Post-Ischemic Bowel Stricture: CT Features in Eight Cases

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Objective: To investigate the characteristic radiologic features of post-ischemic stricture, which can then be implemented to differentiate that specific disease from other similar bowel diseases, with an emphasis on computed tomography (CT) features.

Materials and Methods: Eight patients with a diagnosis of ischemic bowel disease, who were also diagnosed with post-ischemic stricture on the basis of clinical or pathologic findings, were included. Detailed clinical data was collected from the available electronic medical records. Two radiologists retrospectively reviewed all CT images. Pathologic findings were also analyzed.

Results: The mean interval between the diagnosis of ischemic bowel disease and stricture formation was 57 days. The severity of ischemic bowel disease was variable. Most post-ischemic strictures developed in the ileum (n = 5), followed by the colon (n = 2) and then the jejunum (n = 1). All colonic strictures developed in the “watershed zone.” The pathologic features of post-ischemic stricture were deep ulceration, submucosal/subserosal fibrosis and chronic transmural inflammation. The mean length of the post-ischemic stricture was 7.4 cm. All patients in this study possessed one single stricture. On contrast-enhanced CT, most strictures possessed concentric wall thickening (87.5%), with moderate enhancement (87.5%), mucosal enhancement (50%), or higher enhancement in portal phase than arterial phase (66.7%).

Conclusion: Post-ischemic strictures develop in the ileum, jejunum and colon after an interval of several weeks. In the colonic segment, strictures mainly occur in the “watershed zone.” Typical CT findings include a single area of concentric wall thickening of medium length (mean, 7.4 cm), with moderate and higher enhancement in portal phase and vasa recta prominence.

Keywords: Bowel stricture; Stenosis; Ischemic stricture; Bowel ischemia; CT

INTRODUCTION

Post-ischemic strictures are extremely rare sequelae of ischemic bowel disease (1, 2). Post-ischemic strictures present as circumferential segmental ulcers and afferent tubular segment stenosis (3). However, they are at times difficult to differentiate from other bowel diseases such as Crohn’s disease, nonsteroidal anti-inflammatory drug (NSAID)-induced enteropathy and/or cryptogenic multifocal ulcerous stenosing enteritis (CMUSE) secondary...
to overlapping clinical symptoms and histopathological findings (4). If a patient does not have a clear history of ischemic bowel disease, it is difficult to make a diagnosis of post-ischemic stricture formation. Therefore, if characteristic radiologic features of post-ischemic strictures are identified, these “markers” might help differentiate them from other similar bowel diseases.

A few cases of post-traumatic ischemic strictures and ischemic strictures following surgery have been reported (5-7), as well as ischemic stricture as a result of mesenteric vein thrombosis and ischemic enteritis (3, 4, 8-10). However, most of these studies focused on clinical findings. Contrast-enhanced computed tomography (CT) is the diagnostic tool of choice for assessment of the location, nature, extent and etiology of any bowel obstruction thought to be present. Therefore, the purpose of this study was to investigate the CT findings associated with post-ischemic strictures that could be used to differentiate the disease from other bowel diseases. We evaluated a series of eight patients, with available surgical specimens and correlated CT findings with pathologic findings.

MATERIALS AND METHODS

This study was approved by our Institutional Review Board, which waived the requirement for Informed Consent from each patient owing to the retrospective nature of the analyses.

Patients

We searched for the terms “ischemic colitis,” “ischemic enteritis,” “ischemic bowel disease,” “bowel ischemia,” and “ischemic stricture” in abdominal CT reports from July 2005 to August 2015. Of the 2230 patients identified, eight patients (five men and three women; age range, 29–86 years; mean age, 59.3 years) who met the following three criteria were included in the study. The three criteria consisted of: 1) documented CT evidence of both ischemia and the resultant stricture, 2) a medical history negative for previous bowel surgery or other procedures such as intestinal vessel embolization or abdominal trauma, which could result in ischemia; and 3) pathologic confirmation of benign ischemic stricture obtained through segmental resection (n = 5) or through colonoscopy or double-balloon enteroscopy (n = 3).

Clinical Data

Detailed clinical data (compiled from the available electronic medical records) was reviewed. The information included patient gender, chronologic age and a description of the underlying disease process to include symptoms at the time of diagnosis with ischemic bowel disease and at the time of stricture development and vital signs at the time of diagnosis with ischemic bowel disease (blood pressure, heart rate, white blood cell count, and body temperature). The base information also described the nature and duration of the physician-supervised care of ischemic bowel disease and stricture and the time elapsed from the date of diagnosis with ischemic bowel disease and formal identification of stricture development.

