

# CT manifestations of small bowel ischemia due to impaired venous drainage-with a correlation of pathologic findings

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## Abstract

Acute abdominal pain may result from a wide variety of medical and surgical diseases. One of these diseases is small bowel ischemia, which may result in a catastrophic outcome if not recognized and treated promptly. Computed tomography (CT) by its faster image acquisition, thinner collimation, high resolution, and multiplanar reformatted images has become the most important imaging modality in evaluating the acute abdominal conditions. In this article, the author presents a description of the histology of the small bowel, pathophysiology of small bowel change, and a correlation of the pathologic and CT findings of the small bowel injuries due to impaired venous drainage. A convincing correlation of the microscopic mucosal condition with the enhancement pattern of the thickened small bowel wall on CT is useful in definitely describing the mucosal viability.

**Key words:** Computed tomography; Intestines; Ischemia; Small bowel

## Introduction

Intestinal ischemia may be divided into three categories based on the nature of ischemia: Arterial occlusive (60–70%), arterial nonocclusive (20–30%), and venous occlusive (5–10%).<sup>[1]</sup> The clinical outcomes may vary considerably depending on the acuteness, extent, and duration of ischemia. The computed tomography (CT) manifestations are related to the nature of ischemia and the degree of tissue damage.<sup>[1-10]</sup>

In arterial groups, an ischemic attack may persist long enough (persistent arterial insufficiency without

reperfusion),<sup>[8]</sup> resulting in an irreversible injury to the entire thickness of the intestinal wall (transmural necrosis), peritonitis, septic shock, and death. In this condition, the intestinal wall is paper-like thinned and poorly enhanced with or without intramural gas (pneumatosis intestinalis) on CT.<sup>[5-8]</sup> If the blood supply is restored after a certain period of ischemia (transient arterial insufficiency with subsequent reperfusion), the intestinal wall becomes thickened as the reentered blood components extravasate through the damaged microvascular wall into the extravascular space of the intestinal wall. CT shows thickened intestinal wall with various attenuations of the mucosa (enhanced

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or poorly enhanced) and the submucosa (edematous or hemorrhagic).<sup>[8,11]</sup> In the group of impaired venous drainage, the pathophysiology is different from those of the arterial groups.<sup>[1,8,11]</sup> Its CT manifestations may overlap with those of the reperfused status of transient arterial insufficiency.<sup>[8]</sup> The clinical presentations, presence of superior mesenteric venous (SMV) thrombosis, closed-loop small bowel obstruction, and other findings on CT may help differentiate between these two conditions. Among the abovementioned three groups, the first condition has the worst prognosis of the bowel viability and patient survival.<sup>[5,6,11-14]</sup> In the latter two conditions, the thickened wall with a poor mucosal enhancement has higher rates of operation, bowel resection, and bowel necrosis.<sup>[11,14]</sup>

The CT manifestations of various types of intestinal ischemia would be more appropriately discussed separately on the basis of their different mechanisms. Ischemia due to arterial diseases will not be discussed here as they go beyond the scope of this article. Images directly reveal the underlying pathologic processes and reflect the status of the small bowel at the time of imaging acquisition. Small-bowel injuries caused by impaired mesenteric venous drainage have a wide range of causes, clinical presentations, and CT manifestations.

## Causes

The mesenteric venous drainage originates from the centripetal confluence of the intramural vessels, venae rectae, venous arcade, ileocolic, right, and middle colic veins into the SMV.<sup>[15]</sup> Conditions that impair venous drainage of the small bowel may be divided into two

**Table 1: Causes of impairment of superior mesenteric venous drainage**

Inherited or acquired hypercoagulable states
Polycythemia vera, Sickle cell anemia, thrombocytosis
Protein C or S deficiency, Antithrombin deficiency
Paroxysmal nocturnal hemoglobinuria
Slow blood flow
Decreased cardiac output from any cause
Portal hypertension
Inflammation or infection-induced migratory thrombophlebitis
Intra-abdominal abscess
Pancreatitis, appendicitis
Diverticulitis, inflammatory bowel disease
Use of oral contraceptives
Recent surgery
Splenectomy, colectomy, Roux-Y gastric bypass
Closed-loop small bowel obstruction
Volvulus
Incarcerated hernia
Adhesion band
Invasion of the portal vein or SMV by an extrinsic tumor
Hepatocellular carcinoma
Pancreatic carcinoma
Intussusception
Mesenteric fibrosis due to carcinoid or retractile mesenteritis
Idiopathic

groups, namely, intravascular thrombosis and extravascular compression [Table 1].<sup>[9,12,16]</sup> These factors have been well described and are widely familiar. Among these, the condition of closed-loop small bowel obstruction needs a further description. The superior mesenteric artery and vein are closely parallel. An external compression will cause a nearly equal pressure to both the vessels. Because the artery has a much higher blood pressure and a larger thickness, it is more resistant to external pressure. Consequently, the superior mesenteric venous outflow will be first compromised. Unless the compression is tight enough at the beginning of the process to cause a complete quit of the arterial flow, impaired venous drainage will play a major role in the evolution of ischemia in this condition.<sup>[1,8]</sup> The presence of more than one of the listed factors may further increase the risk.

## Clinical presentations

Patients may clinically present with acute or insidious abdominal pain that is out of proportion to physical findings, as well as nausea, vomiting, constipation, and bloody or non-bloody diarrhea. The severity of the presentations depends on the rapidity of their development and on the degree to which venous drainage is impaired.

