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STUDYING THE ASSOCIATION BETWEEN NON-ALCOHOLIC FATTY LIVER DISEASE AND NON-ALCOHOLIC FATTY PANCREAS DISEASE USING TRANSABDOMINAL ULTRASOUND AND ENDOSCOPIC ULTRASOUND

By

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is characterized by hepatic triglyceride accumulation not due to alcohol consumption (<20 g ethanol per day), resulting in steatosis and hepatic inflammation. NAFLD is currently the most common liver disorder, particularly in Western countries. Worldwide, the prevalence of NAFLD is about 25%. Nonalcoholic fatty pancreas disease (NAFPD) is an excessive lipid accumulation in the pancreas in the absence of significant alcohol intake. Endoscopic ultrasound (EUS) can provide detailed images of the entire pancreas. The use of high-frequency US waves and the ability to simultaneously image adjacent organs like the liver and spleen in real-time.

Objective: To determine the possible association between non- alcoholic fatty pancreatic disease (NAFPD) and non-alcoholic fatty liver disease (NAFLD).

Patients and methods: This study was conducted on 100 subjects divided into two equal groups: Group I Included individuals with sonographically proven NAFLD, and Group II (control group): Included healthy individuals with no sonographic evidence of NAFLD. All cases were selected from Internal Medicine Department at Al-Hussein Hospital, Al-Azhar University, during the period from April 2019 to April 2020.

Results: Based on the severity of fatty pancrease, moderate and severe fatty pancrease was significantly associated with older age with p value 0.0001. Fasting-blood-sugar (FBS), erythrocyte sedimentation rate (ESR), and aspartate-aminotransferase (AST) significantly higher in moderate and severe fatty pancreases too with p value 0.0001, 0.001 and 0.0001 respectively. Serum albumin level was the lowest in severe fatty pancreases with p value 0.001. In addition, low-density lipoprotein (LDL), TG and cholesterol was significantly higher in severe fatty pancreases with p value 0.0001, 0.0001 and 0.0001 respectively. High-density lipoprotein (HDL) was the highest in absent fatty pancreases with p value 0.0001. Patient with severe fatty pancrease was only present in fatty liver group with 24%.

Conclusion: Degree of pancreatic steatosis was significantly related to old age, high erythrocyte sedimentation rate, low albumin level, high cholesterol, high triglycerides, and low high-density lipoprotein. Pancreatic steatosis was significantly correlated to presence of fatty liver.

Keywords: Non-alcoholic fatty liver, Non-alcoholic fatty pancreas, Transabdominal ultrasound, Endoscopic ultrasound.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is currently the most common liver disorder, particularly in Western countries. Worldwide, the prevalence of NAFLD is about 25%, with the highest rates reported in South America (31%) and the Middle East (32%), followed by Asia (27%), the USA (24%) and Europe (23%); NAFLD is less common in Africa (14%) *(Younossi et al., 2016)*.

Overall, the prevalence of NAFLD is increasing, particularly in the United States, and it has been projected to become a leading cause of chronic liver disease by 2020 *(Charlton et al., 2011)*. Nonalcoholic fatty pancreas disease (NAFPD) is an excessive lipid accumulation in the pancreas in the absence of significant alcohol intake *(Alempijevic et al., 2017)*.

To date, the pathophysiology of NAFPD remains unclear. There are two potential mechanisms for pancreatic fat accumulation: (i) death of acinar cells, followed by the replacement of adipose tissue; and (ii) intracellular triglyceride accumulation associated with excessive energy balance *(Smits et al., 2011)*.

Nonalcoholic fatty pancreas disease (NAFPD) is usually an incidental finding during transabdominal ultrasound examination. NAFPD may allegedly develop into chronic pancreatitis and further leads to pancreatic cancer *(Hori et al., 2014* and *Rebours et al., 2015)*, and facilitates its dissemination *(Mathur et al., 2011)*.

