Quantitative T2 Mapping of Acute Pancreatitis

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Background: Quantification of the T2 signal by means of T2 mapping in acute pancreatitis (AP) has the potential to quantify the parenchymal edema. Quantitative T2 mapping may overcome the limitations of previously reported scoring systems for reliable assessment of AP.
Purpose: To evaluate MR-derived pancreatic T2 mapping values in AP and correlate them with markers of disease severity.
Study Type: Prospective single-center study.
Population: 76 adults with AP (20–91 years, females/males: 39/37).
Field Strength/Sequence: Fat suppressed multiecho spin-echo prototype sequence to quantify T2 signal at 3T MRI.
Assessment: The severity of AP was assessed clinically, biologically, and by contrast-enhanced CT (CECT) performed 48–72 hours after symptom onset. MRI was then performed ≤24 hours after CT. Two readers blinded to any clinical information independently evaluated the T2 values by placing three regions of interest inside the pancreatic head, body, and tail on the T2 mapping MR sequence. Results were compared with corresponding CECT images as the standard and clinical severity parameters, using the length of hospital stay as our primary endpoint.
Statistical Tests: Continuous variables were compared using the Spearman's rank correlation coefficient, analysis of variance (ANOVA) or Student's *t*-test.
Results: T2 values significantly correlated with the length of hospital stay ($r_s(74) = 0.29$), CT severity index (CTSI) ($r_s(73) = 0.61$; CTSI 0–3: 72 ± 14 msec, CTSI 4–10: 88 ± 15), intensive care unit (ICU) admission ($t(2.77) = -3.41$) and presence of organ failure ($t(6.72) = -3.42$), whereas the CTSI and Ranson score were not significantly related with ICU admission (CTSI: $P = 0.24$; Ranson score: $P = 0.24$) and organ failure (CTSI: $P = 0.11$; Ranson score $P = 0.11$).
Conclusion: T2 mapping correlates with AP severity parameters and is useful for assessing the severity of AP with higher sensitivity than the usual clinical and radiological scoring systems.
Level of Evidence: 1
Technical Efficacy: Stage 2

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Over the past few decades, various scoring systems have been suggested to predict the severity of acute pancreatitis (AP), but no accurate scoring system has been introduced.[1] Although AP often has a mild clinical course, 30% of patients develop severe disease[2] and are subject to a long hospital stay with a high risk of complications, such as infected necrosis or organ failure, with mortality rates up to 30%.[3] Mild AP responds to conservative management, whereas severe AP requires a more aggressive, sometimes surgical approach.[4] Consequently, distinguishing between the two forms is of importance.

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Several clinical and laboratory scoring systems, such as the Ranson criteria, have been designed to accurately correlate complications (eg organ failure and mortality) in AP.[5] The Ranson score is based on 11 prognostic signs (age, blood test values, arterial blood gases, and fluid sequestration) measured on admission, and 48 hours later.[6] CT is the standard imaging modality for confirming clinically suspected AP, staging the disease severity, and assessing complications. The CT severity index (CTSI)[7] is the most widely adopted radiological scoring system in clinical and research settings. The CTSI assesses pancreatic inflammation and the extent of pancreatic necrosis separately.

MRI is an alternative imaging modality and may more accurately assess AP because of the inherently higher contrast-resolution compared with CT.[8] Moreover, MRI is a non-ionizing cross-sectional imaging method, and intravenous gadolinium has a safer profile than the iodinated contrast medium injected for CT.[8,9]

MRI offers the opportunity to go beyond qualitative visual assessment with quantitative imaging sequences, such as T2 mapping. The latter can quantify signal changes reflective of underlying tissue properties and quantify the parenchymal edema that typically occurs in AP. Preliminary studies have shown that the typically increased pancreatic/peripancreatic signal detected on T2-weighted MR sequences has the potential to allow diagnosis and classification of AP.[10–13] In particular, Vietti Violi et al[14] showed that the presence of pancreatic disease is associated with increased T2 relaxation times compared with a healthy pancreas. This quantification offers several advantages, including increased reproducibility and sensitivity than what is obtained by visual assessment of T2-weighted MR images, which is considered especially useful for identifying minor parenchymal changes and, thus, an objective means of staging.[12,14] We aimed to understand if pancreatic T2 values correlate with clinical, biological, and radiological severity parameters, in patients with AP.

