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Impact of Hepatic Steatosis, Psoas Muscle Mass, and Density on Outcomes in Patients with Moderately Severe and Severe Acute Pancreatitis: A Serial Computed Tomography-Based Retrospective Study

Akhil Jerath[1] Nishita Rewari[1] Pankaj Gupta[1] Vaneet Jearth[2] Anupam Singh[2] Jimil Shah[2] Jayanta Samanta[2] Harshal Mandavdhare[2] Vishal Sharma[2] Saroj K. Sinha[2] Usha Dutta[2]

[1] Department of Radiodiagnosis and Imaging, Postgraduate Institute of Medical Education and Research, Chandigarh, India
[2] Department of Gastroenterology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

Address for correspondence Pankaj Gupta, MD, Department of Radiodiagnosis and Imaging, Postgraduate Institute of Medical Education and Research, Chandigarh, India (e-mail: Pankajgupta959@gmail.com).

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Abstract

Background and Objective There is limited literature on the association of fatty liver and muscle mass with outcomes of acute pancreatitis (AP) in Indian patients. We aim to investigate the impact of these parameters on clinical outcomes in AP patients based on serial computed tomography (CT) scans.

Materials and Methods Consecutive patients of AP who had a baseline CT scan within 2 weeks of pain onset and a follow-up CT scan within 2 months of initial CT scan were included. Fatty liver, spleen to liver attenuation difference (AD), psoas muscle area (PMA), and psoas muscle attenuation (PMA_t) were assessed on baseline and follow-up portal venous CT scans. Their association with the length of hospitalization, intensive care unit (ICU) admission, length of ICU stay, surgery, and mortality were assessed using univariate and multivariate analysis.

Results One hundred and ninety-eight patients (143 [72.2%] males, mean age 37.9 [$\pm$12.4] years) were included. The mean interval between the two CT scans was 30 days ($\pm$13.5 days). Twenty-four (12.1%) patients had fatty liver at baseline CT and 58 (29.3%) patients had fatty liver on follow-up CT ($p < 0.001$). The mean AD at the baseline scan was −10.2 ($\pm$12.3) Hounsfield units (HU) and on follow up was −18.8 ($\pm$21.7) HU ($p < 0.001$). PMA in baseline CT was 13.9 ($\pm$5.5) mm^2 and on follow-up was 11.2 ($\pm$4.6) mm^2 ($p < 0.001$). PMA_t on baseline CT was 49.2 ($\pm$8.2) HU and on follow-up was 47.1 ($\pm$12.1) HU ($p < 0.001$). In multivariate analysis, the PMA and PMA_t were significantly associated with the clinical outcomes. There was no association of fatty liver with clinical outcomes. The parameter with the highest area under the curve for predicting clinical outcomes was PMA_t at follow-up CT.

Conclusion Psoas muscle-based assessment is associated with clinical outcomes in patients with AP.

Keywords
- acute pancreatitis
- computed tomography
- fatty liver
- psoas muscle area
- sarcopenia

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Introduction

Acute pancreatitis (AP) is an inflammatory condition of the pancreas having various etiologies.[1] The incidence rate of the disease is on the rise ranging from 13 to 45 cases per 100,000 people with a gradual increase in hospitalization rate. AP generally follows a mild course, but in 20% of the patients, the disease has a moderately severe to severe course, which is associated with significant morbidity. Severe AP has 10 to 20% mortality.[2,3]

Sarcopenia is defined as generalized progressive loss of the skeletal muscle mass. It is termed primary in cases of sarcopenia due to aging and secondary in cases due to chronic illnesses.[4] Computed tomography (CT) imaging is an important method of accurately quantifying skeletal muscle parameters and detecting sarcopenia. Abdominal CT is performed in almost all patients of moderately severe to severe AP at admission and during hospitalization. These CT scans can delineate the parameters like psoas muscle area (PMA) and psoas muscle attenuation, which are indicators of sarcopenia. A recent study reported the association of sarcopenia with worse CT severity scores and larger pancreatic necrosis.[5] Sarcopenia in patients of AP is associated with decreased overall survival, prolonged hospitalization, and higher postoperative complication rate.[5]

Hepatic steatosis is seen in cases of alcoholic and nonalcoholic fatty liver diseases. It is seen frequently in gastrointestinal and pancreaticobiliary diseases, including patients of AP due to the sharing of various factors like ethanol use, diabetes mellitus type II, obesity, and dyslipidemia contributing to its cause.[6] A few previous studies have reported association between fatty liver and severity of AP.[4,6,7] There is limited literature on the association between fatty liver and other clinical outcome of the AP.

