IMAGING IN ABDOMINAL TUBERCULOSIS

IMÁGENES EN TUBERCULOSIS ABDOMINAL

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SUMMARY

In this article we illustrate and discuss the imaging characteristics of abdominal tuberculosis. We present a group of patients assessed with abdominal symptoms and findings suggesting granulomatous disease, using several imaging modalities. The diagnosis was confirmed on the basis of the clinical and histopathological findings. Cases include involvement of several abdominal organs such as the lymphatic system, peritoneum, liver, spleen, kidneys, ureters and pelvic organs.

RESUMEN

En este artículo se discuten e ilustran las características por imagen de la afectación abdominal por tuberculosis. Se presenta un grupo de pacientes evaluados a través de diferentes modalidades diagnósticas con síntomas abdominales y hallazgos imaginológicos sugestivos de infección granulomatosa. Este diagnóstico fue confirmado posteriormente en la evolución clínica y con estudio histológico. Se incluyen casos de afectación en diferentes órganos abdominales, como sistema linfático, peritoneo, órganos pélvicos, hígado, bazo, riñones y uréteres.

Introduction

Abdominal tuberculosis is defined as the isolated or combined involvement of the gastrointestinal tract, peritoneum, omentum, abdominal lymph nodes and solid organs, as a result of the disease (1). Because it mimics different conditions such as proliferative lymph node disease, storage and autoimmune diseases, abdominal tuberculosis is difficult to diagnose. Consequently, histopathology is required to confirm the diagnosis, although in cases of a proven focus of tuberculosis in a different organ (lungs), abdominal findings may be accepted as secondary to this disease (2).

The lung is the organ most commonly involved, considering that abdominal extension is found in only 10% of cases – although this percentage is higher in patients with immune disorders (2). Solid organs are more commonly affected than hollow viscera (1). In most patients with abdominal tuberculosis there is an existing lung focus, identified or not in imaging studies, and the tuberculin test is usually negative, making the diagnosis still more difficult.

Abdominal tuberculosis is more frequent between the third and fourth decades of life. Abdominal infection routes include spread through the bloodstream from a primary pulmonary focus, miliary tuberculosis (with propagation through the infected lymph nodes), intake of the mycobacterium (from sputum or other infected sources such as dairy products), and direct extension into adjacent organs. Peritoneal and abdominal lymph node involvement may occur without compromising other organs (3,4). It is important to know the

Key words (MesH)
Tuberculosis
Tuberculosis renal
Tuberculosis hepatic
Tomography, x-ray computed

Palabras clave (DeCS)
Tuberculosis
Tuberculosis renal
Tuberculosis hepática
Tomografía computarizada por rayos x
patient’s history of infection, epidemiological risk and immune status, because imaging findings of abdominal tuberculosis are non-specific.

**Nodal tuberculosis**

Lymphadenitis is the most frequent manifestation of intra-abdominal tuberculosis and is present in 9% to 93% of cases (5). It usually affects periportal lymph nodes, followed by peripancreatic, inter-aortocaval and mesenteric nodes (6). This is due to the fact that the drainage sites of the organs most commonly affected by tuberculosis are the ileocecal region, right colon, liver and spleen. It may be the only manifestation of tuberculosis.

The routes of dissemination to the lymph nodes include the intake of contaminated food, the bloodstream and the lymphatic system. Lymph node size and appearance vary, with sizes ranging between 1 and 2-4 cm, indicating self-limiting growth. Additionally, they are usually lobulated and clustered. Periportal lymph node involvement manifests as obstructive jaundice, thrombosis or portal hypertension. Cases of renal vascular hypertension have been described because of compression of the renal arteries by the lymph node disease.

In patients with HIV infection in AIDS stage, lymphatic spread is more frequent with involvement of more than one lymph node chain. These patients also suffer more often from generalized symptoms such as fever, sweating and weight loss.