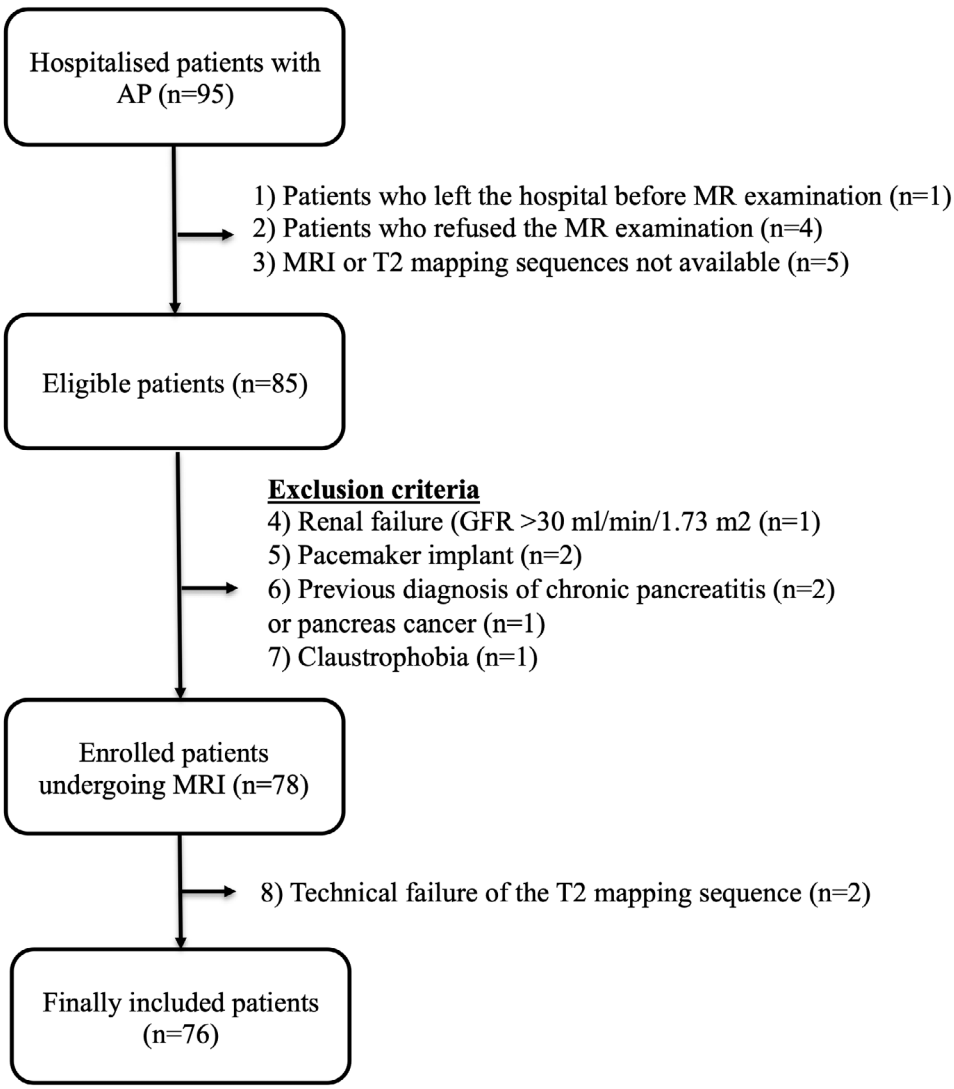

FIGURE 1: Flow chart showing the process of patient inclusion.

Materials and Methods

Patients

This was a prospective, single-center study that followed the Standards for Reporting Diagnostic Accuracy guidelines[15] and was registered in the national portal for human research (SNCTP: 2020–02153). The study protocol (no 2020–02153) was approved by our institutional ethics committee and patients provided written informed consent.

Patients presenting to the Emergency Department for suspected AP between December 2020 and November 2022 were consecutively identified (Fig. 1). AP was defined as the presence of two or more of the following characteristics: abdominal pain, serum amylase or lipase levels ≥3-times the upper limit of normal (>210 U/L and >180 U/L, respectively), and/or characteristic findings of AP on contrast-enhanced CT (CECT).[16] The diagnosis of AP was made according to a standard panel of at least three physicians (emergency physicians, radiologists, and visceral surgeons).[17] The final inclusion criterion was that patients underwent CECT 48–72 hours after symptom onset.

Exclusion criteria were being intubated and ventilated, renal failure with estimated glomerular filtration rate <30 mL/minutes/1.73 m^2, history of allergic reactions to contrast media, pregnancy, age <18 years, general exclusion criteria for MRI, chronic pancreatitis or pancreas cancer, claustrophobia, and inability to cognitively and/or linguistically understand the patient consent sheet.

Patient characteristics were also recorded, including age, sex, and body mass index (BMI).

Parameters of AP Severity

All patients underwent a clinical assessment and laboratory workup at admission and 48 hours later (Table 1). The clinical outcome of AP was reported in regard to complications (intensive care unit (ICU) admission and length of stay, organ failure, minimally invasive interventions, and death). The length of hospital stay was defined as our primary endpoint.

According to our clinical routine, each patient underwent an abdominal CECT examination within 48–72 hours after symptom onset to assess the severity of the AP. The pancreatic CECT protocol included a pancreatic phase at 40 sec and a portal venous phase at 70–75 sec. The severity of the AP was rated as the CTSI determined by the consensus of two readers with 2 and 30 years of clinical experience, respectively. The CTSI was calculated based on a combination of pancreatic inflammation, and degree of pancreatic necrosis as observed on CT, according to the scoring system developed by Balthazar et al.[7]

Exact anatomical location of AP and grade of inflammation was assessed by the same two readers using a specially developed score, the CT localization of inflammation score (CT LocIn Score). We defined the CT LocIn Score (Fig. 2) as the extent of edematous pancreatic enlargement and peri-pancreatic fat stranding evaluated separately for each part of the pancreas (head, body, and tail) as detected on the CT images: 0 = no enlargement nor surrounding fat stranding; 1 = enlargement or surrounding fat stranding; 2 = enlargement and surrounding fat stranding.

