VIA MEDICA
RESEARCH PAPER
ISSN 2450–7458
e-ISSN 2450–8187

Firuze Yalçın[1], Rıdvan Sivritepe[2], Fatma Kulalı[3], Aynur Gül[4], Esra Bora[5], Sema Uçak Basat[6]

[1]Department of Internal Medicine, Hilvan Şehit Halit Şiltak State Hospital, Şanlıurfa, Türkiye
[2]Department of Internal Medicine, Istanbul Medipol University Faculty of Medicine, Istanbul, Türkiye
[3]Department of Radiology, University of Health Sciences Umraniye Education and Research Hospital, Istanbul, Türkiye
[4]Department of Internal Medicine, Mardin Education and Research Hospital, Mardin, Türkiye
[5]Department of Internal Medicine, Karasu State Hospital, Sakarya, Türkiye
[6]Department of Internal Medicine, University of Health Sciences Umraniye Education and Research Hospital, Istanbul, Türkiye

Pancreatic and Hepatic Steatosis in Women with Type 2 Diabetes after Pancreatitis: A Retrospective Cross-Sectional Study

ABSTRACT

Objective: This study aimed to compare the incidence of pancreatic steatosis and hepatic steatosis between patients with/without diabetes suffering from pancreatitis.

Materials and methods: 120 patients between the ages of 18 and 65 years who were hospitalized with pancreatitis were included in the study. The patients were divided into 2 groups: patients with diabetes with pancreatitis (n = 60, Group 1) and patients without diabetes with pancreatitis (n = 60, Group 2). Biochemical blood tests of the patients were analyzed. HU attenuation measurement results on pancreatic abdominal computed tomography (CT) were evaluated retrospectively. The pancreatic-hepatic steatosis status of the patients was recorded. All parameters were compared between the 2 groups.

Results: The study was conducted on a total of 120 female patients with pancreatitis. The average age of the patients was 52.3 years, the mean body mass index (BMI) was 27.3 kg/m^2, the mean HbA1c was 7.4%, and the mean diabetes duration was 4.6 years. The incidence of pancreatic steatosis was found to be statistically significantly higher in Group 1 ($p < 0.05$). While 35.0% (n = 21) of pancreatic steatosis was detected in Group 2, 56.7% (n = 34) of Group 1 were found to have pancreatic steatosis. The mean Hounsfield unit (HU) attenuation differences in the pancreas, corpus, and tail in Group 1 and Group 2 were determined to be statistically significant. ($p = 0.030$, 0.25, and 0.18, respectively). In the correlation analysis, a statistically significant and weak relationship was found between HbA1c and tail and pancreas/spleen values ($p < 0.05$). It was determined that there was a statistically significant and weak relationship between glucose and corpus, tail, pancreas, and pancreas/spleen values ($p < 0.05$).

Conclusions: In our study, pancreatic steatosis was found to be more common in Group 1. However, no significant difference was detected between Group 1 and Group 2 in terms of hepatosteatosis. (Clin Diabetol 2024; 13, 5: 254–259)

Keywords: type 2 diabetes, pancreatitis, pancreatosteatosis, hepatosteatosis

Address for correspondence:
Rıdvan Sivritepe Assoc. Prof.
Department of Internal Medicine, Istanbul Medipol University Pendik Hospital
Bahcelievler Quarter, Adnan Menderes Boulevard No. 31
Pendik Istanbul, Türkiye
Phone: + 90 (545)260 59 57
E-mail: dr.ridvansivritepe@gmail.com
Clinical Diabetology 2024, 13; 5: 254–259
DOI: 10.5603/cd.100237
Received: 13.04.2024 Accepted: 11.07.2024
Early publication date: 11.09.2024

Introduction

Pancreatitis is an inflammatory condition of the pancreas caused by an increase in activated proteolytic enzymes released from pancreatic cells under the influence of various stimuli [1]. Although its incidence is unknown, the incidence of acute pancreatitis is 5-35/100,000 [2]. Alcohol and gallstones are the 2 most common etiological causes of pancreatitis. Septicemia, septic shock, and multiorgan failure are the most common complications [3].

