ABENHA MEDICAL JOURNAL bmj
Print ISSN 1110-208X
Online ISSN 2357-0016

Original article

Role of Fatty Acid Binding Protein 1 in Patients with Non- Alcoholic Fatty Pancreatic Disease with and without Diabetes Mellitus

Reda EI-Badawy[a], Awad M. EL Abd[b], Heba M. Bedair[a], Ghadeer M. Rashad[a]

[a] Department of Hepatology, gastroenterology and infectious diseasess , Benha faculty of medicine, Benha University, Egypt. [b] Department of Medical biochemistry and Molecular biology, Benha faculty of medicine, Benha University, Egypt .

Correspondence to: Heba M. Bedair, Department of Hepatology, gastroenterology and infectious diseasess , Benha faculty of medicine, Benha University, Egypt.

Email:

drhebabedair@yahoo.com

Received: 1 May 2021

Accepted: 19 May 2021

Abstract:

Background: Non-alcoholic fatty pancreatic disease (NAFPD) encompasses a broad range of conditions ranging from pancreatic fat accumulation (fatty pancreas, pancreatic steatosis) to pancreatic inflammation (non-alcoholic steatopancreatitis) and probable pancreatic fibrosis. FABP1 is a 14-kDa protein that is involved in cytoplasmic fatty acid metabolism. Additionally, FABP1 enhances the transport, storage, and use of fatty acids and their acyl-CoA derivatives and may protect against lipotoxicity by accelerating their oxidation or incorporation into TGs and binding other cytotoxic fatty acids. **Aim**: This study aimed to assess the diagnostic role of FABP1 in patients with a non-alcoholic fatty pancreatic disease with and without Diabetes Mellitus. **Method:** This was a cross-sectional study which was carried on 88 subjects who were evaluated through complete history, clinical and biochemical assessment, and abdominal ultrasound. FABP1 was determined using ELISA kits. **Result:** The studied patients showed a mean age of 44.08±12.41 years and a BMI mean of 29.73 ±8.15 kg/m2. 72% of patients were males. FABP1 levels were significantly elevated among diabetic patients with BMI >25 (P=0.02). There was a statistically significant relation between FABP1 expression with high grades of fatty pancreas among non-diabetic subjects with BMI over 25 (P=0.013). At cut-off 0.758, the sensitivity of FABP1 in the prediction of the fatty pancreas was (68.2%), specificity (45.5%), and area under the curve was 0.618 in diabetics and non-diabetics with normal BMI. **Conclusion:** We suggest that FABP may be a good marker for the diagnosis of NAFPD.

Keywords: Non-alcoholic; fatty pancreas disease, Fatty acid-binding protein 1. **Abbreviation:** NAFPD=Non-alcoholic fatty pancreas disease, FABP1 = Fatty acid-binding protein 1, **TGs:** Triglyceridas, BMI: body mass index, ELISA: enzyme-linkedimmunosorbent assay.

Introduction:

Non-alcoholic fatty pancreatic disease (NAFPD) is a condition in which the pancreas accumulates an abnormal amount of lipids in the absence of heavy alcohol use **(1)**. Schaefer **(2)** reported this syndrome for the first time in 1926 and Ogilvie **(3)** used the name 'pancreatic lipomatosis' in 1933 to refer to excessive fat buildup in the pancreas**.**

In comparison to non-alcoholic fatty liver disease (NAFLD), the possible systemic and local implications of excessive pancreatic fat accumulation are not well understood **(4)**.

NAFPD has been observed to be prevalent in both Asian and Western countries. It was observed that 16% of the Chinese population had fatty pancreas in Taiwan **(5)**. In Indonesia, the NAFPD prevalence was 35% in the medical check-up population.

Up to our knowledge, there is no Egyptian published study till now about NAFPD and its relation to obesity or DM with its complications **(6)**. Pancreatic fat content may be involved in a variety of local pathological events, including pancreatic cancer and pancreatitis subtypes **(7)**. NAFPD may progress to chronic pancreatitis, which then progresses to pancreatic cancer, facilitating its spread. NAFPD's pathogenesis is unknown at the moment. Pancreatic fat accumulation has two mechanisms: acinar cell death followed by adipose tissue replacement, and intracellular triglyceride buildup linked with a favorable energy balance **(8).** Fatty pancreas is an emergent problem that needs new markers (to be added to the investigation other than ultrasound) to be used as a simple non-invasive biomarker to aid in diagnosis.