Acute and complete obstruction of the venous outflow is accompanied with prominent abdominal pain and a heightened possibility of intestinal infarction. This situation may occur in patients with an incarcerated hernia, volvulus, or intussusception.

In the case of SMV thrombosis, symptoms may be present 1 day to 6 weeks (estimated mean of approximately 6 days) before diagnosis.<sup>[9,12,17]</sup> Patients may experience acute or gradually increasing abdominal pain. Although this presentation is generally less classic and severe than that of arterial thromboembolism, intestinal infarction and death may still occur.

If the obstruction is gradual and chronic, as it is with tumorous thrombosis of the superior mesenteric or main portal veins, patients rarely report severe abdominal pain. They usually have collateral venous drainages, which may prevent intestinal infarction. It may need more than a few weeks to develop a well collateral circulation in the mesentery and retroperitoneum.<sup>[15]</sup>

## Histology of the Small Intestine

The intestinal wall consists of four layers. From the innermost to the outermost, these are the mucosa, the submucosa, the muscularis externa, and the serosa. Two structures are formed to augment the efficiency of absorption.<sup>[18,19]</sup> First is the permanent shelf-like plicae circulares, also called valvulae conniventes or the Kerckring folds [Figure 1A], which involve both the mucosa and

submucosa. They may achieve a height of 8–10 mm and a thickness of 3–4 mm.<sup>[18]</sup> Hence, they are discernible on CT (shown later). Second is the villus [Figure 1B]. The velvety, blanket-like intestinal mucosa is composed of innumerable villi, each approximately 0.5–1.5 mm long.<sup>[18,19]</sup> Although an individual villus is not recognizable on CT, innumerable slender and sufficiently separated villi are aggregated to form the mucosa. It is visible as an enhancing layer on the thickened submucosa. The core of the villus, called the lamina propria, is covered by a simple columnar epithelium. Between the lamina propria and the surface epithelium is the basal lamina, which is only approximately 0.03–0.07  $\mu\text{m}$  in thickness.<sup>[20]</sup> The mucosa receives 65% of the

blood flow to the intestines<sup>[21]</sup> and is the most vulnerable layer to ischemic injury.

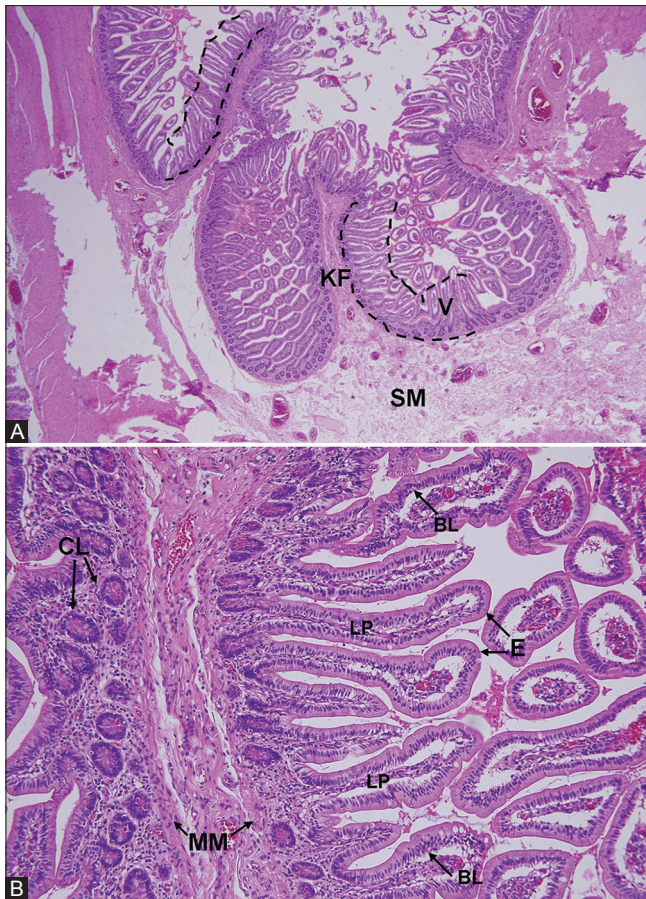
From the viewpoint of visibility on contrast-enhanced CT without high-attenuation oral contrast material, the mucosa, submucosa, and muscularis externa are most clearly distinguished if submucosal fat, edema, or hemorrhage is abundant enough to result in a three-layer appearance (target sign) of the intestinal wall. This three-layer differentiation is uncommonly seen in uncomplicated conditions.

## Pathophysiology

In terms of physiology, tissue volume is kept in a dynamic equilibrium between arterial inflow and venous outflow. When venous outflow is impaired, blood volume in the whole artery-capillary-vein system gradually increases as arterial inflow continues because of its relatively higher pressure. This results in an elevation of the intravascular hydrostatic pressure. Progressive oxygen desaturation of the stagnated blood gradually disrupts the capillary wall and increases its permeability. Intravascular contents begin to leak through the capillary wall into the extravascular interstitial space of the villus. In the early stage, fluid is the predominant leaked component. Lymphatic vessels drain this fluid into the submucosa and further into the mesentery, resulting in mesenteric edema. Then, red blood cells (RBCs) follow as microvascular wall permeability further increases. They gradually become the major component of the lamina propria of the villus as edematous fluid continuously drains out via the lymphatic vessels and arterial blood slowly flows in. The oxygen stored in these stagnant intravascular and extravascular RBCs is progressively consumed until it is unable to maintain the viability of the RBCs, the connective tissue, and the covering epithelium of the villus. The epithelium begins to slough from the villous tip; this is the earliest ischemic injury.<sup>[21]</sup>