Pancreatic steatosis has been defined as a histopathological condition that occurs when fat cells accumulate in the pancreatic tissue. Pancreatic steatosis was first described in 1933. Pancreatic steatosis has been found to play a role in the etiology of type 2 diabetes (T2D), metabolic syndrome, pancreatic cancer, and severe acute pancreatitis [4]

Hepatosteatosis is defined as more than 5% of the liver weight being made up of fat. The etiology of hepatosteatosis includes insulin resistance, obesity, diabetes mellitus, hyperlipidemia, environmental risk factors, and drugs [5].

The frequency of both pancreasteatosis and hepatosteatosis is increased in diabetic patients compared to nondiabetics [5]. There are insufficient data in the literature to determine whether pancreatic and hepatic steatosis accompany diabetic patients with pancreatitis.

This study aimed to compare the incidence of pancreatic steatosis and hepatosteatosis between patients with diabetes and patients without diabetes suffering from acute pancreatitis.

Materials and methods

Study design and participants

The present study was designed as a retrospective cross-sectional study. The study enrolled 120 female patients according to statistical power analysis. We calculated the power analysis as follows: the minimum number of patients required to complete our study, which was calculated using the descriptive statistics with a confidence level of 95% ($\alpha = 0.05$) and a power of 80%, was 120 patients. According to the admission order, female patients who were admitted to our internal medicine outpatient clinic of the University of Health Sciences Umraniye Education and Research Hospital in Türkiye were included in the study.

Inclusion criteria

In total, 120 patients (60 with diabetes with pancreatitis — Group 1; 60 without diabetes with pancreatitis — Group 2) who were hospitalized due to pancreatitis at the University of Health Sciences Umraniye Education and Research Hospital internal medicine clinic between 01/03/2021 and 01/03/2022, were included in the study. Patients between 18 and 65 years of age diagnosed with acute pancreatitis without acute diabetic complications within the last 3 months, and with normal kidney and liver function tests, were included in the study. The patients below 18 years and over 65 years of age, as well as those with acute or chronic infection, neurological disease, history of major surgery, use of drugs associated with pancreatitis, patients with liver cirrhosis, pancreatic tumor, and patients who had undergone pancreatic surgery for any reason were excluded from the study. The demographics, laboratory parameters, and abdominal computed tomography (CT) of the patients were evaluated retrospectively.

Ethical approval

The study was designed in accordance with the principles enshrined in the Declaration of Helsinki. Ethics committee approval numbered 28 and dated 10.03.2022 was received for the study from the University of Health Sciences Umraniye Education and Research Hospital Ethics Committee.

Data collection

Metabolic parameters

Plasma glucose (70–100 mg/dL) enzymatic test, glycated hemoglobin (4,7–5,6%), HPLC, creatinine (< 1 mg/dL) Jaffe' method, CRP (< 3 mg/L) immunoassay, total cholesterol (< 200 mg/dL), high-density lipoprotein (HDL) (40–60 mg/dL), low-density lipoprotein (LDL) (< 130 mg/dL), calcium (8.5–1.03 mg/dL), phosphorus (2.8–4.5 mg/dL), alanine transaminase (10–40 U/L), aspartate transaminase (15–50 IU/L), gamma glutamyl transferase (7-49 U/L), alkaline phosphatase (38–155 U/L), amylase (17–115 U/L), lipase (13–60 U/L), albumin (3.5–5.5 g/dL), and triglyceride (< 150 mg/dL) concentrations were measured using enzymatic colorimetric test, bilirubin (0.3–1.9 mg/dL) diazo reaction, blood urea nitrogen (10–20 mg/dL) using a spectrophotometer, and sodium (135–145 mEq/L) and potassium (3.5–5.5 mmol/L) levels using an ion selective electrode analysis method (ARCHITECT plus Abbott, Illinois, U.S.A.). Hemogram parameters were measured by electrical impedance method with Mindray BC 6800 device, Shenzhen, China.

