

Research article

Peritoneal cavity circumference on computed tomography predicts outcomes in acute pancreatitis

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ABSTRACT

Objectives: We investigated the role of novel intra-abdominal parameters measured by computed tomography (CT) in the prediction of clinical outcomes in acute pancreatitis (AP).

Methods: Patients with AP underwent an abdominal CT scan on admission to define different intra-abdominal parameters (abdominal circumference, peritoneal cavity circumference, intraabdominal visceral fat area, and subcutaneous fat area) at the L2-L3 level using the open-source image analysis software Osirix Lite v.11.0.4 to predict clinical outcomes.

Results: Eighty patients with AP were analyzed. Peritoneal cavity circumference (PCC) was the only variable independently associated with outcomes. PCC showed an area under ROC for prediction of severity in AP of 0.830. A PCC \geq 85 cm increased the risk of severity of AP (RR 15.7), persistent systemic inflammatory response syndrome (RR 9.3), acute peripancreatic fluid collection (RR 6.4), necrotizing pancreatitis (RR 21.50), and mortality (RR 2.4). We found a 4.7-fold increase in the risk of developing severe AP for each 10 cm increase in PCC.

Conclusions: PCC measurement at the L2-L3 level using a non-enhanced abdominal CT scan on admission in patients with AP is useful in the early prediction of severity, persistent systemic inflammatory response syndrome, local complications, and mortality.

1. Introduction

Acute pancreatitis (AP) is the most common gastrointestinal medical condition requiring hospital admission [1]. Patients with AP can present different spectra of disease severity from mild interstitial edematous pancreatitis to severe pancreatitis with local complications [2]. Acute necrotizing pancreatitis can occur in up to 20 % of cases and may be associated with organ failure (38 %), the need for intervention (38 %), and death (15 %) [3].

Currently, there are multiple scores and methods intended to predict

outcomes early in the evolution of acute pancreatitis (AP). Some scores are sophisticated combinations of predictive variables that could improve accuracy but are cumbersome to use, limiting their clinical application [4]. Existing predictive score systems on admission show modest accuracy [4,5]. Some image CT scores report a fairly high accuracy; however, the prediction is made late, 72 h after symptom onset [6]. Since AP is a complex disease and despite the existence of several criteria, it is not easy to predict its subsequent course because often, in patients with the same initial clinical and radiological scores, the clinical course of the disease varies.

Abbreviations: AP, Acute pancreatitis; CT, Computed tomography; VAT, Visceral adipose tissue; ERCP, Endoscopic retrograde cholangiopancreatography; SIRS, Systemic inflammatory response syndrome; Non-S, non-severe; CECT, contrast-enhanced computed tomography; AC, Abdominal circumference; PCC, Peritoneal cavity circumference; VFA, Visceral fat area; ScFA, Subcutaneous fat area; IQR, interquartile range; ROC, Receiver operating characteristic; AUROC, Area under ROC; PPV, Positive predictive value; NPV, Negative predictive value.

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Previous reports assessing the relationship between the severity of AP and intra-abdominal variables such as fat distribution on CT scan are controversial [7–9]. Obesity and particularly visceral adipose tissue (VAT) are implicated with an increased risk of organ failure, persistent systemic inflammatory response syndrome (SIRS), and worse prognosis [10,11]. VAT is found to be a metabolically highly active tissue that exerts a significantly greater inflammatory response than subcutaneous fat through the secretion of a distinct pattern of adipocytokines [12,13]. Furthermore, VAT has been identified to be more metabolically active at the L2 level than other segments [14]. This data interestingly suggests that the peripancreatic fat area at the L2-L3 level may play a key role in the inflammatory response and clinical evolution of AP.

We aimed to readdress the focus on other variables looking for an early and practical method to predict clinical outcomes in AP from admission. The early identification of patients likely to develop persistent organ failure and systemic and local complications is determinant in the decision to transfer patients to specialized centers for meticulous monitoring and management to improve outcome [15]. Therefore, we investigated the role of different intra-abdominal parameters measured by CT scan at the peripancreatic fat level of L2-L3 in the prediction of different clinical outcomes in AP.

2. Patients and methods

We carried out a prospective comparative study in patients with AP admitted from March 2015 to February 2016 to our hospital. The study was performed in compliance with the Declaration of Helsinki for biomedical research and was approved by the Ethics Committee of the School of Medicine and University Hospital. Informed consent was obtained from all participants.

