MR Images of Bowel Wall Thickening in Patients with Minimal to Moderate Cirrhosis: Comparison with Clinical Findings

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Purpose : To evaluate the MR imaging findings of bowel wall thickening in patients with minimal to moderate cirrhosis, and analyze their clinical significances comparing with laboratory findings.

Materials and Methods : We assessed retrospectively the MRI findings of 123 patients with minimal to moderate cirrhosis, and compared these with the clinical laboratory findings. We evaluated the involved sites and MR image findings of thickened bowel wall, as well as the presence of collateral vessels, ascites, and splenic size. These were compared with serum albumin and bilirubin levels, and prothrombin time.

Results : Gastrointestinal wall thickening was detected at 37 sites in 25 patients (20 %), and more frequently detected in moderate cirrhosis (29%) than in minimal cirrhosis (17%). Jejunum and ascending colon were the most common sites of bowel wall thickening; each was involved at 22 and 9 sites, respectively. Ascending colonic wall thickening was more commonly detected in moderate cirrhosis than in minimal cirrhosis. The thickened bowel wall showed symmetric contour, high signal intensity on T2-weighted images, mixed iso- and low signal intensity on T1-weighted images, and homogeneous or target-like enhancement. Serum albumin level was significantly lower in patients with bowel wall thickening (3.3 ± 0.9 g/dl vs. 3.9 ± 0.7 g/dl; $p=0.0024$). Serum bilirubin level was significantly higher in patients with bowel wall thickening (1.7 ± 1.0 mg/dl vs. 1.4 ± 1.2 mg/dl; $p=0.0160$). Bowel wall thickening did not significantly correlate with the presence of collateral vessels, ascites, splenic size, and prolongation of prothrombin time.

Conclusion : In minimal to moderate cirrhosis, the MR imaging evaluation of bowel wall thickening was useful for estimating the severity of cirrhosis and laboratory findings.

Index words : Abdomen
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Introduction

Bowel wall thickening has been reported in patients with cirrhosis by CT, usually presumed to be secondary to edema caused by portal hypertension or oncotic pressure (1 - 3). However, most previous CT studies were performed in patients with severe cirrhosis, and there are no details regarding comparison with clinical findings. In severe cirrhosis, it is difficult to define the affecting factors for bowel wall thickening, because the causes of the bowel wall thickening can be obscured when there are major complications such as recurrent variceal bleeding, bacterial infection with motility disturbance, peritonitis, hepatic failure, and renal dysfunction (4 - 7). Therefore, for the evaluation of potential affecting factors for bowel wall thickening, clinical correlation in patients with minimal to moderate cirrhosis can be helpful.

With the increasing routine use of MR imaging for evaluation of cirrhosis, bowel wall thickening can be noted on MR images. To our knowledge, there are no published reports describing the MR findings of bowel wall thickening compared with clinical findings in patients with minimal to moderate cirrhosis. Therefore, we evaluated the involved sites and MR imaging findings of bowel wall thickening in patients with minimal to moderate cirrhosis, and analyzed their clinical significances comparing with associated radiologic features and laboratory findings. We determined if there were any differences in patients with minimal to moderate cirrhosis, including the following hypotheses:

1. Frequency and sites of bowel wall thickening would differ in patients with minimal versus moderate cirrhosis.

2. Imaging findings such as collateral vessels, ascites and/or splenic size would differ in patients with versus those without bowel wall thickening.

3. Laboratory values, such as serum albumin, serum bilirubin, and/or prothrombin time, would differ in patients with versus those without bowel wall thickening.

Materials and Methods

Patient Selection

In our institution's radiology information system database, 502 patients from a single hepatology practice were found who had MRI examinations for the evaluation of cirrhosis between January 1994 and August 2001. From these patients, hepatic or extrahepatic malignancy was ruled out by preliminary review of the MR images, alpha fetoprotein levels and clinical follow up. Other conditions that may cause gastrointestinal wall thickening, such as cardiac failure, nephrotic syndrome, acute pancreatitis, and inflammatory, ischemic, and neoplastic disease of the gastrointestinal tract, were excluded by review of the patients' clinical records. We selected 128 patients who had available MRI scans and clinical data performed less than 45 days before or after the MRI scan. The degree of liver insufficiency was classified as minimal (A, n=88), moderate (B, n=35), and severe (C, n=5) according to Child-Pugh's criteria. We selected 123 patients (87 men and 36 women: age range, 19 - 77 years; mean age, 51.3 years) with minimal to moderate cirrhosis. The causes of cirrhosis in these patients were as follows: hepatitis C (n=29), hepatitis C and alcohol abuse (n=5), and hepatitis B (n=89).

