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An Overview on Ischemic Colitis Diagnostic & Management Approach

Galwy Abdulrahman Kurkuman^{1*}, Hassan Amer A Alsaluli¹, Omar Ahmed Mohammed Alshehri², Mansour Abdullah M. Alsuayri³, Saad Haif Saeed Algahtani⁴, Khalid Siraj Saad Altalhiyyah⁴, Saeed Abdullah Alahmari⁴, Bader Abdulaziz Alyousef⁵, Felwah Mohammed Yamani⁶

> ¹Faculty of Medicine, Bisha University, Bisha, KSA. ²Vascular Surgery Department, Asser central hospital, Asser, KSA. ³King Abdullah Hospital, Bisha, KSA. ⁴Faculty of Medicine, King Khalid University, Abha, KSA. ⁵Royal College of Surgeons, Dublin, Ireland. ⁶Security Forces Hospital, Dammam, KSA.

ABSTRACT

Ischemic colitis (IC) is the most frequent form of intestinal ischemia, affecting mostly older people with a reported incidence of 16 cases per 100,000 person-years. It is caused by a sudden cut in the blood flow to the colon that compromises its perfusion and functionality. It is transient in most cases and resolves without sequelae, whereas some patients develop colonic necrosis and gangrene that can be life-threatening. Therefore, a high index of suspicion, prompt diagnosis, and treatment are vital in the management of ischemic colitis. We aimed to review the literature to enhance the understanding and awareness of ischemic colitis. We reviewed the literature for ischemic colitis; clinical manifestations, risk factors, and approach to diagnosis. Articles were chosen from the PubMed database, and selected studies were subjected to a thorough review. The clinical features of ischemic colitis differ depending on the clinical circumstances as well as the intensity and duration of the ischemia. A diagnosis of ischemic colitis can often be established clinically based upon history, physical examination, and clinical setting. Moreover, it can be confirmed by a lower endoscopy and abdominal computed tomography (CT) which is beneficial for ruling out other etiologies of abdominal pain.

Keywords: Ischemic colitis, Thumbprinting, Phlebosclerotic colitis, Clinical features, Diagnosis

Corresponding author: Galwy Abdulrahman Kurkuman

e-mail ⊠ Galwwy99@gmail.com Received: 22 December 2020 Accepted: 27 March 2021

INTRODUCTION

Ischemic colitis (IC) is the most common type of intestinal ischemia, targeting mostly older people (Higgins et al., 2004). The incidence of ischemic colitis is reported to be 16 cases per 100,000 person-years, and it has been rising over time (Yadav et al., 2015). It is highlighted by a drop in blood flow to an extent that is not enough to meet the metabolic needs of oxygen and nutrients for cellular metabolism. Most of the patients experience brief, non-gangrenous ischemia that goes away without adverse consequences. Colonic necrosis and gangrene can occur in some people, both of which can be fatal. Prolonged segmental colitis and the formation of stricture are long-term consequences. Several conditions predispose the patient to this disease; however, it can also strike without warning and a clear reason. Because ischemic colitis frequently occurs in people who are debilitated and have several medical conditions, diagnosing and treating it can be problematic (Brandt et al., 2015). In this review, we highlight the risk factors, clinical picture, and diagnosis of ischemic colitis.

MATERIALS AND METHODS

PubMed database had been utilized for the selection process of relevant articles, and the following keys used in the mesh (("Ischemic Colitis"[Mesh]) AND ("Clinical Features"[Mesh]) AND ("Risk Factors"[Mesh] AND "Diagnosis"[Mesh])). For the inclusion criteria, the articles were selected based on including one of the following: ischemic colitis's clinical features, risk factors, and diagnosis. Exclusion criteria were all other articles that did not meet the criteria by not having any of the inclusion criteria results in their topic.

Review

Risk factors

Ischemic colitis is caused by an abrupt, although typically brief, decrease in blood flow that is most noticeable in the "watershed" areas of the colon, where collateral blood flow is restricted. Ischemic colitis is most commonly seen in patients with risk factors for mesenteric ischemia in well-defined clinical situations. Nevertheless, Ischemic colitis can occur without any obvious risk factors. Mentioned below are the most important risk factors for colonic ischemia, which older age individuals most likely tend to have (Chang *et al.*, 2008).

