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Small bowel transmural necrosis secondary to acute mesenteric ischemia and strangulated obstruction: CT findings of 49 patients

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ABSTRACT

Rationale and objectives: Transmural bowel necrosis (TBN) is an uncommon surgical emergency that represents an endpoint of occlusive acute mesenteric ischemia (AMI), nonocclusive AMI and small bowel obstruction (SBO). According to limited evidence, each etiology of TBN might demonstrate a different CT finding. This investigation aimed to 1) identify overall CT findings of TBN, and 2) compare CT findings of TBN in each etiology.

Materials and methods: Forty-nine consecutive adults (mean age, 64.6 years; 26 men) with occlusive AMI, nonocclusive AMI or SBO, and pathologically proven TBN were enrolled. All had a CT scan within 24 h before surgery. Clinical information was compiled from medical records. CT examinations were re-reviewed by two radiologists with disagreements resolved by the third radiologist. Data were analyzed and compared.

Results: Transmural bowel necrosis were secondary to arterial AMI, venous AMI, combined arterial and venous AMI, nonocclusive AMI, and SBO in 6, 5, 2, 10, and 26 patients, respectively. The CT findings were ascites (93.9%), abnormal wall enhancement (91.8%), bowel dilatation (89.8%), mesenteric fat stranding (89.8%), abnormal wall thickness (71.5%), pneumatosis (46.9%) and intrinsic hyperattenuation of bowel walls (22.5%). Portovenous gas, mesenteric venous gas, and pneumoperitoneum were present in 4 patients (8.2%). Bowel wall thickness was the only CT findings that showed a statistically significant difference among the 5 etiologies of TBN ($P = 0.046$).

Conclusions: Most common CT findings of TBN were ascites, abnormal bowel wall enhancement, dilatation, and mesenteric fat stranding. Wall thickness differentiated five etiologies, being most thickened in venous AMI and normal in arterial AMI.

1. Introduction

Transmural bowel necrosis (TBN) represents a late stage of ischemic changes in the bowel. TBN primarily originates from disease processes that reduce blood flow to the gastrointestinal tract, leading to cellular death and ongoing necrosis of the bowel wall layers [1]. Such diseases include occlusive acute mesenteric ischemia (AMI), which can be arterial, venous or combined arterial and venous in

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Abbreviations

AMI	acute mesenteric ischemia
CT	computed tomography
HU	Hounsfield unit
P	probability
PACS	Picture Archiving and Communication System
PV	portal vein
SBO	small bowel obstruction
SD	standard deviation
SMV	superior mesenteric vein
TBN	transmural bowel necrosis

origins, nonocclusive AMI, and strangulated small bowel obstruction (SBO). Suspicion of TBN is crucial to alerting physicians caring for patients with occlusive AMI, nonocclusive AMi or SBO to order an immediate and extensive workup and to plan for potential surgical intervention [2]. The presence of TBN with occlusive AMI, nonocclusive AMI, and SBO impacts the following: the decision to perform surgery, the type of treatment (catheter-directed therapy vs surgical management), the extent of surgical procedures (without vs with bowel resection), surgical subspecialty handling (vascular vs acute care surgeons), and most importantly, the patient's prognosis [2,3]. If TBN diagnosis and treatment are delayed, mortality has been reported in up to 50% of cases [1].

Being widely available and easy to perform, contrast-enhanced computed tomography (CT) is the mainstay imaging modality for patients suspected of having occlusive AMI, nonocclusive AMI, or SBO. CT has demonstrated high accuracy in diagnosing AMI [2,4] and strangulated SBO [5,6]. However, CT's ability to detect TBN as a complication of AMI and strangulated SBO is questionable, as many CT findings are nonspecific and overlap between reversible ischemic change and necrosis [1,7–10]. A lack of bowel wall thickening [1], absent enhancement or hypoenhancement, intrinsic hyperattenuation of the bowel walls [11], and pneumatosis intestinalis [12] have been suggested as TBN signs, although they can be seen in reversible ischemia.

Calame P et al. [8] and Zeng Y et al. [13] recently reported that TBN's appearances might depend on its etiology (i.e., the same CT sign might have a different meaning when present in a different disease process). However, knowledge on this issue is lacking because TBN is uncommon [2,11] and little research has examined this issue [8,9,11,14–18]. Treatment options for TBN have evolved to include multiple subspecialists performing potentially complex procedures. Consequently, determining TBN involvement at occlusive AMI, nonocclusive AMI or SBO diagnosis (i.e., not in the operating room) is essential to develop an appropriate surgical plan and avoid management delay.

This investigation aimed to 1) describe the CT findings of TBN and 2) compare the clinical characteristics and CT features of patients with TBN secondary to arterial, venous, combined arterial and venous occlusive AMI, nonocclusive AMI, and strangulated SBO.

