Transmural Bowel Necrosis From Acute Mesenteric Ischemia and Strangulated Small-Bowel Obstruction: Distinctive CT Features

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OBJECTIVE. The purpose of this study was to assess whether transmural bowel necrosis has distinct CT features based on the three main causes: occlusive acute mesenteric ischemia (AMI), nonocclusive AMI, and strangulated small-bowel obstruction (SBO).

MATERIALS AND METHODS. From January 2010 to December 2017, the records of all patients with a pathologic diagnosis of transmural bowel necrosis were extracted from the pathology department database of a university hospital. The inclusion criteria for the study were presence of transmural bowel necrosis at pathologic examination and available contrast-enhanced CT images obtained within the 24 hours before surgery. Seventy-seven patients were finally included. The CT scans were retrospectively independently reviewed by two abdominal radiologists to identify the classic CT findings of transmural bowel necrosis. Statistical analyses were performed.

RESULTS. Pneumatosis intestinalis was statistically more frequent in nonocclusive AMI (59%) than in occlusive AMI (29%) and strangulated SBO (7%) ($p < 0.01$), as were superior mesenteric venous gas (55%, 29%, and 0%; $p < 0.01$) and portal venous gas (48%, 10%, and 0%; $p < 0.01$). Decreased or absent bowel wall enhancement was more frequent in AMI than in SBO (nonocclusive AMI, 83%; occlusive AMI, 81%; SBO, 56%; $p = 0.02$), as was thinned bowel wall (nonocclusive AMI, 52%; occlusive AMI, 48%; SBO, 18%; $p = 0.02$). Spontaneous hyperattenuation of the bowel wall was more frequent in strangulated SBO (41%) than in nonocclusive AMI (10%) and occlusive AMI (14%) ($p < 0.01$).

CONCLUSION. Transmural bowel necrosis has distinct CT findings according to its three main causes. Occlusive AMI is characterized by an absence of bowel wall enhancement and less mesenteric fat stranding, nonocclusive AMI by a high prevalence of pneumatosis intestinalis and portal venous gas, and strangulated SBO by spontaneous hyperattenuation of the bowel wall and an absence of pneumatosis intestinalis and portal venous gas.

B owel infarction is a life-threatening condition that is always a diagnostic and therapeutic emergency regardless of its cause. Intestinal infarction is caused by a decrease in blood supply to the digestive tract, and according to the severity and duration of ischemia, lesions can range from patchy mucosal necrosis to transmural bowel necrosis [1]. The two main causes of transmural bowel necrosis are acute mesenteric ischemia (AMI) and strangulated small-bowel obstruction (SBO). AMI can be separated into occlusive AMI, which accounts for two-thirds of cases, and nonocclusive AMI, which accounts for one-third of cases [2].

Occlusive AMI is characterized by vascular occlusion of mesenteric arterial or venous trunks. The origin is atheromatous, embolic, or dissecting, and approximately 70% of cases affect the superior mesenteric artery [2]. Nonocclusive AMI is characterized by splanchnic hypoperfusion commonly caused by a low cardiac output state due to sepsis, congestive cardiac failure, hypovolemia, aortic insufficiency, renal or hepatic disease, or cardiac surgery [2, 3]. Transmural necrosis can be the consequence of small-bowel loop strangulation in the context of bowel obstruction. Ischemia is observed in 10% of cases of SBO [4] and is almost exclusively a consequence of closed-loop obstruction [5]. It is usually caused by an adhesive band and less frequently by an external or internal hernia. As a consequence, different clinical contexts can lead to the same pathologic condition, that is, transmural bowel necrosis [1].

The two conditions—AMI and SBO—that can lead to the histologic diagnosis of trans-

mural bowel necrosis seem to have different radiologic features [6]. The typical CT features of bowel infarction due to strangulated SBO include spontaneous hyperattenuation of the bowel wall and mesenteric haziness [4, 7–9]. The CT features of AMI include pneumatosis intestinalis, portal venous gas, and lack of wall enhancement [10, 11]. To our knowledge, the CT features of the occlusive and nonocclusive forms of AMI have never been directly compared, and it is unclear whether these two causes have different patterns at an equivalent degree of pathologic severity. In addition, there is a lack of knowledge regarding CT features associated with reversible mesenteric ischemia [12], so we focused on transmural bowel necrosis to have a strong reference standard. The aim of this study was to assess whether transmural bowel necrosis has distinct CT features according to the three main causes: occlusive AMI, nonocclusive AMI, and strangulated SBO.

