Introduction

Acute pancreatitis (AP) is a common clinical problem. The median mortality of AP is 10% but may reach 20–30% in severe acute pancreatitis (SAP) with multiple organ dysfunction syndromes (1). The most commonly involved organ in AP and particularly in SAP is the gastrointestinal tract, which is correlated with poor outcomes in AP patients (2). Assessing the severity of AP and gastrointestinal tract involvement for the proper management of AP patients are critical components of any strategy designed to reduce AP mortality (3).
Magnetic resonance imaging (MRI) has an excellent resolution of soft tissue, and it has evolved as a valuable tool to non-invasively identify AP complications, trouble shoot difficult cases of AP and assess response to treatment without the radiation exposure. MRI can accurately evaluate the necrosis, hemorrhage, ductal system and inflammation associated with AP (4-6). Compared with traditional examination methods, such as endoscopy, barium meals and CT scans, MRI has some unique advantages, including improved soft tissue contrast, and no ionizing radiation (7). Therefore, the use of MRI to evaluate the gastrointestinal tract is gradually increasing (7-10).

In clinical studies of intestinal complications of AP, González Jiménez et al. (11) described five SAP patients who had duodenal stenosis. Lal et al. (12) reported a case of large bowel obstruction in a 31-year-old postpartum female, secondary to severe gallstone pancreatitis, Liu et al. (2) reported that intestinal mucosal function was injured in early phase of AP especially in patients with organ dysfunction, which may be a stimulus for development of multiple organ dysfunction and correlate with bad outcome in AP patients. Animal experiments (13) revealed that gastric emptying and intestinal peristalsis were decreased by acute necrotizing pancreatitis and undiagnosed gastrointestinal tract damage may lead to fatal outcome if an enterogenic infection occurs. Chen et al. (14) found that 65% of patients of AP complicated with acute gastrointestinal mucosal lesions. In the imaging studies, as early as in 1980, Mendez et al. (15) reported that 5 of 102 AP patients had involvement of the transverse mesocolon and small bowel mesentery in CT examinations. Liu et al. (16) observed that gas volume was significantly elevated in patients with AP in plain abdominal radiographs. Raguwanshi et al. (17) found that 88% AP patients had mesentery, greater omentum and transverse mesocolon abnormalities on CT examination. However, these examinations are not very sensitive and are generally inadequate to demonstrate the full extent of the gastrointestinal tract abnormalities associated with AP and the minute structure changes of gastrointestinal tract could be clearly demonstrated with MRI. Furthermore, MRI doesn’t involve radiation, it can be repeatedly used for follow-up.

We conducted the current study to assess gastrointestinal tract involvement in AP with MRI and analyzed the severity of gastrointestinal tract abnormalities on MRI in relation to those abnormalities evaluated by the MR severity index (MRSI) and the Acute Physiology and Chronic Healthy Evaluation II (APACHE II) scoring system. MR follow up of the gastrointestinal tract was used to evaluate the therapy effect of AP.

Methods

Patient population

Our institutional review board approved this retrospective study and informed consent was waived since this was a retrospective study. The inclusion criteria for the study of AP were: (I) acute history; (II) pancreatitis at first onset; (III) three-fold elevated amylase or lipase, excluding other causes of elevated enzymes; and (IV) abdominal MR examinations within 2 days of the pancreatitis onset. The exclusion criteria were: (I) inability to cooperate when performing MRI; (II) incomplete clinical records; (III) history of chronic pancreatitis; (IV) AP due to pancreatic carcinoma; (V) history of gastrointestinal surgery; and (VI) digestive tract diseases, such as neoplastic lesions or inflammatory disease, and other diseases which could lead to gastrointestinal tract abnormalities (18,19).

Two hundred and thirty-three patients with AP were admitted to our institution between October 2015 and April 2017. Twenty-four patients were excluded, including five patients with severe motion artifacts during MRI scanning, five patients with appendix operations, two patients with esophageal cancer, five patients with gastric cancer, three patients with colorectal cancer, one patient with pancreatic cancer, and three patients with cirrhosis. The final study group consisted of 209 consecutive AP patients, 62 of whom had follow-up MRI.