From a histopathological point of view, there are several stages in the disease: lymphoid hyperplasia, caseification necrosis, capsular destruction, cold abscess formation, and cure with fibrosis and calcification. The imaging appearance of the lymph node disease depends on the pathological phase of the nodes:

On ultrasound, the nodes appear hypoechoic with a more hypoechoic core due to caseification. In a late phase they appear calcified.

On computed tomography (CT) they appear hypodense, mainly at the core, and contrast scans reveal their presence in the periphery, in the non-caseified area. This enhancement pattern is the most frequent and characteristic in tuberculosis, although it is not pathognomonic (Figure 1). This same pattern may be seen in metastatic disease in the ovaries and testicles, lymphoma (especially after treatment), Crohn’s disease, sarcoidosis, and Whipple and Castleman disease.

Other imaging patterns in tuberculous nodal disease include homogenous enhancement or non-enhancement (Figure 2). Magnetic resonance imaging (MRI) may also suggest tuberculous findings when lymphadenopathies appear in the form of confluent masses with central necrosis, accompanied by edema of the surrounding soft tissues. The enhancement pattern is similar to that seen on CT, with central liquefaction. The nodes may appear hypointense in T2 due to the presence of free radicals, secondary to caseification necrosis.

**Genitourinary tuberculosis**

After lymphadenopathy, the urinary tract is the second system most often compromised in extrapulmonary tuberculosis (7). All the organs in the system may be involved: kidneys, ureters, bladder, uterus, cervix, adnexa, prostate, testicles, epididymus and seminal vesicles. It is a significant, though infrequent form of tuberculosis.

It is found in 2% to 20% of all patients with pulmonary tuberculosis. Symptoms are non-specific, leading to late diagnosis and treatment. It is frequently associated with secondary renal failure and destruction or urogenital organs (8). The most important clinical manifestation is the presence of hematuria, which only occurs when the infection affects the bladder in very advanced stages of the disease (9).

Genitourinary tuberculosis occurs as a result of the blood-borne spread of the mycobacterium from the lung to the renal capillaries. Later on, the mycobacterium filters through the urine and colonizes the urothelium. Granulomas are formed inside the renal parenchyma and the mycobacterium may remain
dormant for years and then become activated as a result of any immune disorder. Granulomas may extend to the medulla, then to the papillae and finally break inside the collecting cavities. The prostate and epididymus are infected through bloodstream spread, while the testicles, cervix and seminal vesicles become involved by direct extension from the genitourinary tract (10).

The mycobacterium has been found in the urine of only 15-20% of patients with tuberculosis infection, although not all of them develop the infection. The probability of developing the infection depends on the size of the inoculum, the virulence of the mycobacterium and the patient’s immune status. People with HIV or transplants are at risk for renal tuberculosis because the probability is three times as high as in immunocompetent patients, and their manifestations are more severe, with higher parenchymal and systemic involvement (11).

**Renal tuberculosis**

Once the mycobacterium reaches the renal capillaries, it forms small granulomas that give rise to the miliary form of the disease, which is usually bilateral, and is missed in patients with adequate immunity. The latency period for this miliary infection ranges between 1 and 47 years. After the latency period, if there is any immune alteration, the mycobacterium may infect the rest of the renal parenchyma in an insidious and destructive way, which is usually asymmetrical (destruction is bilateral in up to 30% of cases).

In the acute phase of the infection, the kidney may appear normal on imaging or enlarged because of the growth and coalescence of the granulomas, which appear hypoechoic on ultrasound (US) and hypodense on CT. As the disease advances, renal function declines, and there is delay in uptake, concentration and washout of the contrast medium.

The extension of the disease to the medulla produces papillary necrosis, a characteristic but non-specific finding in renal tuberculosis. On ultrasound, it appears in the form of swollen and hypoechoic papillae due to cavitation or secondary detachment; in the urogram and on CT, it is frequent to find papillae with a moth-eaten appearance. In the late phases, residual calcifications are the main presentation (50%) in the form of stones, caseous debris or replacement of the entire renal parenchyma, giving rise to autonephrectomy.

