Ischemic colitis or more appropriately, vascular disease of the colon, has been known for up to 100 years. Early concern equated this condition to colonic infarction secondary to accidental arterial ligation during surgical procedures. Shaw and Green (1953) reported an episode of infarction after inferior mesenteric artery ligation. Although it was known that an occasional patient manifested a relatively benign process after such arterial ligation, it was not until 1963, when Boley et al. (1963) reported 5 patients with a disease labeled "reversible vascular occlusion of the colon" thus providing emphasis upon the milder component of this problem. Marston et al. (1966) classified this disease into 3 forms: transient, strictures, and gangrene. It is defined as a "reversible condition caused by obstruction of blood flow in intestinal tract mucosa without apparent blockage of the main artery" (Marston et al. 1966). In general, ischemic colitis is more common in the elderly and its prevalence increases with age, but it is also seen in younger patients (Brandt and Boley 2000). Its causes include vascular factors, such as ischemia and embolus, intestinal factors such as constipation, irritable bowel syndrome and history of intestinal surgery (Pescatori et al. 2009), as well as administration of drugs, such as antibiotics, appetite suppressants (phentermine), chemotherapeutic agents (vinca alkaloids and taxanes), constipation inducing medications, decongestants (pseudoephedrine), cardiac glucosides, increasing attention. Marston et al. (1966) classified this disease into 3 forms: transient, strictures, and gangrene. It is defined as a “reversible condition caused by obstruction of blood flow in intestinal tract mucosa without apparent blockage of the main artery” (Marston et al. 1966). In general, ischemic colitis is more common in the elderly and its prevalence increases with age, but it is also seen in younger patients (Brandt and Boley 2000). Its causes include vascular factors, such as ischemia and embolus, intestinal factors such as constipation, irritable bowel syndrome and history of intestinal surgery (Pescatori et al. 2009), as well as administration of drugs, such as antibiotics, appetite suppressants (phentermine), chemotherapeutic agents (vinca alkaloids and taxanes), constipation inducing medications, decongestants (pseudoephedrine), cardiac glucosides,
diuretics, ergot alkaloids, hormonal therapies, statins, illicit drugs, immunosuppressive agents, laxatives, non-steroidal anti-inflammatory drugs, psychotropic medications, serotonin agonists/antagonists and vasopressors. Iatrogenic causes may result in ischemic colitis (Brandt and Boley 1992; Champagne et al. 2007; Steele 2007). While surgery is indicated for the gangrenous form of this disease, transient and structuring forms are often ameliorated by bowel rest, fasting, and/or parenteral fluid administration. The duration of fasting and fluid administration varies among individuals (Gandhi et al. 1996). Significant prolongation of healing is sometimes observed, especially in patients with an ulcer.

Ischemic colitis encompasses a number of clinical entities, all with a final result of insufficient blood supply to a segment or the entire colon. This disease results in ischemic necrosis of a variable severity that can range from superficial mucosal involvement to full-thickness transmural necrosis (Baixauli et al. 2003). Bowel ischemia is mainly a disease of old age, caused by atheroma of mesenteric vessels. Other causes include embolic disease, vasculitis, fibromuscular hyperplasia, aortic aneurysm, blunt abdominal trauma, disseminated intravascular coagulation, irradiation, and hypovolemic or endotoxic shock. Occlusive mesenteric infarction (embolus or thrombosis) has a 90% mortality rate, whereas nonocclusive disease has a 10% mortality rate. Venous infarction occurs in young patients, usually after abdominal surgery. Patients may present with colicky abdominal pain, which becomes continuous. It may be associated with vomiting, diarrhea, or rectal bleeding (Arnott et al. 1999). The colon is the most common site of gastrointestinal ischemia. Colonic ischemia is associated with many precipitating factors and may be due to single-vessel occlusion or global hypoperfusion. The clinical manifestations are often subtle and vary, so a high level of awareness is needed. Special consideration should be given on this disease, in the case of patients, who have recently undergone cardiac or aortic surgery (Parish et al. 1991). Clinical patterns vary from transient colitis to fulminant ischemia with gangrene. Routine biochemical tests are helpful but nonspecific. It is of note that patients with ischemic colitis almost always are presented with lactic acidosis, especially in severe cases (Al Shammeri and Duerrksen 2008). The diagnosis is usually made by history, examination and endoscopy. The condition resolves completely with conservative treatment in most cases, but late diagnosis or severe ischemia can be associated with high rates of complications and death. Once ischemic colitis is diagnosed, serial physical examinations and colonoscopies are helpful for follow-up. Prompt surgery is required for severe episodes, when conservative measures fail, and for patients with chronic symptoms (Brandt and Boley 2000).