2. Materials and methods

2.1. Study design and patients

This retrospective descriptive investigation was performed at a quaternary-care, academic medical center with a 2300-bed capacity. The Institutional Review Board approved the study (approval number Si435/2021) and waived the requirement for informed

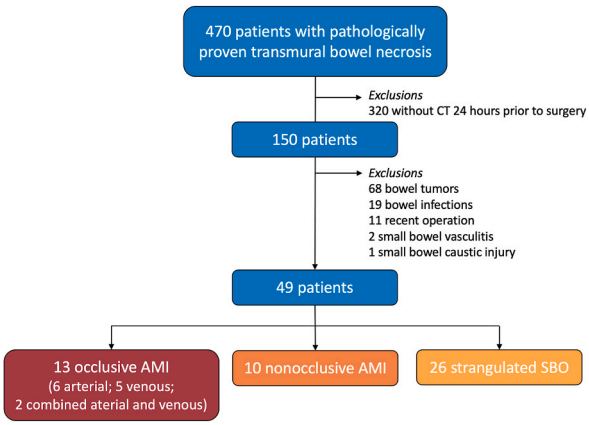

Fig. 1. Flowchart of patient inclusion.

consent due to the minimal risks involved. From January 2010 to June 2021, 470 consecutive adult patients (aged ≥18 years) underwent small bowel resection with a pathological diagnosis of TBN. Patients were excluded if a contrast-enhanced CT was not performed within 24 h preoperatively (n = 320) or they had causative diagnoses other than AMI or SBO (e.g., trauma, enteritis, vasculitis, or malignancy; n = 101). Forty-nine patients were enrolled, as illustrated in Fig. 1.

2.2. Clinical data

Clinical data were collected from electronic medical records (surgical pathology reports and final diagnosis). "Occlusive AMI" encompassed arterial (embolic and thrombotic in origin) and venous obstruction of mesenteric vessels, while "nonocclusive AMI" was indicated by impaired regional perfusion of the bowel from various etiologies (eg, cardiac insufficiency or low-flow states). "Strangulated SBO" was defined as SBO with impeded vascular supply resulting in bowel ischemia or necrosis [3,4,19]. Patient sex, age, clinical history, duration of symptoms, date of presentation, date of small bowel resection, resection site and findings, last follow-up, laboratory results (hemoglobin, white blood cell counts, creatinine, amylase, lactate, and arterial blood pH), site(s) of TBN, and clinical disease course were collated. Dates, time points, and CT examination details were sourced from the hospital's radiological information system.

2.3. Image acquisition

The CT examinations were performed on one of the hospital's 4 multidetector CT scanners: two 64-slice scanners (LightSpeed VCT, GE Healthcare and Discovery CT750 HD, GE Healthcare) and two 256-slice scanners (Revolution CT, GE Healthcare). Scan parameters were 120 kVp and 300 mAs for the 64-slice multidetector CT systems and 250 mAs for the 256-slice multidetector CT systems. The scans used intravenously (IV) administered nonionic iodinated contrast media (320–370 mgI/mL; 1.5–2.0 mL/kg) at 3–4 mL/s via injectors. No oral or rectal contrast media were administered. The CT studies were obtained in an unenhanced phase followed by a portovenous phase of approximately 80 s with the administration of an IV iodinated contrast medium. Four scans had an arterial phase obtained approximately 40 s after contrast administration. An axial-slice thickness of 1.25 mm was acquired from the hepatic dome to the pubic symphysis. Images were sent to a Picture Archiving and Communication System (PACS; Synapse, FujiFilm Corporation).

2.4. CT definitions, reinterpretation, and reference standards

Various CT features were collected from a reinterpretation of the CT images in the PACS. If the patients had more than one CT scans prior to bowel resection, only the one closest to the operation was evaluated. Intrinsic hyperattenuation of bowel walls was assessed on an unenhanced phase as a thickened bowel wall with a CT attenuation higher than normal adjacent loops [9]. "Small bowel wall thickening" was defined as ≥ 2 mm of wall thickness for well-distended loops and ≥3 mm of nondistended loop, while "thinning" was defined as paper-thin or barely visible walls in lung windows [20]. Abnormal bowel wall enhancement could manifest as a decrease or increase in the enhancement of the affected bowel walls, relative to the normal-looking bowel in other areas. This CT finding was believed to reflect perfusion status of the bowel [21]. "Small bowel dilatation" was defined as a loop having an outer transverse diameter ≥2.5 cm [22]. Ascites were considered moderate or large when fluid was present in paracolic gutters or was sufficient to displace bowel loops [23]. Ascitic fluid was identified in the axial plane and the measurements of the CT number were made at the largest fluid pocket that the radiologist-reviewer deemed measurable with a caliper area of at least 1 cm^2 while maintain a distance of at least half of the diameter of the region of interest relative to the surrounding tissues [24].

The portovenous-phase CTs were independently reinterpreted by 2 radiologists with 16 and 17 years of experience and subspecialized in abdominal or emergency radiology, respectively. The arterial and delayed phases were not reviewed. Although the radiologists were aware of the TBN diagnoses and the involved small-bowel segments, they were blinded to the clinical, laboratory, and surgical data and TBN etiologies. Discrepancies were resolved by a third abdominal radiologist with 22 years of experience, which served as a reference standard for CT findings in this investigation. All patients underwent small bowel resection with a pathological diagnosis of TBN.

2.5. Statistical analysis

Descriptive statistics were used for qualitative and quantitative data. Categorical variables (eg, sex, TBN sites, and discharge status) and most CT findings are reported as numbers or percentages. Continuous data (eg, age, duration, and laboratory results) are presented as the mean (standard deviation) for data with normal distribution and the median (interquartile range) for nonnormally distributed data. Depending on their distribution, categorical data were compared using a chi-squared test and continuous variables with 1-way ANOVA or the Kruskal–Wallis test. *One-way ANOVA followed by Bonferroni's* post-hoc pairwise comparisons tests or Kruskal-Wallis test followed by Dunn *Bonferroni's* post-hoc pairwise comparison was used to compare continuous variables among groups. PASW Statistics for Windows, version 18.0 was used for the analyses. Probability (*P*) values less than or equal to 0.05 were considered statistically significant.