Material and Methods
Patient Population
This retrospective study was performed at a single university hospital. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori human research committee approval. This was a retrospective observational study reporting data on a large cohort and not individual patients. Therefore, according to French legislation, no ethics committee approval was required.

From January 2010 to December 2017, the records of all patients with a pathologic diagnosis of transmural bowel necrosis were extracted from the pathology department database. The inclusion criteria were the presence of transmural bowel necrosis at pathologic examination and available contrast-enhanced CT images, defined by at least one portal phase acquisition and an unenhanced abdominal acquisition. The CT examination had to have been performed within 24 hours before surgery to limit bias associated with underestimation of intestinal necrosis. Because its physiopathologic features differ from those of AMI, chronic mesenteric ischemia was excluded, and only acute small-bowel ischemia was included. Acute ischemic necrosis due to mesenteric trauma was excluded because of the different context and imaging features. AMI due to venous obstruction was excluded because of an insufficient number of patients.

Patient data were retrieved from medical records. Occlusive AMI was defined as occlusion of the celiac or superior mesenteric artery or both on CT scans. Nonocclusive AMI was defined as the finding of small-bowel necrosis at pathologic examination without vascular occlusion on CT scans.
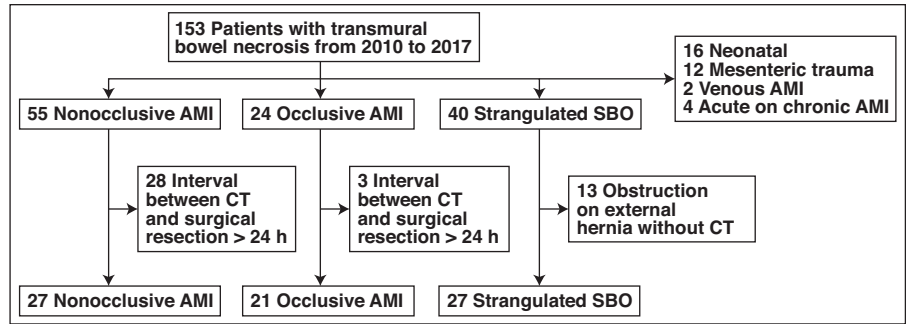

Fig. 1—Flowchart shows patient population. AMI = acute mesenteric ischemia, SBO = small-bowel obstruction.

The cause of AMI was collected retrospectively from clinical-biologic data and surgical results for both occlusive AMI (dissection, atheroma, and embolism) and nonocclusive AMI. Acute bowel necrosis caused by strangulation was confirmed at surgery. The final study included 77 patients (Fig. 1).

CT Protocols
All patients underwent MDCT, but on various equipment, because some patients were referred to our center having already undergone CT (unenhanced and contrast-enhanced series; slice thickness, 1–3 mm). At our institution, CT examinations were performed with 64-MDCT scanners (Brilliance 64, Philips Healthcare; Somatom Definition AS 64, Siemens Healthcare). For AMI, the CT protocol was an unenhanced acquisition and multiphase contrast-enhanced acquisitions. Early arterial phase images were acquired with bolus tracking (ROI located in the aorta), and portal venous acquisition was started 80 seconds after the start of IV contrast administration. For SBO, unenhanced acquisition was performed first [13, 14], followed by a single portal phase. Contrast administration was IV injection of 1.5 mL/kg of nonionic contrast medium at 300–400 mg I/mL through a power injector at a rate of 3–4 mL/s.

Imaging Analysis
CT scans were retrospectively and independently reviewed by two abdominal radiologists (8 and 5 years of experience in abdominal imaging) at a PACS workstation. Readers were aware of the final diagnosis of transmural bowel necrosis but were blinded to the cause. They were also blinded to the patients' clinical, biologic, and surgical features. They reviewed the images using multiplanar reconstructions.

