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CT findings in isolated ischemic proctosigmoiditis

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Abstract The purpose of our study was to describe the CT features of ischemic proctosigmoiditis in correlation with clinical, laboratory, endoscopic, and histopathologic findings. Our study included seven patients with isolated ischemic proctosigmoiditis. Patients were identified by a retrospective review of all histopathologic records of colonoscopic biopsies performed during a time period of 4 years. All patients presented with left lower abdominal quadrant pain, bloody stools, and leukocytosis, and four patients had fever at the time of presentation. Four of seven patients suffered from diarrhea, one of seven was constipated and two of seven had normal stool consistency. The CT examinations were reviewed by two authors by consensus and compared with clinical and histopathologic results as well as with the initial CT diagnosis. The CT showed a wall thickening confined to the rectum and sigmoid colon in seven of seven patients, stranding of the pararectal fat in four of seven, and stranding of the perisigmoidal fat in one of seven patients. There were no enlarged lymph nodes, but five of seven patients showed coexistent diverticulosis and in three of these patients CT findings were initially misinterpreted as sigmoid diverticulitis. Endoscopies and histopathologic analyses of endoscopic biopsies confirmed non-transmural ischemic proctosigmoiditis in all patients. Isolated ischemic proctosigmoiditis often presents with unspecific CT features and potentially misleading clinical and laboratory findings. In an elderly patient or a patient with known cardiovascular risk factors the diagnosis of ischemic proctosigmoiditis should be considered when wall thickening confined to the rectum and sigmoid colon is seen that is associated with perirectal fat stranding.

Keywords Computed tomography · Bowel ischemia · Ischemic colitis · Proctosigmoiditis · Diverticulitis · Diverticulosis

Introduction

Ischemic colitis may occur as a result of proximal or distal occlusions of the visceral arteries due to, for example, arteriosclerosis, athero- or thromboembolism, vasculitis, or small vessel disease. Furthermore, colonic overdistension, hematologic, as well as coagulation disorders and certain drugs may also cause colonic ischemia or at least contribute to its pathogenesis jointly with one or more of the aforementioned factors [1, 2]. Occasionally, colonic ischemia may be caused by intrinsic or extrinsic occlusions of the mesenteric veins, but most commonly ischemic colitis occurs without underlying vascular occlusion, when a low-flow-state and subsequent reactive vasospasm of the visceral arteries lead to a critically disturbed colonic underperfusion. The resulting "non-occlusive ischemic colitis" is mostly self limited, does not usually lead to transmural colonic infarction and typical-

ly affects the colon in a segmental fashion with the splenic flexure and the descending colon most commonly involved [1, 2, 3, 4, 5].

However, occasionally, non-occlusive ischemic colitis may affect only short and atypical colonic segments such as the cecum, the ascending colon, or the rectosigmoid [6, 7, 8]. Although it is not surprising that the rectosigmoid may become affected in non-occlusive colonic ischemia, isolated ischemic proctosigmoiditis seems to be very rare [9]. To our knowledge, CT findings of isolated ischemic proctosigmoiditis have not yet been reported in the radiology literature. Therefore, the purpose of our study was to describe the CT features of isolated ischemic proctosigmoiditis in correlation with clinical, laboratory, endoscopic, and pathologic findings, and to discuss this entity as a rare but important differential diagnosis to acute sigmoid diverticulitis.

Materials and methods

Subjects

Our retrospective study included seven patients with isolated ischemic proctosigmoiditis, identified over a time period of 4 years, who were examined by helical CT. Patients were identified by a retrospective review of all histopathologic records of colonoscopic biopsies performed during this time and found positive for colonic ischemia. Patients with ischemic colitis involving the descending colon or the splenic flexure and patients with bowel ischemia involving any other part of the small or large bowel were excluded. Only those patient were included into our study in whom ischemic changes were confined to the rectum and sigmoid colon endoscopically and radiologically. Five of these patients were women and two were men. Their age ranged from 66 to 87 years (mean age 79 years). All patients had a positive medical history of hypertension, atherosclerosis, and coronary artery disease, and all patients had impaired renal function. Three patients had a positive medical history for diabetes mellitus and two of them also suffered from end-stage renal insufficiency. Four patients suffered from a marginally compensated chronic cardiac insufficiency with ejection fractions ranging from 40 to 50% and one of them had experienced a syncope 1 day before his abdominal complaints began. Two patients suffered from arythmogenic supraventricular tachycardia, whereas only one patient had no risk factors for a low-flow state; however, this patient suffered from a thrombotic thrombocytopenic purpura.

