

Isolated Cecal Necrosis Mimicking a Perforated Peptic Ulcer: A Case Report

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Abstract Isolated cecal necrosis is a rare surgical emergency. In most cases the clinical picture resembles an acute appendicitis, usually occurring in elderly patients who have associated diseases. We believe that this is the first published case of isolated cecal gangrene, mimicking a perforated ulcer. A previously healthy 82-year-old male presented with abdominal pain, stabbing in nature, and of 12-hours duration. The clinical signs and symptoms were consistent with a perforated peptic ulcer. A plain abdominal X-ray disclosed a significant pneumoperitoneum. An emergency laparotomy released a gas under pressure revealing an isolated gangrenous cecum. Perforation of the cecum and fecal soiling were not present. A right hemicolectomy was performed. An intra-operative hemodynamic instability precluded primary anastomosis and a double barrel ileocolostomy was created. The patient was discharged in good health on the 8th postoperative day. This paper presents an original case of isolated cecal necrosis. A high index of suspicion should be maintained and the condition promptly addressed. Any delay is associated with high mortality rate. The procedure of choice is ileocecal resection or right hemicolectomy. The intestinal continuity should be restored through a primary anastomosis unless stump blood supply or peritoneal contamination, or patient's overall condition are specific concerns.

Keywords: *cecal, necrosis, isolated, perforation, ulcer*

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1. Introduction

Ischemic colitis (IC) is the most common form of intestinal ischemia. The left colon is the usual location and rarely involves a full-thickness necrosis. The cecum is less affected but when it does occur, it usually progresses to gangrene [1]. Isolated cecal necrosis (ICN) is predominantly observed in elderly patients with comorbidities. The clinical presentation usually resembles acute appendicitis, making diagnosis of ICN a challenge. Colonoscopy is not applicable when bowel gangrene is suspected. Instead, rapid noninvasive imaging tests of the abdomen such as plain X-ray, ultrasound, and computed tomography, are required. Interestingly, none of these examinations reveal findings specific for colonic necrosis. Therefore, the diagnosis is generally determined during surgery.

To understand why the cecum is vulnerable to ischemia, it is necessary to review its blood supply. Most patients with ICN have no evidence of major vessel occlusion with the condition attributed to a low-flow state, small vessel disease, or both [2]. Rist CB *et al* stated that the cecum, like splenic flexure and rectosigmoid colon, is a "watershed area" with poor blood supply relative to that of the adjacent intestines [3]. The basis for this theory is that the cecum is supplied by the anterior and posterior cecal arteries. These terminal arteries are fed by either the colic,

or ileal branch of the ileocolic artery. Rare anatomical variations can occur: the cecal arteries can arise from an arc between the ileocolic rami; additionally, an accessory appendicular vessel may be present. The latter is known as "the artery of Seshachalam" and branches from the posterior cecal artery. In the absence of such variabilities, which is generally the case, the cecal blood supply is relatively insufficient compared to adjacent bowel segments, which are double-blood supplied [2]. Other anatomical factors that may predispose cecum to nonocclusive ischemia are: (a) absent or insufficient collateral blood flow through the marginal artery [4], (b) long vasa recta with poor collateral blood flow between them [5], (c) the colonic microvasculature plexus is less well developed compared with the small intestines [6].

Human bowel microcirculation is unique in possessing precapillary sphincters and metarterioles. This anatomical feature is sensitive to low flow states, shunting blood circulation. This in turn places the bowels at the risk to ischemic injury [7].

The PubMed Central and Google Scholar online database were browsed to identify English language reports on ICN. Diastatic necrosis and perforations of the cecum due to distal obstruction were excluded. Seventy two reports on post-chemotherapy neutropenic typhlitis were explored but not included in the research analysis. The search revealed 32 publications with 79 cases of ICN. Clinical presentation in 71 cases (89%) was that of acute

appendicitis or right iliac fossa pain; 5 cases (6%) presented as cecal cancer with a mass; 1 case (1%) resembled ileocecal abscess; and 2 patients (3%) had periumbilical or low abdominal pain. No patient in this research presented with a pneumoperitoneum and clinical picture of a perforated peptic ulcer. Therefore, we concluded that our case is without similar reference in the English language literature. Table 1 provides the more specific details of our search (Supporting information 1: Table 1).