This study aimed to investigate the impact of hepatic steatosis and psoas muscle mass and density on the severity and outcomes of AP.

Materials and Methods

Subjects and Study Design

The institute ethics committee approved this single-center retrospective study and the need to obtain informed written consent was waived. Consecutive patients of AP who underwent CT scans between January 2019 and December 2022 were considered for inclusion. Patients who had a baseline CT scan within 2 weeks of pain onset (baseline CT in our center is usually performed between 5 and 7 days) and a follow-up CT scan within 2 months of initial CT scan were considered for inclusion (►**Fig. 1**). Patients with chronic pancreatitis, acute on chronic pancreatitis, and pancreatitis due to pancreatic cancer were excluded.

Baseline Parameters

The age, gender, cause of AP, and severity of AP (based on revised Atlanta classification [RAC]) were assessed.

CT Evaluation

The CT images of the patients were retrieved from the radiology database. CT scans were evaluated by two radiologists (2 years of posttraining experience and a 3rd-year radiology resident) in consensus. The baseline CT scans were assessed for the features of AP and graded according to the modified CT severity index (CTSI) scoring.[8] Liver attenuation was evaluated. In normal patients, the liver has a high attenuation compared to the spleen but hepatic steatosis leads to a reversal of spleen-to-liver attenuation difference. The attenuation of the liver and spleen in Hounsfield units (HU) was assessed by placing five regions of interest (ROIs) of $\geq 1\ cm^2$ on the liver involving different segments and three ROIs on the spleen. These calculations were done on portal venous phase contrast-enhanced CT (►**Fig. 2**). The mean hepatic and splenic attenuation was calculated by taking the arithmetic mean of these values. The spleen-to-liver attenuation difference was calculated by subtracting the liver HU value from the splenic HU. Fatty liver was defined when

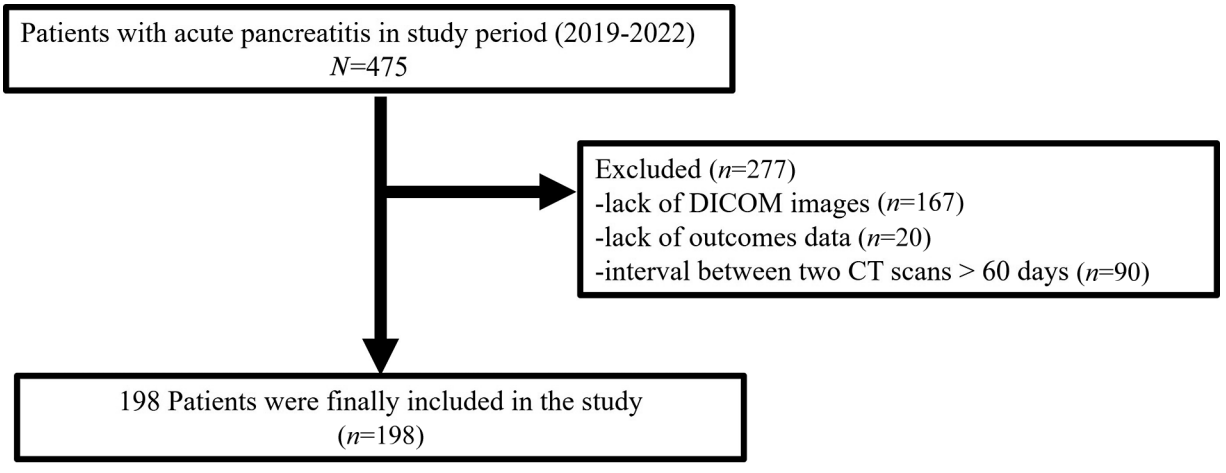

Fig. 1 Flow diagram showing the patients' recruitment.