Pathogenesis

An understanding of the blood supply to the colon is necessary when considering ischemic colitis. The colon is protected from ischemia to a great extent by an abundant collateral blood supply; namely, the colon is nourished by the superior mesenteric artery (SMA), the inferior mesenteric artery (IMA), and the branches of the internal iliac arteries. The SMA gives rise to the middle colic, right colic, and ileocolic arteries, which supply the right colon and the right half of the transverse colon. The IMA branches into the left colic, sigmoid, and superior rectal (hemorrhoidal) arteries, which supply the left half of the transverse colon to proximal rectum. The distal rectum is supplied by inferior and middle rectal (hemorrhoidal) arteries, which are branches of the internal iliac artery. (Brandt and Boley 2000). There is an extensive mesenteric collateral circulation that provides substantial protection from ischemic insults. The SMA and IMA communicate through the marginal artery of Drummond, which runs along the splenic flexure and the arc of Riolan. The marginal artery of Drummond is absent or underdeveloped in 5% of the population, placing the splenic flexure at particular risk of ischemia. If SMA or IMA is gradually occluded, the arc of Riolan or the central anastomotic artery may dilate to compensate and be termed the meandering artery. The IMA and the internal iliac arteries communicate through the superior and middle/inferior rectal (hemorrhoidal) arteries. The dual blood supply of the rectum from the mesenteric and iliac arteries makes it resistant to ischemia. However, certain areas are more vulnerable in some people. The marginal artery of Drummond, which runs along the mesentery within 1 to 8 cm of the colon, is composed by the terminal portions of the branches of the major vascular arcades. This artery can keep the left colon viable when the inferior mesenteric artery is ligated during rectosigmoidectomy. However, the anatomy is vastly variable. For example, the marginal artery of Drummond is occasionally tenuous at the splenic flexure, as described by Griffiths, and indeed is absent at this point in up to 5% of patients (Griffiths 1956). It is poorly developed in the right colon in 50% of the population, explaining the occurrence of right-sided colitis (Sonneland et al. 1958). One or more of the three branches of the superior mesenteric artery may be absent in up to 20% of people. Branches of the inferior mesenteric artery may similarly be absent.

On the other hand, 60% of people have an additional vessel, called the arch of Riolan or meandering mesenteric vessel, which provides communication between the left colic and superior mesenteric arteries. The splenic flexure is a “watershed” area between the areas supplied by the two main arteries. Sierociński found that a 1-2 cm to 2-8 cm area of the splenic flexure was devoid of vasa recta, setting this area predisposed to ischemia (Sierociński 1975). There is also a watershed area at Sudek’s point between the sigmoid colon and the rectum, where the lowest sigmoid branches usually join branches of the superior rectal artery. A third potential watershed area is the right colon, where the marginal vessel is poorly developed in up to 50% of people (Baixauli et al. 2003). Therefore, any portion of the bowel may be affected, but the sites most often affected are the...
Ischemic Colitis

Table 1. Causes of mesenteric ischemia.

<table>
<thead>
<tr>
<th>CAUSES</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoperfusion</td>
<td>Heart failure or prolonged shock of any etiology</td>
</tr>
<tr>
<td>Embolic occlusion</td>
<td></td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td></td>
</tr>
<tr>
<td>Arterial thrombosis</td>
<td></td>
</tr>
<tr>
<td>Venous thrombosis</td>
<td></td>
</tr>
<tr>
<td>Vasculitis</td>
<td></td>
</tr>
<tr>
<td>Thromboangiitis obliterator</td>
<td></td>
</tr>
<tr>
<td>Disseminated intravascular coagulation</td>
<td></td>
</tr>
<tr>
<td>Hypercoagulable states</td>
<td></td>
</tr>
<tr>
<td>Sickle cell disease</td>
<td></td>
</tr>
<tr>
<td>Aortic dissection</td>
<td></td>
</tr>
<tr>
<td>Aortoiliac surgery ischemia</td>
<td>This can be developed if the inferior mesenteric artery (IMA) is sacrificed during an abdominal aortic surgery dissection</td>
</tr>
<tr>
<td>IMA sacrifice</td>
<td>This artery may be sacrificed during colonic resection, although most patients tolerate ligation of the IMA because the collateral circulation is adequate.</td>
</tr>
<tr>
<td>Translumbar aortography</td>
<td></td>
</tr>
<tr>
<td>Cardiac surgery</td>
<td></td>
</tr>
<tr>
<td>Liver transplantation</td>
<td></td>
</tr>
<tr>
<td>Bowel obstruction</td>
<td>Particularly from an incarcerated hernia or volvulus</td>
</tr>
<tr>
<td>Colonic carcinoma</td>
<td></td>
</tr>
<tr>
<td>Trauma</td>
<td></td>
</tr>
<tr>
<td>Drugs</td>
<td>Vasoconstrictors (eg, digitalis), norepinephrine, pseudoephedrine, ergot alkaloids, oral contraceptives, estrogens, various diuretics, antihypertensive medications, nonsteroidal anti-inflammatory drugs, chemotherapeutic agents, alosetron, paclitaxel, meloxicam, cocaine, and amphetamine abuse may be involved.</td>
</tr>
<tr>
<td>Corrosive injury</td>
<td>This usually affects the proximal gut, but in rare cases, it may cause penetrating injury of the transverse colon</td>
</tr>
<tr>
<td>Bowel infections</td>
<td>Necrotizing enteritis</td>
</tr>
<tr>
<td>Radiation injury</td>
<td></td>
</tr>
<tr>
<td>Large-bowel ischemia</td>
<td>In rare cases, this may result from vascular steal syndrome. An example is occlusion of the femoral or iliac artery. The lower extremity involved is supplied by a hypertrophied IMA and hemorrhoidal arteries, with reversed flow in the internal iliac artery.</td>
</tr>
<tr>
<td>Arteriovenous fistula between the mesenteric artery and veins</td>
<td>This is an unusual cause of mesenteric ischemia. Patients with this condition also have portal hypertension.</td>
</tr>
</tbody>
</table>