The readers were asked to analyze the bowel CT features that have been accepted in the literature as associated with transmural necrosis: increased attenuation of the wall of a distended small-bowel loop compared with the neighboring loops on the unenhanced images (present or absent); bowel wall enhancement (decreased or absent to normal, analyzed by compari-

son with the adjacent normal bowel loops); thinned (virtual) or thickened (if ≥ 3 mm) bowel wall; small-bowel dilatation, defined as diameter ≥ 25 mm (present or absent); pneumatosis intestinalis, defined as gas within the bowel wall (present or absent); mesenteric venous gas, defined as presence of gas in bowel drainage vein (present or absent); portal venous gas; peritoneal gas; mesenteric fat stranding (present or absent); and ascites (present or absent). For patients with strangulation, the bowel wall CT features were collected in analysis of the incarcerated small-bowel loop. The results of the comparison of CT features with respect to the different causes of transmural bowel necrosis were those of the reader more experienced in abdominal imaging (reader 1).

Statistical Analysis
Categoric data were expressed as number and percentage and compared by Fisher exact test. Continuous variables were expressed as median and interquartile range and compared by Wilcoxon test. Interreader agreement was analyzed with weighted kappa statistics (0.00–0.20, slight agreement; 0.21–0.40, fair; 0.41–0.60, moderate; 0.61–0.80, substantial; 0.81–1.00, almost perfect). All tests were two-sided, and $p < 0.05$ was considered to be significant. All analyses were performed with SPSS software (version 20.0, IBM SPSS).

Results
Population Characteristics
The characteristics of the 77 patients with transmural bowel necrosis are summarized in Table 1. Fifty patients had AMI, and 27 had strangulated SBO. Among the 21 patients with occlusive AMI, 12 had occlusion due to atherosclerosis, six occlusion due to embolism, and three occlusion due to superior mesenteric artery dissection. Among the 29 patients with nonocclusive AMI, the most common cause of nonocclusive AMI was sepsis, followed by cardiac surgery and decreased cardiac output. There were no significant differences in lactate ($p = 0.20$) or C-reactive protein ($p = 0.48$) lev-

els in patients with occlusive AMI compared with those with nonocclusive AMI. Among the patients with strangulated SBO, the main cause of SBO was adhesions, followed by external internal hernia. Plasma creatinine concentration was higher among patients with nonocclusive AMI than among patients with occlusive AMI.

CT Features

The CT features of small-bowel transmural necrosis according to cause are summarized in Table 2. Pneumatosis intestinalis was more frequent with nonocclusive AMI than with occlusive AMI and strangulated SBO ($p < 0.01$), as were SMV gas ($p < 0.01$) and portal venous gas ($p < 0.01$) (Fig. 2). There was a significantly higher frequency of pneumatosis intestinalis ($p = 0.03$) and portal venous gas ($p < 0.01$) in

the group with nonocclusive AMI than in the group with occlusive AMI (Fig. 3). Presence of a thinned wall was significantly more frequent in the AMI groups than in the strangulated SBO group ($p = 0.02$). Decreased or absent bowel wall enhancement was more frequent in the AMI groups than in the strangulated SBO group ($p = 0.02$). Mesenteric fat stranding was less frequent in occlusive AMI than in nonocclusive AMI and strangulated SBO ($p < 0.01$) (Fig. 4). Spontaneous hyperattenuation of the bowel wall, however, was significantly more frequent in the strangulated SBO group (Fig. 5) than in the AMI groups ($p < 0.01$).

Interobserver Agreement

Interobserver agreement on CT features is presented in Table 3. Regarding the bowel wall

analysis, interobserver agreement was good for wall enhancement ($\kappa = 0.76$) and bowel wall thickening ($\kappa = 0.71$) and poor for parietal thinning ($\kappa = 0.36$). Regarding the analysis of features related to pneumatosis intestinalis, interobserver agreement was excellent to perfect for pneumatosis intestinalis ($\kappa = 0.97$), SMV gas ($\kappa = 1.00$), and portal venous gas ($\kappa = 1.00$).

Discussion

To our knowledge, this is the first study to compare the CT features of transmural bowel necrosis according to its three main causes: occlusive AMI, nonocclusive AMI, and strangulated SBO. The analysis of CT features showed that the prevalence of gas features (pneumatosis intestinalis, SMV gas, and portal venous gas) varied markedly by cause. We found only two (7%) cases of pneumatosis intestinalis in the strangulated SBO group and no cases of SMV gas or portal venous gas, which is in line with previous reports [4, 9, 15]. Interestingly, there were differences in the presence of gas features according to cause of AMI; gas features were significantly more frequent in nonocclusive AMI than in occlusive AMI. This point has never been directly proven, to our knowledge, even if it is consistent with what is reported in the literature [16, 17].