Imaging Techniques

All of the patients who participated in the study underwent contrast-enhanced CT imaging with 16- or 64-multidetector CT scanners (Somatom Sensation 16 or 64 or Somatom Definition, Siemens Medical Systems, Erlangen, Germany), with 3–5 mm. slice-thickness using dual-phase scanning. Arterial and portal venous phase imaging scans (abdominal-pelvic dynamic CT [dynamic CT]) were obtained, with or without oral administration of 1200 mL of a 3% sorbitol solution, or single portal-phase scanning (routine abdominopelvic CT), with or without pre-contrast imaging. For contrast enhancement, intravenous contrast media (2–2.5 mL/kg; iopromide [Ultravist, Bayer Healthcare, Berlin, Germany] or iopamidol [Pamiray, Dongkook, Jincheon, Korea]) was administered at a rate of 2–3 mL/s using an automatic injector. The arterial and portal phase scans were obtained with a 15–25-second delay using the bolus-tracking technique and a fixed 72-second delay, respectively, following intravenous injection of the contrast agent.

Two patients who did not have a complete or high-grade obstruction at the time of imaging underwent a small bowel series, and one patient underwent a double-contrast barium enema of the colon (colon study). The small bowel series was performed after nothing by mouth for at least eight hours, and with the use of a barium and methylcellulose solution. A single-contrast examination was performed to evaluate the entire small intestine. This was followed by a double contrast examination after oral administration of an effervescent agent.
Image Analysis

All eight patients underwent CT scans at the time of diagnosis with post-ischemic stricture, and images were available for each patient. The CT images were retrospectively reviewed by two radiologists, with a consensus being reached regarding their findings. The findings revealed by the imaging studies were evaluated according to the methods established in previous studies as follows (11, 12): location; segment length; thickness of the affected bowel wall (one-side); evidence of thickening of the wall (concentric or non-concentric), enhancement characteristics during arterial and portal phase imaging (homogeneous, with the entire bowel wall enhancing equally; mucosal, with only the innermost layer enhancing; layered, with both the mucosal and serosal layers enhancing and with a central band of relatively reduced enhancement), presence of higher enhancement in portal phase than arterial phase (defined as when enhancement during the portal phase is 15 HU higher than during the arterial phase, or when the enhanced area of the wall is larger during the portal phase as compared with the arterial phase), level of enhancement (decreased, decreased as compared with the adjacent normal bowel wall; mild, similar as compared with the adjacent normal bowel; moderate, more than normal bowel wall but less than nearby vascular structures; marked, markedly increased versus nearby vascular structures). Mesenteric manifestations such as vasa recta prominence or infiltration and ascites were also seen and documented. Bowel obstruction of small and large bowel was evaluated using a 3-point scale (absent, low-grade, or high-grade). A low-grade obstruction was defined as dilatation of the proximal small bowel by 3−4 cm and dilatation of the proximal large bowel by 6−10 cm. A high-grade obstruction was defined as proximal small bowel dilatation of > 4 cm and proximal large bowel dilatation of > 10 cm (13).

Computed tomography images obtained at the time of diagnosis (with ischemic bowel disease were also evaluated). Attenuation was appreciated on pre-contrast images, enhancement characteristics during the portal phase, the level of enhancement during the portal phase, the presence of bowel wall thickening and the segment length of affected bowel were evaluated (for confirmation of abdominal vascular abnormalities). Any mesenteric manifestations of disease, as well as evidence of ascites were also documented. The mesenteric ischemia category was determined by the aggregate of the radiographic findings (14).

Small bowel series and colon studies with barium were also analyzed.

Pathologic Evaluation

Tissue specimens for pathologic evaluation were available, obtained from five patients who underwent surgical resection of the post-ischemic stricture. Tissue samples were reviewed by one pathologist. Another author correlated the pathology identified with the imaging findings.