The control group was selected from the patients in our daily clinical caseload who underwent MRI over the same recruitment period. The inclusion criteria for the control group were as follows: (I) adequate bowel wall visualization; (II) nonspecific abdominal symptoms, such as nausea, vomiting, abdominal pain with diarrhea, bloody diarrhoea; (III) had no other disease which can cause abnormality of gastrointestinal tract, such as cirrhosis, portal hypertensive, ascites and heart failure. The exclusion criteria for control group were as follows: (I) AP; (II) history of neoplasms of digestive system; (III) history of intraperitoneal tumor; (IV) had previous abdominal surgical interventions; (V) other digestive tract diseases, such as inflammatory disease and ileus.

MRI techniques

MRI was performed on a 1.5-T MR scanner with 38 mT/M...
gradients and 120 mT/M per second slope (Signa Excite; GE Medical Systems, Milwaukee, WI, USA) using a phased array torso-pelvis coil. No antiperistaltic agent or enteric contrast was administered. The coverage area of axial scan was apex of the liver to the umbilical. The sequences included axial fast spoiled gradient echo (FSPGR) T1-weighted imaging with fat suppression [repetition time (TR) ms/echo time (TE) ms = 150–170/1.6; flip angle = 80°; matrix = 512x160–192; field of view (FOV) = 26–32 cm; section thickness = 5 mm; number of excitation (NEX) = 1], gradient-echo (GRE) T1-weighted in-phase and out-of-phase MR imaging (TR ms/TE ms = 150/4.4, 2.2; flip angle = 90°; matrix = 256x192–224; FOV = 26–32 cm; section thickness = 5 mm; NEX = 1), respiratory-triggered (R-T) axial fast recovery fast spin-echo (FRFSE) T2-weighted MR imaging with fat suppression (TR ms/TE ms = 10,000–12,000/90–100, TR determined by the frequency of respiration; section thickness = 5 mm; intersection gap = 0.5 mm; matrix = 256x192; NEX = 3; and FOV = 34 cm x 34 cm), coronal and axial single shot fast spin-echo (SSFSE) T2-weighted MR imaging (TE = 90–100 ms; 2 s between slice acquisitions; section thickness = 5 mm; intersection gap = 0.5 mm; matrix = 384x224; one-half signal acquired; and FOV = 33 cm x 33 cm), SSFSE radial series slabs MR cholangiopancreatography (MRCP) (TE = 1,300 ms; 6 s between image acquisitions; section thickness = 40 mm; matrix = 384x224; one-half signal acquired; FOV = 30 cm x 30 cm), and three-dimension (3D) FSPGR dynamic enhanced MR imaging. Dynamic enhanced imaging was performed with an axial fat saturated 3D FSPGR sequence. Gadolinium chelate (Magnevist, Schering Guangzhou Co., China) was administered (0.2 mmol/L per kilogram of body weight) intravenously at approximately 3.5 mL/s by projector (Spectris MR Injection System, Medrad Inc., USA) injection, followed by a 20-mL saline solution flush. First-pass arterial enhancement was optimized with a timing bolus sequence (axial FMPSPGR). Dynamic imaging was performed during breath-holding before the injection (unenhanced), immediately after the injection (hepatic arterial phase), 30 s after the injection (early venous phase), and 1 min after the injection (late venous phase). An additional delayed phase was acquired using 2D FSPGR fat suppression axial T1-weighted imaging.

**MRI interpretation**

The original MRI data were transferred to a workstation (GE, AW4.1, Sun Microsystems, Palo Alto, CA, USA) to be reviewed. Two observers (with 5 and 8 years of experience in interpreting abdominal MRI, respectively) blinded to the laboratory data and clinical outcomes independently reviewed pancreas on the MRI. Another two observers (with 5 and 8 years of experience in interpreting abdominal MRI, respectively) blinded to the laboratory data and clinical outcomes independently reviewed the gastrointestinal tract.

On the MR images, AP was categorized as edematous and necrotic pancreatitis (20). The severity of AP was graded according to MRSI (20). According to MRSI, AP was divided into mild, moderate and severe APs (MRSI was 0–3, 4–6, 7–10 points separately) (20,21). We divided the gastrointestinal tract anatomy into stomach, duodenum, jejunum, ileum, ascending colon (including the ileocecal junction), transverse colon (including the hepatic flexure and splenic flexure), descending colon and the sigmoid colon. Wall thickening was defined as over 10 mm for stomach wall and over 3 mm for intestine, when their lumens were well distended (22-24). Abnormalities of the thickened gastrointestinal tract walls included the degree of thickening, signal intensity change, and symmetric versus asymmetric thickening. Lumen dilatation was defined as diameter over 30 mm for small intestine and over 60 mm for the colon (24).