The patient group suffering from pancreatitis was considered as patients who met at least 2 of the criteria of abdominal pain, pancreatic enzyme elevation, and imaging findings [2].

Group 1 was determined as those who met the diagnostic criteria for diabetes American Diabetes Association criteria, and Group 2 was determined as those who did not meet the diagnostic criteria for diabetes [6].

Radiological evaluation

All patients who were hospitalized due to pancreatitis and included in the study had an upper abdominal CT examination. Measurements were made by a single radiologist. In the radiological evaluation of the abdominal CT scans of the patients, it was examined whether the patients had hepatosteatosis and pancreatic steatosis. Steatosis causes decreased density in the relevant organ on CT. The steatosis level can be measured on CT. Quantitative density measurements were obtained from the spleen (for reference), liver, and pancreas for the diagnosis of hepatosteatosis and pancreatic steatosis. The level of steatosis can be determined using Hounsfield units (HU). However, because there is no designated HU cut-off value for pancreatic steatosis, a negative HU value was used according to the spleen parenchyma by establishing a correlation with the spleen. Measurements were made in 5 segments in the liver, in areas free of vascular structure, bile ducts, calcification, and artifacts, and the average value was calculated. Care was taken to ensure that there were no vascular structures or artifacts in the areas where measurement would be made in the spleen and pancreas. For standardization, the average liver/spleen density value ratio was used for the diagnosis of hepatosteatosis, and the average pancreas/spleen density value ratio was used for pancreatic steatosis. Patients with a liver/spleen density ratio < 1 were diagnosed with hepatosteatosis [7]. For the definition of pancreatic steatosis, the pancreas/spleen density value ratio was accepted as < 0.7 [4].

Statistical analysis

The data were analyzed using the SPSS 25.0 package program. The Kolmogorov-Smirnov test was used to check whether the data were normally distributed. While evaluating the study data, descriptive statistical methods (mean, standard deviation, frequency) as well as t-test and one-way ANOVA test for parametric data, and chi-square, Mann-Whitney U, and Kruskal-Wallis H test for non-parametric data were used. The Friedman test was used to compare the follow-ups of parameters that did not show a normal distribution, and the Wilcoxon signed rank test was used to evaluate pairwise comparisons. Significance was accepted as a p value of 0.05 for all values.

Results

Subject characteristics

The study was conducted on a total of 120 patients, 60 patients with diabetes with pancreatitis and 60 patients without diabetes with pancreatitis. The age average of the patients was 52.3 ± 8.1 (18–65) years, the mean BMI was 27.3 kg/m^2, the mean HbA1c was 7.4%, and the mean diabetes duration was 4.6 years. General characteristics and biochemical parameters of the study groups are shown in Table 1.

Comparison of CT attenuation measurements of patients with diabetes and patients without diabetes

The degree of steatosis between the liver, pancreas, and spleen was measured by abdominal CT examination of the study patients (Tab. 2). It was determined that the mean HU values of pancreas, corpus, and tail were significantly decreased in Group 1 and Group 2.

Comparison of patients in terms of hepatosteatosis

It was determined that there was no statistically significant difference between Group 1 and Group 2 in terms of hepatosteatosis: 76.7% of Group 2 and 85.5% of Group 1. A statistically significant difference was found between the groups in terms of pancreatic steatosis ($p = 0.017$). The incidence of pancreatic steatosis was higher in Group 1: 35.0% of Group 2 and 56.7% of Group 1.

Correlation analysis between laboratory parameters and CT measurement

As a result of the Spearman correlation analysis performed to determine the relationship between laboratory and CT measurement data, it was determined that there was a statistically significant, negative, and weak relationship between HbA1c and tail and pancreas/spleen ratio values ($p < 0.05$). It was determined that there was a statistically significant, negative and weak relationship between glucose and corpus, tail, pancreas, and pancreas/spleen ratio values ($p < 0,05$).

