Review Article

CT Features of Ulcerative Colitis and Crohn's Disease

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Ulcerative colitis and Crohn's disease, collectively known as idiopathic inflammatory bowel disease (IBD), remain a diagnostic and therapeutic challenge. Whereas other inflammatory diseases of the gut are distinguished either by a specific etiologic agent or by the nature of the inflammatory activity, ulcerative colitis and Crohn's disease are disorders with unknown etiologies, uncertain and unpredictable courses, and variable responses to medical and surgical management. Colonoscopy and barium studies are the principle methods of evaluating patients with known or suspected IBD [1]. Both techniques provide superb visualization of the bowel mucosa, abnormal surface patterns, and changes in intestinal caliber [2]. However, these techniques cannot show the transmural extent of inflammation or the intraperitoneal or extraintestinal complications of IBD. In recent years, CT has become indispensable in the assessment of patients with IBD by virtue of its ability to show pathologic changes involving the bowel wall, mesenteric attachments, and adjacent structures [3–5]. For acutely ill patients, CT is often the only study re-quired, providing crucial information for both accurate diagnosis and management of the many complications associated with IBD [6, 7]. The purpose of this article is to review the value and current applications of CT for patients with IBD.

Technical Considerations

Complete opacification of a well-distended gut is mandatory for accurate CT evaluation of the bowel wall thickening and distortion of mural components that are the hallmarks of IBD [8–10]. Nonopacified bowel loops are potential sources of diagnostic error because they can simulate an abscess, mass, or enlarged lymph nodes. The patient should drink 1000 ml of a 2% barium suspension (Readi-CAT 2; E-Z-EM, Westbury, NY) the evening before the CT examination to opacify the colon by the next day. An additional 1000 ml of dilute barium suspension is administered orally over a 1-hr period before the scan to opacify the stomach and small bowel. Dilute (2%) water-soluble contrast material should be used in preoperative and trauma patients as well as those with suspected bowel perforation.

Iodinated contrast material should be administered IV to all patients unless contraindicated because bowel wall contrast enhancement is an important indicator of the degree of mural inflammation and mesenteric engorgement. We give 150 ml of 60% iodinated contrast material, delivered as a monophasic bolus with a power injector at 2 ml/sec. In patients with known or suspected urinary tract complications of IBD, scans also should be obtained before IV contrast material injection. These non–contrast-enhanced scans assist in the visualization of renal stones and intracystic orally administered contrast material resulting from an enterovesical fistula. This pathology might otherwise be obscured by dense iodinated contrast material in the kidneys and bladder.

In patients in whom it is important to differentiate inflammatory from neoplastic colonic disease, rectal air insufflation immediately before scanning is useful in maximizing colonic distension. It allows better assessment of bowel wall homogeneity by minimizing artifacts that may be caused by dense, positive intraluminal contrast material. This technique should be used in conjunction with the IV administration of glucagon to induce intestinal hypomotility, which reduces peristaltic artifacts and assists in the retention of the insufflated air. In the clinical setting of fulminant colitis, however, air insufflation is contraindicated because of the risk of perforation.

Our standard imaging protocol is to obtain scans from the diaphragm through the perineum using 10-mm collimation at 10-mm intervals (pitch, 1:1). In patients with known or suspected IBD, this protocol is modified so

that 5-mm-thick scans at 5-mm intervals (pitch, 1:1) are obtained from the iliac crest through the perineum. If specific bowel loops are of concern, as indicated by other studies, it is important to obtain high-resolution, thin-section images with optimum levels of intravascular contrast enhancement when the affected gut is scanned.

On CT scans, the thickness of the wall of the normal small bowel and colon does not exceed 2–3 mm. This measurement should be made when the gut is fully distended and imaged transaxially because oblique scanning planes may artificially increase this measurement. A wall thickness of greater than 4 mm in any segment of well-distended small bowel and colon is abnormal.

The mesentery and omentum normally contain blood vessels and small lymph nodes smaller than 3–5 mm and fat having an attenuation value of –75 to –125 H [8, 9]. A higher attenuation value indicates the presence of fluid, cellular infiltrate, edema, hemorrhage, or fibrosis.

When patients with IBD are evaluated, it is important to specifically search for the following CT features and associated complications: (1) bowel wall thickness, homogeneity, and contrast enhancement; (2) mesenteric, perirectal, retroperitoneal, and omental fat attenuation and homogeneity; (3) lymph node number and size; (4) extraluminal contrast collections, abscesses, fistulas, and sinus tracts; (5) mesen-

teric or perivisceral masses; (6) size of the presacral space; (7) gallbladder and liver abnormalities, such as gallstones, choledocholithiasis, sclerosing cholangitis, and steatosis; (8) pancreatitis; (9) hydronephrosis and urinary tract stones; (10) sacroiliitis and spinal ligamentous ossification; (11) osteomyelitis; and (12) avascular necrosis of the femoral heads.

Ulcerative Colitis

Ulcerative colitis is characterized pathologically by extensive ulceration and diffuse inflammation of the mucosa. The disease characteristically begins in the rectum and extends proximally to involve part or all of the colon. Early in the disease course, CT scans often are normal; however, with more advanced disease, significant mural pathology usually is present and can be detected on CT scans [7]. CT is not recommended as a primary means of diagnosing ulcerative colitis because of its low diagnostic sensitivity for early disease. Nevertheless, CT is a useful complementary imaging technique because it can detect certain complications and can explain many of the morphologic changes of advanced ulcerative colitis seen on barium enema studies.

Acute Phase: Mucosal Inflammation and Ulceration

The early stages of ulcerative colitis are manifested radiologically and colonoscopically by a granular mucosal pattern attribut-

able to edema, hyperemia, and abnormal mucin production [7]. These changes are beneath the spatial resolution of CT [1, 2]. With progressive disease, severe mucosal ulceration can denude certain portions of the colonic wall, leading to inflammatory pseudopolyps. When sufficiently large, these pseudopolyps can be visualized on CT scans [11, 12] (Fig. 1). Mural thinning, unsuspected perforations, and pneumatosis can be detected on CT scans in patients with toxic megacolon [13] (Fig. 2). In this regard, CT can be quite helpful in determining the urgency of surgery in patients with stable plain abdominal films but with a deteriorating clinical course. Postinflammatory pseudopolyps also can be seen on CT scans [11, 12].

Subacute and Chronic Phases: Mural Involvement

Mural thickening (Fig. 3) and luminal narrowing (Figs. 4 and 5) are common CT features of subacute and chronic ulcerative colitis [14–18]. Although it is easy to understand why the bowel wall becomes thick in a transmural, fibrosing inflammatory disorder such as Crohn's disease, its pathogenesis is not intuitively obvious in ulcerative colitis, which is predominantly a mucosal disease.

In chronic ulcerative colitis, the muscularis mucosae, for unknown reasons, becomes markedly hypertrophied, often by a factor of 40-fold (Fig. 4). Forceful contraction of this hypertrophied longitudinal muscle may pull


A   B
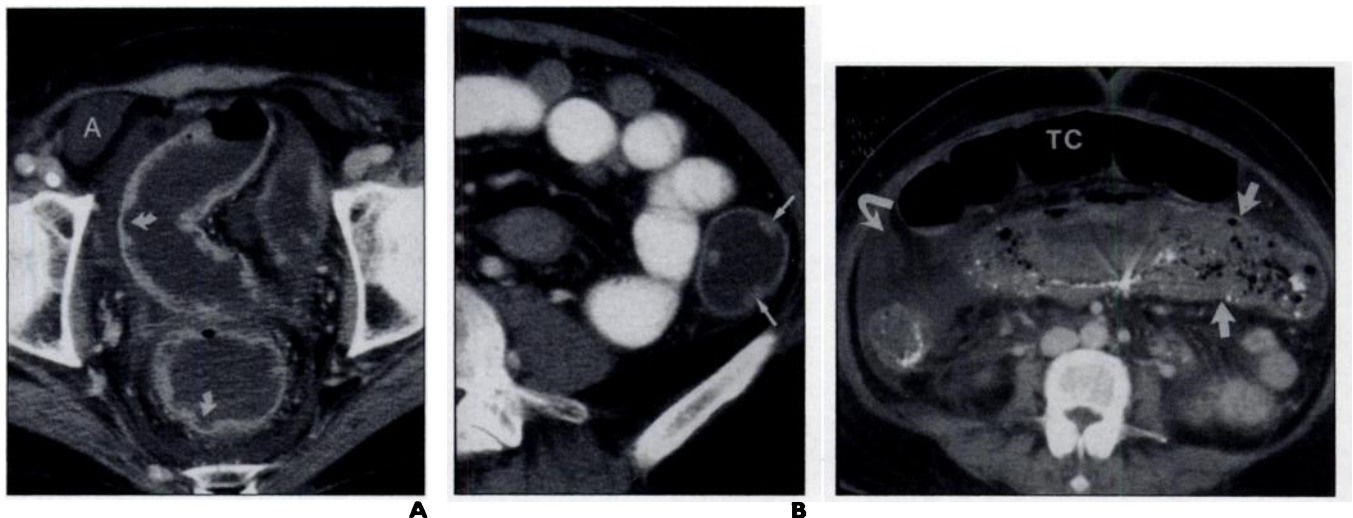
Fig. 1.—Mucosal disease in acute ulcerative colitis.
A, Pelvic CT scan shows diffuse mucosal thickening of fluid-filled rectum and sigmoid. Deep ulcerations (*arrows*) are visualized. Note normal luminal caliber and ascites (A).
B, Magnified CT image of distal descending colon shows residual islands of inflamed mucosa protruding above denuded colonic surface (so-called inflammatory pseudopolyps) (*arrows*).