MR Imaging

All patients underwent MR imaging at 1.5 T (Signa, GE Medical Systems, Milwaukee, Wis). All examinations included axial T1- and T2-weighted MR imaging. T1-weighted imaging included one or more of the following sequences: conventional spin echo (repetition time msec/echo time msec = 400 - 600/11 - 22), in-phase gradient echo (80 - 210/4.2 - 4.6 with 70 °- 90 ° flip angle), and opposed-phase gradient echo (80 - 210/1.8 - 2.5 with 60 °- 90 ° flip angle). T2-weighted imaging included the following sequences: conventional spin echo (1,500 - 3,000/50 - 100), breathing-averaged fast spin echo (3,000 - 7,500/91 - 104 [effective]) with or without fat suppression, and breath-hold fast spin echo (2,500 - 4,200/70 - 138 [effective]). Most patients also underwent multiphasic dynamic imaging after intravenous injection of 0.1 mmol per kilogram of body weight gadopentetate dimeglumine (Magnevist; Berlex Laboratories, Wayne,

NJ), using either T1-weighted opposed-phase gradient-echo imaging as described above, or more recently fat-suppressed three-dimensional spoiled gradient echo imaging (5 - 7/1.3 - 2.1/12 °- 20 °). Other imaging parameters included 256×128 - 256 imaging matrix, usually with use of a rectangular field of view to reduce the number of phase-encoding views, and 5 - 12-mm-thick sections with 2-mm or less section gap. Motion compensation techniques included flow and respiratory compensation. Superior and inferior spatial presaturation pulses and fat saturation were also applied in the majority of patients.

Image Interpretation

Without knowledge of clinical information, the MR images of the patients with cirrhosis were evaluated on teleradiology or film in 103 and 20 patients, respectively. The scans of each patient were read by two readers, both board-certified radiologists. Instances of disagreement among the readers were resolved by consensus.

The stomach, duodenum, jejunum, ascending colon, transverse colon and descending colon were considered as separate anatomic sections of the gastrointestinal tract and were assessed for wall thickening in each patient. The jejunum was identified by the location of its loops in the left upper quadrant and by its abundance of folds. Because the majority of MR scans of cirrhotic patients did not include the pelvis, the sigmoid colon, rectum and ileum were excluded from the assessment. The thickness of bowel wall was measured from the inner lumen to outer serosal layer at an expanded site. The gastric wall was considered thickened if the thickness of the anterior wall of the body exceeded 10 mm (8). The duodenum, jejunum and colon were considered thickened if the wall thickness was more than 3 mm (9).

Whenever gastrointestinal wall thickening was identified, symmetry, signal intensity, and enhancement pattern were assessed. The symmetry of wall thickening was described as concentric if the whole circumference of the gastrointestinal wall was uniformly thickened and as eccentric if the wall of the involved gastrointestinal segment was asymmetrically thickened (9). The signal intensity of thickened bowel wall was described as high, iso, and low signal intense comparing with that of the paraspinal muscles. The enhancement pattern of thickened bowel wall was described as homogeneous if it showed homogeneous attenuation within the thickened wall, and as target-like if it showed alternating rings of high and low attenuation within the thickened wall and, as described by Balthazar (10).

We also evaluated the presence of collateral vessels including perisplenic vessels, splenorenal shunt, periumbilical veins, and gastric varices. Presence of ascites was evaluated on coronal and axial images, and classified as small, moderate, and large amount. Size of the spleen was measured with longest craniocaudal dimension, on coronal images.

Clinical Evaluation

For evaluating clinical manifestations of portal hypertension, we reviewed serum albumin and bilirubin levels, and prothrombin time, obtained less than 45 days (mean duration was 8.2 days; range of duration, 0 - 45 days) before or after MR scans.