2.1. Patients

Patients admitted during the study period were evaluated for inclusion criteria. The diagnosis of AP was based on the presence of at least two out of three of the following criteria: 1) typical abdominal pain consistent with AP; 2) serum amylase and/or lipase greater than three times the upper limit of normal; and/or characteristic findings on abdominal imaging (abdominal CT) [16]. Inclusion criteria were patients with a first episode of AP of any etiology presenting with less than 48 h since the onset of abdominal pain. All patients included in the study were assessed on admission with a non-enhanced short acquisition abdominal CT scan (Light Speed VCT CT99 scanner, General Electric Medical Systems, Milwaukee, WI, USA) extending from L1 to L4 (per protocol) or a full abdominal CT scan (contrast-enhanced or not) at the discretion of the attending physician (due to doubtful diagnosis or etiology or when rapid clinical deterioration was noted). We used a short acquisition abdominal CT scan exclusively for this study. Exclusion criteria for the study were pregnant women, patients with pancreatic calcifications detected on CT scan, patients with clinically evident ascites (grade 2), individuals with concurrent infectious process or diabetic ketoacidosis on admission (it could confound the clinical picture and results in SIRS), and referrals from other hospitals for the management of complications of AP.

The following variables were collected: demographics (age, gender), anthropometrical variables (size, weight, and body mass index), and comorbidities. Etiology of AP was classified in each case as gallstone, alcohol, hypertriglyceridemia, drugs, endoscopic retrograde cholangiopancreatography (ERCP), and others.

2.2. Severity of acute pancreatitis

The severity of AP (mild, moderately severe, and severe), persistent SIRS lasting ≥ 48 h, and the occurrence of local complications were based on the 2012 revision of the Atlanta classification and definitions [2]. Patients were evaluated every day by the attending

gastroenterologist from admission to discharge. An abdominal contrast-enhanced CT (CECT) scan was performed at the discretion of the attending gastroenterologist in those patients with persistent SIRS or organ failure after 7 days from the onset of AP looking for local complications.

We also evaluated the occurrence of acute respiratory distress syndrome (according to the Berlin classification) [17] and acute kidney injury (according to KDIGO definition and classification) [18] as a separate secondary outcome. The time of AP resolution was defined as the time in days since admission to the resolution of AP. The resolution of AP was defined as the resolution or absence of abdominal pain, organ failure, and SIRS in addition to adequate oral feeding tolerance.

2.3. Measurement of abdominal parameters

For the measurement of abdominal parameters and fat distribution, we analyzed abdominal CT scan images using the open-source image analysis software, Osirix Lite v.11.0.4, Fig. 1 A–I. The software allows the segmentation of abdominal tissues based on the Hounsfield value within an attenuation range of -190 to -30 HU which is considered representative of fat density [19,20]. We determined the following parameters: abdominal circumference (AC), peritoneal cavity circumference (PCC), intraabdominal visceral fat area (VFA), and subcutaneous fat area (ScFA). The measurement was made from a single CT scan image at the L2-L3 level; the visceral adipocytes within the omentum at this L2-L3 level are more metabolically active compared to other regions [21,22].

2.4. Statistical analysis

For the purpose of this analysis and to identify variables to predict severe AP cases, we arbitrarily divided the patients into two study groups as follows: mild and moderate-severe classes in the 2012 Atlanta classification were unified in a single group named the “non-severe” (non-S) AP group and the severe class persisted as the “severe” AP group.

Sample size was calculated using a two-proportions difference with a confidence level of 95 % and a power of 80 %. The sample calculation was based on estimating a 30 cm² area difference in intra-abdominal parameters (visceral fat area) between non-S and severe groups. An n of 21 patients per group was estimated. A 2:1 proportion between non-S and severe cases was determined. Once the number of non-S cases was achieved, we included afterward just severe AP cases until the 2:1 proportion was reached.

Statistical analyses were performed with SPSS 20.0 (IBM, Armonk, NY, USA). The normality of the studied variables was assessed with the Kolmogorov-Smirnov test. Continuous variables were expressed as mean \pm standard deviation or median and interquartile range (IQR), depending on whether they were normally distributed; categorical variables were expressed as numbers and percentages. Continuous variables were compared using Student's *t*-test or the Mann-Whitney test, when appropriate. Categorical variables were compared with Fisher's exact test. We used the Spearman rho test to assess the correlation between continuous variables. Logistic regression was used to evaluate the association between the severity of AP and intra-abdominal parameters measured by CT scan. We used univariate models to evaluate individual associations and multivariate models to adjust for confounders and variables of interest. We evaluated the goodness of fit for our model using the Hosmer-Lemeshow test and tested for multicollinearity using the Variance Inflation Factor. Receiver operating characteristic (ROC) curve analysis was applied to evaluate the performance of selected variables for the prediction of severe AP and persistent SIRS, and the area under the ROC curve (AUROC) was calculated. For the best cutoff value selected by ROC analysis, we determined the sensitivity, specificity, positive (PPV) and negative predictive values (NPV). A P value < 0.05 was considered statistically significant.