Fig. 2.—CT scan shows unsuspected perforation and abscess (*straight arrows*) in patient who had fulminant ulcerative colitis and toxic megacolon and who was not improving clinically despite intensive medical management. Patient had sustained clinically silent perforation. This CT scan prompted colectomy and surgical abscess drainage. Curved arrow shows fluid in right paracolic gutter. TC = transverse colon.
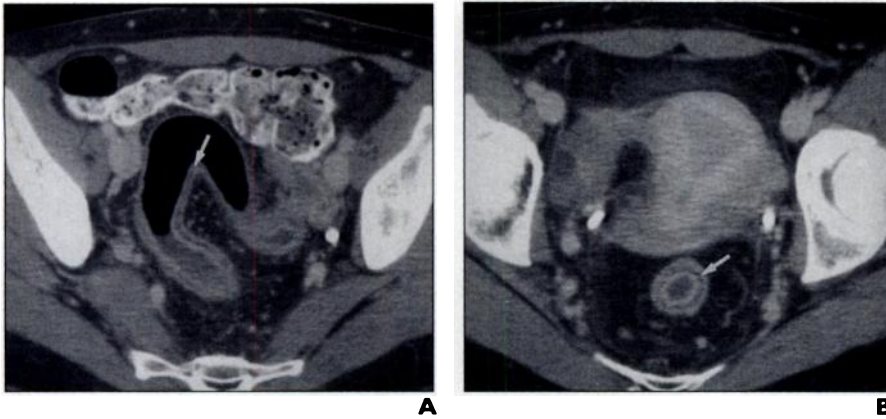
Fig. 3.—Target sign in subacute ulcerative colitis.
A, CT section through rectum and sigmoid shows diffusely inflamed and thickened mucosa. Note that edematous layer of submucosa (*arrow*) of lower attenuation is paralleled by external layer of muscularis propria and internal layer of mucosa, both of which show higher attenuation. Also note prominent blood vessels and lymph nodes in sigmoid mesocolon.
B, Axial CT scan shows rectum with target appearance produced by two higher-attenuation rings (mucosa and muscularis propria) that are separated by edematous submucosa of lower attenuation (*arrow*). This mural stratification is typical of ulcerative colitis.
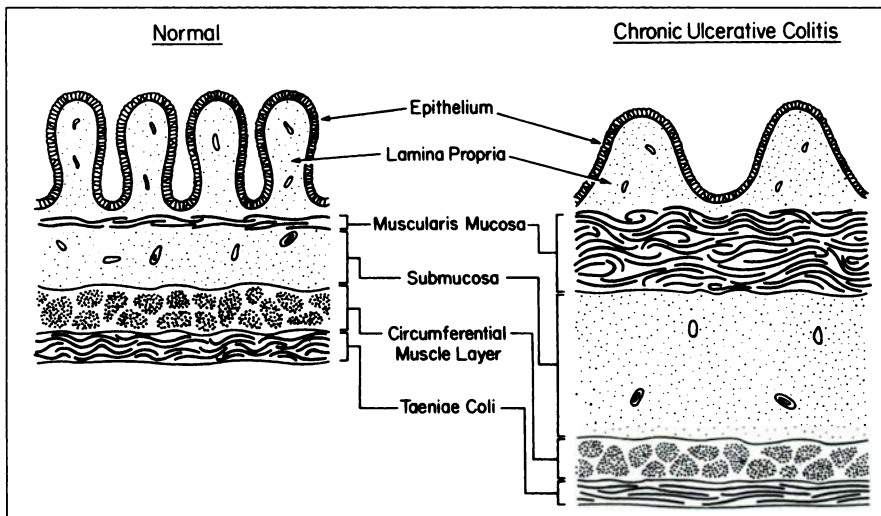
Fig. 4.—Pathologic features of chronic ulcerative colitis indicating morphologic basis of target sign. Diagram shows marked hypertrophy of muscularis mucosae found in chronic ulcerative colitis. This longitudinally oriented muscle becomes chronically contracted, shortening and narrowing involved colon. Submucosa widens because of fatty infiltration, causing further narrowing of lumen. When viewed axially, fatty infiltration of submucosa produces central ring of low attenuation that is surrounded by rings of soft-tissue density representing mucosa internally and muscularis propria externally, which do not undergo fatty infiltration. These rings produce target or halo sign on CT scans. (Reprinted from [20])

the mucosa away from the submucosa, producing diffuse or segmental narrowing of the lumen. This contraction also causes shortening of the colon [7, 19].

The submucosa becomes thickened because of the deposition of fat or, in acute and subacute cases, edema [20]. Submucosal thickening further contributes to luminal narrowing. Additionally, the lamina propria is thickened because of round-cell infiltration in both acute and chronic ulcerative colitides [7, 12].

On axial CT scans (Fig. 5), these mural changes produce a target or halo appearance: the lumen is surrounded by a ring of soft-tissue density (mucosa, lamina propria, and hypertrophied muscularis mucosae), which is surrounded by a low-density ring (fatty infiltration of the submucosa), which in turn is surrounded by a ring of soft-tissue density (muscularis propria) [7, 17, 19]. This mural stratification is not specific and also can be seen in Crohn's disease, infectious enterocolitis, pseudomembranous coli-

tis, ischemic and radiation enterocolitides, mesenteric venous thrombosis, bowel edema, and graft-versus-host disease [21–25].

Certain CT findings can help differentiate granulomatous from ulcerative colitis. Mural stratification (i.e., the ability to visualize individual layers of bowel wall) is seen in 61% of patients with chronic ulcerative colitis but in only 8% of patients with chronic granulomatous colitis [5, 16]. Also, the mean colonic wall thickness in chronic ulcerative colitis is 7.8 mm, significantly smaller than that observed in granulomatous colitis (11 mm) [16]. Finally, the outer contour of the thickened colonic wall is smooth and regular in 95% of ulcerative colitis patients, whereas serosal and outer mural irregularities are present in 80% of granulomatous colitis patients [5].

Rectal and Perirectal Diseases

Rectal narrowing and widening of the presacral space are hallmarks of chronic ulcerative colitis (Fig. 5A). High-resolution CT depicts the anatomic alterations that underlie these rather dramatic morphologic changes. The rectal lumen is narrowed because of the previously described mural thickening that attends chronic ulcerative colitis. As a result, the rectum has a target appearance on axial scans, which should not be mistaken for the external anal sphincter, mucosal prolapse, or the levator ani muscle. The widening of the presacral space is caused by the proliferation of perirectal fat. On CT scans, this fat is characterized by an increased number of nodular and streaky soft-tissue densities and an abnormal attenuation value, 10–20 H higher than that of normal extraperitoneal or mesenteric fat (Fig. 5B). These fatty changes relate to a number of factors, including ex vacuo replacement by fat of the void produced by rectal luminal narrowing and lipodystrophy resulting from an influx of inflammatory cells and edema. Edematous adipose tissue and enlarged lymph nodes often are observed in the perirectal region at the time of abdominoperineal resections in patients with chronic ulcerative colitis [7, 14, 19].

