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The overlooked significance of central obesity in the severity of acute pancreatitis

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Abstract

Central obesity has emerged as a pervasive global concern in recent times. The metabolic and cardiovascular risks associated with central obesity are not as well understood as the risks associated with obesity itself. The aim of the study is to evaluate how central obesity affects the severity of acute pancreatitis (AP). The current research included individuals aged 18 and above who were admitted for the first time to the Gastroenterology Clinic at Fırat University Hospital with a diagnosis of AP during the years 2022 and 2023. Participants were categorized into two groups based on the presence or absence of central obesity, as defined by the World Health Organization (WHO) standards. A comparative analysis was conducted among these groups, concentrating on anthropometric measurements, the Atlanta classification, Ranson score, Charlson Comorbidity Index (CCI), laboratory findings, and clinical outcomes. Patients with a Ranson score of 3 or lower were identified as having mild AP, whereas those with a score exceeding 3 were categorized as having severe AP. Out of the 75 patients who participated in the study, 45 individuals, accounting for 60%, showed signs of central obesity. In the central obesity group, the AP severity was found to be significantly higher according to Ranson scores and the Atlanta classification ($p<0.05$). In analyses based on BMI classification, central obesity was found to independently affect AP severity. A meticulous examination of the data revealed no statistically significant disparities between the study groups with respect to the Charlson Comorbidity Index or the duration of hospital stay. Central obesity has been found to affect AP severity independently of BMI. The consideration of central obesity as an independent risk factor in acute pancreatitis is believed to significantly enhance the prediction of mortality and morbidity in the disease.

Keywords: Central obesity, acute pancreatitis, atlanta classification, ranson score

Introduction

The rising rates of obesity among both adults and children have made it a significant public health concern worldwide. The World Health Organization (WHO) recognizes body mass index (BMI) as the primary measure for categorizing obesity [1].

Many research studies have shown that having excess fat around the abdomen plays a major role in the development of insulin resistance, abnormal lipid levels, and an increased risk of cardiovascular disease (CVD) [2,3]. Furthermore, type 2 diabetes, hypertension, hyperlipidemia, and hyperinsulinemia are more prevalent among individuals with central obesity [4].

The 2017–2018 NHANES data indicates a general increase in the prevalence of obesity across all age groups, with comparable rates of impact on both men and women. However, in countries with abundant food resources, obesity is reported to be more prevalent among men than women [5,6].

At present, the incidence of AP related to gallstones is increasing, largely due to the rising prevalence of obesity [7]. Gallstones have been identified as a contributing factor in 40% to 70% of AP cases [8,9]. Obesity is a significant risk factor for severe AP [10].

The liver produces C-reactive protein (CRP) as an acute-phase reactant when stimulated by interleukin-1 and interleukin-6. A serum CRP level exceeding 150 mg/L within 48 hours is recognized as a dependable indicator for differentiating severe AP from less severe forms [11]. Numerous biochemical indicators have been assessed for their effectiveness in gauging the severity of

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acute pancreatitis. These include urinary trypsinogen activation peptide (TAP), procalcitonin, polymorphonuclear elastase, amylase, lipase, glucose, calcium, antithrombin III, platelet-activating factor, interleukins 1, 6, and 8, as well as tumor necrosis factor (TNF)-alpha [12-14]. It has been established that obesity is a catalyst for inflammatory processes within the body. CRP and various cytokines have been demonstrated to play a role in this process. Consequently, the hypothesis has been postulated that obesity-related inflammation may exacerbate the clinical progression of pancreatitis.

In patients diagnosed with acute pancreatitis, disease severity can be assessed using the Ranson criteria, which consist of 11 parameters. As Ranson scores increase, so does the mortality risk. Specifically, mortality rates have been documented to range from 0% to 3% when the score is less than or equal to 3, from 11% to 15% when the score is greater than 3 and up to 40% when the score is greater than 6 [15-17]. In addition to its well-known impact on general health, obesity is recognized as a major risk factor for developing acute pancreatitis. This study seeks to assess how central obesity affects the severity of acute pancreatitis, regardless of BMI.

Material and Methods

The Non-Interventional Research Ethics Committee at Fırat University granted ethical approval for this study (No: 2024/11-06, Date: 01 August 2024). The research involved patients who were 18 years or older, admitted to the Gastroenterology Clinic at Fırat University Faculty of Medicine Hospital between 2022 and 2023, and monitored for a diagnosis of AP.

