Acute Mesenteric Ischemia Induced by Ligation of Porcine Superior Mesenteric Vein:

Multidetector CT Evaluations

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Rationale and Objectives: To evaluate multidetector computed tomography (MDCT) for detecting the early changes and dynamic evolution of acute mesenteric ischemia (AMI) induced by the ligation of superior mesenteric vein (SMV) in an experimental porcine model.

Materials and Methods: Twelve pigs were randomly assigned to three experimental groups, and one control group with three pigs in each group. After laparotomy, the SMV was separated and ligated in nine pigs and separated without ligation in three controls. MDCT pre- and postcontrast with arterial, venous, and delayed phase scans, and CT angiography reconstructions of mesenteric vessels were carried out at preoperation, 6 hours, 12 hours, and 18 hours after ligation. The findings of mesenteric vessels, bowel, abdominal cavity at pre- and postoperation, and dynamic evolution were correlated with pathology.

Results: AMI-induced pathological changes were identified in all nine experimental pigs. MDCT angiography clearly delineated main trunk of the SMV, peripheral major and minor tributaries up to brushy vasa recta, and the location and shape of ligations. The early ischemic findings were bowel wall thickening, mesenteric edema, ascites, and pronounced bowel enhancement. Superior mesenteric artery and its major branches appeared spasm with poor filling and delayed and prolonged visualization. SMV and its tributaries were poorly delineated with delayed opacification. We also saw thinning of bowel wall, dilatating bowel with fluid, aggravating mesenteric edema and ascites, and poor enhanced bowel over time.

Conclusion: MDCT detects early changes of mesenteric ischemia and its evolution after ligation of porcine SMV, and may find application in early diagnosis of human venous occlusive AMI.

Key Words: Mesenteric ischemia; superior mesenteric vein; tomography; x-ray; computed; animal model.

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Mesenteric venous thrombosis (MVT) remains a lethal disease. It usually results in acute mesenteric ischemia (AMI) and even necrosis if not promptly treated. The 30-day mortality is 20%–27% (1–4). Because of variations in clinical manifestations, courses, and prognosis from different etiology of MVT, it is hard to diagnose acute MVT at an early stage. Improvements in noninvasive imaging techniques, especially multidetector computed tomography (MDCT), may lead to a more favorable outcome of acute MVT (5–7). Identifying patients earlier in their clinical courses can not only avoid intestinal necrosis and unnecessary surgery, but also prompt nonoperative management such as interventional therapy, and hence reduce mortality. According to Zhang et al (7), mortality rate reached up to 39% in patients with acute MVT who underwent surgical therapy, whereas nonoperative management or interventional therapy markedly reduced hospital mortality.

It was shown that sensitivity and specificity of MDCT for MVT were both higher than 90% (8) when MVT occurs in main trunk of superior mesenteric vein (SMV) or portal vein, whereas characteristic changes of AMI usually appeared at a late stage. The value of MDCT remains uncertain in the identification of the early AMI, or in MVT involving predominantly the small mesenteric veins. A retrospective study by Kumar et al (9) suggested that 44% of MVT predominantly involved the small mesenteric veins, which was more difficult to diagnose early, more likely to develop bowel necrosis, and more frequently required surgery.

We conducted this study to evaluate MDCT early changes and dynamic evolution of AMI induced by ligating SMV in an experimental porcine model.

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MATERIALS AND METHODS

Animal Preparation

Twelve female pigs weighing 20–25 kg ages 130–150 days from Shanghai Miaodi Biotechnic Company (License SCXK2005-0002) were housed for acclimation at the research animal care center for 1 week. All experiments were approved by the Hospital Ethics Committee. The animals were assigned randomly to three experimental groups (6-hour group, 12-hour group, and 18-hour group) and one control group ($n = 3$ in each group). The animals were anesthetized with intramuscular injection of 0.2 g ketamine and 0.2 mg diazepam in the thigh and maintained during the procedure by slow instillation of 5% GS 500 mL by mixing the same anesthetic. Intravenous access was obtained by inserting an 18-gauge trochar into an auricular vein.