Carcinoma Complicating Ulcerative Colitis

Patients with ulcerative colitis have a markedly increased risk of developing cancer of the colon and rectum [26]. IBD-related colorectal cancers are notoriously difficult to diagnose early by barium enema and colonoscopy because these neoplasms are frequently scirrhous and flat and are superimposed on a colon that is shortened and narrowed and contains chronically inflamed mucosa. Therefore,
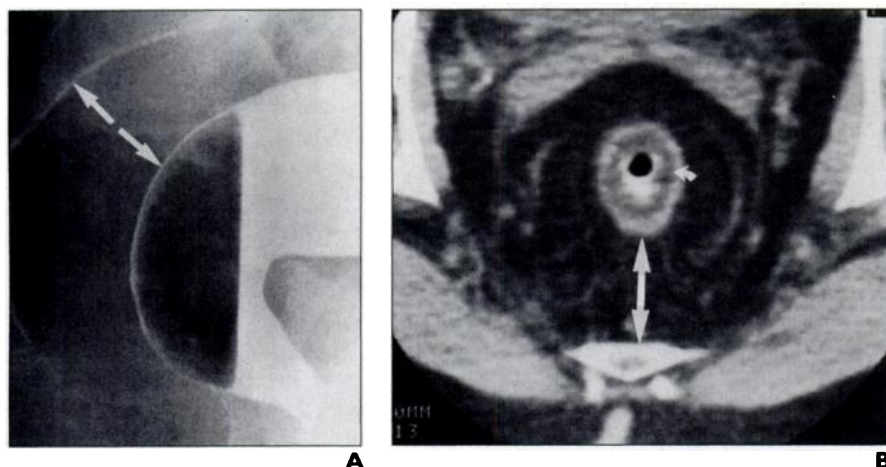
Fig. 5.—CT scan–barium enema correlation for rectal changes in chronic ulcerative colitis.
A, Lateral view of rectum on double-contrast barium enema film shows narrowing of lumen, granular appearance of mucosa, loss of valves of Houston, and significant widening of presacral space (*arrows*).
B, Corresponding pelvic CT scan shows target sign of rectum. Middle, low-density ring (*curved arrow*), which is seen in chronic disease, is attributable to fatty infiltration. Widened presacral space (*straight double-headed arrow*) is filled with abnormal fat with higher-than-normal attenuation and increased number of lymph nodes. (Adapted from [20])

CT scans in patients with long-standing ulcerative colitis should be scrutinized for asymmetric mural thickening, focal loss of mural stratification, and mural thickening of greater than 1.5 cm, all features suggesting malignancy. In patients with established carcinoma, CT is useful for tumor staging.

Crohn's Disease

Crohn's disease can affect any portion of the gastrointestinal tract, most commonly the terminal ileum and proximal colon. The acute, active phase of Crohn's disease is characterized by focal inflammation, aphthoid ulceration with adjacent cobblestoning, a chronic inflammatory reaction with lymphoid aggregates and granulomas that may be transmural, fissures, and fistulas. The chronic, resolving phase of this disorder is associated with fibrosis and stricture formation [27, 28].

Acute Phase: Inflammation

The earliest macroscopic manifestations of Crohn's disease are enlarged lymphoid follicles and aphthoid ulcerations. These changes are demonstrable on double-contrast barium radiography but are beneath the spatial resolution of CT. Consequently, when the disease is limited to the mucosa, CT scans usually are normal. Although inflammatory and postinflammatory pseudopolyps may be identified on CT scans, the assessment of the mucosa is best done with barium studies and colonoscopy, which are more direct and sensitive [11, 12]. CT scans are

more accurate than plain abdominal films for the detection of the intramural gas (Fig. 6) and mural thinning found in toxic megacolon associated with Crohn's disease [13].

Crohn's disease is manifested on CT scans by bowel wall thickening ranging from 1 to 2 cm [5, 14, 15, 29]. This thickening, which occurs in up to 83% of patients, is observed most frequently in the terminal ileum, but other portions of the small bowel, colon, duodenum, stomach, and esophagus may be similarly affected [5].
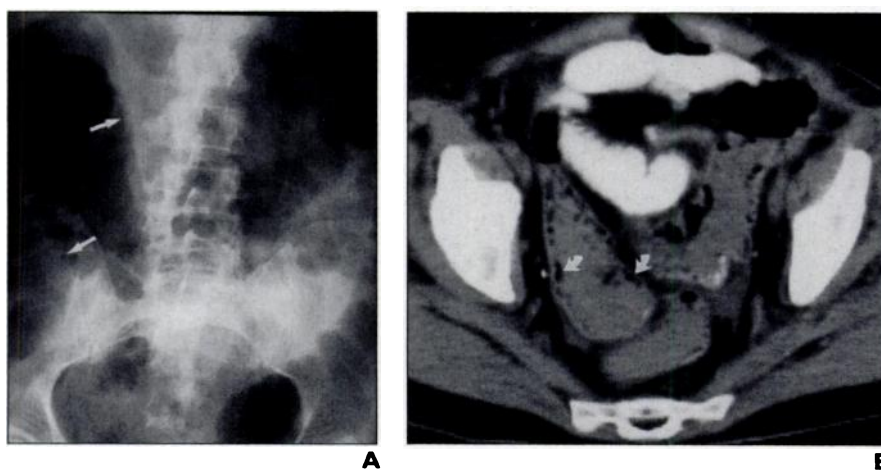
During the acute, noncicatrizing phase of Crohn's disease, the small bowel and colon

Fig. 6.—CT scan–plain film correlation for toxic megacolon complicating Crohn's disease.
A, Plain abdominal radiograph shows diffuse dilatation of colon with blunted haustral folds (*arrows*).
B, CT scan obtained on same day as **A** shows unsuspected intramural gas collections in colon (*arrows*). Patient subsequently underwent colectomy on basis of this finding.

show mural stratification and often have a target or double-halo appearance [28, 29] (Fig. 7). As in ulcerative colitis, a ring of soft-tissue density (corresponding to mucosa) is surrounded by a low-density ring with an attenuation near that of water or fat (corresponding to submucosal edema or fat infiltration, respectively), which in turn is surrounded by a higher-density ring (muscularis propria) [21]. Inflamed mucosa and serosa may show significant contrast enhancement after bolus IV administration of contrast material, and the intensity of enhancement correlates with the clinical activity of the disease.

Two recent papers in the sonography literature have shown that the sonographic demonstration of mural stratification (i.e., the ability to visualize distinct mucosal, submucosal, and muscularis propria layers) indicates that transmural fibrosis has not occurred and that medical therapy may be successful in ameliorating luminal compromise [30, 31]. These studies also have shown that before the onset of fibrosis, the edema and inflammation of the bowel wall that cause mural thickening and luminal obstruction are reversible to some extent [30, 31]. A modest decrease in bowel wall thickness often produces a dramatic increase in luminal cross-sectional area and resolution of the patient's symptoms of obstruction. These studies also have shown that the loss of mural stratification is indicative of transmural fibrosis [30, 31]. This has been our experience with CT (Figs. 7 and 8) as well.

Chronic Phase: Transmural Fibrosis and Cicatrization

In patients with long-standing Crohn's disease and transmural fibrosis, mural stratification

is lost, so that the affected bowel wall typically shows homogeneous attenuation on CT scans (Fig. 9). Homogeneous attenuation of the thickened bowel wall in the presence of good intravascular contrast material levels on thin-section scans suggests irreversible fibrosis; therefore, antiinflammatory agents may not provide a significant reduction in bowel wall thickness. If the affected segments become sufficiently narrow, surgery or stricturoplasty will be necessary to relieve the patient's obstruction.

Mesenteric Involvement

Palpation of an abdominal mass or separation of bowel loops on small-bowel series in patients with Crohn's disease evokes an extensive differential diagnosis: abscess, phlegmon, fibrofatty proliferation or creeping fat of the mesentery, bowel wall thickening, and enlarged mesenteric lymph nodes [32–35]. Each of these disorders has significantly different prognostic and therapeutic implications. This diagnostic dilemma is further complicated by the fact that many patients are receiving immunosuppressive therapy that can mask signs and symptoms. CT can readily differentiate the extraluminal manifestations of Crohn's disease [28, 36].