Ischemic colitis can be described as an inflammatory disease of the colon due to diminished blood flow, leading to bowel wall ischemia and a secondary inflammation. Most of the classifications of intestinal ischemia in the literature are based on the major causative factors. Two mechanisms may cause bowel ischemia: The first and most common is diminished bowel perfusion, due to low cardiac output, often seen in patients with cardiac disease or in cases of prolonged shock of any etiology. The second mechanism is occlusive disease of the vascular supply to the bowel, due to atheroma, thrombosis, or embolism in which the collateral circulation is not adequate to maintain bowel integrity. Regardless of the mechanism, the disease follows the same course. Depending on the cause and severity of the impairment of the bowel blood supply, the morphologic pattern can be arbitrarily subdivided into 3 groups: (1) sigmoid colon, ascending colon and splenic flexure. In systemic low-flow states, the right colon is most often involved, while localized nonocclusive ischemia affects watershed areas, such as the splenic flexure and the sigmoido-rectal junction (Guttormson and Bubrick 1989). Ligation or occlusion of the inferior mesenteric artery produces changes of the sigmoid colon, although changes may be more extensive, if other vessels have been previously occluded. Ischemic colitis isolated to the right colon is often a manifestation of acute ischemia, which may have a more fulminant course. Right-sided ischemic colitis accounts for 8% to 46% of cases (Landreneau and Fry 1990). The small bowel alone, the colon alone, or occasionally both may sustain a hypoxic injury from a variety of causes. Collectively, all these gut hypoxic injuries are designated by the term mesenteric ischemia.
transmural infarction, (2) mural infarction, when the injury extends from the mucosa into the muscularis and (3) mucosal infarction, when ischemic damage is confined to the mucosa (Fisher and Fry 1987). The etiology of mesenteric ischemia is summarized in Table 1 (Stamatakos et al. 2008).

**Transmural infarction**

Transmural infarction usually occurs in the small bowel because it is entirely dependent on the mesenteric blood supply, whereas the large bowel is near the posterior abdominal wall, whose vesicles contribute in creating a collateral blood supply and venous drainage for the large bowel. Transmural infarction frequently involves a long segment of bowel, although in rare cases, skip lesions occur. Transmural infarction is usually the result of thrombosis or embolism of the superior mesenteric artery (SMA), which may only affect the small bowel (approximately one half of the mesenteric ischemia). Mesenteric venous thrombosis is another cause; this may involve both the small bowel and the large bowel. Colonic infarction tends to occur in 2 watershed territories; (1) the splenic flexure, which is the watershed territory between the SMA and IMA blood supply, and (2) the distal sigmoid colon, which forms the watershed area between the IMA and hypogastric artery supply. Regardless of the pathogenesis, the infarcted bowel always appears hemorrhagic. Early in the course of the disease, the bowel appears intensely congested and dusky or purple red in color, with foci of subserosal and submucosal ecchymoses. As the disease progresses, the bowel wall appears edematous, thickened, rubbery, and hemorrhagic. The lumen of the bowel may contain mucus or frank blood. Arterial occlusion usually results in a sharply defined border between the infarced bowel and the normal vascularized bowel, whereas in venous thrombosis, the boundary between infarcted bowel is ill-defined with no clear demarcation between viable and nonviable bowel. Microscopy reveals suffusion of the bowel wall, which tends to mask the underlying ischemic necrosis. Later in the course of the disease, inflammatory infiltration of the bowel wall and ulceration ensue. Bacterial contamination is often present, and bowel perforation may occur within 3-4 days. Arterial occlusion may be difficult to demonstrate histologically, particularly if ischemia is the result of spasm or a low perfusion state superimposed on atheromatous disease.