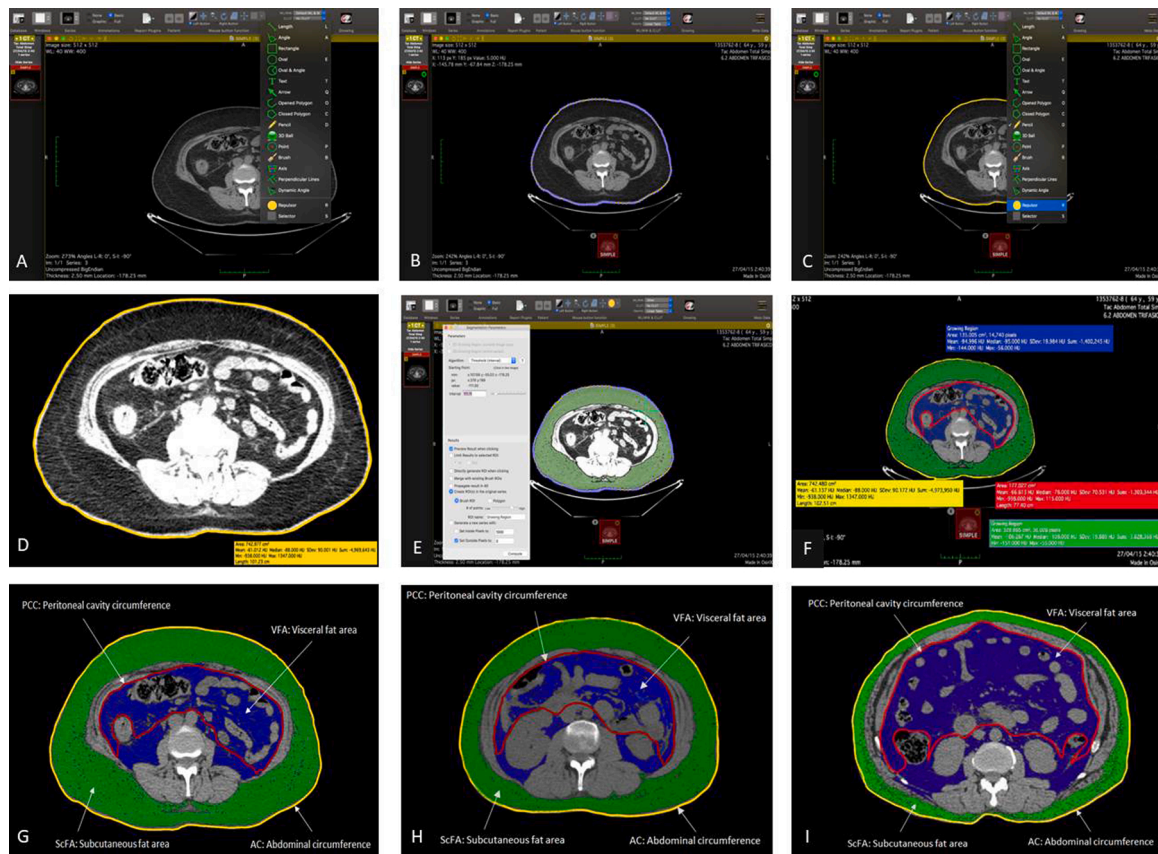


Fig. 1. Methodology for segmentation and measurement of intra-abdominal parameters on CT scan images.