Fibrofatty proliferation of the mesentery.— Fibrofatty proliferation, also known as creeping fat of the mesentery, is the most common cause of separation of bowel loops on small-bowel series in patients with Crohn's disease [3, 7]. On CT scans (Fig. 10), the sharp interface between bowel and mesentery is lost and the attenuation value of fat is elevated by 20–60 H because of the influx of inflammatory cells and fluid. Mesenteric adenopathy, with lymph node size ranging from 3 to 8 mm, also may be present (Figs. 9B and 11). When the lymph nodes are larger than 1 cm, the presence of lymphoma or carcinoma, both of which occur with greater frequency in Crohn's disease, must be excluded [26, 37].

Contrast-enhanced CT scans often show hypervascularity of the involved mesentery, manifesting as vascular dilatation, tortuosity, prominence, and wide spacing of the vasa recta (Fig. 11). These distinctive vascular changes have been called vascular jejunization of the ileum or the comb sign [33]. Identification of such hypervascularity should suggest active disease and may be useful in differentiating Crohn's disease from lymphoma or metastases, which tend to be hypovascular lesions [33].

Abscess.—About 15–20% of patients with Crohn's disease eventually develop an intraabdominal abscess [38, 39]. Abscesses are most frequently associated with small-bowel disease or ileocolitis. Once developed, an abscess can burrow through the adjacent tissue or break open and drain spontaneously into another part of the bowel and/or adjacent organs. Abscesses usually result from sinus tracts, fistulas, perforations, or surgical operations for Crohn's disease [40].
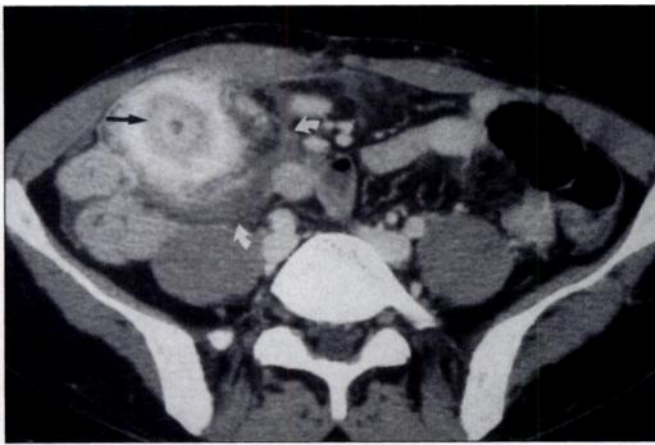

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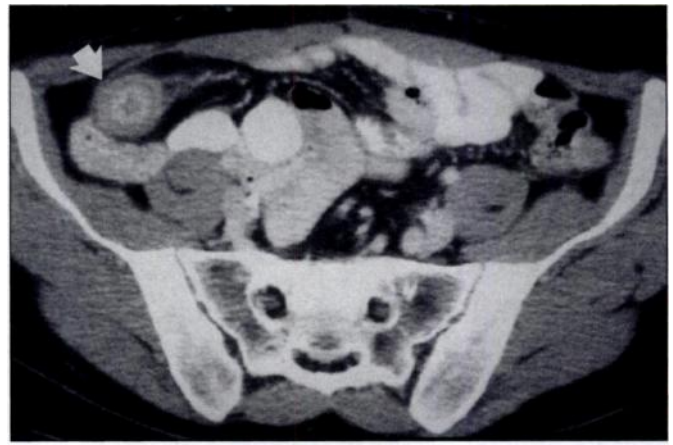
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Fig. 7.—Target sign in acute, noncicatrizing phase of Crohn's disease.
A, Contrast-enhanced CT scan of terminal ileum shows intense enhancement of mucosa and muscularis propria–serosa. Mural stratification is present as edematous, thickened submucosa of low attenuation (*straight arrow*) in relation to other bowel wall layers, producing target sign. Periintestinal fat also shows marked inflammatory change (*curved arrows*). Patient had low-grade small-bowel obstruction and fever.
B, CT scan obtained for same patient as in **A** after 9-week course of antibiotic and steroid therapy shows diminished mural thickening and contrast enhancement of involved ileum (*arrow*) as well as marked reduction of inflammatory change in adjacent mesentery. Patient's symptoms of obstruction improved as well.
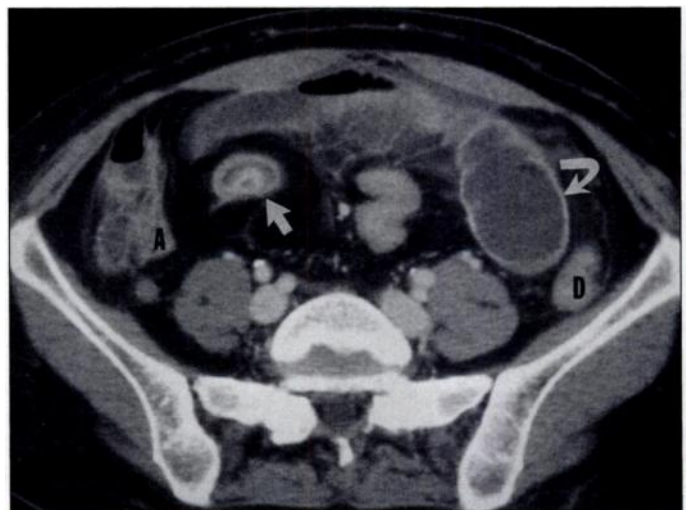
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Fig. 8.—Small-bowel obstruction attributable to Crohn's disease of distal ileum. Contrast-enhanced CT scan shows luminal narrowing and mural thickening of distal ileum (*straight arrow*) causing dilatation of fluid-filled small bowel proximally (*curved arrow*) and collapsed ascending colon (A) and descending colon (D) distal to diseased ileum. Mural stratification of involved segment is maintained. This ability to visualize various layers of bowel wall suggests that stenosis may be reversible with medical therapy.
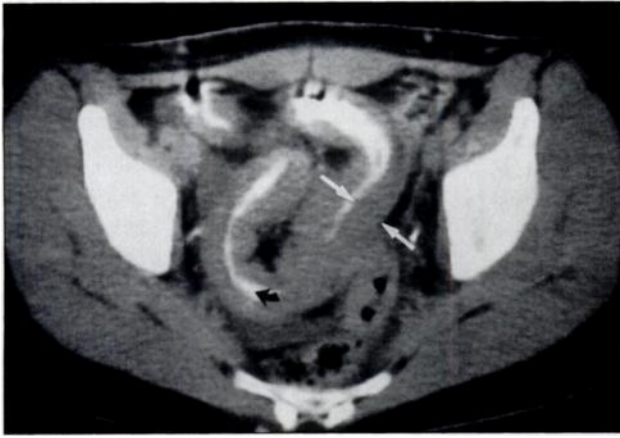
A   **B**

Fig. 9.—Chronic, cicatrizing phase of Crohn's disease in which loss of mural stratification often indicates irreversible transmural fibrosis.

A, Pelvic CT scan shows homogeneous mural thickening (*straight arrows*) of distal ileum associated with luminal narrowing (*curved arrow*) attributable to chronic, cicatrizing fibrosis.

