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CASE REPORT

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Isolated cecal necrosis: Report of two cases

Nizar Kardoun[1,2] | **Zied Hadrich**[1,2] | **Daoud Rahma**[1,2] | **Houssem Harbi**[1,2] | **Salah Boujelben**[1,2] | **Rafik Mzali**[1,2]

[1]Departement of surgery, Habib Bourguiba Hospital, Sfax, Tunisia

[2]Faculty of médecine of Sfax, Sfax, Tunisia

Correspondance
Zied Hadrich, Departement of Surgery, Habib Bourguiba Hospital, Sfax, Tunisia.
Email: hadrich1988@gmail.com

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Abstract

Isolated cecal necrosis is a rare variant of ischemic colitis. Diagnosis is difficult because of nonspecific clinical and radiological findings. It especially affects patients with comorbidities affecting mesenteric perfusion.

KEYWORDS

cecum, emergency, ischemia, necrosis

1 | INTRODUCTION

Isolated cecal necrosis (ICN) is rare, and it typically simulates the presentation of acute appendicitis.[1] It is a rare cause of surgical abdomen frequently seen in the elderly population.[2] These patients may have one or more accompanying diseases. Poor mesenteric perfusion, due either to systemic hypotension (heart failure and chronic renal failure at the top of the list) or to specific pharmacologic agents or drugs, is considered to play a role in the development of ICN.[3] Preoperative diagnosis is difficult because of nonspecific clinical and radiologic findings. The diagnosis of ICN is mostly established on intraoperative findings.[3,4]

We report two cases of isolated cecal necrosis, which were preoperatively diagnosed, in two female patients with a common previous history of hypertension and renal failure.

2 | CASES REPORT

2.1 | Case 1

A 78-year-old woman with a previous history of renal failure, hypertension, dyslipidemia, diabetes mellitus, and atrial fibrillation on acenocoumarol and digoxin presented to the emergency department suffering from an acute abdominal pain, which had gradually migrated to the right iliac fossa. She presented with fever (38°). Cardiopulmonary examination was normal. Abdominal examination revealed tenderness of the right lower abdomen without signs of peritoneal irritation. Laboratory data showed leukocytosis (WBC = 14100/mm) and elevated C-reactive protein (CRP=38 mg/d). The serum creatinine level was 245 µmol/L, and blood urea nitrogen was 29 mmol/L.

A computed tomography (CT) scan of the abdomen and pelvis without contrast showed an image of a dilated cecum with mural thickening, edema, and intramural gas (pneumatosis), while the appendix was intact. Portal venous gas and mesenteric gas as signs of severity were also found (Figure 1), though no perforation or collection was found on the CT scan. The diagnosis of cecal ischemia was suspected. An urgent midline laparotomy was performed.

During laparotomy, an isolated transmural necrosis of the cecum was found. The appendix and the terminal ileum were normal. The cecal examination did not show any evidence of malignancy (Figure 2). The mesenteric vessels were pulsating. The colon, small intestine, abdominal, and pelvic organs were normal. Slight hemorrhagic fluid was found in

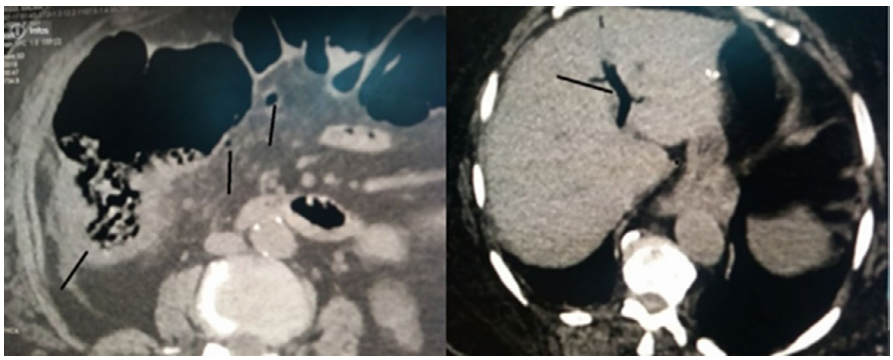

FIGURE 1 Dilated cecum with mural thickening and portal venous gas and mesenteric vessels gas