**Mucosal and Mural infarction**

Mucosal and mural infarction is an ischemic injury that is confined to the inner layers of the intestinal wall. It is usually the result of hypoperfusion rather than occlusive disease. The hypoxic injury may extend deeply, but the serosa is usually spared. Shock and cardiac failure are major causative factors. Many patients with this condition have also received vasoconstricting drugs such as digitalis or norepinephrine. This type of injury may involve any part of the gut and is usually patchy and segmental, unlike transmural infarction, which involves long segments. In some cases, minute intramural thrombi are found, and whether these are the cause or effect of the ischemic injury is not clear. The affected bowel loops may appear dark red or purple as a result of luminal hemorrhage, but serosal hemorrhage, necrosis, or inflammatory exudates, are absent. The mucosa appears hemorrhagic, edematous, and thickened, with superficial ulceration (Stamatakos et al. 2008). Histologic analysis may show vascular dilatation associated with a few extravasated red cells and hemorrhagic necrosis within the superficial layers of the mucosa. However, this necrosis may extend into submucosa and the superficial layers of the muscularis. In the colon, bacterial contamination may produce superimposed pseudomembranous inflammation. The combination of necrosis and bacterial invasion develops when the mucosal barrier becomes defective. Thus, the morphologic changes seen in ischemic colitis may resemble those of pseudomembranous colitides or other inflammatory and/or infective processes (Hwang and Schwartz 2001).

**Epidemiology**

No reliable demographic data describe the incidence of ischemic colitis, worldwide. The incidence is thought to be underestimated, because many mild cases may remain unreported since they are of mild and transient nature. Moreover, many cases are misdiagnosed as inflammatory bowel disease or infectious colitis (Green and Tendler 2005). In contrast, the incidence in patients undergoing abdominal aortic reconstructive procedures has been studied. Hunter and Guernsey reported that as many as 10% of such patients have some degree of ischemic colitis (Hunter and Guernsey 1988). With population aging, the incidence of ischemic colitis is expected to present a further increase. Ischemic colitis is the most common type of ischemic disease affecting the gastrointestinal tract and accounts for 50% of cases. No data suggest that the worldwide incidence or prevalence of ischemic colitis differ between countries. Mortality and morbidity depend on the cause and comorbidities such as underlying cardiac disease, vasculitides, among others. In a study, it was found that approximately 90% of cases of ischemic colitis occur in elderly patients, as well as younger patients (Binns and Isaacsen 1978). The prognosis of ischemic colitis is more favorable than that of other forms of mesenteric ischemia. A transient ischemic episode resolves usually within 1-3 months without sequelae. With significant ischemic injury, long strictures may follow and cause mechanical problems such as bowel obstruction. More severe ischemic trauma may cause bowel gangrene and perforation, but this is a rare phenomenon. In a series of 150 patients with colonic ischemia, Brandt et al. (1981) reported that 44.7% had reversible disease, 18.7% had persistent colitis, 12.7% had ischemic stricture and 18.7% had gangrene or perforation. In 5.3%, follow-up was insufficient. No racial or ethnic predilection for ischemic colitis is reported. The male-to-female ratio in ischemic colitis is approximately 1 : 1. It is already established by the afore-
Ischemic Colitis

Once blood flow drops below a critical threshold, ischemia occurs, with consequences dependent on the individual’s ability to respond by increasing flow. Ischemia is, then, manifested as a spectrum of findings varying from transient intramural and submucosal hemorrhage and edema to gangrene. Ischemic colitis may present in two major clinical patterns: gangrenous (15%-20% of cases) and nongangrenous (80%-85%). In the nongangrenous form, lesions may develop that are transient and reversible, or that progress to chronic and irreversible strictures (10%-15%) or chronic segmental colitis (20%-25%) (Gandhi et al. 1996). The pattern of clinical presentation depends on the cause (Table 2), the extent of vascular obstruction, the speed of ischemic insult, the degree of collateralization, and comorbidity conditions. In most cases, there is no identifiable initiating factor, though a recent history of cardiac or vascular surgery, major systemic illness, or myocardial event may be present (Parish et al. 1991; Longo et al. 1992; Arnott et al. 1999; Fitzgerald et al. 2000). Up to 20% of patients may have associated colonic pathology, such as cancer. Mild to moderate abdominal pain is present in about 60% of cases, is generally abrupt in onset, and is usually described as cramps. Patients often have an urgent desire to defecate. The pain may be associated with diarrhea, frequently followed within 12 to 24 hours by mild bleeding. The blood may be bright red or maroon and is mixed with the stool. The bleeding is not copious—profuse bleeding should suggest another diagnosis. Clinical examination may reveal mild to moderate tenderness over the ischemic segment. If peritoneal signs develop, they are manifested late in the course of the condition and are often very subtle. The white cell count is generally raised, but significant ischemic injury can be present without leukocytosis (Versteeg and Broders 1979; Kaleya and Boley 1995). If acute ischemia leads to infarction, then fever, neutrophilia, and a metabolic acidosis may be present. Severe ischemia may lead to elevated levels of lactate, inorganic phosphate, and alkaline phosphatase. Unfortunately, these are all unreliable indicators for diagnosis and are poor predictors of the presence of ischemic colitis (Kurland et al. 1992). Although ischemic colitis can be classified as a separate entity on the basis of its clinical, radiologic and anatomic features, it is often confused with other colonic disorders. In the acute stage of ischemic colitis, differential diagnosis includes other types of inflammatory bowel disease (eg, Crohn’s disease, ulcerative colitis), colonic injury induced by nonsteroidal anti-inflammatory drugs, pseudomembranous colitis, and various kinds of infectious colitides. Endoscopic biopsy may be instrumental in differentiating ischemic colitis from idiopathic ulcerative colitis and infectious colitides. Moreover, the sharp demarcation between viable and necrotic colonic mucosa seen in endoscopy is a strong indicator of ischemia. In the chronic stage, the stricture of ischemic colitis must be distinguished from that of diverticular disease, carcinoma of the colon, and inflammatory bowel disease. Particular caution should be exercised when the rectum is included in the ischemic insult. Differentiating ischemic from ulcerative colitis can be extremely difficult. Moreover, ischemic and idiopathic ulcerative colitis may coexist (Brandt and Boley 1992). Differentials include adrenal adenoma, adrenocortical carcinoma, adrenal hemorrhage and adrenal metastases (Korobkin 2000).