The change in the bowel wall signal intensity was compared with adjacent normal bowel on T2WI and contrast enhanced T1WI. When the bowel wall signal intensity was close to that of the normal bowel, we defined it as isotense. When the signal intensity was much higher than that of normal bowel wall, it was defined as hyperintense, and when it was much lower than normal bowel wall, it was defined as hypointense.

Although the axial scans did not include lower abdomen, the entire gastrointestinal tract was evaluated on the coronal and sagittal scans.

**APACHE II score**

In all 209 patients, the APACHE II score was calculated using 13 common physiological and laboratory values from the first 48 hours after admission without knowledge of the MRI findings, obtained from the medical records by clinician or nurse. According to the standard of Atlanta, an APACHE II score of 8 was selected as a cut-off point to differentiate between mild and severe cases (25,26).

The medical records of AP patients were reviewed for observing the recovery of gastrointestinal functions, including relief of abdominal pain, recovery of eating, hematuria, amylase recovery and anal passing gas.
Statistical analysis

All of the quantitative data derived from MRI findings are reported as the average of the two observers and qualitative data were used to assess the inter-rater reliability between the two reviewers. Kappa statistics were used to assess the inter-rater reliability between the two reviewers.

The MRSI and APACHE II scores are reported as ranges and as the mean ± standard deviation. The frequencies of gastrointestinal tract abnormalities were calculated in the AP and in the control groups, and the Chi-squared test or Fisher exact test was applied to determine the significance of their difference. Chi-squared test or Fisher exact test was used for the differences in the prevalence of gastrointestinal tract abnormalities in mild, moderate, and severe AP according to the MRSI scores or as mild or severe AP according to the APACHE II scores. The Spearman’s rank correlation coefficient was calculated to test the correlation of the gastrointestinal tract abnormality with the MRSI and the APACHE II scores. Initial and follow up MRI examinations of gastrointestinal tract abnormalities were compared by using the Chi Square Test or Fisher exact test and paired sample t-test.

All statistical tests were calculated using Statistical Package for Social Sciences (SPSS) for Windows (Version 11.5, Chicago, IL, USA). P<0.05 was considered significant.

Results

Patient sample

Of the 209 patients with AP, there were 107 men and 102 women with an average age of 53±16 years (range, 16–82 years). The etiology for AP was biliary in 50% (105/209), high-fat diet in 18% (37/209), alcoholic in 5% (10/209), postoperative status in 1% (3/209), and undetermined in 26% (54/209) of patients. Of the 62 patients with follow up MRI examination, there were 40 men and 22 women with an average age of 53±14 years (range, 22–80 years). The etiology for AP was biliary in 52% (32/62), high-fat diet in 16% (10/62), alcoholic in 13% (8/62), and undetermined in 19% (12/62) of patients. All patients accept fasting until they can tolerate an oral diet.

In the control group, there were 53 men and 47 women with an average age of 46±12 years (range, 13–71 years). Thirty-seven patients were normal on MRI, twenty-four patients had renal cysts, eighteen patients had liver cysts, eleven patients had liver hemangiomas, and ten patients had splenomegaly.

MRI finding of AP

Of the 209 patients with AP, 33% (68/209) received dynamic enhanced MRI. Sixty-two patients had followed up MRI examination in 13±8 days (range, 3–49 days) after admission. The agreement between the observers for MRSI was satisfactory (κ=0.73, P<0.05). Approximately 35%, 59% and 6% of the patients had mild, moderate and severe APs, respectively according to MRSI. The MRSI score averaged 3.1±1.8 points (range, 0 to 10 points).

Approximately 63% (132/209) of the patients with AP had at least one gastrointestinal tract abnormality in the initial MRI examination, compared with 5% (5/100) of the patients in the control group (P<0.05). Among control subject, the gastrointestinal tract abnormalities included smooth mild stomach wall thickening with no mural stratification (r=2), smooth mild duodenum wall thickening with no mural stratification (r=1), dilatation of the gastrointestinal tract (r=2). The agreement of the two raters was generally good (Table 1).

