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Ischemic bowel disease characterized by colorectal mucosal necrosis induced by cardiac arrest

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Keywords: Bowel ischemia. Cardiac arrest. Colorectal mucosal necrosis.

Dear Editor,

The patient was a 60-year-old male. He developed impaired consciousness after drinking alcohol more than 20 days ago and was found 12 hours later to have an altered level of consciousness with vomiting. The vomit was brown in color. Cardiopulmonary resuscitation (CPR) was performed immediately after calling the emergency number (120) and finding the patient in respiratory and cardiac arrest. After 20 minutes of resuscitation, the patient regained supraventricular rhythm but remained in a deep coma. He was transported to the hospital for treatment and was admitted to the intensive care unit (ICU) after endotracheal intubation and CPR. Initial laboratory tests revealed leukocytosis (WBC 17.02 × 10^9/L), markedly elevated inflammatory markers (calcitoninogen 38.6 ng/mL, C-reactive protein 138 mg/L), acute kidney injury (serum creatinine 386 μmol/L), hyperkalemia (6.2 mmol/L), and coagulation dysfunction (D-dimer 85 μg/mL). Due to sepsis, acute renal failure,

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hyperprolactinemia and electrolyte imbalance, the patient was started on bedside continuous renal replacement therapy (CRRT). During hospitalization, the patient developed recurrent bloody stools with gradually increasing frequency and volume. Colonoscopy revealed rectal mucosal detachment and bleeding. His medical history included hypertension and diabetes mellitus.

The patient was admitted to the hospital for further treatment and underwent another colonoscopy, which revealed multiple colorectal ulcers and mucosal detachment. Histopathologic examination revealed lymphocytic infiltration within the lamina propria and inflammatory granulation tissue formation at the lesion sites. After treatment with blood transfusion and intestinal mucosal repair, follow-up colonoscopy showed multiple colorectal ulcers in the healing phase.

Discussion

Systemic ischemia following cardiac arrest compromises intestinal perfusion through compensatory mesenteric vasoconstriction, exacerbating mucosal damage due to the bowel's high metabolic demands [1, 2]. This manifests clinically as ischemic colitis, characterized by abdominal pain and hematochezia. While the splenic flexure - a vascular watershed zone - typically demonstrates greatest susceptibility to ischemic injury (presenting with acute left upper quadrant pain, early bloody stools, and high mortality requiring surgical intervention), rectal ischemia in this case predominated, featuring tenesmus, mucoid bloody stools, and lower abdominal discomfort with better prognosis[3, 4]. This atypical distribution pattern (rectal vs. splenic flexure involvement) may stem from individual vascular anatomical variations, underlying microvascular pathology from chronic hypertension/diabetes, or post-resuscitation hemodynamic redistribution favoring proximal colonic perfusion. Although mucosal healing was achieved through current therapeutic interventions, sustained surveillance remains crucial for monitoring functional recovery and delayed complications.

Conflicts of interest: The authors declare no conflict of interest.

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Author contributions: Zhi HX and Chen JY designed the study. Zhi HX drafted the manuscript. Wang LS revised the manuscript. Yao J and Song Y performed the operation. All authors have read and approved the final manuscript.

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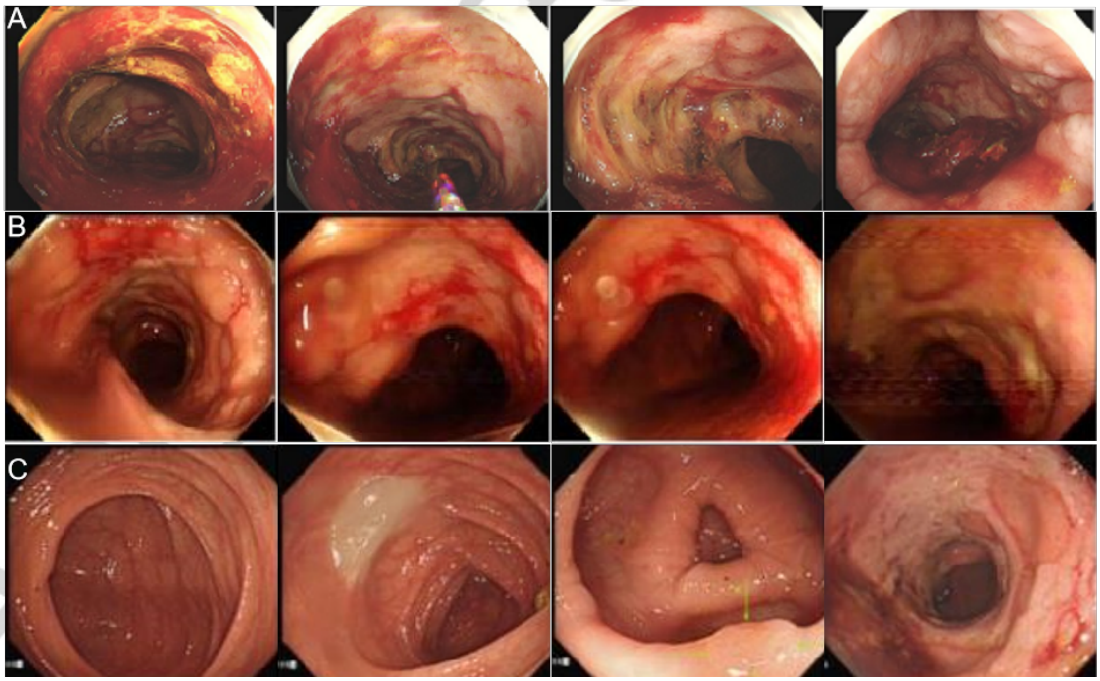

Fig.1 A.First colonoscopy shows rectal mucosal detachment and bleeding B.Second follow-up colonoscopy shows multiple colorectal ulcers and mucosal peeling C.The final review of colonoscopy indicates the healing period of ischemic bowel disease

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