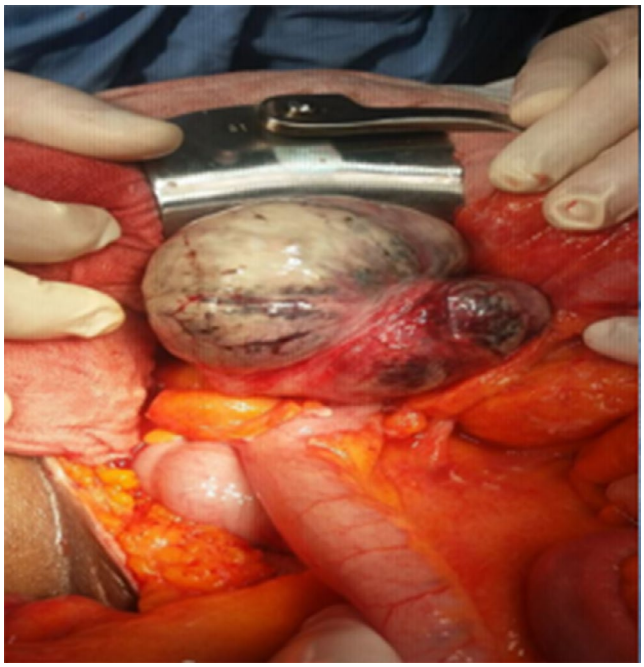

FIGURE 2 Intraoperative imaging

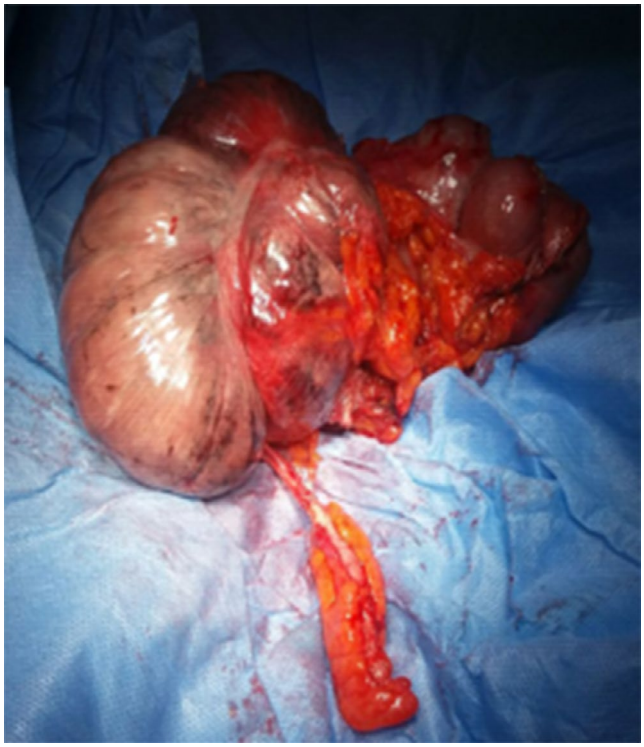

FIGURE 3 Operative specimen

the abdominal cavity, and a warm saline washout was performed. The patient underwent a resection of the cecum (Figure 3) and side-to-side anastomosis using a linear stapler.

A single drain was left in situ. The patient's postoperative course was uneventful, and she was discharged on the tenth postoperative day.

Pathological findings confirmed the transmural ischemic cecum.

A colonoscopy was performed secondarily to check remnant colon, excluding ischemic colitis, or neoplasia.

2.2 | Case 2

A 66-year-old woman with a history of hypertension, diabetes mellitus, coronary artery disease, and renal failure consulted the emergency department for an acute central abdominal pain, which migrated to the right iliac fossa and vomiting. Abdominal examination revealed tenderness in the right iliac fossa without peritoneal signs. Laboratory data showed leukocytosis (WBC 11600/mm3) with elevated C-reactive protein (CRP 143 mg/dL).

A computed tomography (CT) image revealed a cecum surrounded by free air, while the appendix was normal (Figure 4). The preoperative diagnosis was a perforation of the cecum. An urgent surgery was performed.

Via a midline laparotomy, we found a gangrenous lateral cecal wall. The appendix was normal (Figure 5). We performed an ileocecal resection with a double-barrel ileocolostomy in the right iliac fossa.