MR Data Acquisition and Reconstruction

Patients underwent a single MR examination (MAGNETOM Vida, Siemens Healthcare, Erlangen, Germany) of the pancreas within 48–72 hours after the onset of clinical symptoms and ≤24 hours after the CT examination during which the T2-mapping sequence was acquired. All patients were examined in a supine position using an 18-channel body array coil and a 32-channel spine coil. MR parameters included axial (3 mm) and coronal (3 mm) half-Fourier acquisition single-shot turbo spin echo (HASTE) sequences, an axial (6 mm) diffusion-weighted sequence (DWI), axial T1-weighted gradient-echo sequences (VIBE) (3 mm) before and after the intravenous injection of 0.2 cc/kg gadoteric acid, (Dotarem®, Guerbet, Villepinte, France), and a heavily T2-weighted sequence in the coronal oblique plane (50 mm, MR-Wirsungography) with relaxation enhancement (RARE) centered on the head and tail of the pancreas.

TABLE 1. Severity Parameters of AP

Clinical Parameters	Laboratory Parameters	Radiological Parameters
Hospital length of stay	Lipase (13–60) U/L at admission	Computed tomography severity index
Intensive care unit admission	Amylase (13–53) U/L at admission	Radiological complications[a]
Organ failure[b]	White blood cell count (4.0–10.0) G/L at 48 hours	CT localization of inflammation score
Ranson criteria	C-reactive protein (<10) mg/L at 48 hours	
Minimally invasive interventions[c]		
Death		

[a]Radiological complications are complications detected on CT or MRI: gastrointestinal perforation, gastrointestinal bleeding, pseudoaneurysm, and venous thrombosis.
[b]Organ failure: respiratory, cardiovascular, and renal.
[c]Minimally invasive interventions: percutaneous abdominal puncture, percutaneous abdominal and endoscopic drainage, endoscopy, embolization, and venous/biliary stent placement.

Before the intravenous gadolinium injection, a T2 mapping sequence with fat suppression was acquired in the axial plane as follows. A multishot, multiecho spin-echo[18] prototype sequence with radial sampling and a pseudo Golden Angle view ordering[19] was used to acquire k-space using prospective acquisition correction[20] with external triggering (30 slices, 24 echoes with 13 radial spokes each, 512 samples per spoke, 1.2 × 1.2 × 6 mm resolution, number of echoes: 24, echo-spacing: 8.58 msec, acquisition time: 222 sec). According to the individual trigger efficiency of a patient, we chose either a phase scout or a 1D navigator at the liver dome to trigger the acquisition on end-expiration, allowing the patient's free breathing. Notably, this results in varying repetition times (mean: 4678 msec, standard deviation (SD): 1659 msec) depending on the patients' respiratory frequency. For T2 map reconstruction directly on the scanner hardware, an image for each echo in the echo-train was generated using a tiered echo-sharing reconstruction.[21] The low frequencies of k-space were filled with radial spokes from the corresponding echo to retain contrast; whereas, the high frequencies were sampled from all echoes in the echo train (full-tier) to gain sharpness. Next, a monoexponential signal model while ignoring the first echo was fitted on the image data to produce T2 maps.[21] Notably, quantitative mapping in the abdomen is especially challenging due to respiratory and bowl movement. While a systemic analysis of the performance of the T2 mapping method used here is out of the scope of this study, a comprehensive comparison, including this method, was performed by Draveny et al.[22]

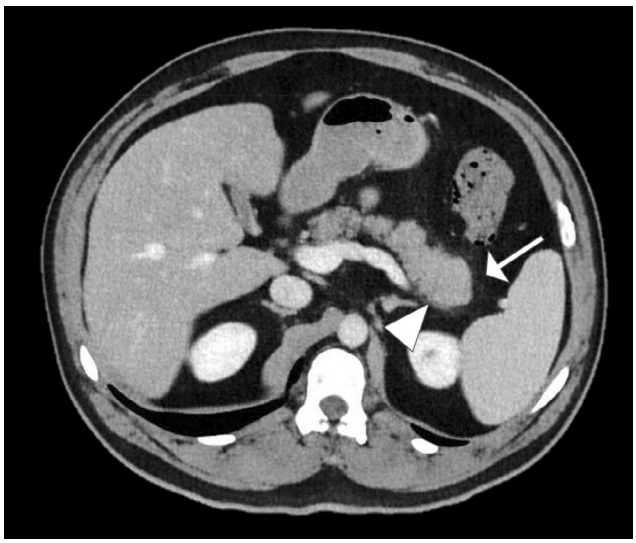

FIGURE 2: Example of assessing the CT localization of inflammation score (CT LocIn Score) in a patient with AP (CTSI 2). This axial contrast-enhanced abdominal CT image shows focal inflammation of the tail of the pancreas with focal parenchyma enlargement (arrow) and subtle peri-pancreatic fat stranding (arrowhead, CT LocIn Score 2). The adjacent corporeal and cephalic parenchyma are normal with preserved pancreatic lobules (CT LocIn Score 0 for both regions). In this patient, T2 values were measured at 74 msec in the tail, clearly higher than those measured in the body (T2 = 47.62 msec), confirming caudal AP.