A midline laparotomy was performed. The small intestine and its mesentery were exteriorized. The distal main trunk of the SMV draining the distal jejunum and ileum and ileocolic vein were ligated. Two or three jejunal venous tributaries remained patent. Separation without ligation of SMV was performed in control animals only.

Imaging Protocols

MDCT pre- and postcontrast scans with arterial phase, venous phase, and delayed phase from the dome of the diaphragm to the pubic symphysis were performed before the operation; at 6 hours postligation in the 6-hour group; at 6 hours and 12 hours postligation in the 12-hour group; and at 6 hours, 12 hours, and 18 hours after ligation in the 18-hour group using a 40-slice scanner (Somatom Sensation 40, Siemens, Erlangen, Germany) with a collimation of 0.6 mm, pitch of 0.75, slice thickness of 1.0 mm, tube rotation of 0.37 seconds, kVp of 120, and mAs of 70. For initiating arterial phase scanning, the time to peak aortic enhancement was first estimated by a minitest bolus of 10 mL of iodinated contrast injected at 4 mL/second (8–13 seconds; mean 11 seconds). Finally, 2 mL/kg of 370 mg I/mL of Ultravist (Bayer Schering Pharma, Guangzhou, China) was power-injected at a rate of 4 mL/second, followed by 25 mL of saline flush, and arterial phase scanning was performed. Venous phase and delayed phase scanning followed after a delay of 35 and 60 seconds, respectively.

Computed tomography angiography (CTA) of the superior mesenteric artery (SMA) and SMV using techniques of volume rendering, maximum intensity projection and thin slab maximum intensity projection, and coronal multiplanar reformation of the abdomen were reconstructed.

CT Data Analysis

The anatomy and changes of mesenteric vessels, bowel, abdominal cavity at pre- and postoperation, and dynamic evolution were evaluated focusing on the following aspects: shape, size, and density of vessels; sites of ligation; thickness, density, and enhancement of bowel wall; width of bowel lumen; amount and density of fluid in bowel lumen and peritoneal cavity; and mesenteric stranding or fluid and intramural of intravascular gas collections. The findings were correlated with the histopathological results. All observations and measurements were performed by two radiologists who showed agreement in all images.

Pathological Analysis

The experimental animals were sacrificed at 6, 12, and 18 hours after operation with an intravenous injection of 10% KCL 20 mL. The control animals were sacrificed at 18 hours. Laparotomy was performed to observe the location and severity of bowel ischemia, which was determined by bowel wall color and thickness, the degree of dilation, inflammatory changes, and vascular changes. A 3-cm segment of distal jejunum, ileum, colon, and normal jejunum were resected and fixed in 10% formalin solution, embedded in paraffin, sectioned, stained with hematoxylin and eosin, and subsequently analyzed for microscopic pathological changes.

Statistical Analysis

The chi-square Fisher exact test was used to test differences between the groups. We considered a P value of $< .01$ to indicate a significant difference after Bonferroni correction.

RESULTS

CTA Manifestations of Normal SMV

MDCT angiography clearly delineated the main trunk of the SMA and SMV and peripheral major and minor branches up to brushy vasa recta in all animals. SMV converged by jejunal vein, ileal vein, and ileocolic vein, joined with the splenic vein to form the portal vein. Three to five jejunal venous tributaries draining the jejunum in the right upper quadrant joined in the main trunk of the SMV from the right side. Ileal veins usually had one or two tributaries in the left lower quadrant or right lower quadrant and jointed the main trunk of the SMV. The ileocolic vein located in the left abdomen transversally joined the SMV from the left side. It had two tributaries that drained the colon at the left abdomen and the distal ileum in the left lower quadrant. More distal tributaries appeared brushy along the main trunk (Fig 1).

