The relation of CT quantified pancreatic fat index with visceral adiposity and hepatic steatosis

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ABSTRACT

Objective: The purpose of this study was to investigate the relation between pancreatic steatosis and visceral adiposity. Furthermore, the study sought to explore the association between pancreatic steatosis, pancreas volume, hepatic steatosis, age, and sex in adults without prior history of pancreatic disease. The research also served to define a cut-off value of visceral fat tissue area (VFA) predicting fatty pancreas.

Material and Methods: CT scans of 98 living-liver donor transplant patients without prior history of pancreatic disease were evaluated for the presence of fatty pancreas. Pancreas volume, VFA, subcutaneous-total FA, VFA/TFA ratios of the patients with and without fatty pancreas were quantified with a semi-automated model on CT. Coexistence of hepatic steatosis was also recorded.

Results: VFA, TFA and VFA/TFA were significantly greater in the fatty group (p< 0.001, p< 0.001, p< 0.001; respectively), and pancreatic steatosis was moderately correlated with VFA, VFA/TFA and TFA with the highest correlation coefficient with VFA (r= -0.715, r= -0.605, r= -0.573, respectively; p< 0.001 for all). A cut-off value of VFA ≥ 107.2 cm^2 estimates pancreatic steatosis with a sensitivity and specificity of 90% (95% CI= 77-96%) and 87.9% (95% CI= 77%-94%), respectively. Pancreas volume was higher in the fatty-group with a mean value of 86.5 ± 17.3 mL (range: 58-119.2 mL, p= 0.097). In multiple logistic regression analyses, pancreatic steatosis was significantly associated with VFA and the male sex (OR= 58.2, 95% CI= 12.2-277.1, p< 0.001; OR= 11.4, 95% CI= 2.1-63.4, p< 0.001; respectively). 77.5% of the fatty pancreas subjects had co-existing hepatic steatosis.

Conclusion: Pancreatic steatosis is related to higher VFA, VFA/TFA and hepatic steatosis. A cut-off value of VFA ≥ 107.2 cm^2 may predict pancreatic steatosis.

Keywords: Pancreas, lipomatosis, liver steatosis, multislice computed tomography, visceral obesity, organ volume

INTRODUCTION

Pancreatic lipomatosis, pancreatic steatosis or fatty pancreas is the accumulation of free fat acids and triglycerides into the pancreatic islet and acinar cells, but preferentially into the interstitium (1-3). The pancreas is a glandular organ, which has both exocrine (acinar and ductal cells) and endocrine (islet cells) functions. Eighty percent of the gland volume is composed of the exocrine component (4). There are various nomenclatures to identify an accumulation of fat including pancreatic lipomatosis, pancreatic steatosis, fatty infiltration, lipomatous pseudohypertrophy, non-alcoholic fatty pancreatic disease, and fatty replacement. However, the term 'fatty replacement' expresses the irreversible damage of glandular islands and the replacement of those with adipocytes (5). Many of those with limited pancreatic steatosis have no major clinical symptoms. In advanced cases, it may lead to exocrine insufficiency and cause clinical symptoms like chronic diarrhea, steatorrhea and weight loss (4). Some studies assume that pancreatic steatosis might cause ductal stones and pancreatitis. In addition, it has been indicated that inflammation due to increased oxidative stress raised by free fat acids metabolism may lead to fibrosis and malignancy in the pancreatic tissue (3,5,6).

There is no single etiologic factor for pancreatic steatosis. It has been assumed that the pathologies related to the pancreas ductal system such as the intraductal calculi and pancreatic tumors may cause fatty pancreas. Pancreatic steatosis also correlates with diabetes mellitus, pancreatitis and the metabolic syndrome, which present the triad of the following features; hyperinsulinemia, hypertension, hyper-

glycemia, hypercholesterolemia, and obesity (4,7). This entity is highly associated with obesity, and it is assumed that visceral fat tissue is a better indicator and predictor of pancreatic steatosis rather than BMI (8).

