Bowel ischemia represents a process of insufficient blood supply of the small or large bowel with the consequences ranging from a transient, totally reversible attack to a lethally catastrophic event. This condition may result directly from arterial occlusion (thromboembolism of the superior mesenteric artery or its branches, vasculitis of various autoimmune diseases, and external compression of the artery by adhesion, volvulus, hernia, and intussusception), hypotension (congestive heart failure, hypovolemia, and sepsis), or vasoconstrictive medications (digitalis, norepinephrine, and ergotamine). Bowel ischemia may also be associated with impaired venous drainage (thrombosis of the mesenteric and portal veins, interference of intramural venous outflow by distention and elevated intraluminal pressure that occurs proximal to a stenotic lesion, and compression of the mesenteric veins by tumor, adhesion, volvulus, hernia, and intussusception) [1–8]. The severity of the ischemic attack depends on the acuteness, duration, degree, and state of the collateral circulation; extent of the involved area; and promptness in correcting the underlying pathologic process. The damage starts with the mucosa, which is most vulnerable to the ischemic insult, extends outward through the submucosa and the proper muscular layer, and ends at the serosa. The extent of the injury may range from mucosal, to mural, to transmural necrosis. We describe different conditions of the ischemic event and their corresponding CT appearances.

CT Manifestations of Bowel Ischemia

Chung Kuao Chou

Pictorial Essay

Fig. 1.—51-year-old man with aortic dissection. Patient did not receive oral contrast material as evidenced by low-density gastric fluid. Contrast-enhanced axial CT scan reveals clear distinction between normally enhanced (arrows) and unenhanced (arrowheads) collapsed small-bowel loops. Normally enhanced duodenum and proximal jejunal loops were supplied by anastomotic branches from gastroduodenal artery and possible minimally patent jejunal artery. Aorta (A) and superior mesenteric artery (SMA) were occluded with thrombi-filled false lumen.

Fig. 2.—63-year-old man with aortic dissection. Contrast-enhanced axial CT scan shows wall of fluid-distended small-bowel loops either normally enhanced (arrow) or totally unenhanced (arrowheads). Unenhanced bowel wall was isodense and not differentiable from intraluminal fluid.

Fig. 3.—62-year-old woman with abdominal angina. Contrast-enhanced axial CT scan shows some small-bowel loops (arrowhead) with bowel wall density lower than other loops (arrow) but higher than luminal fluid. This enhancement is graded as suboptimal, implying that blood flow is present but less than normal.
The CT examinations were performed on a Sytec 4000 scanner (General Electric Medical Systems, Milwaukee, WI). The slice thickness was 7 or 10 mm with a gap of 3 or 0 mm, respectively. Oral contrast medium was not routinely used. The unenhanced images were taken from the diaphragm to the lower borders of the kidneys. The enhanced images were taken from the diaphragm to the symphysis pubis. The IV contrast medium used was Telebrix 30 meglumine (Laboratorie Guerbet-93600, Aulnay and S. Bois, Roissy CdG Cedex, France). The dosage was a rapid manual injection of 100–150 mL.

Persistent Arterial Insufficiency Without Reperfusion (Pale Ischemic Type)

Sometimes, the ischemic event persists long enough without reperfusion and becomes destined to its final outcome: necrosis of the whole bowel wall. The intramural arteriocapillaries first lose part of their volume as the earlier entered blood flows out from the veins, even though some blood may seep back from the veins. At this moment, the CT shows a thin, poorly or suboptimally enhanced bowel wall (Figs. 1–4). Occasionally, detailed ischemic mucosal folds can be seen (Fig. 5). Poor enhancement along the antimesenteric side is suggestive of nonocclusive ischemia (Fig. 6). The intestinal fluid is decreased because the enterocytes cannot produce a normal amount of secretions if the arterial supply is blocked. The bowel wall is first pale and then turns to black and becomes thinned as a result of intravascular volume loss and collapse of necrotic tissue [1, 5]. Not uncommonly, the infarcted bowel is described as grossly dark red or purple and filled with bloody fluid. In fact, this description represents a reperfused instead of a nonreperfused ischemic bowel because there should not be a lot of erythrocytes or plasma extravasating through the damaged and...
CT of Bowel Ischemia

Fig. 8.—57-year-old man with aortic dissection. Contrast-enhanced axial CT scan shows mucosa was edematous and suboptimally enhanced (arrow) compared with other normally enhanced mucosa (arrowhead). Submucosal edema was evident.

Fig. 9.—70-year-old man with septic shock.
A, Unenhanced axial CT scan shows ascending and transverse colonic wall was thickened (arrowheads) and of attenuation.
B, Contrast-enhanced axial CT scan shows mucosa was normally enhanced (arrow) after IV contrast administration. Edematous submucosa (arrowhead) reveals mild enhancement, with increase of approximately 10 H on average, indicating contrast medium extravasation.