Clinical presentation

Three patients were admitted the same day their symptoms began, whereas two patients were admitted 2 days and two patients even 3 days after their symptoms had started. At the time of admission all patients presented with nonspecific clinical findings: All patients suffered from left lower abdominal quadrant pain and rectal bleeding and/or bloody stools. Four patients had mild diarrhea, one had constipation, and two had normal stool consistency. Three patients suffered also from episodes of nausea. Four patients presented with fever ranging from 100°F (=37.8°C) to 102°F (=38.9°C) at the time of admission and all patients showed pathologically elevated white blood cell counts (WBC) ranging from 12,000 to 16,500/mm^3 (mean 12,900/mm^3).

Imaging technique and analysis

All patients underwent CT in order to rule out acute sigmoid diverticulitis, which was initially suspected by the clinicians. The CT scans were performed using a Somatom Plus 4 CT scanner (Siemens, Erlangen, Germany). Since all our patients had impaired renal function, all CT examinations were performed as unenhanced studies only; however, unfortunately no contrast, water, or air was given rectally prior to the CT examinations which were performed using the following parameters: slice thickness 8 mm; table feed 8 mm; and pitch 1. All patients received 500 ml of oral contrast barium suspension, 30 min prior to their examination. The abdominal CT scans were reviewed on hard-film copies by two observers in a consensus fashion. The entire colon was analyzed by the readers and the presence or absence of the following findings was assessed: sigmoidal or rectal wall thickening; stranding of the parasigmoidal or pararectal fat; diverticulosis; enlarged lymph nodes; ascites; abscess formations; pneumoperitoneum; and pneumatosis or portal-venous gas. Finally, CT findings were compared with clinical and laboratory findings, as well as with endoscopic and histopathologic findings, which were obtained within 1–3 days in all patients.

Results

CT findings

All patients showed a rectosigmoidal wall thickening. In three patients it was most pronounced in the sigmoid colon and in the rectosigmoid, whereas the rectum of these patients showed less pronounced changes. The sigmoid colon of these three patients showed a wall thickening ranging from 10 to 12 mm. In one of these patients, there was also some very mild stranding of the parasigmoidal fat, whereas this finding was absent in the other two patients (Figs. 1, 2). The rectum of these three patients showed only a mild wall thickening measuring 5 mm;

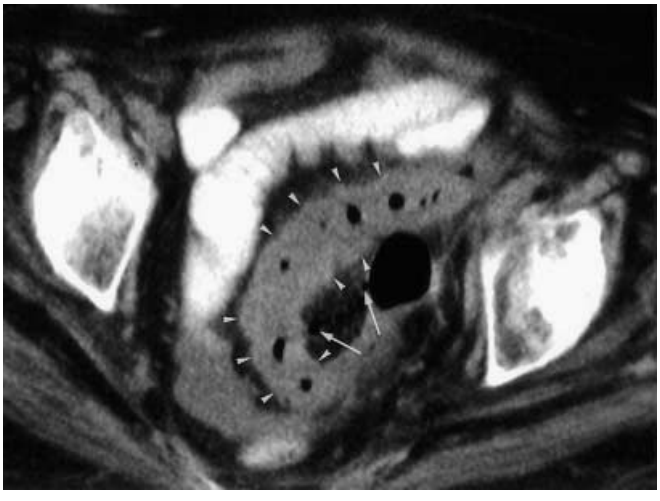

Fig. 1 Unenhanced CT in isolated ischemic proctosigmoiditis shows circumferential wall thickening of the sigmoid colon (*arrowheads*) which may be misinterpreted for sigmoid diverticulitis according to the presence of some diverticula (*arrows*)

