Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2023.242274.2952 Volume 30, Issue 3, May 2024
Manuscript ID ZUMJ-2310-2952 (R1)
DOI 10.21608/ZUMJ.2023.242274.2952
ORIGINAL ARTICLE

Fatty Pancreas; Is It Related to Diabetes Mellitus, Acute Pancreatitis and Pancreatic Cancer.

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Submit Date 12-10-2023
Revise Date 22-10-2023
Accept Date 2023-10-24

ABSTRACT

Background: The accumulation of fat in the pancreas has greater clinical significance. It is unclear, though, how much the intermediate variables influence the fat deposition in these depots. This study aimed to determine the relation of pancreatic steatosis to Diabetes Mellitus, Acute Pancreatitis and Pancreatic Cancer.
Methods: This case-control study was conducted on fifty patients for pancreatic diseases in internal medicine department at Zagazig University Hospitals, equally divided into five groups; group I: 10 cases as a control group, group II: with fatty pancreas (FP), group III: with acute pancreatitis (AP), group IV: with Diabetes Mellitus (DM) and case group V: with pancreatic cancer (PC). Patients were subjected to full medical history, physical examination, laboratory investigations, and non-contrast Computed Tomography (CT).
Results: Age and males were significant increased in group V (P-Value= 0.003 and P=0.019 respectively). Body Mass Index (BMI) (P=0.001) and females (P=0.006) were higher among fatty pancreas patients. Pancreatic steatosis diagnosed by CT in FP representing 100%, in acute pancreatitis 60%, DM 80% and PC 70% of patients, and it was worse in FP group as 40% ware grade 4 (P <0.001). Fatty liver diagnosed by CT in 100% of FP patients, 70% in AP and DM groups and 60% in PC group and was worse in FP group as 80% grade 3 (P <0.001). grades of fatty liver and Low-Density Lipoprotein (LDL) cholesterol significantly independently associated with fatty pancreas (P<0.001). High lipids, diabetes and grades of fatty liver were significantly associated with fatty pancreas (P<0.001) and LDL and fatty liver are independent risk factors (P<0.001).
Conclusion: Pancreatic steatosis is highly related to Diabetes Mellitus, Acute Pancreatitis and Pancreatic Cancer.
Keywords: Pancreatic cancer, Fatty pancreas, Pancreatitis, Diabetes mellitus.

INTRODUCTION

Fatty pancreas (also called pancreatic lipomatosis or Pancreatic steatosis is a term used to describe a variety of diseases, including metabolic syndrome, alcoholism, viral infections, toxins, congenital syndromes, and infiltration of fat into the pancreas, pancreatic inflammation, and development of pancreatic fibrosis [1].

As of right present, the precise prevalence of fatty pancreas is unknown. According to a few epidemiologic research, Asian communities have a prevalence of 16–35% [2].

Hospital-based groups and people with type 2 diabetes mellitus (T2DM) have been found to have a greater prevalence [3].

In Egypt, the annual mortality toll from diabetes is 86,478. The incidence of diabetes among persons

aged 20 to 79 is approximately 15.56%, and the International Diabetes Federation (IDF) calculated in 2013 that 2.2 million Egyptians have pre-diabetes and 7.5 million have diabetes [4]. Insulin-resistant individuals may not be able to meet the increased insulin requirements necessary to develop type 2 diabetes mellitus because of fat accumulation in the pancreatic islets, which results in a decrease in insulin output. Furthermore, in obese non-diabetic participants, a higher percentage of pancreatic fat was linked to elevated insulin levels. This could suggest that the detrimental impact of accumulating pancreatic fat takes a while to show up as decreased β-cell function. It has been determined that pancreatic β-cell damage exists for over ten years prior to the diagnosis of diabetes [5].

Acute pancreatitis (AP) is a common condition with a range of possible clinical consequences. It has a 10–20% mortality rate and can range in severity from a worse, moderate and self-limited illness. Because more active therapy is needed for these individuals, it is important to predict severe cases [6].

Severe Acute Pancreatitis (SAP) was described as acute pancreatitis accompanied by organ dysfunction or localised or regional consequences by the Atlanta Symposium. The majority of SAP deaths are caused by sepsis and multiple organ failure. Patients with sterile necrosis have a mortality rate of 10-12%, but those with infected pancreatic necrosis have a 25–30% mortality rate [7].