The collecting cavities and the renal pelvis may show obstruction and tortuosity secondary to areas of stenosis, or calcifications in the cavities. At the same time, residual calcifications and obstructive uropathy lead to total loss of renal function. It is important to bear in mind that a solid mass may be a manifestation of renal tuberculosis; in this case, the differential diagnosis is a neoplastic lesion, and histology is required.

Xuefang Rui et al. assessed 258 patients with renal tuberculosis and proposed a classification of ultrasound finding into five stages, according to the pathological course (12):

Type I. Nephrectasia. Found in 15% of patients. The capsule is irregular and the renal parenchyma shows multiple, poorly-defined, hypoechoic areas. It may be associated with complex, thick-walled cysts.

**Type II. Hydronephrosis.** Accounts for 14% of cases of renal tuberculosis. There is dilatation of the renal pelvis and collecting cavities. The urothelial walls appear thickened and echogenic.

**Type III. Empyema.** It is present in 8% of cases. The kidney appears enlarged and hypoechoic. At the same time, the cavities are distended with echogenic material inside.

**Type IV. Atrophy and inflammation.** Found in 10% of cases. The kidney is reduced in size and shows irregular contours. Findings are similar to those of chronic nephropathy, but they are unilateral.

**Type V. Calcification.** It accounts for 22% of cases. The kidney is markedly reduced in size with a thin cortical area and multiple areas of dysmorphic calcification replacing the parenchyma (Figure 3).

In summary, urographic and CT-scan findings of renal tuberculosis include moth-eaten calices, parenchymal mass, amputated infundibula, autonephrectomy, thickening of urinary tract walls, parenchymal and cavity calcifications, cavities in the renal parenchyma, hydrocalcinosis, hydronephrosis and hydroureter secondary to segmental stenosis (13).

Secondary amyloidosis may occur in tuberculosis or any other chronic inflammatory disease associated with high concentrations of acute-phase reactants, such as Type A amyloid protein. This disease must be suspected in all patients with tuberculosis beginning with proteinuria, generally within the nephrotic range. The diagnosis is confirmed once amyloid deposits are documented in the renal biopsy (14). Likewise, it is important to take into consideration differential diagnoses for renal tuberculosis, including fungal or drug-related chronic pyelonephritis, renal ischemia and lymphoma.

**Ureteral tuberculosis**

Continuous renal parenchymal involvement leads to bicaluria secondary to the rupture of the renal granulomas into the collecting cavities. This produces a descending infection that
travels through the collecting system. Ureteral involvement is found in up to 50% of cases of renal tuberculosis (15).

At first there is inflammatory thickening of the urothelium followed by areas of stenosis and residual fibrosis. The areas with greatest compromise are the physiologically narrow areas of the ureter: the ureteropelvic and ureterovesical junctions and the middle third at the vascular crossing, because urine stasis increases the contact between the mycobacterium and the walls of the ureter. These stenoses are usually short and multiple (Figure 4).

Figure 4. Volume rendering of an elimination-phase urotomography in a young 19 year-old female. There is evidence of two short segmental stenoses in the left distal ureter (arrow) with proximal dilatation and secondary hydroureterosis (asterisk); additionally, bladder capacity is reduced and shows irregular contours (arrow head) due to bladder and left ureteral tuberculosis.

The ureter may appear dilated as a result of the stenotic areas or because of vesicoureteral reflux, secondary to bacilluria (16). There may also be multiple extensive calcifications in the ureteral tract, giving the appearance of a pipeline ureter.

**Bladder tuberculosis**

In general, the kidney is affected before the bladder, although there have been case reports of tuberculous cystitis without prior renal infection (17). Clinically, there is evidence of hematuria, dysuria and urgent incontinence. In principle, the contaminated urine produces an acute inflammatory process with hyperemia, ulceration, tubercle formation in the area close to the trigone, and subsequent transmural fibrosis (18).