Diagnosis

The diagnosis of ischemic colitis depends on early and repeated evaluation of the patient in conjunction with biochemical, radiological, and endoscopic assessment. The radiologic findings in ischemic colitis are nonspecific and may be seen in other inflammatory disorders of the colon. A reliable diagnosis of ischemic colitis can be made only when radiologic findings are correlated with the patient’s clinical image (Gourley and Gering 2005). When sudden thromboembolism occurs, patients experience abdominal pain localized at the left side of the abdomen, along with tenderness and bloody diarrhea. Severe ischemia may lead to bowel necrosis and perforation resulting in an acute

<table>
<thead>
<tr>
<th>Table 2. Etiologic factors.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Colonic obstruction</td>
</tr>
<tr>
<td>Shock</td>
</tr>
<tr>
<td>Medications</td>
</tr>
<tr>
<td>Digitalis, diuretics, NSAF, catecholamines, estrogens, danazol, gold, neuroleptics</td>
</tr>
<tr>
<td>Major vascular occlusion</td>
</tr>
<tr>
<td>Mesenteric ischemia, trauma</td>
</tr>
<tr>
<td>Small vessel disease</td>
</tr>
<tr>
<td>Diabetes mellitus, vasculitis, rheumatic disease, radiation injury</td>
</tr>
<tr>
<td>Hematologic disorders</td>
</tr>
<tr>
<td>Deficiencies, sickle-cell disease</td>
</tr>
<tr>
<td>Cocaine abuse</td>
</tr>
<tr>
<td>Long distance running</td>
</tr>
</tbody>
</table>
Abdomen and shock. The patient’s signs and symptoms generally resolve within several days. In some cases, a bowel stricture may be developed and cause bowel obstruction. Rarely, chronic ischemic colitis may occur, causing rectal pain and incontinence. Plain abdominal radiography is usually an initial examination undertaken in most cases involving acute abdominal problems. However, it is a useful examination for excluding colon infarction (Scholz 1993). Plain radiographic imaging reveals dilatation of a part of the colon, in early stages of the disease. When both the small bowel and the large bowel are affected by ischemic injury, the small bowel may become dilated with thickening of the valvulae conniventes. Over time, the haustra become thickened and edematous, and it contains a large amount of secretions. With further edema, the haustral markings disappear completely, and the colon acquires a hoselike appearance. Localized pneumatosis coli may be apparent. Eventual tubular narrowing and strictureting appears over a long segment. Even if the initial radiographic findings may be normal in colonic ischemia, it is an invaluable procedure in the differential diagnosis of an acute abdomen (Scholz 1993). Barium enema results are abnormal in 90% of patients with ischemic colitis. Barium enema examination during the acute stage of a vascular insult demonstrates spasm associated with thickening and blunting of the mucosal folds. Multiple mucosal scalloping or thumbprinting are seen along the contours of the bowel. Seen en face, the thumbprints appear as polypoid filling defects. With further progression of mucosal edema, the folds become thickened and ill-defined. Intraluminal secretions are increased. Ulcerations are frequently observed in pathologic specimens of the diseased bowel segment. With diffuse ulceration, the mucosa may be completely effaced. Ulcers may be seen as a serrated mucosa. Deep ulcers are a late finding.