Statistical Analysis

Statistical significance of the site of bowel wall thickening and Child's classification was tested by Fisher Exact test, and statistical significance of all of these associations was tested by using the 2 test. Mean serum albumin and bilirubin levels, and prothrombin time for patients with or without bowel wall thickening were calculated for statistical significance by using Mann Whitney U test. Data were presented as the mean±SD. A P value of 0.05 or less was considered to indicate a statistically significant difference.

Results

MR images

Gastrointestinal wall thickening was seen in 25 (20%) of 123 patients, and in 15 of 88 (17%) patients with minimal cirrhosis and in 10 of 35 (29%) patients with moderate cirrhosis. The bowel wall thickening was detected at 37 sites in 25 patients; jejunum in 22, ascending colon in 9, transverse colon in 3, descending colon in 2 and stomach in one. All 25 patients with bowel wall thickening had wall thickening of jejunum in 16 patients, ascending colon in 3 patients, and contiguous wall thickening of jejunum and ascending colon in 6 patients (Table 1). Wall thickening of the

ascending colon with/without jejunal thickening was more commonly detected in patients with moderated cirrhosis than in patients with minimal cirrhosis ($p < 0.05$), and concomitantly present from ascending to desending colon.

Normal and abnormally thickened bowel wall was demonstrated on T2-weighted images (Fig. 1, 2). All thickened bowel walls showed symmetric contour, and showed high signal intensity on T2-weighted images and mixed iso- to low signal intensity on T1-weighted images (Fig. 3). On contrast enhanced MR images, the thickened bowel wall showed homogeneous enhancement at 19 (51%) sites (Fig. 4), and a target-like pattern at 18 (49%) sites (Fig. 5).

Associated findings

Collateral vessels were detected in 43 (35%) patients with mild and moderate cirrhosis. This included 12 (48%) of 25 patients with bowel wall thickening and 31 (32%) of 98 patients without bowel wall thickening. Collateral vessels tended to be more common in patients with bowel wall thickening, but there was no statistically significant difference between the groups. Ascites was detected in 26 (21%) patients; this was small in 8 patients, moderate amount in 13 patients, and severe in 5 patients. Ascites was seen in 6 (24%)

Table 1. Bowel Wall Thickening Site and Child's Classification

Bowel Wall Thickening	Total	Child's Classification	
		A	B
	n = 123	n = 88	n = 35
Jejunum	16 (13)	13 (15)	3 (9)
Jejunum and Ascending Colon	6 (5)	1 (1)	5 (14)
Ascending Colon	3 (2)	1 (1)	2 (6)
Total	25 (20)	15 (17)	10 (29)

Note.- Numbers in parentheses are percentages
Fisher Exact test p-value: 0.0095

Table 2. Associated Findings and Clinical Findings in Minimal and Moderate Cirrhosis

Findings	Total n = 123	BWT n = 25	Non-BWT n = 98	P-value
Collateral vessels	43 (35)	12 (48)	31 (32)	0.9597
Ascites	26 (21)	6 (24)	20 (20)	0.6946
Splenic size (cm)	12.4±2.6	13.0±2.3	12.2±2.7	0.0642
Serum albumin (g/dl)	3.8±0.8	3.3±0.9	3.9±0.7	0.0024
Serum bilirubin (mg/dl)	1.5±1.1	1.7±1.0	1.4±1.2	0.0160
Prothrombin time (sec)	13.6±1.8	13.8±1.6	13.5±1.8	0.2167

Note.- Numbers in parentheses are percentages, Data present mean±SD
BWT: Patients with bowel wall thickening
Non-BWT: Patients without bowel wall thickening