Creatinine, potassium, CRP, LDH, pancreatic head, corpus, tail, pancreas, and spleen variables, which are among the variables that have a significant effect on the status of hepatosteatosis, were evaluated with backward logistic regression analysis.

Among the variables that have a significant effect on the state of pancreatic steatosis, HbA1c, glucose, total cholesterol, LDL, total bilirubin, pancreatic head, corpus, tail, and pancreas variables were evaluated with backward logistic regression analysis.

Table 1. Comparison of Laboratory Values of Patients with Diabetes and Patients without Diabetes

	Patients without diabetes group	Patients with diabetes group	Total	Z	p-value
	Mean ± SD	Mean ± SD	Mean ± SD		
Age [years]	51.9 ± 6.9	52.9 ± 5.8	52.3 ± 6.2	–0.515	0.658
Height [cm]	166 ± 16.1	166 ± 14.1	166 ± 14.3	–0.582	0.521
Weight [kg]	76 ± 4.9	78 ± 5.1	77 ± 4.2	–0.669	0.109
BMI [kg/m^2]	26.7 ± 3.8	27.9 ± 3.0	27.3 ± 2.5	–1.140	0.818
Diabetes duration [years]	0	4.6 ± 2	4.6 ± 2	–9.446	0
HbA1c (4.7–5.6%)	5.16 ± 0.29	9.7 ± 1.62	7.43 ± 2.56	–9.457	0
Glucose (70–100 mg/dL)	92.18 ± 8.86	219.45 ± 71.51	155.82 ± 81.6	–9.422	0
Urea (5–11 mg/dL)	26.35 ± 7.17	29.9 ± 14.06	28.12 ± 11.25	–1.405	0.16
Creatinine (< 1 mg/dL)	0.69 ± 0.11	0.69 ± 0.2	0.69 ± 0.16	–0.202	0.84
Aspartate Aminotransferase (15–50 IU/L)	105.87 ± 120.63	101.75 ± 131.65	103.81 ± 125.75	–0.501	0.616
Alanine Aminotransferase (10–40 U/L)	127.72 ± 141.44	114.1 ± 161.41	120.91 ± 151.26	–0.281	0.779
Alkaline phosphatase (38–155 U/L)	132.87 ± 106.05	139.37 ± 104.02	136.09 ± 104.65	–0.404	0.686
Gamma glutamyl transferase (7–49 U/L)	210.98 ± 226.38	183.62 ± 231.38	197.18 ± 228.36	–0.478	0.632
Amylase (17–115 U/L)	1085.73 ± 965.95	1041.93 ± 892.8	1063.83 ± 926.44	–0.016	0.987
Lipase (13–60 U/L)	2861.7 ± 3495.21	2973.7 ± 4011.85	2917.7 ± 3746.99	–0.294	0.769
Total Cholesterol (< 200 mg/dL)	172.13 ± 53.04	197.33 ± 76.55	184.73 ± 66.78	–1.979	0.048*
LDL Cholesterol (< 130 mg/dL)	106.13 ± 57.46	139.6 ± 91.43	122.87 ± 77.87	–2.945	0.003*
Triglyceride (< 150 mg/dL)	132.55 ± 98.81	197.5 ± 206.87	165.03 ± 164.69	–2.261	0.024*
HDL cholesterol (40–60 mg/dL)	44.82 ± 12.46	42.22 ± 13.46	43.52 ± 12.98	–0.827	0.408
Sodium (135–145 mEq/L)	139.37 ± 1.83	139.27 ± 1.72	139.32 ± 1.77	–0.471	0.637
Potassium (3.5–5.5 mmol/L)	4.5 ± 0.5	4.38 ± 0.52	4.44 ± 0.51	–1.251	0.211
Calcium (8.5–1.03 mg/dL)	9.21 ± 0.79	9.11 ± 0.78	9.16 ± 0.78	–0.558	0.577
C-reactive protein (< 3 mg/l)	31.8 ± 30.93	22.65 ± 27.86	27.22 ± 29.67	–2.437	0.015*
Lactate Dehydrogenase (90–250 U/L)	281.03 ± 121.08	296.62 ± 203.35	288.83 ± 166.83	–0.286	0.775
Total bilirubin (0.3–1.9 mg/dL)	1.47 ± 1.57	1.39 ± 1.24	1.43 ± 1.41	–0.247	0.805
Direct bilirubin (0–0.3 mg/dL)	0.87 ± 1.35	0.79 ± 0.91	0.83 ± 1.15	–0.165	0.869