Myocardial infarction: IC appears to be linked to myocardial infarction. One study had reported IC in 14 of 100 patients having myocardial infarction and undergone a colonoscopy within 15 days. In most cases, overt or occult bleeding was the reason for a colonoscopy (Cappell, 2004). According to one study, IC caused by a myocardial infarction was linked to greater complications and a worse in-hospital prognosis than IC caused by other causes (Cappell *et al.*, 2006).

Hemodialysis: IC in patients undergoing hemodialysis is usually non-occlusive and caused by diabetes and hemodialysis-induced hypotension (Bender et al., 1995; Flobert et al., 2000).

Drugs: immunomodulators, illicit drugs, and drugs that cause constipation may be best described as culprit agents. The mechanism could be linked to the culprit drug's recognized action or effect. Unless the cause of their ischemia was tied to another condition, patients with past IC should generally avoid any involved drug (Brandt *et al.*, 2015).

Instrumentation A of aortoiliac or surgery: aortic surgery, such as the repair of a ruptured abdominal aortic aneurysm; other types of aortoiliac reconstruction, such as endovascular therapies; and aortic catheterization can all cause IC (Perry *et al.*, 2008; Moszkowicz *et al.*, 2013).

Cardiopulmonary bypass: Ischemic colitis following cardiopulmonary bypass affects fewer than 0.2 percent of patients, however, it is a fatal complication with an 85 percent fatality rate (Tsiotos et al., 1994). Older age, end-stage renal disease, valve surgery, emergency bypass surgery, and inadequate postoperative cardiac output are all risk factors (Allen et al., 1992).

Extreme exercise: Colonic ischemia has been linked to extreme exertion, such as marathon running. Shunting of blood flow away from the splanchnic circulation, together with dehydration, heat, and electrolyte abnormalities such as hyponatremia and hypokalemia are thought to be the cause of ischemia (Brandt *et al.*, 2015).

Hypercoagulability: It is unclear how much acquired or genetic hypercoagulable conditions have a role in the pathophysiology of IC. In other research, the stated prevalence of thrombophilic abnormalities (e.g., antiphospholipid antibodies, factor V Leiden) is higher (Moszkowicz et al., 2013; Tsimperidis et al., 2015).

Clinical presentation

The clinical picture of ischemic colitis varies depending on the clinical situation, as well as the ischemia's onset, duration, and degree. While carefully reviewing the medical and surgical history, including medicines and other drug usages in each

patient, it is critical to evaluate the patient for risk factors linked with ischemic colitis. IC can be transient or persistent. Acute features include colonic edema or bleeding, diarrhea, ischemic ulceration extensive enough as to induce stricture, pancolitis, colonic gangrene, or sepsis, and can vary from mild to severe (Moszkowicz *et al.*, 2013; Cotter *et al.*, 2016). unconscious Individuals, such as those in an ICU, or who are cognitively disabled, such as those with delirium or dementia, may be found difficult to recognize symptoms in (Dorudi & Lamont, 1992). According to a systematic review, the absence of rectal bleeding, peritonitis, or renal impairment predicted low severity, while right-sided colitis was the most significant predictor of severe illness (O'neill & Yalamarthi, 2012).

Acute ischemic colitis

Acute presentation of IC is characterized by the fast development of moderate abdominal cramping and tenderness across the afflicted intestine, which most commonly affects the left side (Longstreth & Yao, 2009). The discomfort may be linked to a strong need to defecate. Unlike ischemia of the small intestine, IC causes cramping discomfort that is felt laterally rather than around the umbilicus and is frequently linked with hematochezia (Longstreth & Yao, 2009; Brandt *et al.*, 2010). Rectal bleeding (bright or maroon blood) or bleeding without pain is also inevitable however, bloody diarrhea generally begins within 24 hours of the start of abdominal pain. IC of the left colon may cause greater bleeding than IC of the right colon (Montoro *et al.*, 2011). Three phases of clinical progression have been identified (Feuerstadt & Brandt, 2010):

Hyperactive phase: Severe pain and frequent passing of bloody, loose stools characterize the early stages of occlusion or hypoperfusion. Blood loss is generally little and does not necessitate a transfusion.