CTA Findings of Mesenteric Ischemia

After the operation, CTA showed the following significant findings: spasm of SMA and its major branches in nine pigs with markedly thinning of caliber in three pigs, opacification of few peripheral arterial branches of small caliber in nine pigs, and delay and prolongation of arterial phase showing visualization of SMA in venous phase and delayed phase in nine pigs.

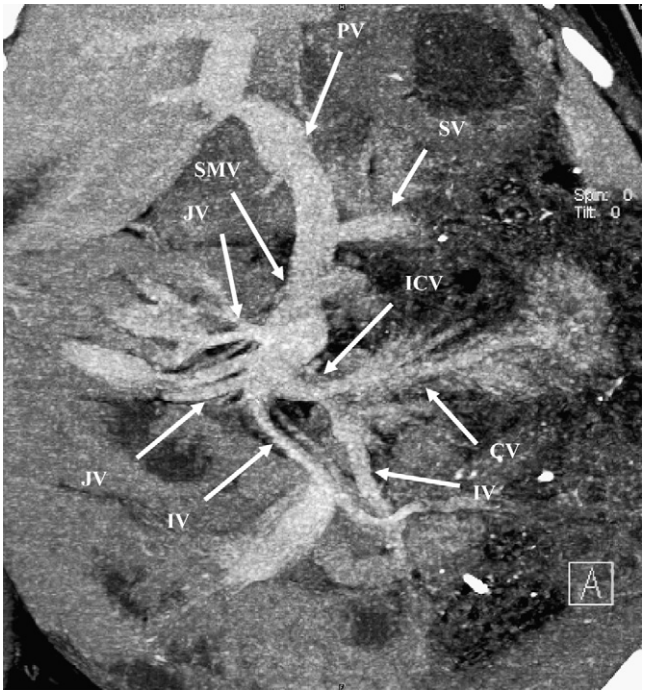

Figure 1. Thin slab maximum intensity projection image shows that the superior mesenteric vein (SMV) was converged by jejunal veins (JV), ileal veins (IV), and ileocolic vein (ICV), which is formed by the colic vein (CV) and IV. The SMV joins the splenic vein (SV) to form the portal vein (PV). The JV are located in the right upper quadrant. IV are in the lower quadrants and continue to SMV. Ileocolic vein is in the left abdomen and transversally joined SMV from the left, and has two tributaries draining the colon at left abdomen and the distal ileum at the left lower quadrant. More distal tributaries appear brushy along the main trunk.

Though contrast-enhanced CT was performed both before and after operation at each time point, we did not find any residual contrast media in the vasculature, even in the bowel distal to ligation in precontrast images at each time point. SMV and its tributaries demonstrated smaller caliber, poor opacification, and delayed visualization after enhancement. CTA readily identified the location of ligation as a nonopacification area with a slim waist. Distal to the ligation, the veins still opacified, with a larger inflexible caliber (Fig 2).

Abnormal CT Findings of Bowel and Abdominal Cavity

Thickening of bowel wall was an early sign of mesenteric ischemia seen in all pigs at 6 hours (100%) and in only one of three pigs (33%) at 18 hours. Bowel wall thinning was a late sign, and was not found in any animals at 6 hours and in two of three pigs (67%) at 18 hours with a typical "paper thin" bowel wall. Bowel dilatation with fluid was found in one of nine pigs (11%) at 6 hours, in two of six pigs (33%) at 12 hours, and all three pigs at 18 hours, with progressively increasing intraluminal fluid. Extravasation of contrast media into bowel lumen was demonstrated in one pig in both the 12-hour and 18-hour groups. Mesenteric edema was identified in nine (100%) and ascites in eight of nine pigs (89%) at 6 hours, which became aggravated with the lapse of time. The density of intraluminal fluid and ascites increased with time. The average CT value of ascites was 101 ± 4 Hounsfield units at 6 hours, 15 ± 3 Hounsfield units at 12 hours, and 15 ± 6 Hounsfield units at 18 hours. Enhancement of bowel wall increased in all nine pigs (100%) at 6 hours and decreased in all six pigs (100%) at 12 hours. Bowel enhancement reduced progressively at 18 hours, with missing enhancement in one pig (Fig 3). Abnormal CT findings of the bowel and abdominal cavity are shown in Table 1.

