

Appendicitis was first described in 1886 by Reginald Fitz. Its etiology is still not completely understood. Fecaliths, incompletely digested food residue, lymphoid hyperplasia, intraluminal scarring, tumors, bacteria, viruses, and inflammatory bowel disease have all been associated with inflammation of the appendix and appendicitis.

Although not proven, obstruction of the appendiceal lumen is believed to be an important step in the development of appendicitis. In some cases, obstruction leads to bacterial overgrowth and luminal distension, with an increase in intraluminal pressure that can inhibit the flow of lymph and blood in some cases. Then, vascular thrombosis and ischemic necrosis with perforation of the distal appendix may occur. Any perforation that occurs near the base of the appendix should raise concerns about another disease process. Most patients who will perforate do so before they are evaluated by surgeons.

Appendiceal fecaliths (or appendicoliths) are found in approximately 50% of patients with gangrenous appendicitis who perforate but are rarely identified in those who have simple disease. As mentioned earlier, the incidence of perforated, but not simple, appendicitis is increasing. The rate of perforated and nonperforated appendicitis is correlated in men but not in women. Together these observations suggest that the underlying pathophysiologic processes are different and that simple appendicitis does not always progress to perforation. Furthermore, some cases of simple acute appendicitis may resolve spontaneously or with antibiotic therapy, and recurrent disease is remotely possible. The relative frequency of these events is unknown.

When perforation occurs, the resultant leak may be contained by the omentum or other surrounding tissues to form an abscess. Free perforation normally causes severe peritonitis. These patients may also develop infective suppurative thrombosis of the portal vein and its tributaries along with intrahepatic abscesses. The prognosis of the very unfortunate patients who develop this dreaded complication is very poor.

### CLINICAL MANIFESTATIONS

More refined approaches to diagnosis, supportive care, and surgical intervention are likely responsible for the remarkable decrease in the risk of mortality from simple appendicitis to currently less than 1%. Nevertheless, it is still important to identify patients who might have appendicitis as early as possible to minimize their risk of developing complications. Patients who have had symptoms for more than 48 h are more likely to perforate.

Appendicitis should be included in the differential diagnosis of abdominal pain for every patient in any age group unless it is certain that the organ has been previously removed (Table 356-1).

The appendix's anatomical location, which varies, directly influences how the patient presents for care. Where the appendix can be "found" ranges from local differences in how the appendiceal body and tip lie relative to its attachment to the cecum (Figs. 356-1 and 356-2), to where the appendix is actually situated in the peritoneal cavity—for example, from its typical location in the right lower quadrant, to the pelvis, right flank, right upper quadrant (as may be observed during

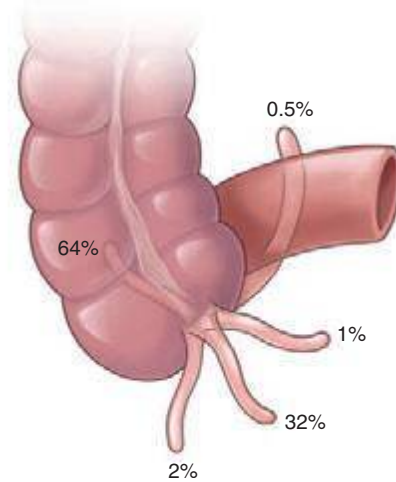


FIGURE 356-1 Regional anatomical variations of the appendix.

pregnancy), or even the left side of the abdomen for patients with malrotation or who have severely redundant colons.

Because the differential diagnosis of appendicitis is so extensive, deciding if a patient has appendicitis can be difficult (Table 356-2). Soliciting an appropriate history requires detecting symptoms that might suggest alternative diagnoses. Patients with appendicitis may not have any abdominal discomfort early in the disease process. Furthermore, many patients may not present with the classically described history or physical findings.

What is the classic history? Nonspecific complaints occur first. Patients may notice changes in bowel habits or malaise and vague, perhaps intermittent, crampy, abdominal pain in the epigastric or periumbilical region. The pain subsequently migrates to the right lower quadrant over 12–24 h, where it is sharper and can be definitively localized as transmural inflammation when the appendix irritates the parietal peritoneum. Parietal peritoneal irritation may be associated with local muscle rigidity and stiffness. Patients with appendicitis

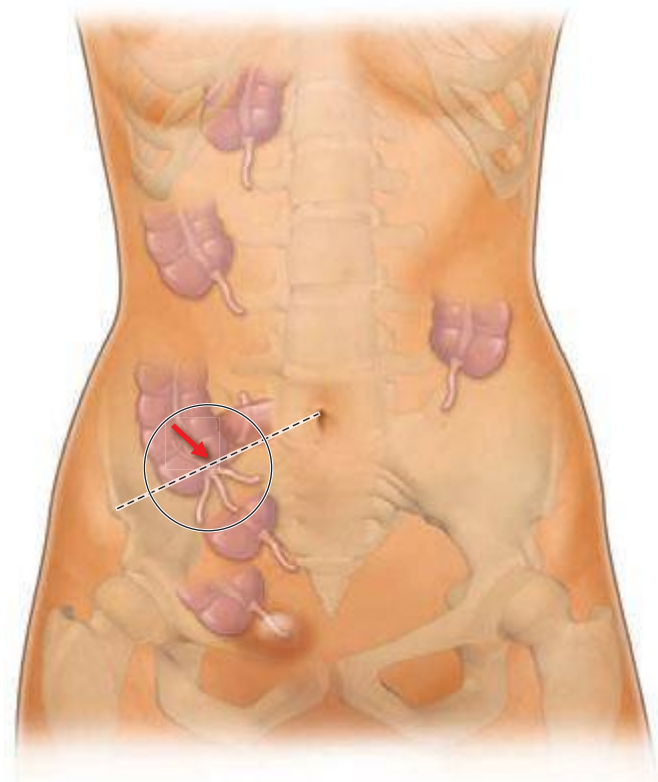


FIGURE 356-2 Locations of the appendix and cecum.

TABLE 356-1 SOME CONDITIONS THAT MIMIC APPENDICITIS

Crohn's disease	Meckel's diverticulitis
Cholecystitis or other gallbladder disease	Mittelschmerz
Diverticulitis	Mesenteric adenitis
Ectopic pregnancy	Omental torsion
Endometriosis	Pancreatitis
Gastroenteritis or colitis	Lower lobe pneumonia
Gastric or duodenal ulceration	Pelvic inflammatory disease
Hepatitis	Ruptured ovarian cyst or other cystic disease of the ovaries
Kidney disease, including nephrolithiasis	Small-bowel obstruction
Liver abscess	Urinary tract infection