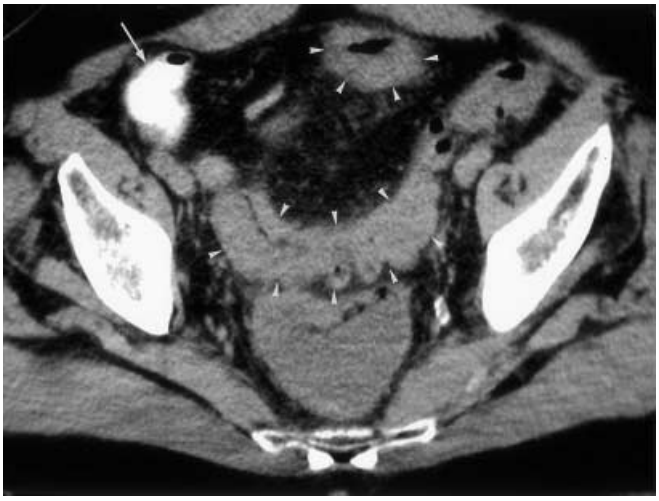

Fig. 2 Unenhanced CT in isolated ischemic proctosigmoiditis shows circumferential wall thickening of the sigmoid colon (*arrowheads*). There is only very mild stranding of the parasigmoidal fat. The cecum appears normal (*arrow*)

however, in two of them there was also stranding of the pararectal fat (Fig. 3).

In one patient, there was moderate wall thickening in the sigmoid colon and rectum, both measuring 6 mm, and mild stranding of the pararectal fat. In the remaining three patients rectosigmoidal wall thickening was most pronounced in the rectum and rectosigmoid, whereas the sigmoid showed only mild and less pronounced changes. The rectal wall of these patients measured from 10 to 12 mm, and in two of these patients there was also pronounced stranding of the pararectal fat (Figs. 4, 5). The sigmoid colon of these three patients showed only a mild wall thickening ranging from 5 to 6 mm (Fig. 6). None of our patients showed ascites, enlarged lymph nodes, abscess formations, pneumatosis, portal-venous gas, or pneumo-peritoneum. The total length of rectal and sigmoid involvement as determined by CT ranged from 10 to 25 cm.

However, only the patient with a thrombotic thrombocytopenic purpura showed a short segment involved (10 cm), whereas the extent of ischemic proctosigmoiditis ranged from 20 to 25 cm (mean 22.5 cm) in the remaining six patients, in whom it was presumably caused by non-occlusive underperfusion. Five patients showed coexistent diverticulosis of their sigmoid colon and in three of them CT findings were initially (prospectively) misinterpreted for acute sigmoid diverticulitis. In the other four patients CT findings were prospectively correctly interpreted as proctosigmoiditis, although initially they seemed more likely to be caused by an infectious etiology than an ischemic one.

Fig. 3a, b Unenhanced CT in isolated ischemic proctosigmoiditis shows circumferential wall thickening of the sigmoid colon (*arrowheads* in **a**). The cecum appears normal (*arrow* in **a**). There is also thickening of rectal wall and stranding of pararectal fat (*arrowheads* in **b**)

Endoscopic and histopathologic findings and clinical outcome

Colonoscopy revealed mucosal edema and erythema, as well as exudates and a friable mucosa involving the rectum and sigmoid in all patients. The extent of ischemic proctosigmoiditis as determined by endoscopy correlated well with the CT findings. The aforementioned endoscopic findings were consistent with non-transmural ischemic colitis and histopathologic analysis of the biopsy specimens, obtained in all patients, confirmed this diagnosis. Infectious proctosigmoiditis was ruled out by stool analysis in all patients. Endoscopy also confirmed the presence of diverticula in five patients, but there were no signs of diverticulitis. Since there was no suspicion of transmural infarction of the rectosigmoid in any one of our patients, surgery was never requested; there-