The specimen revealed localized ischemic area on the antimesenteric side of the cecum. Microscopically, the transmural ischemia was confirmed (Figure 6). The patient's

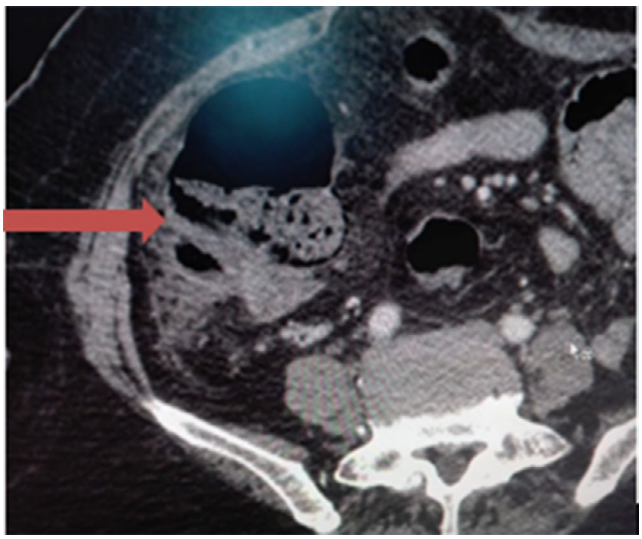

FIGURE 4 Cecum surrounded by free air

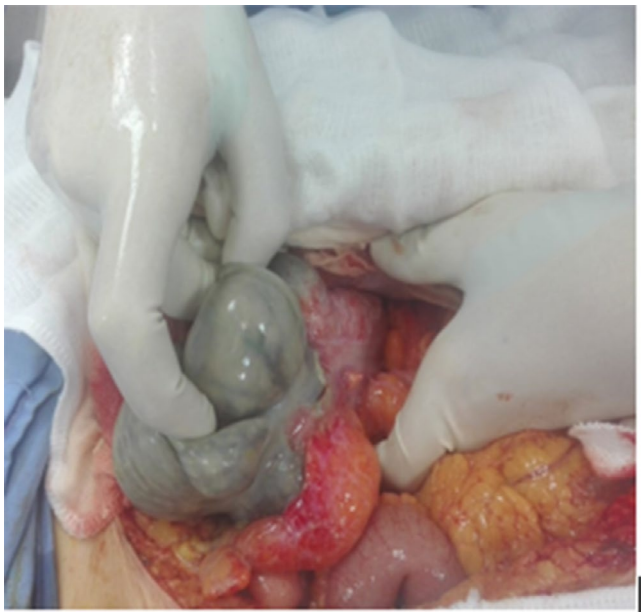

FIGURE 5 Intraoperative imaging

postoperative course was uneventful, and she was discharged on the sixth postoperative day.

3 | DISCUSSION

Isolated cecal necrosis is a rare cause of acute surgical abdomen. Few cases have been reported in the literature and are all case presentations.[2] The largest series is of Gundes E et al, published in 2013 and reported a single-center experience between 1995 and 2011 at Necmettin Erbakan University Meram Medical School's General Surgery Clinic (Turkey).[5] It is frequently seen in the elderly, especially in women with comorbidities.[1,5]

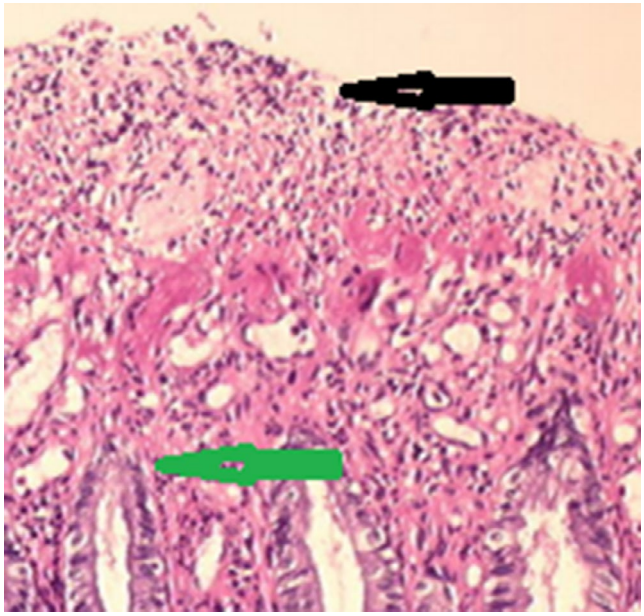

FIGURE 6 Pathological examination. Surface epithelium detached (black arrow); Atrophy of crypts (green arrow)

The most important factor for developing ICN is a presence of the comorbidity, which causes decreased mesenteric perfusion.[1,4] ICN is divided into two groups according to the presence of comorbidities:

Type I (or spontaneous): no identifiable cause of decrease in mesenteric blood flow can be established. Some associated morbidities are usually found, including congestive cardiac failure, ischemic heart disease (Case 2), diabetes, and hypertension (Cases 1 and 2). In some other cases, it can be attributed to drugs such as thiopentone, cocaine, vincristine, prednisolone, ergot, or glypressin.[1,3,4]

Type II (or secondary): decrease in mesenteric flow is identifiable, such as following cardiopulmonary bypass or resuscitation and hypotension in patients undergoing hemodialysis for renal failure. [1,4]

Our two patients could be classified as Type I because they did not have an apparent episode of systemic hypotension.