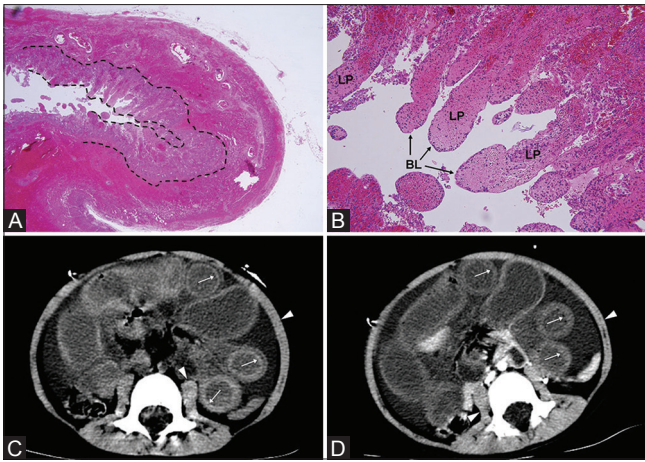
When the villus is entirely denuded of its epithelial covering, it is called a ghost villus [Figure 2A and B]. This state represents mucosal necrosis. The lamina propria of the villus is bloated with extravasated fluid, intact or disrupted RBCs, inflammatory cells, and necrotic tissue. The submucosa is filled with various amounts of edematous and hemorrhagic components; edema usually predominates in the early stage. The Kerckring folds may be stretched and flattened if the submucosa is swollen enough. A massive amount of bloody exudate may leak from the submucosa and disrupted villi into the intestinal lumen, resulting in bloody stool. Finally, ischemic injury involves the muscularis externa and terminates with transmural necrosis of the bowel wall.

If the course is chronic enough, collateral pathways may develop. Examples are cases of cirrhosis-induced portal



**Figure 1 (A and B):** A 44-year-old woman with an adhesion-induced hernia in the pelvic cavity. (A) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 40$ ) shows the surgical margin of a resected small-bowel segment and a normal Kerckring fold (KF). Covering the surface of the fold is an aggregation of numerous villi (V), which forms the mucosa (bounded by dotted lines). These are visible on computed tomography. Although the underlying submucosa (SM) is slightly edematous, the core of the Kerckring fold remains uninvolved. (B) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 200$ ) shows that the villi are slender, covered by epithelium (E), and well separated from each other. They are approximately 0.5–1.0 mm tall. The mucosa at the surgical margin was judged viable, whereas the central portion of the resected bowel was necrotic (not shown). The basal lamina (BL) is barely visible, in contrast to the much thicker muscularismucosae (MM). CL = crypt of Lieberkühn; LP = lamina propria, the core of the villus





**Figure 2 (A-D):** A 3-year-old boy with volvulus. (A) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 40$ ) shows that the columnar architecture of the villi (bounded by dotted lines) is still maintained, even though all of the epithelium has been sloughed off (necrotic mucosa). (B) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 200$ ) demonstrates the swollen lamina propria (LP) of the villi, which is predominantly filled with red blood cells and necrotic tissue. Inflammatory cells are relatively few. The covering epithelia are all disrupted, sloughed, and not visible. This kind of villus is called a ghost villus. These swollen ghost villi are compact together, in contrast to those normal, sufficiently separated slender villi shown in Figure 1. Most villi are still confined by a basal lamina (BL). (C) Non-enhanced computed tomography (CT) scan depicts the target-like, thickened wall with a high-attenuating inner layer (arrow), which is nearly isodense to the adjacent muscles (arrowhead). This finding is compatible with a necrotic mucosa composed of closely contact, RBC-filled ghost villi, as shown in A. The submucosa is edematous or hemorrhagic. (D) On the contrast-enhanced CT scan, the inner layer (arrow) is not enhancing and has become hypoattenuating compared with the abdominal wall muscles (arrowhead), indicating poor blood supply to the mucosa. The edematous submucosa is enhanced and becomes isodense to hemorrhagic mucosa, even though still hypodense to muscles

hypertension, invasion of the portal vein by hepatocellular carcinoma, encasement of the SMV by pancreatic carcinoma, vascular encasement by various degrees of mesenteric fibrosis in carcinoid or retractile mesenteritis,<sup>[22,23]</sup> and others. In these circumstances, injury to the small bowel may be minimal, or it may involve only submucosal edema or congestion. In rare cases, it extends to hemorrhagic necrosis. The process of ischemic injury varies considerably according to the acuity of onset and the completeness of venous occlusion.

### Computed Tomography Techniques

The CT scans were performed on a single-detector spiral scanner (Light Speed, Sytec CT 4000, GE, Milwaukee, WI). The patient was given at most 500 ml of water, depending on the patient's tolerance, as an oral contrast agent approximately 30 minutes before the examination. Positive oral contrast agent was avoided because it can obscure the delicate enhancement pattern of the intestinal wall.<sup>[11,14,24]</sup> A baseline scan from the diaphragm to pelvis

was obtained before the administration of an intravenous contrast medium. Then, a post-contrast scan was started 60–80 seconds later after the beginning of an injection of 100ml nonionized iodinated contrast agent at a speed of 1.5–2 ml/s. The slice thickness was 7.5mm without a gap. The scanning time was 15–20 seconds. All images were interpreted on axial sections. Two or three-dimensional reformatted images were only occasionally obtained.

### Computed tomographic manifestations

With the introduction of high-speed scanning, ultra-thin collimation, high resolution, and multiplanar reformation, multidetector row CT has become the most important imaging modality for evaluating acute abdominal conditions, including ischemia of the small bowel.