In the healing phase when fibrosis sets in, associated flattening and rigidity of the intestinal wall may be observed. The antimesenteric border of the colon becomes pleated because of scarring, and they may appear as sacculations or pseudodiverticula. With a continuing fibrotic process, the affected segment of the large bowel acquires a tubular shape with smooth contours and a concentric lumen. The final outcome is usually a long stricture with proximal bowel dilatation. Nonetheless, when a stricture develops, it is shorter than the original length of the ischemic segment, as seen radiologically. The stricture formation in ischemic colonic disease is smooth and tapering, with a concentric lumen and without shouldering or contour defects. Sacculations are common in ischemic colitis, but skip lesions are rare. Barium enema should be avoided in cases where there is a suspicion of gangrene or perforation. Barium enema, also, makes the later use of angiography or endoscopy more difficult because of residual contrast agent (Ida et al. 1986; Yao et al. 2000). CT is the single best diagnostic modality after the plain radiography, because it can exclude many other causes of abdominal pain and can also establish the diagnosis of intestinal ischemia. CT is often used, as the initial diagnostic test, when assessing patients with nonspecific abdominal pain. It suggests the diagnosis and location or exclude other serious medical conditions and therefore narrow the differential diagnosis possibilities and illustrate the complications. Intrinsic colonic abnormalities cannot be used to diagnose or predict the development of infarction (Balthazar et al. 1999). In non-transmural ischemic colitis, the initial bowel-wall thickening, thumbprinting and pericolonic stranding, with or without peritoneal fluid, can be seen on CT images. In these cases, CT usually demonstrates the double halo or target sign. After reperfusion of the ischemic bowel wall, the sign may be produced by edema in the submucosa and appear as low attenuation or by hemorrhage and appear as high attenuation. If there is total vascular occlusion without reperfusion (infarction), the colonic wall remains thin and unenhancing, associated with dilatation of the lumen. In these cases, CT may demonstrate a thrombus in the corresponding mesenteric vessel. If ischemia is transmural, strictures may form. Occasionally, a toxic megacolon develops. Pneumatosis and/or gas in the mesenteric veins are ominous signs, when associated with bowel wall thickening and are due to bowel infarction. Pneumatosis coli or pneumatosis intestinalis can be diagnosed by demonstrating air bubbles in the colonic or intestinal wall. The gas bubbles are arranged in a linear trend and are best visualized with the window settings for bone or lung (Jones et al. 1982; Balthazar et al. 1999; Barkhausen et al. 1999; Horton et al. 2000; Horton and Fishman 2001; Wiesner and Willi 2001; Thoeni and Cello 2006). MRI is mostly useful for magnetic resonance angiography, particularly in individuals with compromised renal function. Moreover, it has been shown that the sensitivity of MRI in the detection of bowel ischemia is comparable to that of CT and it has the added advantage of not using ionizing radiation. It may be useful in depicting bowel-wall changes and in demonstrating mesenteric vascular abnormalities. As with CT, the additional use of contrast enhancement allows an assessment of the dynamic changes in the bowel wall (Ha et al. 1998). MRI has significant problems in depicting small thromboembolism in small mesenteric vessels (Li et al. 1994).

Ultrasonography is a noninvasive technique that may provide useful information, particularly in investigating chronic mesenteric ischemia. It is unlikely, though, to be definite in excluding a diagnose of mesenteric ischemia. It is undeniable, though, that absence of blood flow in the ischemic colon on Doppler sonography is a better predictor of an unfavorable outcome than early clinical and laboratory findings (Danse et al. 2000). Bowel gas frequently prevents the visualization of changes in colon, which are usually particularly marked around the splenic flexure. In the initial stages, the ischemic bowel may show increased peristalsis, which is at that time reduced. The bowel wall becomes thickened, and nodular and intramural hemorrhage and edema give rise to areas of reduced echogenicity. Gas may also be detected in the portal vein; this is a poor prognostic sign.
Color flow Doppler sonography is effective in demonstrating flow disturbances associated with tortuosity and stenosis at the origin of the celiac axis. The potential for collateralization between the celiac axis, SMA, and IMA is remarkable. As a result, the peak systolic in the celiac axis may be lower or much higher than expected when concomitant SMA occlusion is present. This variation may result in the overestimation or underestimation of the extent of ischemic disease. Images may demonstrate absent or barely visible color flow, absent arterial signals, and thickened bowel-wall loops. Doppler techniques are particularly useful for investigating chronic mesenteric ischemia. Limitations of Doppler analysis of celiac artery and SMA ischemia include that (1) the great potential for collateralization in the splanchic vessels may make an assessment of a single-vessel stenosis difficult, (2) the risk of error increases when the angle of insonation is greater than 60° and (3) careful placement of the sample volume is crucial (Teeffey et al. 1996; Dirkx and Gerscovich 1998).

Angiography has a limited role in cases of colonic ischemia, but it may be invaluable in a few specific indications, such as arteriovenous fistulas and vascular steal syndrome. It usually has no role in the evaluation and management of ischemic colitis, because at the time of symptom onset, colon blood flow has returned to normal. Damage from hypoperfusion is often at the arteriolar level, whereas mesenteric vessels and arcades are patent. There are two exceptions where angiography may have some utility: when acute mesenteric ischemia is considered and cannot be clearly distinguished from ischemic colitis by clinical presentation, or when there is isolated involvement of the right side of the colon, suggesting superior mesenteric artery occlusion (Kaufman et al. 1977; Clark and Gallant 1984; Boos 1992; Cikrit et al. 1996; Yao et al. 2000; Kirkpatrick et al. 2003).