Fig. 4 Unenhanced CT in isolated ischemic proctosigmoiditis shows circumferential thickening of the rectal wall (*arrowheads*) without stranding of the pararectal fat

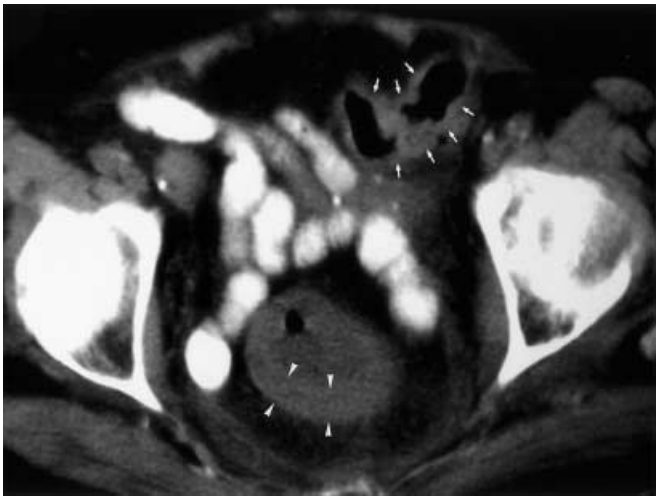

Fig. 6 Unenhanced CT in isolated ischemic proctosigmoiditis shows circumferential thickening of rectal wall (*arrowheads*) as well as mild wall thickening of sigmoid colon (*arrows*)

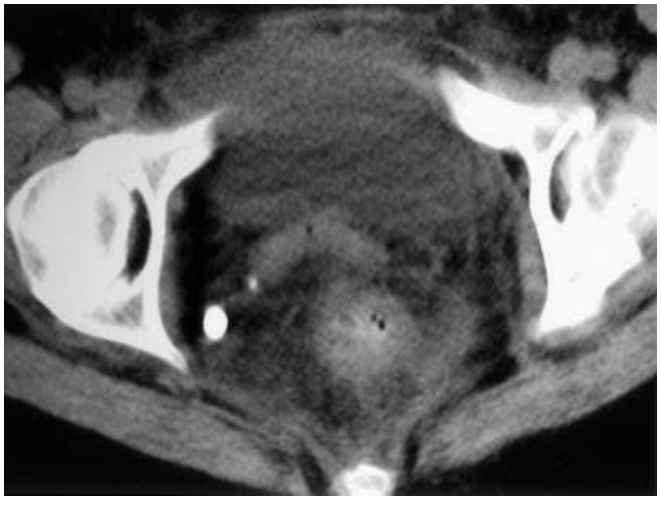

Fig. 5 Unenhanced CT in isolated ischemic proctosigmoiditis shows pronounced stranding of pararectal fat as well as thickening of the rectal wall

fore, all patients were treated conservatively and they all were released in a good condition several days later.

Discussion

Non-occlusive ischemic colitis usually affects the colon in a segmental fashion, with the splenic flexure and the descending colon most commonly involved [2, 5, 10]; therefore, if this particular region of the colon shows a wall thickening at CT, ischemic colitis is usually included in the radiological differential diagnosis; however, if ischemic colitis involves exclusively short and less typical colonic segments, its radiological diagnosis may become challenging, especially in patients with misleading clinical and laboratory findings [7, 8, 9].

The reason why the rectosigmoid may become affected in non-occlusive ischemic colitis is that it represents a watershed area, similarly to the splenic flexure. Although rectosigmoidal ischemia is most commonly associated with ischemic colitis involving the descending colon, ischemic proctosigmoiditis may occasionally occur as an isolated finding [9, 10]. This is interesting, although not surprising, and might be explained on the basis of strong and patent collaterals around the splenic flexure and descending colon, just more pronounced arteriosclerosis of the pelvic arteries than of the superior mesenteric artery, or a combination of these two factors.