2. Case Report

A 82-year-old male presented with a vague abdominal pain of 10 days duration. Prescribed spasmodic tablets provided initial relief until a sudden occurrence of debilitating epigastric pain 12 hours before admission. The pain was constant and quickly spread to the abdomen as whole. His past medical history was insignificant except for chronic constipation. Breath sound were clear but diminished bibasally. The abdomen was bloated, with tenderness, and rebound tenderness. Guarding was throughout, but most pronounced periumbilically and at the right lower quadrant. Bowel sounds were absent, with a rectal examination revealing no abnormalities.

Vital signs on admission were: pulse, 100 min; blood pressure, 100/60; respiratory rate, 25 min; oxygen saturation on air, 94%; axillary temperature, 37.6°C.

Urine output was normal. Blood workups showed leukocytosis of 15.8 thousand/ml, blood sugar of 8.7 mmol/L and mild hypoproteinaemia of 52.8 g/L. A plain thoraco-abdominal X-ray in a supine position revealed a massive pneumoperitoneum with elevation of the diaphragmatic domes (Figure 1).

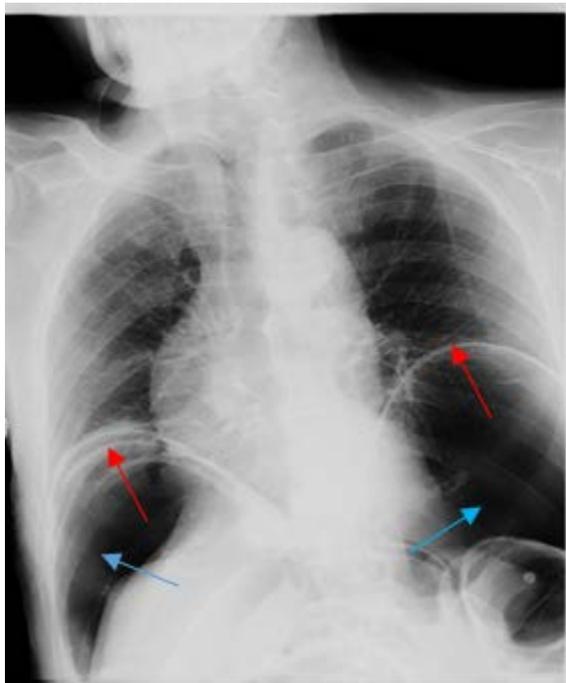


Figure 1. The X-ray on admission (the patient is slightly turned on to his right-hand side)

- Large amount of free intraperitoneal gas (blue arrows).

- Highly elevated diaphragmatic domes (red arrows). The right dome is blurred due to the patient breathing during the exposition.

The patient received a short course of I.V. fluid resuscitation and broad-spectrum antibiotics. This was immediately followed by an upper midline laparotomy which released pressurized gas. The abdomen was found clean with no perforation of the stomach or the duodenum. The small bowels looked normal. The incision was extended downwards and the abdomen thoroughly explored. The complete cecum was found to be necrotic, sharply defined from the terminal ileum, appendix, and ascending colon, all of which appeared normal. The cecum walls were edematous, greenish in discoloration, and collapsed. Cecal perforation was not found and there was no fecal soiling of the peritoneal cavity. Small amount of cloudy peritoneal exudate was found in the rectovesical pouch; a sample taken cultured *Escherichia coli*. There was no evidence of major vascular occlusion. Palpation of the remainder of the colon revealed fecal loading but no distal obstruction. No other abnormalities were found.

A right hemicolectomy was performed. During the operation, the patient became hemodynamically unstable and was started on a Dopamin infusion. The blood supply to the colonic stump was questionable and a “brooked” double barrel ileocolostomy performed (Figure 2). The patient made an uneventful recovery and was discharged healthy 8 days after admission.



Figure 2. The patient's front abdominal wall on the fifth post-operative day (picture taken from the patient's right side).

- The everted (“brooked”) double-barrel ileocolostomy situated to the right of the extended upper midline incision:
 - the ileal end (blue arrow).
 - the colonic mucus fistula (red arrow).

Histological examination of the removed bowel segment revealed hemorrhagic ulceration of the cecal mucosa with transmural necrosis. There was no evidence of vasculitis or cholesterol emboli. The appendix, terminal ileum and ascending colon were intact.

The patient had no complaints at the 1- and 6-month follow-up, reporting a normal appetite with a 2 kg gain in weight. The abdominal wall and ileocolostomy were healthy, with no irritation to the surrounding skin. Abdominal ultrasound revealed no abnormalities and blood tests were normal. After explaining the benefits and

risks of stoma reversal, the patient decided to stay with permanent ileostomy.