The ratio of fatty degeneration in pancreas with pancreatic ductal adenocarcinoma (PDAC) was higher than for pancreases without PDAC (72% vs 44%) *(Tomita et al., 2014)*.

Due to its location in the retroperitoneum, the pancreas is notoriously difficult to image by using conventional radiological techniques. The pancreas cannot be fully imaged with transabdominal US because of over lying air in the stomach and small intestine. CT scans can be used to image the pancreas in its entirety, but CT is inaccurate in measuring pancreatic fat. Pancreatic fat deposits primarily in the interlobular septa. This results in a heterogeneous pattern on CT scans, making determination of tissue density (fat) by using Hounsfield units unreliable *(Wang et al., 2014)*.

Because it allows placing the US transducer in close proximity to the pancreas parenchyma, endoscopic ultrasound (EUS) can provide detailed images of the entire pancreas. The use of high-frequency US waves and the ability to simultaneously image adjacent organs like the liver and spleen in real-time *(Lesmana et al., 2015)*.

This study aimed to determine the possible association between non-alcoholic fatty pancreatic disease (NAFPD) and non-alcoholic fatty liver disease (NAFLD).

PATIENTS AND METHODS

This study was conducted on 100 subjects divided into two equal groups as follow: Group I: Included individuals with sonographically proven NAFLD, and Group II (control group): Included healthy individuals with no sonographic evidence

of NAFLD. All cases were selected from Internal Medicine Department at Al-Hussein Hospital, Al-Azhar University during the period from April 2019 to April 2020.

An informed written consents were taken from all participants in this study after explaining the aim for them after obtaining approval of the ethical committee of Al-Azhar Faculty of Medicine.

Inclusion criteria: Age range from 18 - 70 years old, Both sexes, and patients with fatty liver (sonographically proven).

Exclusion criteria: Alcohol consumption ≥20 gram per day in the past year, chronic pancreatitis, nonvisualized pancreas on ultrasound, receiving drugs causing steatosis (amiodarone, glucocorticoids, valproate, tamoxifen, and methotrexate), and patients with liver diseases other than NAFLD.

All subjects were subjected to the following:

A. Full history taking and thorough clinical examination: including measurement of arterial blood pressure and calculation of the body mass index (BMI).

B. Laboratory investigations including:

1. Complete blood count (CBC)
2. Erythrocyte Sedimentation Rate (ESR)
3. Fasting blood glucose level.
4. Liver biochemical tests including:
    i. Alanine Amino Transferase (ALT) (IU/L).
    ii. Aspartate Amino Transferase (AST) (IU/L).
    iii. Alkaline phosphatase (ALP) (IU/L).
    iv. Albumin (ALB) (G/DL).
    v. Gamma-Glutamyl Transpeptidase (GGT) (IU/L).
5. The lipid profile including (total cholesterol, LDL, HDL and triglycerides).

C. Imaging: All subjects were undergoing abdominal and endoscopic ultrasonographical examination.

Diagnostic criteria for fatty liver disease recommended by *Dasarathy et al. (2013)*:

1. Increased hepatic brightness: defined as a homogenously increased echogenicity or hyperechogenicity.
2. Posterior attenuation of the right lobe.
3. The increased contrast between the right kidney and the liver.
4. The loss of visualisation of the right diaphragm.
5. The diminished visibility of the intrahepatic vessels.

Pancreatic parenchyma prospectively graded through EUS. The classification system was adapted from that used by *Marks et al. (2010)* and *Worthen and Beabeau (2010)*. In addition to assessment of pancreatic echogenicity, we also assessed the pancreas for clarity of the parenchyma and pancreatic duct margins.

Grade I was defined as pancreas in which 80% of the parenchyma was hypoechoic or isoechoic when compared with the spleen, the main pancreatic duct was clearly delineated, and fine, “salt and

pepper" dots in the pancreatic parenchyma were clearly seen.

Grade II was defined as pancreas in which 80% of the parenchyma was hyperechoic when compared with the spleen, the main pancreatic duct was clearly delineated, and fine, salt and pepper dots in the pancreatic parenchyma were clearly seen.

