

# Acute pancreatitis with gradient echo T2\*-weighted magnetic resonance imaging

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**Background:** To study gradient recalled echo (GRE) T2\*-weighted imaging (T2\*WI) for normal pancreas and acute pancreatitis (AP).

**Methods:** Fifty-one patients without any pancreatic disorders (control group) and 117 patients with AP were recruited. T2\* values derived from T2\*WI of the pancreas were measured for the two groups. The severity of AP was graded by the magnetic resonance severity index (MRSI) and the Acute Physiology and Chronic Healthy Evaluation II (APACHE II) scoring system. Logistic regression was used to analyze the relationship between the T2\* values and AP severity. The usefulness of the T2\* value for diagnosing AP and the relationship between the T2\* values and the severity of AP were analyzed.

**Results:** On GRE-T2\*WI, the normal pancreas showed a well-margined and consistently homogeneous isointensity. Edematous AP, as well as the non-necrotic area in necrotizing AP, showed ill-defined but homogeneous signal intensity. AP with pancreatic hemorrhage showed a decreased T2\* value and a signal loss on the signal decay curve. The T2\* value of pancreas in the AP group was higher than that of the control group ( $t=-8.20$ ,  $P<0.05$ ). The T2\* value tended to increase along with the increase in MRSI scores but not with the APACHE II scores ( $P>0.05$ ). AP was associated with a one standard deviation increment in the T2\* value (OR =1.37; 95% CI: 1.216–1.532).

**Conclusions:** T2\*WI demonstrates a few characteristics of the normal pancreas and AP, which could potentially be helpful for detecting hemorrhage, and contributes to diagnosing AP and its severity.

**Keywords:** Magnetic resonance imaging (MRI); pancreatitis; T2\*; T2 star; gradient echo; hemorrhage

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## Introduction

Acute pancreatitis (AP) is an acute inflammatory disease in which the inflammatory process may be limited to the pancreas or may spread to surrounding tissues or even the remote organs, resulting in multi-organ failure and occasionally death (1). Although it often has a mild and self-limiting course, it may be severe, resulting in local and systemic complications carrying a significant risk of death (2,3). When local protective responses fail, AP leases local inflammatory mediators into the systemic circulation,

resulting in systemic inflammatory response syndrome (SIRS) (4). SIRS may cause multiple organ dysfunction and eventually multi-organ failure, which is associated with very high mortality (5). Early accurate diagnosis and assessment of the severity of AP is crucial to determine an early treatment plan and to improve the prognosis of patients with AP.

Currently, the diagnosis and treatment of AP remain to be standardized. The further use of advanced diagnostic tools will potentially help clinicians manage AP at an appropriate

stage (6). In patients with AP, imaging is recommended to confirm the clinical diagnosis, investigate the etiology, and grade the severity of the disease (7). Magnetic resonance imaging (MRI) has an excellent resolution of soft tissue, and it has earned an important role in the diagnosis of AP (7). With the continuous development of MR scanning techniques, multi-echo gradient recalled echo (GRE) T2\*-weighted imaging (T2\*WI) is a relatively new MRI technique. The sequence of a GRE T2\*WI requires high uniformity of the magnetic field and can provide a T2\* value, which can indirectly reflect changes in tissue biochemical components and thus be used for the early quantitative diagnosis of some diseases (8,9).

There have been some studies on the GRE T2\*WI of pancreas showing T2\* was related to iron deposition (10,11). However, until now, there has been no research on the sequence applied in patients with AP. We conducted this study to observe the appearance of the normal pancreas and AP on T2\*WI, to measure the T2\* value of the pancreas, and to discuss the relationship among T2\* values for diagnosing AP and its severity according to the magnetic resonance severity index (MRSI) and the Acute Physiology And Chronic Healthy Evaluation II (APACHE II) scoring system.

## Materials and methods

All authors have access to the study data and have reviewed and approved the final manuscript.

### Ethics statement

This study has been registered in clinical trials (NCT01911689), and at the beginning of this study, it was also approved by the Institutional Review Board of the Affiliated Hospital of North Sichuan Medical College. Informed consent from all participants involved in our study were obtained, this consent was verbal. For all participants, we only add a multi-echo GRE T2\*WI scan after the conventional scan. MRI is radiation-free and the T2\*WI has short scan time, which often finish one scan in a minute. So The Ethics Committee concluded the performance of this study would not cause harm or risk to the subjects, and the ethics committees approved and agreed that verbal consent was sufficient for our study.

### Patient selection

AP patients admitted to our institution between August

2013 and March 2015 were considered for inclusion in this study. The diagnosis criteria of AP require two of the following three features (12): (I) acute typical abdominal pain; (II) threefold elevation of amylase or lipase serum levels, excluding other causes of elevated enzymes; and (III) abdominal imaging examination. The inclusion criteria for patients in this study were as follows: (I) in-patient; (II) acute history; (III) pancreatitis at first onset; (IV) abdominal MR examination. The exclusion criteria in this study were as follows: (I) inability to cooperate when MR imaging was performed; (II) a history of chronic pancreatitis; (III) AP due to pancreatic carcinoma; (IV) hypoproteinemia; and (V) hypoproteinemia or other peritoneal/retroperitoneal infection diseases; and (VI) iron deposition disorder (e.g., diabetes or blood system diseases). A total of 117 patients with AP were enrolled in this study. There were 45 women and 72 men, with an average age of  $50.41 \pm 15.55$  years (range, 15–84 years). All patients had a clinical assessment and laboratory workup on admission.