Intracellular lipids may cause lipotoxicity and consequent damage to islet or acinar cells, while fatty pancreas may be involved in β-cell lipotoxicity and lipoapoptosis. Adipocytes may also have a paracrine effect on the function of acinar and islet cells [8].

Since the 1990s, the incidence of pancreatic cancer has increased. Considering that around 90% of pancreatic cancer cases are incurable at the time of diagnosis and that early identification is essential to reducing the disease's burden [9].

An increasing intrapancreatic fatty deposit (IPFD) during follow-up in patients with intraductal papillary mucinous neoplasm and concomitant pancreatic duct adenocarcinoma (PDAC) may be very useful [10]. The purpose of this study was seeking to ascertain how pancreatic steatosis related to acute pancreatitis, diabetes mellitus, and pancreatic cancer.

METHODS

After taking approval from Institutional Review Board (IRB# 10215-7-12-2022) Zagazig University and written informed consent was obtained from all participants, this case-control study was conducted in Internal Medicine Department at Zagazig University Hospitals, between February 2023 to July 2023. on 40 patients with pancreatic diseases and 10 cases as control group. The study was done according to the Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Inclusion criteria were Age > 18 years. Individuals with pancreatic cancer and fatty pancreas identified by CT. Patients who arrived at the gastrointestinal clinic with upper abdomen pain that was not explained and that was thought to have a pancreatic source. Disqualifying factors: patients who are younger than eighteen. Previous surgery on the pancreas. Patients who consume alcohol. Individuals whose pancreatic boundary is unclear.

The sample was divided into four groups (10 subjects in each group) in addition to 10 subjects as a control group: Group I: Healthy control cases. Group II: Patients with fatty pancreas. Group III: Patients have acute pancreatitis (upper abdominal pain suspected to be pancreatic origin). Group IV: Patient diseased with diabetes mellitus (diagnosed by FBS and Glycosylated Haemoglobin (HbA1c)). Group V: Patient diseased with pancreatic cancer. (diagnosed by CT, Carcino-embryonic antigen (CEA) and Serum cancer antigen 19-9 (CA19-9).

All Patients underwent history taking with special emphasizing on age, sex, occupation, history of drugs causing fatty pancreas like steroid and octreotid, smoking, alcohol. Current symptoms as epigastric pain, vomiting, history of obstructive jaundice, fever, weight loss and diarrhea. Past medical and surgical history. Family history. Complete physical examination including abdominal examination for epigastric tenderness, abdominal masses, and lymph nodes, calculating blood pressure reading and body mass index (BMI). Laboratory Investigations; Complete Blood Count (CBC) with differential count. Total cholesterol, triglycerides, High Density Lipoprotein (HDL) and low-density lipoprotein (LDL). Fasting Blood Sugar (FBS) and HbA1C. Amylase, Lipase. CA19-9, and CEA in suspected pancreatic cancer (focal lesion on CT).

Non contrast computed tomography (CT) was used to diagnose diagnosis and grading of pancreatic cancer, fatty pancreas and fatty liver using multislice CT scanner (Canon Prime Aquilion SP). Fatty pancreas was graded Body mass index (BMI) and bl by CT scan were categorised into five grades (grades 0-4) based on the location of pancreatic involvement. Grade 0: Unaltered look devoid of fat substitution. Grade 1:

Less than 25% of the specified pancreatic area is infiltrated by fat. Grade 2: 25%–50% of a specific pancreatic area was replaced with fat. Grade 3: 50%–75% of a specific pancreatic area is replaced by fat. Grade 4: Associated with fatty infiltration encompassing over 75% of a specific pancreatic area measured for blood pressure [11].