The selected images are imported into the Osirix Lite program in DICOM format (Fig. 1A). The “Pencil” tool is selected from the toolbar and then the contour of the abdominal circumference (AC) is delimited freehand (Fig. 1A-B). The “Repulsor” tool is used to correct irregularities or a lack of precision when delimiting the contour freehand and then the software gives us the measurement results (Fig. 1C-D). Measurement of the subcutaneous fat area (ScFA) and the visceral fat area (VFA) is performed using the “Growing Region” tool by clicking on the subcutaneous and visceral fat area, respectively. A window of “segmentation parameters” is automatically displayed where we can control the attenuation range representative of fat density, thus obtaining coloring and a measurement of the area of interest (Fig. 1E-F). The peritoneal cavity circumference (PCC) is delimited freehand as mentioned before for AC (Fig. 1F). In the particular case of visceral fat, a small variation in density can be observed due to the inflammatory process caused by acute pancreatitis. In this case, multiple individual segmentations can be made in the area of interest using the “Growing Region” tool, then merging and obtaining a single reliable measurement of the area of interest. We showed examples of segmentation and measurement of abdominal CT images in three different cases (Fig. 1G, H, I). The yellow line represents the AC; the red line outlines the PCC; the green and blue areas represent the ScFA and VFA, respectively.

3. Results

A total of 89 patients with a first episode of AP admitted to our hospital were considered for analysis. We excluded 2 patients from the analysis because of chronic pancreatitis (pancreatic calcifications), 2 patients had a different final diagnosis other than pancreatitis, 3 patients were transferred to an outside hospital, 1 patient presented infectious pancreatitis due to salmonella, and finally, 1 with diabetic ketoacidosis at presentation. Eighty patients (54 females and 26 males) were included in the final analysis. The mean age of patients was 39.4 years ± 17.7 (range 15–83 years). The main etiology of AP was biliary disease (78.8 %) and hypertriglyceridemia (11.3 %). Of the 80 patients, 54 were classified as non-S AP and 26 as severe AP. According to the presence of SIRS, 40 (50 %) patients developed persistent SIRS, and 40 (50 %) presented non- or transient SIRS, Table 1.

We compared the demographic data, body mass index, comorbidities, and abdominal parameters measured on abdominal CT scan between patients with severe AP and those with non-S AP and also between patients with persistent SIRS and those with non- or transient-SIRS. Age and the number of comorbidities were significantly higher in those with severe AP and those with persistent SIRS compared with patients with non-S AP and non- or transient-SIRS, respectively, Table 2. Regarding abdominal parameters measured on CT scan, AC, PCC, and VFA were

Table 1

Patients characteristics.

Age, years (mean ± SD)	39.4 ± 17.7
Gender (Female/Male)	54 / 26
BMI, kg/m ² (mean ± SD)	28.4 ± 5.9
No. Comorbidities (median [IQR])	1.0 (2.0)
Diabetes mellitus — no. (%)	19 (23.8)
Hypertension — no. (%)	18 (22.5)
Dyslipidemia — no. (%)	32 (40.0)
Etiology	
Biliary — no. (%)	63 (78.8)
Hypertriglyceridemia — no. (%)	9 (11.3)
Alcohol — no. (%)	4 (5.0)
Post-ERCP — no. (%)	1 (1.2)
Drugs — no. (%)	2 (2.5)
Hypercalcemia — no. (%)	1 (1.2)
Severity of AP	
Non-S — no. (%)	54 (67.5)
Severe — no. (%)	26 (32.5)
Persistent SIRS — no. (%)	40 (50.0)
Mortality — no. (%)	5 (6.2)

BMI: body mass index; ERCP: Endoscopic retrograde cholangiopancreatography; non-S: non-severe; SIRS: Systemic inflammatory response syndrome.

Table 2

Univariate analysis. Comparison of variables between patients with Severe and Non-S acute pancreatitis and also between patients with persistent SIRS and non- or transient-SIRS.

