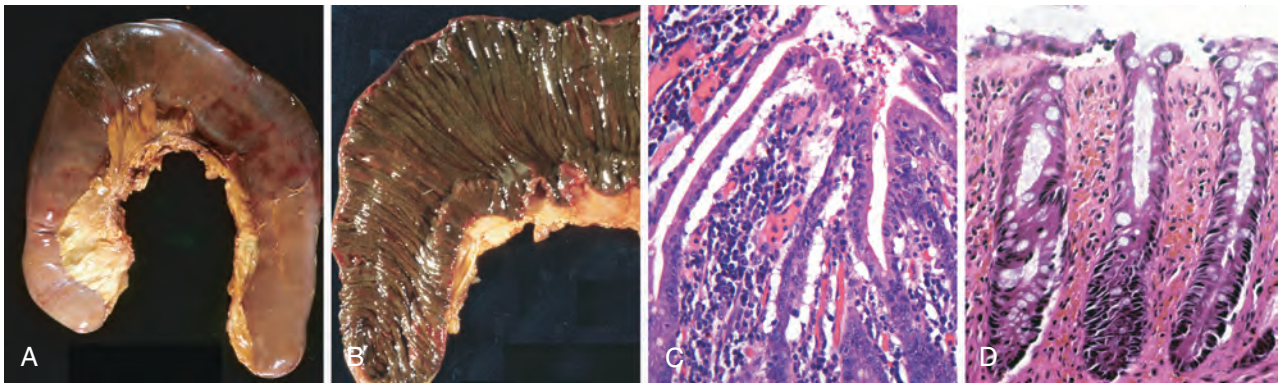

Figure 17-24 Ischemic bowel disease. **A,** Jejunal resection with dusky serosa of acute ischemia (mesenteric thrombosis). **B,** Mucosa is stained with blood after hemorrhage. **C,** Characteristic attenuated villous epithelium in this case of acute mesenteric thrombosis. **D,** Chronic colonic ischemia with atrophic surface epithelium and fibrotic lamina propria.

coli O157:H7 infection, strangulated hernia, or vascular compromise due to prior surgery.

Acute colonic ischemia typically presents with sudden onset of cramping, left lower abdominal pain, a desire to defecate, and passage of blood or bloody diarrhea. The blood loss is usually insufficient to require transfusion, but patients may progress to shock and vascular collapse within hours in severe cases. Surgical intervention, which is necessary in approximately 10% of cases, should be considered if peristaltic sounds diminish or disappear, that is, paralytic ileus, or other features of infarction, such as guarding and rebound tenderness develop. Because these physical signs overlap with those of other abdominal emergencies, including acute appendicitis, perforated ulcer, and acute cholecystitis, the diagnosis of intestinal necrosis may be delayed or missed, with disastrous consequences.

With appropriate management, mortality in the first 30 days is approximately 10%. Mortality is doubled in patients with right sided colonic disease, who have a more severe course in general. This may be because the right side of the colon is supplied by the superior mesenteric artery, which also supplies much of the small intestine. Thus, right sided colonic ischemia may be the initial presentation of more severe disease, including that caused by acute occlusion of the superior mesenteric artery (Fig. 17-24). Other poor prognostic indicators include co-existing chronic obstructive pulmonary disease (COPD) and persistence of symptoms for more than 2 weeks. Happily, most patients recover fully and colonic ischemia does not recur in the majority of cases. Listed below are some additional forms of bowel ischemia, their antecedents and outcomes.

- *Mucosal and mural infarctions* by themselves may not be fatal. However, these often progress to more extensive infarction if the vascular supply is not restored by correction of the insult or, in chronic disease, by development of in adequate collateral supplies. The diagnosis of nonocclusive ischemic enteritis and colitis can be particularly difficult because there may be a confusing array of nonspecific abdominal symptoms, including intermittent bloody diarrhea and intestinal obstruction.
- *Chronic ischemia* may masquerade as inflammatory bowel disease, with episodes of bloody diarrhea interspersed with periods of healing.
- *CMV infection* causes ischemic GI disease due to viral tropism for endothelial cells. CMV infection, which can be a complication of immunosuppressive therapy, is discussed further in Chapter 8.
- *Radiation enterocolitis* occurs when the GI tract is irradiated. In addition to epithelial damage, radiation-induced vascular injury may be significant and produce changes that are similar to ischemic disease. Beyond clinical history, the presence of highly atypical "radiation fibroblasts" within the stroma may provide an important clue to the etiology. Acute radiation enteritis manifests as anorexia, abdominal cramps, and malabsorptive diarrhea, while chronic radiation enteritis or colitis is often more indolent and may present as an inflammatory entero colitis.
- *Necrotizing enterocolitis* (NEC) is an acute disorder of the small and large intestines that can result in transmural necrosis. It is the most common acquired GI emergency of neonates, particularly those who are premature or of low birth weight, and frequently presents when oral feeding is initiated. NEC is discussed in more detail in Chapter 10, but is noted here because ischemic injury is thought to contribute to the pathogenesis.

Angiodysplasia

Angiodysplasia, a lesion characterized by malformed submucosal and mucosal blood vessels, occurs most often in the cecum or right colon and usually presents after the sixth decade of life. Although the prevalence of angiodysplasia is less than 1% in the adult population, it accounts for 20% of major episodes of lower intestinal bleeding; intestinal hemorrhage may be chronic and intermittent or acute and massive.

The pathogenesis of angiodysplasia remains undefined but has been attributed to mechanical and congenital factors. Normal distention and contraction may intermittently occlude the submucosal veins that penetrate through the muscularis propria and can lead to focal dilation and tortuosity of overlying submucosal and mucosal vessels. Because the cecum has the largest diameter of