https://doi.org/10.48047/AFJBS.6.14.2024.6262-6274

African Journal of Biological Sciences

Journal homepage: http://www.afjbs.com

Research Paper Open Access

Study of Fatty Pancreas in Egyptian Patients with Non Alcoholic Fatty Liver Disease: A Cross Sectional Study

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Article History

Volume 6, **Issue** 14, 2024

Received: 10 July 2024

Accepted: 17 August 2024

doi: 10.48047/AFJBS.6.14.2024.6262-6274

Abstract:

Background: NAFLD and fatty pancreas are related to abdominal obesity, insulin resistance, dyslipidemia, diabetes mellitus, and hypertension, which are major etiologies of global health issues and economic burden.

Aim of study: This research was aimed at investigating the fatty pancreas prevalence among Egyptian patients with NAFLD, identifying the risk factors linked to it, and investigate the role of plasma FABP1 as a biomarker for fatty pancreas.

Patients and Methods: The cross sectional study included 200 patients with NAFLD who underwent a further categorization, according to presence of fatty pancreas, measured by ultrasonography.

Result: Fatty pancreas is a common finding in NAFLD patients, with a prevalence of 62.5%. Age, type II DM, NAFLD, metabolic syndrome, overall obesity, central obesity, and serum FABP1 level were all independent predictors of FP. At cut-off > 686, the sensitivity of FABP1 in the prediction of the fatty pancreas was 91.2%, specificity was 94.67%, PPV was 96.6%, NPV was 86.6%, and accuracy 97.7%.

Conclusion: A significant correlation exists between fatty liver and fatty pancreas leading to metabolic consequences such as metabolic syndrome, insulin resistance, and progression of liver disease.

Keywords: NAFLD; fatty pancreas; metabolic syndrome; FABP1

1. Introduction

Obesity represents a significant global health issue that is associated with many metabolic complications, involving type 2 diabetes, metabolic syndrome, non-alcoholic fatty liver disease (NAFLD), as well as cardiovascular disease [1].

NAFLD is now the predominant etiology of chronic liver disease worldwide and is currently expected to affect around 38% of the adult population globally [2].

The pancreas, like the liver, may develop ectopic fat deposits without alcohol use, a condition known as nonalcoholic fatty pancreatic disease (NAFPD), or fatty pancreas (FP) [3].

In recent years, there has been significant attention given to the fat deposition within the pancreas. However, unlike NAFLD, the possible adverse events associated with pancreatic fat accumulation remain not well-established [4].

Ultrasound stands as the most commonly employed imaging technique for evaluating the pancreas and is considered the best method in everyday clinical practice due to its simplicity as well as non-invasiveness [5]. It identifies pancreatic steatosis as having a higher echogenicity in pancreas when compared to the liver or kidney [6].

The biomarkers utilized for pancreatic steatosis detection are still limited. Therefore, imaging could be the cornerstone for diagnosis. Consequently, laboratory markers should be urgently employed to improve diagnostic accuracy due to its simplicity and non-invasiveness [7].

Fatty Acid Binding Protein 1 (FABP1) stands as an essential tissue-specific biomarker. It exhibits a vital role in systemic inflammation [8]. It is primarily expressed on the liver (hepatocyte cytoplasm). However, it has been identified in other sites, involving renal, intestinal, pulmonary, as well as pancreatic tissues [9]. FP emerges as an innovative disease entity, which gained increasing popularity while the obesity epidemic is rising. That's why we carried out this research.

2. Patients and Methods

The cross-sectional study took place at the Tropical Medicine Department, Tanta University Hospital, within a timeframe between August 2020 and March 2023, including 200 patients with non-alcoholic fatty liver disease. They underwent a further categorization, according to fatty pancreas presence, assessed with ultrasonography, into two groups; Group I involved 75 patients who do not have fatty pancreas while Group II involved 125 patients with fatty pancreas. We commenced our research after it got approved by the Ethic Committee of the Tanta ethical committee (Approval code: 34061/8/20). All participants were asked to sign an informed consent prior to participation.

Patients who consumed alcohol excessively, with a dosage over 30 g daily among males and over 20 g daily among females; those who previously have viral hepatitis or others having positive hepatitis B or C virus markers or any other chronic liver disease; those who developed chronic pancreatitis or prior pancreatitis episodes; those having renal disorders or taking medications like amiodarone, cortisone, valproate, methotrexate, were excluded.