	Severity of AP		P	Persistent SIRS		P
	Severe n = 26	Non-S n = 54		Yes n = 40	No n = 40	
Age, years (mean ± SD)	47.7 ± 14.6	35.3 ± 17.7	0.003	43.5 ± 18.0	35.2 ± 16.6	0.036
Gender (Female/male)	16/10	38/16	0.454	24/16	30/10	0.232
Weight, kg (mean ± SD)	80.0 ± 14.9	73.0 ± 17.8	0.055	78.1 ± 14.6	72.9 ± 19.4	0.184
BMI, kg/m ² (mean ± SD)	30.2 ± 4.9	27.5 ± 6.2	0.056	29.3 ± 5.3	27.6 ± 6.4	0.200
No. Comorbidities (median [IQR])	2 (1.0)	1 (1.0)	0.004	1.0 (1.75)	0.5 (1.0)	0.002
Diabetes mellitus — no. (%)	10 (38.5)	9 (16.7)	0.049	15 (37.5)	4 (10.0)	0.008
Hypertension — no. (%)	8 (30.8)	10 (18.5)	0.259	12 (30.0)	6 (15.0)	0.180
Dyslipidemia — no. (%)	13 (50.0)	19 (35.2)	0.231	19 (47.5)	13 (32.5)	0.254
Etiology			0.453			0.130
Biliary — no. (%)	18 (69.2)	45 (83.3)	0.159	28 (70.0)	35 (87.5)	0.099
Hypertriglyceridemia — no. (%)	5 (19.2)	4 (7.4)	0.142	6 (15.0)	3 (7.5)	0.481
Alcohol — no. (%)	2 (7.7)	2 (3.7)	0.592	4 (10.0)	0 (0)	0.116
AC, cm (mean ± SD)	105.6 ± 11.2	95.1 ± 14.2	0.002	101.9 ± 12.8	95.1 ± 14.7	0.032
PCC, cm (mean ± SD)	94.7 ± 11.6	78.8 ± 12.1	<0.001	90.0 ± 12.3	78.0 ± 13.3	<0.001
VFA, cm ² (mean ± SD)	176.2 ± 84.4	123.3 ± 80.6	0.008	159.1 ± 80.6	121.9 ± 86.3	0.049
ScFA, cm ² (mean ± SD)	212.2 ± 86.7	192.6 ± 121.2	0.466	200.5 ± 103.6	197.3 ± 119.2	0.900

Bold characters represent statistical significance.

Non-S: non-severe; BMI: Body mass index; AC: Abdominal circumference; PCC: Peritoneal cavity circumference; VFA: Visceral fat area; ScFA: Subcutaneous fat area.

also significantly higher in those with severe AP and persistent SIRS compared with non-S AP and non- or transient-SIRS patients, respectively, **Table 2**. A significant correlation between PCC and VFA was found, $r = 0.770$, $P < 0.001$.

On multivariate analysis, PCC was the only variable independently associated with the occurrence of severe AP and persistent SIRS, **Table 3**. A ROC curve analysis was used to evaluate the performance of PCC measured by abdominal CT scan to predict the occurrence of severe AP and persistent SIRS on admission. The optimal cutoff value of PCC for the prediction of severe AP was 85 cm, with an AUROC of 0.830 ($p < 0.001$), sensitivity of 85 %, and specificity of 74 % (PPV 61 % and NPV 91 %); the optimal cutoff value for PCC for the prediction of severe AP and or persistent SIRS was 85 cm, with an AUROC of 0.810 ($p < 0.001$), sensitivity 71 %, and specificity 84 % (PPV 84 % and NPV 73 %).

Considering the best cutoff value of 85 cm, we compared different clinical outcomes between three groups according to PCC: < 70 cm, 70–84.9 cm, and ≥ 85 cm. Those with a PCC ≥ 85 cm had a significantly higher rate of severity, persistent SIRS, local complications, and mortality, **Fig. 2**.

A contrast-enhanced abdominal CT scan was performed in 15 patients at the discretion of the attending gastroenterologist in those

Table 3

Multivariate analysis. Risk factors for severity and persistent SIRS in acute pancreatitis.

	Severe AP			Persistent SIRS		
	RR	95 % IC	P	RR	95 % IC	P
Age, years	1.016	0.975 – 1.058	0.448	0.991	0.955 – 1.028	0.617
BMI, kg/m ²	0.878	0.733 – 1.053	0.160	NA	NA	NA
Number of comorbidities	1.215	0.594 – 2.486	0.593	1.518	0.712 – 3.238	0.280
Diabetes mellitus	NA	NA	NA	1.968	0.420 – 9.229	0.390
AC, cm	1.069	0.980 – 1.165	0.132	NA	NA	NA
PCC, cm	1.111	1.029 – 1.198	0.007	1.058	1.012 – 1.107	0.012
VFA, cm ²	0.994	0.983 – 1.004	0.240	NA	NA	NA

Bold characters represent statistical significance. BMI: Body mass index; NA: Not applicable; AC: Abdominal circumference; PCC: Peritoneal cavity circumference; VFA: Visceral fat area.

patients with persistent SIRS or organ failure after 7 days from AP onset looking for local complications. Thirteen patients out of these 15 presented necrotizing pancreatitis. Of those 13 patients with necrotizing pancreatitis, 12 (92.3 %) presented a PCC ≥ 85 cm on the abdominal CT scan performed on admission.