Imaging findings include thickening, mural irregularity and calcifications, reduced bladder capacity and ulcers. On occasions, the disease may manifest in the form submucosal masses mimicking a transitional-cell carcinoma (Figure 5). Other differential diagnoses include schistosomiasis and drug or radiation-associated cystitis (19). In advanced stages of the disease, these patients may require a surgical procedure, like the creation of a neobladder, because bladder capacity may be extremely compromised.

**Genital tuberculosis**

Genital tuberculosis is rare. There are no imaging findings or specific clinical findings, and the consequences are catastrophic. The problem is compounded by a very high rate of female infertility (up to 73%). Spread to these organs is usually blood borne or results from contiguity, although sexually-transmitted infections have also been described (20).

In women, the disease involves the Fallopian tubes in most cases (94%) and usually causes bilateral salpingitis. Other manifestations include menstrual cycle alterations, amenorrhea, infertility, Bartolino gland inflammation with fistula formation, cervical erosions and endometrial thickening. These findings cannot be distinguished from a neoplasm (21). The clinical manifestations may be subtle and go unnoticed in the acute phase, resulting in infertility later on.

Hysterosalpingography shows multiple tubal stenoses and obstructions, endometrial adhesions or deformity of the cavity. Other diagnostic modalities reveal calcified nodules, adnexal calcifications and tubo-ovarian abscesses with extraperitoneal extension, a finding that suggests pelvic tuberculosis (21) (Figure 6).

In males, tuberculosis affects mainly the seminal vesicles or the prostate. Unlike what happens in women, it rarely causes infertility, unless the involvement is bilateral (22). Tuberculous prostatitis may mimic carcinoma, with a rise in prostatic antigen values in 30% of cases and non-specific imaging appearance. Consequently, a biopsy is required in order to make the diagnosis in the majority of cases. On CT and US, it usually appears in the form of hypodense or hypoechoic lesions, respectively, associated with necrotic areas and peripheral calcifications. These lesions appear hyperenhanced with the use of contrast medium. Magnetic resonance may show increased gland volume with a “watermelon” sign on T2, where the gland appears hypointense with high-signal striations, although this appearance is non-specific (23).

The epididymus is one of the male genital organs most frequently affected by tuberculosis because of its rich blood supply. It may also be affected through the lymphatic route, through direct or retrograde extension. Involvement manifests itself in the form of edema and scrotal pain. The portion usually affected is the tail, and in most cases the involvement is unilateral. On ultrasound, the epididymus appears enlarged, hypoechoic and heterogeneous. The involvement may also appear as a solid hypoechoic lesion, undistinguishable from a tumoral lesion (21). Isolated testicular infections with no epididymal involvement are rare. The testicle appears enlarged and hypoechoic, and its margin cannot be distinguished from the epididymus.

**Peritoneal tuberculosis**

Although its true incidence has not been determined (24), peritoneal tuberculosis is sixth among extrapulmonary manifestations of tuberculosis in the United States. The isolated form
is rare, and it is usually associated with extensive abdominal disease (25). Usual associations include immune deficiency, cirrhosis, alcoholism, diabetes, a history of peritoneal dialysis and intravenous drug abuse. It is believed to spread through the bloodstream, but it may occur due to gut perforation or extension from the Fallopian tubes or the lymph nodes (26).

Clinical symptoms may include abdominal pain, bloating and symptoms arising from other organs, including the lungs. Involvement is divided into three types: wet, fibrous and dry. These forms are frequently found together. Wet peritonitis is the most common form of tuberculous peritonitis and consists of free or septated ascites (Figure 7). The second type is fibrous peritonitis, characterized by large omentum and mesenteric masses with fixed intestinal loops. The dry form is less common and consists of mesenteric thickening and lymph node caseification (27) (Figure 8).

CT findings in tuberculous peritonitis include smooth thickening and enhancement of the peritoneum, thickened nodular mesentery, high-density ascites (25-45 HU) and low-density lymph nodes (28). The differential diagnosis includes peritoneal carcinomatosis, amyloidosis, peritoneal mesothelioma and lymphoma (1).