*p-value < 0.05; Mann-Whitney U Test
BMI — body mass index; HbA1c — hemoglobin A1c; HDL — high-density lipoprotein cholesterol; LDL — low-density lipoprotein cholesterol

It was seen that glucose and pancreatic tail variables, which are among the risk factors that have an impact on the state of pancreatic steatosis, form a significant model. The explanatory coefficient of the model is 24.6%. According to the model, glucose increases the risk of pancreatic steatosis by 1.009 times (95% CI: 1.001–1.018).

Discussion

The present study showed that the frequency of pancreatic steatosis in Group 1 was higher than in Group 2, but there was no difference in the frequency of hepatosteatosis. Pancreatic steatosis ranges from simple fat storage and inflammation to the development of pancreatic fibrosis [8]. Diabetes plays an independent role in the progression of visceral fat accumulation and pancreatic steatosis [9]. Studies have reported that pancreatic steatosis is associated with obesity, insulin resistance, pre-diabetes, diabetes, and metabolic syndrome, and that obesity and insulin resistance play an important role in adipocyte infiltration in the steatosis of the liver and pancreas [10].

Loss of β cell mass and function, which leads to the development of diabetes, contributes to the steatosis of the pancreas through triglyceride accumulation [11]. Wu et al. suggested that pancreatic steatosis is strongly associated with metabolic parameters such as abdominal obesity, glucose, and HbA1c [12]. In our study, the average glucose and HbA1c levels of Group 1 were found to be higher than those of Group 2. Pancreatic steatosis was found to be higher in Group 1 than in Group 2. In addition, the average total cholesterol, LDL, triglyceride, and CRP levels of Group 1 were found to be higher than those of Group 2.

Table 2. Comparison of Computed Tomography Attenuation Measurements of Patients with Diabetes and Patients without Diabetes

	Patients without diabetes group	Patients with diabetes group	Total	Z	p-value
	Mean ± SD	Mean ± SD	Mean ± SD		
Pancreas head	73.9 ± 28.13	61.72 ± 34.62	67.81 ± 32	–1.798	0.072
Pancreas corpus	76.93 ± 28.65	62.62 ± 33.43	69.78 ± 31.82	–2.236	0.025
Pancreas tail	76.3 ± 28.55	63.57 ± 30.79	69.93 ± 30.25	–2.37	0.018*
Pancreas total	75.68 ± 27.85	64.07 ± 28.41	69.87 ± 28.61	–2.166	0.03
Spleen	104.73 ± 32.18	101.07 ± 30.07	102.9 ± 31.07	–1.008	0.313
Pancreas/spleen ratio	0.72 ± 0.13	0.64 ± 0.2	0.68 ± 0.18	–2.42	0.016*

Kruskal Wallis test; statistically significant in bold at p-value < 0.05

Computed tomography is an easily applicable method to evaluate pancreatic steatosis [13]. Studies have shown that the HU value in pancreatic steatosis on CT is similar to that of normal adipose tissue [14–16]. Pancreatic steatosis was detected radiologically as a decrease in CT attenuation and a negative HU scale between the pancreas and spleen [17]. In the study conducted by Tushuizen et al. [18], the rate of patients with pancreatic steatosis was found to be statistically higher in patients with diabetes than in patients without diabetes group. In our study, pancreatic attenuation between the pancreas and spleen and a decrease in negative HU value were found to be important findings in terms of pancreatic steatosis. It was determined that the mean HU values of the pancreas, corpus, and tail were significantly decreased in Group 1.