B, CT scan in another patient shows deep ulcers penetrating otherwise homogeneously thickened walls of ascending colon (*small curved arrow*). Terminal ileum (*straight open arrow*) and descending colon (*straight solid arrow*) also are involved. Note increased linear and nodular densities in mesenteric fat around inflamed intestinal segments (*large curved arrow*). Also note prominent lymph nodes seen in small-bowel mesentery.
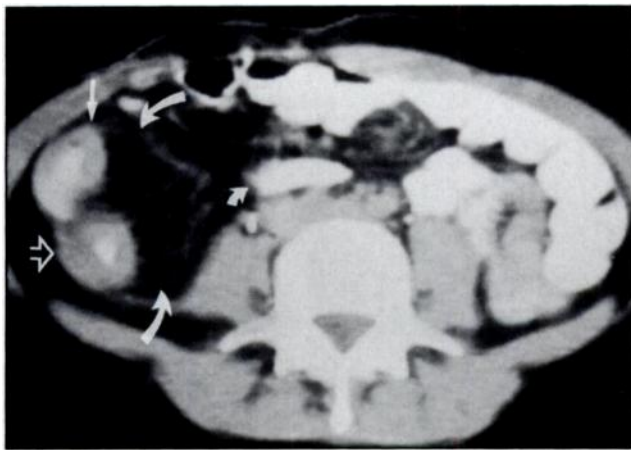
Fig. 10.—Creeping fat of mesentery in Crohn's disease. CT scan shows homogeneously thickened walls of ileum (*straight open arrow*) and ascending colon (*straight solid arrow*). On their medial aspects, note large amount of mesenteric fat with increased number of strandlike densities (*large curved arrows*). Also note separation of normal small-bowel loop (*small curved arrow*) from these diseased segments; this separation has been caused by abnormal mesenteric fat.
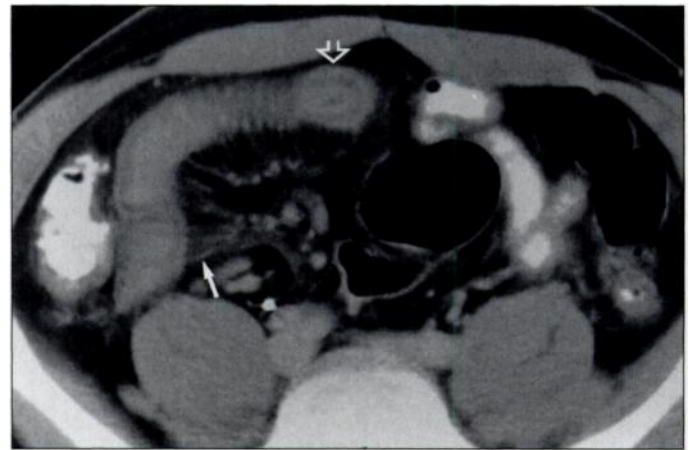
Fig. 11.—Vascular engorgement of ileal mesentery associated with active Crohn's disease. CT scan shows vascular dilatation, tortuosity, and prominence of vasa recta (*solid arrow*) on mesenteric border of ileum, known as comb sign. Diseased ileal loop shows mural thickening and target appearance (*open arrow*). Also note prominent mesenteric lymph nodes.

An intraabdominal abscess may be difficult to diagnose on clinical grounds in patients with Crohn's disease because symptoms may be inconspicuous, masked by corticosteroids, or mistaken for an exacerbation of disease. Barium studies and endoscopy typically can only suggest the presence of an abscess indirectly by a mass effect, spiculation of the mucosa, or identification of a fistula. Also, these studies do not evaluate the ischiorectal fossa, psoas muscle, and solid abdominal organs, common locations of abscess formation [32]. Cross-sectional imaging is required to confirm the diagnosis

and show the full extent and location of the abscess cavity (Fig. 12).

CT is the procedure of choice for both the diagnosis and the percutaneous management of intraabdominal abscesses (Fig. 13). On CT scans, abscesses usually appear as circumscribed round or oval water-density masses with an attenuation of 10–30 H. If the abscess has a well-formed capsule, it may show peripheral contrast enhancement, whereas the liquefied central area of the abscess, which may contain necrotic material, does not. Extraluminal bubbles of air are

important diagnostic features seen in 30–50% of cases [41]. These bubbles may be formed by gas-forming bacteria but most often are secondary to a sinus tract communicating with the skin surface or the gastrointestinal tract. The gas may be distributed as finely dispersed air bubbles throughout a collection or as an air–fluid level.

The treatment of abscesses in patients with Crohn's disease is difficult. When surgically drained, these abscesses reform in 40–90% of cases, with a death rate of 2–4% [42]. In the past, there was a reluctance to percutaneously
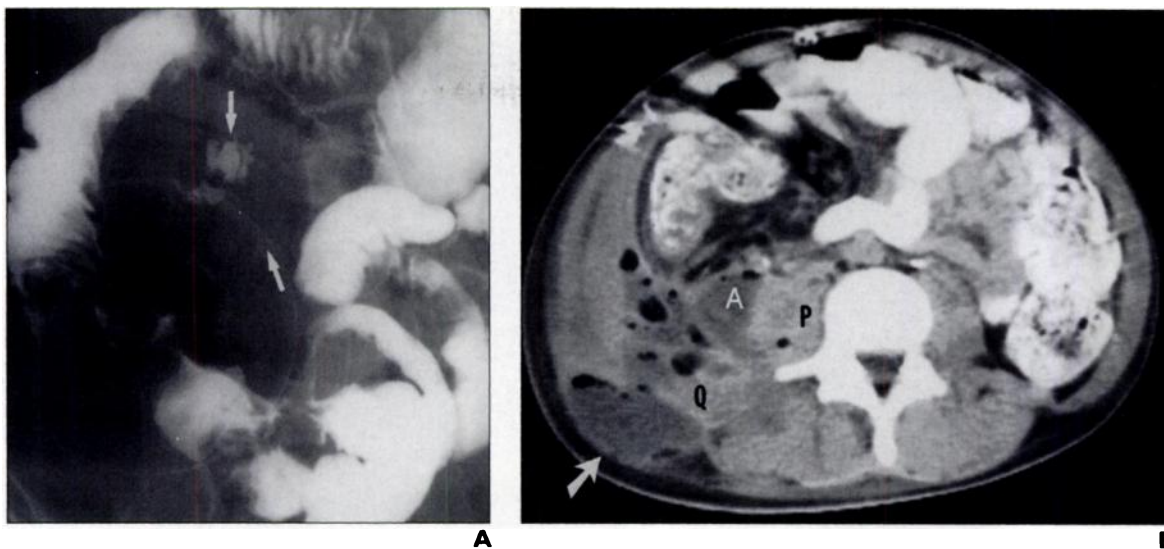
Fig. 12.—Small-bowel series–CT correlation for abscess formation complicating Crohn's ileocolitis.
A, Spot film from small-bowel series shows deformed and narrowed ileocecal region, with extraluminal barium in fistulas (*arrows*). This study provides no hint of large flank abscess.
B, CT scan shows full extent of abscess (A), which involves right retroperitoneal space and adjacent musculature (P = psoas, Q = quadratus lumborum) and extends extraperitoneally into soft tissues of right flank (*arrow*).

drain abscesses in patients with Crohn's disease because these abscesses often have accompanying fistulas and may be multilocular. Recent studies, however, have shown great success in the percutaneous management of these abscesses, either as a temporizing measure or, more often, as definitive therapy [43–47]. Two-stage surgical resections that initially bypass the diseased gut often can be simplified into single-stage surgery. To date, no enterocutaneous fistulas resulting from catheter drainage have been reported.

CT-guided percutaneous abscess drainage may obviate the need for surgery in many cases. Because 70–90% of patients with regional enteritis eventually will require surgery, avoiding an operation for abscess drainage is a tangible benefit of CT [42].

Phlegmon.—A phlegmon is an ill-defined inflammatory mass in the mesentery or omentum that may resolve completely with antibiotics or progress to form an abscess. A phlegmon is another common cause of a mesenteric mass effect in patients with Crohn's disease [7]. On CT scans, a phlegmon produces a loss of definition of surrounding organs and a smudgy or streaky appearance of the adjacent mesenteric or omental fat (Fig. 14).

Fistulas and sinus tracts.—Fistulas and sinus tracts are hallmarks of Crohn's disease, affecting approximately 20–40% of patients [7]. The morphology and anatomic sites of fistulas are protean: enteroenteric, enterocolic, colocolic, enterovesical, enterovaginal, enterocutaneous,

anorectal, duodenopancreatic, gastrocolic, colobronchial, and enterospinal [48–54].

The origin, anatomic course, and sites of communication of sinus tracts and fistulas should first be evaluated by conventional barium studies, excretory urography, cystography, and sinography. These studies, however, may be limited by the fact that the origin of the fistula may be edematous and prevent contrast opacification. Additionally, tiny fistulas may not be seen on barium studies, and evaluation of anorectal fistulas and sinus tracts on such studies often is painful and difficult. CT often is required to show the full extent of these tracts (Fig. 15).

When surgical or percutaneous intervention is planned for symptomatic fistulas, cross-sectional imaging should be done to define precisely their anatomic relationships. Such definition is particularly important for anorectal lesions.