The prevalence of gastrointestinal tract abnormalities was 47% in mild AP, 93% in moderate AP and 100% in severe AP, respectively (among the three groups, P<0.05). Gastrointestinal tract abnormalities were correlated with the MRSI score (r=0.46, P<0.05).

Gastrointestinal tract abnormalities on MRI were noted in 132 patients, including mild smooth gastrointestinal tract wall thickening, mural stratification, and bowel dilatation. Thickened bowel wall was noted in 45 patients, which showed diffuse enhancement in 22 patients and stratified enhancement in 23 patients during the arterial phase after Gd intravenous injection.

Among the AP patients, wall thickening involved duodenum (Figure 1) (27%), ascending colon (26%), stomach (20%), transverse colon (15%), jejunum (Figure 2) (14%) and ileum (6%). Dilatation of the gastrointestinal tract was seen in 8% (Figure 2, Table 2).

During treatment, 62 patients had follow up MRI examination, 16 of whom (26%) had a gastrointestinal tract abnormality on follow up examination, significantly lower than that on the initial MRI (P<0.05) (Figures 3-7). The difference of the frequencies of gastrointestinal tract abnormalities for initial and follow-up MRI examination are shown in Table 3. The difference for the frequencies of stomach, duodenum, jejunum, ileum, ascending and descending abnormal changes between before and after the treatment had statistical significance (P<0.05). There was no difference of transverse colon and bowel dilatation before and after the
It was found that gastrointestinal tract wall thickness was greatly thinner after treatment (P<0.05) (Table 4).

The APACHE II score

Among the 209 patients with AP, the mean APACHE II score was 5.98±4.0 points (ranging from 0 to 23 points). A total of 150 patients had mild AP (APACHE II score <8 points), whereas 59 patients had severe AP (APACHE II score ≥8 points). Approximately 83% (49/59) of the patients with severe AP had gastrointestinal tract abnormalities, significantly more than the 57% (86/150) of patients with mild AP and gastrointestinal tract abnormalities (P<0.05). The presence of gastrointestinal tract abnormalities was slightly correlated with the APACHE II score (r=0.19, P<0.05).

In the follow-up MRI exam, the structure of gastrointestinal tract restored to revert in MRI, coinciding with relief of abdominal pain, recovered appetite, cessation of hematuria, amylase recovery and anal passing gas.

Discussion

In this study, 63% of patients had at least one gastrointestinal tract abnormality on MR imaging. The patterns of the abnormalities included even and mild gastrointestinal tract wall incrassate, mural stratification, stratification enhancement on artery phase after Gd intravenous injection, and dilatation of gastrointestinal tract. The most common abnormalities included thickened duodenal wall (27%), thickened descending colon wall (26%), and thickened stomach wall (20%). The prevalence of gastrointestinal tract abnormalities was 47% in mild AP, 93% in moderate AP and 100% in severe AP, respectively. Gastrointestinal tract abnormalities were correlated with the MRSI score. It was found that gastrointestinal tract wall thickness was greatly thinner after treatment. Our results depicted the patterns and the evolution of gastrointestinal tract in AP by using MRI which may be helpful for early recognition and institution of appropriate therapy to improve the prognosis of AP. We found the prevalence of gastrointestinal tract abnormality was 63% which was higher than previous studies, in which gastrointestinal tract abnormality was exceedingly rare, and
Figure 1 A 60-year-old man with moderate AP. MRSI =3, APACHE II =3. The duodenal wall thickness is 10 mm (A and B, arrows), with stratified enhancement on arterial phase after Gd intravenous injection (C, arrow). The enhancement become diffuse on delayed-phase (D, arrow). AP, acute pancreatitis; MRSI, MR severity index; APACHE II, Acute Physiology and Chronic Healthy Evaluation II.

Figure 2 A 42-year-old woman with moderate AP. MRSI =4, II =8. On axial T2-weighted (A) and T1-weighted (B) images, and coronal T2-weighted image (C) the small intestine is mildly expanded and filled with liquid (arrows). The thickening of the jejunal wall (A, pentagon, short arrows) is smooth and mild, and mural stratification is observed on T2-weighted images. AP, acute pancreatitis; MRSI, MR severity index; APACHE II, Acute Physiology and Chronic Healthy Evaluation II.
only isolated case reports had described them (11,12). Our results demonstrate that the prevalence of gastrointestinal tract abnormality is higher in MRI study than previous trials. The possible reason may be that MRI is more sensitive to depict the slight changes in gastrointestinal tract in AP.