Computed tomography (CT) was used to diagnosis of Fatty Liver Through visual comparison of the vascular and liver attenuation on non-contrast computed tomography (CT), it serves as a straightforward and non-invasive diagnostic tool. a visual grading system based on CT scans that compares the liver's brightness to that of the hepatic vessels (grades 0-4) as per; Grade 0: less than one-third of the liver has no or very little margin blurring, and the hepatic arteries have less attenuation than the hepatic parenchyma. Grade 1: Hepatic arteries with margin blurring in more than one-third of the liver, but with less attenuation than hepatic parenchyma. Grade 2: Hepatic parenchyma and hepatic vessels exhibit the same attenuation. Hepatic vessels in Grade 3 exhibit more attenuation than hepatic parenchyma [12].

STATISTICAL ANALYSIS

For statistical analysis, IBM Inc., Armonk, NY, USA, used SPSS v26. The Shapiro-Wilks test and histograms were employed to determine the normality of the information distribution. Quantitative parametric data were presented as mean and standard deviation (SD) and were analysed by ANOVA (F) test with post hoc test (Tukey). Quantitative non-parametric data were presented as median and interquartile range (IQR) and were analysed by Kruskal-Wallis test with Mann Whitney-test to compare each group. Qualitative variables were presented as frequency and percentage (%) and were analysed utilizing the Chi-square test. A two tailed P value ≤ 0.05 was considered statistically significant.

RESULTS

In table (1) Age was significantly higher in group V (P4 =0.003, post hoc analysis) with 90% ware males. BMI was higher in group II (P <0.001) with female predominance 70%. Smoking and HTN were insignificantly different between the studied groups.

In table (2), WBCs (P <0.001), Lipase and amylase were markedly higher in group III (P5, P8 and P9<0.001. post hoc analysis) Compared to the other groups, group III and group IV had a significantly higher FBS (P2, P5, P9<0.001, P3 =0.005 and P8 =0.003. post hoc analysis). HbA1c considerably increased in group IV (P3 =0.003, post hoc analysis). Group II had much higher levels of TG (P1 =0.011), LDL (P1 =0.005), and total cholesterol (P1 =0.001 post hoc test analysis), while a significantly lower levels of HDL in group II than other groups (P1 =0.047).

In table (3), in group V, CEA and CA19.9 are raised among those patients.

Table (4) Fatty pancreas present in 60% of acute pancreatitis patients, 80% in diabetic patients, 70% in pancreatic cancer patients.

Table (5) Fatty liver present in 100% of fatty pancreas patients (with 80% had high grade fatty liver grade 3), 70% in acute pancreatitis and diabetic patients and 60% in pancreatic cancer patients.

Table (6) showed a strong correlation between fatty pancreas and fatty liver by CT (P <0.00).

.In table (7) among all patients, there was a statistically significant positive correlation between grades of fatty pancreas and all of BMI (r= 0.559, P <0.001), total cholesterol (r= 0.755, P <0.001), triglycerides (r =0.826, P <0.001), LDL cholesterol (r =0.739, P <0.001), serum lipase (r =0.341, P <0.036), fasting blood glucose (r =0.36, P =0.027), HbA1c (r =0.632, P <0.001), and grades of fatty liver (r =0.858, P <0.001), and a statistically significant negative link between HDL cholesterol and grades of fatty pancreas (r= -0.464, P =0.003), and with analysis using linear stepwise regression of factors correlated with grades of fatty pancreas, only grades of fatty liver and LDL cholesterol significantly independently associated with it (P <0.001) (Ts 1).

Table (1): Demographic data of the studied groups

		Group I (n=10)	Group II (n=10)	Group III (n=10)	Group IV (n=10)	Group V (n=10)	P value
Age (years)	**Mean± SD**	34.7± 7.94	43.5±6.1	46.8 ± 5.69	42.4±17.38	51.7±6.33	**0.006***
	Range	22 - 45	38 – 55	39 - 55	19 – 65	43 - 65	
	P value	P1 =0.274, P2 =0.058, P3 =0.405, P4 **=0.003***, P5 =0.941, P6 =0.999, P7 =0.342, P8 =0.850, P9 =0.793, P10 =0.224					
Sex	**Male**	4 (40%)	3 (30%)	4 (40%)	3 (30%)	9 (90%)	**0.038***
	Female	6 (60%)	7 (70%)	6 (60%)	7 (70%)	1 (10%)	