3. Results

3.1. Patients

Forty-nine patients (mean age, 64.6 years; 26 men) met the inclusion criteria. Thirteen had occlusive AMI (6 arterial, 5 venous, 2 combined arterial and venous), 10 had nonocclusive AMI, and 26 had strangulated SBO. Most patients presented within 2 days of symptom onset, and most CT scans were performed within 8 h of presentation. The median white blood cell (14×10^9 cells/L; range 1–12) and serum lactate (4.91 mmol/L; range 1.1–15.3) levels were elevated, and the median pH was low (7.33; range 6.9–7.5). Pathological specimens revealed the ileum as the most frequent TBN site (25/49; 51%), followed by the jejunum (18/49; 36.7%), jejunum and ileum (5/49; 10.2%), and duodenum (1/49; 2%). Out of the total number of patients, ten died during the same admission as the diagnosis. The remaining 39 patients had a median follow-up duration of 55 days. Table 1 details their clinical characteristics and differences according to their TBN etiologies.

3.2. CT features

The most common CT finding of TBN was ascites (46/49; 93.9%). The ascites was moderate or large in 22 patients (22/46; 47.8%), with an average attenuation of 16.9 ± 1.9 HU. Abnormal bowel wall enhancement was present in 45 patients (45/49; 91.8%). Of these, 44 cases had decreased enhancement, and 1 had increased enhancement. Bowel dilatation ($\geq$2.5 cm) and mesenteric fat stranding were observed in 44 patients each (44/49; 89.8%). Thirty-five patients had abnormal bowel wall thickness (35/49; 71.5%), of which 26 had wall thickening and 9 had wall thinning. Pneumatosis was present in 23 patients (23/49; 46.9%). Seven patients had

Table 1
Clinical characteristics of all patients and comparison among different etiologies of transmural bowel necrosis.

	All patients (n = 49)	Arterial AMI (n = 6)	Venous AMI (n = 5)	Combined arterial and venous AMI (n = 2)	Nonocclusive AMI (n = 10)	Strangulated no bowel obstruction (n = 26)	P values
Male sex (n, %)	26 (53.1)	3 (50)	3 (60)	1 (50)	8 (80)	11 (42.3)	0.374
Mean age (years; SD)	64.6 (18.9)	72.2 (12.3)	59.4 (10.7)	75.5 (5.0)	67.0 (18.7)	62.1 (21.8)	0.642
Median durations (range)							
Onset to presentation (days)	2 [1,14]	1.5 [1,3]	5 [1,14]	3 [1,5]	1 [1,2]	2 [1,7]	0.026
Presentation to computed tomography (hours)	8 [1,22]	7 [3,14]	4 [1,17]	13.5 [7,20]	11 [1,22]	7.5 [1,27]	0.607
Computed tomography to bowel resection (hours)	6 [1,24]	8.5 [5,14]	20 [3,24]	9.5 [7,12]	5 [1,13]	4 [1,18]	0.032[a]
Laboratory results							
Hemoglobin (g/dL; mean, SD) (normal; 12–18 g/dL)	12.3 (2.7)	10.5 (3.5)	14.3 (1.3)	13.5 (1.1)	10.5 (2.4)	12.85 (2.5)	0.018[a]
White blood cell count ($\times 10^9$ cells/L; median, range) (normal; 4-11 $\times 10^9$ cells/L)	14 [1,12]	10.9 (1.8, 39.8)	18.5 (2.9, 37.4)	10.0 (5.6, 14.5)	11.8 (5.7, 22.0)	13.5 (3.6, 26.2)	0.708
Creatinine (mg/dL; median, range) (normal; 0.5–1.5 mg/dL)	1.2 (0.4,7.5)	1.5 (0.7, 4.0)	1.3 (0.8, 3.3)	1.4 (1.0, 1.7)	2.4 (1.2, 7.5)	0.9 (0.4, 9.6)	0.005[b]
Amylase (U/L; median, range) (n = 20) (normal; 28–100 U/L)	109 (36, 1621)	–	53.5 (45, 62)	–	330.5 (50, 692)	105 (36, 1621)	0.222
Lactate (mmol/L; median, range) (n = 34) (normal; 0.5–2.2 mmol/L)	4.91 (1.1, 15.3)	5.4 (2.9, 8.7)	2.4 (1.2, 3.0)	2.6 (1.6, 3.5)	4.2 (1.4, 15.3)	3.2 (1.1, 10.6)	0.232
pH (median, range) (n = 32) (normal; 7.35–7.45)	7.33 (6.9, 7.5)	7.4 (7.3, 7.5)	7.4 (7.3, 7.5)	7.3 (7.2, 7.4)	7.3 (6.9, 7.5)	7.3 (7.2, 7.5)	0.700
Site of TBN							0.477
Duodenum	1 [2]	0	0	0	0	1 (100)	
Jejunum	18 (36.7)	2 (33.3)	4 (80)	2 (100)	2 (11.1)	8 (44.4)	
Jejunum and ileum	5 (10.2)	0	0	0	1 [20]	4 (80)	
Ileum	25 (51)	4 (66.7)	1 [20]	0	7 [28]	13 (52)	
Outcome							
Dead (n, %)	10 (20.4)	4 (66.7)	0	0	6 (60)	0	<0.0001
Duration from onset to last follow up (days; median, range) (n = 39)	55 (10, 338)	53 (47, 59)	119 (15, 338)	86.5 (79, 94)	90.5 (29, 111)	43 (10, 424)	0.736

