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General Surgery

PRIMARY ABDOMINAL TUBERCULOSIS, 2 CASE SERIES REPORT AND LITERATURE REVIEW

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ABSTRACT Tuberculosis (TB), an infectious disease caused by Mycobacterium tuberculosis, is a disease that has affected human beings since antiquity and is still a global health problem. The main site of TB is usually in the lung, from where it can spread to other parts of the body. However, it can also present in extrapulmonary forms, the most common being the abdominal.
Abdominal tuberculosis is defined as an infection of the gastrointestinal tract, peritoneum, abdominal solid organs, and/or abdominal lymphatics. Because its clinical symptoms are nonspecific, the diagnosis of abdominal tuberculosis requires a high clinical suspicion, especially in the predisposed population.

KEYWORDS : Tuberculosis; abdome; extrapulmonary tuberculosis.

INTRODUCTION

In 2018, tuberculosis (TB) affected approximately 10 million people worldwide, most of them from developing countries. Due to the effects of globalization and immigration, and the link between the disease and human immunodeficiency virus (HIV) infection, tuberculosis is a major health problem not only in developing countries, but also in developed countries [1]. It is currently considered the most important infectious disease globally, and its spread spreads across all borders. Extrapulmonary tuberculosis occurs in about 20% of diagnoses, with abdominal tuberculosis accounting for about 5% of all cases in the world, being the most common of extrapulmonary tuberculosis [2].

Abdominal tuberculosis is a disease that represents a challenge for early diagnosis, as its nonspecific symptoms can lead to delays in diagnosis and the development of complications, being known as a mimicry for other abdominal diseases. It usually presents in four forms: tuberculous lymphadenopathy, peritoneal tuberculosis (with the possibility of subdivision into acute or chronic, with this further divided into four other forms, such as: ascitic type, fixed fibrotic type, dry plastic type and localized type), tuberculosis gastrointestinal (esophagus, stomach, duodenum, jejunum and cecal and colorectal ileum) and visceral tuberculosis involving solid organs (liver, pancreas, and spleen) [3]. In practice, the various forms can coexist [4]. Surgery is performed in about 15% of cases of abdominal tuberculosis: half of them are performed as acute surgery, including obstruction, abscess formation, perforation, or hemorrhage with the other half as a diagnostic procedure [5].

In addition, a major challenge is co-association with the human immunodeficiency virus (HIV), as HIV is present in up to 50% of TB patients in developing countries, and these are 11 times more likely to develop TB infection [9,11], being also more susceptible to cases of extrapulmonary TB [11]. However, other associations have been responsible for the increase in cases, such as emigration and population aging [5,7,8].

In 2017, the Ministry of Health declared that there were 69,569 new cases reported in Brazil, with an incidence of 33.5 per 100,000 inhabitants [6]. As a higher risk factor, latent tuberculosis infection can also be considered, which corresponds to ¼ of the global population [5].

According to the WHO, TB is the infectious disease with the highest mortality in the world, surpassing even HIV, and has global mortality rates of up to 1.2 million in HIV-negative people [6,10], even with the availability of medicines and prevention programs [8]. It should be considered that a large part of the difficulty in controlling TB is due to the increase in multidrug-resistant TB [3,5,8].

The most vulnerable population are adult men, aged over 15 years, however, it can affect people of both sexes and of all age groups[10].

The main site of involvement is the pulmonary, pulmonary TB, but it can affect any part of the gastrointestinal tract with extrapulmonary manifestations, especially in immunocompromised patients, and in this case, abdominal involvement is the most common, with 5% of all TB cases in the world. Abdominal TB can affect from lymph nodes, gastrointestinal system and peritoneum to solid organs [2,3,4,6,7,8,9,10,12], with the luminal (ileocecal area) and peritoneal forms being the most common [8], as the ileocecal area presents physiological stasis, in addition to a high concentration of lymphoid tissue [12].

Its main risk factors are underlying medical conditions such as cirrhosis, diabetes mellitus, HIV infection, kidney failure and malignancy, medical treatment with steroids and antitumor necrosis factor (TNF) agents, as well as others such as malnutrition, smoking, use of intravenous drugs and alcoholism [2].