TABLE 1: Characteristics of 77 Patients With Transmural Bowel Necrosis

Characteristic	Occlusive AMI (n = 21)	Nonocclusive AMI (n = 29)	Strangulated SBO (n = 27)	p
Age (y)[a]	68.9 (43–89)	69.9 (44–84)	71.9 (44–92)	0.47
Sex				
Male	12 (57)	18 (62)	13 (47)	0.51
Female	9 (43)	11 (38)	14 (53)	
Cause				
Atherosclerosis	12 (57)			
Embolism	6 (28)			
Dissection	3 (14)			
Sepsis		10 (34)		
Cardiac failure		4 (14)		
Cardiac surgery		8 (27)		
Abdominal surgery		2 (7)		
Hemorrhage		2 (7)		
Hypovolemia		1 (3)		
Respiratory insufficiency		1 (3)		
Takotsubo cardiomyopathy		1 (3)		
Adhesions			15 (55)	
External hernia			7 (27)	
Internal hernia			5 (18)	
Blood tests				
Serum lactate level (mmol/L)[a]	4.6 (1.0–7.7)	6.04 (1.4–19)		0.20
No. of patients with serum lactate level > 2 mmol/L	16 (76)	18 (86)[b]		0.53
C-reactive protein (mg/L)[a]	146 (18–375)	173 (92–320)		0.48
Creatinine (μmol/L)[a]	116 (43–275)	198 (40–600)		0.03

Note—Unless otherwise indicated, values are number of patients with percentage in parentheses. The percentages in the AMI columns do not total 100 owing to rounding. AMI = acute mesenteric ischemia, SBO = small-bowel obstruction.
[a]Mean with range in parentheses.
[b]Eight patients did not have a lactate level recorded immediately before CT.
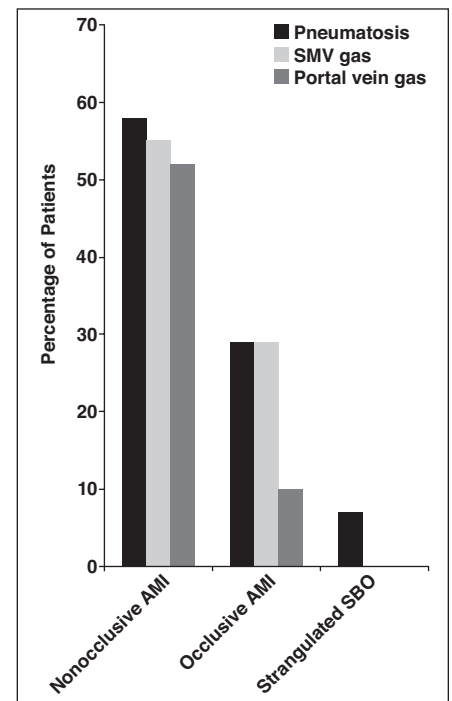

Fig. 2—Graph shows proportion of gas-related features of transmural bowel necrosis. SMV = superior mesenteric vein, AMI = acute mesenteric ischemia, SBO = small-bowel obstruction.

TABLE 2: CT Features of Transmural Bowel Necrosis by Cause

Feature	Occlusive AMI (n = 21)	Nonocclusive AMI (n = 29)	Strangulated SBO (n = 27)	p
Bowel wall				
Spontaneous hyperattenuation	2 (10)	4 (14)	11 (41)	< 0.01
Decreased or absent enhancement	17 (81)	24 (83)	15 (56)	0.06[a]
Thinned wall	10 (48)	15 (52)	5 (18)	0.02
Thickened wall	9 (43)	14 (48)	9 (33)	0.52
Bowel loop dilatation	17 (81)	25 (86)	26 (96)	0.26
Bowel gas				
Pneumatosis intestinalis	6 (29)	17 (59)	2 (7)	< 0.01
SMV gas	6 (29)	16 (55)	0 (0)	< 0.01
Portal venous gas	2 (10)	14 (48)	0 (0)	< 0.01
Extraluminal features				
Pneumoperitoneum	0 (0)	3 (10)	4 (15)	0.11
Mesenteric fat stranding	10 (48)	23 (79)	25 (93)	< 0.01
Ascites	10 (48)	23 (79)	20 (74)	0.06

Note—AMI = acute mesenteric ischemia, SBO = small-bowel obstruction, SMV = superior mesenteric vein.
[a]Value obtained by comparing AMI (occlusive and nonocclusive) with the strangulated SBO group.