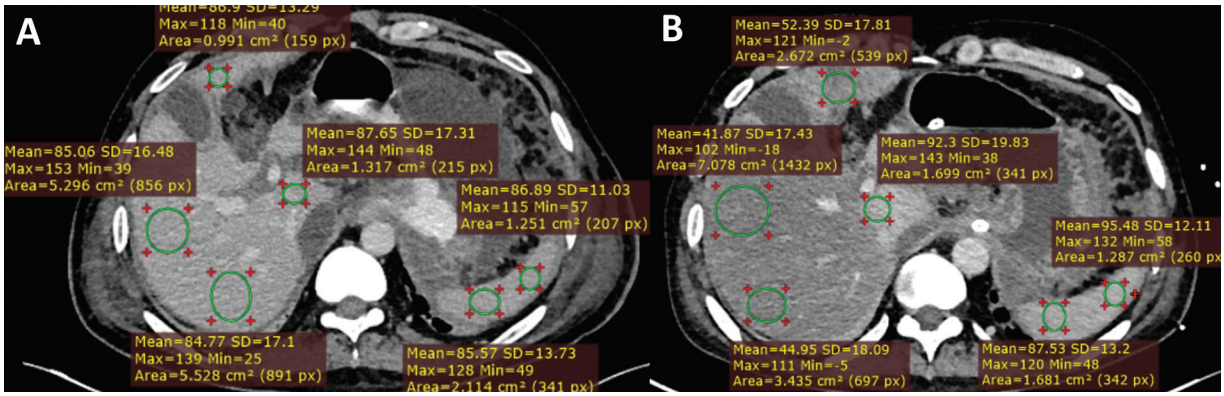

Fig. 2 Contrast-enhanced computed tomography (CECT) scans of a 46-year-old male patient with acute necrotizing pancreatitis. (**A**) Baseline CECT shows spleen-to-liver attenuation difference of 1 Hounsfield units (HU). (**B**) Follow-up CECT of the same patient obtained after 20 days shows spleen-to-liver attenuation difference of 35 HU suggesting fatty change in the liver.

spleen-to-liver attenuation difference was more than 20 HU.[9,10]

Sarcopenia was evaluated by calculating PMA and psoas muscle attenuation on a CT section at the lower border of the L3 vertebra (►**Figs. 3** and **4**). The areas of both psoas muscles were manually measured by radiologists by outlining the psoas muscle and the total PMA was given by adding the two (in square cm). Muscle attenuation was calculated by

