Abdominal imaging manifestations in acquired immunodeficiency syndrome

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Learning objectives

To illustrate to CT, ultrasound, and/or MR features of acquired immunodeficiency syndrome (AIDS) population

To determine the frequency and differential diagnosis of lesions on abdominal imaging in positive HIV-population

Background

As the incidence of AIDS continues to increase and long-term survival improves, it becomes increasingly important for radiologist to understand the spectrum of abdominal disease that may affect patients with AIDS.

Abdominal disorders are a common cause of acute symptomatology in AIDS patients, and patients with AIDS frequently display nonspecific symptoms and physical examination findings. Also, symptoms may be masked by concurrent illness and diminished immune response, making accurate clinical diagnosis difficult.

AIDS patients often exhibit unusual disease processes or unusual presentations of more typical disease. In addition, an immunocompromised states places AIDS patients at risk for developing numerous opportunistic infections and neoplasm.

We revised all of the radiological reports in HIV-population at ten years old in our institution. We encountered 79 cases of abdominal pathology in this population and retrospectively reviewed two senior experienced radiologists who were unaware of radiological findings from the other.

- AIDS-related neoplasia: Kaposi sarcoma (2), non-Hodgkin lymphoma (8), and other (2)
- Mycobacterial infections: tuberculosis (7), and avium-intracellulare complex (6)
- Other bacterial infections: pseudomembranous colitis (8), typhlitis (3), bacillary angiomatosis (2)

- Viral infections: cytomegalovirus (12), herpes simplex (3), HIV (2)

- Fungal infections: candida (11), histoplasma (2)

- Protozoal infections: cryptosporidium (6), Pneumocystis carinii (5)

**Imaging findings OR Procedure details**

**AIDS-RELATED NEOPLASIA**

AIDS patients are at an extraordinary increased risk for certain neoplasm including Kaposi sarcoma (KS), non-Hodgkin lymphoma, and invasive cervical cancer. These neoplasm comprise the three established AIDS-defining malignancies.

KS and lymphoma can affect any part of the gastrointestinal (GI) tract or viscera in the abdomen and pelvis. Whereas the incidence of KS appears to be declining, the incidence of AIDS-related lymphoma (ARL) may be increasing.

Anal cancer and Hodgkin disease occur at increased rates in some populations with AIDS. Cervical and anal cancers have an increased incidence in the AIDS population due to the associated increased incidence of the sexually transmitted human papillomavirus (HPV).

**Kaposi sarcoma**

**Epidemiology**

KS is the most frequent malignancy in HIV-positive patients, occurring with an incidence of 15-50%. There is a 20,000-fold increase in incidence of KS in the HIV population, as KS rarely occurs in the general population.

KS occurs primarily in HIV-positive homosexual or bisexual men, as it appears to be related to a sexually transmitted viral infection with HIV acting as a promoter factor.
herpes virus type 8 has been found in KS tumors and is highly correlated with risk of developing KS.

Unlike opportunistic infections, there is no direct relation between decreased CD4 count and KS, because KS can occur before the CD4 count is diminished.

However, KS most frequently occurs with mild to moderate impairment for the immune system (CD4 of 200-500).

KS is much more aggressive in AIDS than in the rare case that may occur in the general population and can progress rapidly to wide-spread cutaneous and visceral involvement. There is also an increased incidence of secondary neoplasm in AIDS patients with KS.

Pathology

KS originates in the lymphoreticular system. Histological, KS tumors are composed of spindle cells, proliferative endothelial cells, extravased erythrocytes, and vascular clefts. Spindle cells are derived from lymphatic endothelium and are present in all tissues.

Consequently, KS is a multicentre disease that may involve the skin, lymph nodes, GI tract, lung liver, and spleen.

Cutaneous KS is most common and typically precedes GI disease. However, GI and visceral involvement occurs in up to 50% of patients with cutaneous disease. In addition, primary GI KS has reported and skin involvement is not a prerequisite. Up to 5% of patients with KS have GI disease without associate cutaneous involvement.