Grade III was defined as pancreas in which 80% of the parenchyma was moderately more hyper-echoic as compared with the spleen, the main pancreatic duct margins were moderately obscured, and fine, salt and pepper dots in the pancreatic parenchyma were moderately blurry.

Grade IV was defined as pancreas in which 80% of the parenchyma was severely more hyperechoic when compared with the spleen, the pancreas could not be separated from the adjacent fat, the main pancreatic duct margins were severely obscured, and fine, salt and pepper dots in the pancreatic parenchyma were severely obscured.

Also, diagnostic criteria for fatty pancreas are an increase echogenicity of the pancreatic body parenchymal over that of the kidney *(Wang et al., 2014)*.

Statistical analysis:

Statistical analysis was conducted using SPSS 22th edition, continuous variables were presented using mean ± SD and compared using Mann Whitney U test and Kruskal Wallis test. Categorical variables were presented using frequencies and percentages and compared using Chi X2 test. P value <0.05 was considered significant.

Figure Grading of non-alcoholic fatty pancreas. A) No non-alcoholic fatty pancreas— normal pancreas parenchyma. B) Grade I or lightly non-alcoholic fatty pancreas— echogenicity of pancreas parenchyma greater than that of the kidney. C) Grade II or severely non-alcoholic fatty pancreas—echogenicity of pancreas parenchyma greater than that of the kidney but less than retroperitoneal fat. D) Grade III or highly non-alcoholic fatty pancreas— echogenicity of pancreas parenchyma greater than that of the retroperitoneal fat **(Figure 1)**.

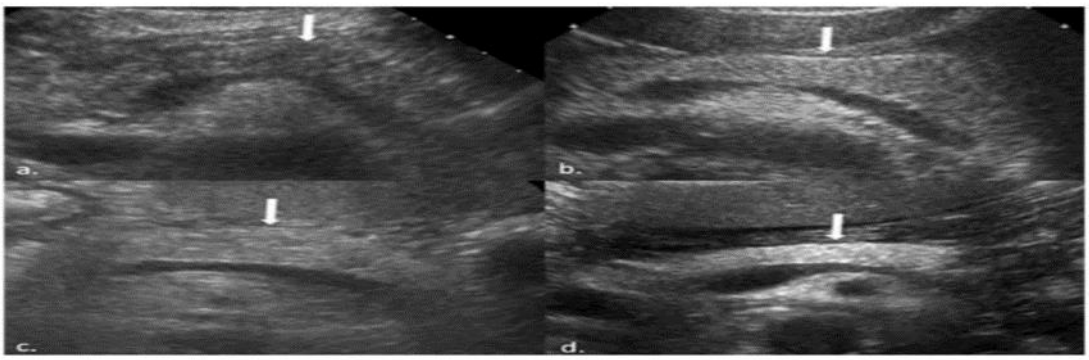
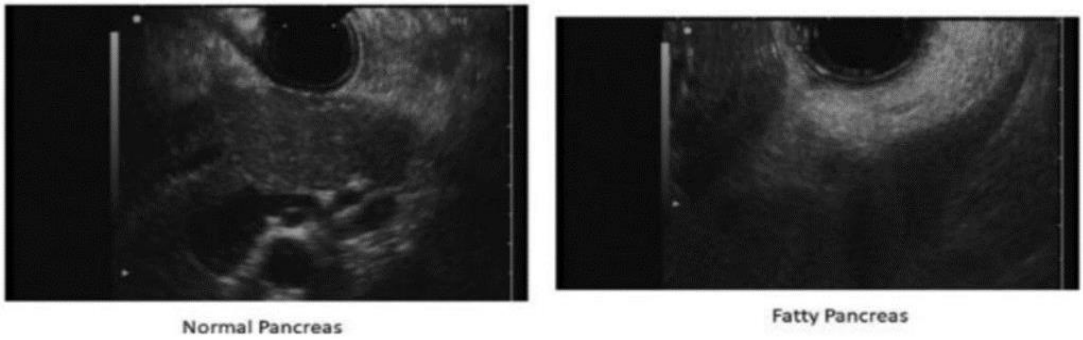


Figure Fat deposition of pancreas on EUS **(Figure 2)**.