The study cohort consisted of adult patients (≥18 years) who presented to the emergency department within 24 hours of symptom onset attributable to AP. The evaluation was conducted exclusively on patients experiencing their initial episode of acute pancreatitis, with those exhibiting iatrogenic or traumatic etiologies being excluded from the study. Furthermore, patients who underwent magnetic resonance cholangiopancreatography (MRCP) within 24 hours of presentation, or endoscopic retrograde cholangiopancreatography (ERCP) due to suspected biliary pancreatitis, were included in the study.

All participants underwent anthropometric assessments: body weight was recorded in kilograms using a calibrated scale with a barrier, while participants wore light clothing and no footwear. Height was measured in centimeters with a stadiometer. BMI was determined by dividing weight in kilograms by the square of height in meters (kg/m²) and classified according to WHO standards into normal, overweight, and obesity classes I–III.

Participants had their waist circumference measured while standing straight with their arms resting at their sides, at the conclusion of a normal breath out. The tape measure was placed midway between the lowest rib and the iliac crest (upper iliac border) in accordance with WHO guidelines, and measurements were taken to the nearest 0.1 cm. Central obesity was identified based on WHO standards, with a waist circumference greater than 102 cm in men and more than 88 cm in women signifying central obesity. All anthropometric measurements were performed by trained nurses or research personnel.

Laboratory and Timing: The initial laboratory samples were collected at the time of presentation to the emergency department (within the first 24 hours). The parameters required for the second set of Ranson criteria were reassessed at 48 hours. The parameters that were measured included complete blood count (WBC, Hct, PLT), glucose, BUN, creatinine, electrolytes (including Ca), AST, ALT, LDH, amylase, lipase, CRP, INR, APTT, lipid panel (TG, HDL, LDL, VLDL), and arterial blood gas/PaO_2 (when indicated). Laboratory values were reported according to the published units of the study (e.g., mg/dL, IU/L).

The Ranson score is determined using eleven clinical and laboratory criteria, evaluated both at the time of admission and 48 hours later, with each positive criterion earning one point. Upon admission, the parameters assessed include: age over 55 years, leukocyte count exceeding 16,000/mm³, blood glucose levels above 200 mg/dL, lactate dehydrogenase (LDH) greater than 350 IU/L, and aspartate aminotransferase (AST) over 250 IU/L. During the first 48 hours, additional factors are taken into account: a hematocrit decrease of more than 10%, a rise in blood urea nitrogen (BUN) exceeding 5 mg/dL, serum calcium below 8 mg/dL, partial pressure of oxygen (PaO_2) under 60 mmHg, a base deficit greater than 4 mEq/L, and fluid sequestration surpassing 6 L. The Ranson score can range from 0 to 11, with scores between 0 and 2 considered mild, and scores of 3 or higher deemed severe in this study. All criteria are assessed both at admission and at the 48-hour mark.

The Charlson Comorbidity Index (CCI) is a recognized instrument for assessing the impact of coexisting medical conditions in patients. Each condition is given a specific weighted score, and the total of these scores forms the CCI. The conditions evaluated in this research included: myocardial infarction (1), congestive heart failure (1), peripheral vascular disease (1), stroke (1), dementia (1), chronic obstructive pulmonary disease (COPD) (1), rheumatic disease (1), peptic ulcer disease (1), diabetes mellitus (1), diabetes with complications (2), hemiplegia (2), gastrointestinal or hematologic cancers such as stomach/intestinal cancer, lymphoma, or leukemia (2), moderate-to-severe renal disease (2), moderate-to-severe liver disease (3), metastatic solid tumor (6), and AIDS (6). In this study, the CCI was determined using the original method, with scores grouped into categories of 0, 1–2, and ≥3 for analysis. The age-adjusted CCI was not utilized; only the original non-age-adjusted CCI was applied.

Statistical Analysis

Data analysis was conducted using SPSS for Windows (V.27, IBM). Categorical variables were represented by frequencies

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and percentages, while continuous variables were evaluated for their distribution patterns. For continuous data that followed a normal distribution, the mean ± standard deviation (SD) was used, whereas data not normally distributed were expressed as the median and interquartile range (IQR). The chi-square test was utilized to explore associations between categorical variables. For continuous data, parametric tests such as the t-test or ANOVA were applied when normality was assumed, and non-parametric tests were used when normality assumptions were not met. Paired t-tests were employed for comparing sequential follow-up measurements. A P value of less than 0.05 was considered to indicate statistical significance.