Rectal and Perianal Pathology

Perianal disease is common in patients with Crohn's disease. Unfortunately, the perianal area often is poorly evaluated by barium studies because the enema tip may be inserted too cephalad or because exquisite anal tenderness may preclude adequate retrograde distension and evaluation. In a recent
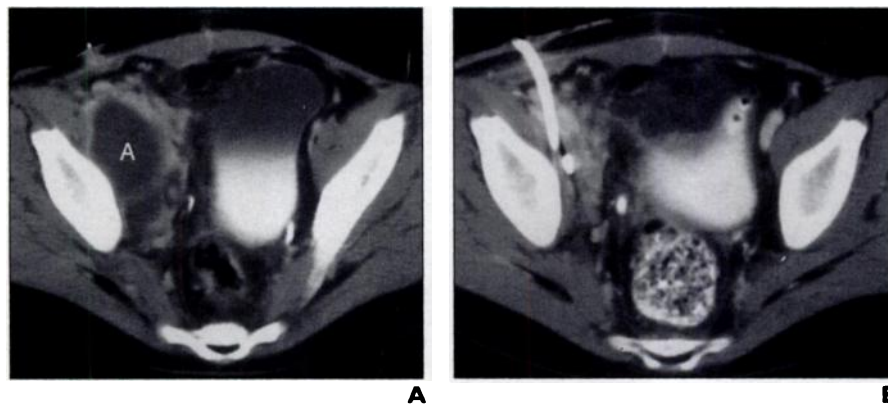
Fig. 13.—CT-directed percutaneous abscess drainage in Crohn's disease.
A, CT scan shows extraperitoneal abscess (A) along posteromedial aspect of right iliopsoas muscle.
B, CT scan obtained after percutaneous insertion of catheter shows complete drainage of abscess.
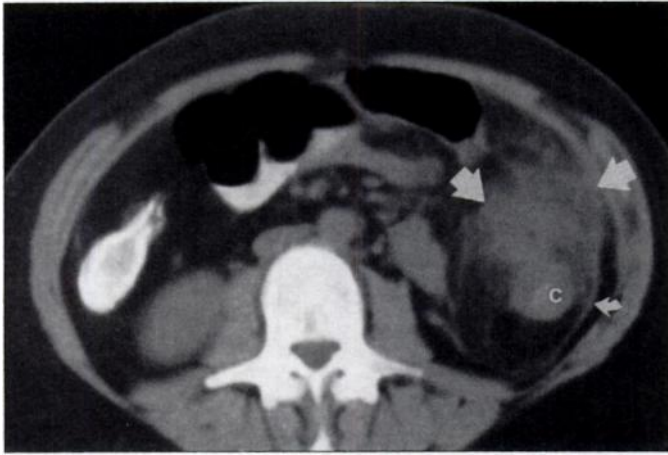
Fig. 14.—Phlegmon associated with Crohn's disease. Unenhanced CT scan shows homogeneous mural thickening of descending colon (c), pericolic phlegmon (straight arrows), and fascial thickening (curved arrow).

their series [55]. MR imaging does offer certain advantages in evaluating anorectal disease by virtue of its ability to directly image fistulas and sinus tracts in the sagittal and coronal planes [56–58]. Transrectal sonography has proven superior to CT in determining the depth of mural involvement in the anorectum and the integrity of the anal sphincter in patients with Crohn's disease [59, 60].

### Neoplasms Complicating Crohn's Disease

Patients with chronic Crohn's disease show an increased incidence of adenocarcinoma and lymphoma of the small bowel and colon, particularly in the bypassed or excluded segments of the gut [37]. Because these bowel loops cannot be studied by conventional barium or endoscopic techniques, CT is helpful in detecting a tumor mass and providing accurate tumor staging.

study, CT showed perirectal–perianal abnormalities in 82% of patients: inflammation of fat planes in the perirectal region and ischiorectal fossa (73%), rectal wall thickening (30%), fistulas or sinus tracts (22%), and abscesses (14%) [55] (Fig. 16). The authors

emphasized that in patients with Crohn's disease, the plane of scanning must continue caudal to the symphysis to include the entire perineum because significant disease was found beneath the level of the symphysis pubis in more than one-third of patients in

Differential Diagnosis of IBD

The differentiation of granulomatous colitis and ulcerative colitis is important in terms of medical management, surgical options, and
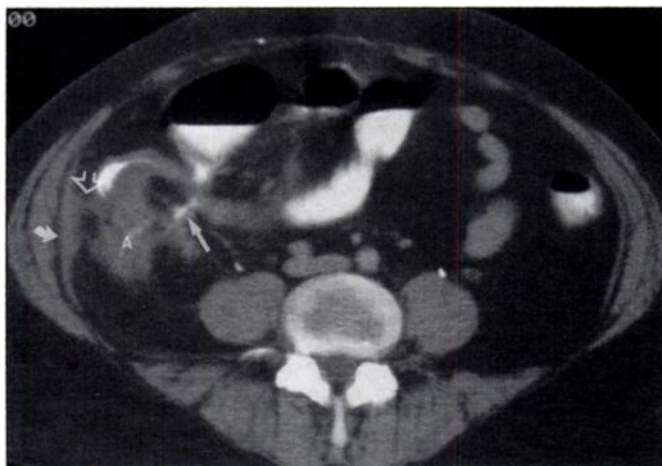
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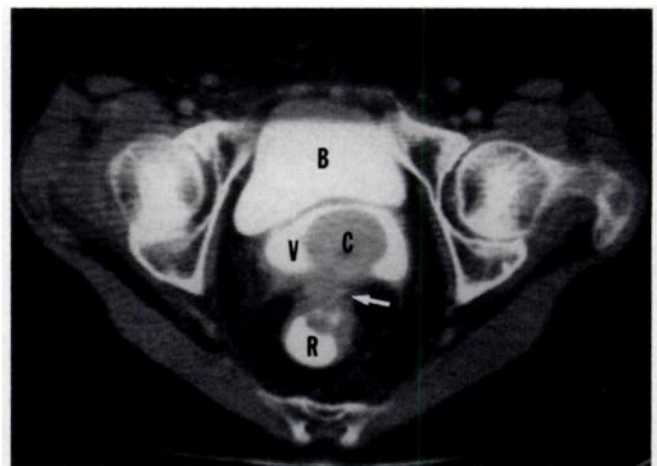
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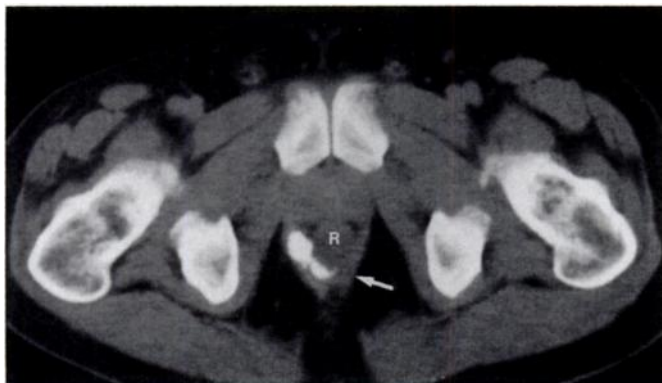
C

Fig. 15.—Fistulas and sinus tracts in three patients.
A, CT scan shows abscess (A) that is associated with fistula to colon (straight solid arrow) and sinus tract (straight open arrow) and that extends into thickened transversus abdominis muscle (curved arrow).
B, Pelvic CT scan shows rectovaginal fistula (arrow). B = bladder; V = contrast material–filled vagina; C = cervix; R = rectum.
C, CT sinogram obtained before surgery shows relationship of patient's rectocutaneous fistula to levator ani muscles (arrow) and rectum (R).

Fig. 16.—Anorectal pathology secondary to Crohn's disease in three patients.
A, CT scan of pelvis shows circumferential, homogeneous mural thickening of rectum (R). Note phlegmon (*arrow*) extending into left perirectal fat. U = uterus.
B, CT scan shows perirectal abscesses (*solid arrows*) associated with sinus tract extending into right buttock (*open arrow*).
C, CT scan of pelvis shows presacral abscess (*curved arrows*) attributable to fistula (*straight arrow*) from rectum (R).

prognosis. This distinction usually can be made on the basis of colonoscopy with biopsy, histologic studies, double-contrast barium enema, determination of disease distribution, and clinical course. CT occasionally can help distinguish these disorders by showing differences in wall thickness, wall density, distribution of colonic involvement, and the presence or absence of small-bowel disease, abscesses, fistulas, and mesenteric fibrofatty proliferation (Table 1).

Idiopathic IBD also must be differentiated from infectious, ischemic, and radiation enterocolitides. A recent study showed considerable overlap in the CT findings for these disorders; however, certain defining features were found [16]. The presence of ascites was more suggestive of an acute rather than a chronic cause of colonic inflammation. Peritoneal fluid was commonly found in acute colitides, particularly pseudomembranous, infectious, and ischemic colitides, and not in chronic IBD. Ascites was only infrequently seen in patients with acute IBD. Submucosal fat deposition on CT scans was primarily found in subacute and chronic colitides, usually ulcerative colitis, and not in acute colitis [16].