CT manifestations of small-bowel changes due to impaired mesenteric venous drainage are symmetric and circumferential wall thickening, and varying attenuations of the thickened wall on non-enhanced and contrast-enhanced scans. Normal thickness of the small-bowel wall depends on the extent of luminal distention. If adequately distended, the wall may be invisible or only 1–2 mm thick, with 3 mm regarded as the upper limit.<sup>[25]</sup> If the lumen is collapsed, the outer diameter of the bowel may reach 20–25 mm (a sum of the Kerckring folds, submucosa, and muscularis externa) in the proximal jejunum and 10 mm in the ileum. The involved segment is usually long in SMV thrombosis or volvulus; of widely variable length in adhesion, segmental twist, or incarcerated hernia; and short in intussusception.

A detailed description of the components of the thickened wall is useful in explaining the different CT appearances. In terms of the pathology, non-neoplastic thickening of the small-bowel wall is most commonly caused by edema or hemorrhage in the submucosa. Relatively uncommon components are fat and gas. Fat deposition may occur in Crohn's disease and ulcerative colitis.<sup>[26]</sup> Intramural gas dissection may be encountered in catastrophic infarction, pulmonary diseases, peptic disease, diverticulosis, steroid use, ileostomy, or other situations.<sup>[8]</sup> Both fat and gas are easily recognized by their extremely low attenuations, and they rarely cause difficulty in differential diagnosis.

Evaluation of the bowel wall attenuation may be aided by a comparison of the bowel wall and the adjacent abdominal wall or psoas muscles [Table 2].<sup>[14]</sup> Normally, the muscles have a density of approximately 40–60 Hounsfield units (HU). It increases by approximately 10–20 HUs after intravenous contrast injection.

### Non-enhanced scans

The thickened wall attenuation is usually homogeneous<sup>[26]</sup> and differentiation of the mucosa from submucosa is difficult. However, if the mucosa is severely hemorrhagic and infarcted, a target-like wall composed of a high-density

inner layer, an edematous low-density middle layer, and an intermediate-density outer wall may be seen on the pre-contrast scan [Figure 2C]. The thickened wall

**Table 2: A comparison of the attenuations of the non-neoplastic, thickened small bowel wall, and the adjacent abdominal wall muscles**

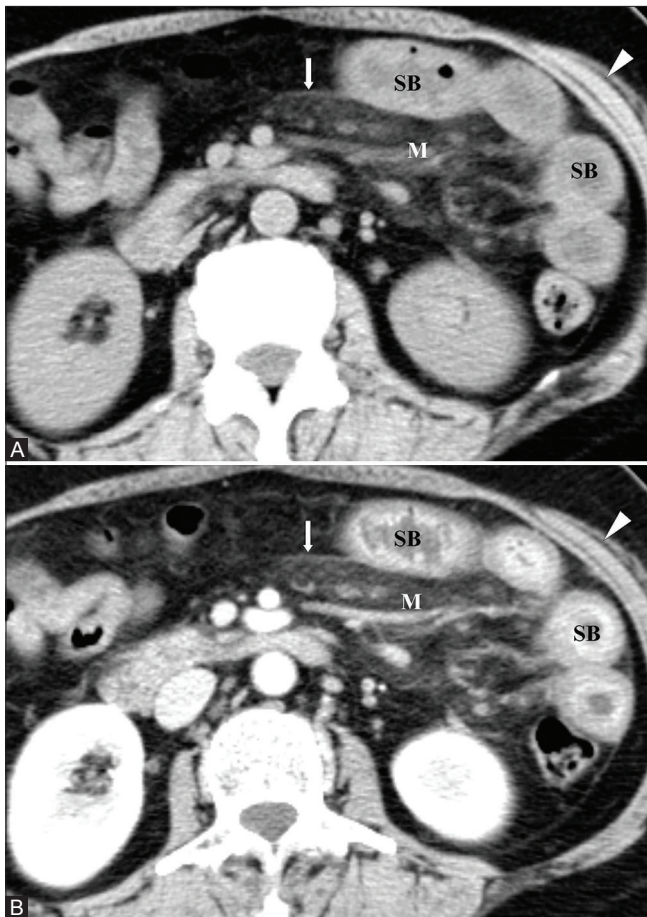
Submucosa	Edematous (10–30HU) - hypodense to muscles (40-60 HU) on pre-contrast scan - more hypodense to the slightly enhanced muscles (50–70HU)
	Hemorrhagic (60–80HU) - isodense or hyperdense to the muscles on pre-contrast scan - hypodense or isodense to the slightly enhanced muscles on post-contrast scan
Mucosa	Indistinguishable from the submucosa on the pre-contrast scan Viable mucosa is hyperdense (>120HU) to the muscles on the post-contrast scan Necrotic mucosa is invisible and indistinguishable from the submucosa on the post-contrast scan

attenuation may be similar to that of fluid or lower than that of the muscles if edema is the predominant component (10–30 HUs). If its density is similar to or slightly higher than that of the muscles, it is compatible with a substantial hemorrhagic component (60–80HUs).