Image Segmentation and T2 Value Acquisition

Two readers with 5 and 15 years of experience with abdominal MRI, respectively, and blinded to any clinical results separately analyzed the T2-mapping sequences of all patients on a picture archiving and communication system workstation (PACS; Carestream Vue, v. 11.4; Carestream Health, Rochester, NY). Each reader determined the mean T2 values and SD by manually drawing one region of interest (ROI) in each part of the pancreatic parenchyma (head, body, and tail; $n = 3$ ROIs; Fig. 3). The readers were instructed to

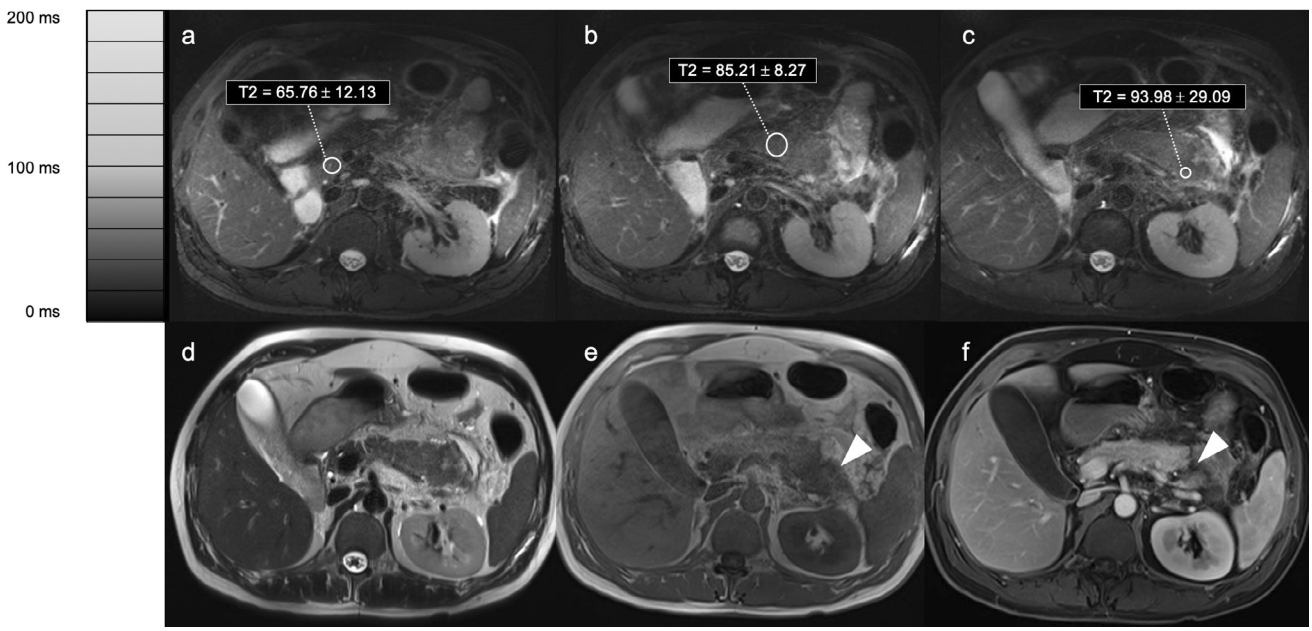

FIGURE 3: Axial images of the reconstructed T2 map (a–c) and corresponding T2-weighted image (d) and precontrast (e) and postcontrast T1-weighted fat-saturated images (f) in a patient with AP CTSI 6. Each of the two readers determined the mean pancreatic T2 values and standard variation by manually drawing regions of interest within the head (a), body (b), and tail (c) of the pancreatic parenchyma avoiding pancreatic necrosis (arrow head) by referring to other sequences (d–f).

draw the largest possible ROI in each area while avoiding the pancreatic duct, vessels, focal lesions, pancreatic necrosis, and zones showing partial volume effects referring if necessary to postcontrast sequences. The readers also reported any pancreatic duct dilatation, which was defined by a maximal diameter >4 mm. For the latter, the two readers discussed any discordance until reaching consensus.

Statistical Analysis

Statistical analyses were performed using software R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). The T2 values separately and independently determined by each reader were compared using the interclass correlation coefficient (ICC) to assess inter-reader reproducibility. Reliability was classified as poor (ICC <0.5), moderate (ICC between 0.5 and 0.75), good (ICC between 0.75 and 0.9), or excellent (ICC >0.90).[23]

The data from the two readers were averaged for the subsequent analyses. The mean T2 values for the three pancreatic anatomical regions (head, body, and tail) in the same patient were compared, as well as across all study patients. The T2 values in each patient as measured separately for each of the three pancreatic anatomical regions were correlated with the CT LocIn Score, which had been specially developed to localize the inflammation on CT images.

In each patient, the T2 values were measured separately for the three pancreatic anatomical regions and averaged for the subsequent analyses. The mean T2 values averaged from the two readers and the three pancreatic anatomical regions were correlated with age, sex, BMI, and AP severity using the length of hospital stay as the main outcome, as well as laboratory values, clinical and radiological outcome as exploratory outcomes. The CTSI and Ranson score were also correlated with length of hospital stay, lipase at admission, and clinical and radiological outcomes.