CASE REPORT

CASE 1

A 20-year-old woman admitted to the adult emergency room at the HSPM complaining of a progressive increase in abdominal volume for 1 month, associated with diffuse abdominal pain of low intensity and

dyspnea, which worsened when changing position. She reported weight loss of 5 kg during the period and watery diarrhea of a small volume without mucus or blood. She reported intermittent episodes of unmeasured fever and denied other gynecological or urinary symptoms.

She denied a recent history of infectious and contagious diseases or travel to endemic places.

She reported a pathological history of hyperthyroidism due to Graves' disease for 5 years, under regular treatment with methimazole, 10 mg/day, having been discontinued for 1 month when she began to present symptoms. He stated smoking and occasional alcoholism.

On physical examination, she was emaciated, pale and dyspneic. Globular abdomen, massive to percussion, positive Piparotte's sign, depressible and slightly painful in hypogastrium with sudden negative decompression and no signs of peritoneal irritation or involuntary defense.

On admission, he presented mild leukocytosis of 11,000 leukocytes with 15% of rods (VR= 3800 – 11.00), C-reactive protein 12.6 mg/l (VR= 8 mg/dl), alterations in thyroid hormones T4L = 12.5; TSH = 0.003 and other initial biochemical tests within normal limits. Chest X-ray showed mild hypo transparency in the left hemithorax, with opacification of the homo lateral costophrenic sinus suggestive of pleural effusion. Abdominal ultrasound showed a large amount of free fluid in the abdominal cavity. Transvaginal pelvic ultrasound showed topical and normal-sized ovaries.

Abdominal tomography showed diffuse peritoneal thickening, nodular peritoneal and omental thickening, with characteristics of carcinomatosis and enlarged lymph nodes, in addition to a large amount of free fluid in the abdominal cavity, without showing a probable neoplastic lesion (Figure 1).

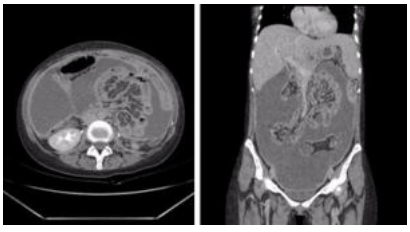

Figure 1. Computed tomography of the abdomen with IV contrast showing nodular peritoneal and omental thickenings A) Axial section, B) Sagittal section.

Initially, the hypotheses of polyserositis due to autoimmune disease and peritoneal carcinomatosis due to a neoplasm of undetermined origin were raised.

Left thoracentesis performed on the 1st day of hospitalization (DIH) with a yellow citrine fluid output whose biochemical analysis showed to be an exudate. Diagnostic paracentesis performed on the 9th IDH, in which ascitic fluid analysis revealed 470 cells/mm3, with lymph monocytic predominance (93%) and GASA = 0.3, indicating the fluid's exudative character. Oncotic cytology of both liquids was negative for neoplastic cells and ADA= 32 (VR = 40 U/L). Rheumatic markers, rheumatoid factor, ANA and Anti-DNA and tumor CEA, Alphafetoprotein, CA 19.9 negative, except for CA 125 = 390 (VR = 35U/ml). Serology for HIV, Hepatitis B and C, and syphilis are also negative. Colonoscopy showed lymphoid hyperplasia in the cecum and mild mucosal irregularity in the terminal ileum. The biopsy performed at the site found only lymphoid follicular hyperplasia.

Finally, the digestive tract surgery team indicated the performance of diagnostic video laparoscopy due to the diagnostic doubt of the neoplasm of origin of carcinomatosis and ascites. A biopsy of two fragments of the peritoneum measuring about 2 cm performed, the first in the right flank region and the second in the right iliac fossa, whose pathological examination showed peritoneal tuberculosis and absence of neoplastic cells in the appropriate sample.

Treatment for tuberculosis started with rifampicin, isoniazid, pyrazinamide and ethambutol, responding satisfactorily to treatment, with a reduction in ascitic volume, weight gain, improvement in diarrhea and improvement in general health.

CASE 2

A 24-year-old woman diagnosed with deep pelvic endometriosis followed up with a gynecologist for abdominal pain for 8 months began to experience worsening abdominal pain, which was nonspecific and progressively worsened, associated with increased abdominal volume. The patient had undergone magnetic resonance imaging of the pelvis 8 months ago, which showed only lesions in the pelvis and middle rectum suggestive of endometriosis.

She underwent laboratory tests and chest X-rays that were initially normal, with no signs of pulmonary disease. The new pelvic MRI showed the presence of nodular lesions in the pelvic and peritoneal region and large omentum suggestive of peritoneal carcinomatosis with moderate/large amounts of abdominal fluid.