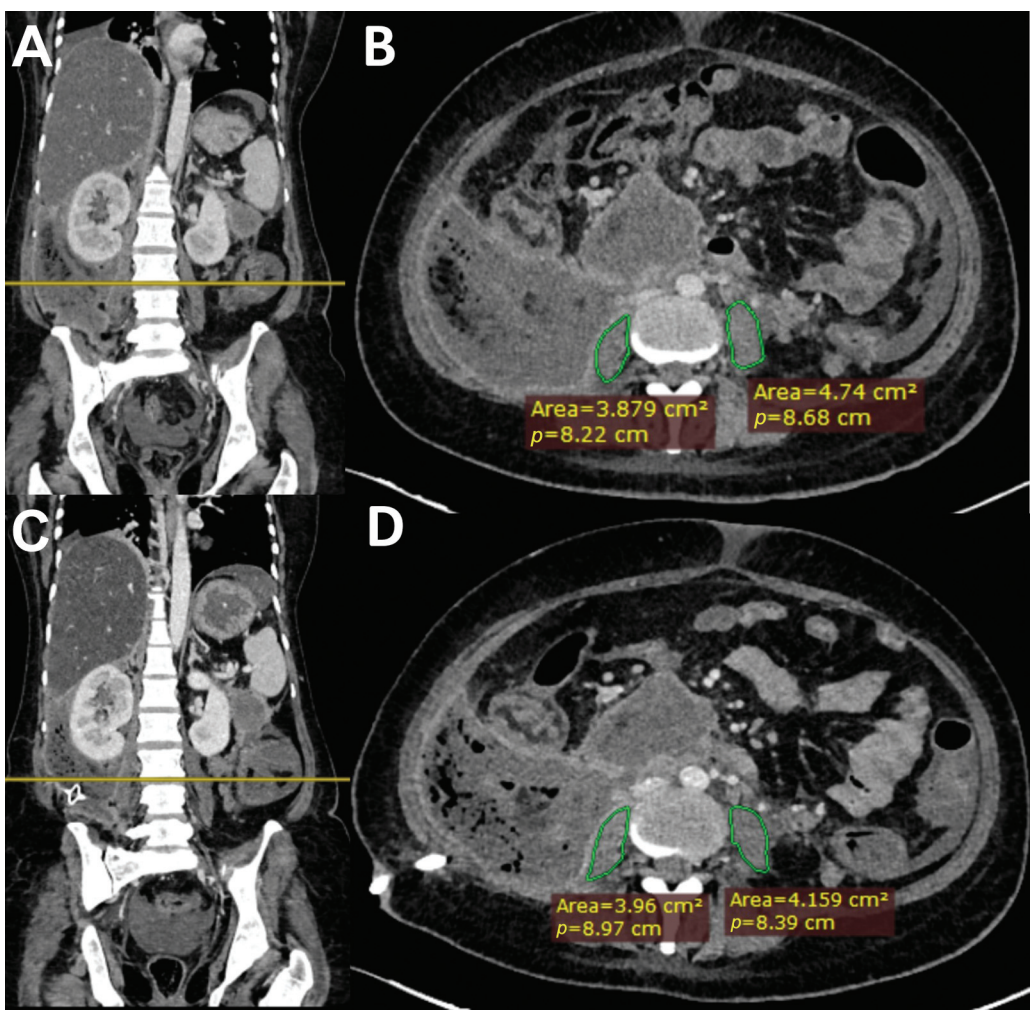

Fig. 3 Contrast-enhanced computed tomography (CECT) scans of a 49-year-old female with acute necrotizing pancreatitis. Psoas muscle area was calculated by drawing closed polygons around both the right and left psoas muscle at the lower border of the L3 vertebra. (**A**) and (**B**) show coronal and axial images of the baseline CECT of the patient. The mean psoas muscle area at baseline CECT was 4.31 cm^2. (**C**) and (**D**) show coronal and axial images of follow-up CECT of the same patient obtained after 18 days. The mean psoas muscle area was reduced to 4.06 cm^2.

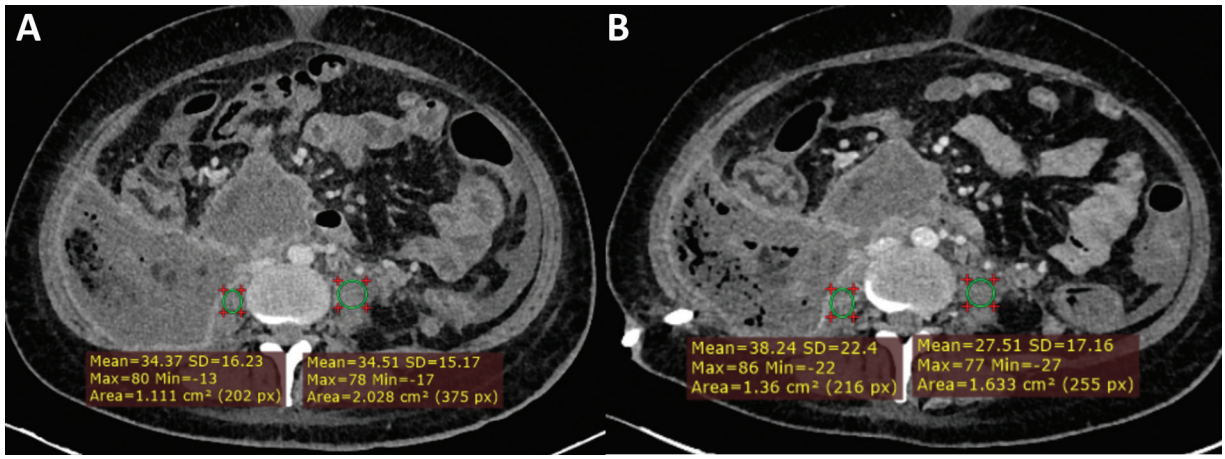

Fig. 4 Contrast-enhanced computed tomography (CECT) scans of the same patient as in ►**Fig. 3**. Psoas muscle attenuation was calculated by drawing regions of interest (ROIs) in both the right and left psoas muscles at the lower border of the L3 vertebra. (**A**) shows an axial image of the baseline CECT scan. The mean psoas muscle attenuation was 34.4 Hounsfield units (HU). (**B**) shows an axial image of a follow-up CECT scan obtained after 18 days. The mean psoas muscle attenuation was reduced to 32.5 HU.

drawing two ROIs (one in each psoas muscle), measuring ≥ 1 cm^2. Mean attenuation was calculated by taking the arithmetic mean of these values of the right and left sides and represented in HU values.