Results

The study included 75 patients aged 21 to 94 years, of whom 40 (53.3%) were female and 35 (46.7%) were male, with a mean age of 60.52 ± 15.83 years. Participants were stratified into two groups based on WHO criteria: those without central obesity (n=30) and those with central obesity (n=45). A significant association was observed between central obesity and female sex (p=0.001). In the non-central obesity group, males comprised 73.3% of patients, whereas females represented 71.1% of the central obesity group. No statistically significant difference in mean age was detected between the two groups (p=0.429) (Table 1).

A meticulous examination revealed no statistically significant disparities between the groups with and without central obesity with respect to triglyceride (p=0.249), HDL (p=0.666), LDL (p=0.504), VLDL (p=0.122), and total cholesterol (p=0.204) levels (see Table 2). Table 2 presents the biochemical comparisons between the groups.

There was a notable difference in Ranson scores, with patients in the central obesity category showing higher scores than those without central obesity (p=0.013). Additionally, when comparing the overweight group (BMI 25–29.9 kg/m²) to the obesity class I group (BMI 30–34.9 kg/m²), the latter had higher Ranson scores; however, this difference was not statistically significant (p>0.05) (Table 3).

As illustrated in Table 4, a comparison has been made between patients who have been classified according to their body mass index and those who have been classified according to the Atlanta classification for acute pancreatitis.

In the central obesity group, mild pancreatitis was observed in 84.4% of cases according to the Ranson score, whereas severe pancreatitis was detected in 15.6% of cases. Conversely, cases within the non-central obesity group exhibited mild pancreatitis. A statistically significant difference was observed in the Ranson score between the central obesity and non-central obesity groups (p=0.037). Furthermore, the rate of severe pancreatitis was found to be significantly higher in patients with central obesity compared to those without central obesity (see Table 5).

A lack of statistically significant discrepancy was identified in the duration of hospital stay among subjects with and without central obesity (p=0.369).

A comparative analysis revealed that 10% of patients in the non-central obesity group and 15.6% of those in the central obesity group had a documented history of cholecystectomy. A lack of statistically significant discrepancy was identified when comparing the groups with respect to cholecystectomy history (p=0.731). Within the non-central obesity group, the most prevalent etiological cause was identified as idiopathic, accounting for 53.3% of cases. The etiology of the remaining cases included biliary causes (30.0%), hypertriglyceridemia (10.0%), and DPP-4 inhibitor use (6.7%).

The most prevalent etiological cause in the central obesity group was idiopathic, accounting for 53.3% of cases. The etiology of the remaining cases included biliary causes (33.3%), DPP-4 inhibitor use (8.9%), and hypertriglyceridemia (4.4%). An examination of etiological causes revealed no statistically significant disparities between the groups with and without central obesity (p=0.844) (see Table 6).

In the non-central obesity group, the most prevalent weight category was overweight, observed in 80% of cases, while 20% of individuals were classified as normal weight. Within the cohort classified as central obesity, the predominant obesity category was Class 1, constituting 88.9% of the observed cases. Class 2 obesity was observed in 6.7% of cases, and Class 3 obesity in 4.4% of cases.

Table 1. Comparison of groups according to demographic and anthropometric characteristics

Parameters	Non-central obesity (n=30)	Central obesity (n=45)	p
Age [Mean±SD]	58.73 ± 18.43	61.71 ± 13.93	0.429[1]
Gender			**0.001[2]**
Female (n, %)	8 (26.7)	32 (71.1)	
Male (n, %)	22 (73.3)	13 (28.9)	
BMI [Median (min-max)]	26 (19–29)	31 (30–45)	**<0.001[3]**

1Student's t-test, 2Chi-square test, 3Mann-Whitney U test, p<0.05

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Table 2. Comparison of parameters between groups with and without central obesity