AMI = acute mesenteric ischemia; SD = standard deviation.
Values in brackets represent percentage unless specified otherwise.
Pairwise comparison identified a single pair of significant difference (NOMI vs. venous AMI; adjusted p value = 0.021).
[a] Pairwise comparison did not identify significant differences between groups.
[b] Pairwise comparison identified a single pair of significant difference (NOMI vs. strangulated obstruction; adjusted p value = 0.002).

portovenous gas, mesenteric venous gas, or pneumoperitoneum (each finding in 4 patients: 4/49; 8.2%). Of these, one had all 3 findings, two had 2 out of 3 findings (mesenteric and portovenous gas) and the rest had 1 out of 3 findings. In the unenhanced phase, 11 patients had intrinsic hyperattenuation of the bowel walls (11/49; 22.5%). Table 2 aggregates the CT findings of TBN.

For patients with occlusive AMI, Table 3 details their CT findings and Fig. 2 aggregates findings according to the subcategories of arterial (n = 6), venous (n = 5), and combined arterial and venous (n = 2) occlusions.

Three patients, including one case of non-occlusive mesenteric ischemia and two cases of arterial acute mesenteric ischemia, underwent more than one CT scan before undergoing bowel resection surgery. The CT scans were performed between 2 and 13 days prior to the index CT. Out of the total, two scans showed normal-appearing small bowel loops, while the other scan showed slight hyperenhancement of small bowel walls.

3.3. Comparison of TBN by etiology

A comparison was made of the clinical characteristics and CT features of the patients classified into 5 groups based on their TBN etiologies. Statistically significant differences were found for the onset-to-presentation duration between venous AMI and NOMI (longer in the former), serum creatinine levels between NOMI and strangulated SBO (higher in the former), and death at admission (higher rates in arterial AMI and NOMI than others). Sex, age, other laboratory results, and TBN sites did not differ among the 5 groups.

Among the five etiologies of TBN, bowel wall thickness was the only CT finding that exhibited a statistically significant difference (P = 0.046). Wall thickening was observed most frequently in cases of venous AMI (5/5; 100%), and less commonly in nonocclusive AMI (6/10; 60%), strangulated SBO (13/26; 50%), and arterial AMI (2/6; 33.3%). Normal wall thickness was identified in 4 out of 6 patients with arterial AMI (66.7%) and all 2 cases of combined arterial and venous AMI, but not in venous AMI and in only 20.0–23.1% of nonocclusive AMI and strangulated SBO cases. This suggests that bowel wall thickness is a useful diagnostic criterion in differentiating among the various etiologies of TBN.

4. Discussion

4.1. Overall CT findings

This investigation demonstrated various CT findings of TBN and identified a few features that tended to be found in selected TBN etiologies. Although the differences in the CT features of TBN for the 5 etiologies did not reach statistical significance, our study still extends the currently limited knowledge on this topic. Few TBN investigations and a meta-analysis have been performed, and most were conducted from 2015 onward (Table 4) [8,11,13,15–17,25–30]. This increase in research might reflect an advance in the

Table 2
Computed tomographic features of all patients and comparison among different etiologies of transmural bowel necrosis.

	All patients (n = 49)	Arterial AMI (n = 6)	Venous AMI (n = 5)	Combined arterial and venous AMI (n = 2)	Nonocclusive AMI (n = 10)	Strangulated obstruction (n = 26)	P values
Intrinsic high attenuation of bowel walls on unenhanced CT (n, %)	11 (22.4)	1 (16.7)	2 (40)	0	1 [10]	7 (26.9)	0.597
Enhancement (n, %)							0.900
Decreased	44 (89.8)	5 (83.3)	4 (80)	2 (100)	10 (100)	23 (88.5)	
Normal	4 (8.2)	1 (16.7)	1 [20]	0	0	2 (7.7)	
Increased	1 [2]	0	0	0	0	1 (3.8)	
Wall thickness (n, %)							0.046
Thinning	9 (18.4)	0	0	0	2 [20]	7 (26.9)	
Normal	14 (28.6)	4 (66.7)	0	2 (100)	2 [20]	6 (23.1)	
Thickening	26 (53.1)	2 (33.3)	5 (100)	0	6 (60)	13 (50)	
Bowel dilatation (n, %)	44 (89.8)	6 (100)	4 (80)	1 (50)	9 (90)	24 (92.3)	0.304
Bowel diameter (cm; mean, SD) (n = 44)	3.4 (0.7)	3.2 (0.6)	3.3 (0.2)	–	3.2 (0.4)	3.7 (0.7)	0.223
Pneumatosis (n, %)	23 (46.9)	3 (50)	2 (40)	0	7 (70)	11 (42.3)	0.374
Superior mesenteric venous gas (n, %)	4 (8.2)	1 (16.7)	0	0	1 [10]	2 (7.7)	0.869
Portal venous gas (n, %)	4 (8.2)	1 (16.7)	0	0	2 [20]	1 (3.8)	0.446
Pneumoperitoneum (n, %)	4 (8.2)	1 (16.7)	0	0	0	3 (11.5)	0.647
Mesenteric fat stranding (n, %)	44 (89.8)	5 (83.3)	5 (100)	1 (50)	9 (90)	24 (92.3)	0.345
Ascites (n, %)	46 (93.9)	5 (83.3)	5(100)	1 (50)	10 (100)	25 (96.2)	0.059
Moderate/large amount of ascites (n, %) (n = 46)	22 (47.8)	3 (60)	1 [20]	0	3 [30]	15 (60)	0.238
Attenuation of ascites (Hounsfield Units; median, range) (n = 46)	15 (4, 47)	18 [4,20]	12 [7,15]	–	15 (5, 47)	15 [5,29]	0.345

AMI = acute mesenteric ischemia, SD = standard deviation.
Values in brackets represent percentages unless specified otherwise.