Any part of the GI tract may be affected; however, involvement of the duodenum is most common. KS occurs in the submucosal portion of the bowel; however, more advanced tumors may extend into the mucosa and may result in acute ulceration, hemorrhage, perforation, or obstruction.

As lesions are primarily submucosal, endoscopic biopsy is often of low yield, and CT diagnosis of primary lesions and associated adenopathy becomes more essential.

Nodal involvement
KS tumors are typically hypervascular due to the predominant histologic feature of disorganized vascular channels. Therefore, on contrast-enhanced CT, a characteristic finding of KS is hyperattenuating lymph nodes (Figure 30) on page 42. Nodes are of increased attenuation, greater than that of adjacent muscles, and similar to adjacent enhancing vessels. In fact, the most frequent cause of hyperattenuating nodes in AIDS is KS, accounting for up to 85% of cases. Less frequent causes of hyperattenuating nodes on CT include mycobacterial infection (9%), AIDS lymphoma (3%), and angiomunoblastic lymphoma (3%) (Figures 1 and 2).

Of patients with KS, up to 68% have hyperattenuating nodes. Isoattenuating nodes and hypoattenuating nodes are found in 29 and 3% cases, respectively. Central necrosis within nodes in KS is atypical.

Lymphadenopathy in the abdomen and pelvis occurs primarily in the retroperitoneum, mesentery, or groin.

**Extranodal involvement**

Extranodal involvement of KS in the abdomen is primarily in the form of discrete nodules or masses involving the bowel that may be seen on CT. GI KS lesions are initially flat and become nodular, bull's-eye lesions with progression. Later, polypoid masses with irregular (Figure 3) on page 15 fold thickening may be seen.

GI tract focal masses with segmental, nodular bowel wall thickening in association with bulky hyperattenuating, enhancing lymph nodes are typical of KS.

Although KS can involve solid organs, this is usually in the form of microscopic infiltration along vascular tracts, rather than of discrete lesions in the liver and spleen. Rarely, small low-attenuation hepatosplenic nodules with variable enhancement may be seen. Nonspecific hepatosplenomegaly occurs in up to 41%. If present, hepatomegaly is mild to moderate and splenomegaly is often marked.

**AIDS-related lymphoma**

**Epidemiology**
The second most common malignancy in AIDS is lymphoma, affecting 4%-10% of HIV-positive patients. Non-Hodgkin lymphoma occurs much more commonly than Hodgkin diseases in the HIV-positive population.

AIDS patients have a 60-fold increased risk for non-Hodgkin lymphoma than the general population. Although KS occurs predominantly in homosexual men, ARL occurs in all AIDS risk groups.

As with KS, there is no direct relation between the CD4 count and the incidence of ARL. ARL may occur in the HIV-positive patient in the mid or late phases of infection, and the CD4 count is usually moderately depressed. The etiology of ARL is not proven but may be relate to re-activation of Epstein-Barr virus.

ARL is very aggressive, often with widely disseminated disease at the time of initial presentation including extensive lymphadenopathy and extranodal disease. Extranodal disease occurs in up to 98% of cases of ARL.

The extent of disease and the pattern of involvement in the abdomen in ARL differ from that seen in non-Hodgkin lymphoma in the general population, as more atypical manifestations of non-Hodgkin lymphoma occur in AIDS. For example, liver lesions may be seen in up to 45% of patients with AIDS versus only 5-10% of patient without AIDS.

**Nodal involvement**

Abdominal lymphadenopathy is present in 56% of cases of ARL. Nodes are typically large and bulky *(Figure 4)* on page 16 and the vast majorities, of 88%, of enlarged lymph nodes are of soft tissue attenuation *(Figure 30)* on page 42

Central necrosis or hypoattenuation within nodes is seen in fewer than 12% of cases. Hypoattenuation nodes favor a diagnosis of mycobacterial infection rather than of ARL, as will be discussed subsequently. Massive adenopathy, with nodes larger than 3 cm in diameter, in HIV-positive patients is due to ARL in up to 90% of cases.