Pathologic Findings

Gross observation. At 6 hours after operation, autopsy found that the affected bowel appeared pink with normal peristalsis in three pigs and distended with spasm of the proximal jejunum in one of three pigs. Involved bowel wall and its mesentery looked thick in all three pigs. Ascites was seen in two of three pigs. At 12 hours, similar bowel changes presented with slightly weak peristalsis and deepening color. At 18 hours, ascites was found in all three pigs, with sanguineous fluid in one pig. Thinned bowel wall was detected in two of three pigs with cyanotic color. No venous thrombosis was found in SMV, even distal to the ligation at three different time points.

Histological findings. AMI changes were identified pathologically in all nine experimental pigs. At 6 hours after SMV ligation, the mucosal and submucosal layers showed congestion, edema, and venous dilatation. The focal mucosal necrosis was detected in one pig. At 12 hours, congestion and edema in all layers of bowel wall, focal necrosis and hemorrhage in mucosa, venules dilatation and infiltration of inflammatory cells (mainly lymphocytes) in the submucosa were found. At 18 hours, more severe ischemic changes included diffuse necrosis, ulcerations in mucosa, interruption and dissolving of muscle fiber, and fibrous exudation in the serosa (Fig 4).

DISCUSSION

MVT accounts for 5–15% of AMI (8). The most commonly associated risk factors are portal hypertension; hypercoagulation; trauma; intraabdominal inflammatory conditions including pancreatitis, appendicitis, diverticulitis, diffuse peritonitis, and parasitic infestation; and recent surgery, especially splenectomy; bowel obstruction; and oral contraceptives (11). Many cases are idiopathic. AMI develops when MVT is associated with a lack of adequate venous collaterals, which results in the development of intestinal mucosal edema and subsequent arterial hypoperfusion.

To our knowledge, dynamic evolution of MVT in CT and magnetic resonance imaging manifestations has not been previously reported, although some studies investigated pathophysiologic and angiographic changes (12–14). In our initial experiment, we investigated the effect of ligation site on

