study performed by Dobrin et al., [13] who investigated 62 patients, 31.3% of them were diabetic, mean of waist circumference was 117.25cm.

Also in our study there was a significant increase in bodyweight in groups IA & II in comparison to group IB & this agreed with Kowk et al., [10].

Regarding the systolic blood pressure, it was significantly higher in group IA in comparison to groups IB & II. This was partially in accordance with the result of Aller et al., [14] who investigated 195 patients for NASH. They reported that the mean systolic Blood pressure was  $138 \pm 22.5$ .

As for the diastolic blood pressure, it was significantly higher in group IA in comparison to groups IB & II. This was in accordance with Kowk et al., [10] who reported that diastolic blood pressure was higher in diabetic patients with obesity.

According to ALT, in our study there was a significant increase in group IA & IB in comparison to group II, this disagreed with Kowk et al., [10] who reported that there was no significant differences according to ALT between diabetic and obese patients in his study. Kowk et al., [10] performed their study on 2466 patients, which is a large number and this may explain the difference.

This was close to the mean of ALT in Aller et al., [14] study which was  $67 \pm 41.9$ . On the other hand the mean ALT of Dorbin et al., [13] study was higher  $91 \pm 49$ .

In our study there was a significant positive correlation between fibrosis and duration of diabetes in patients without obesity & that agreed with the results of Kowk et al., [10] and Giorda et al., [9].

Our study showed a significant positive correlation between fibrosis & Triglycerides in patients with obesity. This was in accordance with Giroda et al., [9] who declared that there was a correlation between fibrosis & level of triglycerides in diabetic patients. Also this agrees with the results of Kowk et al., [10].

In our study BMI showed a significant positive correlation with fibrosis in diabetic patients with central obesity. This agreed with the results documented by the following authors: Fierbinteanu - Barticevici et al., [15]; Kowk et al., [10] and Giorda et al., [9].

In our study there was no significant correlation between cholesterol level in the studied groups and liver fibrosis. This agrees with Kowk et al., [10] and Mikolasevic et al., [16]. While the results of Giroda et al., [9] were opposite to ours, as they documented that there was no significant correlation between serum cholesterol and liver fibrosis.

In our study there was no significant correlation between fibrosis & LDL and this agreed with Kowk et al., [10]. On the contrary Giroda et al., [9] reported that there was significant correlation between fibrosis and LDL.

As for HDL, there was no significant correlation between it and fibrosis & this disagreed with Kowk et al., [10] and Giorda et al., [9] who found that there was a positive correlation between fibrosis and decrease HDL. In our study the mean age of the patients was about 48 years old, while the mean age of studied groups of Giorda et al., [9] was about 64 years old and this may explain the difference.

According to FBG there was a significant positive correlation with fibrosis in diabetic patients with central obesity & this agreed with Mikolascevic et al., [16] who reported that fibrosis was higher in patients with high FBG. While our results were opposite to these reported by Kowk et al., [10] who documented that there was no correlation between fibrosis and FBG in patients with type 2 diabetes.

#### Conclusion:

Steatosis was higher in diabetic patients than patients with obesity only (controls). Fibrosis was higher in diabetic patients with obesity than controls. So diabetes could be a leading etiological factor for development of steatosis and fibrosis in diabetic obese patients. Prevention of steatosis and fibrosis in patients with diabetes and obesity may be done by good control of blood glucose level, body weight and lipid profile. More studies are needed on larger number of patients to focus on fibroscan and its relation to other methods for assessment of steatosis & fibrosis in diabetic patients. Regular follow up by fibroscan for diabetic patients may be beneficial, taking into consideration that it is a non-invasive investigation. Restoring to Normal values of steatosis & fibrosis might be a potential therapeutic target in follow up of diabetic patients.

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## إستخدام الفيبروسكان لتقييم أمراض الكبد الدهنية غير الكحولية فى مرض الإعتلال السكرى من النوع الثانى

المرض السكرى من مجموعة من الأمراض الأيضية التى تتميز بارتفاع السكر فى الدم الناتج عن خلل فى إفراز الأنسولين أو عمل الأنسولين أو كليهما ويترافق إرتفاق السكر المزمن فى الدم بالضرر على المدى الطويل بالعجز وفشل الأجهزة المختلفة وخاصة العينين والكليتين والأعصاب والقلب والأوعية الدموية.

إن أمراض الكبد الدهنية الغير كحولية وأنواعها غالباً ما تحدث فى الأشخاص الذين يعانون من السمنة أو المرض السكرى من النوع الثانى أو إختلال نسبة الدهون بالدم أو مقاومة الجسم للأنسولين.

المرض السكرى من النوع الثانى يعد هو أخطر وأهم الأسباب لأمراض الكبد الدهنية غير الكحولية يعتبر مقاومة الجسم للأنسولين هى العامل الأساسى لتطور وتسلسل حدوث المرض حيث أنه يوجد تسعون بالمئة من مرض السكرى من النوع الثانى يعانون من أمراض الكبد الدهنية غير الكحولية.

وهذه الدراسة الهدف منها هو تقدير نسبة التليف الكبدى فى هؤلاء المرضى. وقد كان تقدير نسبة التليف الكبدى فى هؤلاء المرضى يعتمد على العينة الكبدية وفحص خلايا الكبد وأنسجة. وقد كان هذا يعرض المرضى للتدخل لأخذ عينات وكان هذا لا يصلح لجميع المرضى بالرغم من كثرة إنتشار هذا المرض على مستوى العالم وفى العقود الأخيرة كانت هناك محاولات لتقدير نسبة التليف فى هؤلاء المرضى ولكن دون تدخل مع المريض.

وقد أستخدام الفيبروسكان هو نوع من أنواع الموجات الصوتية التى تستخدم لقياس نسبة التليف فى الكبد دون تدخل مع المريض فى أوروبا وأنحاء أخرى من العالم وأصبح محل إهتمام كبير.

النتائج: ارتفاع ذو قيمة إحصائية لنسبة تشبع الكبد بالدهون فى مرض الاعتلال السكرى من النوع الثانى مقارنة بهؤلاء الذين يعانون من زيادة الوزن فقط.

ارتفاع ذو قيمة إحصائية لنسبة تشبع الكبد بالدهون فى مرضى السكر من النوع الثانى وزيادة الوزن مقارنة بها فى هؤلاء الذين يعانون من مرض السكر من النوع الثانى ولكن دون زيادة فى الوزن.

ارتفاع ذو قيمة إحصائية فى نسبة التليف الكبدى فى المرضى الذين يعانون من مرض السكر من النوع الثانى وزيادة الوزن مقارنة بهؤلاء الذين يعانون من زيادة الوزن فقط.

الاستنتاج والخلاصة: من تقييم نتائج هذه الرسالة ومقارنتها بنتائج الباحثين الآخرين فى نفس المجال يمكن التوصل إلى إمكانية استخدام تقنية الفيبروسكان كدلالة لقياس نسبة التصلب الكبدى فى مرض السكر من النوع الثانى والتنبؤ بالتليف الكبدى.