The same calculations were then repeated on the follow-up scan.

Clinical Outcomes

Length of hospitalization, intensive care unit (ICU) admission, length of ICU stay, surgery, and mortality were recorded.

Statistical Analysis

Statistical analysis was carried out using IBM Statistical Package for the Social Sciences Statistics, version 29, and MedCal, version 23. The categorical variables were reported as proportion and percentages. The continuous variables were expressed as mean with standard deviation. Categorical variables were compared using the chi-square test or Fischer's exact test. Quantitative variables were compared using the Student's *t*-test or Mann–Whitney *U* test. Paired quantitative variables were compared using paired *t*-tests. The correlation was measured using Pearson's or Spearman's correlation based on the data distribution. Receiver operating characteristics (ROC) curves were plotted to assess the area under the curve (AUC). Multivariable analysis was performed using linear or logistic regression. All statistical analyses were carried out at a 5% level of significance and a two-tailed *p*-value of < 0.05 was considered significant.

Results

Baseline Characteristics

One hundred and ninety-eight patients met the inclusion criteria (►**Fig. 1**). There were 143 (72.2%) males and 55 (27.8%) females. The mean age was 37.9 (± 12.4) years. The major causes of AP were alcohol (45.5%) and gallstones (41.9%). Among the 198 patients, 83 (41.9%) had moderately severe and 115 (58.1%) had severe AP. The mean CTSI was 8.8 ± 1.6. The mean interval between the two CT scans in our study was 30 days (± 13.5 days).

Twenty-four (12.1%) patients had fatty liver at baseline CT and 58 (29.3%) patients had fatty liver on follow-up CT ($p < 0.001$). The mean attenuation difference at the baseline scan was –10.2 (± 12.3) HU and on the follow-up scan was –18.8 (± 21.7) HU with a mean difference of 8.47 HU, which was statistically significant ($p < 0.001$). PMA in baseline CT was 13.9 (± 5.5) mm^2 and on follow-up CT was 11.2 (± 4.6) mm^2 with a mean difference of 2.64 mm^2, which was statistically significant ($p < 0.001$). The mean attenuation of psoas muscle on baseline CT was 49.2 (± 8.2) HU and on follow-up was 47.1 (± 12.1) HU with a mean difference of 2.09 HU, which was statistically significant ($p < 0.001$).

The mean hospital stay was 37.6 (± 22.9) days. Out of these patients, 100 patients (50.5%) required ICU admission. The mean ICU stay was 7.9 (± 12.2) days. Of the 198 patients, 30 (15.2%) underwent surgery and 56 (28.3%) succumbed to the disease (►**Table. 1**).

Association with Outcomes (►Tables 2–4)

Fatty Liver

Fatty liver at baseline or follow-up CT was not associated with clinical outcomes.

However, the spleen-to-liver attenuation difference at follow-up CT scans was significantly associated with the severity of AP ($p = 0.007$).

Psoas Muscle Parameters

PMA at baseline was significantly associated with ICU stay ($p = 0.003$) and mortality ($p = 0.043$). Psoas muscle attenuation at baseline was associated with the severity of the disease ($p = 0.023$) and mortality ($p < 0.001$). Psoas muscle attenuation on follow-up was associated with the severity of AP ($p = 0.023$), ICU stay ($p = 0.001$), surgery ($p = 0.007$), and mortality ($p < 0.001$).

Table 1 Patient characteristics

Parameter	Frequency (%)
Male	143 (72.2)
Female	55 (27.7)
Mean age (± SD) in years	37.9 (± 12.4)
Etiology	
Alcohol	90 (45)
Gallstones	83 (42)
Idiopathic	13 (6.5)
Post ERCP	6 (3)
Drug induced	4 (2)
Hypercalcemia	2 (1)
RAC severity	
Moderately severe	83 (42)
Severe	115 (58)
Mean interval between two CT scans (± SD) in days	30 (± 13.5)
Mean CTSI (± SD)	8.8 (± 1.6)
Fatty liver at baseline	24 (12)
Fatty liver at follow-up	58 (29)
Mean spleen to liver attenuation difference at baseline (± SD)	10.2 (± 12.3)
Mean spleen to liver attenuation difference at follow-up (± SD)	18.8 (± 21.7)
Mean psoas muscle area at baseline	13.9 mm^2 (± 5.5)
Mean psoas muscle area at follow-up	11.2 mm^2(± 4.6)
Mean psoas muscle attenuation at baseline (± SD)	49.2 HU (± 8.2)
Mean psoas muscle attenuation at follow-up (± SD)	47.1 HU (± 12.1)
Mean length of hospital stay (± SD) in days	37.6 (± 22.9)
Number of patients admitted in ICU	100 (50)
Mean length of ICU stay (± SD) in days	7.9 (± 12.2)
Surgery	30 (15)
Mortality	56 (28)