Computed tomography (CT) is the first line imaging modality used to diagnose suspected pancreas disease. Pancreatic fat index, which shows the difference between mean pancreatic and splenic attenuation (P-S) value and quantified on non-enhanced CT, has been shown to be an indicator of pancreatic steatosis. It has been histologically demonstrated that P–S is significantly correlated with the amount of pancreatic fat component (9). In comparison with other imaging modalities, CT can measure the pancreas volume and amount of abdominal fat component easily and accurately. Instead of two-point measurements, pancreas volume can be measured more accurately with new automatic segmentation models on CT as well as fat tissue quantification. The aim of this study was to investigate the relation between pancreatic steatosis and visceral adipose tissue and to define a cut-off value of visceral fat tissue area on CT, which might show pancreatic steatosis. Additionally, the study also aimed to explore the relation between pancreas steatosis, pancreas volume, hepatic steatosis, age, and sex in adults without prior history of pancreatic disease. To the best of our knowledge, the cut-off value of visceral tissue area for the pancreas steatosis is presented for the first time.

MATERIAL and METHODS

Study Population

This retrospective study was approved by the Clinical Research Ethical Committee of our institution (Ankara University, School of Medicine; ref no: i4-224-20, date: 22.04.2020). CT scans of living-liver donor transplant patients and the institution's radiology information system/picture archiving and communication system (Centricity 5.0 RIS-i, GE Healthcare, Milwaukee, WI) were utilized to identify study population. One hundred and three patients, without chronic alcohol consumption or chronic diseases (diabetes mellitus-DM) in their medical records, examined between December 2016 and December 2018 were included into the study. Patients with acute-chronic pancreatitis, pancreatic tumors, previous pancreatic surgery, ductal dilatation, diffuse or more than one speckled parenchymal calcification or ductal calcifications were excluded from the study. Those etiologic factors were deemed in the literature as factors that induce pancreatic steatosis and affect pancreas volume (4). After addressing all of the exclusion criteria, 98 patients without pancreatic disease were included into the study.

CT Acquisition Parameters and Assessment of Pancreas-Hepatic Steatosis and Volume

All examinations were performed on a 64-detector (Toshiba Aquilion 64, Otowara, Japan) scanner. In our institution, living-liver donor CT protocol consists of non-contrast images followed by arterial,

portal, and hepatic venous phases. The post-contrast series were obtained with scan delay times of 30 seconds (arterial phase), 75 seconds (portal phase) and 180 seconds (hepatic venous phase) after the start of IV contrast agent administration. A volume of 1.5-2 mL/kg of intravenous (IV) non-ionic iodinated contrast agent (350 mg/mL Omnipaque; GE Healthcare, Oslo, Norway) and 40 mL of saline were injected via antecubital vein with an 18-gauge cannula at a rate of 4 mL/s. The following CT scan parameters were performed: 120 kVp, collimation of 64 x 0.5, 1 mm section thickness, 0.8 mm reconstruction interval. An automatic exposure control system was used with a range from 100 to 400 mA.

Pancreatic steatosis and hepatic steatosis were assessed by non-enhanced axial CT images (Figure 1). The pancreatic parenchymal attenuation was measured from five different sections (head, corpus, tail, neck and uncinate process) by using an area of 0.5 cm^2 region of interest (ROI). Splenic attenuation was acquired from three different regions by using 1 cm^2 of ROI. Mean CT attenuation value of the splenic-pancreatic parenchyma were calculated by using the average of those measurements. Vascular structures were avoided in the measurements. Care was taken not to involve the peripheral section of the pancreas to avoid partial volume effect. The measurements were performed close to the splenic vein in patients in whom the margins of the pancreas were poorly defined from the adjacent retroperitoneal fat. The difference between the pancreas-splenic attenuation was utilized to determine pancreatic steatosis. Pancreatic steatosis (fatty pancreas group) was defined if the difference was ≤ -5 Hounsfield Unit (HU) (10). The patients with pancreas-splenic attenuation difference > -5 HU comprised the non-fatty pancreas group. Liver attenuation index (LAI), which estimates hepatic steatosis in non-enhanced CT, is the difference between mean hepatic parenchymal attenuation value and mean splenic atten-
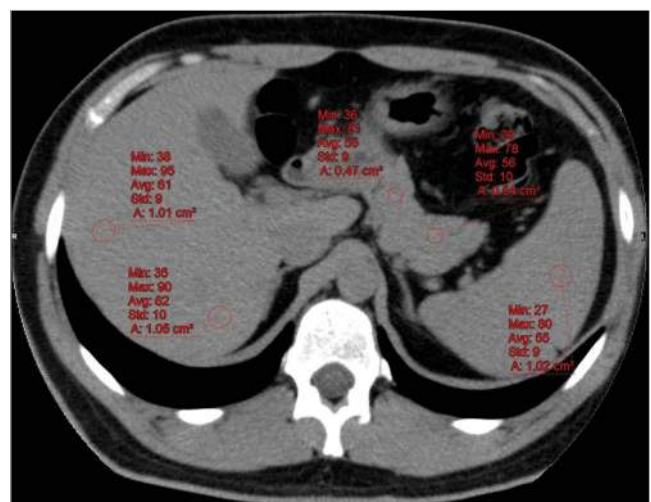