Extraintestinal Complications of IBD

Extraintestinal complications develop in one-quarter to one-third of patients with IBD [61]. These complications can be divided into three categories: (1) those that are intimately related to the activity or extent of disease and that are responsive to therapy directed at the bowel disease (e.g., arthritis or iritis); (2) those whose course is independent of the severity of the underlying bowel disease (e.g., sclerosing cholangitis or ankylosing spondylitis); and (3) those that result from inadequate or disordered intestinal function (e.g., cholelithiasis or nephrolithiasis) [61].

Hepatobiliary Complications

The most frequent, serious complications of extraintestinal IBD occur in the liver and the biliary tract [62–65]. As a rule, these complications do not correlate with disease activity, duration, or severity, with the exception of fatty infiltration, which tends to occur in patients who are more seriously ill, debilitated, and malnourished or who are on hyperalimentation therapy.

Fatty liver is found on liver biopsy in 20–50% of patients with IBD and may be caused

by fat malabsorption, hyperalimentation, sepsis, protein-losing enteropathy, malnutrition, and corticosteroids. CT is an excellent means of showing hepatic steatosis noninvasively [62].

About 30–50% of patients with Crohn's disease develop gallstones, especially in the presence of extensive terminal ileal disease or after ileal resection. These patients form lithogenic bile as a consequence of bile salt malabsorption or loss through the enterohepatic circulation [62]. Although sonography is the primary means of detecting gallstones, CT is superior for detecting choledocholithiasis.

Primary sclerosing cholangitis occurs in 1–4% of patients with ulcerative colitis and less commonly in those with Crohn's disease [64]. Although the sensitivity of CT for the diagnosis of sclerosing cholangitis has not been established, CT can directly visualize the fibrous mural thickening of the larger bile ducts that characterizes this disease. Other CT signs suggesting sclerosing cholangitis include focal duct dilatation, discrepancy between the sizes of the intrahepatic and extrahepatic bile ducts, focal clustering of intrahepatic ducts, and discontinuous areas of minimal intrahepatic bile duct dilatation (Fig. 17). These findings are best appreciated on contrast-enhanced scans. CT offers three major advantages in evaluating patients with known or suspected sclerosing cholangitis [66]. First, it is a noninvasive technique, an important consideration for patients who need multiple serial examinations. Second, CT can visualize the entire biliary tract in cases in which strictures obstruct the flow of contrast medium during cholangiography, occasionally leaving large portions of the intrahepatic bile ducts unexamined. Finally,

TABLE 1	CT Features of Ulcerative Colitis and Crohn's Disease		
CT Feature	Ulcerative Colitis	Crohn's Disease	p
Wall thickness (mm) (mean ± SD)	7.8 ± 1.9	11.0 ± 5.1	<.0002
Submucosal fat (% of patients)	61	8	.0001
Isolated right colon involvement (% of patients)	0	38	<.0004
Mesenteric fibrofatty proliferation (% of patients)	0	49	<.0004
Abscess (% of patients)	0	35	<.0007

Note.—Adapted from [16].
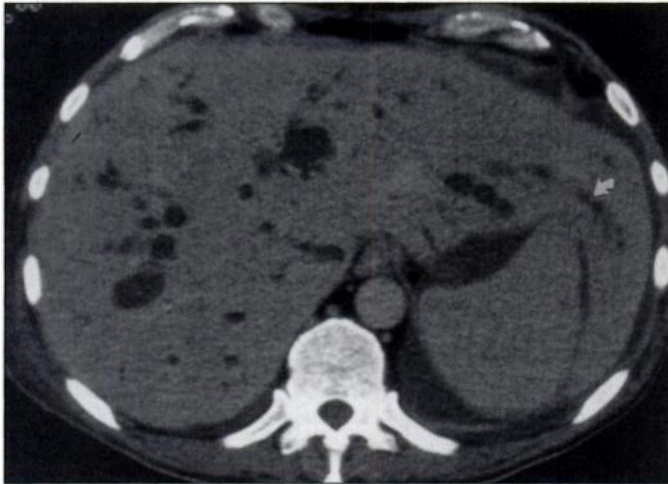
Fig. 17.—Sclerosing cholangitis associated with inflammatory bowel disease. CT scan shows focal clustering of dilated intrahepatic ducts associated with discontinuous areas of minimal intrahepatic bile duct dilatation. Note beaded appearance of left ductal system (arrow).

CT can depict complications of sclerosing cholangitis, such as secondary biliary cirrhosis and portal hypertension, as well as soft-tissue masses associated with cholangiocarcinoma. About 1.5% of patients with ulcerative colitis develop cirrhosis attributable to chronic active hepatitis, sclerosing cholangitis, pericholangitis, or any combination of these [65].

Hepatic abscesses are well-described complications of Crohn's disease [67, 68]. Hepatic abscesses most commonly develop in patients with long-standing disease but may occur as the initial manifestation of Crohn's disease. Steroids and other immunosuppressive agents, perforations, intraabdominal abscesses, and anastomotic leaks are all predisposing factors

for the development of hepatic abscesses in patients with Crohn's disease [68]. CT is the premier means of diagnosing hepatic abscesses and guiding the percutaneous drainage of suitable collections (Fig. 18).

Pancreatic Complications

Approximately 1–2% of patients with Crohn's disease develop pancreatitis attributable to a variety of causes: (1) drugs, such as steroids, azathioprine, and metronidazole; (2) choledocholithiasis; (3) fistula from the adjacent gut; (4) sclerosing cholangitis; (5) sphincter of Oddi dysfunction or stenosis of the descending duodenum leading to obstruction of the duct or reflux of duodenal

contents into the duct; and (6) autoantibodies against pancreatic acinar cells [69, 70]. Regardless of the cause, CT is needed to help confirm the diagnosis of pancreatitis and, more importantly, its complications.

Urinary Tract Complications

Between 2% and 10% of patients with Crohn's disease develop nephrolithiasis attributable to water and electrolyte losses from diarrhea, malabsorption, and high ileostomy output [71, 72]. Oxalate stones are the most common stones to develop but may not be sufficiently dense to be visible with conventional radiology techniques.

Hydronephrosis may develop in patients with Crohn's disease for a variety of reasons: calculous disease, obstruction attributable to the inflammatory effect of an abscess (Fig. 19) or phlegmon, or the mass effect of creeping fat of the mesentery encroaching on the ureter. CT is useful in detecting both hydronephrosis and the obstructing mass or stone.

Fistulas may develop between the diseased gut and the kidney(s) in patients with Crohn's disease, leading to a renal or perinephric abscess. More commonly, enterovesical fistulas develop [48, 72]. Conventional studies detect fewer than 50% of enterovesical fistulas, whereas CT has a nearly 90% success rate [72]. The presence of air in the bladder in the absence of a Foley catheter or previous instrumentation is diagnostic of an enterovesical fistula.

Musculoskeletal Complications

Arthritis, one of the most common extraintestinal manifestations of IBD, is manifested as peripheral arthritis, sacroiliitis–spondylitis,