Abbreviations: CT, computed tomography; CTSI, computed tomography severity index; ERCP, endoscopic retrograde cholangiopancreatography; HU, Hounsfield units; ICU, intensive care unit; RAC, revised Atlanta classification; SD, standard deviation.

Multivariate Analysis

Length of hospital stay: The only factor significantly associated with length of hospitalization was the PMA at baseline CT ($p = 0.036$).

ICU admission: Age ($p = 0.042$), PMA at baseline CT ($p = 0.001$), PMA at follow-up CT ($p = 0.045$), and psoas muscle attenuation at follow-up CT were significantly associated with ICU admission.

Length of ICU stay: The length of ICU stay was significantly associated with PMA at baseline CT ($p = 0.003$), psoas muscle attenuation at baseline ($p = 0.017$), and at follow-up CT ($p = 0.001$).

Surgery: Surgery was significantly associated with PMA at baseline ($p = 0.018$), PMA at follow-up ($p = 0.046$), and psoas muscle attenuation at follow-up CT ($p = 0.015$).

Mortality: PMA at baseline CT ($p = 0.019$), psoas muscle attenuation at baseline CT ($p = 0.032$), and RAC severity of AP ($p = 0.026$) were significantly associated with mortality.

Correlation (►Tables 5 and 6)

There was no significant correlation between spleen-to-liver attenuation difference at baseline or on follow-up CT scans with length of hospital and length of ICU stay. There was a significant positive correlation between PMA at baseline with length of ICU stay (correlation coefficient = 0.193, $p = 0.007$). There was a significant negative correlation between psoas muscle attenuation at baseline (correlation coefficient = 0.252, $p < 0.001$) and at follow-up CT scans with length of ICU stay (correlation coefficient = 0.319, $p < 0.001$).

Table 2 Association of fatty liver with outcomes

Outcomes	*p*-Value
Fatty liver at baseline	
RAC severity	0.919
Length of hospital stay	0.598
ICU stay	0.872
Length of ICU stay	0.671
Surgery	0.058
Mortality	0.792
Fatty liver at follow-up	
RAC severity	0.342
Length of hospital stay	0.542
ICU stay	0.392
Length of ICU stay	0.506
Surgery	0.385
Mortality	0.121

Abbreviations: ICU, intensive care unit; RAC, revised Atlanta classification.

Table 3 Association of spleen to liver attenuation difference with outcomes

Outcomes	*p*-Value
Spleen to liver attenuation difference at baseline	
RAC severity	0.072
ICU stay	0.796
Surgery	0.724
Mortality	0.893
Spleen to liver attenuation difference at follow-up	
RAC severity	0.007
ICU stay	0.351
Surgery	0.456
Mortality	0.144

Abbreviations: ICU, intensive care unit; RAC, revised Atlanta classification.

Table 4 Association of psoas muscle area with outcomes

Outcomes	*p*-Value
Psoas muscle area at baseline	
RAC severity	0.270
ICU stay	0.003
Surgery	0.119
Mortality	0.043
Psoas muscle area at follow-up	
RAC severity	0.588
ICU stay	0.536
Surgery	0.733
Mortality	0.807

Abbreviations: ICU, intensive care unit; RAC, revised Atlanta classification.

Table 5 Association of psoas muscle attenuation with outcomes

Outcomes	*p*-Value
Psoas muscle attenuation at baseline	
RAC severity	0.023
ICU stay	0.073
Surgery	0.967
Mortality	0.001
Psoas muscle attenuation at follow-up	
RAC severity	0.023
ICU stay	0.001
Surgery	0.007
Mortality	0.001

Abbreviations: ICU, intensive care unit; RAC, revised Atlanta classification.