Figure 1. The measurement of mean attenuation values of the liver, spleen and pancreas parenchyma on non-enhanced CT in different sections by using region of interest (ROI).
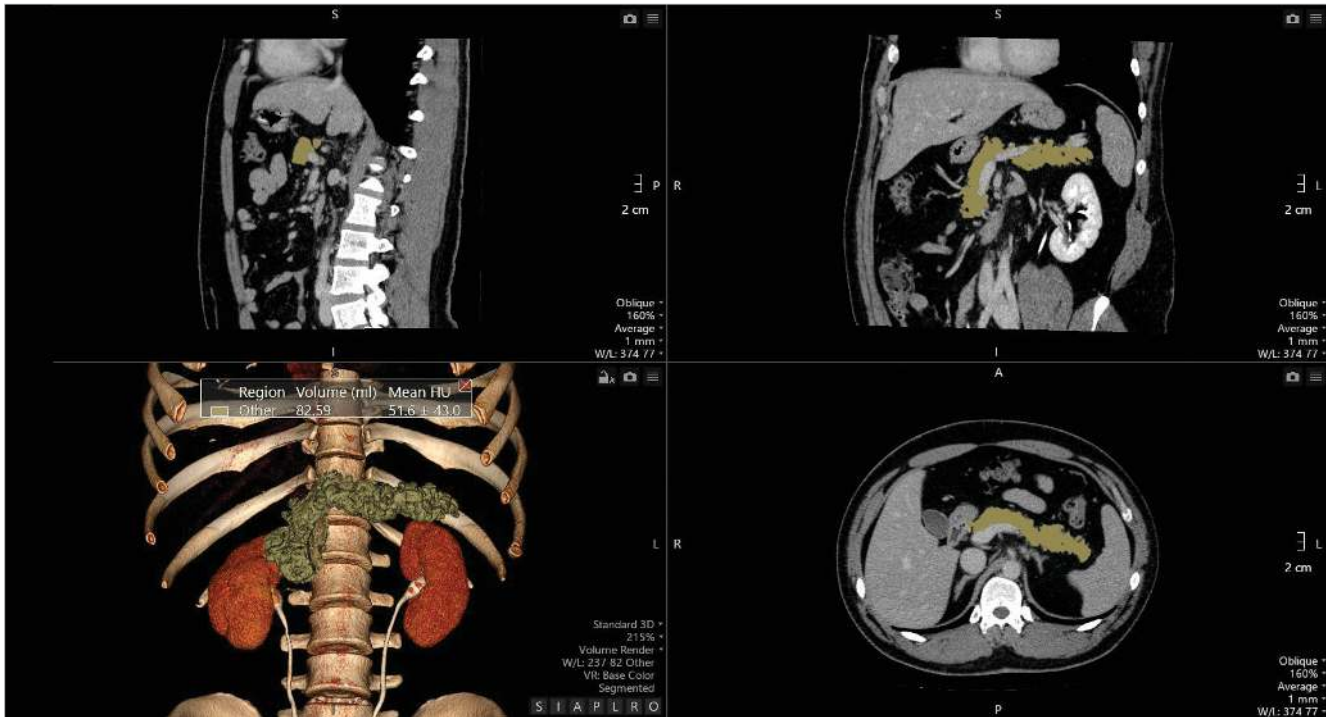
Figure 2. Pancreatic volume measurement by a semi-automated model in a 55-year-old male with pancreatic steatosis. Note that VFA was calculated as 240 cm^2.

uation value. Mean hepatic parenchymal attenuation is calculated by placing 1 cm^2 of ROI in five different regions of the hepatic parenchyma. LAI > 5 HU identifies the absence of significant steatosis (11). The subjects with LAI ≤ 5 HU composed the hepatosteatotic group.