**Extranodal involvement**

The most common extranodal site of ARL involvement in the abdomen and pelvis is the GI tract, with an incidence of up 74%. 

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Bulky, fungating masses involving the GI tract may be detected by CT.

Bowel involvement is often multifocal. Although virtually any segment of the GI tract may be involved, the stomach (Figure 5) on page 17 and small bowel (Figures 6 and 7) are most frequently involved. CT findings may include focal or circumferential bowel wall thickening with (Figure 8) on page 20 or without excavated masses.

Extranodal ARL also may involve the liver, spleen, and/or kidneys. Less common sites of disease include the adrenal glands, pancreas, lower genitourinary system, peritoneum (Figure 9), on page 21 and omentum.

In ARL, the liver and spleen are most often normal in size with mild hepatosplenomegaly occurring in the minority of cases. In fact, moderate or marked hepatosplenomegaly favors a diagnosis of an infectious process including mycobacterial infection or histoplasmosis rather than ARL.

Focal hypoattenuating hepatic lesions occur in up to 45% of cases (Figures 10, 11, and 12). Hepatic lesions occur in 33% of cases with GI involvement and may be present without adenopathy (Figures 13, 14, and 15). Splenic lesions are found in 7% of cases and splenomegaly is identified in only 4% of cases (Figures 16, 17, and 18).

Other neoplasm

Cloacogenic carcinoma arises from the cloacogenic epithelium, with is found in the transition zone between anal and rectal mucosa. The GI literature suggests an increase in the occurrence of anorectal carcinoma in homosexual men.

Smooth-muscle tumors of the GI tract and liver in AIDS patients have been reported. However, there is no epidemiologic data that indicate an increased prevalence of smooth-muscle tumors in the AIDS population.

**OPPORTUNISTIC INFECTIONS**

In HIV-positive patients, atypical manifestations or typical infections and unusual infectious processes produce a wide variety of CT findings. Overall, abdominal lymphadenopathy in the HIV-positive population is much more commonly due to infection than to neoplasm, with infection accounting for up to 64% of cases of adenopathy.
The most common infectious cause of abdominal adenopathy in AIDS is Mycobacterium tuberculosis (MTB) infection, accounting for more than 50% of cases. Overall, infection with MTB or Mycobacterium-avium intracelullare complex (MAC) account for up to 85% of infectious causes of adenopathy in AIDS.

The patient with AIDS and acute abdominal pain (Figure 31) on page 43 represents a difficult clinical problem. Often critically ill, these patients require immediate surgical or medical care. Unusual diseases and atypical presentations or common disorders complicate the clinical evaluation.

**Mycobacterial infections**

**Epidemiology**

Whereas MTB affects immunocompetent and immunosuppressed patients, abdominal MAC disease solely affects immunosuppressed patients.

MTB infection occurs with increased frequency in patients with normal or mildly depressed CD4 counts in the range of 200-500 cells/mm3. In contrast, although MAC is one of most common opportunistic bacterial pathogens to infect AIDS patients, infection in the abdomen occurs only after the immune system is severely compromised (CD4 count falls below 100 cells/mm3).

**Nodal involvement**

Abdominal lymphadenopathy occurs in the vast majority of HIV-positive patients, with mycobacterial infection occurring in 89% of cases of MTB and nearly 100% of cases of MAC. The most useful CT feature to distinguish lymphadenopathy due to MTB and MAC is the presence or absence of central low density or necrosis. Central hypodensity within nodes is seen in up to 93% of MTB but in only 11-14% of cases of MAC (Figure 32).

