Ischemic Colitis Following Abdominal Aortic Aneurysm Surgery

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Ischemic colitis (IC) following abdominal aortic aneurysm surgery has been well-reported in the literature as a highly lethal complication. The overall incidence of IC is between 1 and 7%, the incidence being highest following repair of ruptured abdominal aortic aneurysms. Mortality rates with this complication have been shown to be as high as 54%. With such a high incidence and mortality rate, prevention, early detection and treatment are of significant importance in the management of this complication. This paper discusses primary and secondary prevention of IC following abdominal aortic aneurysm surgery. Primary prevention is targeted at the preoperative and intraoperative levels. Preoperative prevention involves looking at the preoperative risk factors, both anatomic and hemodynamic subgroups. Several techniques of intraoperative detection of IC reported in the literature are also discussed. Secondary prevention involves early detection of IC and management in the postoperative period to allow for institution of supportive measures. Important early warning signs of postoperative IC, as well as the reported use of flexible sigmoidoscopy to reliably predict and diagnose the complication, are addressed.

INTRODUCTION

Ischemic colitis (IC) following abdominal aortic aneurysm (AAA) repair is a serious complication associated with a high mortality. It most frequently involves the sigmoid colon (40%), while the descending colon (26%), rectum (12%), transverse colon (6%), ascending colon (9%) and small bowel (9%) are less frequently involved (1). AAA repair often requires division of a patent inferior mesenteric artery (IMA), and this is the most important factor in the development of postoperative IC (2). Ligation of a patent IMA is usually safe (3). However, some patients do not have adequate collateral circulation from the superior mesenteric artery (SMA) or hypogastric arteries to maintain perfusion to the left side of the colon (2,3). Assessment of collateral blood flow intraoperatively may appear to be adequate at the time, but may not be sufficient in the postoperative period when the patient has hypovolemia, hypotension, or a low cardiac output.

INCIDENCE AND MORTALITY

The overall incidence of clinically evident postoperative IC has been between 1% and 7% (1 to 2% detected clinically; 7% detected by routine colonoscopy) (1,2,4). Intestinal ischemia may occur after abdominal aortic reconstruction for occlusive disease, but is more likely to occur after repair of ruptured aneurysmal disease, presumably because of lesser development of mesenteric collateral circulation and associated systemic hypertension. The incidence of transmural colonic infarction following repair of ruptured AAAs is approximately 7% in those who survive the repair, with colonic ischemia detected in approximately 60% of such cases (4). Whatever the incidence, the mortality rate of IC in patients who have survived AAA repair is very high. In a recent report of veterans from the United States who developed such a complication, the overall mortality was 54% (5). Two-thirds of these patients required a colectomy and in this subgroup the mortality rate was 89% (5).

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PREOPERATIVE PREVENTION

Numerous preoperative risk factors have been proposed to be associated with an increased risk of postoperative IC. These can be divided into three groups: anatomic, hemodynamic, and other (1).

Anatomic

Factors such as a history of prior colectomy (causing collateral interruption); evidence of celiac, SMA, hypogastric, or mesenteric artery occlusive disease; presence and extent of an aneurysm; and the presence of a concomitant retroperitoneal hematoma compressing the viscera and blood supply have all been associated with the development of IC following AAA repair (1,6,7). On preoperative arteriography, retrograde filling of the SMA from the IMA suggests inadequate collateral blood flow and thus places such patients at higher risk for postoperative IC. If the IMA is found to be occluded or severely stenosed, ligation has been shown to carry little risk (1).

Hemodynamic

The most important hemodynamic risk factors include preoperative hypotensive shock and hypoxemia (1,6,7). Patients with renal disease also have a higher risk (7).

Other

Other risk factors noted in the literature include advanced age of the patient, inadequate preoperative bowel preparations for the AAA surgery (4), as well as previous pelvic radiation therapy for malignancy (8).

INTRAOPERATIVE PREVENTION

Several intraoperative risk factors have been proposed to predict the increased risk of postoperative IC (2,4,7). Prolonged operating and aortic cross-clamping time were both independently associated with the complication (2,4). Operative traction and trauma to the intestines were also associated, as they may have resulted in an ischemic low-flow situation (7).

In a cohort study by Bjork *et al.*, it was also shown that aortobifemoral grafting was an independent risk factor for IC (7). Reconstruction with a tube graft or an aortoiliac graft, preserving flow to the internal iliac arteries, prevented postoperative IC.

Ligation of one of the internal iliac arteries was also a risk factor, the risk being more pronounced if both were sacrificed. Several authors have claimed that ligation of the open IMA constitutes a major risk factor for postoperative IC, although no specific evidence has been presented (1,3). Intraoperatively, the presence of IMA backbleeding does not guarantee protection. The value of the ratio of IMA stump pressure to systemic stump pressure in predicting ischemia is controversial. However, several studies have shown that aggressive intraoperative revascularisation of the colon by IMA reimplantation, pelvic revascularisation, or both can significantly diminish the incidence of IC to as low as 3%, as detected by routine colonoscopy. All of these cases were mild and non-fatal (2,3,7,8).