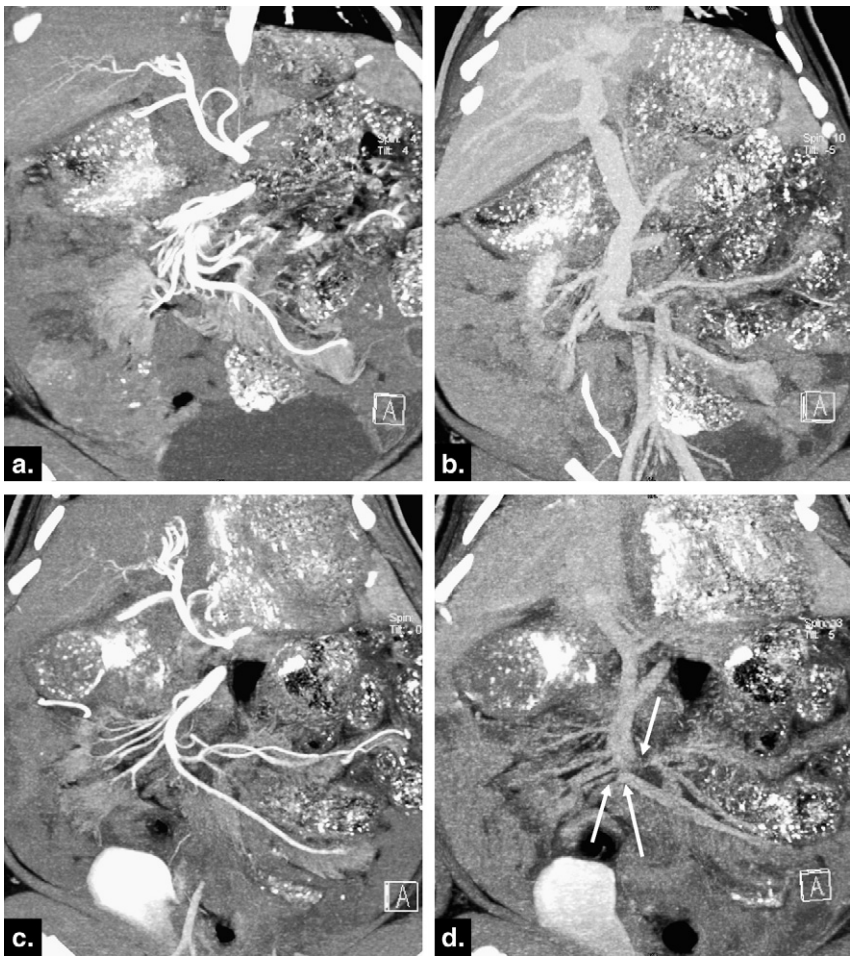

Figure 2. Thin slab maximum intensity projection (TSMIP) images of arterial phase **(a)** and venous phase **(b)** before operation clearly show fully filled main trunk and branches of the superior mesenteric artery (SMA) and superior mesenteric vein (SMV). TSMIP images of arterial phase **(c)** and venous phase **(d)** at 6 hours after operation show spasm of SMA and its major branches with markedly shrunk caliber, poor filling, and fewer branches. SMV and its tributaries demonstrate smaller caliber and poor and delayed opacification. The sites of ligation appear as a nonopacification area with the shape of slim waist (*arrows*). Distal to the ligation, the veins still opacify with a larger inflexible caliber.

successfully duplicating MVT model. When the main trunk of the SMV near the confluence of the splenic vein was ligated, all animals died 2–3 hours after operation. At autopsy, the visible changes included bowel and mesentery swelling, with normal color and peristalsis, SMV congestion, and ascites. No intestinal necrosis and perforation occurred. It was supposed that the animals died from circulatory collapse due to the reduced blood volume returning to the heart, hypovolemia, and hemoconcentration. When the ligation happened at slightly distal site and kept one to two major jejunal venous tributaries patent, the animals survived 6–8 hours after operation with no intestinal necrosis seen. When we ligated one or two major ileal veins, no marked abnormality was found in subjacent bowel and peritoneal cavity at 6 hours, 12 hours, 18 hours, or 36 hours on CT imaging. The laparotomies, performed at 18 hours and 36 hours, respectively, showed that only slight edema in subjacent bowel and mesentery presented. The bowel retained its normal color and peristalsis with no obvious pathologically ischemic change found in mucosa. We presumed that collateral circulation that existed extensively between occluded and nonoccluded bowel and mesentery prevented the development of bowel ischemia. Finally, on the basis of initial experimental results, we ligated distal main trunk of SMV, which drained the distal jejunum and ileum and ileocolic veins, keeping two to three major jejunal veins patent. AMI changes were identified pathologically in all nine experimental pigs. Bowel distal to ligation showed cyanosis, congestion, edema, wall thickening, hemorrhage, and necrosis. The experimental model provided venous ischemia with overall degree of ischemic insult roughly correlated with the duration of venous ligation by histologic evaluation. The changes after 6 hours corresponded to early ischemic damage, mainly wall edema, which may be reversible. After 12 hours, the changes were more severe, likely with permanent injuries. If anticoagulant therapy was promptly instituted, injured bowel may return to normal by fibrotic repair. After 18 hours, complete bowel necrosis was present. All animals were tolerant during the study with stable vital signs.