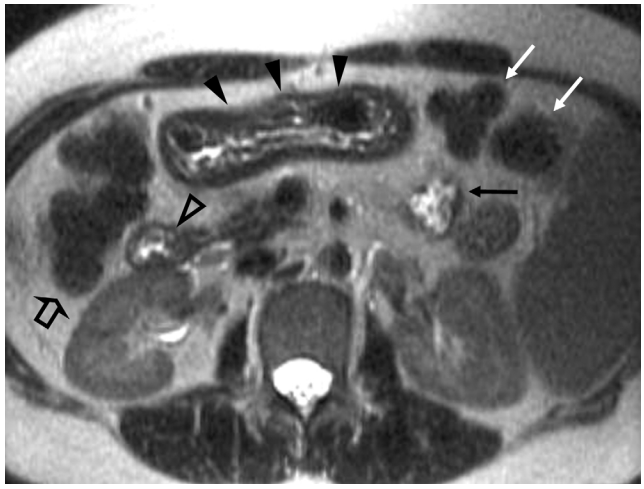


Fig. 1. 60 year-old woman with minimal cirrhosis. T2-weighted single-shot fast SE MR image (/185) shows normal walls of stomach (black arrowheads), duodenum (open black arrowhead), jejunum (black arrow), ascending colon (open black arrow) and transverse colon (white arrows).

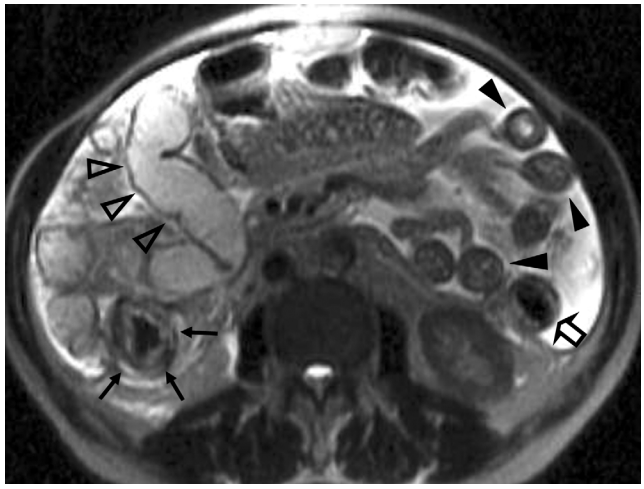
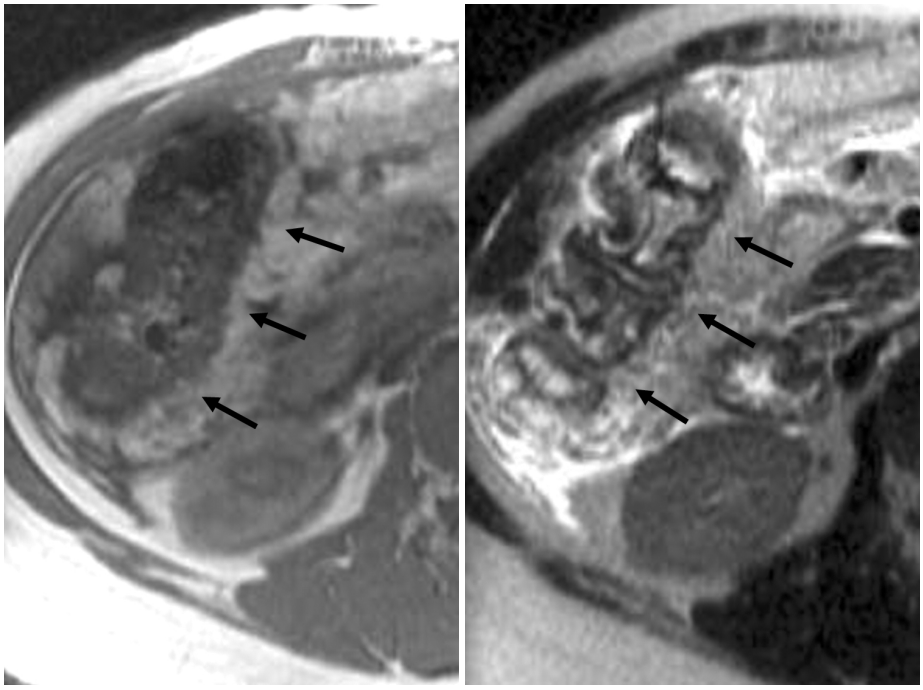


Fig. 2. 68 year-old man with moderate cirrhosis. T2-weighted single-shot fast SE image (/186) shows symmetric wall thickening with high signal intensity in jejunum (black arrowheads), ascending colon (black arrows), and descending colon (open black arrow). Markedly dilated right-sided small bowel (open black arrowheads) suggests associated ileus.

patients with bowel wall thickening and in 20 (20%) patients without bowel wall thickening, respectively (not statistically significant). Mean splenic size was 13.0 ±2.3 cm in patients with bowel wall thickening, and 12.2±2.7 cm in patients without bowel wall thickening (not significantly different).