Another factor in developing ICN is the presence of a variation in cecal blood supply. The anterior and posterior cecal arteries mainly supply the cecum. These arteries often arise from the vascular arcade between the ileal branch and colic branch of the ileocolic artery, while in the others, these arteries arise directly from the ileal or colic branch.[2,4] If this arcade is absent, the cecal blood supply is considered deficient. In addition, the vasa recta supplying the cecum are the longest because this segment of bowel has the widest diameter, which makes the cecum vulnerable to ischemia [4]

Cecal necrosis on acute colonic pseudo-obstruction, known as Ogilvie syndrome, is a distinct entity. As the cecum and ascending colon dilate and the luminal diameter increases, wall tension in the colonic mucosa increases

proportionately. This increased wall tension leads to colonic ischemia, fluid, and bacterial translocation, and eventually cecal perforation.

In contrast to ischemic colitis and colonic infarction, which usually affect the colon in a segmental fashion with the left colon most commonly involved, isolated cecal infarction represents an uncommon and less well-known entity. It may present a diagnostic challenge, as it is an unusual and rather atypical presentation of acute colonic ischemia. Cecal infarction typically presents with right lower abdominal quadrant pain, and it may be associated with leukocytosis or even fever.[6] Therefore, diagnosis is difficult because patients present with right-sided abdominal pain and tenderness suggesting appendicitis, cecal diverticulitis, stercoral perforation, or cecal carcinoma. Nowadays, there is no specific serum marker for colonic ischemia.

CT shows nonspecific findings, although cecal wall thickening with isolated pneumatosis coli is strongly suggestive of the diagnosis.[6,7] Cecal infarction typically presents as isolated low-attenuating circumferential cecal wall thickening.[4] Depending on the severity of involvement, there may also be some pericecal inflammation, stranding of the adjacent mesenteric soft tissue, or ascites. If such abnormalities involve the region of the appendix, radiologic differentiation from acute appendicitis with cecal involvement becomes more difficult, but as long as the findings concern only the cecum, the correct diagnosis may be suspected radiologically. Furthermore, CT findings in such cases may rarely resemble neoplastic changes, but the acute clinical presentation of patients with cecal infarction will usually allow differentiation of this condition from, for instance, cecal tumor.

Diagnostic laparoscopy is considered as a useful option to make a definitive diagnosis and to implement a surgical strategy, which includes incision type.[4,8]

The use of colonoscopy in patients with suspected ischemic colitis is controversial. Bradbury et al. cautioned that colonoscopy may decrease colonic perfusion because of increased transmural pressure.[3,9] We did not perform colonoscopy in any of our two patients.

Resection of a necrotic bowel segment is the main objective of the surgical treatment. Cecal resection or right hemicolectomy are the options to choose from according to the extent of necrosis in the cecum and evidence of peritonitis.[8,10]

Diagnostic laparoscopy may be useful for diagnosis and treatment. Based on the results of diagnostic laparoscopy, we can choose the appropriate approach.

If the clinical history and examination lead to suspicion of cecal necrosis, surgery should be performed immediately. Laparoscopy (outside contraindications to Laparoscopy) or a midline abdominal incision must be made to allow exploration of all of the intra-abdominal organs and intestine.

4 | CONCLUSION

Isolated ischemic cecal necrosis is an infrequent variant of ischemic colitis. The diagnosis should be considered when an elderly patient presents with right lower quadrant pain, particularly if cecal wall thickening is noted on abdominal CT scan. If evidence of peritonitis exists, right hemicolectomy with anastomosis can be performed with satisfactory results.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Z Hadrich: conceived the idea for the document and contributed to the writing and editing of the manuscript. N Kardoun: contributed to the writing and editing of the manuscript. R Daoud: reviewed and edited the manuscript. H Harbi: reviewed and edited the manuscript. S Boujelben: contributed to the literature review, manuscript writing, editing, and review of the manuscript. All authors read and approved the final manuscript.

ETHICAL STATEMENT

Personal data have been respected.

DATA AVAILABILITY STATEMENT

Personal data of the patient were respected. No data are available for this submission.

ORCID

Zied Hadrich https://orcid.org/0000-0002-5048-3755

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