RESULTS

Clinical Findings for the Eight Patients with Post-Ischemic Stricture

Clinical findings are summarized in Table 1. Six patients (75.0%) had underlying diseases such as diabetes mellitus, hypertension, chronic renal failure including kidney transplantation, cardiac disease and/or a previous history of cancer. The main symptoms attributed to ischemic bowel disease were abdominal pain (patient #3, 4, 6, 7, and 8) and diarrhea (patient #1, 2, 3, and 4). One patient reported hematochezia (patient #2). Vital parameters were stable in all patients at the time of diagnosis with post-ischemic stricture except for one patient (patient #1); patient #1 was found to have low blood pressure (systolic and diastolic blood pressure, 92 and 42 mm Hg, respectively). This patient could not be medically cleared to undergo surgery due to comorbidities; conservative management with antibiotics was felt to be the best treatment plan for this patient. Regarding treatment of ischemic bowel disease, five patients (5/8, 62.5%; patient #3, 4, 6, 7, and 8) received anticoagulation therapy, which included administration of Warfarin and Heparin. Two patients (2/8, 25.0%; patient #1 and 2) were treated with antibiotics. One patient (1/8, 12.5%; patient #5) was treated conservatively, which included nothing by mouth. One patient (patient #3), diagnosed with superior mesenteric artery occlusion on the initial CT scan, underwent exploratory laparotomy. However, no abnormalities associated with the bowel loops were identified on exploratory laparotomy, and bowel loop resection was not performed. This patient was then treated with anticoagulation therapy.

Post-ischemic stricture developed a mean of 57 days (range, 14−144 days; median, 48 days) after bowel ischemia occurred. At the time of diagnosis with post-ischemic stricture of the bowel loops, six patients had abdominal pain (6/8, 75.0%). For treatment of the strictures, five
patients (5/8, 62.5%) underwent surgery and one patient (patient #5) underwent endoscopic balloon dilation. One patient (patient #2), who had been considering surgical intervention because conservative care had proven ineffectual, could not undergo surgery because of a number of comorbidities and the presence of pneumonia.

**Table 1. Clinical Features of Group of Eight Patients with Post-Ischemic Strictures**

| Patient No. | Sex | Age | Underlying Disease | BP (mm Hg) | HR (bpm) | BT (°C) | WBC (10³ mm⁻³) | Symptoms at Time of Stricture Development | Stricture Treatment | Time (Days) *
|-------------|-----|-----|-------------------|------------|----------|---------|----------------|------------------------------------------|-------------------|---------
| 1           | M   | 67  | DM; hypertension; CRF; complete AV block | 92/42      | 79       | 36.2    | 10.7           | LOC during defecation                      | Subtotal colectomy | 98      |
| 2           | F   | 67  | DM; atrial fibrillation; congestive heart failure | 90/64      | 87       | 36.3    | 4.3            | Abdominal pain; diarrhea                   | Antibiotics        | 43      |
| 3           | F   | 63  | Hypertension; atrial fibrillation | 107/66     | 124      | 38.3    | 7.8            | Hypoalbuminemia                            | Conservative treatment | 144     |
| 4           | M   | 67  | Previous glottic cancer; atrial fibrillation; mitral valve replacement; RA and LA thrombi | 151/103    | 103      | 36.0    | 10.3           | Abdominal pain                             | Small bowel R & A  | 15      |
| 5           | M   | 86  | Bile duct resection due to CBD cancer; hypertension; DM | NA         | NA       | NA      | NA             | Post-prandial abdominal pain; constipation  | Balloon dilation   | 57      |
| 6           | M   | 55  | None | NA         | NA       | NA      | NA             | Abdominal pain                             | Small bowel R & A  | 14      |
| 7           | F   | 41  | Kidney transplantation; hypertension | 127/87     | 89       | 37.1    | 6.4            | Abdominal pain; vomiting                    | Small bowel R & A  | 32      |
| 8           | M   | 29  | None | 132/82     | 90       | 37.0    | NA             | Abdominal pain                             | Small bowel R & A  | 53      |

*Time elapsed from ischemic event to stricture development, †Systolic and diastolic blood pressure, ‡At time of ischemic bowel disease. AV = atrioventricular, BP = blood pressure, BT = body temperature, CBD = common bile duct, CRF = chronic renal failure, DM = diabetes mellitus, HR = heart rate, LA = left atrium, LOC = loss of consciousness, NA = not available, RA = right atrium, R & A = resection and anastomosis, WBC = white blood cell count