Laboratory parameters	No-central obesity Median (min-max)	Central obesity Median (min-max)	p
WBC [Mean ± SD]	10.88±3.78	12.62±4.39	0.079[2]
Lymphocytes	1.83 (0.5–10.3)	1.55 (0.5–14.3)	0.766[1]
Neutrophils [Mean ± SD]	7.86 ± 3.65	9.66 ± 3.96	0.051[2]
Platelets	240 (63–479)	264 (126–728)	**0.021[1]**
Urea	37 (7–109)	30 (10–104)	0.095[1]
Creatinine	0.9 (0.4-1.9)	0.8 (0.4–1.8)	0.006[1]
Glucose	118 (63–300)	140 (81–350)	0.062[1]
AST	68.5 (13–539)	147 (14–781)	0.108[1,**]
ALT	63 (5–559)	98 (11–1008)	0.328[1,**]
Total Bilirubin	1.0 (0.2–7.2)	1.1 (0.2–6.3)	0.901[1]
Direct Bilirubin	0.4 (0.1–5.4)	0.5 (0.1–4.1)	0.875[1]
Amylase	571.5 (105–3900)	929 (70–4030)	0.249[1]
Lipase	688 (34–5480)	700 (36–5400)	0.961[1]
Calcium [Mean ± SD]	8.89 ± 1.03	9.12 ± 0.65	0.248[2]
CK-MB	21.5 (12–151)	23 (9–180)	0.867[1]
Troponin	4.6 (2-85)	2.9 (1–75)	0.143[1]
Lactate	2.35 (1–5.3)	2 (1–6.7)	0.286[1]
INR	1.01 (0.9–1.5)	1.00 (0.8–1.4)	**0.019[1]**
APTT	26.25 (18–48.5)	23 (12–35)	**0.008[1]**
CRP	37.5 (3–211)	30 (3–211)	0.841[1]
Trıglycerıde	113 (48–1100)	100 (34–964)	0.249[1]
HDL [Mean±SD]	46.25 ± 15.16	47.72 ± 13.85	0.666[2]
LDL [Mean±SD]	98.57 ± 32.74	104.56 ± 40.89	0.504[2]
VLDL	23 (9.6–233)	20 (6–192)	0.122[1]
Cholesterol [Mean±SD]	196.63±143.23	167.26±46.42	0.204[2]

[1]Mann-Whitney U test, [2]Student's t-test, WBC: White Blood Cells, AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase

Table 3. Comparison of acute pancreatitis severity according to the ranson classification in patients classified as overweight (BMI 25–29.9 kg/m²) and obesity class I (BMI 30–34.9 kg/m²) based on body mass index

Group	Mild	Severe	p
Obesity class 1	33	7	**0.07**
Overweight	24	0	
Total	57	7	

Mild pancreatitis was defined as a Ranson score of 0–2, whereas severe pancreatitis was defined as a Ranson score of ≥3

Table 4. Comparison of acute pancreatitis severity according to the atlanta classification in patients classified as overweight (BMI 25–29.9 kg/m²) and obesity class I (BMI 30–34.9 kg/m²) based on body mass index

Group	Mild	Moderate	Severe	p
Obesity class 1	8	29	3	**0.0002**
Overweight	17	7	0	
Total	25	36	3	

Mann-Whitney U test

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Table 5. Comparison of groups according to ranson severity and CCI index comorbidity level

Variables	No-central obesity, n (%)	Central obesity, n (%)	p
Ranson			
Mild	30 (100.0)[a]	38 (84.4)[b]	0.037[1]
Severe	0 (0.0)[a]	7 (15.6)[b]	
Charlson Comorbidity Index			
0	6 (20.0)	4 (8.9)	
1-2	14 (46.7)	19 (42.2)	0.331[2]
3-4	9 (30.0)	21 (46.7)	
5 ≤	1 (3.3)	1 (2.2)	

[1]Fisher's exact test, [2]Fisher–Freeman–Halton exact test, a, b: Indicates the group(s) from which the difference originates

Table 6. Comparison of groups according to etiological causes

Etiological causes	No-central obesity, n (%)	Central obesity, n (%)	p
Hypertriglyceridemic	3 (10.0)	2 (4.4)	0.844
Biliary	9 (30.0)	15 (33.3)	
DPP-4 inhibitor use	2 (6.7)	4 (8.9)	
Idiopathic	16 (53.3)	24 (53.3)	

Fisher–Freeman–Halton exact test

Discussion

This study aimed to assess the influence of central obesity on the severity of AP. Our findings indicated that patients with central obesity exhibited significantly higher disease severity, as determined by both the Ranson score and the Atlanta classification, compared to those without central obesity.