Colonoscopy, although invasive, is the most sensitive diagnostic test for ischemic colitis. Significant mucosal changes can be seen, and biopsies can be taken when necessary, especially when investigating chronic patterns of colonic ischemia. Extreme care is needed during colonoscopy, particularly in patients with acute colonic ischemia. Frank necrosis is manifested by black bowel wall, and this is an indication to stop the colonoscopy procedure and proceed with laparotomy. Hemorrhagic nodules suggest less severe ischemia and are seen early in the course, as they are rather transient. Nonspecific findings include superficial ulceration, mucosal friability, edema, erythema and luminal narrowing (Habu et al. 1996). As changes in the color of the mucosa may reflect the severity of ischemia, viability of the colon, and prognosis, Church (1995) has proposed a management algorithm based on findings at colonoscopy.

Bowel ischemia has always been difficult to be accurately evaluated during surgery (Horgan and Gorey 1992); hence, new intraoperative tests and ways of using currently available diagnostic modalities are under investigation. These include intraoperative photoplethysmography (Ouriel et al. 1988), tonometry (Schiedler et al. 1987) and sampling of inferior mesenteric blood (Delaney et al. 1999). None of these, however, is established in clinical practice at this time. Serum D-lactate, a bacterial product that translocates across the ischemic intestinal wall, appears to be significantly elevated in patients with acute mesenteric ischemia (Murray et al. 1994) and has been used to help prediction of ischemic colitis after repair of a ruptured abdominal aortic aneurysm (Poeze et al. 1998). Intestinal fatty acid-binding protein has also been shown to be elevated in patients with mesenteric infarction (Fried et al. 1991). Alpha-glutathione S-transferase (alpha-GST) belongs to a family of enzymes involved in the detoxification of a range of toxic and foreign compounds within the cell. It has been reported as a marker of ischemia (Calman et al. 1958), an isolated rise in alpha-GST indicated segmental ischemia, while massive elevation with coincident elevation of transaminase levels suggested a global, hypoperfusional, nonocclusive type of ischemia. More recently, scintigraphic methods have been used in the diagnosis of ischemic colitis. In-111 or Tc-99m labeled leukocyte scintigraphy has been studied and demonstrated successful imaging of bowel infarction, yet the localization mechanism still remains unclear. It is suggested that the presence of polymorphonuclear leukocytes in the inflammatory response to tissue ischemia, as a result of reperfusion injury may play the primary role (Statthaki et al. 2008). Tc-99m (V) DMSA is a low-molecular weight complex that has been used successfully in the scintigraphic diagnosis of infarction (Lee et al. 1998). But, in conclusion, data suggest that Tc-99m (V) DMSA has no possible role in the detection and diagnosis of ischemic colitis (Statthaki et al. 2008).

**Treatment**

Most cases of ischemic colitis do not underline any identifiable cause. However, a high index of suspicion is required in patients with abdominal pain, diarrhea, rectal bleeding, and abdominal tenderness who have a possible precipitating cause. Serial physical examinations and repeated endoscopy may be required for the diagnosis and ongoing management (Church 1995). If the physical examination does not suggest gangrene or perforation, the patient is treated expectantly. Very mild cases can be managed on an outpatient basis with liquid diet, close observation, and antibiotics. For inpatients, a combination of intravenous fluids and bowel rest is recommended to reduce intestinal oxygen requirements. Parenteral nutrition should be considered for patients who do not respond immediately and those who are poor candidates for surgery (Brandt and Boley 2000) as they may need prolonged bowel rest. Broad-spectrum antibiotics have been recommended by many authors, as there is experimental evidence that this reduces the length and severity of bowel damage (Brandt and Boley 1992). It is critically important to maximize intestinal perfusion. Therefore, digitalis and other vasoconstrictors are withdrawn or minimized, if possible, and cardio-
ac output is maximized by adequate fluid resuscitation (Binns and Issacson 1978; Kim and Gewertz 1987). Steroids have no role in the treatment of acute ischemia, and they only serve in masking the development of peritoneal signs and delay a possible necessary laparotomy. Likewise, oral cathartics and bowel preparations should not be given because of the risk of precipitating colonic perforation or toxic dilation of the colon (Gandhi et al. 1996). Persistent unexplained sepsis or pyrexia increases concern about infarction of the bowel. Assuming there is no full-thickness ischemia on the initial endoscopy, repeated endoscopy should be regularly performed until the ischemic changes improve or until there is a change in the clinical condition, which mandates other investigations or laparotomy.