In fact, in MTB almost all patients will have at least a few nodes with central low attenuation after intravenous contrast administration (Figure 19) on page 31. In contrast, most nodes in MAC infection are of homogeneous soft tissue attenuation, as the immune system is not capable of granuloma formation.
In MTB, abdominal lymphadenopathy is located primarily in the mesentery, retroperitoneum, and splenic hilum. Mesenteric nodes tend to be significantly larger than in MAC. Nodes in MAC infection are usually smaller than in MTB, and cluster of normal-size nodes are frequently present.

**Extranodal involvement**

The GI tract may be involved in AIDS patients with mycobacterial infections. In MTB, any portion of GI tract may be involved; however, the ileocecal region is the most common site of involvement *(Figure 20)*. This may be seen on CT as thickening of the ileocecal valve, the medial wall of the cecum, and the terminal ileum.

Extranodal disease also may be manifest by multiple focal lesions in the liver, spleen, kidneys, and/or pancreas. An important distinction is that disseminated MTB disease may involve the peritoneum and omentum *(Figure 21)*, with resultant peritonitis and peritoneal implants. This does not occur in MAC infection.

**Pseudomembranous colitis**

With the widespread availability and use of antibiotic, pseudomembranous colitis has become a more cause of acute abdominal pain. Pseudomembranous colitis is an acute inflammation reaction of the colon that occurs as a complication of antibiotic use and subsequent alteration of the normal colonic flora. Overgrowth by Clostridium difficile results in ulceration and sloughing of mucosal pseudomembranes.

Often, the patient has symptoms and signs more suggestive of acute infection, such as fever, chills, and an elevated count of white blood cells. As with CMV colitis, severe cases of pseudomembranous colitis can progress to toxic megacolon, ischemic bowel, and perforation.

The CT findings of pseudomembranous colitis are nonspecific, and many findings, including bowel wall thickening, are indistinguishable from those of CMV colitis. However, unlike CMV colitis, changes in pseudomembranous colitis are restricted to the colon.

Homogeneous, circumferential edema of the bowel wall, extending from cecum to anus is quite characteristics of pseudomembranous colitis *(Figure 22)* and the degree of bowel wall thickening is greater than that usually encountered in other forms of colitis.
Bacillary angiomatosis

Bacillary angiomatosis (BA) is a pseudoneoplastic condition that almost exclusively occurs in AIDS patients. It represents a hemangioproliferative response to infection with Bartonella bacilli (B. henselae and B. Quintana). The angioplastic disorder consists of cutaneous lesions with systemic involvement.

In BA, there is a marked vascular proliferation in involved lymph nodes, with resultant enhancement after intravenous contrast, similar to KS. However, unlike KS, BA usually occurs in patients with a very low CD4 count (lower than 100 cells/mm3).

In the abdomen, the liver and spleen also may affect. Peliosis hepatitis, or hepatic BA, may form in response to infection with B. henselae and can be fatal. Peliosis represents sinusoidal dilatation with multiple blood-filled cystic lesions in the liver. On CT, this may identified as multiple, small, low-attenuation lesions throughout the liver and spleen.

The differential diagnosis of multiple, small, low-attenuation hepatosplenic lesions in AIDS includes multifocal abscesses, such as due to bacterial or fungal infections, and neoplasm such as KS or ARL.

The constellation of findings or hyperattenuating adenopathy, large hypoattenuating hepatosplenic lesions, and severe immunocompromised state favors a diagnosis of BA (Figure 33) on page 45

Viral infections

Cytomegalovirus

The most common life-threatening opportunistic viral infection in AIDS is cytomegalovirus (CMV) infection. Clinical CMV is typically due to reactivation and occurs in AIDS patients when the CD4 count is less than 100 cells/mm3. Clinical disease due to CMV is identified in up to 40% of patients with advanced HIV disease.