Pancreas volume was calculated on portal venous phase by a semi-automated model (voxel-based volume calculation). In our institution, the axial thickness for non-enhanced series was 5 mm in living-liver donor transplant patients, which is not adequate for pancreatic volume calculation. In this model, threshold-based segmentation algorithms were used facilitating the separation of pancreas parenchyma from the surrounding tissues (12). After selecting a region of interest, pixels of analogous attenuation were highlighted by the software (Figure 2). If the model highlights the surrounding tissues same as the pancreas parenchyma, the erroneously selected structures margins were outlined manually by the radiologists (AGC) on axial images and excluded from the highlighted area. Then the post-processing software (Vitrea Version 7.4, Vital images, Minnetonka, Minnesota, United States) automatically calculated the total parenchymal volume. The mean processing time for pancreatic volume measurement was approximately 19 minutes.

Measurement of Body Fat Composition

The automated fat analysis software program (Vitrea Adcanced Vizualization CT Fat Measurement) quantified the total (TFA), visceral (VFA) and subcutaneous (SFA) fat tissue areas on one axial image at the level of L3-L4 disk. The adipose tissue between the anterior side of the vertebra and abdominal wall muscle was defined as visceral fat, and the area between the skin and abdominal wall was segmented as subcutaneous fat. -30 HU and - 190 HU were arranged as threshold values defining the adipose tissue (13). An area of interest selected the similar pixels of those adjusted attenuation values and then highlighted those adipose tissues separately. The program automatically calculated SFA, VFA, TFA, VFA/TFA and waist circumference.

Statistical Analysis

The difference between the two groups for normally distributed continuous variables were evaluated by Student's t test. Differences between the two groups for nominal variables were analyzed using the Chi Square test. Receiver operating characteristic (ROC) curves were used to describe the performance of diagnostic value of the variables. The area under the corresponding curves was calculated as described by Hanley and McNeil (14). Degree of association between continuous variables was calculated by Pearson's correlation coefficient. In order to define the risk factors of the outcome variable, multiple logistic regression analysis was used, and adjusted odds ratios were calculated. p-values less than 0.05 were considered significant.

RESULTS

Study population consisted of 38 females (38.2%) and 60 males (61.2%) with a mean age of 35.9 ± 8.7 (min-max; 20-59) years.

Figure 3. Pancreas volume in a 48-year-old female without fatty pancreas. Pancreas volume was smaller compared to the fatty subject shown as Figure 2. (pancreas volume of the nonfatty pancreatic subject; 62.36 mL, pancreas volume of the fatty pancreatic subject; 82.59 mL).

Fatty pancreas was detected in 40 patients of the study population. There was a statistically significant difference between the two groups in terms of sex (p< 0.001). No statistically significant difference was observed between the two groups in terms of age (p= 0.098). Pancreas volume was lower in the non-fatty group with a mean value of 80.7 ± 16.2 mL (range; 48-114.2 mL), but this difference did not reach statistically significant levels (Figure 3, p= 0.097). Mean VFA value in the fatty group was 173.6 ± 67.6 cm^2, while the mean in non-fatty group was 66.7 ± 46.9 cm2. VFA, TFA and VFA/TFA were significantly larger in the fatty group as compared to the non-fatty group (p< 0.001, p< 0.001, p< 0.001; respectively). Fatty pancreas subjects were found to have higher values of SFA (mean ± SD, 155.8 ± 52.3 cm^2); however, this difference was not statistically significant (p= 0.121). Table 1 summarizes the demographic data and pancreas volume-abdominal fat area measurements of the study population.

In ROC analysis, patients with VFA > 107.2 cm^2 (area under the curve [AUC] ± standard error [SE]: 0.920 ± 0.029, p< 0.001) appeared much more likely to have pancreatic steatosis. For this cut-off value (VFA > 107.2 cm^2), sensitivity, specificity, and positive (PPV) and negative predictive values (NPV), as well as their 95% Confidence Intervals (CI), were calculated as 90% (77-96%), 87.9% (77%-94%), 83.7% (95% CI: 70-91.9%), and 92.7% (95% CI: 82.7-97.1%) respectively. Mean VFA/TFA ratio was found to be 0.52 ± 0.09 in the fatty pancreas group. A cut-off value of 0.42 for VFA/TFA in the discrimination of pancreas steatosis was found to allow the appropriate combination of sensitivity 87.5% (73.9-94.5%) and specificity 72.4% (59.8-82.2%), with AUC being 0.856 ± 0.037 (95% CI: 0.784-0.927, p< 0.001). PPV and NPV was 68.6% (55-79.7%) and 89.4% (77.4-95.4%), respectively.