	P value	P1 =0.639, P2 =1.000, P3 =0.639, P4 **=0.019***, P5 =0.639, P6 =1.000, P7 **=0.006***, P8 =0.639, P9 **=0.019***, P10 **=0.006***					
BMI (kg/m^2)	**Mean ± SD**	23.3 ± 2	30.4±3.2	26.2 ± 2.97	22.4 ± 3.06	21.4±2.07	**<0.001***
	Range	20 - 26	26 – 35	22 - 33	18 – 27	19 - 25	
	P value	P1 **<0.001***, P2 =0.136, P3 =0.945, P4 =0.526, P5 **=0.010***, P6 **<0.001***, P7 **<0.001***, P8 **=0.024***, P9 **=0.002***, P10 =0.922					
Smoking	**Yes**	4 (40%)	6 (60%)	2 (20%)	3 (30%)	5 (50%)	0.384
	No	6 (60%)	4 (40%)	8 (80%)	7 (70%)	5 (50%)	
HTN	**Yes**	1 (10%)	4 (40%)	5 (50%)	4 (40%)	3 (30%)	0.393
	No	9 (90%)	6 (60%)	5 (50%)	6 (60%)	7 (70%)	

ANOVA (F) test with post hoc test (Turkey) and Chi-square test

BMI: body mass index, HTN: hypertension, P1: P value between group I and II, P2: P value between group I and III, P3: P value between group I and IV, P4: P value between group I and V, P5: P value between group II and III, P6: P value between group II and IV, P7: P value between group II and V, P8: P value between group III and IV, P9: P value between group III and V, P10: P value between group IV and V, *: significant as P value ≤ 0.05

Table (2): Laboratory investigations of the studied groups

	Group I (n=10)	**Group II (n=10)**	**Group III (n=10)**	**Group IV (n=10)**	**Group V (n=10)**	**P value**	**Normal range**
WBCs (*10^9/L)	7.15±2.06	7.63 ± 2.11	14.53±1.43	7.24±1.23	10.86±2.08	**<0.001***	
	P1 =0.976, P2 **<0.001***, P3 =1.000, P4 **<0.001***, P5 **<0.001***, P6 =0.989, P7 **=0.002***, P8 **<0.001***, P9 **<0.001***, P10 **<0.001***						
Lipase (U/L)	118.7±32.95	152.7±31.71	395.8±93.79	138.9±32.01	235.8±61.55	**<0.001***	**Up to 115**
	P1 =0.658, P2 **<0.001***, P3 =0.927, P4 **<0.001***, P5 **<0.001***, P6 =0.981, P7 **=0.015***, P8 **<0.001***, P9 **<0.001***, P10 **=0.003***						
Amylase (U/L)	98.8 ± 9.38	109.2 ± 8.87	334.8±130.53	92.5 ± 28.88	137.7±16.95	**<0.001***	**Up to 210**
	P1 =0.995, P2 **<0.001***, P3 =0.999, P4 =0.608, P5 **<0.001***, P6 =0.972, P7 =0.829, P8 **<0.001***, P9 **<0.001***, P10 =0.463						
FBS (mg/dL)	102 ± 10.81	122.3±15.88	201.2 ± 51.24	150.2±28.78	138.4±21.57	**<0.001***	**70-110**
	P1 =0.536, P2 **<0.001***, P3 **=0.005***, P4 =0.058, P5 **<0.001***, P6 =0.225, P7 =0.734, P8 **=0.003***, P9 **<0.001***, P10 =0.895						
HbA1c (%)	5.79 ± 0.26	6.89 ± 0.43	6.56 ± 0.59	7.45 ± 1.87	6.3 ± 0.55	**0.004***	**4.5-6.4**
	P1 =0.084, P2 =0.367,P3 **=0.002***, P4 =0.743, P5 =0.933, P6 =0.672, P7 =0.628, P8 =0.230, P9 =0.971, P10 =0.064						
Cholesterol (mg/dL)	140.5±33.11	264.6±51.11	220.3 ± 47.43	196.2±98.52	211 ± 65.12	**0.002***	**<200**
	P1 **<0.001***, P2 =0.052, P3 =0.295, P4 =0.109, P5 =0.524, P6 =0.128, P7 =0.332, P8 =0.912, P9 =0.997, P10 =0.984						
TG (mg/dL)	112.2±17.31	224.9±43.23	177.5 ± 48.17	184.2±130.78	177.74±74.16	**0.028***	**<150**
	P1 **=0.011***, P2 =0.290, P3 =0.203, P4 =0.287, P5 =0.606, P6 =0.730, P7 =0.610, P8 =1.000, P9 =1.000, P10 =1.000						
HDL (mg/dL)	54.6±6.22	39.6±11.14	42.8±15.49	50.5±8.95	43.3±14.24	**0.038***	**35-60**
	P1 **=0.047***, P2 =0.179, P3 =0.934, P4 =0.215, P5 =0.973, P6 =0.246, P7 =0.954, P8 =0.587, P9 =1.000, P10 =0.647						
LDL (mg/dL)	113.8±18.46	187.1±35.89	144.2 ± 52.75	160.6 ± 66.47	146.88±32.89	**0.013***	**60-130**
	P1 **=0.005***, P2 =0.551, P3 =0.148, P4 =0.468, P5 =0.216, P6 =0.674, P7 =0.273, P8 =0.922, P9 =1.000, P10 =0.958						