Continuous variables were compared using the Student's *t*-test, analysis of variance (ANOVA) or Spearman's rank correlation coefficient. We used the following general structure to report Spearman's correlation in American Psychological Association format: r_s (df) = [*r* value], *P* = [*P*-value]. The degrees of freedom (df) was calculated as n-2. The performance of T2 mapping for prediction of hospital stay >5 days was assessed using sensitivity, specificity, and area under the receiver operating characteristic (ROC) curve (AUC). A Mann–Whitney-Wilcoxon test was then used to determine whether the AUC was significantly different from 0.5. The "5 days" threshold was chosen because it corresponds to the average length of stay for pancreatic diseases according to the Swiss tariff system for remuneration of acute diseases.[24] Differences were considered significant at $P < 0.05$.

Results

Patients

A total of 95 consecutive patients diagnosed with AP were admitted to the visceral surgery department between December 2020 and November 2022. The process of patient inclusion is shown in Fig. 1. Our final study population consisted of 76 patients (39 women; mean age 53.17 ± 17.89 years, range: 20–91 years). The baseline characteristics of our patients are provided in Table 2.

TABLE 2. General Characteristics of the Study Population

	Patients *n* = 76
Female	39 (51%)
Mean age (years)	53.17 (SD: 17.89)
Etiology of AP	
Biliary stone	34 (45%)
Alcohol	12 (16%)
Post-ERCP	11 (15%)
Drugs	3 (4%)
Hypertriglyceridemia	1 (1%)
Iatrogenic	1 (1%)
Unknown	14 (18%)
AP severity parameters	
Mean hospital length of stay (days)	7 (SD: 11)
Intensive care unit admission	3 (4%)
Mean intensive care unit length of stay (days)	34 (SD: 54)
Organ failure[a]	9 (12%)
Minimally invasive interventions[b]	
No interventions	67 (88%)
One intervention	6 (8%)
Two or more interventions	3 (4%)
Death	1 (1%)
Mean Ranson criteria	2 (1%)
Mean computed tomography severity index	2 (SD: 2)
CTSI 0–3: mild AP	57 (75%)
CTSI 4–6: moderate AP	18 (24%)
CTSI 7–10: severe AP	1 (1%)
Radiological complications[c]	
No complications	72 (95%)
One complication	3 (4%)
Two or more complications	1 (1%)

AP = acute pancreatitis; CTSI = computed tomography severity index; Post-ERCP = postendoscopic retrograde cholangiopancreatography; SD = standard deviation.
[a]Organ failure: respiratory, cardiovascular, and renal.
[b]Minimally invasive interventions: percutaneous abdominal puncture, percutaneous abdominal and endoscopic drainages, endoscopy, embolization, and venous/biliary stent placement.
[c]Radiological complications are complications detected on CT or MRI: gastrointestinal perforation, gastrointestinal bleeding, and pseudo-aneurysm.

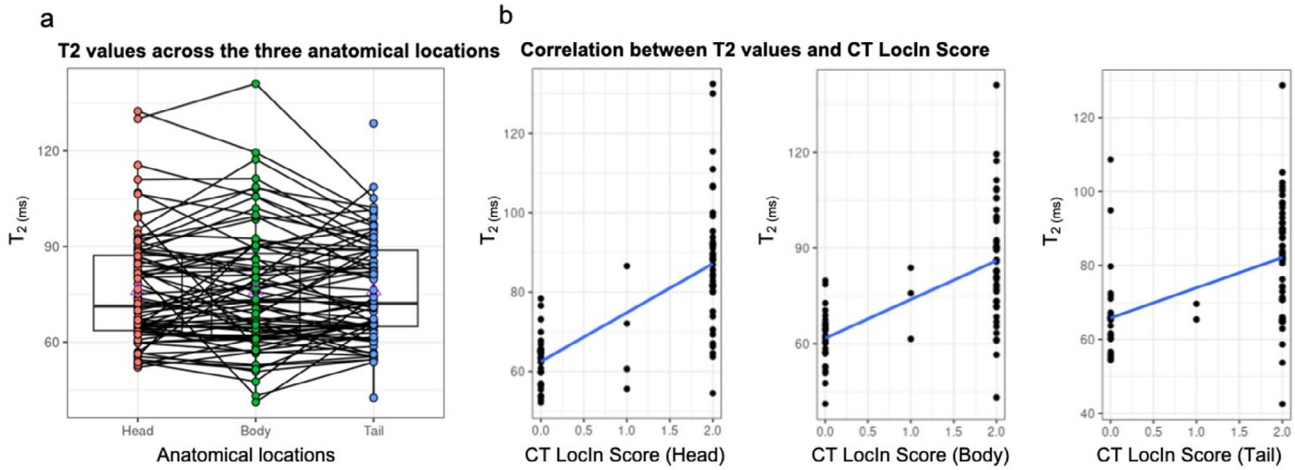

FIGURE 4: Boxplots of mean T2 values across the three anatomical locations (a) and scatterplots of the correlation between mean T2 values and CT LocIn Score across the three anatomical locations (b).