RESULTS

Total of 100 participates were recruited in our sample, and divided into two groups fatty liver and control group with a ratio 1:1, They had a mean age 38.3 ± 10.7 years old. There was no gender discrepancy in our sample as females represented 46% and males represented 54%. Using the trans-esophageal Ultrasound has revealed that 24% of the included participants were not having fatty pancreases, while 40% were mild, 24% were moderate and 12% had severe fatty pancreases.

Participants had a mean hemoglobin level 13.3 ± 1.6 gm/dl, mean TLC 7.9 ± 2.1 103/cc, mean platelet count 318.1 ± 76.1 103/cc, mean FBS 120.5 ± 41.6 mg/dl and mean ESR 17.5 ± 4.8 mg/dl. Regarding liver function profile, mean AST 19.6 ± 4.6 mg/dl, mean ALT 21.1 ± 5.0 mg/dl, mean Albumin 4.2 ± 0.5 mg/dl, mean ALP 72.8 ± 23.4 mg/dl and mean GGT 16.0 ± 6.2 mg/dl. Lipid profile showed a mean Cholesterol 181.9 ± 41.5 mg/dl, mean HDL 40.2 ± 6.5 mg/dl, mean LDL 102.9 ± 32.7 mg/dl and mean TG 172.8 ± 65.8 mg/dl **(Table 1)**.

Table (1): Trans-abdominal and trans-esophageal ultrasound features, demographics and laboratory findings of the included participants

Characteristics*	All participants (n=100)	Control	Cases	p-value**
		Fatty liver-Absent (n=50)	Fatty liver-Present (n=50)	
Age (year)	38.3±10.7	33.2±9.5	43.3±9.5	<0.01
Sex (female)	46 (46%)	24(52.2%)	22(47.8%)	0.69
Fasting blood glucose (mg/dl)	120.5±41.6	108.9±32.9	132.0±46.4	<0.01
Erythrocyte sedimentation rate	17.5±4.8	17.4±5.2	17.6±4.4	0.75
Hemoglobin (gm)	13.3±1.6	13.4±1.7	13.2±1.4	0.77
Blood Count				
Total leucocytic count (x1000/L)	7.9±2.1	7.2±1.8	8.5±2.2	<0.01
Platelets (x1000/L)	318.1±76.1	335.3±75.2	301.0±73.6	0.05
Liver Functions				
AST (IU/L)	19.6±4.6	18.9±4.1	20.2±4.9	0.26
ALT (IU/L)	21.1±5.0	20.4±3.3	21.7±6.3	0.16
Albumin (G/DL)	4.2±.5	4.3±0.5	4.0±0.4	<0.01
ALP (IU/DL)	72.8±23.4	73.1±25.9	72.4±20.9	0.72
GGT (IU/DL)	16.0±6.2	15.4±6.3	16.6±6.2	0.39
Lipid Profile				
Total Cholesterol(mg/dl)	181.9±41.5	160.0±28.2	203.7±41.3	<0.01
HDL- Cholesterol(mg/dl)	40.2±6.5	41.7±6.4	38.6±6.3	0.01
LDL- Cholesterol(mg/dl)	102.9±32.7	93.4±33.0	112.4±29.8	<0.01
Triglycerides (mg/dl)	172.8±65.8	154.6±61.9	191.0±65.2	<0.01
Fatty pancreases				
Absent: (Score =0-1)	24 (24.0%)	16 (32.0%)	8 (16.0%)	<0.01
Mild: (Score =2)	40 (40.0%)	24 (48.0)	16(32.0%)	
Moderate: Score =3)	24 (24.0%)	10 (20.0%)	14 (28.0%)	
Severe: Score =4)	12(12.0%)	0 (0.0%)	12 (24.0%)	

*Values expressed as mean±SD or n (%)

** p-value calculated using student T-test for continues variables or Chi X2 for binary variables

ALT= Alanine Amino Transferase; AST= Aspartate Amino Transferase; ALP= Alkaline phosphatase; GGT=Gamma-Glutamyl Transpeptidase.