In this study gastrointestinal tract wall thickening was...
Figure 5 A 57-year-old man, MRSI =3, APACHE II =6. (A) The ascending colon wall was thickened in the initial exam (arrow); (B) 6 days after admission, ascending colon wall became thin (arrow). MRSI, MR severity index; APACHE II, Acute Physiology and Chronic Healthy Evaluation II.

Figure 6 A 54-year-old man, MRSI =6, APACHE II =4. (A) The intestinal tract was expanded and filled with gas and liquid (arrows); (B) 6 days after admission, the dilatation has resolved (arrows). MRSI, MR severity index; APACHE II, Acute Physiology and Chronic Healthy Evaluation II.

Figure 7 A 63-year-old woman, MRSI =4, APACHE II =3. (A) The ileal wall is thickened in the initial exam (arrow); (B) 10 days after admission, ileum wall became thin (arrow). MRSI, MR severity index; APACHE II, Acute Physiology and Chronic Healthy Evaluation II.
the most common finding in AP patients. Inflammatory substances can stimulate the gastrointestinal tract in patients with AP, leading to the excessive output of gastric juices and subsequent mucosal and submucosal edema (27). Some patients exhibit a three-layer structure referred to as the “target sign” (28). Tolan et al. (29) considered this pattern acute wall edema, and noted a direct correlation with inflammatory activity. In this study we found most AP patients exhibited gastrointestinal tract wall thickening, some with a stratified appearance on MRI that was similar to the “target sign” (28). After treatment, the mural stratification resolved on follow-up MRI. Our results suggested that the thickened intestinal wall in AP patients is derived from acute wall edema and can resolve soon after treatment.

We found frequent mild smooth thickening of the intestinal wall, similar to that of other non-tumorous bowel wall thickening patterns. Ramalho et al. (30) observed that the wall thickness usually ranges between 5 and 10 mm for patients with small bowel Crohn's disease, similar to the range we noted (Table 2) in AP patients. Bowel wall thickening has been observed in bowel ischemia edema, bleeding and infection, although this thickness was unrelated to disease severity (31). Gastrointestinal tract wall thickness reduced after treatment, suggesting resolved edema after effective treatment.

We noted two patterns of wall enhancement in the initial exam; diffuse, and stratified. Tolan et al. (29) proposed that the patterns of wall enhancement could help determine the likely level of disease activity. Tolan et al. reported that stratified enhancement was observed in combination with submucosal edema in active disease and that diffuse enhancement commonly represented transmural inflammation. Low-level inhomogeneous enhancement was often observed in fibrosis (22). We consider that the wall thickening we observed with AP resulted from active

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<th>Follow-up (cases) (%)</th>
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<td>Dilatation of gastrointestinal tract</td>
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Table 3

### Table 3

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<th>Follow-up (mm)</th>
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<td>Thickened descending colon wall</td>
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Table 4

### Table 4

AP, acute pancreatitis; MRI, magnetic resonance imaging.

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inflammation, and that this inflammation was transmural in some cases. In the follow-up examination, we noted smooth mild wall thickening with no mural stratification, confirming that active inflammation could resolve soon after treatment.

AP inflammation can spread through connected anatomic compartments, or across biofilms damaged by pancreatic enzymes (32,33). The closer to the pancreas, the more obvious the impact on the affected tissues and organs. As duodenum, ascending colon and descending colon are retroperitoneal and stomach, jejunum and transverse colon are close to the pancreas (34), we found these organs could be frequently involved in this study. In contrast, the sigmoid colon, far from the pancreas, showed no involvement in this study.

The pattern of wall thickening is not sufficient to identify the cause of thickened bowel wall, although a characteristic distribution of involvement may help identify specific diseases. For example, thickening of the ascending colon and jejunum are common in cirrhosis (18). Intestinal mechanical obstruction usually occurs in the small intestine (35). Crohn's disease usually localizes to the distal ileum (29). The most commonly affected intestine in ulcerative colitis is the left colon (9). Mesenteric vascular lesions are associated with ischemic bowel disease (36). Immune responses and hypoalbuminemia often affect both the small and large intestines (37). The most commonly involved segment is the large intestine in inflammatory enteritis (38). In blunt abdominal trauma, bowel injury in included bowel wall transaction discontinuity, extraluminal air, focal bowel wall thickening, free peritoneal fluid, mesenteric infiltration or hematoma on CT examination. Duodenal was most commonly affected bowel in pancreatic injuries (39). In this study we found the most commonly affected intestines in AP were duodenum and descending colon.