Pneumatosis intestinalis has been extensively studied in terms of prognostic value in transmural bowel necrosis but less in terms of pathophysiologic features [18–20]. Pneumatosis intestinalis and portal venous gas are consequences of bowel wall damage and appear to be linked to the amount of blood flow in the ischemic bowel wall. In strangulated SBO, venous and arterial flow stops; in occlusive AMI only arterial flow stops; and in nonocclusive AMI, there is continuous venous and arterial flow. Therefore, during nonocclusive AMI the persistence of blood flow may allow transport of gas from the gut toward the liver. This may explain why the presence of gas is much more frequent in nonocclusive AMI and why ultrasound detection of portal venous gas has become an important factor in ICUs for identifying patients in whom AMI develops [21, 22]. There were no significant differences in lactate levels between the occlusive AMI group and the nonocclusive AMI group.

In contrast, spontaneous hyperattenuation of the bowel wall was significantly more frequent in the strangulated SBO group than in the AMI groups. This finding has been described in small-bowel ischemia associated with SBO [13, 23]. Spontaneous hyperattenuation of the bowel wall is thought to result from submucosal or transmural hemorrhage caused by mesenteric venous occlusion, as in strangulated SBO, but this radiologic feature can also be due to reperfusion after arterial AMI (with blood extravasation through damaged vessels). Decreased or absent bowel wall enhancement and presence of a thinned wall were less frequent in strangulated SBO than in AMI, which highlights that intestinal ischemia in strangulation is first of venous and then of arterial origin.

Mesenteric fat stranding was less frequent in occlusive AMI than in nonocclusive AMI and strangulated SBO. Mazzei et al. [24] specifically studied the diagnostic value of mesenteric features in AMI. Their findings were similar to ours, such as that mesenteric fat stranding can be the consequence of multiples factors. It is usually the consequence of elevation of mesenteric pressure, which explains why it is almost always observed in strangulated SBO, but it can also be a consequence of transmural infarction or be linked to a reperfusion syndrome.

Interobserver agreement on radiologic signs of AMI has been the topic of various studies because of the subjective and nonre-
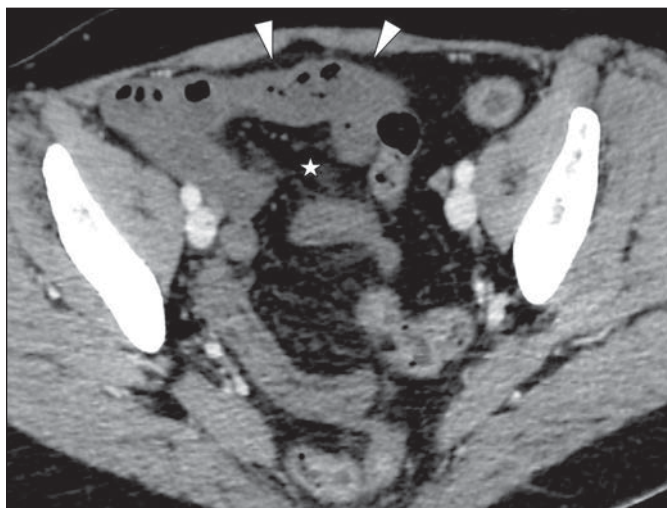