Based on the severity of fatty pancreases, moderate and sever fatty pancreases was significantly associated with older age with p value 0.0001. While FBS, ESR and AST was significantly higher in moderate and severe fatty pancreases too with p value 0.0001, 0.001 and 0.0001 respectively.

Serum albumin level was the lowest in severe fatty pancreases with p value 0.001. In addition, LDL, TG and Cholesterol was significantly higher in severe fatty pancreases with p value 0.0001, 0.0001And 0.0001 respectively. While HDL was the highest in absent fatty pancreases with p value 0.0001 **(Table 2)**.

	No Score (0-1) (n=24)	**Mild Score (2) (n=40)**	**Moderate Score (3) (n=24)**	**Severe Score (4) (n=12)**	p-value**
Age (year)	27.3±7.7	35.6±5.7	48.9±7	48±7.3	<0.01
Sex (female)	12 (50%)	22 (55%)	8 (33.3%)	4 (33.3%)	0.28
Fasting blood glucose (mg/dl)	97.4±12.5	107.1±26.3	144.9±39.2	162.3±67.8	<0.01
Erythrocyte sedimentation rate	15.6±3.6	16.4±4.5	19.8±5.3	20.5±3.8	<0.01
Hemoglobin (gm)	13.6±1.9	13.1±1.5	13.4±1.6	12.8±1	0.62
Blood Count					
Total leucocytic count (x1000/L)	7.1±1.6	7.7±2	7.9±2.2	10.1±1.6	<0.01
Platelets (x1000/L)	370.1±60.7	310.3±71.2	285±83.6	306.7±55.9	<0.01
Liver Functions					
AST (IU/L)	18.8±4.9	17.9±3.5	21.7±4.9	22.8±3.3	<0.01
ALT (IU/L)	21.5±6.1	20.2±4.2	21.5±4.7	22.3±6.1	0.19
Albumin (G/DL)	4.4±0.4	4.3±0.4	4±0.5	3.8±0.3	<0.01
ALP (IU/DL)	65.7±18.2	75.3±25.3	76.2±24.3	71.8±24.5	0.45
GGT (IU/DL)	13.1±6.3	16.5±5.8	16.5±4.7	19.2±8.2	0.051
Lipid Profile					
Total Cholesterol(mg/dl)	150.1±23.1	164.8±20.9	209.3±31.5	247.5±33.6	<0.01
HDL-Cholesterol(mg/dl)	47.1±5.2	41.2±4.3	35.5±3.6	32.2±1.3	<0.01
LDL- Cholesterol(mg/dl)	77.8±14.4	94.9±28.7	129.7±30.8	126.7±22	<0.01
Triglycerides (mg/dl)	110.2±37.2	157.3±46.1	217.3±49	260.3±38.4	<0.01
Fatty liver (present)	8 (16.0%)	16(32.0%)	14 (28.0%)	12 (24.0%)	<0.01

*Values expressed as mean±SD or n (%)

** p-value calculated using Analysis of Variance (ANOVA) for continues variables or Chi X2 for binary variables. ALT= Alanine Amino Transferase; AST= Aspartate Amino Transferase; ALP= Alkaline phosphatase; GGT=Gamma-Glutamyl Transpeptidase.

Gender distribution was not significantly different between study groups nor severity of fatty pancreases **(Table 3)**.