Fig. 10.—64-year-old man with paroxysmal atrial fibrillation. Superior mesenteric arteriogram obtained during venous phase shows contrast medium stasis in thickened wall (arrow) consistent with contrast extravasation into edematous submucosa during reperfusion stage.

Fig. 11.—70-year-old woman with atrial fibrillation.
A, Unenhanced axial CT scan shows intermediate density–thickened transverse colonic wall (arrowhead).
B, Contrast-enhanced axial CT scan obtained after IV contrast administration shows bowel wall was enhanced (arrowhead), with increase of approximately 30–40 H on average, consistent with contrast medium extravasation into hemorrhagic wall.
ruptured microvascular wall into the mucosa, submucosa, or bowel lumen if the arterial supply is severely reduced, either occlusively or nonocclusively, without a subsequent reperfusion taking place. Microscopically, inflammatory cell infiltration in response to bacterial invasion is much more prominent than RBC extravasation in the nonreperfused attenuated wall. The evolution of bloody diarrhea or bloody intraluminal fluid is most likely due to an outpouring of the reperfused blood from the infarcted mucosa or submucosa into the lumen. Even though the residual blood in the capillaries that flows back from the venules may cause extravasation of RBC in the mucosa (lamina propria of the villi) or scattered hemorrhagic foci in the submucosa or subserosa, it is unlikely for this small amount of hemorrhage to cause considerable wall thickening. As bacteria proliferate and more gas is produced, the intraluminal gas may dissect into the necrotic wall (pneumatosis intestinalis), spread through the mesenteric veins, and finally flow into the portal veins (Fig. 7).

**Transient Arterial Insufficiency with Subsequent Reperfusion (Hemorrhagic Type)**

If the pathologic processes were corrected (by lysis of the embolus, reestablishment of blood pressure, release of external compression, or prompt development of collateral circulation), the reentered blood might cause different CT appearances, depending on the degree of disruption of the vascular wall integrity. The intestinal microvessel derives its oxygen supply through direct diffusion from the blood. When the arterial supply is insufficient for a certain period and returns later, the microvascular endothelium and the mucosal epithelium become damaged, and the permeability increases proportionately to the duration of oxygen deprivation and the degree of the reperfusion injury. If the degree is mild, only water molecules leak into the extravascular space and cause a mucosal or submucosal edema appearance on CT (Fig. 8). When the damage becomes more severe, the molecules of contrast medium follow the previously escaped fluid and cause various degrees of mucosal or submucosal enhancement (Figs. 9 and 10). As the ruptures between the damaged endothelial cells further enlarge, the RBC also leak, resulting in a thickened soft-tissue-density bowel wall with or without mucosal enhancement (Figs. 11 and 12). The thickened mucosal folds or thumbprinting appearance seen radiologically are caused by submucosal edema or hemorrhage. The mucosa may remain intact or become necrotic. In the case of reperfusion, the bowel is grossly dark red, the wall is thickened, and the lumen is largely filled with bloody fluid in contrast with appearances of the nonreperfused condition.

**Impaired Venous Drainage**

When the mesenteric venous drainage is impaired, the intravascular volume increases, and the hydrostatic pressure rises as the arterial blood continues flowing into the capillary bed and venules of the bowel and mesentery. The elevated hydrostatic pressure causes the molecules of water or contrast material, or...
ally engorged during this condition. The mesenteric veins are usu-
more common and prominent. Mesenteric vas-
more tightly, the wall enhancement might be
less than the venous outflow. Thus, the CT ap-
venous drainage (Fig. 15A). The relatively rich
vascular engorgement usually occurs with impaired
venous pressure, the arterial inflow is usually
increased if RBC are released into
venous drainage (Fig. 15A). The relatively rich
arterial supply may contribute to increased in-
the density of the intraluminal fluid (asterisk) in 40-year-old woman was consistent with, but not diagnostic of, bloody contents, which was confirmed by surgical specimen.

Ischemia Due To Closed-Loop Small-
rectiovascularendotheliumintothesubmucosa,
appearing as submucosal and mesenteric eda
blood flow or thrombosis of the arterial supply may contribute to increased in-
creased. If the compression is tight enough from
the beginning of the obstruction, the wall might
be thin and totally unenhanced, similar to that of
total fluid filling of the lumen (Fig. 15C). This appearance may oc-
more than the venous outflow. Thus, the CT ap-
venous outflow. Thus, the CT ap-
may result from increased vascular permeability as a result of oxygen deprivation or elevated in-
the latter is more common and prominent. Mesenteric vas-
venous hydrostatic pressure caused by im-
venous drainage. Both the artery and vein are compressed.
Because the arterial pressure is higher than the venous pressure, the arterial inflow is usually
nonreperfused ischemia (Fig. 15B).

Mesenteric edema or hemorrhage (Fig. 15A) may result from increased vascular permeability as a result of oxygen deprivation or elevated in-
and subsequent bowel necro-
Figs. 13 and 14). These appearances are similar to those of reperfusion ischemia previously de-
the mechanism is different from that of a directly arterial origin, which is
caused by oxygen-deprived and free rad-
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