Inter-Readers Reproducibility

The ICC of the obtained MRI T2 values was good between the two readers for the overall analysis (0.85, 95% confidence interval (CI) = 0.77–0.90), as well as for each anatomical location (0.80, 95% CI = 0.75–0.84). Student's *t*-test showed no significant differences between the mean measurements of the readers ($P = 0.09$, mean T2 value of Reader 1 = 75 ± 18 msec, range: 50–123 msec, mean T2 value of Reader 2 = 78 ± 19 msec, range: 48–128 msec).

T2 Values Across the Three Anatomical Locations and CT LocIn Score

The ANOVA revealed no significant difference in means across the three anatomical locations ($P > 0.99$). The mean T2 values were 76 ± 18 msec (range: 52–133 msec), 76 ± 19 (range: 41–141 msec), and 76 ± 16 msec (range: 43–129 msec) in the head, body, and tail of the pancreas, respectively.

However, T2 values in the same patient for the three different anatomical regions of the pancreas correlated significantly and positively with the presence of inflammation using the specially developed CT LocIn Score as shown in Fig. 4 (head: $r_s(74) = 0.74$; body: $r_s(74) = 0.66$; tail: $r_s(74) = 0.52$; all three anatomical regions together: $r_s(226) = 0.64$).

T2 Values and Correlation with Patient Characteristics and AP Severity

No significant correlation was found between the T2 values and patient age ($r_s(74) = 0.18$; $P = 0.13$), sex (male: 77 ± 18 msec, female: 75 ± 14 msec; $t(65) = -0.44$; $P = 0.66$), BMI ($r_s(74) = 0.06$; $P = 0.61$) or main pancreatic duct dilatation (presence of dilatation: 86 ± 21 msec, absence of dilatation: 75 ± 15 msec; $t(9) = -1.54$; $P = 0.16$).

TABLE 3. Correlation Between T2 Values and AP Severity

Spearman's Rank Correlation Coefficient	r_s	*P*
Length of hospital stay (days)	0.29	**0.01***
Length of hospital stay (days) without the outlier[a]	0.26	**0.02***
CTSI	0.61	**<0.01***
Ranson criteria	0.01	0.94
Lipase at admission (U/L)	0.25	**0.03***
Amylase at admission (U/L)	0.25	**<0.05***
Leukocytes at 48 hours (G/L)	0.46	**<0.001***
CRP at 48 hours (mg/L)	0.55	**<0.001***
Student's *t*-Test	***t***	***P***
Hospital stay <5 and ≥5 days	3.37	**0.001***
CTSI (0–3 vs 4–10)	3.87	**<0.001***
ICU admission	−3.41	**<0.05***
Organ failure[b]	−3.42	**0.01***
Radiological complications[c]	−0.31	0.78
Minimally invasive interventions[d]	−2.26	0.05

CRP = C-reactive protein; CTSI = computed tomography severity index; ICU = intensive care unit.
*Significant values are in bold.
[a]Defined as the only patient with severe AP followed by a fatal outcome.
[b]Organ failure: respiratory, cardiovascular, and renal.
[c]Radiological complications are complications detected on CT or MRI: gastrointestinal perforation, gastrointestinal bleeding, pseudo-aneurysm.
[d]Minimally invasive interventions: percutaneous abdominal puncture, percutaneous abdominal and endoscopic drainages, endoscopy, embolization, venous/biliary stent placement.