Fatty acid-binding proteins (FABPs) are a class of tiny, highly conserved lipid chaperone molecules with a wide variety of activities **(9)**. They have the potential to be used as biomarkers of tissue damage. FABP is detectable in serum when there is continuous damage. Each protein in this family is named after the tissue from which it was isolated, and prominent members of this group include liver fatty acid-binding protein (L-FABP), intestinal fatty acid-binding protein (I-FABP), heart fatty acid- binding protein (HFAP), and epidermal fatty acid-binding protein (E-FABP) **(10)**. FABP1 facilitates the transportation, storage, and use of fatty acids and their acyl- CoA derivatives and may act as a lipotoxicity protectant by promoting their oxidation or incorporation into TGs and binding other cytotoxic-free fatty acids **(11)**. We speculated that FABP can be a diagnostic marker for NAFPD. This study aimed to determine the role of FABP1 in patients with Non-Alcoholic Fatty Pancreatic Disease.

Patients and methods:

Study population

This was a cross-sectional study, conducted on 88 subjects who attended AL Helal Hospital in Shebin El Kom, Menoufia, during

the period from March 2020 till December 2020. The Ethical Committee of the Benha Faculty of Medicine approved the study. Informed written consent was taken from all patients after explaining the aim to them. This study population was divided as follow:

Group 1: included 22 subjects with normal BMI, Non-diabetics.

Group 2: included 22 patients with normal BMI, Diabetics.

Group 3: included 22 patients with BMI over 25, Non-diabetics.

Group 4: included 22 patients with BMI over 25, Diabetics.

Inclusions Criteria: Normal BMI and obese people with and without DM type 2. All subjects aged from 18-70 years old. Both sexes were included.

Exclusions Criteria: Patients with a history of chronic pancreatitis or previous attacks of acute pancreatitis and/or admission to the hospital.

The patients were evaluated clinically through full medical history and clinical examination as blood pressure and body mass index (BMI)**.** Laboratory investigations included complete blood count (CBC), fasting blood sugar, HbA1c%, HCV antibody & HBVs antigen, liver profile, lipid profile, and serum Insulin level, which was used for calculating insulin resistance (IR). HOMA-IR was calculated using the given mathematical equation; HOMA-IR = fasting insulin (mU/ml) x fasting plasma glucose (mmol/l)/22.5 (**12).**

Fatty Acid Binding protein1 (FABP1**)** was evaluated by the enzyme-linked immunosorbent assay (ELISA), and the deviation from the normal will be correlated with other investigations and clinical manifestations of the subjects. Abdominal Ultrasound was done for detection of fatty pancreas by expert radiologist**.** The echogenicity of the pancreas is divided into four grades **(13).**

- **Grade 0**: Similar echogenicity of the pancreas to the kidney parenchymal.
- **Grade1**: Higher echogenicity of the pancreas than in the kidney if the operator can see both in the same view in the transverse epigastric scan with a slight move to the right. If the pancreas and kidney cannot be visualized simultaneously, the radiologist compares the kidney to the liver and then the liver to the pancreas.
- **Grade 2**: A significant increase in echogenicity of the pancreas but lower than the echogenicity of retroperitoneal fat.
- **Grade 3**: the echogenicity of the pancreas is similar to or higher than the retroperitoneal fat.

NAFPD is diagnosed when the pancreas appeared as grade 1 to 3.

Statistical analysis:

Data were collected, tabulated, statistically analyzed using SPSS version 22 **(Inc, Chicago, Illinos, USA).** Quantitative data were summarized as mean ($\underline{X}$), standard deviation (SD), and range. Qualitative data were summarized as numbers and percentages. To study the association between two qualitative variables, the Chi-square test (χ^2) was used. ANOVA (f) test was used to compare quantitative variables of three or more groups. Kruskal-Wallis test was used for comparing quantitative variables of three or more groups not normally distributed. Spearman's correlation (r) was used to test the association between two quantitative variables. A *p* value of < 0.05 was considered significant.