Diagnostic video laparoscopy indicated for biopsy of pelvic and peritoneal lesions, with multiple biopsies of the peritoneum, pelvic and omentum, sent for anatomopathological examination and collection of ascitic fluid, where a PCR test performed to detect M. tuberculosis, which resulted in positive. The biopsy showed a pattern of non-caseating granulomas, with intense lymphocytes infiltrating, confirming the diagnosis of abdominal tuberculosis.

DISCUSSION

Formerly, several cases of abdominal TB occurred as a direct result of consuming Mycobacterium bovis in unpasteurized milk. Currently, most cases of abdominal TB are the result of reactivation of primary pathology caused by Mycobacterium tuberculosis [4,7].

There are four means by which TB is able to reach the abdomen. Firstly, tubercle bacilli can enter the gastrointestinal tract, GIT, by ingestion of milk or sputum, gastrointestinal tuberculosis. The mucosal layer of the gastrointestinal tract may be infected with the bacilli, and epithelioid tubercles develop in the lymphoid tissue of the submucosal. Caseous necrosis of the tubercles leads to ulceration of the overlying mucosa, which at a later stage spreads to the deeper layers, to the adjacent lymph nodes and to the peritoneum. The histological hallmark of TB is caseation necrosis into granulomas. Sporadically, these bacilli enter the portal circulation or the hepatic artery, entering solid organs such as the liver, pancreas, and spleen. Hematogenous dissemination from the tubercular focus to other parts of the abdomen, such as kidneys, lymph nodes and peritoneum is the second route of infection, visceral tuberculosis. The third route includes direct spread to the peritoneum from adjacent infected locus, including the uterine tubes or adnexa, peritoneal tuberculosis. And finally, abdominal tuberculosis can spread through the lymphatic channels of infected nodules, tuberculous lymphadenopathy [3,5,7].

The main pathological mechanisms of gastrointestinal tuberculosis are ingestion of contaminated material in the form of active pulmonary TB, reactivation of a quiescent intestinal focus resulting from hematogenous dissemination in childhood, or direct extension from other organs, such as contagion through contiguous infected lymph in the uterine tubes [3]. This form mainly affects the esophagus, stomach, duodenum, jejunum, ileum and colorectal. The pathological response of this form of the disease can show in three forms, ulcerative, hypertrophic or ulcer-hypertrophic structure[9]. Ingested bacilli pass through Peyer's patches of the intestinal mucosa and are carried by macrophages through the lymphatic vessels to the mesenteric lymph nodes, where they remain lethargic. Reactivation of the disease, especially in the immunocompromised including diabetes, those with Acquired Immune Deficiency Syndrome and renal failure, can lead to abdominal tuberculosis, with dissemination of the bacteria to the peritoneum or intestine [4,13].

Esophageal involvement is attributed to ingestion of sputum and may occur by dissemination of infection from the lungs, hematogenous spread by a small endobronchial lesion, spinal or mediastinal nodules infected at the level of the carina. This type of extrapulmonary TB usually manifests itself due to extrinsic compression of the lymphadenopathy, causing compression or narrowing of the esophagus [2,3]. The stomach is not such a common organ in abdominal TB, because the bactericidal property of gastric acid, scarcity of lymphoid tissue in the gastric wall and thick intact gastric mucosa hinder the spread of bacteria [4,14]. Furthermore, when there is a successful stomach infection, the routes of irradiation may be hematogenous or in adjacent lymph nodes [3]. The macroscopic pathology is characterized by transverse ulcers, fibrosis, thickening

and narrowing of the intestinal wall, enlarged and entangled mesenteric lymph nodes, omental thickening and peritoneal tubercle. Histology shows numerous granulomas that may or may not be caseous and often alcohol-acid-resistant bacilli cannot be found if there is a low mycobacterial load [2,3,4,11].