A

B
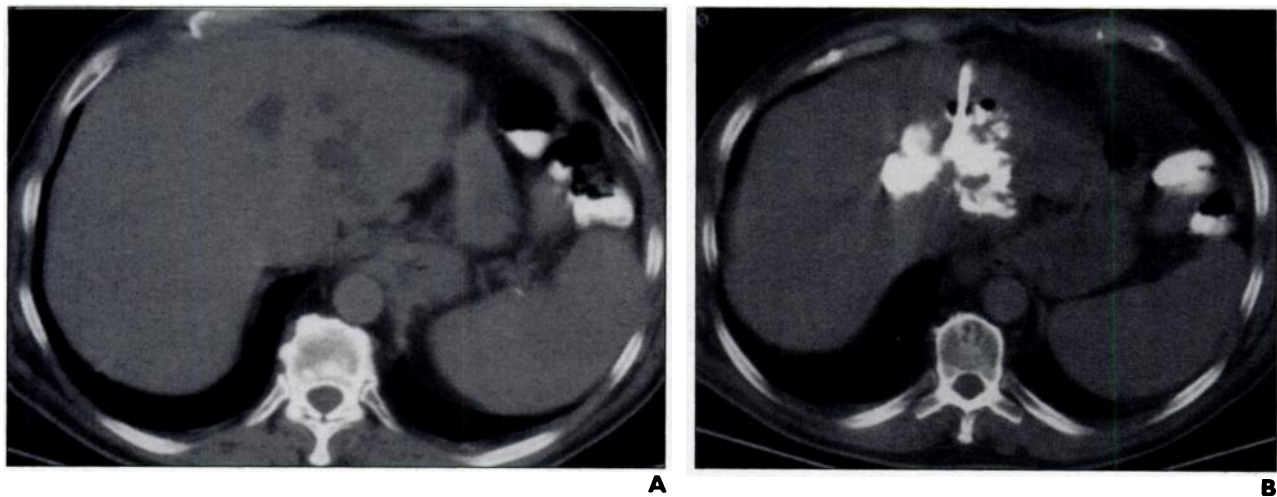
Fig. 18.—Liver abscess complicating Crohn's disease.
A, CT scan shows multiple areas of low attenuation in left hepatic lobe.
B, CT scan taken after injection of contrast material through percutaneous drainage catheter shows multilocular liver abscess.
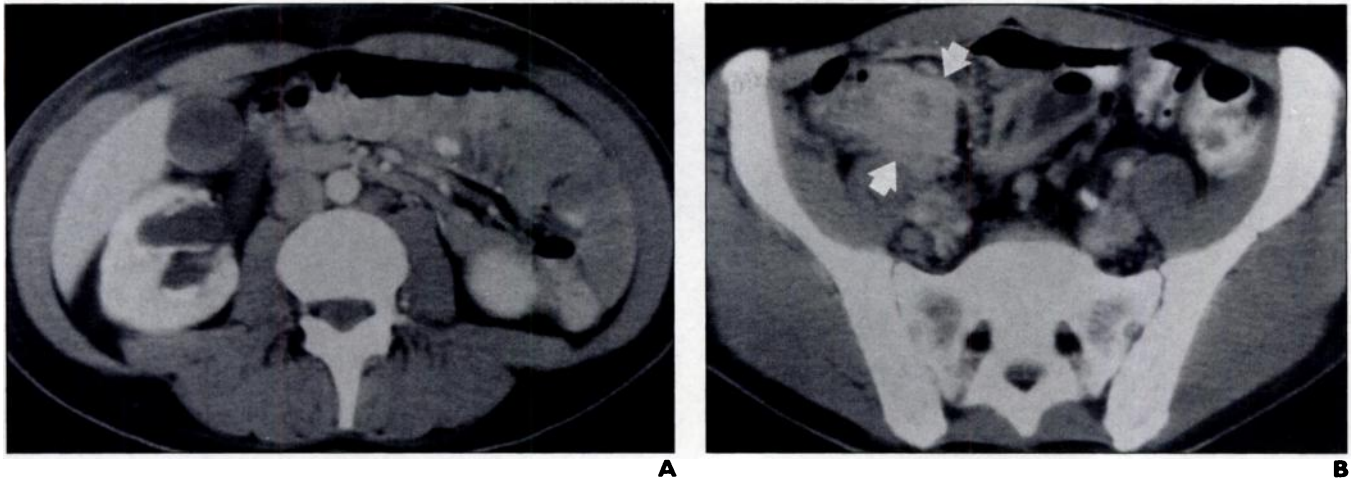
Fig. 19.—Crohn's disease abscess causing hydronephrosis.
A, CT scan of midabdomen shows right-sided hydronephrosis.
B, CT scan of iliac fossae shows right lower-quadrant abscess (*arrows*) secondary to Crohn's ileitis and causing right ureteral obstruction.
C, Retrograde pyelogram confirms level of right ureteral obstruction (*arrow*).

or both. The radiologic findings of enteropathic peripheral arthritis usually are minimal and are best seen, if at all, on conventional radiographs. IBD-related changes in the axial skeleton are similar to changes seen with ankylosing spondylitis [73–75] (Fig. 20). CT can detect the subtle changes of early sacroiliitis before they become apparent on plain films: bilateral, usually symmetric joint narrowing with osseous erosions and then sclerosis, more pronounced on the iliac side of the articulation. Between 10% and 30% of patients with IBD have symptomatic sacroiliitis; some of them experience significant disability [76]. In a recent study, CT showed the presence of asymptomatic sacroiliitis in 32% of patients with IBD, although it was identified by plain film radiography in only 10% of patients [77]. The CT identification of asymptomatic sacroiliitis may enable the earlier introduction of therapy to inhibit disease progression or permit the earlier diagnosis of IBD in a previously

Fig. 20.—Crohn's disease–associated ankylosing spondylitis. Pelvic CT scan photographed at bone window shows partial bony ankylosis of right sacroiliac joint (*straight arrow*). Note mural thickening (*curved arrows*) of distal ileal loop.
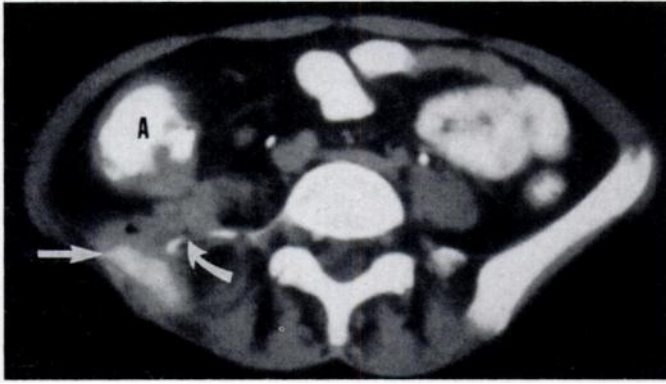
Fig. 21.—Osteomyelitis of right iliac crest complicating Crohn's ileocolitis. CT scan shows mural thickening of ascending colon (A), with retrocolic extension of inflammatory change (*curved arrow*) into pelvic wall and destruction of right iliac bone (*straight arrow*).

unidentified host. There appears to be no correlation between the presence of sacroiliitis and the type, duration, or severity of IBD [77].

Osteonecrosis has been reported as a rare complication of IBD in the following clinical settings: during or after corticosteroid therapy; during total parenteral nutrition, especially with lipid emulsions; and, most recently, as a direct complication of the disease without other precipitating factors [78, 79]. CT studies often can show early signs of avascular necrosis before plain film studies. CT abnormalities include subtle alterations in the trabecular pattern, joint space integrity, femoral head contour, and acetabulum, which may be undetectable or poorly defined on plain films. However, MR imaging is the best technique for establishing the diagnosis of avascular necrosis.

Septic arthritis of the hip can complicate a psoas muscle abscess or a retroperitoneal abscess progressing through the greater sciatic notch. MR imaging and CT show these changes before they can be recognized on plain films.

The iliac bone (Fig. 21) and the sacrum are the most frequent sites of osteomyelitis in patients with Crohn's disease [80, 81]. Iliac and sacral bone infections are almost invariably the result of an adjacent pelvic abscess or enteric fistula, so the CT diagnosis of osteomyelitis and pelvic abscess usually is made concurrently. CT findings for osteomyelitis include cortical bone destruction, intraosseous gas, increased attenuation of the bone marrow, narrowing of the medullary cavity, serpentine drainage tracts, and the presence of an involucrum or sequestrum.

A spinal epidural abscess from fistulization of a presacral or psoas muscle abscess in patients with Crohn's disease has been reported [82]. Prevertebral, intraforaminal, and epidural gas may be seen on CT scans.

Crohn's disease complications now account for 73% of all psoas muscle abscesses [83]. A

right psoas muscle abscess may develop secondary to terminal ileal disease, and a left psoas muscle abscess can result from sigmoid or jejunal involvement. Most patients with psoas muscle abscesses have well-established Crohn's disease, but the clinical manifestations may be nonspecific. Occasionally, psoas muscle abscesses are seen at the initial presentation of Crohn's disease. CT has emerged as the best technique for the diagnosis and percutaneous management of psoas muscle abscesses [84, 85].

Primary rectal sheath abscesses also have been reported as a complication of Crohn's disease and may be diagnosed and treated with percutaneous drainage under CT guidance [86].

Conclusion

CT is the premier imaging procedure for evaluating the mural and extraintestinal manifestations of IBD. Although barium studies have been and remain the primary radiographic means of diagnosing IBD, CT plays a critical role in detecting abscesses, in differentiating various causes of mesenteric abnormalities, and in discovering the extraintestinal complications that so often afflict IBD patients.

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