Patients without any pancreatic disorders were recruited as controls. They underwent upper abdominal examinations in our hospital as part of physical examinations or assessments for other diseases. The exclusion criteria for this control group were as follows: (I) pancreatic diseases; (II) cirrhosis of the liver; (III) cholecystitis; (IV) biliary tract stones; (V) peritoneal effusion; (VI) abdominal or retroperitoneal tumor; and (VII) iron deposition disorder (e.g., diabetes or blood system diseases). In total, 51 patients comprised the control group. There were 31 men and 20 women, with an average age of  $50.90 \pm 17.83$  years (range, 11–81 years).

### MRI techniques

All patients underwent an MRI scan within three days of admission. All MRI examinations were performed with a 3.0T scanner (Discovery MR 750; GE Medical Systems, Milwaukee, Wis) in the supine position. The sequences included two-dimensional coronal and axial single-shot fast spin-echo (SSFSE) T2-weighted; axial fast-recovery fast-spin-echo (FRFSE) T2-weighted with fat suppressed; water-only images generated from axial slab three-dimensional (3D) axial liver acquisition with volume acceleration (LAVA) MR imaging (T1WI); SSFSE radial series slab magnetic resonance cholangiopancreatography (MRCP); and multi-echo GRE T2\*WI. GRE T2\*WI was obtained in two breath-holds, and the parameters used were as follows: repetition time (TR), 57.3 ms; TE, 1.4, 3.5, 5.7, 7.8, 10.0, 12.2, 14.3, 16.5, 18.6, 20.8, 22.9, 25.1, 27.2, 29.4, 31.5, 33.7 ms; flip angle, 25°;

**Table 1** MRI parameters at 3.0T

Scanning sequences	TR (ms)	TE (ms)	Flip angle	Section thickness (mm)	Intersection gap (mm)	Matrix	FOV (cm)
AX 3D LAVA-Flex	4.2	2.6/1.3	15–20°	5	0	384×224	26–33
AX FRFSE T2WI	10,000–12,000	90–100	90°	5	0.5	256×192	36×34
COR SSFSE T2WI	2,500–3,500	80–100	90°	5	0.5	384×256	39×33
AX SSFSE T2WI	2,500–3,500	80–100	90°	5	0.5	320×256	39×33
MRCP	3,045	1,300	90°	40	40–50	384×224	32×34
AX 3D LAVA C+*	4.2	2.6/1.3	15–20°	5	0	384×224	26–33

\*, dynamic enhanced imaging is indicated with \*. MRI, magnetic resonance imaging; SSFSE, single-shot fast spin-echo; FRFSE, fast-recovery fast-spin-echo; LAVA, liver acquisition with volume acceleration; FOV, field of view; MRCP, magnetic resonance cholangiopancreatography; TR, repetition time; TE, echo time.

**Table 2** MRSI scoring system

Prognostic indicators	Characteristics	Points
Pancreatic inflammation	Normal pancreas	0
	Focal or diffuse enlargement of the pancreas	1
	Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2
	Single, poorly defined fluid collection or phlegm on	3
	Two or more poorly defined collection or presence of gas in or adjacent to the pancreas	4
Pancreatic necrosis	No necrosis	0
	<30%	2
	30–50%	4
	>50%	6

MRI, magnetic resonance imaging; MRSI, magnetic resonance severity index.

matrix, 256×192; section thickness, 4 mm; overlap, 2.5 mm; and phase field of view (FOV), 0.8 mm. Other parameters of conventional sequences used are in *Table 1*.

### *MRI image interpretation*

The original MR imaging data were loaded onto a workstation (advantage workstation 4.4; GE Healthcare) for reviewing. Two observers (with 5 and 8 years of experience in interpreting abdominal MR images) who were blinded to the laboratory data and clinical outcomes independently reviewed the MR images.

On the MR images, AP was defined as edematous and necrotizing pancreatitis. Pancreatic necrosis was defined as a well-margined area of signal intensity different from the signal intensity of a normal pancreas in a non-enhanced image along with the absence of enhancement in enhanced imaging (13). AP was graded as mild (0–3 points), moderate (4–6 points) or severe (7–10 points) according to the MRSI, which was derived from the CT-severity index (14) (*Table 2*).