**Hepatic tuberculosis**

Liver involvement is frequent in miliary tuberculosis. It manifests as liver insufficiency and hepatomegaly. It may be found on occasions in immunocompetent patients without lung disease (29,30). In miliary tuberculosis, spread to the liver is...
through the hepatic artery. In its localized forms, the route of propagation may be the portal vein (31).

The macronodular form (tuberculoma and abscesses) is infrequent and the diagnosis may be difficult when it occurs. The most common symptoms include right upper quadrant pain, fever, anorexia and weight loss (32). Elevated transaminases may be present in two thirds of the cases. It is often accompanied by anemia and elevated globular sedimentation rate. CT findings include multiple small hypodense nodules in the miliary form (Figure 9) and gross calcification in the chronic stages. The macronodular form presents with larger single or multiple hypodense non-enhancing nodules. These nodules may coalesce to form abscesses. Biopsies of these lesions may reveal the presence of granulomas. Tissue culture provides bacteriological confirmation (33).

The gall bladder is rarely infected with tuberculosis because the normal mucosa and the wall of the gall bladder are resistant to the mycobacterium. Involvement is usually associated with severe abdominal tuberculosis affecting the peritoneum, the mesentery and the lymph nodes. On imaging, the appearance is non-specific, showing a thick-walled gall bladder or a central soft tissue mass. It must be considered as part of the differential diagnosis for gall bladder carcinoma or adenomyomatosis (34).

**Splenic tuberculosis**

The spleen is the fifth organ most commonly infected in miliary tuberculosis after the lung, the liver, the lymph nodes and the bone marrow (35). Splenic tuberculosis occurs mainly in immunosuppressed patients. It manifests in the form of low-density micronodules and splenomegaly on CT. On ultrasound there is a diffuse increase in echogenicity and multiple hypoechoic or echogenic nodules. Calcifications may be identified in the chronic forms (1,5,36).

The differential diagnosis in the miliary form includes metastasis, lymphoma, sarcoidosis and fungal infection (37). Another
rare form is the macronodular form that manifests as a single nodule (tuberculoma) or as multiple 1-3 cm nodules. On ultrasound, this lesion appears with variable echogenicity and on CT it appears hypodense with minimal peripheral enhancement (1).

The primary isolated involvement of the spleen is rare. However, there are some case reports in the literature (38,39), all of them involving immunocompromised patients, with varying presentations of micronodular or macronodular patterns, or tuberculous abscesses (Figure 10).

**Adrenal tuberculosis**

Adrenal tuberculosis occurs only in 6% of patients with active tuberculosis. In most cases it is bilateral but asymmetrical (40). In the acute and subacute stages of the disease there is evidence of diffuse and homogeneous increase in the size of the gland, or a hypodense core with peripheral enhancement, suggesting necrosis. It may also appear as a solid mass with non-specific characteristics; however, a solid mass with preserved glandular contours is more suggestive of granulomatous disease rather than neoplasia (41).

In the later stages, the size of the gland diminishes and is replaced by gross calcifications (Figure 11). The differential diagnosis includes metastasis, lymphoma, primary neoplasia and hemorrhage. Because of progressive destruction of both glands, the clinical findings suggest Addison’s disease (42).

**Pancreatic tuberculosis**

Pancreatic tuberculosis is extremely rare and is normally due to miliary propagation. It is more frequent in AIDS patients. The lesion is found in the head or the neck of the pancreas, although tail lesions have also been described. Clinical manifestations include weight loss, low-grade fever, anorexia and upper abdominal pain. However, unlike pancreatic carcinoma, pancreatic tuberculous lesions rarely produce back pain or obstructive jaundice (43).

Pancreatic tuberculosis may be focal or diffuse. In the focal form, it appears as a poorly-defined mass, whereas in the diffuse form there is an overall increase in gland size, with or without stenosis of the main pancreatic duct.