Paralytic phase: The pain normally lessens, becomes more constant, and diffuse during the paralytic phase. The abdomen tends to be more tender and distended without bowel sounds.

Shock phase: A damaged, gangrenous mucosa begins to leak massive amounts of fluid, protein, and electrolytes. Dehydration, shock, and metabolic acidosis may occur, necessitating immediate surgical intervention. Luckily, only 10 to 20% of people are affected by this most severe type.

Laboratory studies: Complete blood count, metabolic panel, and coagulation investigations are all commonly acquired during the initial examination of a patient with abdominal discomfort or gastrointestinal bleeding. Although they are not diagnostic for colonic ischemia, they can help with disease severity evaluation (Theodoropoulou & Koutroubakis, 2008). Even though elevated blood lactate, lactate dehydrogenase (LDH), creatine phosphokinase (CPK), or amylase may suggest severe tissue injury, there are no particular laboratory indicators for ischemia. Hemoglobin levels that are lower than normal might indicate intestinal blood loss. In a patient with signs and symptoms of IC, a white blood count of more than 20,000 cells/microliter and metabolic acidosis are strongly indicative of IC with infarction(Montoro et al., 2011). To rule out an infectious cause of bloody diarrhea, fecal polymerase chain

reaction tests for pathogens should be conducted. Clostridioides difficile infection seldom causes bloody diarrhea, however, C. difficile superinfection can occur when IC is established (Theodoropoulou & Koutroubakis, 2008).

Plain abdominal radiography: A nonspecific plain abdominal radiograph is commonly acquired in the assessment of abdominal discomfort. Only advanced ischemia is associated with distension or pneumatosis. In a limited percentage of individuals with mesenteric infarction, signs such as thumbprinting (indicating submucosal edema) and bleeding might be seen. Nevertheless, radiographic findings showing ischemia, when present, may indicate a poorer prognosis than those without such findings (Smerud et al., 1990).

Chronic ischemic colitis

Recurrent abdominal discomfort, bloody diarrhea, weight loss from protein-losing enteropathy, periodic bacteremia, prolonged sepsis, or symptomatic colonic strictures are all symptoms of chronic persistent colonic ischemia. Chronic ischemic colitis affects around 20% of people who have recurrent ischemia (Center *et al.*, 1998). Chronic ischemia can lead to segmental ulcerating colitis or strictures, which usually appear three to six months after the onset of symptoms. Colonoscopy may be necessary to confirm chronic colitis or stricture in symptomatic lesions. It is important to keep an eye on ischemic strictures that do not cause any symptoms. Without treatment, few strictures will dissolve in 12 to 24 months. If partial blockage symptoms appear, segmental resection is recommended (Kim *et al.*, 2017).

Long-distance runners may develop chronic ischemic colitis, which manifests as lower-abdominal discomfort, diarrhea, and minor bleeding. Rehydration and repair of metabolic imbalances are generally adequate treatments (Moses, 2005). Another variant that causes chronic IC is Phlebosclerotic colitis. It is an uncommon kind of ischemic colitis characterized by venous blockage due to fibrotic sclerosis and calcification of the mesenteric veins' walls. It usually implicates the right colon, and its symptoms normally go away on their own (Jan & Yang, 2008).

Diagnostic approach

Patients with any of the risk factors listed above, as well as lower abdomen discomfort, bloody diarrhea, or hematochezia, should be suspected of colonic ischemia; however, these symptoms are vague. IC is frequently suspected based on the patient's history, physical examination, and clinical situation. When imaging is possible, it is usually validated with computed tomography (CT) scan of the abdomen (Montoro *et al.*, 2011). A conclusive diagnosis must be determined in the operating room for patients who come with fulminant gangrenous colonic ischemia, peritonitis, and/or colon perforation. Lower endoscopy (sigmoidoscopy or colonoscopy) is the best diagnostic procedure for identifying colonic ischemia and distinguishing it from other causes of abdominal pain and bloody stools in certain acute situations and for people with persistent symptoms (Park *et al.*, 2007; Montoro *et al.*, 2011).