Clinical findings

Serum albumin level was significantly lower in patients with bowel wall thickening: 3.3±0.9 g/dl vs. 3.9±0.7 g/dl ($p < 0.05$). Serum bilirubin level was higher in patients with bowel wall thickening: 1.7±1.0 mg/dl vs. 1.4±1.2 mg/dl ($p < 0.05$). Mean prothrombin time was 13.8±1.6 sec and 13.5±1.8 sec in patients with and without bowel wall thickening, respectively (not significantly different).



a b

Fig. 3. 58 year-old man with minimal cirrhosis. A. T1-weighted fast spoiled gradient-echo image (180/4, 80 ° flip angle) shows mixed iso- and low signal intensity of thickened ascending colon (arrows). B. T2-weighted single-shot fast SE image (/185) shows internal high signal intensity of thickened ascending colon (arrows).

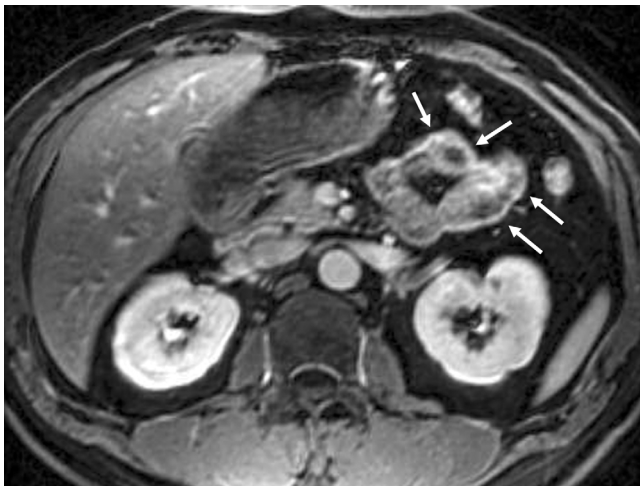


Fig. 4. 34 year-old man with minimal cirrhosis. Contrast enhanced gradient-echo MR image (6/2, 15 ° flip angle) shows homogeneous enhancement in thickened jejunum (arrows).

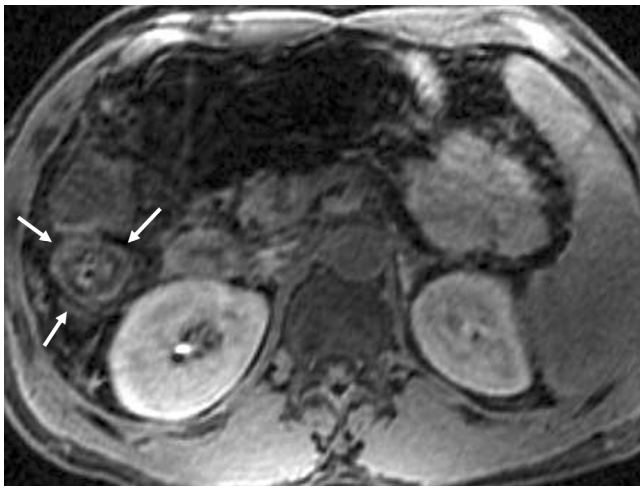


Fig. 5. 58 year-old man with minimal cirrhosis. Contrast enhanced gradient-echo MR image (6/2, 15 ° flip angle) shows target-like enhancement in thickened ascending colon (arrows).