2.2. Anthropometric measurements

The height as well as body weight were assessed while wearing light clothes in order to get the Body Mass Index (BMI). Waist circumference (WC) was assessed by a non-elastic tape, at the midpoint between the iliac crest and the lowest rib, while the individual was standing and at the normal exhale end. Hip circumference (HC) was measured at the buttocks' broadest region.

2.3. Biochemical parameters

Every participant underwent clinical assessment as well as lab testing. The latter involved measuring:

- Serum bilirubin, transaminases, albumin, fasting plasma glucose, HbA1c, as well as fasting lipid profile.

- The chemiluminescent sandwich immunoassay (CLIA technique method) was employed for assessing serum insulin levels. This was done utilizing the fully automated LIAISON® XL device (LIAISON® Insulin 310360: Diasorin S.P.A, Verecelli, Italy) for the quantitative measurement of insulin. The sensitivity of the kit ranged from 0.23 to 0.61 µIU/ml, and its assay range fell between 0 and 500 µIU/ml. The standard reference range for serum insulin, as determined by the specific kit employed, was 3.21-16.32 µIU/ml.

- The homeostasis model assessment (HOMA-IR) was calculated utilizing fasting blood glucose as well as fasting insulin levels, according to the formula: HOMA-IR = fasting insulin level (µU/L) multiplied by fasting glucose level (mg/dl), divided by 405 [10].

- FABP1 was measured utilizing a sandwich enzyme-linked immunosorbent assay (ELISA) kit supplied by SunRed Company (catalogue no. 201-12-3752). The kit's sensitivity indicated 5.526 ng/L, while its assay range fell between 6 ng/L and 1800 ng/L. As per the manufacturer's instructions, this kit offers a high level of sensitivity with an exceptional specificity for detecting FABP1. Additionally, there were no notable cases of cross-reactivity or interference with other analogues. The standard reference range for serum FABP1, as determined by the ELISA kit, fell between 120 and 400 ng/L.

2.4. Definition of metabolic syndrome

The metabolic syndrome diagnosis adhered to the criteria set by the NCEP-Adult Treatment Panel III (ATP III). The diagnostic criteria were established if the patient met three or more of the following conditions:

- Visceral obesity (WC equal to or more 102 cm for males, or WC equal to or more 88 cm for females),
- Elevated triglyceride levels (≥ 150 mg/dL) or receiving treatment for dyslipidemia,
- Low levels of HDL cholesterol (< 40 mg/dL for males, < 50 mg/dL for females) or receiving treatment for dyslipidaemia,
- High blood pressure (≥ 130/85 mmHg) or receiving treatment for hypertension, as well as fasting glucose levels (≥ 100 mg/dL) or previously diagnosed with T2DM.

2.5. Imaging:

The FibroScan® 502 device developed by Echosens, equipped with both M and XL probes, was employed for assessing steatosis with Controlled Attenuation Parameter (CAP) while fibrosis was evaluated with Liver Stiffness Measurement (LSM). Both LSM as well as CAP were acquired from the same hepatic parenchyma region. The operators of Transient elastography were unaware of the clinical data.

All participants underwent an abdominal US examination conducted by a skilled radiologist utilizing a high-resolution ultrasound machine (Toshiba TUS-A500 Aplio S.N: W7C2232110) equipped with a 5 MHz convex-array probe. The radiologist was unaware of the demographic, clinical, as well as biochemical data of the participants. If the pancreas echogenicity was higher than the kidney echogenicity, it is characterized as fatty pancreas. Otherwise, it is classed as non-fatty pancreas. The term "fatty pancreas" was classified into three levels. Level 1 refers to a higher echogenicity of the pancreas compared to renal tissues. Level 2 indicates a higher pancreas echogenicity compared to the kidneys, but slightly lower than the echogenicity of the retroperitoneal fat. Level 3 describes a pancreas echogenicity that is similar to or higher than the echogenicity of the retroperitoneal fat [11].

2.6. Statistical analysis

Our team analyzed data statistically with IBM SPSS statistics® version.20. The Student’s t-test was employed for comparing among both groups; the multiple logistic analysis was employed while analyzing independent correlation factors associated with fatty pancreas; the ROC curve for FABP1 diagnostic assessment; the Pearson correlation test was utilized for assessing correlation. P value of below 0.05 was deemed statistically significant.

