



Colon Ischemia: An Update for Clinicians

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CME Activity

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Abstract

Colon ischemia (CI) is an underrecognized entity associated with high morbidity and mortality. Establishing the diagnosis and initiating appropriate and timely treatment is critical for improving outcomes. Colon ischemia is a disease spectrum that requires a full understanding for recognition and treatment. This review outlines the full spectrum of CI management from initial presentation to medical and surgical treatment.

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Colon ischemia (CI), as opposed to acute mesenteric ischemia (AMI) that affects the small bowel, is the most common type of mesenteric ischemia.¹ Colon ischemia is the cause of 15% of all patients hospitalized for acute lower gastrointestinal bleeding, predominantly affecting the elderly.² *Colon ischemia* can be defined as a decrease in blood flow to a level insufficient to maintain colonocyte metabolic function. It has a spectrum of manifestations including reversible colopathy (submucosal or intramural hemorrhage or edema), transient

colitis evidenced by mucosal ulceration, chronic colitis, stricture, gangrene, and fulminant universal colitis. Colon ischemia has become the preferred terminology rather than ischemic colitis, as some patients do not have a documented inflammatory phase.³ Colon ischemia is occurring more often, with a recent population-based study finding the incidence to be 16 cases per 100,000 person-years, which is a 4-fold increase over the past 34 years.⁴ It is more common in women than in men,⁴ with mortality rates ranging from 4% to 12%.⁴⁻⁶

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Despite the increasing incidence of CI, a timely diagnosis remains a challenge for clinicians. A high index of suspicion and prompt management are important for improving outcomes. This diagnostic challenge is highlighted by a study performed in an emergency department, which found that only 10% of patients with CI presenting with abdominal pain and bloody diarrhea are correctly diagnosed at the time of the presentation.⁷ Proposed reasons for this low initial diagnostic rate include the broad differential diagnosis, the nonspecific presenting symptoms in patients with multiple coexisting medical conditions, and difficulty in identifying an inciting or precipitating cause of CI. The etiology, clinical manifestations, diagnosis, and treatment of CI will be reviewed, with emphasis on a systematic evidence-based approach to management, incorporating the latest clinical guidelines³ and most recent research.

ETIOLOGY AND RISK FACTORS

The 3 main mechanisms responsible for CI include nonocclusive CI, embolic and thrombotic arterial occlusion, and mesenteric venous thrombosis (MVT). Nonocclusive CI, caused by hypoperfusion of the mesenteric microvasculature, is by far the most common mechanism, occurring in 95% of patients.³ This type of ischemia is usually most prominent at the “watershed” areas (ie, splenic flexure and rectosigmoid junction)⁸; however, any segment of the colon can be affected.⁵ The rectum is uncommonly involved because of a dual blood supply from both splanchnic and systemic arterial systems.⁹ Typically, nonocclusive CI is transient; however, prolonged severe ischemia causes necrosis of the mucosal layer with potential for transmural infarction.¹⁰ Colonic injury is related to both the hypoxic component during the episode of decreased blood flow and the sequelae of reperfusion, which is mainly seen after partial ischemia. Here, reperfusion results in the release of oxygen free radicals and other toxic by-products.¹¹ Less commonly, CI can result from arterial thromboemboli or from MVT, which almost always involves the proximal colon.¹²

Colon ischemia typically occurs in well-defined clinical settings, particularly in patients with vascular risk factors. These include diabetes mellitus, coronary artery disease, and peripheral vascular disease.^{4,13} However, it can also occur

without identifiable risk factors. The heterogeneous risk factors for CI support a multifactorial pathogenesis and emphasize the importance of a careful assessment of the medical, surgical, and medication/drug use history in every patient with CI. Irritable bowel syndrome (IBS), constipation, and surgical procedures such as abdominal aortic aneurysm repair are known risk factors.¹⁴⁻¹⁶ Interestingly, patients with IBS exhibited increased ischemic hypersensitivity as compared with the general population, which may account for the disproportionate number of patients with IBS presenting for medical evaluation of CI.¹⁷ Eliciting a thorough medication history is important, especially focused on constipation-inducing medications (eg, opioids), immunomodulators (eg, azathioprine and type I interferons), and illicit drugs (eg, cocaine).¹⁸⁻²⁰ In addition, neuromodulating and vasoconstricting medications, such as quetiapine and rizatriptan, are recognized precipitants of CI.^{21,22}

Evaluation of thrombophilia as a cause of CI should be considered in young patients and all patients with recurrent CI.^{23,24} The degree to which acquired or hereditary hypercoagulable states contribute to the pathogenesis of CI is not well understood. However, the prevalence of antiphospholipid antibodies, plasminogen activator inhibitors, and factor V Leiden sequence variations are increased in patients with CI.²⁴

CLINICAL MANIFESTATIONS

The clinical manifestations of CI vary depending on the extent and duration of ischemia. Most patients have self-limiting, nongangrenous ischemia, which typically resolves completely.²⁵ However, approximately 10% of patients develop colonic necrosis and gangrene, which can be life-threatening.²⁶ These patients have a more protracted hospital course and tend to develop long-term complications, such as chronic ischemic colitis or strictures.

Patients with CI typically present with abrupt, cramping abdominal pain of mild to moderate severity that often affects the left side of the abdomen. This is often accompanied by an urgent desire to defecate and the subsequent passage of bloody diarrhea within 24 hours.^{5,6} Colon ischemia should be considered when the presenting symptoms are abdominal pain and bloody diarrhea.^{5,6} Compared with ischemia affecting the small intestine,

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