Case Report

Peritoneal Tuberculosis Mimicking Peritoneal Carcinomatosis

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ABSTRACT

Tuberculosis is a serious infection that can appear in many forms and presentations. Here, we highlight a case of a 13 year old patient with a three-month history of nonspecific abdominal pain whose symptoms persisted after treating Typhoid and H. pylori infections. She had subsequent computed-tomography imaging notable for nodular thickening of the omentum and ascites concerning for possible carcinomatosis. Diagnostic laparoscopy with peritoneal biopsy revealed abdominal tuberculosis, and she had resolution of symptoms following appropriate medical therapy. We discuss the risk factors, clinical features, and work-up in the diagnosis of peritoneal tuberculosis.

INTRODUCTION

Worldwide, Tuberculosis (TB) is a global epidemic causing 1.3 million deaths among HIV negative patients in 2017. [1] It is estimated that 10 million people developed TB in 2017. Abdominal tuberculosis (TB) comprises up to 5% of all TB cases.[2]It is the sixth cause of extrapulmonary TB, after lymphatic, genitourinary, osteoarticular, miliary and meningeal. Abdominal TB includes involvement of the gastrointestinal tract, solid organs, lymph node, pancreatobiliary system and peritoneum.[3]

Abdominal TB is frequently mistaken with other abdominal disease such as inflammatory bowel disease, advanced ovarian cancer, peritoneal carcinomatosis, mycotic, yersinia and amoeba infections. Clinically, patients present with acute or chronic symptoms including abdominal pain, fever, diarrhea, weight loss, anorexia, and fatigue. Peritoneal TB often manifests as increased abdominal girth from ascites. On physical examination, patients often show abdominal tenderness, hepatomegaly and ascites. Risk factors of peritoneal TB includes cirrhosis, diabetes, malignancy, HIV, peritoneal dialysis, and administration of TNF inhibitors.

Peritoneal TB disease manifests in different types. It can be associated with ascites, enclosed or fibrotic with omental and mesenteric thickening. 90% of peritoneal TB is associated with ascites.[4] Peritoneal TB can occur following hematogenous spread from a primary pulmonary focus, active pulmonary disease, miliary TB or infected lymph nodes. Diagnosis of peritoneal TB still remains a challenging diagnosis for clinicians.

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CASE PRESENTATION

We present the case of a 13-year-old premenarchal female who presented to clinic on 7/4/18 in with a chief complaint of diffuse abdominal pain for 3 months. She also endorsed non-productive cough, nausea, vomiting, diarrhea, and subjective fevers. She had no past medical or surgical history, was not on any medications, and did not have any pertinent family history. On exam, she was afebrile and had normal vitals. She was in no acute distress. Her head and neck exam was unremarkable, and she had a normal cardiopulmonary exam. Her abdominal exam was notable for mild diffuse tenderness to palpation as well as a positive fluid wave suggestive of ascites.

Her peripheral blood film exam showed microcytic hypochromic RBCs with presence of anisocytosis, mild thrombocytosis with presence of few microclots, mild leukocytosis with absolute neutrophilia and toxic granulations, and her reticulocyte count was 0.3% (normal 0.5%-2%). Lab values of note included a WBC count of 12.8 (normal 3.5-10), Hgb of 9.8 (normal 11.0-18.0), Hct of 32.7 (normal 36.0-65.0, platelets of 660 (normal 150-500). Her ESR was 54. Her total, direct, and indirect bilirubin were within normal limits, as were her ALT, AST, and ALP. She was negative for malaria and sickle cell, positive for H. pylori, and reactive for S. typhi O and S. paratyphi BO, CO, and CH on Widal test. Imaging was notable for a normal Chest X-Ray (CXR) and an ultrasound of the abdomen and pelvis which showed an enlarged homogeneously hyperechoic liver with moderate diffuse hepatic steatosisand ascites. Her gallbladder, spleen, kidneys, bladder, uterus, and ovaries were of normal size and appearance.

Based on her initial presentation, she was diagnosed with typhoid and paratyphoid fevers, a cough, H. pylori, ascites, and hepatomegaly. She was given Vitakid, Trisoliv (tricholine citrate and andrographispeniculata) for hepatic dysfunction, Ascovent (ambroxolacefyllinate) for cough, Trigan-d (Dicyclomine, Paracetamol) for abdominal pain, Cefixime for typhoid fever, Hematinic with folic acid for anemia, and Pylo-kit (Lansoprazole + Tinidazole + Clarithromycin) for H pylori.

On 17/4/18, she returned with no resolution of her symptoms. Her physical exam was unchanged from prior. Repeat lab results were notable for a WBC count (13.4 from 12.8), a mildly improved Hgb (10.2 from 9.8), and worsening thrombocytosis (940 from 660). She was negative for BrucellaAbortus at that time.

On 24/5/18, a CT scan of the abdomen and pelvis was performed, which revealed marked nodular enhancing peritoneal and omental thickening along with mild ascites, suggestive of possible peritoneal carcinomatosis (Figure 2).