Table 3
Details of computed tomographic features in patients with occlusive acute mesenteric ischemia.

Characteristics Etiology	Case No.	Jejunal involvement	Intrinsic high attenuation of bowel walls	Decreased enhancement	Wall thickening	Dilatation	Superior mesenteric or portal venous gas	Pneumoperitoneum	Mesenteric fat stranding	Moderate/large ascites
Arterial	23	no	yes	yes	no	yes	no	no	yes	yes
	26	no	no	yes	no	yes	no	no	yes	yes
	27	yes	no	no	no	yes	no	no	no	absent
	28	yes	no	yes	yes	yes	yes	no	yes	no
	33	no	no	yes	no	yes	yes	yes	yes	yes
	34	no	no	yes	yes	yes	no	no	yes	no
Venous	7	yes	no	no	yes	yes	no	no	yes	no
	16	yes	yes	yes	yes	yes	no	no	yes	no
	40	no	no	yes	yes	yes	yes	no	yes	no
	43	yes	no	yes	yes	yes	yes	no	yes	yes
	48	yes	yes	yes	yes	no	no	no	yes	no
Arterial and venous	2	Yes	no	yes	no	yes	no	no	no	absent
	39	yes	no	yes	no	no	no	no	yes	no

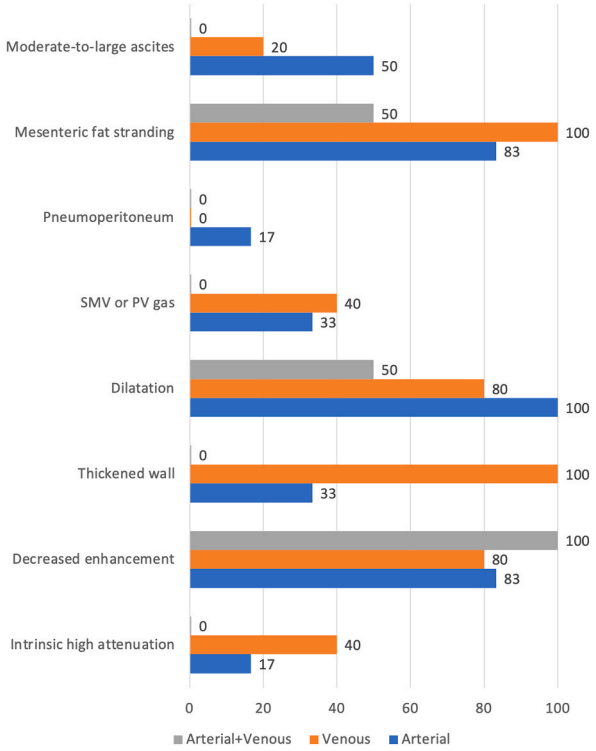

Fig. 2. CT findings among 13 patients with occlusive acute mesenteric ischemia (6 arterial, 5 venous and 2 combined arterial with venous). X-axis and values to the right of each bar represent percentages of each finding relative to the number of patients in that etiology.

knowledge of the treatment options for patients with bowel ischemia. With endovascular therapy becoming a viable option, the importance of differentiating between irreversible ischemia (TBN) and reversible ischemia has recently been emphasized [3].

Our investigation identified that the frequent CT features of TBN were ascites, an absent or decreased bowel wall enhancement, bowel dilatation, mesenteric fat stranding, and abnormal bowel wall thickness. These features are consistent with those described in other studies. Calame P et al. [8] found that small bowel dilatation, mesenteric fat stranding, decreased/absent bowel wall enhancement, and ascites were the most frequent findings in TBN when all etiologies (AMI and SBO) were combined. Atre ID et al. [16] recently reported that the most frequent CT findings of 26 pathologically proven TBN patients (21 from arterial AMI) were mesenteric fat stranding and absent/decreased bowel wall enhancement. Abnormal bowel wall enhancement in TBN was often depicted as decreased or absent, representing a direct sign of vascular impairment of the small bowel. This feature was also considered the most reliable CT sign of bowel ischemia irrespective of viability [31]. Nakashima K et al. [11] found the sign predictive of TBN, but with only a modest sensitivity of 67%.

Although nonspecific, mesenteric fat stranding, fluid and ascites were prevalent in TBN of any etiology, with prevalence ranging from 47.6% to 96.2% [8,11,16,17,25–27,29,30]. These conditions result not only from the elevated mesenteric pressure typical of strangulated SBO but also from transmural infarction or even a reperfusion state [8]. Fat stranding, fluid, and ascites in the presence of SBO often suggest a complication with relatively modest sensitivity (58%–88%) and specificity (76%–90%) [32], while those that occur in AMI suggest a higher disease severity (ie, potential TBN). However, these 2 findings were not reported by Emile SH et al. [17] or Milone M et al. [28], who identified bowel dilation and wall thickening as their most frequent CT findings of TBN, respectively. Differences in the frequency of each CT finding might stem from variations in patient inclusion criteria, proportions of each TBN etiology in study cohorts, CT definitions used, and reviewing physicians' judgments.