The features of pancreas in the control group and AP group were observed on GRE T2\*WI, and the three segments of the pancreas were evaluated: the head, body, and tail. The regions of interest (ROIs) were placed in a corresponding anatomical position on the GRE-T2\*WI. For each segment, the ROI was placed into a central slice as large as possible, avoiding the main pancreatic duct and blood vessels, and the T2\* values obtained by copying ROIs in the 3 regions were averaged to provide a regionally independent T2\* value. In each subject, the T2\* values from the three segments are denominated as T2\*-head, T2\*-body, and T2\*-tail. The average of the T2\*-head, T2\*-body, and T2\*-tail is calculated as the T2\* value of the whole pancreas. In acute necrotic pancreatitis, we measured the T2\* value in the non-necrotic area in the same manner. In other words, if the necrosis is in the pancreatic tail, the average of the T2\*-head and T2\*-body values is the T2\* value of the whole pancreas. This calculation process for the T2\* value was completed using post-processing software in the workstation directly after scanning.

### *The APACHE-II score*

In clinical practice, the physician typically uses the APACHE II to evaluate the severity of AP (15). AP was graded as mild (0–7 points) or severe AP ( $\geq 8$  points) according to the APACHE II scoring system. An APACHE II score  $\geq 8$  points indicates much higher rates of morbidity and mortality (16). The APACHE II score of all 117 patients was calculated according to the laboratory data and clinical outcomes by two physicians, each with 4 years of experience in treating digestive diseases.

### *Statistical analysis*

Data derived from the MR images were expressed as the average of the two observers' findings, and the T2\* value was averaged by multiple measurements. Any discrepancies in the discrete data were discussed by the two observers until a consensus was reached. Kappa statistics were used to assess the inter-rater reliability of the MRSI scoring system. This statistic is generally interpreted as follows: a kappa value equal to or greater than 0.81 indicates very good agreement; a kappa value ranging from 0.80 to 0.61 indicates good agreement; a kappa value ranging from 0.60 to 0.41 indicates moderate agreement; and a kappa value of less than 0.41 indicates poor agreement.

The mean  $\pm$  SD and range were used to express the continuous variables. A paired-sample *t*-test and the Spearman rank correlation coefficient were used to analyze the T2\* value in the Control group. An independent *t*-test was used to determine the differences for the T2\* value between the Control group and AP group as well as the relationship between the T2\* value and the severity of AP according to MRSI and the APACHE II scoring system. The receiver operating characteristic (ROC) curve was used to analyze the usefulness of the T2\* value in diagnosing AP and necrotizing AP. Logistic regression analysis was used to test the association of the T2\* value with AP.

All statistical tests were performed using the Statistical Package for Social Sciences (SPSS) for Windows (Version 13.0, Chicago, IL, USA). P values  $< 0.05$  were considered significant.

## **Results**

### *Patient sample*

In the 117 patients with AP, the etiology of AP included gallstones in 49.57% (58/117) patients, alcohol abuse in

11.11% (13/117) patients, high-fat meals in 8.55% (10/117) patients, postoperative status in 1.71% (2/117) patients, and no specified etiology in 29.06% (34/117) patients.

Of the 51 patients in the control group, 21 patients had no abnormalities in the abdomen, 17 patients had hepatic cysts, 4 patients had hepatic hemangiomas and 9 patients had renal cysts according to MRI.

### *Findings of normal pancreas on T2\*WI*

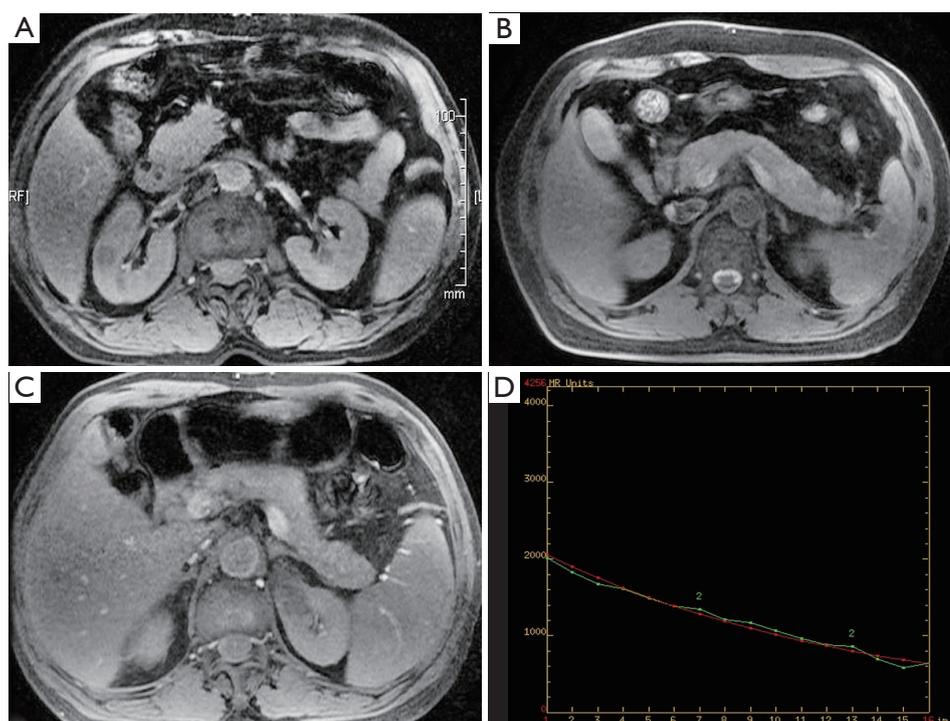
In the 51 patients in the control group, the pancreas showed a well-marinated and consistently homogeneous isointensity on GRE-T2\*WI. The corresponding signal decay curve is relatively smooth (*Figure 1*). The T2\*-head, T2\*-body and T2\*-tail averaged  $22.74 \pm 3.45$  ms,  $20.51 \pm 3.40$  ms and  $19.92 \pm 3.89$  ms, respectively. The average T2\* value was  $21.06 \pm 2.64$  ms for the whole pancreas in Control group. The T2\* value was significantly different between the head and body ( $t=3.59$ ,  $P=0.001$ ) and between the head and the tail ( $t=4.297$ ,  $P<0.001$ ), but no significant difference was found between the body and the tail ( $t=1.30$ ,  $P=0.20$ ).