A study compared patients who were overweight (BMI 25–29.9 kg/m²) without central obesity to those with Class I obesity (BMI 30–34.9 kg/m²) who had central obesity. The Ranson scores did not show any significant differences between these groups. However, using the Atlanta classification, it was found that the occurrence of moderate and severe acute pancreatitis was notably higher in patients with central obesity. Additionally, severe acute pancreatitis was only seen in the Class I obesity group according to the Atlanta criteria. These results indicate that relying solely on BMI may not be adequate for predicting the severity of acute pancreatitis, and central obesity might be a more crucial factor to consider.

The present study aligns with prior research suggesting that obesity exerts a significant influence on the prognosis and mortality of AP. However, we found that BMI alone was insufficient for determining AP severity according to the Atlanta classification, as severe pancreatitis did not develop in patients who were overweight based on BMI but did not have central obesity. According to the Atlanta classification, patients with Class 1 obesity, as defined by BMI and concomitant central obesity, exhibited a higher propensity for developing severe AP compared to those with Class 2 or Class 3 obesity. These findings underscore the notion that central obesity is a more significant predictor of health outcomes than BMI. Additionally, the absence of a substantial discrepancy between the groups with regard to the CCI suggests that the CCI may be an inadequate tool for evaluating the severity of AP. This finding indicates a potential need for refinement of the CCI as a diagnostic instrument.

As Johnson et al. [18] reported, visceral adiposity significantly exacerbates inflammation in AP. In a similar vein, Papachristou et al. [19] discovered that obese patients suffering from acute pancreatitis exhibited elevated levels of both CRP and Ranson scores. This finding suggests the possibility that AP may present with a more severe and potentially fatal course in these individuals.

In the present study, central obesity was found to be most prevalent among female patients across all patient groups (p=0.001), a finding that aligns with the results of the NHANES 2017–2018 survey [20].

Studies have reported elevated CRP levels in obese people [21, 22]. However, the findings of the present study did not align with these results. The observed discrepancy may be attributable to the fact that patient classification was based on central obesity as opposed to overall obesity. Additionally, some patients were prescribed anti-inflammatory medications for the management of pain.

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In the present study, PLT values were found to be elevated in the central obesity group, while INR and APTT values were reduced (p=0.021, p=0.019, and p=0.008, respectively). These findings are consistent with the data reported by Elena Campello et al. [23], which indicate an increased risk of hypercoagulability in obese individuals. This finding indicates that, in addition to elevated inflammation, central obesity may also be a contributing factor to an increased risk of vascular complications.

According to Sadr-Azodi et al. [24], individuals with central obesity have a higher risk of developing AP, although a clear association between overall obesity and AP onset has not been established. The present study yielded analogous results, indicating that, in accordance with the Atlanta classification, central obesity is a more significant predictor of acute pancreatitis severity than BMI.

Among the patient groups examined, it was notable that the most common etiological cause was "unknown." This finding indicates that MRCP performed in the early stages may be inadequate for detecting all instances of microlithiasis. Consequently, the employment of advanced diagnostic methodologies, such as endoscopic ultrasonography, is imperative to avert the underdiagnosis of biliary-related AP.

Conclusion

In the cohort of patients diagnosed with acute pancreatitis, the most prevalent BMI category within the central obesity group was identified as "obesity class 1" (88.9%), while 80% of individuals in the non-central obesity group were classified as "overweight." According to the Atlanta classification, patients with central obesity and a BMI of 30–34.9 kg/m² exhibited more severe AP compared to those with a BMI of 25–29.9 kg/m² and no central obesity. The limitations of the present study are as follows: first, the lack of EUS in identifying the etiology of AP, particularly in cases related to microlithiasis; second, the relatively small sample size; and third, the absence of central obesity among patients in the overweight group.

In summary, it is proposed that central obesity should be regarded as an independent risk factor in the clinical management of patients with AP and incorporated into early risk stratification.

Ethical Approval
Approved by the Non-Interventional Research Ethics Committee at Fırat University (No: 2024/11-06, Date: 01 August 2024).

Patient Consent
Informed consent was waived due to the retrospective and non-interventional design of the study.

Conflict of Interest
The authors declare no conflict of interest.

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