Fig. 3—67-year-old woman with transmural bowel necrosis due to occlusive acute mesenteric ischemia. CT image shows typical findings: absence of bowel wall enhancement (*arrowheads*) of dilated bowel loop and absence of mesenteric fat stranding and ascites (*star*).
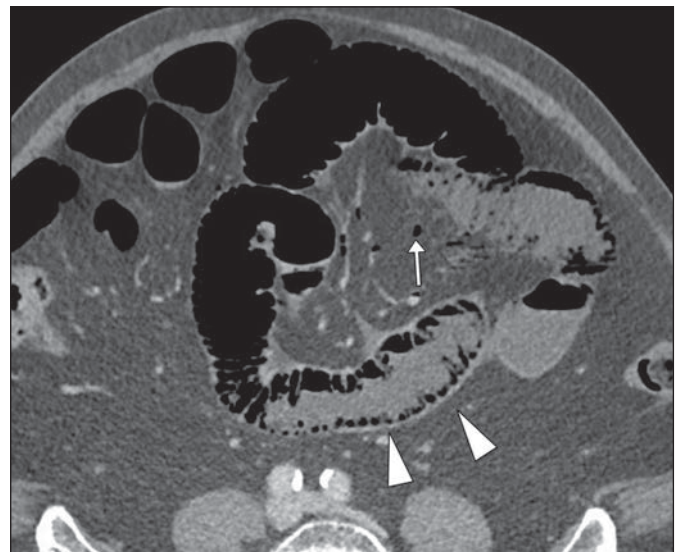

Fig. 4—57-year-old man with transmural bowel necrosis due to nonocclusive acute mesenteric ischemia. CT image shows typical findings: intestinal pneumatosis (*arrowheads*) and mesenteric vein gas (*arrow*). Bowel wall enhancement, thinned wall, and dilatation of bowel loop are absent.

producible nature of the CT features. CT has a crucial role, however, in the diagnosis of small-bowel necrosis. In our study, the greatest interobserver agreement was observed for the presence of bowel gas (κ = 0.97, 1.0, and 1.0), which may explain why this feature is so important in suspected bowel ischemia even if it is nonspecific [25]. Analysis of decreased or absent bowel wall enhancement showed substantial interobserver agreement (κ = 0.76), which was higher than in most of the literature, which reports kappa values ranging from 0.20 to 0.62 [17]. This can be explained by the fact that we evaluated only bowel resection specimens, which have a high prevalence of being nonenhancing. Kärkkäinen et al. [26] had already reported that interobserver agreement was higher among patients who underwent bowel resection. Additionally, all patients in our study underwent CT with both unenhanced and enhanced acquisitions, which published studies have shown improves interreader agreement [14, 17]. Regarding wall thinning, interobserver agreement was poor (κ = 0.36), which could be explained by the high rate of pneumatosis intestinalis, which impeded analysis of the wall thickness.

In our study, the prevalence of nonocclusive AMI compared with that of occlusive AMI was higher than that in the literature. The prevalence of nonocclusive AMI varies between 5% and 41% among all types of AMI [18, 27–31]. In our series, the proportion of nonocclusive AMI was greater than that of occlusive AMI, possibly because we evaluated only resected bowel, but it seems to reflect daily practice at our institution.

Nonocclusive AMI is a challenge for intensivists because its diagnosis is frequently delayed, its prognosis is extremely poor [28], and the presentation is not specific. Patients receiving sedation are frequently unable to describe digestive symptoms, and their condition can be confounded by hypovolemic or septic shock. Ultrasound performed in the ICU may help clinicians to better identify patients presenting with strong evidence of nonocclusive AMI, for whom abdominal CT with contrast injection is required. In a pragmatic approach, we found that patients presenting with nonocclusive AMI

and transmural bowel necrosis frequently had bowel loop dilatation (86%), ascites (79%), and portal venous gas (48%), all signs easily identified with US [21]. In addition, 93% (27/29) of the patients with nonocclusive AMI and transmural bowel necrosis had at least one of these three signs. Therefore, among critically ill patients with clinically and biologically suspected nonocclusive AMI, evidence of bowel loop dilatation, ascites, or portal venous gas at ultrasound should be followed by urgent abdominal CT and surgical evaluation. The absence of these three signs at US examination, however,