4.2. Differentiating CT features of TBN according to etiologies

Bowel wall thickness was the only CT finding that showed a statistically significant difference among the five etiologies of TBN. Specifically, wall thickening was observed more frequently in cases of TBN secondary to venous AMI than in those caused by other etiologies. Conversely, normal wall thickness was more commonly identified in cases of TBN secondary to arterial and combined arterial and venous AMI. This observation may be explained by the fact that in mesenteric venous occlusion, wall thickening is generally more pronounced than in cases of purely arterial AMI due to intramural edema and hemorrhage resulting from venous congestion [1].

Additionally, the following observations were made: 1) intrinsic hyperattenuation of the bowel walls on unenhanced CT was seen in cases of strangulated SBO (Fig. 3A–D), 2) strangulated SBO cases exhibited larger bowel diameter and a moderate to large amount of

Table 4
Computed tomographic appearances of transmural bowel necrosis of the small bowel in previous literature.

No.	First author (years)	No. of patients	Bowel abnormalities						Mesenteric abnormalities		Abnormal gas		
			Absent/decreased enhancement	Increased enhancement	Wall thickening	Wall thinning	Intrinsic high attenuation	Dilatation	Fat stranding or fluid	Ascites	Pneumatosis	SMV-PV gas	Pneumoperitoneum
1	Calame (2020)	21	17 (80.95)	NR	9 (42.9)	10 (47.6)	2 (9.5)	17 (80.9)	10 (47.6)	10 (47.6)	6 (28.6)	6 (28.6)	0
2	Wang (2019)	58	42 (72.4)	NR	34 (58.6)	18 (31.0)	NR	40 (68.9)	NR	50 (86.2)	16 (27.6)	6 (10.3)	NR
3	Verdot (2021)	74	53 (71.6)	11 (14.9)	17 (22.9)	24 (32.4)	2 (2.7)	56 (75.7)	NR	NR	27 (36.5)	20 (27.0)	NR
4	Calame (2020)	29	24 (83.8)	NR	14 (48.3)	15 (51.7)	4 (13.8)	25 (86.2)	23 (79.3)	23 (79.3)	17 (58.6)	16 (55.2)	3 (10.3)
5	Muratsu (2020)	18	NR	NR	NR	NR	NR	NR	NR	15 (83.3)	17 (94.4)	12 (66.7)	1 (5.6)
6	Perez-Garcia (2017)	47	33 (70.2)	3 (6.4)	30 (63.8)	NR	NR	33 (70.2)	NA	29 (61.7)	41 (87.2)	23 (48.9)	10 (21.3)
7	Calame (2020)	27	15 (55.6)	NR	9 (33.3)	5 (18.5)	11 (40.7)	26 (96.3)	25 (92.6)	20 (74.1)	2 (7.4)	0	4 (14.8)
8	Rondenet (2018)	25	9 (36)	NR	16 (64)	NR	14 (56)	23 (92)	25 (100)	24 (96)	1 (4)*	*	2 [8]
9	Nakashima (2015)	16	13 (81.3)	NR	7 (43.8)	NR	5 (31.3)	NR	15 (93.8)	8 (50)	4 (25)**	NR	**
10	Xu (2022)	45	NR	NR	36 (80)	NR	NR	NR	43 (95.6)	40 (88.9)	NR	NR	NR
11	Atre (2022)	26	23 (88.5)	3 (11.5)	11 (42.3)	14 (53.9)	NR	17 (65.4)	25 (96.2)	19 (73.1)	17 (65.4)	11 (42.3)	3 (11.5)
12	Calame (2020)	77											
13	Emile (2018)	73	NR	NR	34 (46.6)	NR	NR	45 (61.6)	NR	39 (53.4)	11 (15.1)	NR	NR
14	Milone (2013)	121	NR	NR	94 (77.7)	NR	NR	62 (51.2)	NR	NR	40 (33.1)	NR	NR

Number on the first column: 1 = study on arterial AMI; 2 = study on venous AMI; 3–6 = studies on nonocclusive AMI; 7–10 = studies on strangulated SBO; 11–14 = studies on a mixture of these etiologies. For the studies #6 and #11, values represent TBN of small and large bowel. Studies #1, #4, #7 and #12 are the same investigations but the information was broken down into etiologies for the purpose of this table.
NR = not reported, PV = portal vein, SMV = superior mesenteric vein.
Values in bracket represent percentages.
*Pneumatosis or SMV-PV gas.
**Pneumatosis or pneumoperitoneum.

ascites, and 3) nonocclusive AMI cases had a higher proportion of abnormal gas, including pneumatosis intestinalis and portovenous gas (Fig. 4A–D). Although these observations did not reach statistical significance ($P = 0.304$ to 0.597), they provided valuable insights into the CT features of TBN.