We also evaluated the occurrence of acute respiratory distress syndrome (according to Berlin classification) and acute kidney injury (according to KDIGO definition and classification). Those with a PCC ≥ 85 cm had a significantly higher rate of ARDS and AKI, **Fig. 3**. The correlation analysis (Spearman Rho) between PCC and respiratory (PaO₂/FiO₂) and renal function (creatinine) at 48 h from admission were as follows: PaO₂/FiO₂, $r = -0.605$, $P < 0.001$; and creatinine, $r = 0.457$, $P < 0.00$. No patients with a PCC ≥ 85 cm presented normal respiratory function (paOFiO₂ ≥ 400) and all patients with a PCC < 85 cm showed normal renal function (creatinine ≤ 1.4 mg/dL). There was a significant correlation between the PCC and the time of AP resolution, $r = 0.337$, $P = 0.004$. The median (IQR) time of AP resolution was shorter in patients with a PCC < 85 cm than those ≥ 85 cm (5 days [3.0] vs 7 days [10.0], $P = 0.004$).

The cutoff of PCC ≥ 85 cm increased the risk of developing severe AP (RR 15.714, 95 % CI 4.607–53.598), persistent SIRS (RR 9.333, 95 % CI 3.338–26.101), acute peripancreatic fluid collection (RR 6.364, 95 % CI 1.866–21.705), necrotizing pancreatitis (RR 21.500, 95 % CI 2.632–175.614), and mortality (RR 2.419, 95 % CI 1.848–3.168). After adjusting for age, BMI, comorbidities, gender, VFA, and AC, we found a statistically significant association between the severity of AP and PCC (OR 4.72, 95 % CI 1.734–12.871). We found a 4.7-fold increase in the risk of developing severe pancreatitis for each 10-cm increase in PCC, **Fig. 4**.

4. Discussion

PCC measured by a non-enhanced short acquisition abdominal CT scan is a novel highly reproducible method not previously described that predicts clinical outcomes in AP on admission, such as severity, persistent SIRS, local complications, and mortality. To our knowledge, this is the first prospective study that predicts outcomes through a practical and easy reproducible parameter on admission in patients with their first episode of AP with less than 48 h of clinical evolution. Patients with a PCC ≥ 85 cm have a high risk of morbidity and mortality, so these patients should be transferred to specialized centers or area for meticulous monitoring and management to improve outcomes.

Previous reports assessing the relationship between the severity of

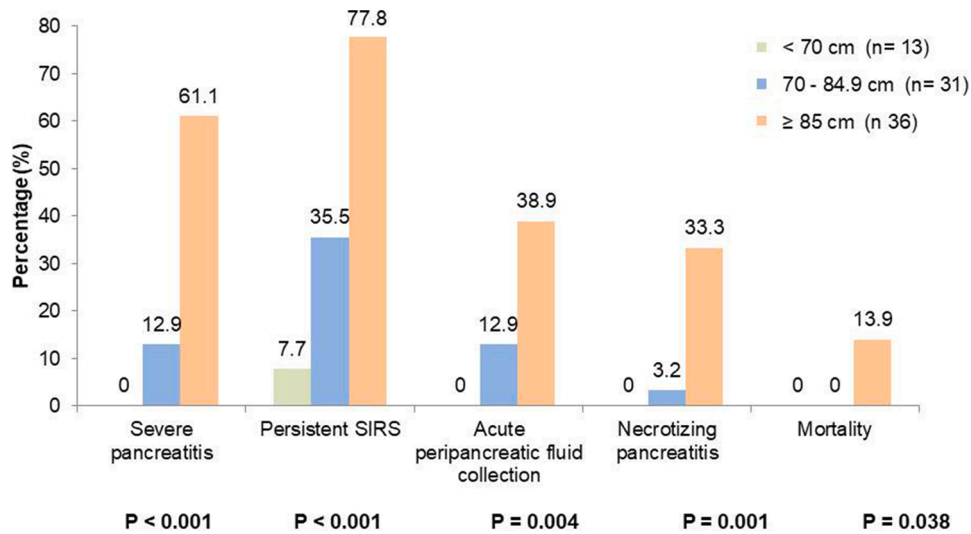


Fig. 2. Comparison of complications rates of acute pancreatitis between different peritoneal cavity circumference (PCC) groups.