Table (3): Comparison of gender distribution based on liver and pancreases pathology

Groups \ Gender		Male		Female		P value
		N	%	N	%	
Group	Control	26	48.1%	24	52.2%	0.688
	Fatty liver	28	51.9%	22	47.8%	
Fatty pancreases	No	12	22.2%	12	26.1%	0.286
	Mild	18	33.3%	22	47.8%	
	Moderate	16	29.6%	8	17.4%	
	Severe	8	14.8%	4	8.7%	

Chi X2 test showed significantly different distribution of fatty pancreases severity level based on fatty liver presence with p value 0.001. Patient with severe fatty pancreases was only present in fatty liver group with 24%.

Table (4): Correlation between fatty liver and severity of fatty pancreases

Fatty pancreases \ Groups	Control		Fatty liver		P value
	N	N %	N	N %	
Normal	16	32.0%	8	16.0%	0.001
Mild	24	48.0%	16	32.0%	
Moderate	10	20.0%	14	28.0%	
Severe	0	0.0%	12	24.0%	

Binary regression model showed highly significant correlations between presence of fatty liver and severity of fatty pancreases with Odds ratio 2.45, 95% CI 1.5-4.031 and p value 0.0001 (Table 5).

Table (5): Univariate analysis of binary Logistic Regression Analysis between Fatty Pancreas (dependent factor) and fatty liver (independent factor)

Variable	Odds Ratio (95% Confidence Interval)	p-value
Fatty pancreases	2.46 (1.50, 4.03)	<0.001

Fatty pancreases as dependent factor (present or absent), fatty liver as independent factor.

DISCUSSION

In the current study, participates had a mean age 38.3 ± 10.7 years old. There was no gender discrepancy in our sample as females represented 46% and males represented 54%. Patients who are diagnosed with fatty liver was older in age when compared to control group with mean age 43.3 ± 9.5 in fatty liver groups versus 33.2 ± 9.5 years old in control group with p value 0.0001. These results were similar to studies conducted by

Rosso et al. (2011) and *Paul and Shihaz (2020),* who have identified a significant correlation between fatty steatosis and advanced age. However, age was not a predictor for pancreatic steatosis in another study *(Patel et al., 2013).*

It is reported that the prevalence of pancreatic staetosis increase after the age of 50 years mainly in women, due to increased intake of hormone replacement therapy, oral contraceptives, steroids and antiretrovirals *(Wong et al., 2014).*

However, in our study the mean age of diagnosis was younger that reported in literature, this may be due to hepatitis C virus is endemic in Egypt, however, many national campaigns had been organized to increase the data attained about prevalence and indolent cases of HCV in the last decade *(El-Kassas et al., 2018).*

Hyperechogenic pancreas can be seen in both pancreatic fibrosis and in fatty pancreas. Pancreatic steatosis can be classified into four grades by identifying patterns of pancreas echogenicity in abdominal ultrasound *(Paul and Shihaz, 2020).*

Grade 0: when pancreas and renal echogenicity are similar; grade 1: when pancreas echogenicity is increased and is slightly higher than in the kidney; grade 2: when substantial increase in pancreas echogenicity than renal echogenicity but the retroperitoneal fat echogenicity is more than pancreatic echogenicity; and grade 3: the pancreas echogenicity is ≥ retroperitoneal fat echogenicity *(Lee et al., 2013).*

In the present study, using the abdominal Ultrasound has revealed that 24% of the included participants were not having fatty pancreases, while 40% were mild, 24% were moderate and 12% had severe fatty pancreases.

Findings of the current study revealed that severity of fatty pancreases was significantly associated with older age with p value 0.0001. As well as, FBS, ESR and AST was significantly higher in moderate and severe fatty pancreases too.

These findings were similar to ones reported by *Lee et al. (2013),* who stated that insulin resistance, visceral fat, triglyceride, and alanine aminotransferase (ALT) are higher degree of fat deposition in the pancreas.

Lee et al. (2013) found that the presence of fatty pancreas along with fatty liver concurrently in many cases. They suggested that fatty pancreas might be the initial indicator of "ectopic fat deposition" and as an early marker of insulin resistance, which is a key element of fatty liver and/or metabolic syndrome. *Al-Haddad et al. (2013)* confirmed that hepatic steatosis is the strongest predictor with an odds ratio almost 14 times higher than normal populations.