In the duodenum, the focus may be intrinsic, extrinsic or both [15,16]. The extrinsic form is secondary to lymphadenopathy in loop C, the intrinsic form presents as ulcerative, hypertrophic or hypertrophic ulcerative form[1]. The involvement of loop C of the duodenum occurs by contiguous invasion or extrinsic compression of adjacent lymph nodes. It is most observed by extrinsic compression of adjacent lymphadenopathy[9]. Ulceration can cause stenosis and fistulas [2]. Because of physiological stasis, fluid, and electrolyte absorption and abundance of lymphoid tissue (Peyer's patches), the ileocecal region is concomitantly involved in most cases of abdominal tuberculosis. Caseous necrosis remains a very important criterion for the histological diagnosis of intestinal tuberculosis. Because of physiological stasis, fluid and electrolyte absorption, decreased digestive activity and abundant lymphoid tissue (Peyer's patches) [3,4,7]. In the early stages of ileocecal TB, there is slight thickening of the ileum and cecum. In the late stages, there is eccentric thickening involving the medial cecal wall and the ileocecal valve, with regional lymphadenopathy and changes.

In the early stages of ileocecal TB, there is mild thickening of the ileum and cecum. In the late stages, there is eccentric thickening involving the medial cecal wall and the ileocecal valve, with regional lymphadenopathy and inflammatory changes [2]. The most present finding in colorectal TB is the presence of ulcers, which are linear or fissure, transverse or circumferential and are covered with white or opaque yellow exudates [17]. These ulcers usually have abnormal mucosa, with erythema, oedema, irregular border and nodularity.

The main means of dissemination of visceral tuberculosis is by the hematogenous route and the main target organs are the spleen, liver, gallbladder, genitourinary system and kidney [3,9]. Hepatosplenic involvement arises as a result of hematogenous dissemination from a primary site of infection or by local dissemination from the gastrointestinal tract [18,19]. The two main forms of hepatosplenic TB are the common micronodular-miliar type and the rarer macronodular type. In the liver, tubercle bacilli arrive through the hepatic artery in miliary TB and through the portal vein in macronodular TB [2]. In the gallbladder, its wall and mucosa are resistant to M. tuberculosis. Therefore, infection denotes severe diffuse abdominal TB, with a thickened gallbladder wall and possibility of intraluminal soft tissue mass [2,20,21]. Mycobacterium tuberculosis can spread to the genital system via the blood or lymphatic route. Granulomas develop in the fallopian tubes and subsequently in the other genital organs [2, 4]. The collecting system is the most related site in renal tuberculosis. Primarily, only a few calyxes are involved, with calyceal deformity or papillary necrosis. Eventual cure and fibrosis lead to multiple stenosis, configuring an irregular caliectasis. In cured or chronic renal tuberculosis, atrophy, progressive hydronephrosis and calcifications may occur [2].

Peritoneal tuberculosis has a hematogenous dissemination origin, and may be secondary to lymph node rupture, gastrointestinal dissemination or tubal involvement. It encompasses the peritoneal cavity, the mesentery, and the omentum. It is described by tubercles that appear as white “seedlings” on the parietal and visceral surfaces of the peritoneum. Uniquely, peritoneal tuberculosis is divided into three types: The wet ascitic type is associated with abundant amounts of free or loculated fluid in the abdomen, with a high density ascites due to increased protein content of the inflammatory exudate, with a straw coloration. Often there is peritoneal enhancement [4]. The fixed fibrotic type is characterized by involvement and thickening of the omentum and mesentery, and is described by the presence of tangled bowel on imaging. Loculated ascites may be present. And, the dry plastic type is marked by a peritoneal fibrous reaction, peritoneal nodules and presence of adhesions [3,22]. It is termed 'plastic peritonitis' when there is infiltration and thickening of the omentum and intestinal walls, with formation of associated caseous masses [23,24] The three types may occur simultaneously [9]. Also, abdominal 'cocoon' can be reported as a fourth type of peritoneal TB because of its distinct clinical presentation and imaging on examination, leading to therapeutic implications [12].

And finally, abdominal tuberculous lymphadenopathy commonly follows drainage from the contaminated organs, although it may affect any lymph node in the abdomen. In general, most lymph node infection occurs in mesenteric nodules, omental nodules, nodules at the porta hepatic, along the celiac axis, and in peripancreatic locations. The most common route of primary infection results from an initial exposure to MTB bacilli, in which they multiply in the lungs, giving rise to the Ghon complex. The secondary route of bacilli dissemination is the lymph nodes that drain the bacilli, and the infection is able to spread from the lymphatic system into the bloodstream. Another route of transmission is secondary to ingestion of material together with associated intestinal tuberculosis. This case occurs during primary tuberculosis infection or as a result of reactivation of dormant locus. Finally, the hematogenous route of transmission is characterized by contiguous spread through the abdominal organ [9].