One thing we need to pay special attention to is AP patients with inflammatory bowel disease (IBD). Antonini et al. (40) found that patients with IBD have increased risk of AP. In this study, no AP patients had IBD. AP and IBD may have similar presentation, therefore a pancreatic disease could not be recognized in patients with IBD. However, elevation of pancreatic enzymes only, without symptoms nor imaging suggestive of pancreatitis, is not an indication to start a treatment but is recommended to follow up in first instance. On the other hand, patients with IBD presenting with pancreatitis-like abdominal pain should be investigated to rule out a concomitant pancreatic disease. Liu et al. (16) observed that gas volume of gastrointestinal tract in was significantly elevated in patients with AP. In this study, we also found some AP patients had dilatation of the gastrointestinal tract with gas. But it was a nonspecific finding and can occur in various diseases (41).

In this study, no patient had gastrointestinal fistula or bowel obstruction which is rarely complication of SAP but one that clinicians should be aware of due to its high mortality. According to the previous research, the sites of fistula may involve any site of gastrointestinal, either in localization or diffusion (42-44). Most of the cases were confirmed clinically 4–8 weeks after onset of the disease, which suggests that the development of gastrointestinal fistula is associated with the long-term effects of the pancreatic or peripancreatic inflammation and infection (44). In our study, the finding is in disagreement with the results, none of AP patients has gastrointestinal fistula or obstruction. In our study, AP patients had a small number of SAP patients (6%) and rather short-term follow up (62 patients had follow up MRI examination in 13±8 days after admission). As we found in our study, colonic and duodenal were the most easily involved structures. Colonic and duodenal fistulas are the two commonest forms of fistula in SAP. For the clinical outcome, the occurrence of colonic fistula and obstruction is associated with worse outcome (45).

Optimal feeding has always been regarded as an important means to improve the outcome in AP patients. Starting early enteral feeding as soon as possible was thought to be important, but until recently, no scientific study was available to determine the exact time and it remains debatable (46,47). In clinical therapy, if patients can tolerate an oral diet without abdominal pain, nausea, vomiting, or ileus, they may be allowed to have oral food (48). In this study, all patients accept fasting until they can tolerate an oral diet, we found the gastrointestinal tract abnormalities resolve when their abdominal symptoms improved significantly. Gastrointestinal tract monitoring with MRI may provide objective index for the recovery of bowel function, exact time of enteral feeding and curative effect.

MRSI exhibits advantages in evaluating local complications (49). APACHE II scores are the “traditional” multifactorial scoring systems for predicting the severity, pancreatic necrosis, and mortality of AP (50). In this study we found that gastrointestinal tract abnormalities on MRI increased with the increasing severity of AP. Gastrointestinal tract abnormalities were correlated with the MRSI and APACHE II scores. We speculate that gastrointestinal tract abnormalities were mainly due to massive pancreatic fluid leakage.
There are several limitations of this study. Firstly, some patients with AP were treated by conservative therapy including fasting and nasogastric drainage, and gastrointestinal preparation cannot be made before MRI examination. This has a great influence on observing the intestinal wall. In order to increase the accuracy of the study, scans from a control group of patients without AP were included in this study. Secondly, the time interval between MRI examination and the onset of AP is variable, which may have affected the prevalence of gastrointestinal tract and the MRSI scores. Therefore, we performed MRI within 48 h after admission to minimize variability.

In conclusion, gastrointestinal tract plays an important role in the development of AP. Early recognition and institution of appropriate therapy may improve prognosis of AP. Gastrointestinal tract abnormalities on MRI are common in AP and they are positively correlated with the severity of AP on MRI and may add value for determining the severity of AP. MRI can be used to monitor treatment effects of gastrointestinal tract abnormality and can provide objective index for the recovery of bowel function, exact time of enteral feeding and curative effect.

Acknowledgements

None.

Footnote

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

Ethical Statement: Our institutional review board approved this retrospective study and informed consent was waived since this was a retrospective study.

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