On ultrasound, the lesion appears as a focal hypoechoic mass and, on CT, it appears as a low-attenuation, peripherally-enhancing mass. An abscess may form on the site of the lesion and later on it may be replaced by residual calcifications. Pancreatic involvement due to tuberculosis may be associated frequently with peripancreatic lymphadenopathies, making diagnosis much more difficult. On MRI, the lesions appear hypointense in T1 and hypo or hyperintense in T2. The common biliary duct and the
common pancreatic duct appear normal in the focal form, and may appear stenotic in the diffuse form (44). In the majority of cases, the diagnosis is made by histopathology because of the need to rule out pancreatic carcinoma, focal chronic pancreatitis and metastasis (45).

**Intestinal tuberculosis**

Intestinal tuberculosis is not very frequent and its incidence is unknown: in close to 80% to 90% of all patients with abdominal tuberculosis there is intestinal involvement. The mycobacterium may also invade the gut mucosa through four different routes, including swallowing infected sputum, bloodstream spread from the lung, intake of contaminated food or milk, and contiguity spread (46).

There is gross evidence of three lesional patterns: small submucosal ulcers, pseudonodular hypertrophic lesions or a mix of both patterns, and an ulcerated hypertrophic or “cobblestone” pattern, as shown with barium studies (47). Ulcers are multiple, small, with poorly differentiated and raised edges, usually larger than those found in Crohn’s disease. The regions usually involved, in descending order, are the ileocecal valve, ileum, caecum, ascending colon, jejunum, the rest of the colon, rectum and duodenum, and the stomach.

**Ileocecal tuberculosis**

The most frequent location is the ileocecal region (90%). This does not happen by accident and is explained on the basis of the venolymphatic stasis and the abundance of lymphoid tissue found in this area. The mycobacterium penetrates the mucosa and remains in the submucosal lymphoid tissue where an inflammatory response is triggered, with lymphangitis, endarteritis, granuloma formation, caseous necrosis and mucosal ulceration.

The clinical manifestations are highly non-specific and include abdominal pain, anorexia, asthenia, fever, nocturnal sweating, weight loss, diarrhea, constipation, rectal bleeding, right iliac fossa mass, and ascites. The presence of ascites may be a useful clue in differentiating this condition from Crohn’s disease, where ascites is not a common finding.

Radiological signs are non-specific and the differential diagnosis with Crohn’s disease, lymphoma or cecal carcinoma is difficult. Barium studies during the early stages may show spasm and reactive hypermotility of the ileocecal valve, longitudinal submucosal ulcers of the small intestine and transverse colonic ulcers along the path of the lymphoid follicles, as well as ileocecal valve incompetence. In later stages there is thickening of the valve lips and a larger gap between the valve and the thickened terminal ileum (Fleischner’s sign).

In advanced states, due to fibrosis, there are symmetrical annular stenoses (napkin ring) and obstruction associated with tethering and shortening. The terminal ileum is narrow and stiff, the ileocecal valve expands with a shortened tapered caecum (Sterlin’s sign), representing acute inflammation overimposed on chronic inflammation of the same segment. Later on there is cecal retraction outside the iliac fossa into the hepatic flexure. Deep fissures and fistulae may form in the final stages of the disease.

On US there is evidence of concentric mural thickening of the caecum and the ileum, edema and absence of peristalsis (Figure 12). Additional, though non-specific findings include ascites, lymphadenopathies and thickening of the omentum. Abdominal CT is the best test for assessing intra- and extraluminal involvement. The most frequent finding is asymmetrical thickening of the ileocecal region and the medial wall of the colon, with or without proximal intestinal dilatation. Lymphadenopathies are multiple and appear hypodense in the center due to caseous necrosis (Figure 13).

The differential diagnosis for this ileocecal valve involvement includes amebiasis, lymphoma, Crohn’s disease, cecal carcinoma and sarcoidosis. Ileocecal tuberculosis may extend to the appendix and produce acute or chronic appendicitis (48). Endoscopic findings include ulcers, stenoses, pseudopolyps, fibrous bands, fistulae and ileocecal valve deformity (49).