Results:

The studied subjects were 88 patients, with a mean age of 44.08±12.41 years. The mean BMI was 29.73±8.15 kg/m^2. 72% of patients were males, and 27% were females. 21.6% of them were smokers, 17.1% had hypertension. 71.6% were manual workers, and 27.4% were employers. Also, most of the studied patients (84.1%) were living in urban areas **(Table 1)**. Almost all studied parameters showed significant differences between groups, while no significant differences were reported between groups regarding Hb %, PLT count, and S. albumin (**Table 2)**.

The level of FABP1 increased in non-diabetic patients with BMI >25 with statistically significant differences versus other groups (**Table 3)**. There was increased grading of the fatty pancreas in diabetic patients either with normal BMI or with BMI >25 with a statistically significant difference (**Table 4)**. Also, there was a statistically significant relation between FABP1 expression with high grades of fatty pancreas among non diabetic subjects with BMI over 25 (**Table 5),(Figure 3,4,5).**

At cut-off 0.758, the sensitivity of FABP1 in the prediction of the fatty pancreas was (68.2%), specificity (45.5%) and area under the curve was 0.618 in diabetics and non-diabetics with normal BMI (**Figure 1)**. At cut-off 0.80, the sensitivity of FABP1 in the prediction of the fatty pancreas was (54.5%), specificity (54.5%), and area under the curve was 0.382 in non-diabetics and diabetics with BMI > 25 **(Figure 2)**. There was no correlation between FABP1 and other studied variables except for T. bilirubin,D. bilirubin in non-diabetic patients with BMI > 25, and the platelet count in diabetics with normal BMI (**Table 6).**

Table (1): Demographic characteristics of the studied groups (n=88).

	The studied groups (88) No.	 %
Gender (No ,%)		
Male (No ,%)	64	72.7
Female (No ,%)	24 Mean ±SD	27.3 Range
Age/year	44.08±12.41	18.0-69.0
BMI (kg/m^2)	29.73±8.15	18-43
Special habits		
Smoking	19	21.6
No-smoking	69	78.4
Blood pressure		
Hypertensive	15	17.1
Not Hypertensive	73	82.9
Occupation		
Employee	25	28.4
Manual worker	63	71.6
Residence		
Rural	14	15.9
Urban	74	84.1

Table (2): Comparison between the studied groups regarding laboratory investigations.