Discussion

Gastrointestinal wall thickening was detected in 20% of patients with minimal to moderate cirrhosis, less than in previous studies that found this in 37% to 63% of patients with cirrhosis (1, 2). In our study, bowel wall thickening was more frequently detected in moderate cirrhosis (29%) than in minimal cirrhosis (17%), this suggests that frequency of bowel wall thickening was depended on the severity of cirrhosis in the study populations. Therefore, in our cases, low incidence of bowel wall thickening is likely to be the result of exclusion of the severe cirrhosis. Although jejunal wall thickening was most commonly detected in minimal to moderate cirrhosis, ascending colonic wall thickening was more frequently detected in moderate cirrhosis than in minimal cirrhosis. Therefore, presence of wall thickening ascending colon with/without jejunal thickening can be considered as suggestive findings of moderate cirrhosis.

Most frequently involved sites of the bowel wall thickening were jejunum and ascending colon, this suggests that the superior mesenteric vein is more commonly affected by portal hypertension than the inferior mesenteric vein. The inferior mesenteric vein drains blood from the left colon via the splenic vein, splenorenal shunt and short gastric varices, and these collateral pathways can explain why the left colon might have more pathways for decompression than the right colon (2). However, in spite of the same drainage pathway to the superior mesenteric vein, it is not clear why the jejunum is more affected by increased pressure of the superior mesenteric vein than the ascending colon. This may be secondary to differences in drainage gravity; the jejunal vein has a larger angle to the superior mesenteric vein than the mid-colic vein, and needs more drainage power against gravity than the mid-colic vein. Moreover, arterial communication through the collateral arc between mid-colic and left colic arteries can be decreased the flow influence to the ascending colon in patients with portal hypertension. This can be the reason for concomitant wall thickening of transverse and descending colon: wall thickening of transverse colon was always associated with wall thickening of ascending colon, and wall thickening of descending colon was associated with wall thickening of ascending and transverse colon. This is consistent with a previous study that reported that if the ascending colon was normal, no wall thickening was seen in the transverse or descending colon (1).

Although portal hypertensive gastropathy has been reported as more than 50%, on endoscopic findings (11), because of lack of gastric expansion and superficial location of the lesion, the lesions are rarely detected on CT or MR images. In our study, gastric wall thickening was only detected in one patient who had diffuse wall thickening of the entire intestine.

Radiologic findings of bowel wall thickening in cirrhosis has been reported as uniformly thickened, irregularly narrowed, or slightly effaced and dilated on barium study (3, 12), and as concentric contour change on CT scan (1). In this study, thickened bowel wall showed symmetric contour, and high signal intensity on T2-weighted images, and mixed iso- and low signal intensity on T1-weighted images. This MR signal intensity is likely to be from bowel wall edema due to venous congestion, consistent with the reported histopathologic findings of the thickened bowel wall in cirrhosis (13, 14). On contrast enhanced MR images, thickened bowel wall showed a homogeneous or target-like enhancement. Although previous study showed no patients with ring-like enhancement in cirrhosis (1, 3), target-like enhancement was detected at the same frequency as homogeneous enhancement in our study. The target sign suggests intramural edema with progressed venous congestion.

Although collateral vessels are important signs of portal hypertension (15, 16), we did not find statistically significant correlation between bowel wall thickening and presence of collateral vessels. This result suggests that vascular decompression through collateral vessels may be insufficient for reducing the bowel wall thickening in cirrhosis. Ascites tended to be more frequently detected in patients with bowel wall thickening than in patients without bowel wall thickening, but there was no significant difference between the groups. The most likely pathogenic mechanism of ascites is portal hypertension causing increased hydrostatic pressure within the mesenteric veins, however, several systemic vasoactive factors and aldosterone catabolism are thought to play a major role in the development of ascites (15, 17 - 19). Although size of the spleen in patients with cirrhosis has been

found to correlate with the severity of hepatic functional reserve (16), we found there was not significant relationship between the splenic size and bowel wall thickening. Even a patient without bowel wall thickening may have an enlarged spleen, bowel wall thickening can be disappeared in the same patient with consistent splenic size.