### *Findings of AP on conventional MRI and T2\*WI*

In the 117 patients with AP, 79.49% (93/117) of the patients were diagnosed with edematous AP, whereas 20.51% (24/117) of the patients were diagnosed with necrotizing AP on MRI. The agreement between the observers for the MRSI score was very good ( $\kappa=0.84$ ). The MRSI score was  $3.84 \pm 1.75$  points (range, 0–10 points). A total of 35.04% (41/117), 58.97% (69/117) and 5.98% (7/117) of the patients had mild, moderate and severe AP according to MRSI, respectively.

On GRE T2\*WI, the pancreas in edematous pancreatitis shows an ill-defined and enlarged but homogeneous signal intensity, and the signal decay curve is relatively smooth (*Figure 2*). However, there were also two patients with clinically confirmed AP (2/117, 1.71%) who had no evidence on the conventional sequence; the T2\* value had increased visually with one had T2\* values of the head, body and the tail being 20.143, 34.031 and 33.212 ms, respectively.

For necrotizing pancreatitis, the non-necrotic area manifests as well as edematous pancreatitis. The signal decay of a necrotic area no longer universally hold, particularly for hemorrhage areas; a local hemorrhage could result in a decreased T2\* value and signal loss on the signal decay curve (*Figures 3,4*). In the cohort, 10.25%



**Figure 1** The upper abdomen T2\*WI for a 38-year-old male with normal pancreas. The pancreas can be visualized clearly by GRE T2\*WI and shows a well-marinated and consistently homogeneous isointensity (A-C). The T2\* values of the pancreas can also be measured. The corresponding signal decay curve of the pancreatic tail was also derived (D). GRE, gradient recalled echo; T2\*WI, T2\*-weighted imaging.

(12/117) of patients had elevated signal intensity in the water-only images generated from LAVA-flex, suggesting hemorrhage. Hemorrhage occurred in the pancreatic parenchyma, which manifested as hyperintensity on T1-weighted LAVA-Flex imaging and as corresponding signal loss and a decreased T2\* value on T2\*WI. The T2\* value of the pancreatic hemorrhage lesion was  $11.80 \pm 2.30$  ms, which was significantly lower than that of control group ( $t = -13.94$ ,  $P < 0.001$ ). In the 12 patients with hemorrhage, there were 5 patients whose hemorrhage occurred in the peripancreatic area, which lacked the characteristic appearance on the GRE T2\*WI. The T2\* values of the non-necrotic area for the T2\*-head, T2\*-body and T2\*-tail were  $26.85 \pm 6.66$  ms ( $n = 114$ ),  $26.02 \pm 6.04$  ms ( $n = 110$ ) and  $24.46 \pm 5.57$  ms ( $n = 106$ ), respectively, with an average of  $25.86 \pm 4.90$  ms for whole pancreas in AP patients.