Pearson's correlation analysis demonstrated that the mean attenuation difference of the pancreas-spleen parenchyma negatively correlated with VFA, VFA/TFA and TFA (r= -0.715, r= -0.605, r= -0.573, respectively; p< 0.001 for all). Among the abdominal fat tissue compartments, this relationship is most significant with VFA. However, the attenuation difference was not related with SFA and the pancreas volume (p= 0.086 and p= 0.239). Table 2 demonstrates the correlations between pancreas steatosis (difference of pancreas-splenic mean attenuation value) and abdominal fat tissue compartments and pancreatic volume. Univariate analyses showed that VFA, TFA, waist circumference, and male sex were significant risk factors for pancreas steatosis (Table 3). Multiple logistic regression analyses adjusted for age confirmed that higher VFA (≥ 107.2 cm^2) independently predicted pancreas steatosis (Table 4). No association was found between pancreas steatosis and TFA and SFA in the multivariate analysis.

Out of the 98 patients, 43 had hepatic steatosis (43.9%). Of the 40 patients with pancreatic steatosis, 31 (77.5%) had co-existing hepatic steatosis. Hepatic steatosis was related to fatty pancreas (p< 0.001).

DISCUSSION

Previous studies have shown that fatty pancreas is associated with BMI and visceral fat tissue (10,15). In a later study, the authors have proposed that visceral fat tissue is better indicator and predictor of pancreatic steatosis compared to BMI (8). However, to the best of our knowledge, no cut-off value of VFA

Table 1. Demographic data and pancreas volume-abdominal fat area measurements of the study population

	Fatty Group (P-S ≤ -5 HU)	Non Fatty Group (P-S > 5 HU)
Sex F/M, n	3/37	35/23
Age, mean ± SD, years	37.5 ± 7.5	34.7 ± 9.2
TFA, cm^2, mean ± SD (min-max)	329.4 ± 102.5 (149.6-577.2)	202.9 ± 92.6 (22.3-472.5)
VFA, cm^2, mean ± SD (min-max)	173.6 ± 67.6 (52.8-345.3)	66.7 ± 46.9 (11.7-260.3)
SFA, cm^2, mean ± SD (min-max)	155.8 ± 52.3 (79.8-287.3)	136.1 ± 66.8 (10.6-357.7)
VFA/TFA	0.52 ± 0.09 (0.30-0.70)	0.33 ± 0.14 (0.07-0.61)
Waist circumference	98.3 ± 8.2 (84.2-119.9)	87.8 ± 7.6 (70.8-109.2)
Pancreas volume, mL (min-max)	86.5 ±17.3 (58-119.2)	80.7 ± 16.2 (48-114.2)
F: Female, M: Male, P: Pancreas, S: Spleen, VFA: Visceral fat area, TFA: Total fat area, SFA: Subcutaneous fat area.		

Table 2. Correlation coefficients (r) between the variables

	TFA (p)	SFA (p)	VFA (p)	VFA/TFA (p)	Waist Circumference (p)	Pancreas volume (p)
Pancreas-spleen MAV, r	- 0.573 (< 0.001)	-0.174 (0.086)	-0.715 (< 0.001)	-0.605 (< 0.001)	-0.570 (< 0.001)	-0.120 (0.239)
TFA, r		0.781 (< 0.001)	0.866 (< 0.001)	0.348 (< 0.001)	0.903 (< 0.001)	0.084 (0.410)
SFA, r			0.363 (< 0.001)	-0.264 (0.009)	0.613 (< 0.001)	-0.128 (0.208)
VFA, r				0.730 (< 0.001)	0.855 (< 0.001)	0.229 (0.024)
VFA/TFA, r					0.485 (< 0.001)	0.287 (0.004)
Waist Circumference, r						0.259 (0.010)

MAV: Mean attenuation value, VFA: Visceral fat area, TFA: Total fat area, SFA: Subcutaneous fat area.