3. Discussion

The patient displayed signs, symptoms, and radiological findings consistent with a perforated peptic ulcer. Hindsight revealed that this was a clinical mimicry of ICN. The procedure of choice in this situation is ileocecal resection or right hemicolectomy with primary ileocolic anastomosis. Our patient became hemodynamically unstable during the second half of the operation and the colonic stump showed signs of ischemia. This precluded primary anastomosis because of high risk of anastomotic leakage.

Two questions arise concerning the present case: (1) *Why was the massive necrosis limited to the cecum?* Referring to the discussed blood supply to the cecum, other aspects may have contributed to the nonocclusive ischemic insult: age-related microvascular disease and colonic perfusion affected by the patient straining from constipation. The latter exerts pressure on the cecal veins reducing the arteriovenous oxygen gradient in the colonic wall [8]. Chronic constipation was found in one study to be strongly associated with ischemic colitis [9].

(2) *How did gas leave the cecum in the absence of a perforation?* Ischemic injury affects the wall bowel from the inside out. Mucosa ceases to be a reliable barrier against bacteria, toxins, and cytokines. Consequently, they invade bowel wall and blood circulation, a process referred to as translocation [10]. We speculate that the evolving full-thickness necrosis created weak micro-spots through which colonic gas rapidly entered the peritoneal cavity before the development of macro-perforation.

We were unable to reach a preoperative diagnosis which resulted in choosing an inappropriate abdominal incision. The patient had a clear need for emergency surgery and any delay for additional imaging tests could have been detrimental [11]. Moreover, such tests would not have altered our management plan.

Most individuals with ICN, as in this case, present days after the initial ischemic insult. In a series of 36 patients with colonic ischemia it was proven that full-thickness gangrene was fatal in 71% of the patients, while mucosal necrosis only associated with an 88% survival rate [12]. Therefore, an increased awareness of this rare entity and earlier operative intervention would improve the results of treatment.

4. Conclusions

Our case of ICN did not present a management challenge. Namely because the clinical picture was indistinguishable from a perforated peptic ulcer. It is of note, that the majority of patients display vague symptoms before development of transmural necrosis. Ignorance of this entity, an ambiguous preoperative diagnosis, with intrinsic caution of operating on a fragile patient, may result in a delayed treatment with an unfavorable outcome. Therefore, a high relevance in patients with certain

comorbid conditions and prompt surgical intervention are the key steps towards successful outcomes.

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Statement of Competing Interests

The authors have no competing interests.

List of Abbreviations

IC: ischemic colitis; ICN: isolated cecal necrosis; CT: computed tomography.

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Supporting Information 1

Table 1. Literature search of cases of isolated cecal necrosis reported in the English language literature