#### *T2\* values in the diagnosis and evaluation of the severity of AP according to MRSI*

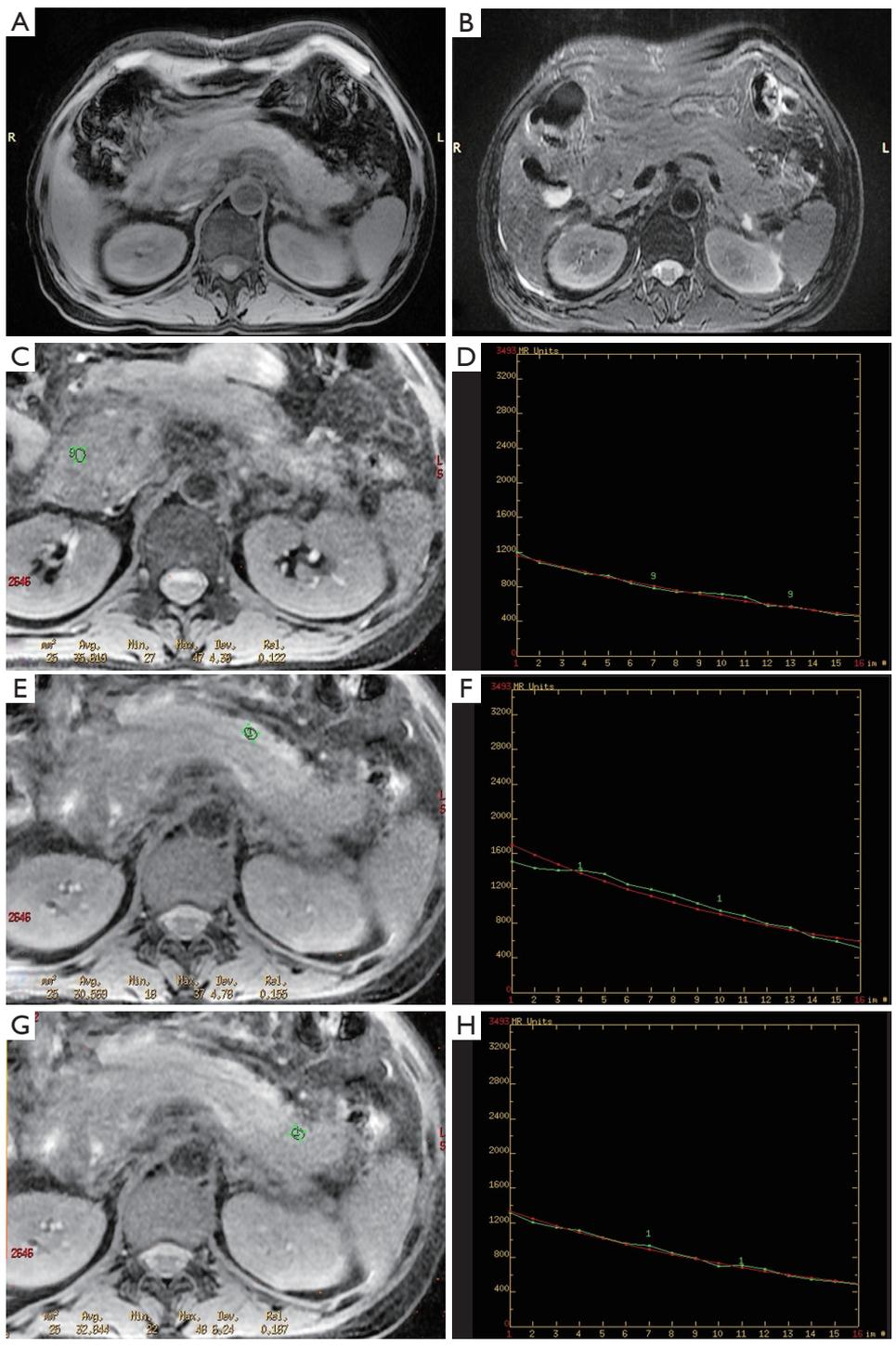
The T2\* value of the AP group was significantly higher than that of the control group ( $t = -8.02$ ,  $P < 0.001$ ). For a

diagnosis of AP, the area under the ROC curve of the T2\* value is 0.80 ( $P < 0.001$ ) (Figure 5). In regression models, AP was associated with a one standard deviation increment in the T2\* value (OR = 1.37; 95% CI: 1.22–1.53;  $P < 0.001$ ).