Early detection of intestinal ischemia before infarction develops remains difficult. In recent years, however, the widespread availability of MDCT; technical advances in MDCT temporal, spatial, and contrast resolutions; and the resulting high quality of CTA with three-dimensional reformatted techniques have improved significantly visualization of the mesenteric vascular anatomy, thrombus, and, at the same time, extravascular abnormalities and indirect signs of

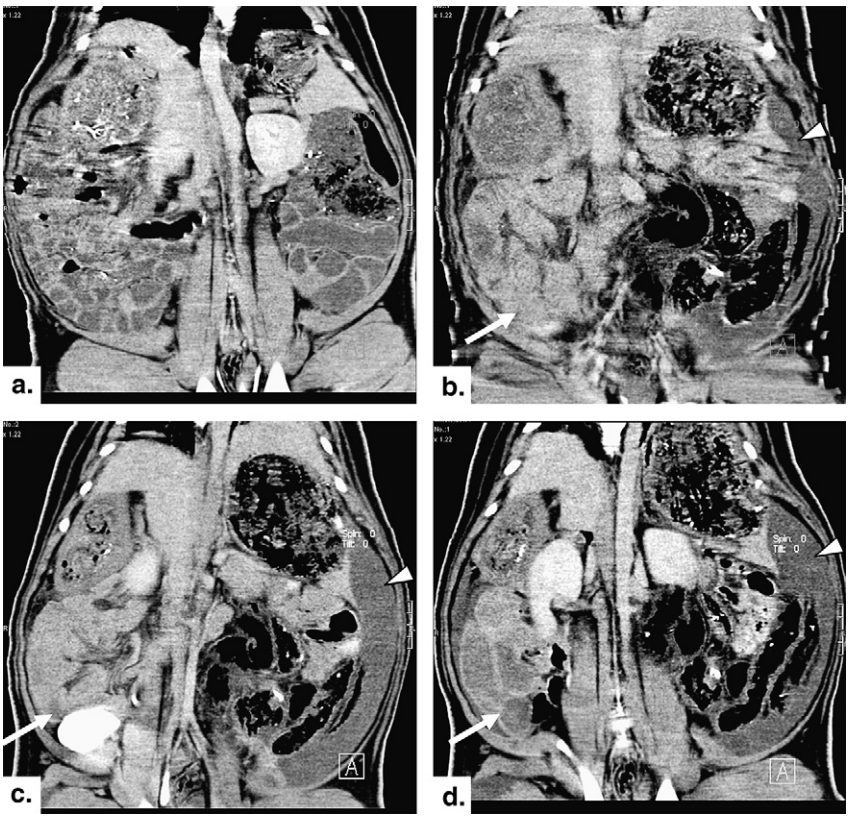

Figure 3. Coronal multiplanar reformation images at preoperation **(a)**, 6 hours **(b)**, 12 hours **(c)**, and 18 hours **(d)** after the ligation show dynamic changes with dilatating bowels with fluid and aggravating ascites.

intestinal ischemia (5,6). Some studies have shown that CT was diagnostic in 90% of patients with MVT (1,2), whereas other investigators reported that the role of CT was not same in two different MVTs (9). They found that conventional or single-slice spiral CT had a sensitivity of 97% in MVT with splenic or portal vein involvement, but only 67% of sensitivity in isolated MVT with predominant involvement of the small mesenteric veins. Our results suggest that MDCT angiography can clearly delineate not only main trunk of SMV, peripheral major and minor tributaries up to brushy vasa recta, but also the postoperative abnormalities of the mesenteric vessels.