Abdominal imaging

The initial imaging examination conducted in patients presenting promptly with signs of intestinal ischemia is usually a CT scan of the abdomen with intravenous contrast. CT results are not always specific, and scans may appear normal at first. Edema and a segmental pattern of thickening of the gut wall are common observations ("thumbprinting" or "target" or "double-halo" appearance). Rather than continuous ischemia, these alterations usually reflect the first occurrence of transient ischemia and subsequent reperfusion damage. However, they are not exclusive to ischemia and can also be present in infectious colitis caused by C. difficile or Crohn's disease (Alpern et al., 1988). Other CT scan abnormalities comprise abnormal gut outlines, mesenteric inflammation with fat stranding, and free peritoneal fluid (Romano et al., 2007).

Nonetheless, CT can distinguish colonic ischemia from nonischemic causes of abdominal discomfort, or it may signify permanent transmural infarction, which necessitates colon removal. In the latter stages of colonic ischemia, pneumatosis coli or pneumoperitoneum suggesting perforation may be detected, however, these symptoms are not exclusive to colonic ischemia (Menke, 2010; Milone et al., 2013). Hepatic portal venous gas is an uncommon radiographic finding that has been linked to bowel necrosis, especially in individuals who have other clinical and radiological signs of necrosis. In benign conditions, however, pneumatosis coli and portal venous gas can also be observed (e.g., post-surgical or endoscopic manipulation) (Nelson et al., 2009). A large arterial embolic or venous blockage more compatible with acute mesenteric ischemia may be seen using standard CT imaging. CT angiography may be a better first imaging investigation if a significant arterial blockage is suspected (Menke, 2010).

Lower endoscopy

A colonoscopy (full or restricted) confirms the diagnosis of colonic ischemia and, if feasible, should be done in all patients suspected of having IC. Moreover, to minimize severe distention that might lead to colon perforation, lower endoscopy should be done with little air insufflation. If colonic ischemia is suspected, the test should be done sooner rather than later (within 48 hours of initial presentation), but not in patients who have acute peritonitis on physical examination or imaging investigations that show permanent ischemic damage (Houe *et al.*, 2010).

Colonoscopy is effective in detecting mucosal lesions, allowing biopsy of questionable regions while avoiding interfering with later arteriography. Colonoscopy can reveal ischemic colitis; however, it cannot distinguish between transmural and clinically insignificant mucosal ischemia (Longo et al., 1996; Houe et al., 2010). Edematous, friable mucosa, erythema, and intermittent pale patches are common colonoscopic findings in the acute context (Zou et al., 2009). Submucosal hemorrhage is represented by bluish hemorrhagic nodules, which are similar to "thumbprints" found on radiologic investigations. Cyanotic mucosa with scattered hemorrhagic erosions or linear ulcerations are signs of more severe Pseudomembranous colitis with yellowish circular plaques or confluent membranes is seen in a small percentage of individuals and is not related to C. difficile infection (Dignan & Greenson, 1997). The segmental distribution, quick transition between damaged and non-injured mucosa, and rectal sparing all point to ischemia rather than inflammatory bowel illness. The "single-stripe sign," which is a single linear ulcer running

down the colon's longitudinal axis, may also point to an ischemic etiology of colitis (Zuckerman *et al.*, 2003).

Biopsies performed from afflicted regions may reveal nonspecific alterations such as bleeding, granulation tissue, and capillary thrombosis, with crypt destruction, and pseudopolyps, crypt abscesses, all of which might be mistaken for Crohn's disease. Mucosal atrophy and patches of granulation tissue can be detected in the chronic phase of ischemic colitis. Extensive transmural fibrosis and mucosal atrophy can be seen in a postischemic stricture biopsy (Price, 1990; Mitsudo & Brandt, 1992).

CONCLUSION

Ischemic colitis has different clinical manifestations depending on the clinical situation as well as the extent and duration of the ischemia. Of notice, suspected cases of ischemic colitis should be in patients with risk factors for colonic ischemia, lower abdominal discomfort, and/or blood per rectum. Ischemic colitis is frequently diagnosed clinically based on the history, physical examination, and clinical context. A diagnosis of ischemic colitis can be confirmed by a lower endoscopy, most often a colonoscopy. Abdominal computed tomography (CT) is beneficial for ruling out other causes of abdominal pain.

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