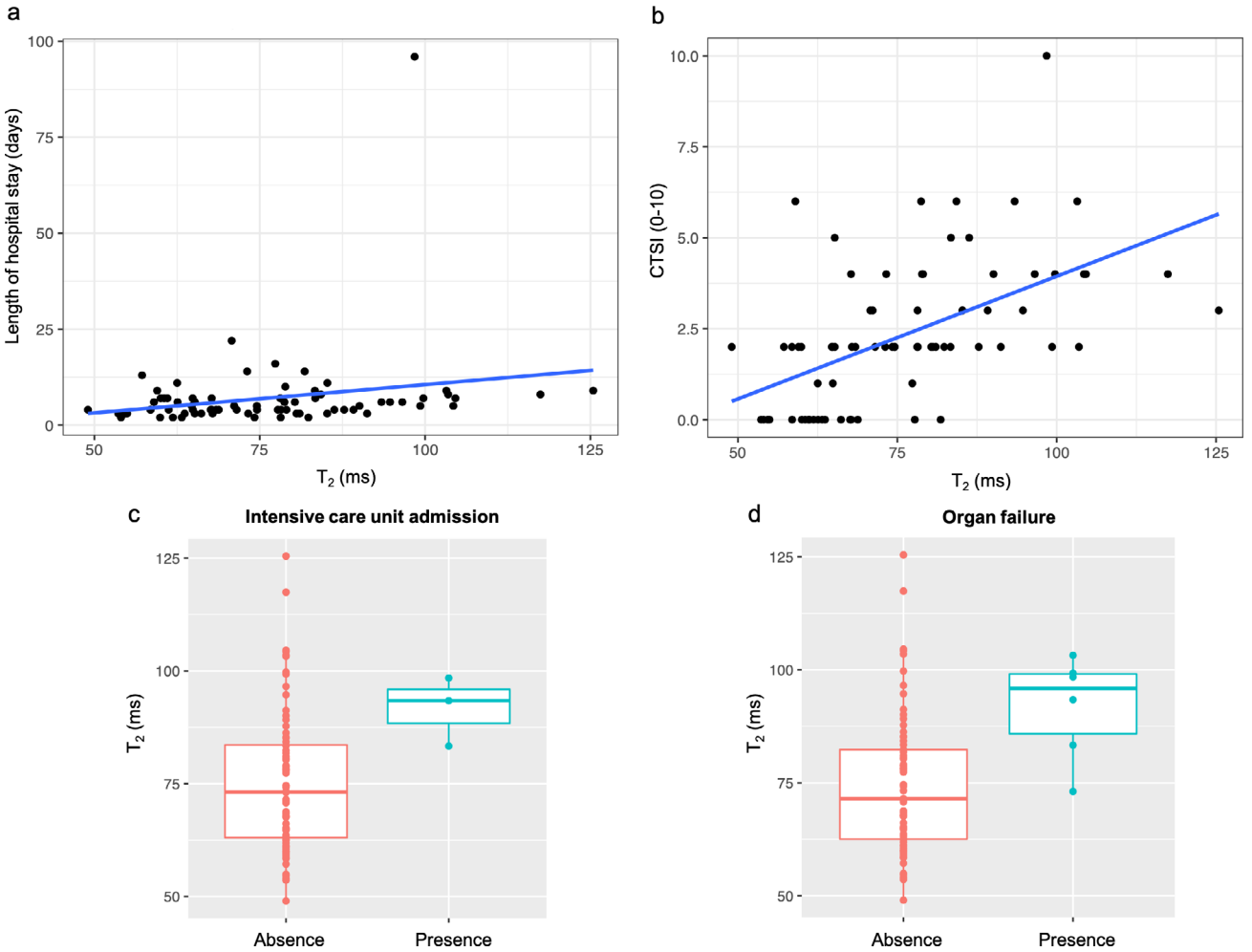

FIGURE 5: Histograms and charts of the significant correlations between mean T2 values and parameters of AP severity, such as the length of hospital stay (a), CTSI (b), admission to the intensive care unit (c), and organ failure (d).

Table 3 and Fig. 5 summarize significant correlations between T2 values and parameters of AP severity. Spearman correlation revealed a significant positive association between T2 values and the length of hospital stay ($r_s(74) = 0.29$), even after withdrawal of the only outlier ($r_s(73) = 0.26$), which was a patient with severe pancreatitis and fatal outcome. There was also a significant difference in T2 values between patients hospitalized <5 and ≥5 days (length of hospital stay <5 days: 70 ± 12 msec, length of hospital stay ≥5 days: 81 ± 16 msec; $t(65) = 3.37$). The ROC curves illustrated in Fig. 6 show the predictive power of the T2 values for a hospital stay <5 days compared with CTSI and the Ranson score. The AUC was 0.68 (CI: 0.56–0.80) for the T2 values, 0.71 (CI: 0.60–0.82) for the CTSI and 0.69 (CI: 0.58–0.81) for the Ranson score with no statistically significant difference between the three AUCs. A Mann-Whitney-Wilcoxon test revealed that the AUCs for the three parameters were significantly different from 0.5.

For the exploratory outcomes, T2 values correlated significantly with the CTSI ($r_s(73) = 0.61$; CTSI 0–3: 72 ± 14 msec, CTSI 4–10: 88 ± 15; $t(29.05) = -3.87$), ICU admission (ICU admission: 92 ± 8 msec, no ICU admission: 75 ± 16 msec; $t(2.77) = -3.41$), and presence of organ failure (presence of organ failure: 92 ± 11 msec, absence of organ failure: 75 ± 16 msec; $t(6.72) = -3.42$). We found a statistically significant relationship between T2 values and laboratory values (lipase and amylase at admission and leukocytes and CRP at 48 hours). Corresponding Spearman correlation coefficients (r_s) and P values are provided in Table 3. Comparisons between T2 mapping, CTSI, and the Ranson score are shown in Table 4.

Discussion

These results show that pancreatic T2 values significantly correlate with the severity of AP and with the length of the patients' hospital stay. Thus, T2 mapping has the potential to distinguish mild (CTSI 0–3) from moderate to severe (CTSI 4–10) AP and help predict hospital stays longer than 5 days. Pancreatic T2 values did not depend on anatomical location, but we found a significant correlation between the T2 values and localization of inflammation in the three anatomical

regions of the pancreas. Furthermore, the measured T2 values did not depend on age, BMI, or sex and showed good inter-reader reproducibility.