The early ischemic findings in vessels appear as follows: the vasospasm of SMA and its major branches display poor filling, small caliber, delayed and prolonged visualization; SMV and its tributaries display poor and delayed opacification with reduced caliber. These findings are consistent with digital subtraction angiography findings reported by other investigators (15,16). They observed intraluminal defect, poor and delayed opacification or nonopacification in mesenteric vein, vasoconstriction of SMA, and prolonged visualization of arterial arcades. In our study, the location and shape of ligations are correctly identified as nonopacification with slim waist configuration. The veins distal to the ligation

TABLE 1. Abnormal Computed Tomography Findings of Bowel and Abdominal Cavity and Dynamic Evolution

Abnormal Computed Tomography Findings	6 Hours (n = 9)	12 Hours (n = 6)	18 Hours (n = 3)	Controls (n = 3)
Thickening of bowel wall	9 (100%)*	5 (83%)*	1 (33%)	0
Thinning of bowel wall	0 (0)	1 (17%)	2 (67%)	0
Bowel dilatation with fluid	1 (11%)	2 (33.3%)	3 (100%)	0
Mesenteric edema	9 (100%)*	6 (100%)*	3 (100%)	0
Ascites	8 (89%)*	6 (100%)*	3 (100%)	0
Increased enhancement of bowel wall	9 (100%)*	0 (0)	0 (0)	0
Reduced enhancement of bowel wall	0 (0)	6 (100%)*	3 (100%)	0

*P < .01 vs. controls.

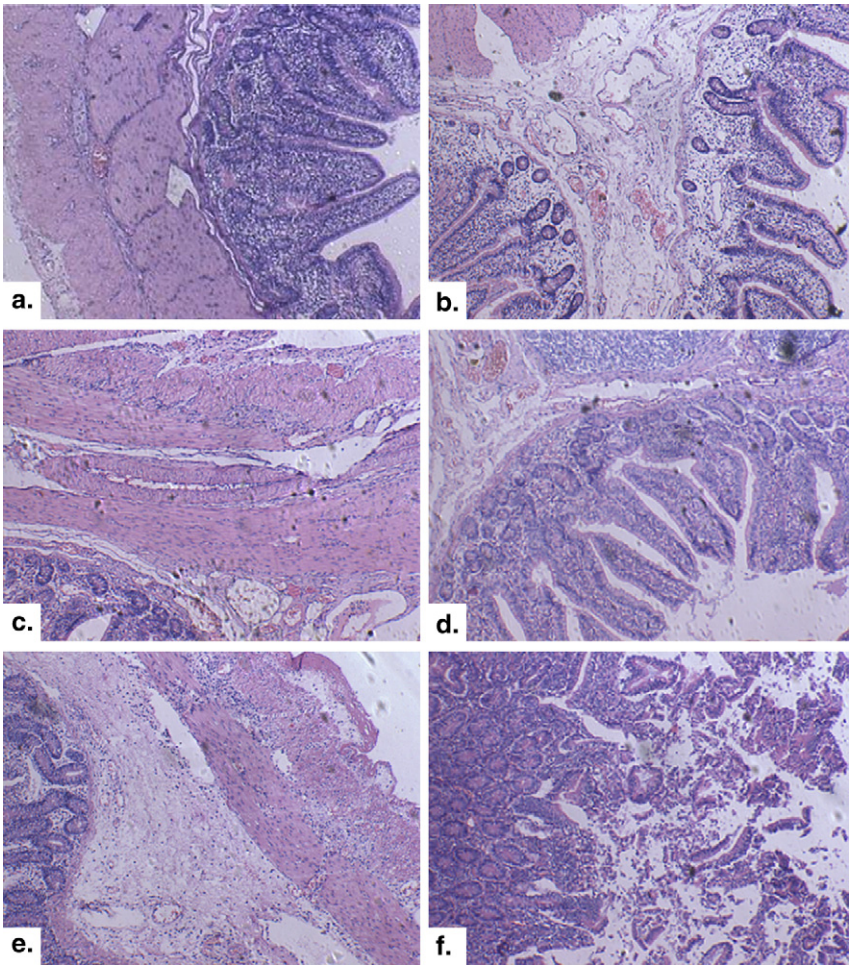

Figure 4. Normal bowel wall shows intact mucosal layer and submucosal layer **(a)**. At 6 hours after ligation of superior mesenteric vein, congestion and edema are seen in the submucosal layer with intact mucosal layer **(b)**. At 12 hours, the entire bowel wall shows congestion and edema with focal mucosal necrosis. More striking venule dilatation and infiltration of inflammatory cells appear in submucosal layer **(c, d)**. At 18 hours, there are diffuse mucosal necrosis and ulcerations in mucosa, interruption and dissolving of muscle fiber, and fibrous exudation in the serosa **(e, f)**.

expand and remain opaque, with a stiff shape. With the prolonging duration of ischemic insult, no previously mentioned abnormalities change markedly.