Diagnosis

From a diagnostic point of view, abdominal TB does not present clinical signs, laboratory, radiological, bacteriological and histopathological tests specific and that are the gold standard for diagnosing it, therefore, the approach must be individually adjusted to each patient according to the symptomatology [3,4,9].

In general, the most common laboratory findings found in abdominal TB include increased ESR, normochromic anemia, and hypoalbuminemia. Ascitic fluid analysis reveals an exudate, with a protein concentration > 3 g/dL, cell counts between 150-4000µL with a predominance of lymphocytes. The ratio of ascitic fluid to blood glucose is < 0.96 and the serum ascitic albumin gradient is < 1.1 g/dL [3,4].

Associated with laboratory tests, abdominal ultrasonography, abdominal CT and diagnostic laparotomy are the tests most used in the investigation and diagnostic elucidation of abdominal TB. Radiological findings are nonspecific and may vary according to the presentation of the disease.

In tuberculous lymphadenopathy, the most common form of presentation of the disease, the lymph nodes involved are the omental, mesenteric and peripancreatic chains. In early stages, where there is an increase in lymphoid proliferation, lymph nodes are enlarged with homogeneous enhancement on CT. With the progression of the disease, the central region of the nodules evolves with caseous necrosis, being seen in the image as a hypo attenuating center and with peripheral capsular enhancements. In the final stages (cure), the nodes undergo fibrosis and calcifications can be observed [2,3,6,12].

Gastrointestinal tuberculosis is a rare manifestation of abdominal TB and can affect any organ in the gastrointestinal tract. Esophageal TB presents irregular wall thickening, mucosal ulceration, fistula formation, diverticula, and strictures on CT, while ultrasound may show asymmetric thickening with loss of esophageal wall stratification. Gastric TB presents on CT with involvement of the gastric antrum and distal body, where hypertrophic pyloric strictures, wall thickening, ulceration and fibrosis are seen. Duodenal TB is demonstrated on CT by signs of obstruction, since the duodenum is affected by the compression of the adjacent lymphadenopathy. There may also be hypertrophic involvement with thickening of the duodenal walls, ulcerations that lead to strictures and fistulas. The ileocecal region is the most common site of involvement in abdominal TB (80-90% of cases).

On CT, thickening can be seen involving the medial cecal wall and ileocecal valve, with regional lymphadenopathy and inflammatory signs. Fleischer's sign, which demonstrates an incompetent valve of a narrowed terminal ileum, and Sterling's sign, which reveals a shortened and rigid cecum, may also be present. In colorectal TB, the cecum is the most affected site and colonoscopy is the exam of choice, which reveals ulcers surrounded by erythematous, edematous mucosa and with nodularities and fissures involved by exudates. On CT, stenosis can be seen. Splenic TB is seen on USG with the presence of a hyperechoic spleen and/or liver and can be classified as micronodular or macronodular according to the size of the nodules. The micronodular shape reveals small hypo echoic nodules on USG that present minimal nodular enhancement on CT contrast injection. The macronodular form, on the other hand, shows larger nodules that can be single or multiple, hypo dense with peripheral highlights on CT and hypo echoic on US. Importantly, the micronodular form is more related to pulmonary miliary TB. Liver TB is also divided into micro and macronodular according to imaging findings, which are similar to the splenic form. The biliary tree is rare in abdominal TB, but when

present, it reveals thickening and irregularities of the bile duct on CT. It shows dilation segments alternating with biliary strictures that are similar findings to PSC and cholangiocarcinoma. Pancreatic TB is uncommon and can be seen on CT as a solitary lesion with multiple cystic components in the head of the pancreas and peripancreatic lymphadenopathies [2,3,12].

Peritoneal TB is the second most common form of presentation of abdominal TB. The wet type is the most common and presents on CT with hyperdense ascites. The fibrotic type evidences mesenteric thickening and omental enhancements and a tangle of bowel loops that can progress to an abdominal 'cocoon' or encapsulating sclerosing peritonitis, which presents with bowel obstruction. On CT, this 'cocoon' is seen as a part or all of the small intestine with or without involvement of the large intestine, surrounded by a mantle with soft tissue density. Finally, the plastic type is characterized by fibrous adhesions of intestinal loops with mesenteric thickening and caseous mesenteric lymphadenopathy [2,3,12].