**Table 2. Radiographic Features of Group of Eight Patients with Post-Ischemic Strictures**

| Patient No. | Site             | Length (cm) | Wall Thickness (mm) | Enhancement of Strictures on CT Scan | Obstruction |
|-------------|------------------|-------------|---------------------|-------------------------------------|-------------|
| 1           | Splenic flexure of colon | 15          | 5                   | Con                                 | Mild Homogeneous Moderate Mucosal High-grade |
| 2           | DC-SC junction   | 9           | 6                   | Con                                 | Moderate Mucosal Moderate Homogeneous Low-grade |
| 3           | Ileum           | 6           | 6                   | Con                                 | Moderate Mucosal Moderate Mucosal Low-grade |
| 4           | Ileum           | 6           | 7                   | Con                                 | Moderate Mucosal Moderate Mucosal High-grade |
| 5           | Ileum           | 12          | 4                   | Non-con                             | NA Marked Mucosal High-grade |
| 6           | Ileum           | 4           | 5                   | Con                                 | Moderate Mucosal Moderate Layered Low-grade |
| 7           | Ileum           | 4           | 6                   | Con                                 | NA Moderate Homogeneous Low-grade |
| 8           | Jejunum         | 2           | 3                   | Con                                 | Moderate Mucosal Moderate Homogeneous High-grade |

Con = concentric, DC = descending colon, SC = sigmoid colon
Radiographic Features of Post-Ischemic Stricture and Prior Ischemic Bowel Disease

CT evidence of post-ischemic stricture are summarized in Table 2. Each patient had one stricture only. The mean length and thickness of the affected bowel was 7.4 ± 4.8 cm and 5.3 ± 1.3 mm, respectively. Most post-ischemic strictures initially developed in the ileum (5/8, 62.5%), followed by the jejunum (1/8, 12.5%), splenic flexure of the colon (1/8, 12.5%) and finally the junction of the descending colon and the sigmoid colon (1/8, 12.5%). Most strictures displayed concentric thickening. For evaluation of post-ischemic strictures, six patients underwent dynamic CT scanning which included both the arterial phase and portal phases. Two patients underwent routine abdomino-pelvic

Fig. 1. 67-year-old man with multiple pre-existing and underlying pathologies (including diabetes mellitus, hypertension, chronic renal failure, and complete AV block) (patient #1) who suffered loss of consciousness during defecation.

A. CT scan performed (at time of diagnosis of ischemic bowel disease) shows some areas of decreased enhancement (arrows) in splenic flexure of colon, which is consistent with ischemic colitis. Patient had diarrhea at that time, and vital parameters were stable. Patient was managed conservatively, with antibiotics. B. Coronal arterial and portal phase images obtained 98 days after ischemic event show better bowel wall enhancement in portal phase than arterial phase (mild homogeneous enhancement in arterial phase and moderate mucosal enhancement in portal phase) (arrows) as well as vasa recta prominence around site of stricture. C. Colon study shows thickened folds (“thumb printing”), which is typical finding in cases of ischemic colitis. Patient underwent subtotal colectomy. D. Gross specimen of resected large bowel reveals approximately ten centimeters long segmental stricture (arrows) with dilatation of proximal bowel segment. This corresponds with CT image findings (arrows in B) and colon study (arrows in C). AV = atrioventricular
CT scans limited to the portal phase. On the arterial phase images (available in six), the strictures visualized displayed mild mucosal enhancement (1/6, 16.7%) or moderate mucosal enhancement (5/6, 83.3%). Of the six strictures with both arterial and portal phase images available, four strictures visualized displayed higher enhancement in portal phase than arterial phase (4/6, 66.6%). On the portal phase images, seven strictures (7/8, 87.5%) visualized displayed moderate enhancement and one (1/8, 12.5%) displayed marked enhancement. Four strictures (4/8, 50%) visualized displayed mucosal enhancement, three (3/8, 37.5%) displayed homogeneous enhancement, and one (1/8, 12.5%) displayed layered enhancement. Vasa recta prominence was appreciated in all patients (8/8). Four patients (4/8, 50.0%) were diagnosed with low-grade obstruction and four patients (4/8, 50.0%) were diagnosed...
high-grade obstruction. One patient, with colonic stricture caused by ischemic colitis (patient #1), had a colon study performed which revealed “thumbprinting,” ulcers, and hastral loss with smooth tapering in the narrow portion of the colon (Fig. 1). Two patients with small bowel strictures (patient #4 and 6) had a small bowel series performed and the studies revealed fold effacement and segmental luminal narrowing in the small bowel loops (Fig. 2).