About 20% of patients with acute ischemic colitis require surgery. Indications include ongoing sepsis refractory to medical management, signs of peritoneal irritation, diarrhea and bleeding lasting more than 10 to 14 days, evidence of pneumoperitoneum on imaging, endoscopic evidence of full-thickness ischemia, or protein-losing enteropathy that goes on longer than 2 weeks (Boley 1990). Without surgery, the risk of perforation is high. At laparotomy, the diagnosis is confirmed and the involved segment of the colon is resected. It is crucial to check for the viability of the mucosal margins of the resected specimen, as the serosal surface of the bowel may look surprisingly well-perfused. Some authors have reported the use of intraoperative techniques to exclude colonic ischemia, such as Doppler ultrasonography, intraoperative colonoscopy with laser Doppler, intraoperative photoplethysmography, oxygen electrodes, pulse oximetry of transcolonic oxygen saturation, and intravenous fluorescein for determination of viability (Maupin et al. 1989; Bergman et al. 1992). Many of these studies were evaluated at the time of aortic surgery in an effort to predict the development of ischemia (Schiedler et al. 1987; Ouriel et al. 1988; Horgan and Gorey 1992; Delaney et al. 1999). Generally, surgery involves colectomy with end-colostomy or ileostomy, leaving the distal bowel, as a Hartmann stump or a mucous fistula, if poor perfusion of the distal bowel end precludes overseeing. Intestinal ischemia-reperfusion leads to circulatory shock of the area, involved. Although, intravascular volume depletion is considered to be the main mechanism for the circulatory derangement, the pathophysiology of this type of shock and of the distant organ dysfunction is still not completely comprehended. Douzinas et al. (2003) have reported that reducing arterial oxygen content during reperfusion following splanchnic ischemia may provide a possible strategy to control the development of oxidative injury induced by ischemia-reperfusion. Hypoxic reperfusion represents an intervention that, when applied after ischemia and early reperfusion, may attenuate the triggering of a multifactorial cascade leading to the production of various reactive products that cause further tissue injury (Douzinas et al. 2004). Depending on how much of the colon is involved, the stoma may have to be permanent. In one series, 75% of patients who underwent resection and stoma formation for segmental involvement of the colon were able to have their stomas closed, versus only a third of patients with total colonic involvement (Longo et al. 1997). As a group, patients who require surgery are more critically ill, and the associated mortality rate is 30% to 60% (Binns and Issacson 1978; Brandt and Boley 1992). As far as total parenteral nutrition is concerned, if a patient who would otherwise require surgery has a concurrent or recent myocardial infarction or major medical contraindications to surgery, a trial of long-term parenteral nutrition and intravenous antibiotics may be considered as an alternative, but less-than ideal, form of treatment.

The clinical course of chronic ischemic colitis is reversible in about half of the cases. In about two thirds of patients with a reversible injury, the symptoms resolve in 24 to 48 hours, and endoscopic and radiographic investigations confirm healing within 2 weeks. In severe but reversible injury (eg, in segmental ulcerative colitis), the colon may take 1 to 6 months to heal (Brandt and Boley 1992). In the other third of cases, however, the damage is too severe to heal, leading to chronic segmental colitis or strictures. In such cases, the patient may have persistent diarrhea, rectal bleeding, protein-losing enteropathy, or repeated episodes of sepsis, which may lead to perforation. Gangrene occurs in about 15% of patients and requires laparotomy within hours. Others (20%-25%) develop chronic segmental ulcerating colitis, while about 10% subsequently develop strictures. Fulminant pancolitis due to ischemia is rare, occurring in only 1%. Total colonic ischemia without rectal involvement occurs in up to 18%, and this requires surgery in all cases; the mortality rate is 75%, even with surgery (Longo et al. 1997). Endoscopy may suggest segmental colitis. Biopsy specimens of bullae show submucosal hemorrhage and edema, while intervening areas reveal nonspecific inflammation. Venous congestion, mucus depletion and injury of the crypt’s architecture and surface epithelial cells are also common. However, the diagnosis is not always easy and chronic ischemic colitis can easily be mistaken for inflammatory bowel disease.

Compounding the difficulty, pseudopolyposis may be present in patients with ischemic colitis. Patients may also develop ischemic strictures, which are classically smoother than neoplastic strictures; however, differentiation is not always easy and resection may be required, both for treatment of symptoms and to obtain a definitive histopathologic diagnosis. Mildly symptomatic chronic disease frequently responds to supportive management. In contrast to acute ischemia, chronic ischemia may respond to topical steroid preparations. Resectional surgery is generally reserved for patients for whom conservative supportive therapy fails and for those with recurrent episodes of colitis or with symptomatic strictures. As in surgery for acute ischemia, the resected specimen must be examined to confirm that the mucosa is normal at the resection margins, and pulsatile...
bleeding must be identified at the bowel ends. Surgery in patients with chronic ischemia usually is curative, and development of further ischemic disease is rare (Baixauli et al. 2003).

Prognosis

People with ischemic colitis are hospitalized. Initially, nil per os should be instructed, so that the intestine can rest. Instead, intravenous fluids, electrolytes, and nutrients are administered. Antibiotics are often given to prevent infection that might follow the inflammation. Within a few days, antibiotics are usually stopped and eating is resumed. More than 50% of people with ischemic colitis improve and recover over a period of 1 to 2 weeks. However, when the interruption of blood supply is more severe or more prolonged, the affected portion of the large intestine may have to be surgically removed. Ischemic colitis often occurs without an obvious predisposing event, may involve all segments of the large intestine, and frequently requires surgery. While its course may be self-limited, elderly and diabetic patients, as well as those developing ischemia following aortic surgery or hypotension, continue to present a poor prognosis (Binnis and Isaacson 1978).

References


Horgan, P.G. & Gory, T.F. (1992) Operative assessment of intesti-