Isolated ischemic proctosigmoiditis accounts for only approximately 5% of all cases of ischemic colitis, and it has been reported to occur following abdominal aortic surgery or implantation of aortic stents, secondary to vasculitis, due to myointimal hyperplasia of mesenteric veins and following radiotherapy [1, 9, 11, 12, 13, 14]. Nevertheless, isolated ischemic proctosigmoiditis is most commonly related to non-occlusive conditions and this is supported by our findings, where six of seven patients had at least one reason for a low flow state and subsequent non-occlusive rectosigmoidal ischemia.

The clinical presentation of patients with ischemic proctosigmoiditis may be unspecific and misleading since they often present with left lower abdominal quadrant pain, bloody stools, elevated white blood cell counts, or fever; the latter is not surprising, since it is well known that in ischemic colitis the disrupture of the mucosal barrier may lead to inflow of intraluminal bacte-

ria and toxins from the gut. As a consequence and since ischemic proctosigmoiditis usually occurs in elderly patients, the constellation of the aforementioned clinical and laboratory findings may easily evoke the clinical suspicion of acute sigmoid diverticulitis and, therefore, many of these patients are examined by CT.

The CT findings of ischemic colitis have extensively been described in the radiology literature, and although they are not absolutely specific, they often allow determination of the correct radiological diagnosis in an appropriate clinical setting [10]. The CT findings in ischemic colitis consist of hyper- or hypoattenuating bowel wall thickening, paracolic stranding, absence of bowel wall enhancement, or pneumatosis intestinalis, with predominant involvement of splenic flexure and descending colon [10, 15]. However, if the CT findings in ischemic colitis consist predominantly of a wall thickening confined to the sigmoid colon and/or rectum, and if more specific CT findings, such as pneumatosis intestinalis, are missing, several other differential diagnoses must also be taken into consideration including sigmoid diverticulitis, infectious or ulcerative proctitis/proctosigmoiditis, and sigmoid carcinoma [15].

Proctosigmoiditis can be distinguished from sigmoid carcinoma on CT according to the fact that the latter typically shows a shorter segment involved, a finding which also allows differentiation between sigmoid carcinoma and sigmoid diverticulitis by CT. Since the length of bowel wall involvement in proctosigmoiditis is even higher than in sigmoid diverticulitis, and since, in contrast to sigmoid carcinoma, ischemic proctosigmoiditis does not show associated enlarged lymph nodes, its radiological differentiation from sigmoid carcinoma may be even more reliable [16].

Contrarily, CT differentiation between proctosigmoiditis and sigmoid diverticulitis may become more challenging if proctosigmoiditis presents mainly with a thickening of the sigmoid colon, rather than with a rectal wall thickening, and especially if the patient shows coexistent diverticulosis. Fortunately, ischemic sigmoiditis is most commonly associated with ischemic proctitis (i.e., proctosigmoiditis) and, therefore, it is the presence of a rectal wall thickening and/or stranding of the pararectal fat that will allow to differentiate proctosigmoiditis from acute sigmoid diverticulitis by CT. The fact that in contrast to acute sigmoid diverticulitis stranding of the perisigmoidal fat seems to be uncommon in ischemic proctosigmoiditis may also help to distinguish these two entities, although perisigmoidal fat stranding in ischemic sigmoiditis/proctosigmoiditis will be absent only as long as more pronounced or even transmural infarction of the sigmoid and/or superinfection has not occured.

Surely, it may theoretically happen that only the sigmoid or only the rectum is involved by infection, inflammation or ischemia. Therefore, differentiation between isolated ischemic sigmoiditis and acute sigmoid diverticulitis may become impossible by CT if there is more pronounced ischemia of the sigmoid colon with perisigmoidal fat stranding and associated diverticulosis. Furthermore, radiological differentiation between ischemic, infectious or ulcerative proctitis or proctosigmoiditis will also be impossible on the basis of CT features alone. However, according to the fact that ischemic colitis has been reported to be the most common form of colitis in patients aged over 50 years, an advanced age of patients and the presence of known hematologic and/or cardiovascular risk factors will clearly favor ischemia in such cases, rather than an infectious or ulcerative etiology, which would be more typical for a younger patient population [17, 18].