Preoperative angiography rarely demonstrates the IMA, despite the fact that patent arteries were found in almost 50% of the patients undergoing aortic reconstruction in one study (3). Perhaps this is due to the anterior location of the takeoff of the IMA and its small size. Nevertheless, an inability to visualize the IMA by preoperative angiography does not suggest that the patent vessel can be ligated. Therefore, objective assessment of the adequacy of the blood flow to the left colon or reimplantation of the IMA is necessary (7).

There are several techniques that can be used intraoperatively to assess the adequacy of collateral blood flow to the colon, although there is a lack of a universally accepted method (2-4,7). Good results have been reported with each of these. They include the use of continuous Doppler ultrasonography to assess colonic blood flow by qualitatively judging signal changes with temporary IMA occlusion, as well as the use of colonic photoplethysmography (PPG) for the same purpose (2,3). Absence of fluorescence after intravenous injection of fluorescein has been associated with a high risk of postoperative IC, whereas "patchy" perfusion has not (2-4). Intramural pH measurement by endoluminal tonometry is another method used to detect colonic ischemia intraoperatively (2,4,7). This technique employs a silicone tonometer inserted per rectum to measure luminal pCO_2 changes, which are correlated with pH and are in equilibrium with colonic wall pCO_2 values. Studies have shown that patients who developed IC had mean pH values ranging from 6.86 to 7.11 (2), suggesting that acidosis, presumably from hypoxemia, is evident.

POSTOPERATIVE DETECTION AND MANAGEMENT

Early recognition of postoperative IC is important to allow for institution of supportive measures. Grossly bloody or Hemoccult-positive diarrhea is the most common early manifestation of IC. It usually occurs one to two days after AAA surgery, although it can appear as late as 2 weeks in some patients (2,4). Diarrhea develops because of hypermotility and loss of absorptive capacity of the colon. It occurs in 66% to 75% of patients with IC, and 50% of these are bloody (9,10). In addition, fever of uncertain origin, unexplained leukocytosis or thrombocytopenia, persistent metabolic acidosis, and abdominal distention can all be early warning signs of IC (5,9,10). None of these parameters have a sufficient sensitivity to be used alone in the detection process, but in the aggregate presentation they are important.

In 1979, Forde *et al.* (11) reported the use of flexible colonoscopy to establish the diagnosis of IC. In this study, it was found that the endoscopic appearance correlated well with subsequent histopathology studies. Several subsequent au-

thors have also recommended the use of routine postoperative lower endoscopies for survivors of ruptured AAA repairs (6,10). However, a positive effect on ultimate survival has not been demonstrated. A recent study has recommended the use of early flexible sigmoidoscopy as the most appropriate technique for diagnosis (after the onset of clinical warning signs), and for monitoring of intestinal ischemia (10). Flexible sigmoidoscopy has been shown to reliably predict full-thickness ischemia (9,10). Although postoperative IC may involve the ascending colon, transverse colon or small intestine in up to 20% of cases, involvement of the rectum, sigmoid colon or descending colon is almost always present (10). Furthermore, 95% of the lesions affecting the left colon are within reach of the sigmoidoscope. Advantages of flexible sigmoidoscopy include the ability to perform it at the bedside, its relative ease and speed of performance, enhancement of mucosal visualisation, and extent of examination when compared to rigid proctosigmoidoscopy. In patients in whom sigmoidoscopy reveals nonconfluent ischemia limited to the mucosa only, the recommended management consists of close observation, optimization of hemodynamic status and serial endoscopic examinations (9-11).

Treatment for IC following AAA repair is similar to that for IC in other patients, although the threshold for colectomy should be lower to avoid possible perforation (2,4). For early mild IC, conservative therapy including IV fluids, bowel rest, and broad-spectrum antibiotic treatments should be continued until the ischemic injury heals (2-4). IC that progresses despite supportive measures requires immediate colon resection. Primary anastomosis is contraindicated due to the potential for leakage and contamination of the aortic prosthesis (4,5).

CONCLUSION

There are several risk factors for IC that can be identified in a patient who undergoes AAA repair surgery, either emergently or electively. The most important preoperative risk factor is hypotensive shock and hypoxemia, as occurs with ruptured AAAs. Thus, a patient who is clinically hypoxemic and hypovolemic should be resuscitated adequately with oxygen and fluids prior to surgery. The most important intraoperative factors contributing to ischemic bowel include prolonged aortic clamping time and ligation of the blood supply to the bowel, mainly the IMA. Many studies have already shown that intraoperative revascularisation of the colon, either by IMA reimplantation, or pelvic revascularisation, or both, can significantly reduce the incidence of postoperative IC. Finally, in patients who present with several preoperative and intraoperative risk factors, early detection is the key to the prevention of colonic infarction. Early detection of a combination of symptoms including diarrhea, fever, leukocytosis, metabolic acidosis and abdominal distention is very important. Patients who present with these clinical findings after AAA surgery should arouse a high level of suspicion for IC and flexible sigmoidoscopy is recommended.

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