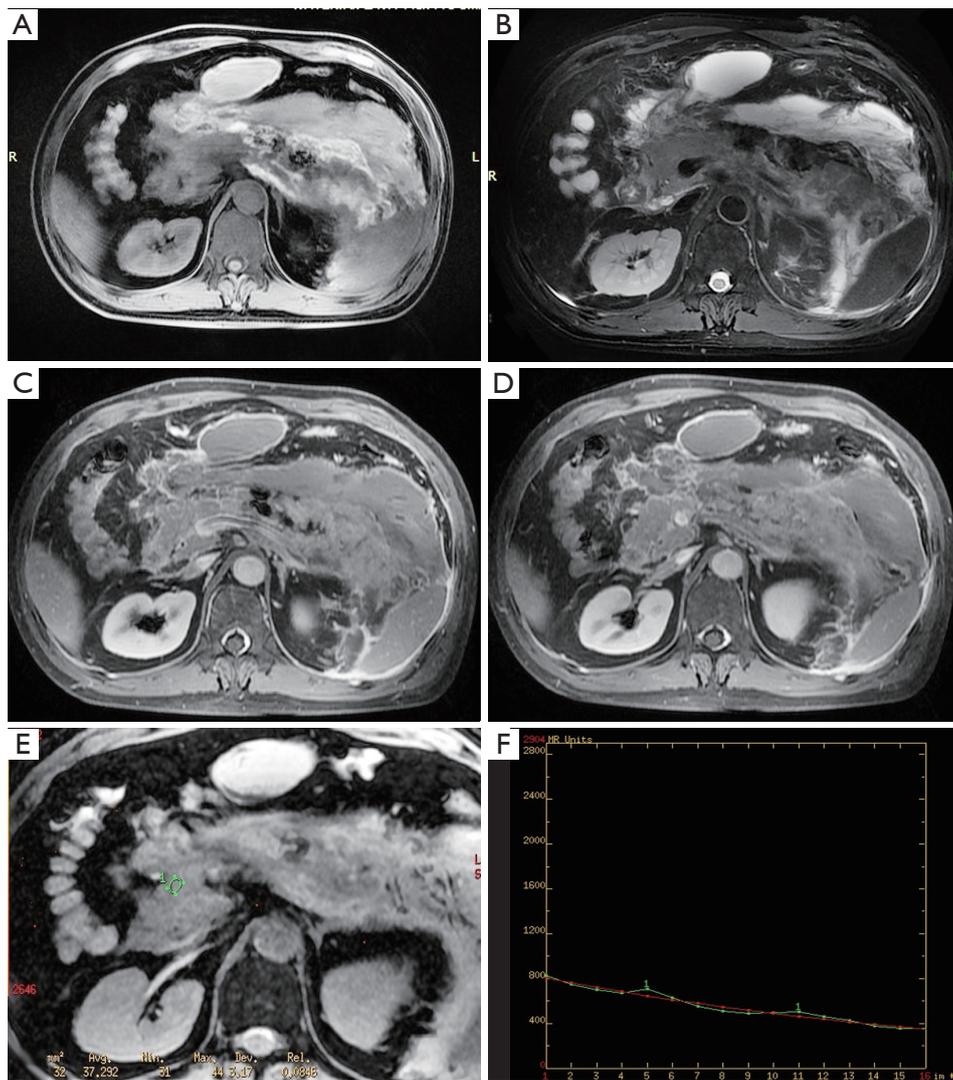
The T2\* value shows significant difference between edematous AP and necrotizing AP ( $25.28 \pm 4.56$  vs.  $28.10 \pm 5.59$  ms;  $t = 2.57$ ,  $P = 0.01$ ). For diagnosing necrotizing AP, the area under the ROC curve of the T2\* value is 0.66 ( $P = 0.02$ ). The T2\* values were  $24.08 \pm 4.41$ ,  $26.49 \pm 4.91$  and  $29.99 \pm 3.83$  ms in mild, moderate and severe AP according to MRSI, respectively. The T2\* values increase along with the gradual increase in the MRSI score (Table 3) and are significantly different between mild and moderate AP ( $P = 0.011$ ) as well as mild and severe AP ( $P = 0.002$ ); however, there are no significant differences between moderate and severe AP ( $P = 0.071$ ).

#### *The relationship between the T2\* value and the severity of AP according to APACHE II*

Among the 117 patients with AP, the mean APACHE II score was ( $5.10 \pm 3.51$ ) points (range, 0–14 points). A total



**Figure 2** A 45-year-old male with acute edematous pancreatitis. T1WI and T2WI depict evidence of AP: an enlargement of the pancreas and inflammatory exudation in the peripancreatic fat (A,B). The T2\*-head, T2\*-body and T2\*-tail values were 35.619 ms, 30.569 ms and 32.844 ms, respectively, and their corresponding signal decay curves were derived (C-H). AP, acute pancreatitis.



**Figure 3** A 45-year-old male with acute hemorrhagic necrotizing pancreatitis. Pancreatic necrosis can be found on T1WI (A), T2WI (B) and enhancement images (C,D). The T2\* value measured in the non-necrotic area was 37.292 ms (E,F).

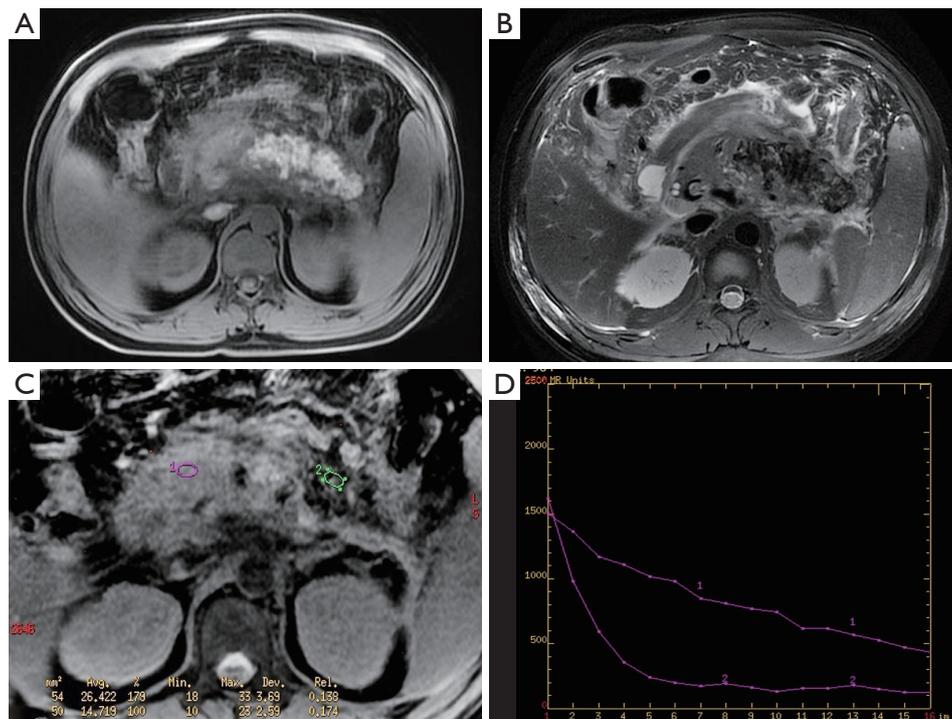
of 89 patients had mild AP (APACHE II score <8 points), whereas 28 patients had severe AP (APACHE II score  $\geq$ 8 points). The T2\* value was  $25.73 \pm 4.80$  and  $26.25 \pm 5.26$  ms in mild and severe AP according to APACHE II, respectively. There is no significant difference between mild and severe AP ( $t = -0.49$ ,  $P = 0.63$ ).