CT findings of bowel segment and vascular structure at the time of diagnosis with ischemic bowel disease are summarized in Table 3. On CT images obtained at the time of diagnosis with ischemic bowel disease, high attenuation of the bowel wall was seen on pre-contrast images in two patients (2/8, 25%; patient #7 and 8), which is consistent with intra-mural hemorrhage. Images obtained from four patients (4/8, 50.0%) displayed decreased enhancement of the ischemic bowel wall. Images from five patients (5/8, 62.5%) possessed a focal wall defect or a focal decrease in enhancement. Classification by ischemic category resulted in three patients (3/8, 37.5%) being diagnosed with venous occlusion, and four patients (4/8, 50%) diagnosed with non-occlusive mesenteric ischemia (including ischemic colitis). Only one patient (1/8, 12.5%) was diagnosed with arterial occlusion with reperfusion injury. This patient underwent a negative exploratory, because all bowel segments were found to be normal in color. The mean length of the ischemic bowel among all patients was 22.4 ± 12.0 cm.

**Histopathological Findings**

Five patients (5/8, 62.5%) underwent surgical resection of the stricture (patient #1, 4, 6, 7, and 8). Histopathological findings of the tissue samples included deep ulceration, chronic trans-mural inflammation, submucosal and subserosal fibrosis (scarring) and crypt distortion of the mucosa. Three of five specimens (3/5, 60%) showed dilatation of submucosal venules and capillaries (patient #6, 7, and 8). An organizing abscess was identified in one of the patients (patient #4).

**DISCUSSION**

To our knowledge, no study has focused on the radiologic features of post-ischemic strictures. A few cases of post-ischemic stricture have been identified and clinical findings have mainly been described in case reports (4, 8-10, 15). The cases in this study highlight several important aspects of post-ischemic stricture. First, post-ischemic stricture develops in any bowel segment and mainly develop in the watershed zone, particularly in colon. Second, post-ischemic strictures usually developed within several weeks of an ischemic event. Third, post-ischemic strictures developed in one bowel segment only. Fourth, the appearance of a post-ischemic stricture is typically concentric wall thickening of medium length with moderate and higher enhancement in portal phase than arterial phase; vasa recta prominence is also noted.

Most previously reported cases of post-ischemic stricture

| Patient No. | Vessels | Length (cm) | Enhancement of Bowel Wall | Ischemic Category |
|-------------|---------|-------------|---------------------------|-------------------|
| 1           | Moderate atherosclerotic change at aorta and IMA proximal | 20 | Decreased; wall defect | Ischemic colitis (nonocclusive) |
| 2           | Mild atherosclerotic change at aorta | 20 | Decreased | Ischemic colitis (nonocclusive) |
| 3           | Moderate atherosclerotic change at aorta; focal thrombosis at mid SMA | 40 | Mild Mucosal | Arterial occlusion |
| 4           | Mild atherosclerotic change at aorta and SMA | 10 | Moderate Mucosal; focal decreased | Nonocclusive mesenteric ischemia |
| 5           | Moderate atherosclerotic change at aorta and SMA | 14 | Decreased; wall defect | Nonocclusive mesenteric ischemia |
| 6           | IMV obliteration | 10 | Decreased | Venous occlusion |
| 7           | Thrombosis in SMV and portal vein | 25 | Mild Homogeneous; wall defect | Venous occlusion |
| 8           | Thrombosis in SMV and portal vein | 40 | Mild Layered; wall defect | Venous occlusion |

IMA = inferior mesenteric artery, IMV = inferior mesenteric vein, SMA = superior mesenteric artery, SMV = superior mesenteric vein
developed secondary to venous ischemia (9, 16); a few reported cases developed secondary to ischemic enteritis or ischemic colitis (3, 17). In our study, various types of ischemic bowel disease were found to be responsible for the development of post-ischemic stricture. These included non-occlusive ischemia, arterial occlusion, and venous occlusion. Most patients evaluated in previous studies were of advanced age with underlying conditions such as cardiac disease or diabetes mellitus. If the patients were relatively youthful, they were usually found to suffer from coagulation disorders and portal vein or superior mesenteric vein obstruction. As was the case in studies previously undertaken, most of our patients were of relatively advanced chronologic age, and suffered from pre-existing and underlying conditions and illnesses. None, however, suffered from any disease process associated with hypercoagulation. This condition could have been identified in one young patient #8 (diagnosed with superior mesenteric vein thrombosis) (Fig. 3).

Post-ischemic strictures have been reported on less often than post-traumatic ischemic strictures in case reports and in radiology research. Radiographic findings associated with post-ischemic strictures are similar to those that have been reported for post-traumatic ischemic strictures (5, 18-20). The mechanism of injury for the two types of ischemic stricture is similar, in that focal segmental ischemia develops at the mesentery and bowel wall (21). Reported post-traumatic ischemic strictures developed between one and eighteen weeks after trauma; this is similar to the findings in our study. Also similar to the findings in our study, post-traumatic ischemic strictures displayed thickened folds (“thumbprinting”) on barium
study and most occurred as a single stricture measuring 5 to 10 centimeters in length. A few patients with post-traumatic ischemic strictures reportedly had very short (2 cm) and very long (18 cm) strictures, as well as multiple strictures. Most of the post-ischemic strictures evaluated in our study displayed concentric wall thickening (7/8, 87.5%) with high-grade or low-grade obstruction (8/8, 100%), although two previously reported cases revealed no proximal dilatation associated with the colonic post-traumatic stricture (18).