Hypoalbuminemia, specifically a serum albumin level of 2 g per 100 ml or lower, has been reported as the commonest cause of noninflammatory intestinal edema (3, 12, 20). The low serum albumin leads to a decrease in the colloidal osmotic pressure with leakage of intravascular fluid across capillary membranes into the interstitial tissues (3). In our study, serum albumin level was significantly lower in patients with bowel wall thickening than in patients without bowel wall thickening. These results suggest that serum albumin level is one of the major affecting factors for bowel wall thickening in cirrhosis. Plasma bilirubin concentration varies directly with bilirubin production and inversely with hepatic bilirubin clearance. The impaired hepatic function may cause changes in hepatic secretion of biliary lipids and bilirubin, and elevated serum bilirubin concentrations in patients with cirrhosis can result from overproduction of bilirubin and a concomitant decrease of biliary transport capacity (21). In our study, mean bilirubin level was significantly higher in patients with bowel wall thickening than in patinets without bowel wal thickening. Therefore, presence of the bowel wall thickening can be provided the important indicator of hypoalbuminemia and elevated sereum bilirubin levels in patients with minimal to moderate cirrhosis.

Prothrombin time is a screening test for abnormalities of the extrinsic coagulation pathway, and prolonged prothrombin time is associated with increased risk of hemorrhage (22, 23). Although bleeding tendency may play a role for bowel wall thickening with intramural oozing and congestion, in our study, mean prothrombin time was not significantly different between patients with or without bowel wall thickening.

This study has some limitations. First, most of our patients had viral hepatitis, so our population probably does not represent the entire spectrum of cirrhosis. Second, this was a retrospective study and there was no histopathologic proof or endoscopic correlation. Although endoscopic evaluation with biopsy is useful for diagnosis of bowel wall disease, in cirrhotic patients, it is limited according to the bleeding tendency from varices, thrombocytopenia or prolonged prothrombin time. To minimize this limitation, we excluded malignant disease and included only those patients without clinical evidence of other gastrointestinal disease.

In conclusion, most commonly involved sites of bowel wall thickening were jeunum and ascending colon in minimal to moderate cirrhosis, and ascending colonic wall thickening was more commonly detected in moderate cirrhosis than in minimal cirrhosis. Symmetrically thickened bowel wall shows high signal intensity on T2-weighted images, mixed iso- and low signal intensity on T1-weighted images, and homogeneous or target-like pattern on contrast enhanced images. Low serum albumin and high bilirubin levels were significantly correlated with presence of bowel wall thickening in minimal to moderate cirrhosis.

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경증과 중등도 간경화 환자에서 장관 비후의 MR 영상: 임상소견과의 비교

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목적: 경증과 중등도 간경화 환자에서 장관 비후의 MR 영상 소견을 분석하고, 혈액검사 소견과 비교하여 임상적 의미를 알아보고자 한다.

대상과 방법: 경증과 중등도 간경화 환자 123명을 대상으로 MR영상소견을 분석하고 혈액검사 소견과 비교하였다. MR 영상 소견은 장관벽 비후, 우회혈류의 유무, 복수, 비장의 크기를 분석하였다. 이 소견들과 혈청 단백질, 담즙색소 수치, 혈액응고(프로트롬빈) 시간을 비교하였다.

결과: 위장관벽 비후는 25명 환자에서 37개의 위치에서 있었고, 경증 간경화(17%) 보다 중등도 간경화(20%)에서 더 높게 나타났다. 장관벽 비후의 가장 흔한 위치는 공장과 상행결장이었고 각각 22명과 9 명에서 나타났다. 상행 결장의 비후는 경증 간경화 보다 중등도 간경화에서 더 높게 나타났다. 비후된 장관벽은 대칭적이며 T2강조영상에서 고신호강도를 보였고 T1강조영상에서 동등신호강도와 저신호강도가 섞인 신호강도를 나타냈으며 균질하거나 과녁형 조영증강을 보였다. 혈청 단백질은 장관벽 비후가 있는 환자에서 의미있게 낮은 수치를 보였다(3.3±0.9 g/dl vs. 3.9±0.7 g/dl; p=0.0024). 혈청 담즙색소는 장관벽 비후가 있는 환자에서 의미있게 높은 수치를 보였다(1.7±1.0 mg/dl vs. 1.4±1.2 mg/dl; p=0.0160). 장관벽 비후는 우회혈류의 유무, 복수, 비장의 크기, 혈액응고 시간의 지연과 의미있는 관계가 없었다.

결론: 경증과 중등도 간경화에서 MR영상 소견은 간경화의 중증도와 혈액검사 소견을 평가하는데 유용하였다.

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