The vascular abnormalities of MVT are identified exactly by MDCT angiography. However, because of the absent time-dependent evolution, acuteness, duration, severity, and prognosis cannot be determined depending on these abnormalities alone without combining the indirect findings in bowel and abdominal cavity. Imaging findings related to the pathophysiology of intestinal vascular injury from venous etiology may be divided into four phases (17). Spastic reflex ileus with homogeneous thickened bowel wall and without bowel distention is identified in the first phase. In the second phase is a hypotonic reflex ileus with progressive intestinal intramural and mesenteric edema. CT findings are related to bowel wall thickness, pronounced enhanced mucosa, and submucosal edema that display a typical "target sign" (17,18). The third phase is characterized by mesenteric vascular engorgement and edema with formation of venous collateral vessels. CT may reveal thickened bowel wall and mesentery, ascites, and mesenteric engorgement. In the fourth phase, intestinal necrosis and peritonitis occur (17,19). Absence of wall enhancement, presence of peritoneal fluid and gas in vascular branches, bowel wall, or peritoneum are typical CT findings.

The present study shows that early indirect ischemic findings are bowel wall thickness, mesenteric stranding, pronounced bowel enhancement, and ascites with the sensitivity of 100% in the three former findings, 89% in the latter. Among these findings, bowel wall thickness and pronounced bowel enhancement have a relatively high specificity of 67% and 100%, respectively, which are consistent with a previous report (20). In addition, no "target sign" in thickened bowel is identified. The causes may be rapidly progressive ischemia in this animal model with relatively small animals. At a late stage, findings with relatively high sensitivity and specificity of 67% and 100%, 100% and 89%, and 100% and 100% are thinning bowel wall, bowel dilatation with fluid, and reduced wall enhancement, respectively.

Although the previously mentioned results are obtained from the acute occlusion of the SMV, in a routine clinical setting, thrombosis usually develops gradually in the SMV system, which leads to subacute or chronic venous mesenteric ischemia and corresponding indolent course and nonspecific manifestations. The involved bowel may develop enough collateral circulation to prevent necrosis development. However, those cases that develop into bowel necrosis are due largely to such factors as rapidly propagating thrombus and completely obstructing venous lumen that extends extensively to peripheral tributaries or to the main trunk, even the

portal vein. Under such circumstances, effective collateral circulation cannot be established and venous drainage from the bowel is compromised. The disease progresses rapidly and intestinal necrosis develops early. The pathological course and clinical and CT manifestation from ischemia to infarction are similar to those findings observed in this study.

This study has several limitations. First, no evidence of thrombus distal to the ligation was found, as we initially anticipated. Therefore, no thrombus in the venous branches or vasa recta was observed by MDCT angiography. The prerequisites of thrombosis are not only very slow or nearly stopped blood flow, but also a hypercoagulable state. Second, contrast-enhanced CT performed both before and after operation at each time point of the study may result in residue accumulation of contrast medium, especially in bowel distal to ligation, which may produce artificial bowel wall enhancement. We did not find any contrast media in the vasculature, even in the in bowel distal to ligation in precontrast images after surgery. Third, the small sample size with limited number of animals compromises statistical power. The extrapolation of the results from this animal study into human application should be cautious and must be validated further with clinical studies.

In conclusion, this animal study has shown that human venous occlusive AMI can be duplicated successfully in the porcine by SMV ligation. MDCT and CTA can be used to diagnose this venous AMI at an early stage by detecting direct and indirect early changes in vessels and in the bowel.

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