CT findings associated with post-ischemic stricture reflected the underlying pathology. Surgical specimens were available for five patients. Of the four patients for whom arterial and portal phase CT images were available (in addition to tissue samples), three patients (3/4, 75%) showed higher enhancement in portal phase than arterial phase, likely due to fibrosis and submucosal capillary dilatation. The radiographic findings associated with post-ischemic strictures mimicked those of various intestinal diseases such as Crohn’s disease, NSAID-induced enteropathy and/or CMUSE (4). A previous report on strictures associated with Crohn’s disease showed that it was relatively uncommon at the time of diagnosis. The inflammatory stricture showed layered enhancement during the parenchymal phase of MRI, and active inflammatory stenotic lesions were usually seen in combination with fistulas, abscesses, and the comb sign (vasa recta prominence) (22, 23). However, stricture in Crohn’s disease is difficult to differentiate from post-ischemic strictures based on pathologic findings. The pathologic findings in five patients (with surgically resected specimens) in our study were found to be very similar to strictures associated with Crohn’s disease: transmural inflammation, crypt distortion, submucosal fibrosis, and ulcer. There were no non-caseating granulomas associated with post-ischemic stricture in this study; however, more than half of the strictures associated with Crohn’s disease do not contain non-caseating granulomas in the pathologic specimens (24, 25). Clinical and radiological findings could be used to differentiate between post-ischemic strictures and strictures associated with Crohn’s disease. Patients with strictures associated with active Crohn’s disease usually possess terminal ileum involvement, skipped areas, and more than one segment with inflammation (26). Patients with strictures associated with chronic Crohn’s disease usually show other bowel sequelae such as pseudosacculation and antimesenteric border shortening.

Nonsteroidal anti-inflammatory drug-induced enteropathy or CMUSE usually presents as short segmental strictures (diaphragm-like stricture) (27-29). The post-ischemic strictures in our study possessed a longer length than those of the previously mentioned diseases (7.0 ± 3.8 cm), although the concentric wall thickening was similar. Patients with NSAID-induced enteropathy have a history of NSAID use. Most reported CMUSE patients possessed more than two strictures with a history of recurrent strictures.

There were several limitations to our study. First, this study could not completely exclude selection bias due to its retrospective nature. Cases with severe bowel ischemia could not avoid emergency surgery. Conversely, cases without significant obstruction might be overlooked. Second, our analyses included only a small sample of patients. However, post-ischemic stricture is so rare that limited case reports are available. This analysis of radiographic findings associated with post-ischemic strictures could be the beginning of research targeting the differentiation of post-ischemic strictures from other bowel diseases. Third, the CT protocol was heterogeneous in our study. Therefore, there were some limitations on assessment of the enhancement degree and presence of bowel obstruction objectively. Fourth, the frequency with which post-ischemic strictures may occur may be underestimated. According to one previous report of colon ischemia, about 12% of patients with severe colonic ischemia healed with stricture formation. Also, strictures might be reversible or irreversible, and characteristically lead to few obstructive symptoms (30). Therefore, patients with mild symptoms or reversible stricture might not be appropriately diagnosed.

We included only those who had a CT scan at the time of ischemic bowel disease, and had pathologic or endoscopic confirmation of benign stricture of clearly post-ischemic stricture. In addition, those patients who had history of surgery or procedure, which can develop ischemic stricture, were excluded because these cases are not usually difficult to be differentiated from other inflammatory bowel diseases. Our aim is to identify CT findings associated with post-ischemic strictures which might be implemented to differentiate between that disease and other, similar bowel diseases (through patients with definite post-ischemic stricture). Our study does not reflect the exact frequency of post ischemic stricture.

In conclusion, post-ischemic stricture may develop in the ileum, jejunum, or colon within several weeks of an ischemic event. In the colon, strictures mainly develop...
in the watershed zone. Typical CT findings to indicate the condition include a single area of concentric wall thickening of medium length, with moderate and higher contrast enhancement in portal phase than arterial phase and vasa recta prominence.

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