In our study, pancreatic steatosis was found to be higher in Group 1 than in Group 2. However, no difference was detected in terms of hepatosteatosis between the 2 groups. In another study, it was observed that pancreatic and liver attenuation was negatively correlated with glucose and HbA1c levels [18]. In the present study, hepatosteatosis was not found to be statistically different in Group 1. In our study, it was determined that there was a statistically significant and weak relationship between HbA1c and tail and pancreas/spleen ratio values. It was determined that there was a statistically significant and weak relationship between glucose and corpus, tail, pancreas, and pancreas/spleen ratio values.

Pancreatic and liver fat are closely related to obesity, metabolic syndrome, and diabetes. [19]. In our study, pancreatic steatosis appeared to be more common in Group 1. However, no significant difference was detected between Group 1 and Group 2 in terms of hepatosteatosis, and in the regression analysis, it was determined that glucose and pancreatic tail parameters influenced the risk of pancreatic steatosis.

Study limitations

The present study had some limitations. Our study was a retrospective cross-sectional study. Thus, it took a short observation period to establish a causal relationship for the significant association between pancreatic and hepatic steatosis and pancreatitis. It can be generalized to clinical settings through multicenter and longer-term prospective studies. Our study was from a single center; therefore, our results may not represent all patients with T2D. The patients included in our study were adult, middle-aged women; consequently, it is unclear whether our findings also apply to men in this age group. Another limitation of the study is that the measurements and comparisons were carried out only once.

Despite these limitations, our study shows the relationship between pancreatic steatosis and patients with pancreatitis T2D, and it is a valuable study, the likes of which has not been conducted before.

Conclusions

Diabetes mellitus is closely related to pancreatic and liver fat. In our study, pancreatic steatosis was detected twice as often in patients with diabetes with pancreatitis compared to patients without diabetes with pancreatitis, but it was found that there was no difference in the frequency of hepatosteatosis between the groups.

Article information

Data availability statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics statement

Ethics committee approval numbered 28 and dated 10.03.2022 was received for the study from the

University of Health Sciences Umraniye Education and Research Hospital Ethics Committee.

Author contributions

Conception: FY, RS; Design: FY, RS; Fundings: FY, RS; Materials: FY, RS, FK; Data collection and/or Processing: FY, RS, FK; Analysis and/or Interpretation: FY, RS; Literature review: FY, RS, AG, EB; Writer: FY, RS, AG, EB; Supervision: RS, SUB; Critical review: RS, SUB

Funding

No funding, donations, or other support was received for the study.

Conflict of interest

The authors declare no conflict of interest.