Gastrointestinal involvement is the most common extra ocular manifestation of CMV infection in AIDS, occurring in up to 30% of patients. Within the abdomen and pelvis, the predominant site of involvement is the colon, and CMV colitis causes significant patient morbidity due to persistent diarrhea, fever, weight low, and abdominal pain.
The most common CT feature of CMV colitis is marked colonic wall thickening. Classically, disease involves the right colon and cecum and may extent into the terminal ileum, although pancolitis or isolated left colonic involvement may occur. Colonic wall thickening occurs in up to 92% of affected patients and is typically circumferential and heterogeneous with mural stratification and submucosal hypoattenuation (Figures 23 and 24).

With a mean mural diameter of 15 mm, wall thickening is significantly greater than that seen in ulcerative colitis, Crohn disease, or pseudomembranous colitis. Unlike in pseudomembranous colitis, the terminal ileum may be involved in CMV infection.

Lymphadenopathy is not a predominant feature of CMV colitis (Figure 33) on page 45. Lymphadenopathy in association with ileocecal disease should suggest MTB infection rather than CMV.

The least common radiologic manifestation of abdominal CMV infection is that of an inflammatory mass or pseudotumor. This most common arises in the stomach and may be mistaken for KS, lymphoma, or carcinoma.

CT is useful in evaluating the major complications of CMV colitis including ischemia, perforation, or toxic megacolon.

Thus, the cause of AIDS-related cholangiopathy remains uncertain. Common radiologic findings include gradual and regular stenosis of the distal common bile duct, so-called papillary stenosis, and dilatation of extra- and intrahepatic bile ducts.

**Herpes simplex virus**

Because infection and latency occur in peripheral nerves accessible to direct inoculation, the major sites of GI tract involvement are the oral cavity, esophagus, rectum and anus.

A common radiologic manifestation at all sites is the appearance of multiple, small, discrete ulcers in an otherwise normal black-ground mucosa. Esophageal ulcers in the AIDS patients tend to be slightly larger than those that occur in the immunocompetent host, whose intact immune system prevents enlargement of ulcers.

**HIV**
HIV has been implicated as a cause of ulcers of the GI tract, most commonly in the esophagus. HIV-related ulcers are not an AIDS-defining illness, but it is included here because of its clinical importance and because of the increasing recognition of this entity.

HIV-related ulcers are relatively large (>2 cm) and are solitary, although small, multiple ulcers have also been uncommonly described.

Fungal infections

Candida albicans

Candida albicans is a commensal organism in the GI tract. Mucosal candidiasis is extremely common in individuals with advanced HIV disease. In the GI tract, the esophagus is the most common site of infection (frequency in AIDS patients is 10-20% in the USA and is as high as 80% in developing countries).

The common radiologic finding of candidal esophagitis includes discrete linear or irregular filling defects that tend to be longitudinally oriented. These filling defects represent the heaped-up areas of mucosal plaques that consist of necrotic debris and colonies of Candida albicans.

Radiological findings associated with disseminated GI candidiasis small hypodenses lesions in the liver, spleen, and kidney (Figure 25). on page 38

Histoplasmosis

Histoplasma capsulatum is a common opportunistic pathogen in AIDS patients in endemic areas. Disease is typically self-limited in the normal host. However, disseminated histoplasmosis has been reported with increasing frequency in AIDS patients, and the incidence of diseases increase as the CD4 count declines to less than 100.

GI involvement occurs in up to 75% of those with disseminated disease. Although any segment of the GI tract may be involved, the colon is the most sites. Disease primarily affects the ascending colon, often with extension into the terminal ileum.

CT findings in histoplasmosis infection may include segmental circumferential colonic wall thickening, pericolic inflammatory change, and regional adenopathy (Figure 33) on page 45. Colonic histoplasmosis infection may produce apple-core lesions or
strictures than may mimic colonic neoplasm (Figures 26, 27, and 28). Retroperitoneal and mesenteric adenopathies are present in up to 80% of patients, and enlarged nodes may be of soft tissue attenuation or hypoattenuating as in the case of MTB infection.