Conclusion

In elderly patients or patients with known hematologic or cardiovascular risk factors a diagnosis of isolated ischemic proctosigmoiditis should be considered if wall thickening confined to the rectum and sigmoid is seen in association with perirectal fat stranding. This may initiate an early endoscopic examination of affected patients and avoid an unnecessarily aggressive surgical approach to this rare condition, which requires surgery only in exceptional cases with transmural rectosigmoidal infarction.

References

1. Haglund U, Bergqvist D (1999) Intestinal ischemia: the basics. Langenbecks Arch Surg 384:233–238
2. Longo WE, Ballantyne GH, Gusberg RJ (1992) Ischemic colitis: patterns and prognosis. Dis Colon Rectum 35: 726–730
3. Reilly PM, Bulkley GB (1993) Vasoactive mediators and splanchnic perfusion. Crit Care Med 21:55–68
4. Fisher DF Jr, Fry W (1988) Collateral mesenteric circulation. Surg Gynecol Obstet 164:487–492
5. Wiesner W, Willi UV (2001) Nonocclusive ischemic colitis in a 12-year-old girl: value of unenhanced spiral computed tomography. Int J Colorectal Dis 16:55–57
6. Rist CB, Watts JC, Lucas RJ (1984) Isolated ischemic necrosis of the cecum in patients with chronic heart disease. Dis Colon Rectum 27:548–551
7. Landreneau RJ, Fry WJ (1990) The right colon as a target organ of nonocclusive mesenteric ischemia. Arch Surg 125:591–594
8. Yilmaz EN, Vahl AC, van Rij GL, Vink GQ, Lange-De Klerk ES, Brom HL, Rauwerda JA (1999) The renin-angiotensin system in swine during hypovolaemic shock combined with low-flow ischaemia of the sigmoid colon. Cardiovasc Surg 7:539–544

9. Bharucha AE, Tremaine WJ, Johnson CD, Batts KP (1996) Ischemic proctosigmoiditis. Am J Gastroenterol 91: 2305–2309
10. Balthazar EJ, Yen BC, Gordon RB (1999) Ischemic colitis: CT evaluation of 54 cases. Radiology 211:381–388
11. Jaeger HJ, Mathias KD, Gissler HM, Neumann G, Walther LD (1999) Rectum and sigmoid colon necrosis due to cholesterol embolization after implantation of an aortic stent-graft. J Vasc Interv Radiol 10:751–755
12. Okada M, Konishi F, Sakuma K, Kanazawa K, Koiwai H, Kaizaki Y (1999) Perforation of the sigmoid colon with ischemic change due to polyarteritis nodosa. J Gastroenterol 34: 400–404
13. Savoie LM, Abrams AV (1999) Refractory proctosigmoiditis caused by myointimal hyperplasia of mesenteric veins: report of a case. Dis Colon Rectum 42:1093–1096
14. Norotsky MC, Shackford SR (1992) Rectosigmoid ischemia following blunt abdominal trauma in a patient treated with radiation therapy: case report. J Trauma 33:931–932
15. Horton KM, Corl FM, Fishman EK (2000) CT evaluation of the colon: inflammatory disease. Radiographics 20: 399–418
16. Chintapalli KN, Chopra S, Ghiatas AA, Esola CC, Fields SF, Dodd GD III (1999) Diverticulitis versus colon cancer: differentiation with helical CT findings. Radiology 210:429–435
17. Marston A, Pheils MT, Thomas ML, Morson BC (1966) Ischaemic colitis. Gut 7:1–15
18. Brandt L, Boley S, Goldberg L, Mitsudo S, Berman A (1981) Colitis in the elderly. Am J Gastroenterol 76:239– 245