In the current study, serum albumin level was the lowest in severe fatty pancreases with p value 0.001. In addition, LDL, TG and Cholesterol was significantly higher in severe fatty pancreases, while HDL was the highest in absent fatty pancreases.

These results were consistent with many studies conducted on obese population and metabolic syndrome, as pancreatic steatosis is significantly correlated to obesity, metabolic syndrome and impaired lipid metabolism which is most commonly presented as low HDL

level, hypertriglyceridemia, and hypercholesterolemia *(Olufadi and Byrne, 2012)*.

Conclusion of these studies supported that pancreatic steatosis and its severity is significantly correlated with high LDL level, low HDL, high cholesterol level and high triglycerides *(Musso et al., 2012* and *Van Raalte et al., 2013)*. This was in contrast to study conducted by *Patel et al. (2013),* who reported no significant difference in lipid profile findings based on severity of pancreatic steatosis.

There was a significantly different distribution of fatty pancreases severity level based on fatty liver presence. Patient with severe fatty pancreases was only present in fatty liver group with 24%. This was consistent with a study conducted by *Chang et al. (2018)* who stated that Non-alcoholic fatty liver disease (NAFLD) was found to be positively correlated with pancreatic steatosis with more liability to develop pancreatic cancer.

Our results showed that binary regression model had a significant correlation between presence of fatty liver and severity of fatty pancreases.

Another study revealed that pancreatic steatosis is common in patients with NAFLD and pancreatic fat content positively correlates with liver steatosis grading determined by histology *(Nacif et al., 2018)*.

In addition, patients with histology determined liver fibrosis have significantly less pancreatic fat infiltration than those without evidence of liver fibrosis *(Patel et al., 2013)*. Fatty infiltration in pancreas causes β-cell dysfunction, which may also lead to hepatic steatosis and pancreatic fat also may play a role in the development of non-alcoholic steatohepatitis (NASH) *(Van Geenen et al., 2010)*.

With the increasing prevalence of NAFLD worldwide, pancreatic steatosis will probably also become increasingly common. Pancreatic fat may induce local effects in the liver that affect the progression of NAFLD. Clinicians performing endoscopic ultrasounds have noted a significant prevalence of pancreatic steatosis *(Sepe et al., 2011)*.

Many of these patients may have undiagnosed NAFLD; however, there is little information to guide what clinical management, if any, is required in these patients. There are no data about pancreatic fat in patients with biopsy-proven NAFLD, and this study fills that gap. This study illustrates that there is a strong association between pancreatic fat and liver steatosis. In addition, it suggests that steatosis and lipotoxicity may lead to fibrosis of the pancreas as well as the liver *(Hernando et al., 2012)*.

CONCLUSION

Degree of pancreatic steatosis was significantly related to old age, high ESR, low albumin level, high cholesterol, high triglycerides, and low HDL. Pancreatic steatosis was significantly correlated to the presence of fatty liver.

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دراسة العلاقة بين تدهن الكبد غير الكحولى و تدهن البنكرياس غير الكحولى باستخدام الموجات فوق الصوتيه علي البطن و السونار بالمنظار

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خلفية البحث: يتميز مرض الكبد الدهني غير الكحولي بتراكم الدهون الثلاثية في الكبد وليس بسبب استهلاك الكحول (أقل من 20 جرامًا من الإيثانول يوميًا)، مما يؤدي إلى حدوث تنكس دهني وإلتهاب الكبد. ويعد مرض الكبد الدهني غير الكحولي حاليًا أكثر إضطرابات الكبد شيوعًا، خاصة في الدول الغربية. ويبلغ معدل إنتشار مرض الكبد الدهني غير الكحولي في جميع أنحاء العالم حوالي 25٪. ومن المتوقع أن يصبح سببًا رئيسيًا لمرض الكبد المزمن. ومرض البنكرياس الدهني غير الكحولي هو تراكم مفرط للدهون في البنكرياس في غياب من تناول الكحول بكميات كبيرة. ويمكن أن توفر الموجات فوق الصوتية صورًا مفصلة للبنكرياس بالكامل مع إستخدام الموجات الصوتية عالية التردد والقدرة على تصوير الأعضاء المجاورة مثل الكبد والطحال.