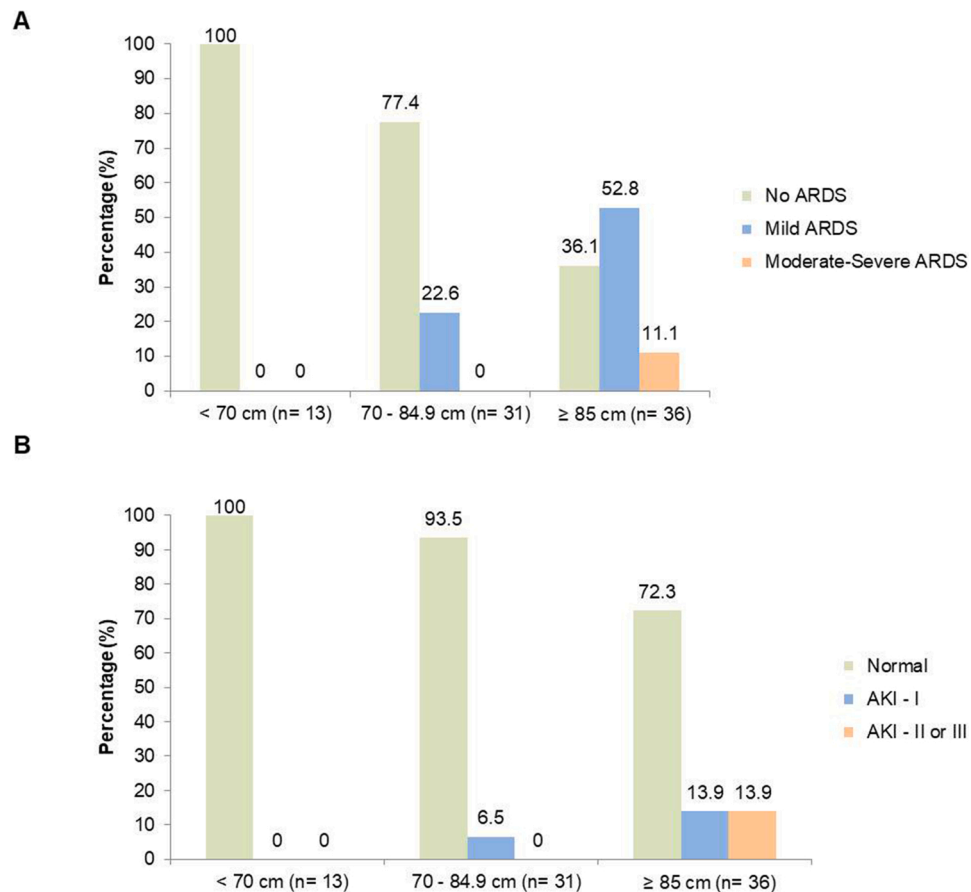


Fig. 3. A. Comparison of respiratory function in acute pancreatitis according to peritoneal cavity circumference, P < .001. B. Comparison of renal function in acute pancreatitis according to peritoneal cavity circumference, P < 0.01.

AP and intra-abdominal variables such as fat distribution on CT scan are controversial. O’Leary et al. showed a significant association between the visceral fat volume and the occurrence of severe AP (p = 0.003), systemic complications (p = 0.003), and mortality (p = 0.019) [9]. However, this association did not persist as significant in the multivariate analysis. In addition, they did not specify the time at which the CT scan was performed during hospitalization. We believe this is an important limitation because it may be an important variation in the

measurement of fat volume and other anthropometric parameters at different times of AP evolution. In another study, Hall et al. [8] retrospectively assessed abdominal fat distribution (subcutaneous, retroperitoneal, and intra-abdominal) with severity and mortality in AP; however, there was not a significant association between fat distribution and these clinical outcomes [8]. They, however, reported a significant association between retroperitoneal fat an intra-abdominal fat with several scores of severity [8]. In another retrospective study, Yashima