Diagnosis by biopsy can be performed guided by endoscopic ultrasound or performed by open or laparoscopic surgery. The presence of abdominal TB is histologically suggested by the presence of caseification necrosis in granulomas [3].

Treatment

The mainstay of treatment for abdominal TB is anti-tuberculosis chemotherapy. The therapeutic regimen is the same used in pulmonary TB with a total duration of six months, with the first two being administered an association between rifampicin, isoniazid, pyrazinamide and ethambutol and the last four months being a regimen only of rifampicin and isoniazid [2, 3,4,7,9,10,22].

Surgical treatment is reserved for cases that evolve with complications, such as obstructions, ulcers, fistulas, perforations, hemorrhages, and abscesses or for diagnostic confirmation, since patients are at high risk of postoperative complications as they are chronic and malnourished, which makes them unfavorable candidates for the surgical approach [2,3,4,7,9,10,22].

There are three types of surgeries most commonly used to treat gastrointestinal TB. In the first type, surgery is done to address the intestinal segments involved, such as an enteroenterostomy or an ileus transverse colostomy. The second type are segmental resections, such as the ileocecal, which is indicated for the treatment of obstructions caused by ileocecal TB. The third type is stenoplasty to preserve the intestinal regions affected by stenotic lesions with obstructive signs [3,4].

Prognosis

There is not much information about the prognosis of patients with abdominal TB. However, it is known that early diagnosis and initiation of therapy are fundamental factors for a good outcome, with later diagnosis and delay in starting treatment for the disease associated with a higher mortality rate [1,2,7, 6]. Another factor that directly affects treatment success is a positive response to the action of anti-TB agents [7,6]. Furthermore, it has been reported that some patients may develop drug-induced hepatitis for abdominal TB. In this case, patients should be treated with alternative anti-TB agents such as streptomycin, levofloxacin, and ethambutol; and should be treated in an attempt to reverse the liver injury picture [6,7].

Laparoscopy should be performed to investigate patients with abdominal TB who show signs of acute complications that require surgical intervention. However, it is necessary to pay attention to the risk of iatrogenesis, as many complications can occur during this intervention, such as intestinal perforation, septic shock and respiratory failure [5,6]. As a result, mortality rates rise and patients end up with a poor prognosis, not because of the abdominal TB itself, but because of the surgical and drug management itself.

Complications

The main complications related are intestinal obstruction — total, partial, or refractory to other treatments — [1,2,4,7,9,11,12,13,14,15], intestinal perforation [1,2,3,4,9,11,12,13,15], hemorrhages/internal bleeding [2,3,4,9,12,13,15], fistula [1,11,12,13,15], intestinal malabsorption (which manifestations can be: anemia, lack of vitamin B12 and steatorrhea), bowel habit change [2], infectious process [3], abscess [9,11] and peritonitis [12].

Just as laparoscopic surgery, other diagnostic procedures can also cause complications. The peritoneal percutaneous perforation blindly made is one example of these, in which there is a chance of occurring intestinal perforation, hemorrhage, and development of an infectious process. To lower the risks it is indicated that the patient is stable and that the diagnosis of ascites is made. Another example is the ileocecal resection surgery caused by ileocecal obstruction during the anti-TB treatment. The various complications that can occur at the postoperative are anastomosis mistake, fecal fistula, intra-abdominal sepsis, persistent obstruction, dehiscence, and local infectious process [2]. Furthermore, even performing laparoscopic surgery in the attempt to resolve all the acute clinical manifestations that need surgical intervention, the patient can present persistent episodes of those complications, such as intestinal obstruction, ventral hernia and local infectious process [12].

CONCLUSION

Abdominal tuberculosis generally presents with nonspecific clinical manifestations, therefore it is a medical challenge for both diagnosis and treatment, being thus diagnosed at late stages, a priori due to suspicion of peritoneal carcinomatosis, which has clinical and tomographic peculiarities similar to those of pulmonary TB. Of all reported cases, abdominal tuberculosis is the most common type of extrapulmonary tuberculosis, and it can present in different clinical forms.

The treatment is well accepted with anti tuberculosis therapy, but in cases of complications such as obstructive, perforation and hemorrhagic conditions, surgical intervention is necessary. The evolution of the disease varies according to the start of treatment, with late diagnosis predisposing to a worse prognosis.

Abdominal tuberculosis remains a very relevant problem in all populations and needs further studies to deepen the disease.

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