Moderate to marked hepatosplenomegaly is seen in up to 50% of patients, and multiple low-attenuation hepatosplenic lesions may be present. Involvement of the omentum or mesentery with thickening, stranding, or fine nodularity similar to tuberculosis infection also has been reported.

Protozoal infections

Cryptosporidium

Cryptosporidiosis is the most common protozoal infection in AIDS, occurring in 10-20% of patients with a CD4 count of less than 200. Diarrhea is the predominant symptom of these infections.

Barium studies and CT examinations are usually normal; however, occasionally fluid-filled loops of bowel and/or mild proximal small bowel fold thickening may be seen. Lymphadenopathy is not a significant manifestation.

Cryptosporidium organism have been implicate as a cause of AIDS-related cholangiopathy. The bile duct strictures shown radiologically are similar to those caused by CMV

Pneumocystis

Pneumocystis carinii is a eukaryotic microbe that is believed to be either a protozoon or a fungus. The lung is the portal of entry, and pneumonia, which is the most common manifestation of P. carinii infection, occurs in about 75 of AIDS patients in the USA.

Imaging findings of extrapulmonary pneumocystosis commonly include visceral and nodal calcification, and the liver and spleen are the most common sites. US findings or multiple, tiny echogenic foci within the liver parenchyma have been reported to be a sign of an early stage of infection. Evolution of splenic involvement has been observed on CT scans, with multiple lesions of varying size and low attenuation being described as the starting point. These lesions become smaller or disappear and may appear progressively calcified (Figure 29), on page 41 in either a rimlike or punctuate fashion, on follow-up CT scans.
Fig. 1: Figure 1. Castelman disease. Axial CT image (arterial phase). Differential diagnosis with nodal involvement of Kaposi sarcoma

Fig. 2: Figure 2. Castelman disease. Axial CT image (renal elimination phase). Differential diagnosis with nodal involvement of Kaposi sarcoma
Fig. 3: Figure 3. An irregular thickening of rectal wall. Kaposi sarcoma was performed at the biopsy
Fig. 4: Figure 4. Coronal reconstructions CT show large masses and bulky masses in a patient HIV-positive. AIDS-related lymphoma
**Fig. 5:** Figure 5. A patient with known high-grade B-cell NHL. Selected CT image showing extensive wall thickening of the gastric antrum. Note the liver lesions (segments IV and V).
Fig. 6: Figure 6. Sonography illustrates a hypoechoic mural concentric thickening of the ileum wall.
Fig. 7: Figure 7. CT scan confirms the mural concentric thickening
Fig. 8: Figure 8. Axial CT scan show a thickening of ileum wall with extensive cavitation in a AIDS patient
Fig. 9: A 55 years-old man with recurrent abdominal pain. Axial enhanced CT image shows omental caking. Laparoscopy biopsy demonstrated high-grade lymphoma.
**Fig. 10:** Figure 10. 36 years old man with AIDS and upper right quadrant pain and fever. Abdominal ultrasound shows a hypoechoic mass within the right lobe of the liver.
Fig. 11: Figure 11. 36 years old man with AIDS and upper right quadrant pain and fever. Abdominal enhanced CT confirms the hypodense hepatic lesion and assess ductal intrahepatic biliary dilatation
**Fig. 12**: Figure 12. Abdominal enhanced CT six months after chemotherapy shows mass regression.
Fig. 13: Figure 13. An 35-years-old man with 1 year of history fever, weight loss and abdominal pain. Ultrasound detected a large hypoechoic mass into the right lobe liver. HIV-positive.
Fig. 14: Figure 14. An 35-years-old man with 1 year of history fever, weight loss and abdominal pain. Axial CT images detected a large hypodense mass into the right lobe liver. HIV-positive. Biopsy confirm that is primary extranodal lymphoma
Fig. 15: Figure 15. An 35-years-old man with 1 year of history fever, weight loss and abdominal pain. T2-weighted images demonstrate a hyperintensity rim
**Fig. 16:** Figure 16. 83 years old women with abdominal pain. Abdominal ultrasound shows a heterogeneous non well-defined mass within the upper portion of the spleen.