ANOVA (F) test with post hoc test (Turkey)

P1: P value between group I and II, P2: P value between group I and III, P3: P value between group I and IV, P4: P value between group I and V, P5: P value between group II and III, P6: P value between group II and IV, P7: P value between group II and V, P8: P value between group III and IV, P9: P value between group III and V, P10: P value between group IV and V, *: significant as P value ≤ 0.05

Table (3): CEA and CA19-9 of Group V (n=10)

		Group V (n=10)
CEA (ng/mL)	**Mean ± SD**	7.76 ± 1.05
	Range	0 - 2.9 ng/mL
CA19-9 (U/mL)	**Mean ± SD**	83.4 ± 6.35
	Range	< 37 U/mL

*CEA Carceno Emberyonic Antigen. CA19.9 Cancer Antigen 19.9.

Table (4): Pancreatic steatosis grading by CT of the studied groups

	Group I (n=10)	Group II (n=10)	Group III (n=10)	Group IV (n=10)	Group V (n=10)	P value
Grade 0	10 (100%)	0 (0%)	4 (40%)	2 (20%)	3 (30%)	**<0.001***
Grade 1	0 (0%)	0 (0%)	2 (20%)	1 (10%)	6 (60%)	
Grade 2	0 (0%)	0 (0%)	1 (10%)	2 (20%)	1 (10%)	
Grade 3	0 (0%)	6 (60%)	3 (30%)	4 (40%)	0 (0%)	
Grade 4	0 (0%)	4 (40%)	0 (0%)	1 (10%)	0 (0%)	
P1 <0.001*, P2 =0.035*, P3 =0.010*, P4 =0.005*, P5 =0.017*, P6 =0.125, P7 =0.001*, P8 =0.649, P9 =0.162, P10 =0.059						

Chi-square test *: significant as P value ≤ 0.05

P1: P value between group I and II, P2: P value between group I and III, P3: P value between group I and IV, P4: P value between group I and V, P5: P value between group II and III, P6: P value between group II and IV, P7: P value between group II and V, P8: P value between group III and IV, P9: P value between group III and V, P10: P value between group IV and V, *: significant as P value ≤ 0.05

Table (5): Fatty liver by CT of the studied groups

	Group I (n=10)	Group II (n=10)	Group III (n=10)	Group IV (n=10)	Group V (n=10)	P value
Grade 0	10 (100%)	0 (0%)	3 (30%)	3 (30%)	4 (40%)	**<0.001***
Grade 1	0 (0%)	0 (0%)	2 (20%)	2 (20%)	5 (50%)	
Grade 2	0 (0%)	2 (20%)	2 (20%)	2 (20%)	0 (0%)	
Grade 3	0 (0%)	8 (80%)	3 (30%)	3 (30%)	1 (10%)	
P1 <0.001*, P2 =0.013*, P3 =0.013*, P4 =0.014*, P5 =0.064, P6 =0.064, P7 =0.001*, P8 =1.000, P9 =0.219, P10 =0. 219						