4.3. Intrinsic hyperattenuation of bowel walls on unenhanced CT in strangulated SBO

Our investigation found intrinsic hyperattenuation of the bowel walls on unenhanced CT scans in 40% of patients with venous occlusive AMI, and 26.9% with strangulated SBO. These proportions were much higher than that for patients with arterial AMI (16.7%), nonocclusive AMI (10%), and combined arterial and venous AMI (0%). Although the differences did not reach statistical significance in our population, other studies reported similar findings. The reported prevalence of intrinsic hyperattenuation of the bowel walls among patients with strangulated SBO complicated by TBN has ranged between 31.3% and 56% [8,11,27]. Additionally, Calame P et al. [8] found this condition significantly more frequently in TBN secondary to strangulated SBO than in other etiologies. Intrinsic bowel wall hyperattenuation is a classic feature of this condition, irrespective of bowel viability [9]. This finding might be explained by the intramural hemorrhage and hemorrhagic infarction that occur in TBN [1], although the condition can be seen in anticoagulation or after irradiation [9,11]. Another limitation in the use of this sign is the subjectivity of its evaluation (i.e., limited agreement among radiologists) [9,11].

4.4. Larger bowel diameter and moderate/large ascites in strangulated SBO

In our patient cohort, the mean bowel diameter was larger and the proportion of moderate/large amounts of ascites was higher among patients with TBN secondary to strangulated SBO. These findings are consistent with earlier studies showing a prevalence of ascites ranging from 50% to 96% [8,11,27]. In addition, ascites was found to be an independent predictor of strangulated SBO in the study of Xu W et al. [30]. Bowel dilatation is expected in patients with SBO, especially when it is complicated by strangulation. It possibly arises from a lost contractability with TBN, resulting in adynamic ileus, while the viable segments may have spastic contraction [33]. Although ascites is a common CT feature of complicated SBO, its presence does not allow differentiation between TBN and reversible ischemia [1,32]. In contrast, the absence of ascites has been suggested as a solid sign to exclude strangulation [5, 34].

4.5. Abnormal gas in nonocclusive AMI

Pneumatosis intestinalis occurred in a much higher proportion of our TBN cases with nonocclusive AMI (70%) than in other etiologies (38.5% and 42.3%). This result aligns with previous investigations on nonocclusive AMI complicated by TBN. They reported a prevalence of pneumatosis intestinalis ranging between 36.5% and 94.4%, SMV-PV gas between 27% and 66.7%, and

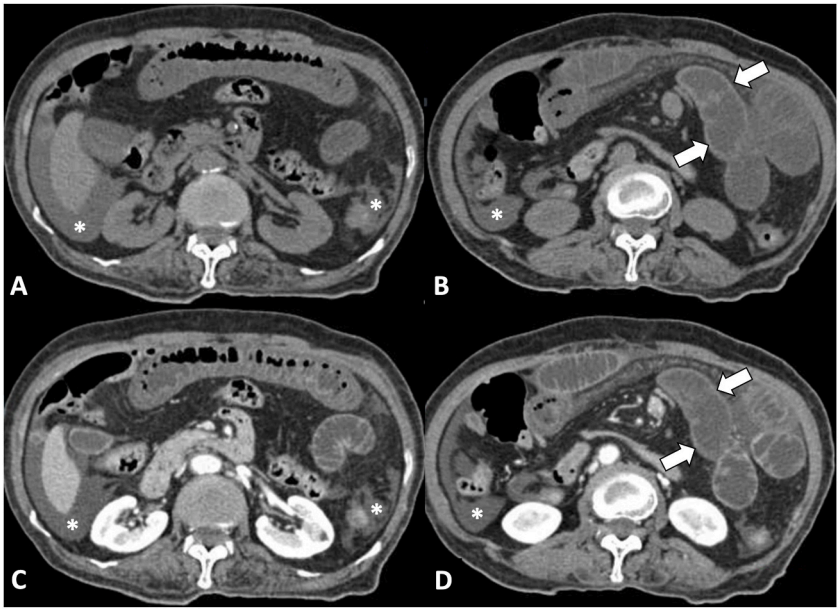

Fig. 3. Intrinsic hyperattenuation of bowel walls, absent wall enhancement, and ascites in a 27-year-old woman with transmural bowel necrosis secondary to strangulated small bowel obstruction. Axial unenhanced CT images (**A and B**) show dilatation of jejunal loops disproportionate to the ileum, with thickened walls and high attenuation of one of the jejunal walls (arrows), mesenteric fat stranding and ascites (asterisks). On portovenous-phase images (**C and D**), absent enhancement of this loop is observed.