Table 3. Univariate analyses of the risk factors

	OR	95% CI	p
Age	1.04	0.99-1.09	0.112
Sex (male)	18.76	5.17-68.10	< 0.001
TFA	1.01	1.00-1.02	< 0.001
SFA	1.00	0.99-1.01	0.125
VFA	1.03	1.02-1.04	< 0.001
VFA ($\geq$ 107.2 cm^2)	65.57	17.86-240.71	< 0.001
VFA/TFA ($\geq$ 0.42)	18.37	6.11-55.19	< 0.001
Waist circumference	1.18	1.10-1.26	< 0.001

VFA: Visceral fat area, TFA: Total fat area, SFA: Subcutaneous fat area, OR: Odds ratio, CI: Confidence interval.

Table 4. Multiple logistic regression analyses of the risk factors showing statistically significant difference

	OR	95% CI	p
SEX (male)	11.4	2.1-63.4	< 0.001
VFA ($\geq$ 107.2 cm^2)	58.2	12.2-277.1	< 0.001

OR: Odds ratio, CI: Confidence interval, VFA: Visceral fat area.

quantified on CT has previously been proposed for fatty pancreas.

There are some hypotheses on ectopic fat storage. Ectopic fat accumulation in undesirable sites (such as the liver, the pancreas, the heart) might be due to the dysfunction of subcutaneous fat tissue and as a result, insufficient storage of fat tissue in the subcutaneous fat tissue occurs. The dysfunction of subcutaneous fat tissue signals especially triglycerides to move toward the internal organs (16). Additionally, it has been shown in obese animal models that the dysfunctional visceral adipocyte they present produces inflammation and insulin resistance. Simi-larly, it has been shown in humans that visceral adipose tissue is metabolically active and secretes enzymes including leptin, endotrophin and inflammatory cytokines such as tumor necrosis factor alpha (TNFα), IL-6, IL-8 and insulin-like growth factor (IGF-1), all of which lead to inflammation. Inflammation within the visceral adipose tissue is associated with systemic insulin resistance and metabolic syndrome (17). As mentioned before, metabolic syndrome is one of the strongest etiologic factors contributing to fatty pancreas. Nevertheless, the pathophysiology of pancreatic steatosis is not as well-understood as it is in the liver (18).

This topic was first investigated by Ogilvie in 1933, where he demonstrated that obese subjects had a higher degree of adiposity in their pancreas compared to controls in his cadaveric study (19). Later, in large autopsies series, the relationship between the amount of pancreatic fat and obesity and age was performed (20). We did not find a correlation between age and pancreas steatosis, but our cohort was younger and limited in a narrow scale (range; 20-59 years) and presented a smaller sample size. This may be the reason why we could not find an association between fatty pancreas and age. Studies in the literature have also defined a correlation between pancreas volume and steatosis and added that pancreatic volume is influenced by steatosis and obesity. They have shown that an incremental increase in the pancreas volume is due to the increase in fat volume in the parenchyma histologically (21). In concordance with their results, we found that pancreas volume was 7% greater in the fatty group. The current study demonstrated that abdominal fat tissue (except subcutaneous fat tissue) and fatty pancreas are related, with the highest correlation being with VFA. In ROC analysis, we defined a cutoff value of ≥ 107.2 cm^2 that estimated fatty pancreas. Similarly, a recent MRI (magnetic resonance imaging) study has shown the highest correlation between fatty pancreas and visceral fat tissue compared to other abdominal fat compartments (22), and the study has also pointed out that BMI is not a good predictor of pancreatic steatosis with a very low correlation coefficient (r) of 0.12. Similarly, Staff et al. have claimed in a pediatric population study that VFA and pancreas fat fraction have a significant association whereas BMI does not (23).

In the current study, 43.9% of the study population had concomitant hepatic steatosis. Of the 40 patients with pancreatic steatosis, 77.5% of the patients demonstrated co-existence of fatty liver, which is quite similar to previous reports in the literature. Our literature review identified citations that demonstrated a positive relationship between nonalcoholic fatty pancreas disease and fatty hepatic disease (10,24,25). The liver and pancreas arise from foregut endoderm in embryologic life. Since the pancreas has the same embryological origin and close anatomical relationships to the liver, fat accumulation might be analogous in the pancreas and liver parenchyma (3). Additionally, the pancreas, liver and visceral fat tissue are controlled by the same vagal neurons (26).