Chi-square test *: significant as P value ≤ 0.05

P1: P value between group I and II, P2: P value between group I and III, P3: P value between group I and IV, P4: P value between group I and V, P5: P value between group II and III, P6: P value between group II and IV, P7: P value between group II and V, P8: P value between group III and IV, P9: P value between group III and V, P10: P value between group IV and V, *: significant as P value ≤ 0.05

Table (6): Association between fatty pancreas and fatty liver by CT of the studied patients (n =50)

Fatty liver	Fatty pancreas					P value
	Grade 0 (n=19)	Grade 1 (n=9)	Grade 2 (n=4)	Grade 3 (n=13)	Grade 4 (n=5)	
Grade 0 (n=20)	18 (94.7%)	1 (11.1%)	0 (0%)	1 (7.7%)	0 (0%)	**<0.001***
Grade 1 (n=9)	1 (5.3%)	5 (55.6%)	1 (25%)	2 (15.4%)	0 (0%)	
Grade 2 (n=6)	0 (0%)	2 (22.2%)	0 (0%)	3 (23.1%)	1 (20%)	
Grade 3 (n=15)	0 (0%)	1 (11.1%)	3 (75%)	7 (53.8%)	4 (80%)	

Chi-square test *: significant as P value ≤ 0.05

Table (7) Correlation between fatty pancreas patients and the studied parameters among all patients (n=40):

	r	p
Age (year)	0.146	0.381
BMI (kg/m^2)	**0.559**	**<0.001****
Cholesterol (mg/dl)	**0.755**	**<0.001****
Triglycerides (mg/dl)	**0.826**	**<0.001****
LDL cholesterol (mg/dl)	**0.739**	**<0.001****
HDL cholesterol (mg/dl)	**-0.464**	**0.003***
Lipase (U/L)	**0.341**	**0.036***
Amylase (U/L)	0.124	0.458
FBG(mg/dl)	**0.36**	**0.027***
HbA1c (%)	**0.632**	**<0.001****
CEA	0.234	0.578
CA 19-9	0.577	0.134
Grades of fatty liver	**0.858**	**<0.001****

r Spearman rank correlation coefficient *p<0.05 is statistically significant
**p≤0.001 is statistically highly significant

DISCUSSION

In the present study, the age and male sex were significantly higher in PC group (Mean age 51.7±6.33) than other groups, while in fatty pancreas and diabetes mellitus groups there was female predominance as it is related to sedentary life, obesity and hyperlipidaemia and lack of exercise.

The relationship between pancreatic steatosis and type 2 diabetes mellitus, obesity, and chronic pancreatitis (CP) showed an association by Tirkes et al [13]. The distribution of sex among patients with and without T2DM and CP was equal. Patients with and without T2DM were similar in age, while those in the CP group were older.

BMI was significantly increased in fatty pancreas group than in other groups. it was mainly related to hyperlipidaemia and fat deposition in different body organs.

In a study by Tirkes et al. (2019) showed that pancreatic fat fraction showed a moderate positive correlation with visceral adipose tissue (VAT) (r = 0.54). and the correlation of pancreatic fat with the subcutaneous adipose tissue (SAT)was weak (r = 0.23) and visceral-to subcutaneous adiposity ratio (V/S) (r = 0.26).

In disagreement with our study, Hoogenboom et al [14] in a study on PDAC patients, showed that in total 32 cases were matched to 117 controls, the mean BMI of the patients and controls were in the overweight range (p = 0.723; 29.6 and 29.2, respectively), (21.4 in our study). In the same study, 71.9% of patients had a fatty pancreas, compared to 45.3% of controls (70% in our study). FBS was considerably higher in the DM group than in the control group and significantly higher in the AP group than in the other groups. In DM group, FBS and HbA1c was noticeably higher group than control group.

Corresponding to our research, Sharma et al [15] on a CP and non-Cancer groups showed a relative hyperglycemia among cancer patients. The study shows association of FBG level in relation to tumour volume, grade, and time to PDAC diagnosis. Individuals experience hyperglycemia on average for 36 to 30 months prior to PDAC diagnosis; this information should be included in early detection efforts.