	Non-diabetics (n=44)		**Diabetics (n=44)**		**ANOVA test**	
	Normal BMI	**BMI > 25**	**Normal BMI**	**BMI > 25**		
	Mean ±SD	Mean ±SD	Mean ±SD	Mean ±SD	**T**	**P values**
WBCs**(c/mm^3)**					1: 0.95	1: 0.35
					2: 3.14	2: 0.003**
	6.2±2.21	6.85±2.35	6.29±2.14	8.51±2.52	3: 0.14	3: 0.89
					3: 2.25	4: 0.029 *
Hb**(g/dl)**					1: 0.85	1: 0.40
					2: 0.28	2: 0.78
	12.48±1.56	12.89±1.64	12.48±1.78	12.63±1.75	3: 0.0	3: 1.0
					4: 0.51	4: 0.62
PLT**(c/mm^3)**	251.77±63.09	213.95±83.61	231.0±57.83	240.64±73.11	1: 1.69	1: 0.09
					2: 0.49	2: 0.63
					3: 1.14	3: 0.26
					4: 1.13	4: 0.27
FBG(mg/dl)	95.18±7.66	195.55±74.0	97.95±6.37	207.32±78.05	1: 6.33	1: < 0.001 **
					2: 6.55	2: < 0.001 **
					3:1.31	3: 0.20
					4: 0.54	4: 0.61
HbA1c(%)	4.24±0.18	8.15±1.75	4.26±0.26	8.65±1.90	1: 10.41	1: < 0.001 **
					2: 10.73	2: < 0.001 **
					3: 0.27	3: 0.79
					4: 0.90	4: 0.37
ALT(**IU/dl**)	24.27±10.63	39.5±23.18	56.18±13.25	41.41±19.94	1: 2.8	1: 0.008 **
					2: 2.89	2: 0.006 **
					3: 8.81	3: < 0.001 **
					4: 0.29	4: 0.77
AST(**IU/dl**)	20.86±9.67	37.77±33.29	55.27±14.56	59.82±25.41	1: 2.29	1: 0.027 *
					2: 0.73	2: 0.47
					3: 9.23	3: <0.001 **
					4: 2.47	4: 0.018 *
GGT**(U/l)**	29.68±11.75	59.41±23.32	76.45±13.36	72.64±15.02	1: 5.34	1: <0.001 **
					2: 0.89	2: 0.38
					3: 12.33	3: < 0.001 **
					4: 2.24	4: 0.03 *
ALP(**IU/l**)	85.55±20.57	132.0±42.61	161.64±17.81	147.64±19.66	1: 4.61	1: <0.001 **
					2: 2.48	2: 0.017 *
					3: 13.12	3: < 0.001 **
					4: 1.56	4: 0.13
T. bilirubin**(mg/dl)**	0.79±0.21	0.87±0.25	0.79±0.20	0.98±0.19	1: 1.17	1: 0.25
					2: 3.28	2: 0.002 **
					3: 0.0	3: 1.0
					4: 1.68	4: 0.10
D. bilirubin**(mg/dl)**	0.42±0.12	0.41±0.16	0.26±0.19	0.44±0.14	1: 0.22	1: 0.83
					2: 3.57	2: 0.001 **
					3: 3.27	3: 0.002 *
					4: 0.67	4: 0.50
Albumin**(g/dl)**	4.29±0.28	3.86±0.38	4.11±0.29	5.75±8.33	1: 4.17	1: <0.001 **
					2: 0.92	2: 0.36
					3: 2.01	3: 0.051
					4: 1.06	4: 0.38
Cholesterol**(mg/dl)**	293.86±125.3 3	201.5±65.18	357.41±158.8 3	234.36±99.92	1: 3.07	1: 0.004 **
					2: 3.08	2: 0.004 **
					3: 1.47	3: 0.15
					4: 1.29	4: 0.20
TG**(mg/dl)**	100.27±26.66	102.32±34.49	101.91±30.34	133.32±113.0	1: 0.22	1: 0.83
					2: 1.26	2: 0.22
					3: 0.19	3: 0.85
					4: 1.23	4: 0.23

HDL**(mg/dl)**	32.45±8.78	37.09±7.46	32.23±6.87	37.5±9.26	1: 1.89 2: 2.15 3: 0.10 4: 0.16	1: 0.07 2: 0.04 3: 0.92 4: 0.87

P1:Non diabetic (N BMI, BMI > 25)

P2:Diabetic (N BMI, BMI > 25)

P3: N BMI (Diabetic, Non diabetic)

P4: BMI> 25 (Diabetic, Non diabetic)

Table (3): Comparison of the studied groups regarding FABP1 levels.

	Non-diabetics (n=44)		**Diabetics (n=44)**		ANOVA test	
	Normal BMI	**BMI>25**	**Normal BMI**	**BMI>25**	t	P-value
	Mean ±SD	**Mean ±SD**	**Mean ±SD**	**Mean ±SD**		
FABP1 level**(ng/ml)**	0.60±0.24	0.99±0.71	1.42±1.12	0.85±0.85	1: 2.42 2: 1.9 3: 1.52 4: 1.31	1: 0.02 * 2: 0.06 3: 0.14 4: 0.20

F: ANOVA F test,

P1:Non diabetic (N BMI, BMI > 25)

P2:Diabetic (N BMI, BMI > 25)

P3: N BMI (Diabetic, Non diabetic)

P4: BMI> 25 (Diabetic, Non diabetic)

Table (4): Comparison of the studied groups regarding different grades of fatty pancreas.

	Non-diabetics (n=44)		**Diabetics (n=44)**		**Sig. test**	
	Normal BMI	**BMI>25**	**Normal BMI**	**BMI>25**	**Test**	***P Value***
Grades of fatty pancrease						
No	6(27.3)	0(0.0)	0(0.0)	0(0.0)	FET_1:7.84	P_1: 0.05
I	5(22.7)	5(22.7)	0(0.0)	0(0.0)	FET_2:1.09	P_2: 0.30
II	6(27.3)	7(31.8)	7(31.8)	4(18.2)	FET_3:16.02	P_3:<0.001 **
III	5(22.7)	10(45.5)	15(68.2)	18(81.8)	FET_4:7.91	P_4: 0.013 *

X^2: Chi-square FET: Fishers exact test.