**TABLE 3: Interreader Agreement on CT Features of Transmural Bowel Necrosis (n = 77)**

Feature	Reader 1[a]	Reader 2[a]	κ[b]
Bowel wall			
Spontaneous hyperattenuation	17 (22)	9 (12)	0.59 (0.42, 0.72)
Decreased or absent enhancement	58 (75)	53 (69)	0.76 (0.65, 0.84)
Thinned wall	30 (39)	27 (35)	0.36 (0.46, 0.72)
Thickened wall	32 (42)	25 (32)	0.71 (0.58, 0.81)
Bowel loop dilatation	67 (87)	66 (86)	0.73 (0.60, 0.82)
Bowel gas			
Pneumatosis intestinalis	25 (32)	26 (34)	0.97 (0.95, 0.98)
Superior mesenteric vein gas	22 (29)	22 (29)	1.00 (1.00, 1.00)
Portal venous gas	16 (21)	16 (21)	1.00 (1.00, 1.00)
Extraluminal features			
Pneumoperitoneum	6 (8)	5 (6)	0.91 (0.86, 0.94)
Mesenteric fat stranding	59 (76)	61 (79)	0.50 (0.31, 0.65)
Ascites	52 (68)	51 (66)	0.64 (0.49, 0.76)

[a]Values are number with percentage in parentheses.
[b]Values in parentheses are 95% confidence limits.
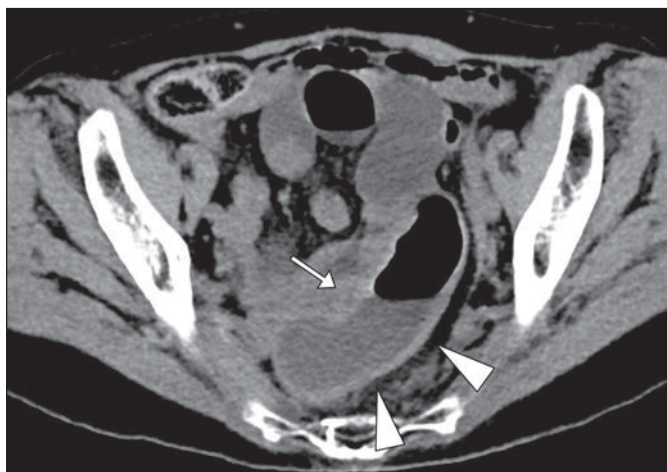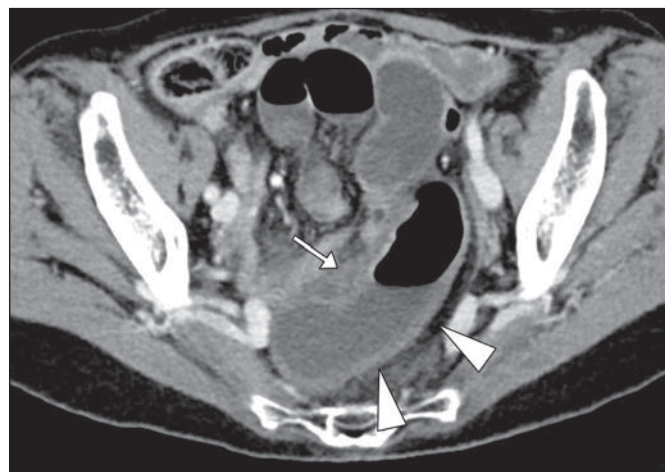
Fig. 5—85-year-old man with transmural bowel necrosis due to strangulated small-bowel obstruction. Examples of typical findings.
A, Unenhanced CT image shows hyperattenuation of incarcerated bowel-wall loop (*arrowheads*) and mesenteric fat stranding (*arrow*).
B, Contrast-enhanced CT image shows mesenteric fat stranding (*arrow*) and dilated bowel loop (*arrowheads*).

makes nonocclusive AMI and transmural bowel necrosis unlikely.

Besides the retrospective design, our study had limitations. First, the readers were blinded to the cause of bowel necrosis, but in most cases the cause was easily visualized on CT images. The differences observed in CT features with respect to cause could have been due to different degrees of necrosis, but we selected cases of small-bowel resection to avoid this bias and excluded all patients with a greater than 24-hour delay between CT and resection. In addition, lactate values were not different between patients with occlusive AMI and those with nonocclusive AMI. Finally, we were unable to study the CT features of transmural bowel necrosis in occlusive AMI of venous origin because of an insufficient number of cases.

Conclusion

In this study, we identified distinctive patterns of CT features according to cause of transmural bowel necrosis. Occlusive AMI is characterized by an absence of bowel wall enhancement and less mesenteric fat stranding, nonocclusive AMI by a high prevalence of pneumatosis intestinalis and portal venous gas, and strangulated SBO by spontaneous hyperattenuation of the bowel wall and an absence of pneumatosis intestinalis and portal venous gas.

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