Compared to other groups, group II had significantly higher levels of cholesterol, TG, and LDL and significantly lower levels of HDL. Also compared to other groups, group II had significantly higher levels of cholesterol, TG, and LDL and significantly lower levels of HDL. Consistent with our research,

Melitas et al [16] revealed that the pancreatic steatosis group had greater triglycerides than the control group.

In group V (Patient have pancreatic cancer), CEA and CA19.9 were raised among those patients.

In agreement with our study, Sharma et al [15] showed that CA19-9 was higher in CP than control group.

Pancreatic steatosis grading by CT showed pancreatic steatosis present in 60% of acute

pancreatitis patients, 80% in diabetic patients, 70% in pancreatic cancer patients.

Hoogenboom et al [14] showed 71.9% and 45.3%, among PDAC patients and controls respectivly had pancreatic steatosis on CT (p = 0.0094).

Fatty liver by CT showed fatty liver present in 100% of fatty pancreas patients (80% of them had high grade fatty liver, grade 3), 70% in acute pancreatitis and diabetic patients and 60% in pancreatic cancer patients.

IN consistence with our research, Tirkes et al [13] showed that chronic pancreatitis patients had higher fatty liver compared with the non-chronic pancreatitis group. A higher fatty liver was also seen in patients with T2DM compared with those without T2DM.

The current investigation found a strong correlation between fatty liver and fatty pancreas. by CT.

Melitas et al [16] found that pancreatic fat is closely related to metabolic syndrome and hepatic steatosis but its effect has been poorly understood, and its clinical implications remain vaguely defined, it is likely that the entity of metabolic syndrome with features of pancreatic steatosis and hypertriglyceridemia with their associated manifestations of chronic pancreatitis, including ductal lithiasis, will be widely appreciated.

On a Correlation between fatty pancreas patients and the studied parameters among all patients, There was statistically significant positive correlation between fatty pancreas and all of BMI, lipid profile, fasting blood glucose, HbA1c and grades of fatty liver and on doing a linear stepwise regression analysis of the positive factors only grades of fatty liver and LDL cholesterol significantly independently associated with it, indicating that increased BMI and lipids results in ectopic lipid deposition in tissues, such as the heart, skeletal muscles, liver, and pancreas.

Tirkes et al [13] investigation, which supported our findings, demonstrated a relationship between elevated BMI, abdominal obesity, and pancreas fat.

The major limitation of our study was inability to assess the diseases histologically thereby inability to correlate our CT findings with histopathological findings, but this is not possible anyway for most patients due to limited facility and risky procedure. Also, the assessment of the disease requires a larger sample size. A strength of our study was its design, which provided us with the opportunity to employ CT as a readily available tool for assessing fatty pancreas in all patients at all stages. The results of this study will provide ideas for new research, and further studies are needed with a larger number of patients.

CONCLUSION

Acute pancreatitis, pancreatic cancer, and diabetes mellitus are closely associated with pancreatic steatosis.

Author Contributions:

H.I.M: Conception and design, drafted the manuscript, data collection. M.A.I: radiological data collection and interpretation. S.J.Z: interpretation of data, critically reviewed the manuscript and language logistics. N.F.I: Conception and study design, interpretation of data, critically reviewed the manuscript. All authors provided final approval for submission and publication.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Funding information

None, declared.

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(Ts 1): Linear stepwise regression analysis of factors significantly associated with fatty pancreas (n=40):

	Unstandardized Coefficients		Standardized Coefficients	t	P	95.0% Confidence Interval	
	B	Std. Error	Beta			Lower	Upper
(Constant)	-1.135	0.351		-3.232	0.003*	-1.848	-0.422
Grades of fatty liver	0.765	0.095	0.658	8.060	<0.001**	0.572	0.957
LDL cholesterol	0.012	0.003	0.378	4.627	<0.001**	0.007	0.017

*p<0.05 is statistically significant **p≤0.001 is statistically highly significant

To Cite :

Zaghloul, S., Mohammed Ashour, H., Amine Ibrahim, M., Ibrahim, N. Fatty Pancreas; Is it related to Diabetes Mellitus, Acute Pancreatitis and Pancreatic Cancer.. *Zagazig University Medical Journal*, 2024; (851-858): -. doi: 10.21608/zumj.2023.242274.2952