3. Results

Two hundred patients with non-alcoholic fatty liver disease were included and underwent a categorization into two groups, based on existence of FP; Group I involved 75 patients without fatty pancreas while Group II involved 125 patients with fatty pancreas. There were 75 (37.5%) patients without FP, while of the 125 (62.5%) patients with FP, 31 (15.5%), 44 (22%), and 50 (25%) were grade I, II, and III, respectively. Baseline clinical as well as laboratory data were compared among the two groups, showing significant differences except for sex, total bilirubin, and INR (Table1&2).

Table (1). The demographic and clinical characteristics of patients in the studied groups.

		Group 1 Non FP		Group II FP		P
Age (years)	Range	19-58		35-67		<0.001
	Mean ±SD	41.373±10.393		48.840±8.138		
Sex	Male	37	49.33	73	58.40	0.212
	Female	38	50.67	52	41.60	
Current smoking	No	47	62.67	58	46.40	0.026
	Yes	28	37.33	67	53.60	
HTN	No	58	77.33	78	62.40	0.028
	Yes	17	22.67	47	37.60	
Type 2 DM	No	65	86.67	34	27.20	<0.001
	Yes	10	13.33	91	72.80	
Metabolic syndrome	No	46	61.33	15	12.00	<0.001
	Yes	29	38.67	110	88.00	
BMI (kg/m^2)	Range	23.1 - 36.9		21.6 - 44.9		<0.001
	Mean ±SD	29.424 ± 4.484		35.163 ± 5.560		
WC (cm)	Range	82 - 125		85 - 138		<0.001
	Mean ±SD	100.707 ± 11.90		113.464 ± 10.98		
HC (cm)	Range	89 - 138		95 - 148		<0.001
	Mean ±SD	110.13 ± 12.044		123.000 ± 11.44		
Waist/hip ratio (WHR)	Range	0.78 - 1.04		0.8 - 1.09		0.149
	Mean ±SD	0.914 ± 0.040		0.923 ± 0.047		

Fatty Acid Binding Protein 1 (FABP1) was statistically higher among patients with FP than patients without FP (938.176±172.568 vs.500.173± 158.547; p-value <0.001), as illustrated on (Table 2). At a cut-off > 686, the sensitivity of FABP1 in the prediction of the fatty pancreas was 91.2%, specificity was 94.67%, PPV was 96.6%, and NPV was 86.6%, with an accuracy 97.7% (Figure 1).

Table (2). Laboratory data among the two studied groups.

		Group 1 Non FP (n=75)	Group II FP (n=125)	P
ALT (IU/L)	Range	11 – 112	11 - 143	<0.001
	Mean ±SD	32.37 ± 14.24	55.07 ± 31.78	
AST (IU/L)	Range	12 -86	11 - 115	<0.001
	Mean ±SD	27.34 ±12.45	46.93 ± 24.01	
GGT (IU/L)	Range	20 - 37	21 - 37	<0.001
	Mean ±SD	25.34 ± 3.32	28.93 ± 3.09	
Serum albumin (g/dl)	Range	3.7 - 5	3.5 - 4.8	<0.001
	Mean ±SD	4.29 ± 0.3	4.05 ± 0.32	
Total bilirubin (mg/dl)	Range	0.26 - 1.2	0.3 - 1.2	0.089
	Mean ±SD	0.80 ± 0.19	0.86 ± 0.24	
FBG (mg/dl)	Range	78 - 215	85 - 267	<0.001
	Mean ±SD	109.86 ±20.41	139.00 ±31.80	
HbA1C (%)	Range	4.6 - 8.5	4.9 - 13.1	<0.001
	Mean ±SD	5.643 ± 0.839	7.608 ± 1.724	
Fasting insulin (µIU/ml)	Range	2.98 -30.2	7.6 - 35.12	<0.001
	Mean ±SD	13.052 ± 6.276	19.474 ± 5.840	
HOMA- IR	Range	0.68 - 10.96	1.9 - 16.59	<0.001
	Mean ±SD	3.602 ± 2.150	6.775 ± 3.042	
Cholesterol (mg/dl)	Range	114 - 340	104 -345	0.002
	Mean ±SD	211.627 ± 44.314	233.724±49.079	
Triglycerides (mg/dl)	Range	82 - 280	124-376	<0.001
	Mean ±SD	184.46 ± 43.96	214.920±40.112	
LDL (mg/dl)	Range	82 - 270	40-274	0.032
	Mean ±SD	129.405 ± 35.814	141.510±39.700	
HDL (mg/dl)	Range	29 - 77	20.7-64	0.001
	Mean ±SD	50.107 ± 8.680	46.087±8.276	
FABP1 (ng/ml)	Range	105 - 758	542 - 1284	<0.001
	Mean ±SD	500.173 ± 158.547	938.176 ± 172.568	