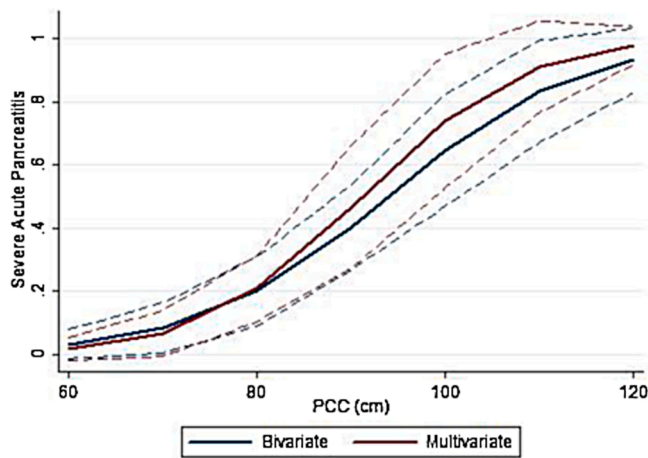


Fig. 4. Risk of severe acute pancreatitis based on PCC measure by CT scan. The output is the unadjusted and adjusted (age, gender, BMI, comorbidities, AGV, and AC) regression coefficient plots. The y-axis is the proportion of patients who will have severe pancreatitis on a scale of 0% to 100 % based on the PCC represented in the x-axis on a 10 cm scale interval (60 to 120 cm). The solid lines represent bivariate (blue line) and multivariate (red line) analysis; dashed lines are the 95 % CI.

et al. reported that VAT was independently associated with severity in AP (OR 1.02, 95 % IC 1.01–1.03, $p = 0.003$). They reported VAT values $>100 \text{ cm}^2$ were significantly associated with poor clinical evolution and outcome [7].

VAT is metabolically active tissue with a greater activity correlation at the L2 level than other intra-abdominal segments [14]. This data suggests that peripancreatic visceral fat at the L2-L3 level may play a greater role in the inflammatory response in AP than other fat segments. The fact that the PCC was the only parameter independently associated with outcomes in our study, made us believe that other intraabdominal variables exist more than just fat distribution contributing to the development of severity in AP and that directly contribute to PCC size measurement.

A case-control study quantifying the intestinal gas using plain abdominal radiographs within the first 24 h of hospital admission in patients with AP demonstrated a higher intestinal gas quantity in those with AP compared with controls, and in severe AP compared with mild AP cases [23]. The amount of intestinal gas correlates with the severity of AP (assessed by Ranson score: $r = 0.762$, $P < 0.01$; and APACHE II score: $r = 0.801$, $P < 0.01$), and is also associated with local complications (pancreatic or peripancreatic infections) [23]. The larger amount of intestinal gas might be explained by alterations in gut function and peristalsis secondary to the inflammatory response [24,25]. The increased level of intestinal gas may reflect the development of small intestinal bacterial overgrowth secondary to a delay in intestinal transit in AP [24]. This in consequence may contribute to the development of intra-abdominal hypertension favoring an increase in intestinal permeability to bacteria and endotoxins [26,27].

Because of these alterations in gastrointestinal motility, a change in the intestinal gas pattern, and intestinal permeability present in the early phase of AP, our findings are quite relevant because we could predict on admission the clinical evolution of patients by measuring PCC on a non-enhanced short acquisition abdominal CT scan. Based on our results and previous reports, the PCC reflects the VFA and intestinal gas content and seems to be an accurate and early surrogate of the dynamic and pathophysiological changes occurring in the peripancreatic intra-peritoneal visceral fat and gut function.

Our study presented several limitations. The number of patients in this study was small; future studies with a large number of patients are needed to validate our findings and draw safe conclusions. We did not correlate other intra-abdominal parameter segments with clinical

outcomes. We neither determined the amount of intestinal gas in the selected CT scan images; in addition, other parameters such as organ size or volume that could influence final PCC measurement, were not assessed. We did not demonstrate that comorbidities such as diabetes, hypertension, or dyslipidemia had an association with severity; these may be explained by the fact that our population was younger (39.4 ± 17.7 years of age) and had a shorter period of comorbidity evolution compared with other reports [7–10].

In summary, PCC measurement at the L2-L3 level using a non-enhanced short acquisition abdominal CT scan on admission in patients with AP is useful in the early prediction of severity, persistent SIRS, local complications, and mortality.

CRedit authorship contribution statement

Roberto Monreal-Robles: Conceptualization, Formal analysis, Methodology, Supervision, Validation, Writing - original draft, Writing - review & editing. **Ana E. Kohn-Gutiérrez:** Investigation, Software, Writing - review & editing. **José Sordia-Ramírez:** Data curation, Investigation. **Julian A. Zúñiga-Segura:** Data curation, Investigation. **Javier A. Palafox-Salinas:** Investigation, Software, Writing - review & editing. **Sylvia de la Rosa-Pacheco:** Methodology, Formal analysis. **Guillermo Elizondo-Riojas:** Project administration, Resources, Investigation. **José A González-González:** Conceptualization, Investigation, Supervision, Validation.

Declaration of Competing Interest

The authors declare that they have no conflicts of interest or financial disclosures. They also agree to allow the corresponding author to serve as the primary correspondent with the editorial office, to review the edited typescript, and proof.

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