REFERENCES

1. Habtezion A. Inflammation in acute and chronic pancreatitis. Curr Opin Gastroenterol. 2015; 31(5): 395–399, doi: 10.1097/MOG.0000000000000195, indexed in Pubmed: 26107390.
2. Szatmary P, Grammatikopoulos T, Cai W, et al. Acute Pancreatitis: Diagnosis and Treatment. Drugs. 2022; 82(12): 1251–1276, doi: 10.1007/s40265-022-01766-4, indexed in Pubmed: 36074322.
3. Mederos MA, Reber HA, Girgis MD, et al. Acute Pancreatitis: A Review. JAMA. 2021; 325(4): 382–390, doi: 10.1001/jama.2020.20317, indexed in Pubmed: 33496779.
4. Ramkissoon R, Gardner TB. Pancreatic steatosis: an update. Curr Opin Gastroenterol. 2019; 35(5): 440–447, doi: 10.1097/MOG.0000000000000566, indexed in Pubmed: 31343416.
5. Engin A. Non-Alcoholic Fatty Liver Disease. Adv Exp Med Biol. 2017; 960: 443–467, doi: 10.1007/978-3-319-48382-5_19, indexed in Pubmed: 28585211.
6. ElSayed NA, Aleppo G, Aroda VR, et al. 2. Classification and Diagnosis of Diabetes: Standards of Care in Diabetes-2023. Diabetes Care. 2023; 46(Suppl 1): S19–S40, doi: 10.2337/dc23-S002, indexed in Pubmed: 36507649.
7. Zeb I, Li D, Nasir K, et al. Computed tomography scans in the evaluation of fatty liver disease in a population based study: the multi-ethnic study of atherosclerosis. Acad Radiol. 2012; 19(7): 811–818, doi: 10.1016/j.acra.2012.02.022, indexed in Pubmed: 22521729.
8. Paul J, Shihaz AV. Pancreatic steatosis: a new diagnosis and therapeutic challenge in gastroenterology. Arq Gastroenterol. 2020; 57(2): 216–220, doi: 10.1590/s0004-2803.202000000-27, indexed in Pubmed: 32490903.
9. Ahbab S, Ünsal A, Ataoğlu HE, et al. Prediabetes and Type 2 Diabetes are Independent Risk Factors for Computed Tomography-Estimated Nonalcoholic Fatty Pancreas Disease. Clinics (Sao Paulo). 2019; 74: e1337, doi: 10.6061/clinics/2019/e1337, indexed in Pubmed: 31664423.
10. Wagner R, Jaghutriz BA, Gerst F, et al. Pancreatic Steatosis Associates With Impaired Insulin Secretion in Genetically Predisposed Individuals. J Clin Endocrinol Metab. 2020; 105(11): 3518–3525, doi: 10.1210/clinem/dgaa435, indexed in Pubmed: 32725157.
11. Tariq H, Nayudu S, Akella S, et al. Non-Alcoholic Fatty Pancreatic Disease: A Review of Literature. Gastroenterology Res. 2016; 9(6): 87–91, doi: 10.14740/gr731w, indexed in Pubmed: 28058076.
12. Wu WC, Wang CY. Association between non-alcoholic fatty pancreatic disease (NAFPD) and the metabolic syndrome: case-control retrospective study. Cardiovasc Diabetol. 2013; 12: 77, doi: 10.1186/1475-2840-12-77, indexed in Pubmed: 23688357.
13. Koç U, Taydaş O. Evaluation of pancreatic steatosis prevalence and anthropometric measurements using non-contrast computed tomography. Turk J Gastroenterol. 2020; 31(9): 640–648, doi: 10.5152/tjg.2020.19434, indexed in Pubmed: 33090101.
14. Begovatz P, Koliaki C, Weber K, et al. Pancreatic adipose tissue infiltration, parenchymal steatosis and beta cell function in humans. Diabetologia. 2015; 58(7): 1646–1655, doi: 10.1007/s00125-015-3544-5, indexed in Pubmed: 25740696.
15. Caldart F, de Pretis N, Luchini C, et al. Pancreatic steatosis and metabolic pancreatic disease: a new entity? Intern Emerg Med. 2023; 18(8): 2199–2208, doi: 10.1007/s11739-023-03364-y, indexed in Pubmed: 37462859.
16. Blaho M, Dítě P, Kunovský L, et al. Fatty pancreas disease: clinical impact. Vnitr Lek. 2018; 64(10): 949–952, indexed in Pubmed: 30590942.
17. Pinnick KE, Collins SC, Londos C, et al. Pancreatic ectopic fat is characterized by adipocyte infiltration and altered lipid composition. Obesity (Silver Spring). 2008; 16(3): 522–530, doi: 10.1038/oby.2007.110, indexed in Pubmed: 18239594.
18. Tushuizen ME, Bunck MC, Pouwels PJ, et al. Pancreatic fat content and beta-cell function in men with and without type 2 diabetes. Diabetes Care. 2007; 30(11): 2916–2921, doi: 10.2337/dc07-0326, indexed in Pubmed: 17666465.
19. Pezzilli R, Calculli L. Pancreatic steatosis: Is it related to either obesity or diabetes mellitus? World J Diabetes. 2014; 5(4): 415–419, doi: 10.4239/wjd.v5.i4.415, indexed in Pubmed: 25126389.