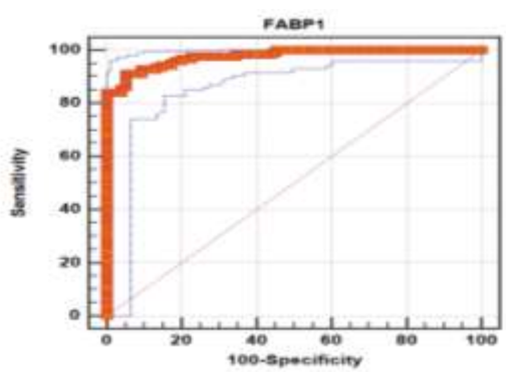


Fig. (1): ROC curve of FABP1 level for prediction of fatty pancreas

FABP1 showed a positive correlation with age, AST, ALT, GGT, BMI, WC, FBG, HbA1C%, HOMA-IR, total cholesterol, serum triglyceride, and LDL as well as hepatic steatosis and liver fibrosis. Conversely, a statistically significant negative association was documented between

FABPI and HDL while no statistically significant variance was observed between FABP1 and other numerical data (Table 3).

Table (3). Correlation between FABP1 and other numerical variables.

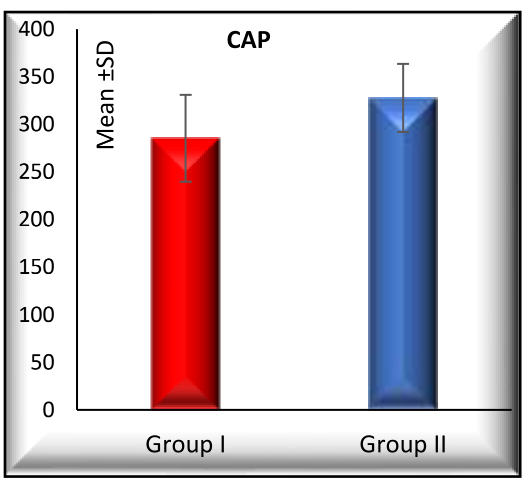
Correlation		
	FABP1	
	r	**P-value**
Age (years)	0.354	<0.001
BMI (kg/m^2)	0.554	<0.001
WC (cm)	0.497	<0.001
ALT(IU/l)	0.509	<0.001
AST(IU/l)	0.568	<0.001
GGT(IU/l)	0.608	<0.001
Total bilirubin (mg/dl)	0.132	0.062
Hemoglobin (mg/dl)	-0.128	0.070
Platelet (X10^9)	-0.111	0.119
FBG (mg/dl)	0.552	<0.001
HbA1C%	0.680	<0.001
Fasting insulin (µIU/ml)	0.597	<0.001
HOMA- IR	0.627	<0.001
TC (mg/dl)	0.322	<0.001
TG (mg/dl)	0.450	<0.001
LDL (mg/dl)	0.288	<0.001
HDL (mg/dl)	-0.265	<0.001
Hepatic steatosis (dB/m)	0.584	<0.001
Liver fibrosis (kPa)	0.591	<0.001

We analyzed that the grades of steatosis were noticeably greater in patients with fatty pancreas (p-value <0.001) (Table 4; Figure 2). FP cases exhibited a significantly greater fibrosis score (4.860 ± 1.256 vs. 6.99 ± 2.768; p-value <0.001) and exhibited a significantly greater frequency of advanced fibrosis (p-value <0.001) (Table 4; Figure 3).

Table (4). The relationship between fatty pancreas and liver steatosis and fibrosis.