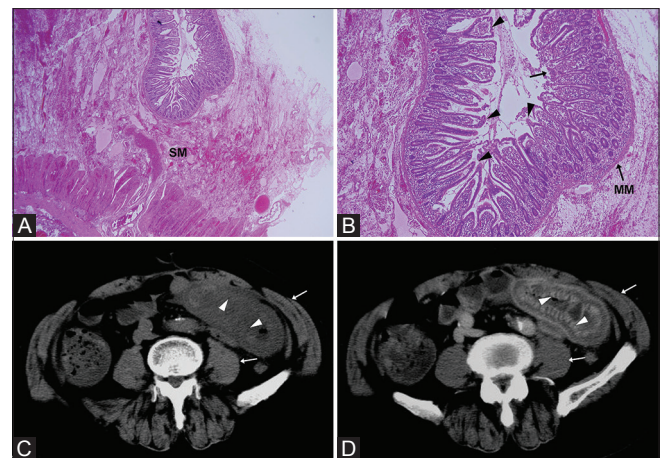
**Contrast-enhanced scans**

The submucosa may only show mild enhancement because its capillary network is much less than that of the mucosa. The density of the edematous or hemorrhagic submucosa is lower than that of the adjacent abdominal wall or psoas muscles [Figure 2D] on the contrast-enhanced scans, as the muscles enhance normally. A chronically congested or hyperemic wall has low attenuation compared with that of the muscles before intravenous contrast enhancement. It shows strong, whole-thickness enhancement, and becomes isodense or hyperdense to the muscles on the post-contrast scan [Figure 3].

Of special importance is the density of the inner layer of the thickened wall, which is supposed to reflect the status of the mucosa.<sup>[11,14]</sup> Because the mucosa receives 65% of the blood flow to the intestines, a normally structured and perfused mucosa should be the brightest layer (>120 HUs) of the thickened wall, regardless of the edematous or hemorrhagic submucosa, on the contrast-enhanced scans.



**Figure 3 (A and B):** A 58-year-old woman with mesenteric panniculitis, which is a preceding process of retractile mesenteritis. Varying degrees of mesenteric fibrosis may constrict venous branches and result in impaired venous drainage. (A) Non-enhanced scan. The small bowel wall (SB) is symmetrically thickened and hypoattenuating relative to the muscles (arrowheads). The mesenteric fat (M) is diffusely hyperattenuated to the other fatty tissues and partly covered anteriorly by the thickened visceral peritoneum (arrow) of the mesentery. (B) On this contrast-enhanced scan, the congested wall has become hyperdense to the muscles



**Figure 4 (A-D):** A 70-year-old man with adhesion-induced strangulation. (A) Photomicrograph (hematoxylin and eosin stain, original magnification ×40) shows an edematous and hemorrhagic submucosa (SM). (B) Photomicrograph (hematoxylin and eosin stain, original magnification ×100) shows some villi with ulceration of their tips (arrow), evidence of earliest ischemic necrosis. However, most villi (arrowheads) remain slender and architecturally intact. MM = muscularis mucosae. (C) Non-enhanced scan shows a thickened wall (arrowhead) that is somewhat homogeneous in attenuation and darker than the muscles (arrow). (D) On contrast-enhanced computed tomography, attenuation of the normally enhancing mucosa (arrowhead) is higher than that of the muscles (arrow). The swollen Kerckring folds closely contact together, forming a black-white interlacing appearance composed of the enhancing mucosa and submucosal edema. The patient received surgical enterolysis of the adhesion with segmental small-bowel resection. The pathologic specimen was not judged as necrotic



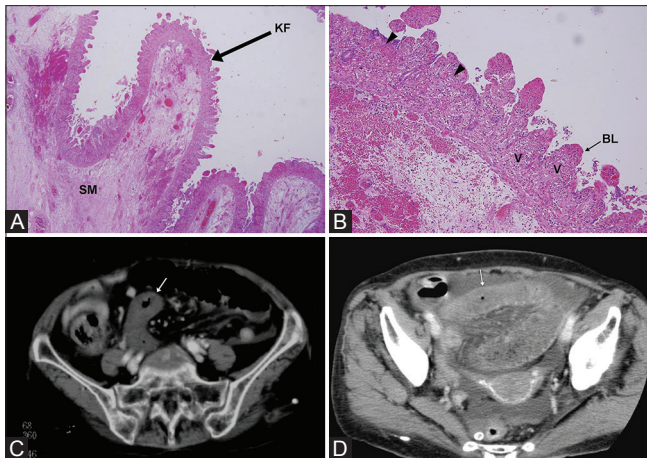
An adequately enhancing inner layer represents a normal blood supply to the normally structured mucosa, and the bowel tends to be viable [Figure 4]. However, the lack of an enhancing inner layer may represent poor blood circulation in the mucosa, which may have become necrotic or even sloughed [Figures 2, 5 and 6]. The non-enhancing mucosa is indistinguishable from the submucosa and not visible on CT. This finding represents further injury of the bowel wall (at least mucosal necrosis), and it results in a prognosis worse than that associated with normal inner-layer enhancement.<sup>[14]</sup>

Surgical intervention, small-bowel resection and necrosis, and death may happen more commonly in patients whose CT scans show poor enhancement of the thickened wall than in those with normal enhancement.<sup>[11,14]</sup> However, absence of inner-layer enhancement is not proof that the bowel is nonviable [Figure 7]. This is because ischemia-induced bowel necrosis may be mucosal, mural, or transmural depending on the severity of injury. In mucosal necrosis, damage is limited to the mucosa. Mural necrosis involves both the mucosa and the submucosa, or even part of the muscularis externa. When the entire thickness of the muscularis externa is involved, transmural necrosis occurs, and bowel perforation is unavoidable. Small bowel affected