ROC Curves (►Fig. 5)

Severity: The AUC was the largest (0.624 [95% confidence interval [CI]: 0.552–0.692]) for psoas muscle attenuation at follow-up CT scan followed by psoas muscle attenuation at baseline CT (0.609 [95% CI: 0.537–0.678]).

Table 6 Correlation of attenuation difference and psoas muscle indices with outcomes

Parameters	Length of hospital stay		Length of ICU stay	
	Pearson's coefficient	*p*-Value	Pearson's coefficient	*p*-Value
Liver AD at baseline CT	0.064	0.373	0.041	0.563
Liver AD at follow-up CT	−0.069	0.332	−0.119	0.096
PMA at baseline CT	0.129	0.070	0.193	0.007
PMA at follow up CT	0.011	0.872	0.042	0.557
PM attenuation at baseline CT	−0.084	0.239	−0.252	0.001
PM attenuation at follow-up	−0.139	0.050	−0.319	0.001

Abbreviations: AD, attenuation difference; CT, computed tomography; ICU, intensive care unit; PMA, psoas muscle area; PM, psoas muscle.

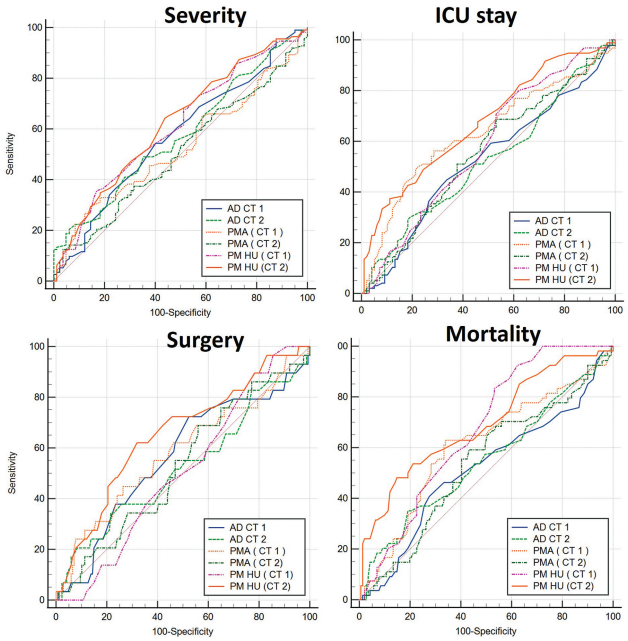

Fig. 5 Receiver operating characteristic curves for the severity of acute pancreatitis, intensive care unit (ICU) stay, surgery, and mortality.

ICU stay: The AUC was the largest (0.674 [95% CI: 0.603–0.739]) for psoas muscle attenuation at follow-up CT scan followed by PMA at baseline CT (0.632 [95% CI: 0.560–0.700]).

Surgery: The AUC was the largest (0.654 [95% CI: 0.583–0.721]) for psoas muscle attenuation at follow-up CT scan followed by PMA at baseline CT (0.582 [95% CI: 0.510–0.652]).

Mortality: The AUC was the largest (0.692 [95% CI: 0.622–0.756]) for psoas muscle attenuation at follow-up CT scan followed by psoas muscle attenuation at baseline CT (0.668 [95% CI: 0.597–0.734]).

Discussion

The present study investigated the effect of the presence of fatty liver and sarcopenia (PMA and psoas muscle attenuation) on the outcomes of the patients of AP. The analysis of these parameters is an evolving technique and can be easily obtained from abdominal CT images. CT is an integral imaging modality used in patients of AP for diagnosis and follow-up.

Our study suggested no association between the presence of fatty liver with the severity and outcomes of AP. This was contradictory to various studies done in the past, which recommend fatty liver to be an independent risk factor predicting the severity and mortality in AP.[11–13]

The mean PMA in our study was $13.9 \pm 5.5\ mm^2$ at baseline scan and $11.2 \pm 4.6\ mm^2$ on follow-up scans. It was lower as compared to a similar study done in the Chinese population in 2023 with a mean PMA of $15.28\ mm^2$.[14] The PMAs in the present study were lower as compared to a large sample of healthy patients from India who had no other comorbidities.[15] The mean difference in PMA between baseline and follow-up CT in our study was $2.64\ mm^2$ ($p < 0.001$), which suggested a decrease in PMA throughout the disease. It was similar to the findings of a study from New Zealand.[16]

In our study, the presence of ICU stay and mortality were significantly associated with PMA at baseline CT. It was contradictory to the abovementioned Chinese study that reported no association between ICU stay and PMA. Also, there was no association between PMA and the severity of the disease in the Chinese study.[14] There is no previous study that has evaluated the association of PMA with mortality in AP.