	Group 1	**Group II**	**P**

		Non FP (n=75)		FP (n=125)		
CAP (dB/m)	Range	238 - 400		247 - 400		<0.001
	Mean±SD	285.38±45.56		327.656±35.825		
LSM (kPa)	Range	2.1 - 10.3		3.1 - 14.5		<0.001
	Mean±SD	4.860 ± 1.256		6.990 ± 2.768		
		N	**%**	**N**	**%**	**P-value**
Steatosis grade	**S1**	25	33.33	2	1.60	<0.001
	S2	27	36.00	25	20.00	
	S3	23	30.67	98	78.40	
Fibrosis grade	**F0**	58	77.33	39	31.20	<0.001
	F1	14	18.67	43	34.40	
	F2	2	2.67	15	12.00	
	F3	1	1.33	21	16.80	
	F4	0	0.00	7	5.60	



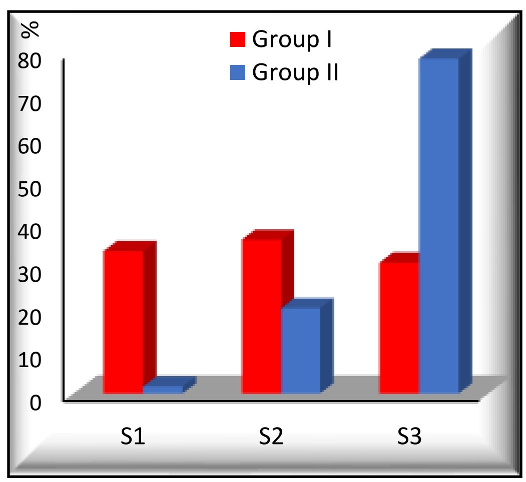


Fig. (2): The relationship between fatty pancreas and NAFLD.

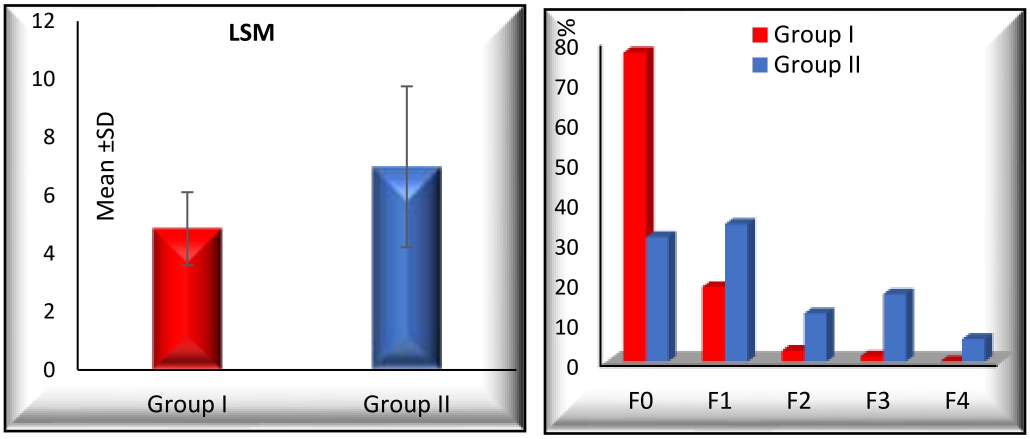


Fig. (3): The Relationship between fatty pancreas and liver fibrosis.

The FP cases underwent a further categorization into two subgroups according to pancreatic fat grade measured by US, namely, mild FP group (GI) and severe FP group (GII+GIII). Age, HTN, central obesity, metabolic syndrome, and HDL showed no variance by the grade of pancreatic fat (Table 5).

Table (5): Clinical, laboratory and metabolic parameters based on pancreatic fat grade.