| Authors, Year, ^(ref) | Case No | Age | Associated disease | Preoperative diagnosis | Major vascular occlusion | Clinical presentation |
|--|---------|---------------|--|---------------------------|--|--|
| Carnevale, N. <i>et al</i> , 1973 ⁽¹⁾ | 1 | - | Hypertension, renal failure | no | No; Cholesterol micro embolization | Acute appendicitis |
| Hakami, M, 1976 ⁽²⁾ | 2 | 8;11 | Acute rheumatic arthritis | no | no | Acute appendicitis |
| Hargrove, W.C. <i>et al</i> , 1978 ⁽³⁾ | 2 | 65;65 | Open heart surgery | 1 no; 1 yes (bariumenema) | no | Acute appendicitis |
| Varki, A.P. <i>et al</i> , ⁽⁴⁾ (Plus 72 similar publications on Post-chemotherapy Neutropenic Typhlitis) | 1 | 48 | Acute leukemia | no | no | Right iliac fossa pain |
| Gerlock, A.J. <i>et al</i> ,1981 ⁽⁵⁾ | 1 | 53 | Vascular embolization | yes | Yes (ileocolic artery) | Right iliac fossa pain |
| Rist, C.B. <i>et al</i> ,1984 ⁽⁶⁾ | 3 | 59;74;89 | Chronic heart failure | no | no | Acute appendicitis |
| Hubens, A. <i>et al</i> ,1987 ⁽⁷⁾ | 3 | 62;75;76 | Atherosclerotic vascular disease | no | No; Spontaneous cholesterol micro embolization | Acute appendicitis |
| Chan, T. <i>et al</i> ,1988 ⁽⁸⁾ | 1 | 59 | Atherosclerotic vascular disease; cerebral angiography | no | No; Iatrogenic cholesterol micro embolization | Cecal cancer |
| Ter Borg, F. <i>et al</i> ,1990 ⁽⁹⁾ | 1 | 27 | Mucormycosis; Acute leukemia | no | no | Right iliac fossa pain and a mass |
| Pashkova, V.S. <i>et al</i> ,1992 ⁽¹⁰⁾ | 1 | - | Myeloma with amyloidosis | no | no | Acute appendicitis |
| Borra, S. <i>et al</i> ,1995 ⁽¹¹⁾ | 3 | 61;65;69 | End-stage renal disease | no | no | Acute appendicitis |
| Elnakadi, I. <i>et al</i> ,1998 ⁽¹²⁾ | 1 | 54 | Mucormycosis; Acute leukemia | no | no | Acute appendicitis |
| Schuler, J.G. <i>et al</i> ,2000 ⁽¹³⁾ | 5 | 57-91 | Hypertension;Diabetes; Coronary artery disease; | no | no | Acute appendicitis, Cecal cancer, Viscus perforation |
| Simon, A.M. <i>et al</i> ,2000 ⁽¹⁴⁾ | 2 | 65;67 | Hypertension; Diabetes; Coronary artery disease; | yes (CT) | no | Acute appendicitis |
| Wiesner, W. <i>et al</i> ,2002 ⁽¹⁵⁾ | 1 | 58 | End-stage renal disease; Diabetes mellitus | yes (CT) | no | Acute appendicitis |
| Hasana, S. <i>et al</i> ,2004 ⁽¹⁶⁾ | 3 | 71 | Open heart surgery | no | no | Acute appendicitis |
| Perko, Z. <i>et al</i> ,2006 ⁽¹⁷⁾ | 1 | 73 | Hypertension | no | no | Acute appendicitis |
| Watanabe, T., <i>et al</i> ,2006 ⁽¹⁸⁾ | 1 | 71 | Cerebral infarction | no | no | Right iliac fossa abscess |
| Baig, W.W. <i>et al</i> ,2008 ⁽¹⁹⁾ | 1 | - | Mixed fungal infection; End-stage renal disease | yes (colonoscopy) | no | Right iliac fossa mass |
| Kiyak, G. <i>et al</i> ,2008 ⁽²⁰⁾ | 1 | 61 | Hypertension;Diabetes; Coronary artery disease; | no | no | Acute appendicitis |
| Ruiz-Tovar, J. <i>et al</i> ,2008 ⁽²¹⁾ | 1 | 82 | Hypertension;Diabetes; | no | no | Cecal cancer |
| Trinca, K.D. <i>et al</i> ,2008 ⁽²²⁾ | 1 | 58 | Cocaine and crack abuse | no | no | Acute appendicitis |
| Dirican, A. <i>et al</i> ,2009 ⁽²³⁾ | 4 | 46-68 | End-stage renal disease COPD | no | no | Acute appendicitis |
| Hennawy, H.M. <i>et al</i> ,2009 ⁽²⁴⁾ | 1 | 57 | Hypertension | yes (angio-CT) | yes (ileocolic artery) | Periumbilical pain |
| Xiaofeng, P. <i>et al</i> ,2011 ⁽²⁵⁾ | 12 | mean age 68.1 | Hypertension; Diabetes; Coronary artery disease; | no | no | 8 Ac.appendicitis 4 Lower abdominal pain |
| Manipadam, J.M. <i>et al</i> ,2012 ⁽²⁶⁾ | 1 | 29 | T-cell lymphoma | no | no | Lower abdominal pain |
| Botaa, B. <i>et al</i> ,2013 ⁽²⁷⁾ | 1 | 9 | Healthy child (the only known case) | no | no | Acute appendicitis |
| Gundes, E. <i>et al</i> ,2013 ⁽²⁸⁾ | 13 | 51-84 | Chronic heart failure End-stage renal disease | no | no | Right iliac fossa pain |
| Hunter, J.P. <i>et al</i> ,2013 ⁽²⁹⁾ | 1 | - | - | - | - | Acute appendicitis |
| Karpuzi, A. <i>et al</i> ,2014 ⁽³⁰⁾ | 1 | 84 | Chronic heart failure | no | no | Acute appendicitis |
| Çakar, E. <i>et al</i> ,2014 ⁽³¹⁾ | 6 | 35-80 | End-stage renal disease; Chronic heart failure; Aorto-femoral bypass | no | no | Acute appendicitis |
| Özer, E. <i>et al</i> ,2015 ⁽³²⁾ | 1 | 86 | Trichiniasis | no | no | Acute appendicitis |
| This case | 1 | 82 | No associated diseases | no | no | Peptic ulcer perforation with pneumoperitoneum |

*CT: computed tomography

Searched electronic databases: PubMed Central and Google Scholar.

See below for the reference list of this search.

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