Right lower quadrant pain in the immunocompromised patient: diagnostic imaging

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Learning objectives

Clinical entities presenting with acute abdomen in immunocompromised patients have particular characteristics because apart from common diseases as in the general population, they have an increased frequency of certain unusual processes and both clinical and laboratory tests may be harmless even if it is a severe disease.

For all these facts imaging tests become especially important in these cases.

We present a review of their radiological findings for the proper characterization and differential diagnosis of this pathologies

Background

We reviewed the imaging in pathologies that cause acute pain in FID, focusing on those particularly important in immunocompromised patients. All these entities have common radiological findings which makes essential to guide their diagnosis the knowledgement of the medical history and the treatment received by the patient supported by radiological tests.

Neutropenic colitis:

Neutropenic enteritis syndrome represents a disease whose etiology is multifactorial and is characterized by a transmural hemorrhagic necrotic inflammation of the that occurs in immuno suppressed patients, usually with severe neutropenia. The drugs commonly used in cancer treatments involved in its etiology are 5-FU, paclitaxel and docetaxel chemotherapy mainly but any immunosuppresor agent may produce it.

Radiologically it is characterized by a circumferential mural thickening of the colon, especially the right, affecting more often the cecum, which has low attenuation edema, associating stranding of pericolonic fat. In addition there may be ascites, dilated bowel loops due to paralytic ileus or cecal pneumatosis. (Figures 1 and 2)

The small intestine can be also affected. (Figure 3)

Traditionally the treatment was always surgical but now conservative treatment is preferred (antibiotics, supportive and rest of the digestive tract).
**Pseudomembranous colitis:**

It is an infectious entity due to Clostridium difficile, which is a Gram (+), anaerobic spore-forming bacillus. Pathogenic *C. difficile* strains produce several known toxins.

Its pathogenesis is due to the production of exotoxin A (enterotoxic) and B (cytotoxic) which are necessary for the diagnosis. It is mainly associated with the use of antibiotics, which destroy the normal gastrointestinal flora, it appears after 5-10 days of antibiotic therapy onset, but it can occur at any time, even 10 weeks after cessation of the treatment.

Diagnosis is made by demonstrating the presence of toxins in feces or with stool culture and can also rely on colonoscopy (that is very nonspecific) and CT, which should be performed with oral and IV contrast.

It is characterized by a large mural thickening (often more than 10mm), frequently in the form of diffuse pancolitis and wall nodularity. (Figure 4) In CT studies the most specific sign of colitis by *C. difficile* but less frequent, is the accordion sign, shown by the oral contrast trapped in the folds of the intestinal edematous nodular mucosa (Figure 5).

In addition is frequently accompanied by stranding of fat and ascites as other common entities in immunocompromised patients. The small intestine is very rarely affected and is more common in the rectum, sigmoid and descending colon. Treatment ranges from the suppression of the antibiotic or undergoing with metronidazole therapy or vincristine in severe cases. Surgery is rarely needed.

**Toxic megacolon:**

A rare but serious complication of inflammatory bowel disease and pseudomembranous colitis. It is also associated to treatment with vincristine. Radiographically is characterized by a marked colonic distension, especially in the transverse colon. (> 5.5 cm) and edema with thickening of the colonic wall (which measure is 4 mm in normal conditions), with loss of the normal haustral pattern. (Figures 5 and 6)

**Pneumatosis intestinalis:**

Pneumatosis intestinalis has been described associated with numerous chemotherapeutic agents and especially bevacizumab. It is secondary to an increased permeability of the mucosa in connection with immunosuppression.

Patients are usually asymptomatic, being an incidental finding. Most cases are solved by removing the injurious agent. Subserosal or submucosal gas in the intestinal wall is radiologically seen appearing mainly as cystic collections (Figures 7 and 8)
**Ischemic colitis:**

It is due to intestinal necrosis being the patient very symptomatic. It is a surgical emergency characterized by bowel wall thickening with gas in the submucosa or subserosa with cystic or band distribution with intense mucosal enhancement associated in a reversible phase and there is no enhancement when the schema is fully established.