P1:Non diabetic (N BMI, BMI > 25)

P2:Diabetic (N BMI, BMI > 25)

P3: N BMI (Diabetic, Non diabetic)

P4: BMI> 25 (Diabetic, Non diabetic)

Table (5): Relation between FABP1 expression and different grades of fatty pancreas among thestudied groups.

			FABP1				ANOVA test	
			Mean ±SD		Range		F	*P value*
Non-diabetics (n=44)	NormalBMI	Grades on pancreas NoI II III	1.07 0.67 0.75 1.49	0.87 0.17 0.40 0.98	0.36 0.46 0.11 0.70	2.79 0.89 1.36 2.58	1.48	0.25
	BMI>25	Grades on pancreas I II III	0.51 0.43 0.76	0.13 0.15 0.23	0.36 0.16 0.35	0.70 0.59 1.24	7.03	0.005**
Diabetics (n=44)	NormalBMI	Grades on pancreasII III	1.39 1.43	0.97 1.21	0.70 0.41	3.0 4.31	St t= 0.06	0.95
	BMI>25	Grades on pancreas II III	0.62 0.90	0.20 0.93	0.38 0.29	0.82 4.41	St t= 0.59	0.56

*significant

Table (6): Correlation between FABP1 and other variables among studied groups.

	FABP1 level							
	Non-Diabetics subjects				**Diabetics patients**			
	Normal BMI		BMI over 25		Normal BMI		BMI over 25	
	R	*P value*	r	*P value*	r	*P value*	r	*P value*
Age(years)	0.13	0.55	-0.02	0.93	-0.05	0.84	-0.22	0.33
BMI(kg/m2)	-0.001	0.997	0.004	0.99	-0.13	0.56	0.11	0.62
SBP	-0.32	0.14	0.38	0.09	-0.15	0.52	-0.16	0.48
DBP	-0.498	**0.018***	0.31	0.16	0.033	0.88	0.20	0.39
WBCs**(c/mm^3)**	0.083	0.71	0.07	0.77	-0.17	0.45	-0.26	0.24
Hb **(g/dl)**	0.15	0.51	-0.03	0.91	-0.28	0.22	0.43	**0.046***
PLT**(c/mm^3)**	-0.13	0.56	0.19	0.39	-0.65	**0.001****	-0.33	0.13
FBG**(mg/dl)**	0.02	0.95	-0.06	0.79	0.22	0.32	-0.12	0.59
HbA1c**(%)**	-0.06	0.79	0.14	0.53	-0.22	0.32	0.15	0.50
ALT(**IU/l**)	0.03	0.89	-0.30	0.18	-0.19	0.39	-0.20	0.36
AST(**IU/l**)	0.001	0.997	-0.13	0.56	-0.006	0.98	-0.12	0.61
GGT(**U/l**)	-0.009	0.97	0.10	0.68	-0.08	0.73	-0.04	0.86
ALP(**IU/l**)	0.04	0.88	0.28	0.21	-0.15	0.51	-0.017	0.94
T bilirubin**(mg/dl)**	-0.06	0.80	-0.45	**0.035***	0.17	0.46	-0.12	0.58
D bilirubin**(mg/dl)**	0.14	0.54	-0.47	**0.029***	0.03	0.90	-0.34	0.13
Albumin**(g/dl)**	-0.098	0.67	0.37	0.09	-0.10	0.65	-0.12	0.60
Cholesterol**(mg/dl)**	0.39	0.08	-0.19	0.40	-0.07	0.76	0.043	0.85
TG**(mg/dl)**	-0.31	0.16	-0.14	0.53	-0.21	0.36	-0.18	0.43
HDL**(mg/dl)**	-0.24	0.28	-0.03	0.88	0.13	0.56	-0.11	0.63
S. insulin	0.09	0.69	0.14	0.54	-0.14	0.55	-0.05	0.83