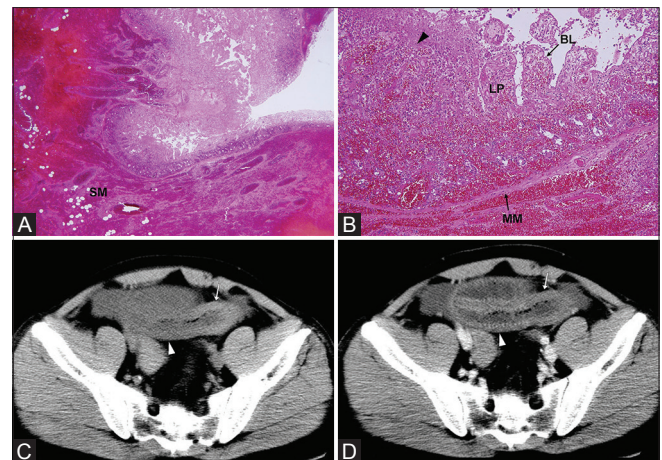
by only mucosal or mural necrosis may survive without a need for resection, though late-occurring fibrosis-induced stricture may be a complication. The CT manifestation and clinical outcome of a case with SMV thrombosis presented in another report<sup>[27]</sup> were compatible with the above descriptions. The post-contrast CT scan of that case revealed poor inner layer enhancement of a symmetrically and circumferentially thickened small-bowel segment, which was darker than the abdominal wall muscles. The patient received anticoagulant therapy and recovered without surgery. However, she developed fibrotic stricture months later. The CT picture and clinical course were consistent with an ischemic injury limited to mucosal or mural necrosis.

Persistent enhancement of the thickened wall<sup>[28]</sup> is possibly related to slow blood flow and delayed extravasation of molecules of the contrast material into the extravascular interstitial space. This is corresponding to the angiographic demonstrations of a prolongation of the entire arterial phase and intense opacification of the thickened bowel wall.<sup>[29]</sup> This picture might not be perceived unless the enhancement of the other uninvolved segments has faded on a scan delayed long enough [Figure 8].

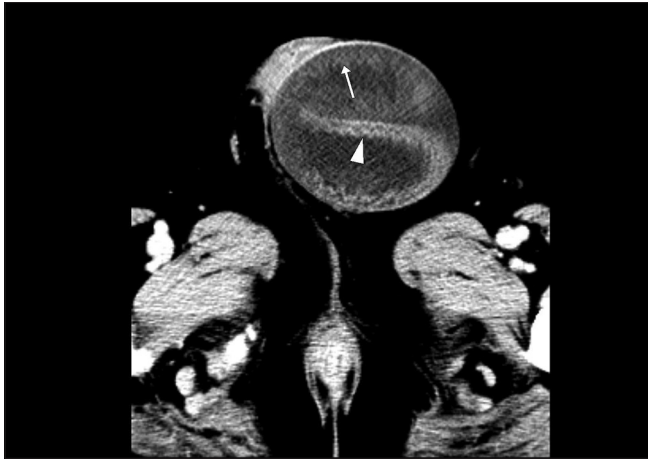
In addition to the different appearances of the bowel, CT can show underlying pathologic processes that can impair



**Figure 5 (A-D):** (A–C) An 83-year-old woman with adhesion-induced strangulation. (A) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 40$ ) shows a predominantly edematous submucosa (SM) and a severely swollen Kerckring fold (KF). (B) On this photomicrograph (hematoxylin and eosin stain, original magnification  $\times 200$ ), ghost villi (V) are swollen and compacted together. Their mean height is approximately 0.5 mm, similar to that of normal villi. Boundaries of some villi are intact; others are lost (arrowheads). The villi are filled with red blood cells, inflammatory cells, and necrotic tissue. BL = basal lamina. (C) Contrast-enhanced computed tomography (CT) scan shows a thickened wall (arrow) of homogeneous attenuation without a recognizable, enhancing inner layer. The thickened wall is hypodense to the adjacent muscles. This appearance is consistent with mucosal necrosis. The patient recovered after surgery. (D) Contrast-enhanced CT scan in an 81-year-old woman with episodes, pathologic results, and an outcome similar to those of the patient in A–C. The thickened ileum (arrow), approximately 2 cm in outer diameter, showed no inner-layer enhancement



**Figure 6 (A-D):** A 42-year-old man with superior mesenteric venous thrombosis. (A) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 40$ ) shows the submucosa (SM) stuffed with red blood cells (RBCs). (B) Photomicrograph (hematoxylin and eosin stain, original magnification  $\times 200$ ) shows ghost villi that are swollen and compacted. Some are still confined by a basal lamina (BL), whereas others (arrowhead) are disrupted and merged together. The lamina propria (LP) is filled with RBCs and necrotic tissue. MM = muscularis mucosa. (C) On non-enhanced computed tomography (CT), attenuation is higher on the left half (arrow) of the thickened wall than on the right (arrowhead), and it is similar to the adjacent muscles. This finding suggests an increased hemorrhagic component in the left half. (D) On contrast-enhanced CT scan, the right half (arrowhead) has normally enhancing mucosa, including Kerckring folds, and is now clearly seen. The left half (arrow) does not show similar inner-layer and fold enhancement (an appearance consistent with mucosal necrosis), and it is hypoattenuating relative to the muscles