The ischemia associates bowel dilatation, ascites and other signs as venous occlusion, arterial, porto mesenteric gas, etc. It can occur in any part of the intestine. (Figures 10 and 11)

**Diarrhea post-chemotherapy:**

Gastrointestinal enteritis secondary to chemotherapy is mainly associated with bowel toxicity due to treatment with 5-FU, floxuridine, irinotecan, cetuximab, EGFR agents and VEGF receptor agents. (Figure 12)

**Enteritis:**

It is a complication of radiotherapy for cancer of the rectum, cervix, uterus, prostate, bladder or testicles that can affect the small intestine or the bulk. It is usually progressive and can be acute or chronic.

The acute form usually occurs during the application of radiotherapy or shortly after it (3-6 weeks) and it manifests as diarrhea, abdominal cramps and nausea. These symptoms are due to transient inflammatory phenomena and are usually solved within 2-6 weeks of stopping the treatment. The diagnosis of acute radiation enteritis is clinical and requires no additional exploration.

In imaging it is characterized by an intense enhancement, edema and thickening of the bowel loops wall. (Figure 13)

The chronic form may appear months or years after receiving radiotherapy and is mainly characterized by fibrotic irreversible and potentially serious phenomena. Chronic enteritis appears, in general, six months after stopping the treatment. Proctitis usually occurs in about 1 year after stopping the treatment, and for enteritis the latency period is often longer, with a mean of 5 years, in any case the time between treatment and enteritis may vary between 2 months and 30 years. The clinic depends on the affected bowel segment.

The rectum and sigma are one of the most frequently damaged segments by radiotherapy. The proctitis or proctosigmoiditis due to radiation causes diarrhea with tenesmus, urgency and distal hematochezia due to the presence of telangiectatic lesions that easily bleed.
The involvement of the small intestine may be severe and lead to intestinal obstruction episodes due to stenosis, perforation and intra-abdominal abscesses, bacterial overgrowth, fistulas, bleeding or malabsorption in cases of extensive involvement. A review of the patient history to know the characteristics of the treatment received is essential for making the diagnosis of radiation enteritis.

If radiation enteritis is suspected small bowel transit (preferably with enteroclysis) and computed tomography are the techniques of choice.

Chronic intestinal lesions secondary to radiation are irreversible and lead to fixed intestinal segments with stenosis and decreased distensibility. (Figure 14)

**Graft versus host disease (GVHD):**

Occurs after a bone marrow transplant while the patient is still neutropenic. It is important to differentiate it from other gastrointestinal complications because its treatment also involves immunosuppression. Its diagnosis is anatomic pathological. In imaging techniques it is characterized by intestinal dilation, mucosal enhancement and increase of the intestinal wall thickness that can be discontinuous. Ascites and rarefaction of perilesional fat is less frequent. No enlarged adenopathies are found. (Figures 15 and 16)

**Tumors and perforations:**

Primary lymphoma of the ileocecal region is usually presented as areas of homogeneous wall thickening with poor enhancement, other times polypoid lesions that can cause invaginations are seen. Bowel fistula is a classic complication. (Figures 17 and 18)

**Infections:**

Infections due to enteroinvasive bacteria, mycobacteria, viruses (CMV, herpes) can cause similar symptoms to acute appendicitis, especially if it involves the ileocecal area as in the case of *Yersinia enterocolitica, Campylobacter jejuni, and Salmonella enteritidis.*

The radiological findings are nonspecific, like mural thickening of the bowel wall, with homogeneous enhancement and locoregional lymph nodes. (Figures 19 and 20)