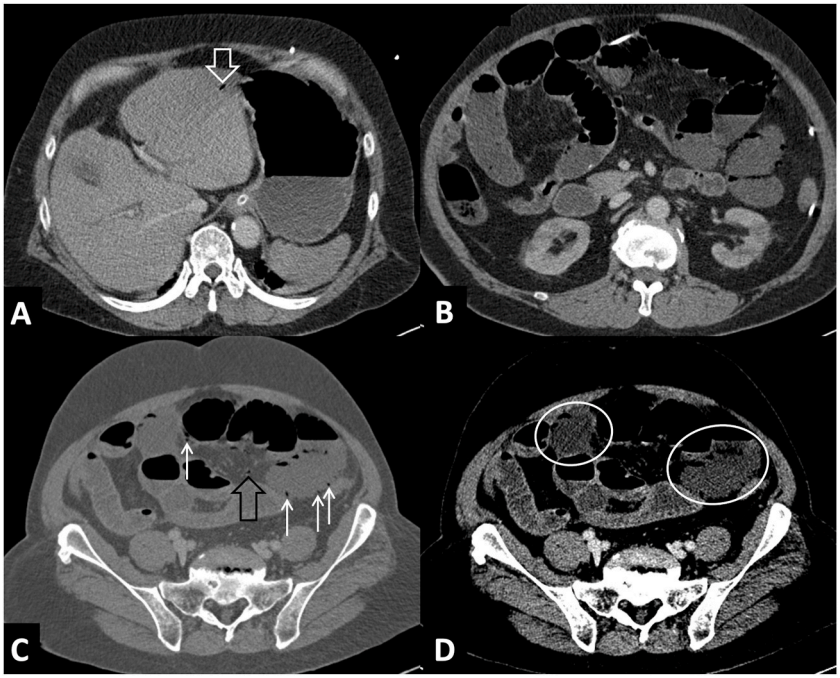

Fig. 4. Pneumatosis intestinalis, mesenteric, and portal venous gas in a 59-year-old man with transmural bowel necrosis secondary to nonocclusive mesenteric ischemia. Axial contrast-enhanced CT images in a portovenous phase (**A and B**; soft-tissue window) show an air bubble in the peripheral portal venous branch (open white arrow in **A**) and diffuse dilatation of small bowel without a transition point (**B**). Pneumatosis intestinalis (thin arrows in C) and mesenteric venous gas (open black arrow in **C**) are better shown using a wide window width setting (**C**). A narrow-window-width axial image (**D**) shows a clear distinction between jejunal loops without wall enhancement (in circles) and the rest.

pneumoperitoneum between 5.6% and 21.3% [8,15,25,26]. Calame P et al. [8] found abnormal gas significantly more frequently in TBN with nonocclusive AMI. In their investigation, pneumatosis was present in 58% of TBN patients with nonocclusive AMI (vs 28.6% of patients with occlusive AMI and 7.4% with strangulated SBO). Verdot P et al. [15] and Perez-Garcia C et al. [26] identified 27 (36.5%) and 20 (27.0%) cases (out of 74), and 41 (87.2%) and 23 (48.9%) cases (out of 47), of pneumatosis and portovenous gas, respectively. This high incidence of abnormal gas in TBN cases with nonocclusive AMI might be explained by blood flow to the bowel wall being maintained despite ischemic necrosis. The flow would allow gas to pass through the submucosa, muscular layers, and mesenteric veins [15]. It is important to note that while pneumatosis intestinalis was frequently observed in TBN secondary to nonocclusive AMI, this finding was not specific for TBN and should be interpreted in conjunction with bowel wall enhancement. A recent letter to the editor by Calame P et al. [35] highlighted the significance of assessing bowel wall enhancement in the presence of pneumatosis intestinalis. Specifically, normal bowel wall enhancement in cases with pneumatosis intestinalis would suggest an early-intermediate stage of nonocclusive AMI, rather than a more complicated case of TBN. Our results support their findings, as all seven of our nonocclusive AMI cases with pneumatosis intestinalis also exhibited decreased bowel wall enhancement.

5. Limitations

A small sample size and heterogeneous patient group limited this retrospective investigation despite retrieving almost 10 years of data. The analysis was consequently insufficiently powered to detect differences in the CT findings of the 5 TBN etiologies. Our literature review revealed that most prior studies had a limited number of patients (between 16 and 121). Small samples result from the need to Ref. [1] have pathological proof of TBN diagnoses and [2] perform surgery within an appropriate period from CT scanning (typically 24 h). Although the use of surgical pathology as an inclusion criterion created a selection bias, it ensured that our TBN diagnoses were definitive.

As we did not investigate TBN related to reversible ischemia, our conclusions were limited to TBN, and it was understood that there would be overlaps of the CT findings of TBN and reversible ischemia. The issue of overlapping CT appearances is multifaceted. First, the etiologies and pathogeneses of TBN play a central role. In addition, disease severity, location of the involved bowel segments (small vs large bowel), the presence and degree of hemorrhage in the bowel walls, and superinfection are involved. We had 3 radiologists reinterpret all CT examinations, but it was impossible to blind them from the TBN etiologies as occlusive AMI and SBO are generally unmistakable in CT images. Furthermore, it should be noted that the non-blinded nature of the image re-review process may have led to some CT signs, such as intrinsic wall hyperattenuation, being unconsciously identified. Despite the standard acquisition protocol for patients presenting with suspected acute mesenteric ischemia at our institution including an arterial phase, most CT scans were performed without this phase. The reason for this was the absence of clinical suspicion of bowel ischemia on the referring physician's

side. However, we believed this reflected the challenges in making a clinical diagnosis of this condition. In addition, almost half of the scans were performed for suspected bowel obstruction therefore the arterial phase was not routinely included. Because of heterogeneity of CT protocols, the arterial and delayed phases were not reviewed for findings.

In conclusion, our study found that the most frequent CT findings of TBN were ascites, abnormal bowel wall enhancement, bowel dilatation, and mesenteric fat stranding. Among the five etiologies of TBN, the only significant differentiating factor was bowel wall thickness, with thickening being identified most frequently in cases of TBN secondary to venous AMI and normal bowel wall thickness being found mostly in cases of TBN secondary to arterial and combined arterial and venous AMI.

Author contribution statement

Watanya Jaidee; Rathachai Kaewlai: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Wanwarang Teerasamit; Piyaporn Apisarnthanarak: Performed the experiments; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Napaporn Kongkaewpaisan; Sirinya Panya: Contributed reagents, materials, analysis tools or data; Wrote the paper.

Data availability statement

The authors do not have permission to share data.

Additional information

No additional information is available for this paper.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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