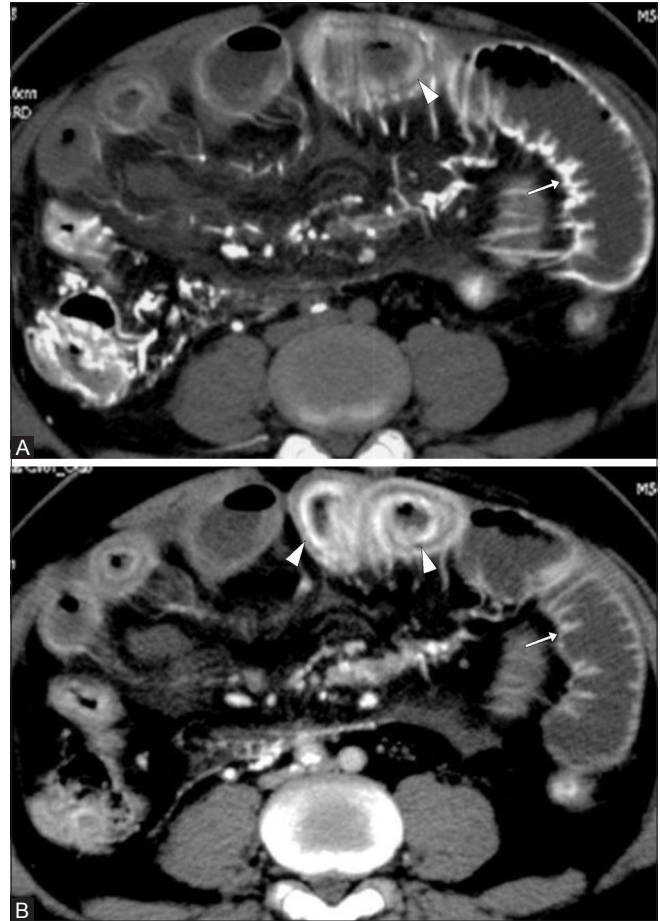
**Figure 7:** A 73-year-old man with an incarcerated left inguinal hernia. The covering mucosa of upper segment (arrow) is poorly enhancing on contrast-enhanced computed tomography. The patient recovered after surgical reduction without resection of the small bowel. This outcome indicates that transmural necrosis did not occur, though the mucosa might have been damaged or necrotic. The normally enhancing mucosa of the lower segment (arrowhead) was about 1 mm thick. The heaved Kerckring fold, approximately 4–6 mm tall, is composed of a dark edematous submucosa (like a mountain) and covered by a bright mucosa (like a forest)

venous drainage. In SMV thrombosis, the vein may be completely non-opacified or rim-enhanced [Figure 9]. The enhancing rim represents either blood flow around the thrombus or a markedly enhanced and hyperemic venous wall.<sup>[12]</sup>

In closed-loop small-bowel obstruction, volvulus results in the typical whirl of the superior mesenteric vessels and associated bowel. An internal hernia through a congenital or acquired foramen of the mesentery or ligaments may be recognized because of the special course of the splanchnic vessels<sup>[30-33]</sup> or the unusual locations of bowel loops.<sup>[32,33]</sup> Common appearances of closed-loop obstruction due to any cause include a circumscribed or U-shaped configuration of distended loops that are nearly completely filled with fluid [Figure 10]. Another common appearance is a radial distribution of fluid-filled dilated loops with convergence of the associated stretched and engorged vessels (spoke-wheel sign).<sup>[34]</sup> Images may also depict fusiform tapering (beak sign) of the two ends of the dilated loop [Figure 11], a triangular shape of the ends of the bowel, or two collapsed adjacent loops.<sup>[35,36]</sup>

Helical and multidetector-row CT can sometimes provide two-dimensional reformatted images to add details regarding the transition site of the obstruction [Figure 11].<sup>[36]</sup> Intussusception shows protrusion of the intussusciens, mesenteric fatty tissue, and vessels into another small bowel segment [Figure 12].

Other abdominal findings include mesenteric fat stranding due to edema or hemorrhage, engorgement of the mesenteric



**Figure 8 (A and B):** A 54-year-old man with superior mesenteric venous thrombosis. (A). Early phase of computed tomographic arteriography. The mucosa of the target-like, thickened wall (arrowhead) is hypodense to that of the uninvolved segment (arrow). (B). Delayed phase shows a contrary appearance. The mucosa of the thickened wall (arrowhead) becomes hyperdense to that of the uninvolved segment (arrow). These appearances reflect a slow blood inflow and delayed extravasation of molecules of the contrast material into the extravascular space, resulting in a prolonged mucosal enhancement in contrast to that of the normally circulated segment

vessels, an increased outer diameter of the bowel due to wall thickening or luminal distention, and serosanguinous peritoneal fluid. This serosanguinous ascites is extravasated from the bowel wall and mesentery; it represents increased severity of bowel injury.

### Pitfalls of Interpretation

Pseudothickening of the normal small-bowel wall is sometimes encountered in two conditions. This appearance results from highly vascularized Kerckring folds, which are more abundant in the jejunum and gradually diminish in size and number toward the ileum. Normally, the jejunum has a thicker wall and a more intense enhancement than the ileum.

First is in the non-distended jejunum. The normal Kerckring folds may reach a height of 8–10 mm and





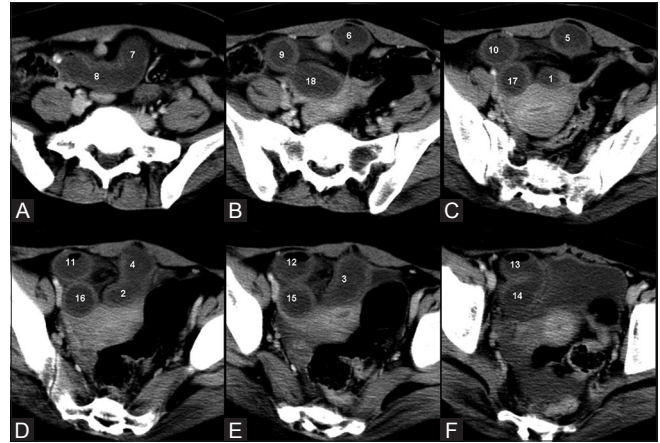
**Figure 9:** A 30-year-old woman who received total colectomy. Contrast-enhanced scan shows a nonoccluding thrombus (arrowhead) in the superior mesenteric vein and an adjacent abscess (arrow)