## Discussion

In this study, we found that GRE-T2\*WI of pancreas is feasible; reliable image quality combined with T2\* values can contribute to diagnosing AP and determining its severity. The T2\* value of the AP group was higher than

that of the Control group, both for necrotizing AP and edematous AP, and it had a tendency to increase gradually along with the increasing MRSI scores. Furthermore, the addition of GRE-T2\*WI to conventional MR imaging may help in detecting potential hemorrhages.

On GRE-T2\*WI, the normal pancreas showed a well-marinated and consistently homogeneous isointensity, and the corresponding signal decay curve was relatively smooth. The T2\* values of the head, body and tail of normal pancreas were  $22.74 \pm 3.45$ ,  $20.51 \pm 3.40$ ,  $19.92 \pm 3.90$  ms, respectively, and the T2\* values were significantly higher for the head than for the body and tail. Our results agree with other related reports (17-19), which use DWI to research



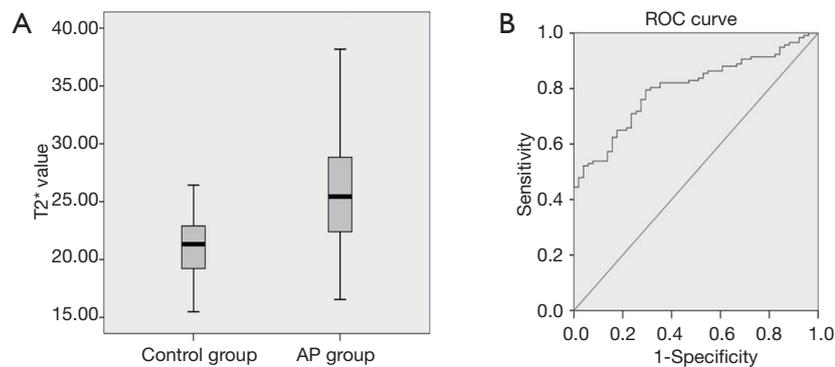
**Figure 4** A 48-year-old male with severe acute pancreatitis (AP). The irregular streak and sheet signal intensity in the pancreas show hyperintensity on fat-suppressed T1WI images (A) and hypointensity on T2WI images (B), which suggest hemorrhage. The T2\* values can be measured in the area with and without hemorrhage (C) and were 14.719 ms and 26.422 ms, respectively. The greater decrease in the corresponding signal decay curve can be seen in the hemorrhage area (D).

the different ADC values of the head, body, and tail of normal pancreas. These different values may be explained by the differences in tissue composition, with an increasing number of islets of Langerhans and a higher density of fatty components toward the tail (18). Another possible reason is the different blood supply to the head, body and tail of normal pancreas (20).

Edematous AP, as well as the non-necrotic area in the necrotizing AP, shows enlarged and ill-defined but homogeneous signal intensity. The T2\* value of the AP patient group was  $25.86 \pm 4.90$  ms. AP is an acute inflammatory disease; as in other inflammatory diseases, the pathological changes are mainly edema, hyperemia and inflammatory cell infiltration. Cell edema will lead to an increase in hydrogen protons, which can prolong T2\* relaxation and results in an increased T2\* value (21,22). In this study, we found that the T2\* value in the AP group was higher than that in the control group, and for diagnosing AP, the area under the ROC curve of the T2\* value was 0.801. The logistic regression analysis more

reliably demonstrated the usefulness of the T2\* value in the diagnosis of AP. Our results indicate that T2\*WI can reveal the inflammation of pancreas in AP and is helpful for a quantitative diagnosis of AP. In our study, 1.69% (2/117) of the patients showed normal pancreas on the MR image but with an increased T2\* value. Thus, we have evidence to speculate that an increased T2\* value can help in the diagnosis of AP. In future studies, the results of this work need to be confirmed with a larger population.