Nonetheless, this study has some limitations. The major limitation of the current study is that pancreas steatosis was not evaluated histologically. However, the study conducted by Kim et al. has assumed that the difference between pancreas and spleen mean attenuation values on CT is significantly correlated with the intrapancreatic fat accumulation histologically (9). They have also added that non-enhanced CT is a useful tool in the non-invasive assessment of pancreas steatosis. The retrospective design of the current study with a limited study population is one of the other limitations. Large scale prospective studies which comprise histopathological evaluation are warranted to strengthen the current study's results. Additionally, we were not able to obtain weight and height data for the study population from the institution's medical records. Therefore, we could not correlate our results with BMI. In the current study, we observed that age was not a risk factor for fatty pancreas. Our cohort was younger and had a smaller sample size, which may explain why we could not find an association between fatty pancreas and age. On the other hand, another study with a larger study population has demonstrated a weak positive correlation between the pancreatic fat fraction and age (r= 0.33, p= 0.01) (22).

CONCLUSION

CT attenuation index, which defines pancreatic steatosis, is moderately correlated with VFA but not with SFA. A cutoff value of VFA ≥ 107.2 cm^2 and VFA/TFA ≥ 0.42 exhibited high specificity and sensitivity ratios to define pancreatic steatosis. Additionally, hepatic and pancreatic steatosis, which are the main ectopic fat accumulation sides, are related to each other. Further large-scale studies are needed to validate the current study findings.

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BT ile hesaplanan pankreatik yağ endeksinin viseral obezite ve karaciğer yağlanması ile ilişkisi

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ÖZET

Giriş ve Amaç: Çalışmanın amacı, pankreas yağlanması ile viseral yağ doku miktarı arasındaki ilişkiyi araştırmaktır. Ayrıca, normal yetişkinlerde pankreas yağlanması, pankreas hacmi, karaciğer yağlanması, yaş ve cinsiyet arasındaki ilişkileri de araştırmayı amaçlamaktadır. Aynı zamanda, yağlı pankreası öngören viseral yağ dokusu alanı (VFA) için cutoff değeri bulmayı amaçlamaktadır.

Gereç ve Yöntem: Pankreas hastalık öyküsü olmayan 98 canlı karaciğer nakli vericisinin BT incelemeleri yağlı pankreas varlığı açısından değerlendirildi. BT'de yarı otomatik model ile pankreas hacmi, VFA, subkutan-total FA, VFA/TFA oranları hesaplandı. Eşlik eden karaciğer yağlanması kaydedildi.

Bulgular: VFA, TFA ve VFA/TFA oranları yağlı pankreas grubunda anlamlı olarak daha yüksekti (sırasıyla; p< 0,001, p< 0,001, p< 0,001) ve pankreas yağlanması; VFA, VFA/ TFA ve TFA ile orta derecede korele olup VFA en yüksek korelasyon katsayısına sahipti. (r= -0,715, r= -0,605, r= -0,573; sırasıyla p< 0,001). VFA ≥ 107,2 cm^2 cutoff değeri pankreas yağlanmasını %90 duyarlılık (%95 GA= %77-96) ve %87.9 özgüllükle (%95 GA= %77-94) tahmin eder. Yağlı pankreas grubunda ortalama pankreas hacmi 86,5 ± 17,3 mL (aralık; 58-119,2 mL, p= 0,097) ölçülmüş olup daha yüksek bulundu . Çoklu lojistik regresyon analizinde pankreas yağlanması; VFA ve erkek cinsiyet ile anlamlı derecede ilişkiye sahipti (OR= 58,2, %95 GA= 12,2-277,1, p< 0,001; OR= 11,4, %95 GA= 2,1-63,4, p< 0,001;). Yağlı pankreas grubunun %77,5'inde eşlik eden hepatik steatoz vardı.

Sonuç: Pankreas yağlanması; yüksek VFA, VFA/TFA oranları ve hepatik steatoz ile ilişkilidir. VFA ≥ 107.2 cm^2 cutoff değeri pankreas yağlanmasını öngörebilir.

Anahtar Kelimeler: Pankreas, yağlanma, karaciğer yağlanması, multidedektör bt, viseral obezite, organ hacmi