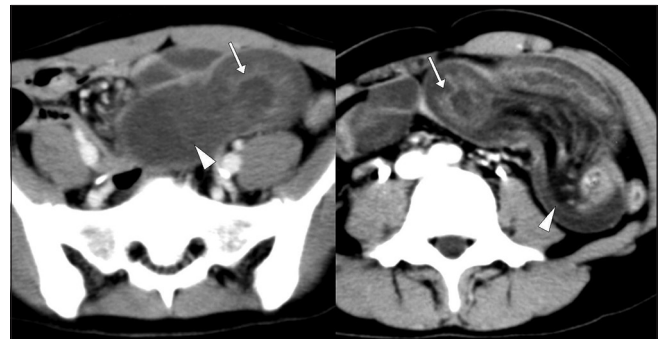
**Figure 11:** A 34-year-old man with an adhesion-induced closed-loop obstruction. Coronal reformatted image clearly shows a beak sign and the constriction point (arrows), and submucosal edema (arrowheads) of the wall

a width of 3–4 mm.<sup>[18]</sup> When these numerous folds crowd together in a non-distended jejunum, they may result in a wall thickened to 9–12 mm (plus the thickness of the submucosa and muscularis externa), with an outer diameter of approximately 18–25 mm. On the pre-enhancement scan, its density is lower than or similar to that of the muscle [Figure 13]. After the intravenous administration of contrast medium, scans show homogeneous and intense enhancement of the entire wall, which becomes brighter than the muscles. This finding should not be misinterpreted as a thickened wall without inner-layer enhancement, which suggests mucosal necrosis.

The second condition occurs with cross-sectional imaging of a distended jejunum filled with low-attenuating fluid [Figure 14]. Because of a partial volume-averaging effect, normal folds may look like a poorly enhancing or target-like



**Figure 10 (A-F):** A 32-year-old woman with an adhesion-induced internal hernia. (A–F) Contrast-enhanced skipped, nonadjacent images. The circumscribed segment is totally filled with fluid and can be successfully traced from one end (1) to the other (18). Both ends are in close contact. The wall is approximately 2.75–3.75 mm thick. It is poorly enhancing and darker than the muscles. The patient received small-bowel resection and recovered. The specimen showed hemorrhagic necrosis of the full thickness of the wall



**Figure 12:** A 23-year-old woman with polyposis-induced intussusception. Mesenteric vessels and fatty tissue are clearly seen in the bowel lumen. The thickened submucosa is either covered by normally enhancing, viable mucosa (arrow) or poorly enhancing, necrotic mucosa (arrowhead) which is invisible. The pathologic specimen showed transmural necrosis

thickened wall. To avoid this mistake, it is better to measure the wall thickness on a longitudinal section of the small bowel.<sup>[11]</sup>

## Conclusion

Offering fast imaging and further improved images of the entire abdominal cavity, helical and multidetector row CT can show multiple various manifestations of small bowel ischemia due to impaired venous drainage, including detailed information about a thickened wall. Normal mucosal enhancement implies better blood circulation in the thickened wall than poor enhancement does. The prognosis is better with the former condition than with the latter. CT can also reveal underlying abnormalities, such as SMV thrombosis, closed-loop small-bowel obstruction, and intussusception.





**Figure 13 (A and B):** A 30-year-old man with nonspecific abdominal pain. (A) On this non-enhanced scan, the collapsed small bowel (arrow) has a wall thickness larger than 1cm and an outer diameter of approximately 25mm. Its attenuation is lower than that of the adjacent muscle (arrowhead). (B) Strongly enhancing bowel (arrow) is brighter than the muscles (arrowhead) on this contrast-enhanced scan. The patient recovered uneventfully

#### Essentials

1. Symmetrical and circumferential wall thickening of the small bowel is the typical CT manifestation of small bowel change due to impaired venous drainage. However, it may occur in a wide variety of other pathologic processes.
2. A well-enhanced inner layer, generally agreed to be the mucosa, of the thickened small bowel wall usually represents an adequate blood supply and implies a good prognosis.
3. A poorly enhanced inner layer suggests a necrotic mucosa and implies a worse prognosis even though not necessarily to the extent of transmural necrosis.
4. A practical and reliable method of evaluating the attenuation value of the bowel wall is a direct visual comparison of the bowel wall and the adjacent abdominal wall or psoas muscles. Hemorrhagic mucosa/submucosa is brighter than muscles on pre-enhanced images and darker on post-contrast images. Sometimes, a Hounsfield unit measurement is needed.



**Figure 14:** Same patient as in Figure 13. Kerckring folds (arrows) of the longitudinally sectioned, distended bowel is approximately 4–6 mm high and 2–3 mm thick, with a 2–3 mm interfold distance. The section thickness is 7.5mm. A transverse scan of the wall (rectangles) would involve two folds at most like that of the adjacent, continuous, transversely sectioned bowel (arrowheads); this can mimic a target sign or a poorly enhancing thickened wall. This appearance should not be interpreted as wall thickening

5. Pseudothickening may be encountered in cross-sectioned images of the small bowel. Longitudinal-sectioned scans would more definitely show the wall thickening.

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#### Conflicts of interest

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