r: correlation coefficient

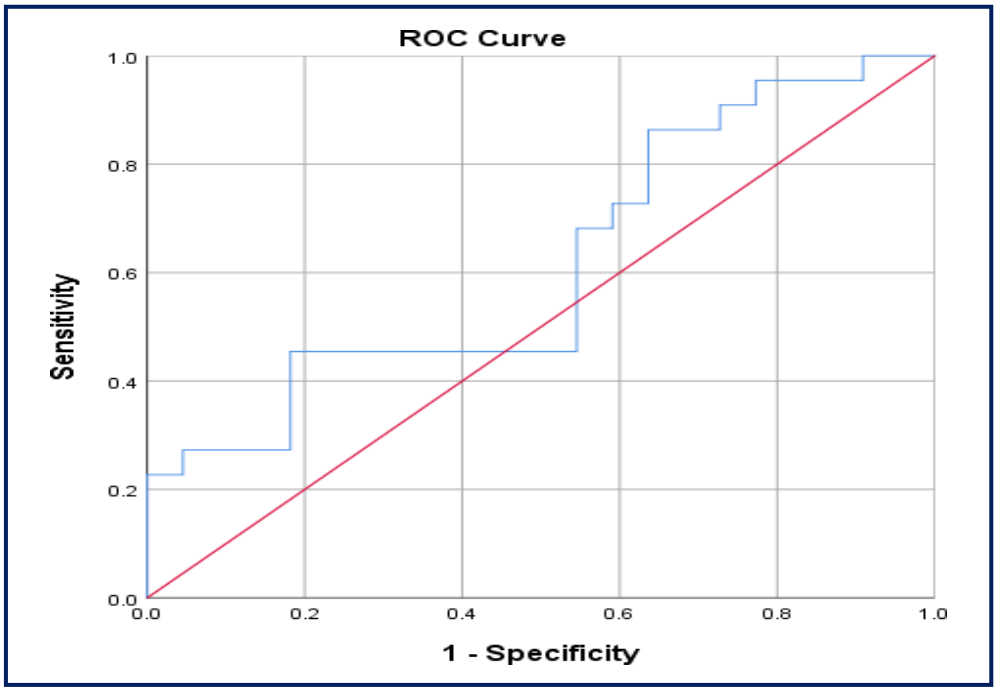

Fig 1: ROC curve of FABP1 level for prediction of fatty pancreas in diabetics, non diabetics with normal BMI.

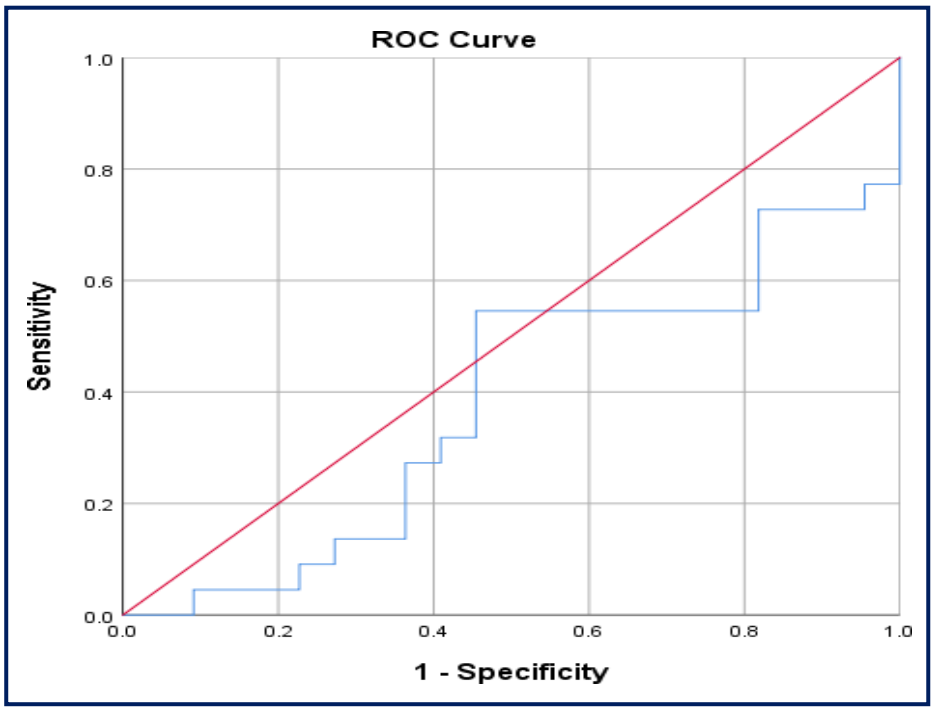

Fig 2: ROC curve of FABP1 level for prediction of fatty pancreas in non diabetics, diabetics with BMI >25 .