	Fatty pancreas		P
	Mild FP (n=31)	Severe FP (n=94)	
	Mean±SD	Mean±SD	
Age (years)	48.387±9.649	48.989 ±7.628	0.722
Male sex (N %)	14 (45.16%)	64 (68.09 %)	0.022
Smoking (N %)	11 (35.48%)	56 (59.57%)	0.020
HTN (N %)	12 (38.71%)	35 (37.23%)	0.883
Type 2 DM (N %)	13 (41.94%)	78 (82.98%)	<0.001
BMI (kg/m2)	33.290±5.172	35.884±5.437	0.021
Central obesity (N %)	29 (93.55%)	86 (91.49%)	0.714
MetS (N %)	25 (80.65%)	85 (90.43%)	0.146
ALT(IU/l)	33.516±15.962	62.189±32.533	<0.001
AST(IU/l)	29.677±10.931	52.631±24.464	<0.001
GGT(IU/l)	26.839±2.491	29.628±2.969	<0.001
Albumin (g/dl)	4.248±0.261	3.990±0.314	<0.001
FBG (mg/dl)	120.903±24.755	144.968±31.710	<0.001
HbA1C%	6.298±1.095	8.039±1.679	<0.001
Insulin (µIU/ml)	16.707±4.742	20.387±5.902	0.002
HOMA- IR	4.928±1.853	7.384±3.117	<0.001
TC (mg/dl)	218.581±42.218	238.718±50.350	0.047
TG (mg/dl)	201.581±42.043	219.319±38.682	0.032
LDL (mg/dl)	127.161±34.948	146.243±40.202	0.020
HDL (mg/dl)	46.216±8.727	46.045±8.169	0.921
CAP (dB/m)	306.774±36.421	334.543±33.016	<0.001
LSM (kPa)	5.110±1.033	7.610±2.882	<0.001

A multivariate logistic regression analysis was done for identifying the main independent predictors of fatty pancreas. A high serum FABP1 level, age, type II DM, NAFLD, metabolic syndrome, overall obesity and central obesity were the main independent predictors of fatty pancreas (Table 6).

Table (6): Factors predict fatty pancreas analyzed by multiple logistic-regression analysis.

	Odd's ratio	95.0% C.I. for Odd's ratio		P-value
Age	1.110	0.989	1.247	0.028
Smoking	0.765	0.335	2.959	0.058
Type 2 DM	1.260	0.828	3.197	0.019
HTN	0.631	0.086	4.633	0.651
Metabolic syndrome	1.118	0.354	5.605	0.013
NAFLD	1.025	0.997	1.054	0.036
Overweight and obesity	1.106	0.017	3.247	0.019
Central obesity	1.046	0.101	2.213	0.041
Waist/height ratio	0.178	0.001	0.644	0.350
HOMA- IR	0.849	0.447	1.612	0.617
HbA1C	0.492	0.120	2.026	0.326
TC (mg/dl)	0.983	0.950	1.017	0.332
TG (mg/dl)	0.995	0.972	1.018	0.653
LDL (mg/dl)	1.008	0.974	1.044	0.641
HDL (mg/dl)	0.961	0.857	1.078	0.499
FABP1	2.026	1.013	4.038	<0.001

4. Discussion

Fatty pancreas has lately gained attention as being related to obesity, diabetes, as well as metabolic syndrome [12]. The fatty pancreas prevalence among Asians ranges from 16% to 35% [13], while 50% to 80% of NAFLD patients are known to have fatty pancreas [14].

Although the first identification of FP was documented in the 1930s, its clinical implications were not thoroughly investigated for many years since most physicians considered this condition to be harmless and without any significant future clinical effects. Recent studies have shown that FP is linked to other comorbid disorders [15]. Hence, we undertook this research to investigate its occurrence among Egyptian patients with NAFLD, determine the factors that contribute to its development, and explore the potential of plasma FABP1 levels as a biomarker for fatty pancreas.

Our research revealed a significant incidence of fatty pancreas among adult patients with non-alcoholic fatty liver disease (NAFLD), with a detection rate of 62.5%. It is evident that in this study, focusing on individuals with NAFLD, there was a much greater occurrence of FP compared to earlier studies that screened the general population (16-35%) [13]. The mean age of FP patients showed a significant increase in comparison to that without FP (48.840±8.138 vs. 41.373±10.393; p <0.001). Juliyanti et al. (2017) [16] along with Weng et al. (2018) [17] also addressed similar findings, showing that the FP occurrence becomes more common as individuals get older. This could be attributed to a decrease in pancreatic parenchymal volume associated with aging, as well as factors, involving atherosclerosis, reduced blood flow, metabolic dysfunction exacerbated by age-related metabolic slowdown, and ectopic fat deposition induced by prolonged dyslipidemia [18]. Consequently, advancing age is regarded as a significant risk factor for the FP development.