In our study, the mean psoas muscle attenuation was 49.2 ± 8.2 HU on the baseline scan and 47.1 ± 12.1 HU on the follow-up scan. It was significantly higher as compared to a recently conducted multicenter study in Europe, which recorded a mean muscle attenuation of 29.35 ± 4.16 HU in severe AP.[17] A Chinese study in 2021 on patients of AP calculated a mean muscle attenuation of 38.24 ± 3.92, which was lower than our study.[3] These findings were replicated again in another study in 2023 in the similar Chinese population, which showed a mean muscle attenuation of 36.07 ± 3.94 HU.[18] The higher psoas muscle attenuation in our study compared to these Chinese studies can be because of different patient characteristics. These studies included only severe AP patients while our study included patients of varying severities of AP (moderate as well as severe AP).

Our study suggested a significant association between psoas muscle attenuation at baseline CT with the severity of the disease as well as mortality. The muscle attenuation on follow-up CT was associated significantly with the severity of AP, presence of ICU stay, surgery, and mortality. A similar observation was reported by another European study, which suggested the association of low muscle attenuation with the severity of the disease.[17] The association between muscle attenuation with increased mortality was similar to a multicenter Dutch study in 2017 by van Grinsven et al who found a distinct association between muscle attenuation and mortality in AP patients ($p = 0.001$).[19] Zhou et al in 2021 reported a similar association between low muscle attenuation and mortality in AP patients.[3] The association between the need for ICU admission and increased mortality with low muscle attenuation was also noted in the United States-based study by Trikudanathan et al in 2021.[20]

Low muscle attenuation usually results from increased fatty infiltration of the muscle, which is known as myosteatosis or increased water infiltration representing muscle edema. Skeletal muscles are an important source of leptin, which not only prevents deposition of lipids in peripheral tissues but is also an anti-inflammatory agent. The reduced muscle attenuation impairs skeletal muscle function along with reduced leptin production. The disturbance in the inflammatory environment due to cytokines imbalance explains this association between low muscle attenuation and severity and mortality in AP.[21] The effect of leptin as an anti-inflammatory agent has been studied in animal models where exogenous supplementation of leptin reduced expression of proinflammatory factors and nitric oxide, which resulted in reduced severity in AP.[22]

Our study also reported a significant attenuation difference between baseline and follow-up CT.

The other school of thought suggests that severe inflammatory response syndrome (SIRS) in AP patients results in myosteatosis. The release of multiple proinflammatory

agents in AP provides high oxidative stress on the muscle mitochondria resulting in altered fatty acid metabolism resulting in fat deposition. This fatty deposition thus results in low muscle attenuation as the disease progresses.[23] It is also supported by a recent study in patients of colorectal cancer, which suggested an association between host SIRS and reduced muscle attenuation and myosteatosis.[24]

Our study has a few limitations which need to be acknowledged. First, this was a retrospective study with no set timing for the acquisition of CT. Second, the scans were evaluated by two radiologists in consensus and the intra- and interobserver agreement were not assessed. Third, anthropometric parameters were not available for patients. So, we could not calculate the psoas muscle index. Finally, the impact of edema on psoas muscle parameters in our results as well as the previous studies is still unknown.

In conclusion, our study did not find the association of fatty liver with outcomes of AP. However, PMA indices are associated with clinical outcomes. Larger prospective well-planned studies may confirm as well as supplement the findings reported by us.

Ethics Approval

Institute Ethics committee of PGIMER, Chandigarh, India approved this study.

Data Availability Statement

All data associated with the manuscript has been presented in the paper.

Authors' Contributions

A.J., N.R., P.G.: Collected data, performed statistical analysis, wrote the initial draft, and revised the manuscript. V.J., A.S., J.S., J.S., H.M., V.S., S.K.S., U.D.: Collected data and revised the manuscript.

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None.

Conflict of Interest

None declared.

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