Three studies have reported the T2 relaxation time of the pancreas in healthy volunteers, with a mean ranging from 43 to 63 msec,[10,11,13] lower than what was measured in our study. Comparing these T2 values with those obtained in our study is difficult because we only included patients with AP. More recently, Vietti Violi et al and Wang et al showed that the presence of pancreatic disease is associated with increased T2 values. They reported T2 values of 71 ± 9 msec for patients with diffuse pancreatic disease and 58 ± 11 msec for patients with chronic pancreatitis, which are lower than the values for AP patients in our study, possibly due to the more fibrous pancreatic parenchyma that we encountered in our patients.[14,25] Furthermore, one should consider systematic biases between studies due to the use of different T2 quantification methods.[22] Several factors can affect the T2 relaxation time estimate, including choice of pulse sequence, preparation pulses, field strength, and even the number of acquired k-space lines.[26]

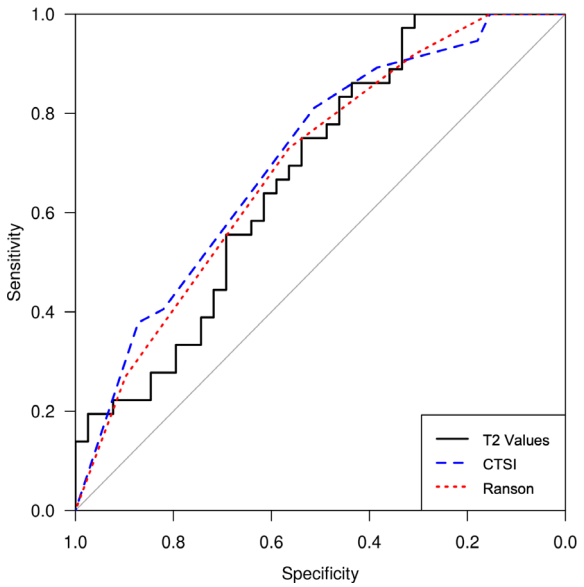

FIGURE 6: ROC curves of the performance of T2 mapping, CTSI and Ranson score for prediction of hospital stay <5 days. The AUC of this ROC curves was 0.68 (CI: 0.56–0.80, $P < 0.01$) for the T2 values, 0.71 (CI: 0.60–0.82, $P < 0.01$) for the CTSI and 0.69 (CI: 0.58–0.81, $P < 0.01$) for the Ranson score with no statically significant difference between the three AUCs.

We did not find any relationship between T2 values and anatomical location, between T2 values and patient characteristics data (sex, age, and BMI), or between T2 values and main pancreatic duct dilatation. Vietti Violi et al reported higher T2 values in the tail and lower values in the head, with a significant correlation between T2 values and age and between T2 values and pancreatic duct dilatation, which we primarily attribute to the T2 hyperintensity of fat; this may have been unequally distributed in the pancreatic parenchyma of their patients.[14] Our T2 values were devoid of fat hyperintensity, because we used fat suppression, and they were mainly determined by the acute parenchymal edematous inflammation, as indicated by the correlation between T2 values and the localization of the inflammation determined by the CT LocIn Score.

In agreement with our results, Upadhyay et al and Vietti Violi et al demonstrated good to high reproducibility within and across different readers.[13,14]

Similar to our T2 values, the two traditional scoring systems, the CTSI and Ranson score, correlated significantly with length of hospital stay and lipase at admission. However, unlike the CTSI and Ranson score, T2 values correlated

TABLE 4. Comparison of Scoring Systems for AP Severity

	T2 Mapping	CTSI	Ranson Score
Length of hospital stay (days)	**$P = 0.01$***	**$P = 0.01$***	**$P < 0.01$***
Lipase at admission (U/L)	**$P = 0.03$***	**$P = 0.03$***	**$P < 0.05$***
ICU admission	**$P < 0.05$***	$P = 0.24$	$P = 0.24$
Organ failure[a]	**$P = 0.01$***	$P = 0.11$	$P = 0.11$
Radiological complications[b]	$P = 0.78$	$P = 0.14$	$P = 0.46$
Minimally invasive interventions[c]	$P = 0.05$	$P = 0.15$	$P = 0.18$

CTSI = computed tomography severity index; ICU = intensive care unit.
*Significant values are in bold.
[a]Organ failure: respiratory, cardiovascular and renal.
[b]Radiological complications are complications detected on CT or MRI: gastrointestinal-perforation, gastrointestinal-bleeding, pseudo-aneurysm.
[c]Minimally invasive interventions: percutaneous abdominal punctures, percutaneous abdominal and endoscopic drainages, endoscopy, embolization, and venous/biliary stent placement.

significantly with ICU admission as well as presence of organ failure and showed a statistical trend for minimally invasive interventions. Thus, T2 mapping has the potential to select patients at risk of complications with more sensitivity than the usual clinical and radiological scoring systems. Furthermore, T2 mapping is less invasive because it does not require arterial puncture (Ranson score) or radiation exposure (CTSI). In addition, T2 mapping does not require the intravenous injection of any contrast medium.

Limitations

First, we had to exclude patients who were directly admitted to the ICU because of the clinical severity of their AP and, thus, were not able to undergo the MRI. This explains the low number of patients with severe AP compared with the total number of patients with AP. Moreover, patients with severe AP generally have large areas of pancreatic necrosis which limits the interpretation of our measured T2 values. The second limitation is the use of CECT as the standard instead of any pathological reference standard. Finally, there was a delay between the CT and MR examinations. Although we kept this delay as short as possible (≤24 hours by definition), the disease may have progressed in that time frame.

Conclusion

We showed the potential of T2 mapping for assessing the severity of AP. T2 mapping can differentiate mild from moderate AP and help predict the length of hospital stay. As a less invasive alternative to the traditional scoring systems, we recommend the assessment of the AP severity using T2 mapping in clinical routine. Further studies should investigate the use of T2 mapping in patients with severe AP, ideally in multicenter studies.

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