الهدف من البحث: تحديد العلاقة المحتملة بين مرض البنكرياس الدهني غير الكحولي ومرض الكبد الدهني غير الكحولي.

المرضي وطرق البحث: أجريت هذه الدراسة على 100 شخص مقسمين إلى مجموعتين متساويتين: المجموعة الأولى تضمنت أفرادًا ثبتت إصابتهم بمرض الكبد الدهني غير الكحولي، والمجموعة الثانية (المجموعة الضابطة) تضمنت أفرادًا لا يوجد دليل بالموجات فوق الصوتية على الاصابه بالكبد الدهني غير الكحولي. وقد تم أخذ موافقات خطية مستنيرة من جميع المشاركين في هذه الدراسة بعد توضيح الهدف منها. تم عرض بروتوكول الدراسة على لجنة أخلاقيات كلية طب الأزهر وتمت الموافقة عليه.

نتـــائج البحـــث: تـــم تقســـيم المرضـــي إلـــي مجمـــوعتين متســـاويتين: مجموعـــة الكبـــد الـــدهني ومجموعـــة الـــتحكم، وكـــان متوســـط أعمـــارهم 38.3 ± 10.7 ســـنة. حســـب شـــدة البنكريـــاس الـــدهني، وإرتـــبط البنكريـــاس الـــدهني المعتـــدل والشـــديد بشـــكل كبيـــر مـــع تقـــدم العمـــر بقيمـــة 0.0001 p. بينمـــا كـــان ســـكر الـــدم الصـــائم و معـــدل ترســـيب الـــدم و انزيمـــات الكبـــد أعلـــى بشـــكل ملحـــوظ فـــي البنكريـــاس الـــدهني المعتـــدل والشـــديد أيضًـــا بقيمـــة 0.0001 p و 0.001 و 0.0001 علـــى التـــوالي. كـــان مســـتوى الألبـــومين فـــي الـــدم هـــو الأقـــل فـــي البنكريـــاس الـــدهني الحـــاد بقيمـــة 0.001 p. بالإضـــافة إلـــى ذلـــك، كـــان الكوليســـتيرول الضـــار و الـــدهون الثلاثيـــة و الكوليســـتيرول أعلـــى معنويًـــا فـــي البنكريـــاس الـــدهني الحـــاد بقيمـــة 0.0001 p و 0.0001 و 0.0001 علـــى التـــوالي. بينمـــا كـــان الكوليســـتيرول الغيـــر ضـــار الأعلـــى فـــي غائـــب البنكريـــاس الـــدهني بقيمـــة p0.0001. وكـــان المرضـــى الـــذين يعـــانون مـــن البنكريـــاس الـــدهني الشـــديد موجـــودين فقط في مجموعة الكبد الدهني بنسبة 24٪.

الإســـتنتاج: كانـــت درجـــة التـــنكس الـــدهني فـــي البنكريـــاس مرتبطـــة بشـــكل كبيـــر بالشـــيخوخة، وارتفـــاع ســـرعة الترســـيب، وانخفـــاض مســـتوى الألبـــومين، وارتفـــاع الكوليســـترول، وارتفـــاع الـــدهون الثلاثيـــة، وانخفـــاض الـــدهون الغيـــر ضـــاره. ارتـــبط التنكس الدهني في البنكرياس بشكل كبير بوجود الكبد الدهني.

الكلمـــات الدالـــة: الكبـــد الـــدهني غيـــر الكحـــولي، البنكريـــاس الـــدهني غيـــر الكحـــولي، الموجات فوق الصوتية عبر البطن، الموجات فوق الصوتية بالمنظار.