Fig 3: Grade I fatty pancrease

Fig 4: Grade II fatty pancrease.

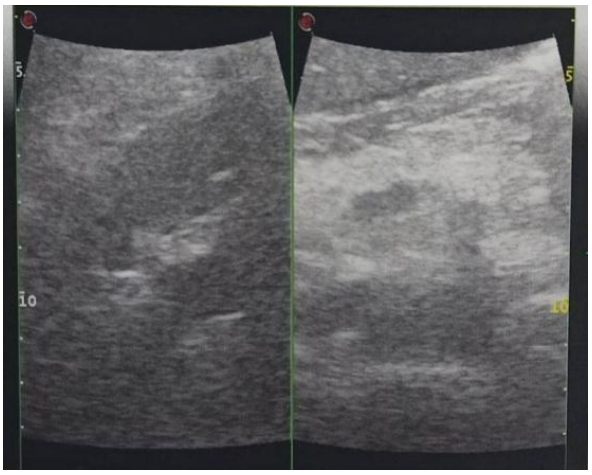

Fig 5: Grade III fatty pancrease

Discussion:

The mean age of the studied patients was 44.08 ±12.41 years. This is in line with **another** study**,** which showed that NAFPD patients' mean age was significantly higher (44.15) than non NAFPD subjects (39.12) **(14).**

In line with this finding, another study reported that age was positively correlated with the fatty pancreas **(15)**. Lesmana and co-workers **(6)** reported that the presence of fatty pancreas was related to age increase (> 35 years). This could be related to a dysfunctional lipid metabolism being exacerbated by age-related metabolic slowing and aggravation of ectopic fat deposition caused by prolonged dyslipidemia (**16**).

In the current study, NAFPD was more frequent in males than females (64 versus 24) with a male to female ratio (2.6:1). This male predominance came in agreement with **others (6).** Men are at greater risk for NAFPD due to increased visceral fat deposition, whereas women had increased subcutaneous (gluteal femoral) lipid deposition.

In the current study, BMI mean was (29.73 ±8.15). This agreed with El-Badawy and colleague **(14). ,** who concluded that the disease was associated with high BMI, with a

significant difference (P < 0.001) between fatty pancreas and non-fatty pancreas groups (30.4 kg/m^2 vs. 27.3 kg/m^2, respectively) **(14)**. Also, the current study showed a significant difference as regard ALT, AST,GGT, ALP, T. bilirubin, and D. bilirubin, which were higher in the diabetic patients either with normal BMI or with BMI >25 kg/m^2 in (**Table 2)**. This is in line with **other study (4)**, who reported an association between liver enzymes including AST, ALT, and γ GGT and fatty pancreas **(4).** On the other hand, others showed that no significant associations were found between fatty pancreas and AST, ALT, and γ GGT levels (**5**).

The present study revealed that high levels of serum cholesterol in diabetics and non-diabetics with normal BMI were statistically significant. This came in line with another study (**5**), where it was reported significantly higher total cholesterol (P < 0.001), TG (P < 0.001), LDL-C (P < 0.001), VLDL-C (P < 0.001), cholesterol/HDL (P=0.007),

and LDL/ HDL ratios (P=0.024) in the fatty pancreas group.

The level of FABP1 increased in non-diabetic patients with BMI >25, with a statistically significant difference compared to other groups. This came in line with the results from the study done in 2019 (17), who reported a significant positive correlation between FABP1 and BMI and fasting blood sugar.

This study revealed increased grading of the fatty pancreas in diabetic patients either with normal BMI or with BMI >25 with statistically significant difference.

The present study showed a statistically significant relation between FABP1 expressions with high grades of fatty pancreas among non-diabetic subjects with BMI over 25.

Conclusions:

There was an association between Non-alcoholic fatty pancreas and diabetes mellitus with increasing fatty pancreas grading. FABP may be a good marker for the diagnosis of NAFPD.