AP with hemorrhage in the pancreatic parenchyma but not in the peripancreatic area shows a decrease in the T2\* value and a signal loss on the signal decay curve. The addition of these image characteristics to conventional MRI can aid in the qualitative diagnosis of AP and in the detection of hemorrhage. In clinical practice, the basic treatment for AP is conservative medical therapy. Therefore, it is difficult to obtain histopathological evidence of hemorrhage (23). Although CT and angiography can detect hemorrhage, the main disadvantage of these techniques is ionizing radiation (7). Romanova *et al.* (24) reported that CT



**Figure 5** The boxplot of T2\* value and the ROC curve. (A) The boxplot of the control group and AP group. It shows that the T2\* value of the AP group is higher than that of the control group; (B) the ROC curve (to analyze and evaluate the T2\* value to diagnose AP). The area under the ROC curve of the T2\* value is 0.80 ( $P < 0.01$ ). AP, acute pancreatitis; ROC, receiver operating characteristic.

**Table 3** The T2\* value in different severities of AP according to MRSI

Statistics	MRSI		
	Mild	Moderate	Severe
T2* value	24.08±4.41	26.49±4.91	29.99±3.83
T value	-2.580*	-3.335**	-1.830***
P value	0.011*	0.002**	0.071***

\*, represents mild vs. moderate; \*\*, represents mild vs. severe, \*\*\*, represents moderate vs. severe. MRI, magnetic resonance imaging; AP, acute pancreatitis; MRSI, magnetic resonance severity index.

is also less sensitive than MRI in detecting hemorrhage. To our knowledge, the hyperintensity on T1-weighted images with fat suppression often refers to the hemorrhage (25,26), but investigators have reported that the T1 hyperintense signal may vary with the combination of blood, high protein content, fat, and calcification (27). In this case, GRE T2\*WI was used for the differential diagnosis. Breakdown products of hemorrhage have a large paramagnetic effect or recalled magnetic susceptibility effects, which can change the uniformity of the local magnetic field, as shown in the decrease of the T2\* value and the signal loss on the signal decay curve (9). Thus, the addition of GRE-T2\*WI to conventional MR imaging can contribute to the detection of hemorrhage. The peripancreatic hemorrhage lacked a characteristic appearance on GRE T2\*WI, which may be due to peripancreatic inflammatory exudation or even fluid collection.

The T2\* value in necrotizing AP is higher than that in edematous AP, and the ROC curve (area =0.66,  $P=0.02$ ) also

suggests the usefulness of the T2\* value in the diagnosis of necrotizing AP. Failure in the microcirculation led to a histopathologically significant increase in acinar cell necrosis and edema level in AP (28). We can speculate that the pancreatic cell edema level is higher in necrotizing AP than in edematous AP.

The MRSI was derived from the CT severity score index developed by Balthazar (29), which included the severity scores of pancreatic inflammation and pancreatic necrosis. In this study, we found that the T2\* values increased along with the increase in MRSI scores. Although the T2\* value had a tendency to increase gradually along with the MRSI scores, there was no significant difference between moderate and severe AP ( $P=0.071$ ). One possible reason is that the higher the AP severity is according to the MRSI, the more severe the inflammation of the pancreas is. As mentioned above, the T2\* value increases in necrotizing AP, and the moderate and severe AP group also contained some patients with necrotizing AP. Another possible reason is that the sample size was not large enough to attain statistical significance in each of its separate arms.

APACHE II has the advantage of reflecting systemic complications. Patients with higher APACHE II scores often have more serious general conditions (30). In this study, we found that there was no statistically significant difference between mild and the severe AP according to the APACHE II score. One possible reason for this result may be that the T2\* value reflects only local pancreatic features, which are hardly affected by the APACHE II.

Our study suffers from several limitations: (I) detection bias. We chose a relatively small ROI. Schwenzer *et al.* (31) studied the iron deposition of pancreas using ROIs ranging

from 1.0–1.4 cm<sup>2</sup>; small ROIs allow flexibility when measuring the T2\* value, which is useful to avoid the main pancreatic duct, large vessels, etc. Measurement precision can be improved by averaging the values of repeated measurements; (II) performance bias. The second limitation is that the time interval between the MRI and the onset of AP was variable, which may have affected T2\* values and the MRSI scores. We performed MRI examinations within three days after admission to minimize this variability. In addition, the APACHE II score was calculated by several physicians and nurses, which may have led to variations between the different observers. However, this likely does not affect the main results of the study; (III) selection bias. Some diseases, such as chronic anemia, diabetes, etc., may lead to iron deposition in pancreas. Some recruited patients did not have a clear medical history, which can lead to selection bias. In our study, all of the patients met the inclusion and exclusion criteria.

In summary, GRE T2\*WI demonstrated some characteristics of the normal pancreas and AP, which was helpful for detecting hemorrhage. The addition of GRE T2\*WI to conventional MR can contribute to diagnosing AP and determining its severity. Because it provides a quantitative diagnosis and evaluation of AP, GRE-T2\*WI may be a feasible and effective tool for AP.

## Acknowledgements

None.

## Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

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