**Images for this section:**
Neutropenic colitis

Fig. 1: Intravenous contrast-enhanced CT scan in a transplantation patient shows a thickening of the cecal wall (up to 23mm), with surrounding fat stranding. There is not stenosis of the lumen.
Fig. 2: US image that shows an important symmetric thickening of the cecum with stenosis of the lumen associated and fluid in right lower quadrant.
**Fig. 3:** The patient presented with right lower quadrant pain, fever, diarrhea and he was undergoing chemotherapy. Multi-detector row CT scan shows marked circumferential cecal wall thickening with submucosal. Notice that also marked thickening of the ileal wall with surrounding fat stranding and mesenteric fluid also are seen.
**Fig. 4:** Immunocompromised patient undergoing antibiotic therapy who presents diffuse watery diarrhea and abdominal cramps. Coronal oblique reformatted and transverse intravenous contrast-enhanced CT scan images showing extensive wall thickening throughout the colon, with "thumbprinting," low attenuation from mucosal and submucosal edema, irregular mucosal contour with polypoid protrusions, pericolonic fat stranding, and ascites.
Fig. 5: Transverse CT image in a man with pseudomembranous colitis. Marked wall thickening throughout the colon is seen. The accordion sign (arrows) is seen in the transverse colon, and ascites is also noted.
**Fig. 6:** Transverse CT image shows markedly distended transverse colon (14.5 cm diameter) with an ahastral pattern and feces inside.
Fig. 7: Plain abdominal radiograph: there is gross dilatation of the transverse colon which measures approximately 10cm in maximum diameter. There is also evidence of pseudopolyp formation (black arrows).
**Fig. 8:** Patient with benign asymptomatic pneumatosis of the right colon discovered at routine imaging. He was under bevacizumab chemotherapy. Scout view and coronal CT images demonstrating air (arrows) in the wall of right colon, with normal wall enhancement. There is no intraperitoneal fluid collection.
Patient with cetuximab (for colorectal cancer) induced bowel toxicity.

Benign pneumatosis intestinalis

*Fig. 9:* Coronal oblique reformatted and transverse intravenous and oral contrast-enhanced CT scan images showing extensive pneumatosis intestinalis in the wall of ascending colon. The patient was asymptomatic.
Fig. 10: The patient after an episode of severe hypotension presented intense abdominal pain due to mesenteric ischaemia. Coronal oblique reformatted intravenous contrast-enhanced CT scan image showing cecum wall thickening with edema, thumbprinting and small amount of fluid in paracolic gutter. There is no pneumoperitoneum Angiography demonstrated a focal estenosis (78%) of the superior mesenteric artery.
Fig. 11: Transverse intravenous contrast-enhanced CT scan images showing air in the wall of ascending and transverse colon. Notice also the fat stranding and ascites in patient B.
Fig. 12: Patients immunocompromised who developed abdominal pain with new-onset diarrhea. A) Coronal contrast-enhanced CT image reveal diffuse dilatation of fluid-filled small bowel loops. Notice the small amount of fluid in right lower quadrant. (green arrow) B) Coronal contrast-enhanced CT image shows mild small-bowel wall thickening with uniform increased enhancement.
Fig. 13: Patient with a history of radiation therapy in pelvis. Axial contrast-enhanced CT scan images show diffuse bowel wall thickening, with submucosal edema and increased contrast enhancement. The lumen is preserved.
Fig. 14: Fat halo sign in chronic radiation enteritis. Intravenous contrast-enhanced CT scan demonstrates ascites and a segment of cecum with a band of lower attenuation, fat (arrow).
Fig. 15: Axial contrast-enhanced CT scan. Small bowel loops with water halo sign and marked mucous enhancement (blue arrow). Other bowel segments demonstrate a normal paper-thin wall. Ascites (green arrow)
Fig. 16: US image showing a concentric thickening of a segment of small bowel. The lumen is preserved.
**Fig. 17:** Patient with diffuse large B-cell lymphoma. Multi-detector row CT scan images show marked homogeneous and symmetric thickening of the cecal wall. There is no stenosis of the lumen. There is fat stranding, which is, however, less severe than the wall thickening. Notice a big abscess located in the right paracolic gutter. There is no fat plane of separation between the abscess, the abdominal wall musculature and the adjacent right iliopsoas muscle, which seems to be infiltrated.
**Fig. 18:** Adenocarcinoma in ascending colon. Coronal contrast-enhanced CT images showing the carcinoma and an abscess with stranding of the surrounding fat.
**Fig. 19:** Axial contrast-enhanced CT scan reveals stratified symmetric thickening of the terminal ileum. The coronal reformated scan image show large regional and mesenteric lymphadenopathies (arrows). The patient had an infection due to Tropheryma whippelii.
Infectious enteritis

Terminal ileitis due to *Campylobacter infection* in a 28-year-old patient. Multi-detector row CT scan shows marked thickening of the cecum (arrowheads) and terminal ileum (arrows) with preservation of a layered enhancement pattern. Note the small regional lymph nodes and the absence of fat stranding.

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Fig. 20
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Conclusion

Abdominal pain in the right lower quadrant in an immunocompromised patient is a potentially very serious entity, which correct diagnosis relies on imaging, especially ultrasound and CT.

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