References:

1- Alempijevic T, Dragasevic S, Zec S, Popovic D, Milosavljevic T. (2017): Nonalcoholic fatty pancreas disease. Postgrad Med J; 93:226-230.

2- Schaefer JH. (1926): The normal weight of the pancreas in the adult human being: A biometric study. Anat Rec.;32(2): 119-32.

3- Ogilvie RF.(1933): The islands of langerhans in 19 cases of obesity. J Pathol Bacteriol; 37: 473–481.

4- Lee JS, Kim SH, Jun DW, Han JH, Jang EC, Park et al (2009): Clinical implications of fatty pancreas: correlations between fatty pancreas and metabolic syndrome. World journal of gastroenterology: WJG. 15(15):1869.

5- Wang CY, Ou HY, Chen MF, Chang TC, Chang CJ. (2014): Enigmatic ectopic fat: prevalence of nonalcoholic fatty pancreas disease and its associated factors in a Chinese population. J Am Heart Assoc; 3:297–304.

6- Lesmana CR, Pakasi LS, Inggriani S, Aidawati ML and Lesmana LA(2015): Prevalence of Non-Alcoholic Fatty Pancreas Disease (NAFPD) and its risk factors among adult medical check- up patients in a private hospital: a large cross-sectional study. BMC gastroenterology; 15(1):174.

7- Mathur A, Zyromski N, Pitt H, Walker J, Saxena R, Lillemoe K et al (2009): Pancreatic Steatosis Promotes Dissemination and Lethality of Pancreatic Cancer. Southern Surgical Association article| Volume 208, ISSUE 5, P989-994.

8- Smits MM, van Geenen EJ. (2011): The clinical significance of pancreatic steatosis. Nat Rev Gastroenterol Hepatol; 8: 169–177.

9- Amiri M, Yousefnia S, Forootan FS, Peymani M, Ghaedi K, Esfahani MH (2018): Diverse roles of fatty acid binding proteins (FABPs) in development and pathogenesis of cancers. Gene; 676:171-83.

10-- Abdulaziz BA, Abdu SA, Amin AM, El Menyawi AK, Ahmed A, Khalil MA etal (2019): Assessment of Liver Fatty Acid Binding Protein (L-FABP) as a Diagnostic Marker in Non-Alcoholic Fatty Liver Disease. Open Journal of Gastroenterology; 9(6):113-24.

11-Wang G, Bonkovsky HL, de Lemos A, Burczynski FJ. (2015): Recent insights into the biological functions of liver fatty acid binding protein 1. Journal of lipid research. 2015 Dec 1;56(12):2238-47.

12-Gutch M, Kumar S, Razi SM, Gupta KK, Gupta A. (2015): Assessment of insulin sensitivity/resistance. Indian journal of endocrinology and metabolism. 2015 Jan;19(1):160.

13-Marks WM, Filly RA, Callen PW. (1980): Ultrasonic evaluation of normal pancreatic echogenicity and its relationship to fat deposition.Radiology;137:475–9.

14-El-Badawy R, Yousef A, Elkholy H and Mustafa A (2020): Studying the Association between Non-Alcoholic Fatty Pancreatic Disease and Non-Alcoholic Fatty Liver Disease. Benha Medical Journal.

15-Wang D, Yu XP, Xiao WM, Jiao XP, Wu J, Teng DL et al (2018): Prevalence and clinical characteristics of fatty pancreas in Yangzhou, China: A cross-sectional study. Pancreatotomy; 18(3):263-8.

16-Wallace TM, Levy JC, Matthews DR (2004):An increase in insulin sensitivity and basal beta- cell function in diabetic subjects treated with pioglitazone in a placebo-controlled randomized study. Diabet Med; 21:568–76

17-Abdelkader A, Al-Dahshan M, Ali M, Othman M, Al-Zohairy Y (2019) : Non Alcoholic Fatty Pancreatic Disease Significance, Importance and Risk factors. International Journal of Science and Research (IJSR) ; 7.583.

To cite this article: Reda EI-Badawy, Awad M. EL Abd, Heba M. Bedair, Ghadeer M. Rashad. Role of Fatty Acid Binding Protein 1 in Patients with Non- Alcoholic Fatty Pancreatic Disease with and without Diabetes Mellitus. BMFJ 2023;40(3):662-673.