**Esophageal tuberculosis**

Esophageal tuberculosis is found most frequently in AIDS patients and manifests in the form of dysphagia and painful swallowing. It occurs by swallowing of infected sputum and invasion into the mucosa, usually through a pre-existing mucosal lesion. It may also occur through the bloodstream, lymphatic drainage or direct invasion from the lymph nodes or the lungs.

Non-specific findings include esophagitis, mucosal thickening, ulcers, plaques, fistulae and stenoses, these latter two in the late stages of the disease. There is also extrinsic compression due to adjacent lymphadenopathies, mainly at the carina. In the late stages there is diverticular formation due to traction resulting from chronic fibrotic changes and stenosis. CT is used to determine mediastinal extension of the disease.

**Gastric tuberculosis**

Gastric involvement in tuberculosis is extremely rare and is due to extension from the lymph nodes or through the
bloodstream. The antrum and distal corpus of the stomach are the sites most commonly affected. Barium studies reveal two patterns: an ulcerative pattern mimicking peptic ulcer disease, and a hypertrophic pattern mimicking a malignancy. Additionally, adjacent lymphadenopathies may produce extrinsic compression. Complications include outflow tube obstruction and fistula formations. The CT scan reveals regional lymphadenopathies and submucosal masses.

**Duodenal tuberculosis**

This form of tuberculosis is found in 2% of all patients with intestinal tuberculosis. It usually involves the third and fourth portions of the duodenum. It manifests in the form of compression secondary to adjacent lymphadenopathies, and may become complicated with fistulae secondary to chronic mucosal inflammation. The CT scan reveals thickening of the duodenal folds, regional lymphadenopathies and thickening of the mesenteric root.

**Jejunal tuberculosis**

The isolated involvement of the jejunum is rare and is almost always associated with peritonitis. Imaging characteristics are non-specific, with the presence of ulcers, thickened folds and stenosis.

**Colonic tuberculosis**

Isolated colitis with no ileocecal valve involvement is rare. It occurs in 9% of intestinal tuberculosis cases. The clinical manifestations are non-specific and include diarrhea and weight loss. It usually affects one colonic segment (ascending, transverse or descending). Pancolitis is unusual and it is difficult to differentiate from ulcerative colitis (50).

Barium studies show spike formation, spasms and stiffness in the early stages. In the late stages there is stenosis and fistulae formation. Ultrasound plays a very limited role due to gas interposition, but it may show concentric mural thickening. In contrast, CT is the most useful method, revealing mural thickening, obstruction or subobstruction secondary to stenosis, lymphadenopathies and ascites.

**Tuberculosis of the abdominal aorta and its branches**

Tuberculosis of the aorta and its branches is extremely rare. Although any segment of the aorta may be involved due to bloodstream spread or direct extension from adjacent lymphadenopathies, more cases have been described in the abdominal portion (51). Likewise, the disease may manifest in the form of arteritis with areas of stenosis or aneurysmal dilations, mainly of the sacular type. Aortal dissections may also occur, although less frequently.

Vascular granulomatous infection must be suspected in patients with a history of disseminated tuberculosis and associated abdominal aneurysm. Characteristically, these aneurysms are not associated with calcifications, unlike those of atherosclerotic origin.

**Conclusion**

Although the clinical and imaging manifestations of abdominal tuberculosis may mimic other pathologies, a high degree of suspicion is required in populations at risk. The prevalence of the disease is still high in our setting. Abdominal manifestations of tuberculosis depend on the patient’s immune status and they may appear late, with the resulting sequels for the organ involved. The most acute manifestations are usually accompanied by pulmonary infection. Imaging recognition helps guide the clinician in the search and demonstration of the mycobacterium.
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Received for evaluation: September 18, 2010
Accepted for publication: October 20, 2010