**Fig. 17:** Figure 17. 83 years old women with abdominal pain. Abdominal CT confirms the spleen mass and show that transgresses the spleen capsule, and heterogeneous mass affects the entire spleen.
**Fig. 18:** Figure 18. 83 years old women with abdominal pain. Axial MR (T1WI) proves the different components of the spleen mass.
Fig. 19: Figure 19. Extensive hypoattenuating adenopathy is present in the retroperitoneum. Note central water attenuation with peripheral enhancement of nodes.
Fig. 20: Figure 20. Contrast-enhanced CT in the right lower quadrant shows lobulated wall thickening of the cecum with extension into the terminal ileum. There is associated pericolic inflammatory change.
**Fig. 21:** Figure 21. Peritoneal involvement in a HIV-positive patient, and MTB infection
**Fig. 22:** Figure 22. CT scan shows eccentric thickening of the colon. Note the severe pericolonic stranding around the transverse colon.
Fig. 23: Figure 23. Cytomegalovirus. Coronal CT reconstructions
**Fig. 24:** Figure 24. Cytomegalovirus colitis. Sagital CT reconstructions

**Fig. 25:** Figure 25. Contrast-enhanced CT scan of an immunocompromised patient shows multiple rounded areas of decreased attenuation scattered throughout the spleen and liver
Fig. 26: Figure 26. Colon carcinoma simulated colonic histoplasmosis. Axial CT image
Fig. 27: Figure 27. Colon carcinoma simulated colonic histoplasmosis. Barium study
**Fig. 28:** Figure 28. Colon carcinoma simulated colonic histoplasmosis. Ultrasonography image
Fig. 29: Figure 29. Tiny spleen calcifications in an immunocompromised patient with Pneumocystis carinii disseminated infection
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**Fig. 30:** Figure 30
Table 2. Acute abdomen in AIDS population

<table>
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<td>✓ Antritis: Cryptosporidium</td>
<td>✓ Mycobacterium avium-intracellulare</td>
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**Intestinal perforation**

**Intestinal obstruction**

✓ Kaposi sarcoma
✓ Lymphoma

Fig. 31: Figure 31
Table 3. CT and clinical findings in mycobacterial infections

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<tr>
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MTB: Mycobacterium tuberculosis. MAC: Complejo Mycob. avium–intracellulare

Fig. 32: Figure 32
Table 4. CT and clinical findings in other opportunistic infections

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<th>Histoplasmosis</th>
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<td>No</td>
<td>Colon &gt; Small bowel</td>
<td>Colon &gt; Small bowel</td>
</tr>
<tr>
<td>Liver/Spleen</td>
<td>No</td>
<td>No</td>
<td>Moderate HSM</td>
</tr>
<tr>
<td>Lesions liver/spleen</td>
<td>Low-attenuation</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Peritoneum/omentum</td>
<td>No</td>
<td>No</td>
<td>Yes (rare)</td>
</tr>
<tr>
<td>Other</td>
<td>Cutaneous disease</td>
<td>Inflammatory mass</td>
<td>Stricture</td>
</tr>
</tbody>
</table>

BA: Bacillary angiomatosis. CMV: Cytomegalovirus

Fig. 33: Figure 33
Conclusion

Computed tomography (CT) provides the most comprehensive radiologic evaluation of the abdomen and pelvis in this setting.

It is therefore important for radiologists to be aware of the abnormalities that may be seen with CT due to opportunistic infections or AIDS-associated neoplasia in the abdomen and pelvis.

Knowledge of the patient's CD4 count largely affects the differential diagnosis.

Personal Information

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Fig. 1: Figure 1. Uxia, Iria, and Andrea. My daughters and niece
References


Carucci LR et al. (2005) Hepatic peliosis (bacillary angiomatosis) in AIDS: CT findings. Abdominal Imaging 30(6): 738-740