The present research revealed a significant difference in ALT, AST, as well as GGT levels among both groups under investigation. These findings align with Okada et al. (2021) [21], but contradict the results of Wang et al. (2018) [22], who did not see any significant correlations between FP and AST, ALT, and GGT levels. This variation may be attributed to that all of our patients have NAFLD, as well as the diverse ethnic origins along with sample sizes. The serum albumin level exhibited a significant decrease among patients with FP in comparison to others without this condition. The findings of our research indicate that FP cases exhibited a significantly elevated fibrosis score and a greater occurrence of advanced fibrosis.

Our research addressed that the average BMI value exhibited a significant increase among FP cases compared to cases without FP. The lean patients rate differed significantly among FP and non-FP cases (6.4% vs. 29.3%, $p < 0.001$). Among the eight lean NAFLD cases within the FP group, three of them exhibited severe FP. Being one of the easiest ectopic fat to be detected, NAFLD could be an early predictor for metabolic conditions, even among cases with normal body weight. Hence, an early NAFLD detection offers chances to manage the NAFPD progression [23]. BMI measures do not differentiate between trunk and visceral obesity, although anatomical fat distribution is of prime importance due to its varying metabolic implications [24]. Our research provides evidence for a correlation between FP and visceral obesity, as shown by an increase in waist circumference (WC).

Markers associated with lipid metabolism and glucose metabolism exhibited strong associations with the FP severity. The presence of FP has been linked to hyperlipidemia, which is characterized by elevated levels of total cholesterol, triglycerides, as well as LDL cholesterol, along with a reduction in HDL cholesterol. Additionally, FP patients showed significantly greater FBG, HbA1c, fasting insulin, HOMA-IR. Similarly, a cohort research conducted by Wong et al. (2014) also revealed that cases developing both FP and NAFLD had a greater HOMA-IR in comparison to those with either condition alone [25].

There is strong evidence that metabolic syndrome, which includes conditions such as obesity, diabetes, dyslipidemia, as well as hypertension, is linked to both NAFLD and NAFPD [26]. The current research revealed a significant correlation between fatty pancreas and the occurrence of metabolic syndrome and its components ($p < 0.001$). Metabolic syndrome was present in 88% of individuals diagnosed with FP and in 90.4% of severe FP cases. Our study's results were consistent with the findings of Singh et al. (2017), which showed that FP was linked to a significantly higher risk of arterial hypertension, DM, as well as metabolic syndrome [7].

Our research indicated that FP cases exhibited a significantly greater fibrosis score and were more likely to have advanced fibrosis. This is not unexpected considering FP is closely correlated with metabolic syndrome and its components. Furthermore, when FP cases were subdivided based on pancreatic fat grade, FP itself seemed not to be associated with NASH and advanced fibrosis, but the presence of extensive FP resulted in an increased likelihood of advanced fibrosis (all the cases with significant fibrosis $\geq$ F2 were in the severe FP group).

Furthermore, we ran a multivariate logistic regression analysis and discovered that serum FABP1 level, age, type II DM, NAFLD, metabolic syndrome, overall obesity and central obesity were the significant independent predictors of fatty pancreas. Serum FABP1 revealed the greatest OR in comparison to the other factors. Therefore, serum FABP1 could be a significant marker of fatty pancreas. This was similar with the results of Soliman et al. (2023) [27] who observed elevated serum FABP1 levels in FP cases, suggesting FABP1 may be a suitable diagnostic marker for detecting NAFPD. We addressed that serum FABP1 levels were highly linked with BMI, visceral obesity, glucose as well as lipid parameters. This came in harmony with Shi et al. (2012), who reported significant increases in FABP1 among healthy obese subjects in comparison to normal weight subjects as the "paradoxical" increase of serum FABP1 in obese subjects could be a compensatory up-regulation of the human body to counteract the metabolic stress imposed by obesity [28]. Our limitation is the method we utilized for detecting FP. US depends mostly on operators, though the same radiologist assessed the participants. In contrast, US remains a qualified method in a skilled hand. Additionally, its non-invasiveness, speed, as well as affordability makes it a good tool in clinical application in comparison with MRI, CT, along with EUS.

5. Conclusion:

Fatty pancreas shows a greater prevalence among patients with NAFLD. This indicates that similar risk factors could have an impact on the pathogenesis of both conditions. FABP1 could be an important marker of FP.

Conflict of Interest:

No conflicts of interest declared.

Acknowledgments:

This research was carried out without funding.

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