Upon presetting to clinic on 29/5/18, the patient endorsed continued abdominal pain, severe enough to prevent her from attending school. At that time, she denied vomiting and diarrhea but had persistent cough and endorsed weight loss. She had been taking Ascovent, Cefixime, hemanitic with folic acid, pylo-kit, trigan-d, trisolive, and vita-kid as prescribed. On physical exam, she then had abdominal distension with identifiable nodular masses, as well as new small palpable axillary lymph nodes. She appeared well but pale, and she had abdominal tenderness with mild guarding and mild rigidity. Her bowel sounds were present but scant. Her heart and lung exam were unremarkable. Labs showed a resolved leukocytosis, resolved anemia, and improving thrombocytosis (522 from 940). Given this presentation along with the CT findings, the differential included tuberculous abdomen, peritoneal carcinomatosis, and abdominal sarcoid. A diagnostic laparoscopy was planned for further evaluation.

She was admitted to the hospital on 31/5/18 and her diagnostic laparoscopy with peritoneal biopsy was

performed that day. On operative visualization, the patient had evidence of advanced carcinomatosis of parietal and visceral peritoneum, free fluid in the pelvis, uterus and ovaries adherent to the pelvic wall, and liver with multiple adhesions on the diaphragmatic surface. Two masses on the parietal peritoneum were excised and sent for pathology, which showed organizing peritoneal adhesions composed of expanded inflamed granulation tissue. The inflammatory cell infiltrate within the granulation tissue was characterized by loose aggregates of epithelioidhistiocytes with central liquefactive necrosis. Numerous foreign body-type and Langhans-type multinucleate giant cells were noted within the granulomata. Scattered lymphocytes, plasma cells, and histiocytes were also noted around the granulomata. There was no evidence of neoplasm. The Ziehl-Neelsen stain was positive for acid fast bacilli. The PAS and Grocott stains were negative for fungi. In conclusion, we found necrotizing granulomatous inflammation secondary to mycobacterial infection, consistent with active tuberculosis.

The patient was placed on prednisolone for one week and 4-FDC tuberculosis treatment (rifampicin, isoniazid, pyrazinamide, and ethambutol) for a plan of six months per standard tuberculosis treatment protocol.

She was seen in follow up on 20/7/18. She endorsed resolved pain, denied weight loss, cough. Her abdominal distention and tenderness to palpation had significantly improved. The patient was then seen in follow up on 24/8/18 and was doing well. She had no complaints or concerns. Her abdomen was soft, non-tender, non-distended with no hepatosplenomegaly, no masses, and no rebound or guarding. She remained adherent to treatment up to that point and will continue to the end of her six month course.

Figure 1: A posteroanterior chest x-ray of the patient at initial presentation appears normal and does not indicate any concern for pulmonary tuberculosis.

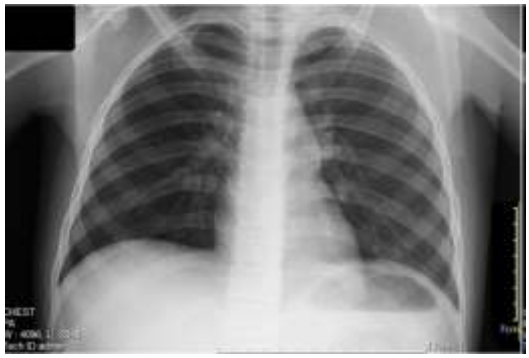


Figure 2: A representative cross-sectional image from the patient's computed tomography of her abdomen and pelvis is significant for nodular omental thickening and free abdominal fluid.

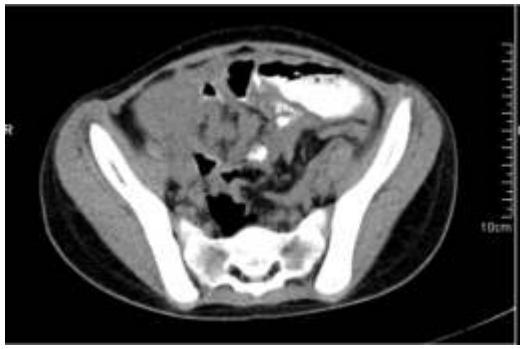


Figure 3. An intraoperative image of the patient's endoscopic procedure is notable for multifocal nodularity of the omentum. In this image, the camera is facing towards the pelvis with the bowel is at the inferior border of the image and the abdominal wall at the superior border of the image.

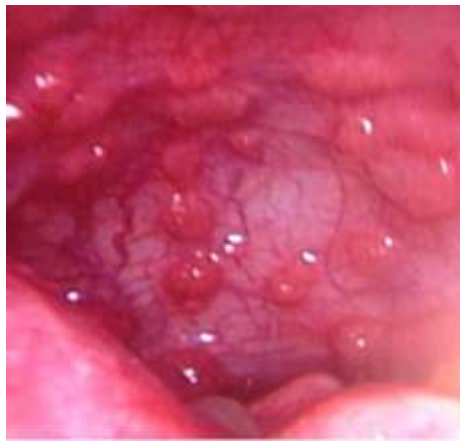
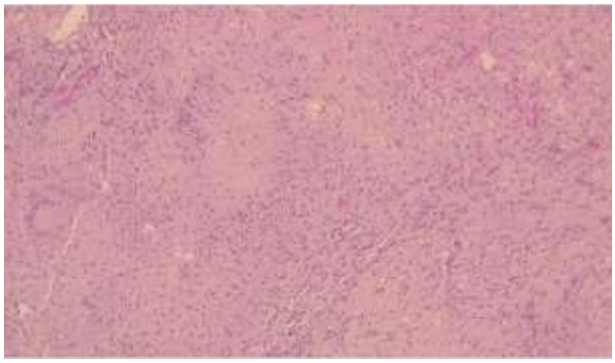
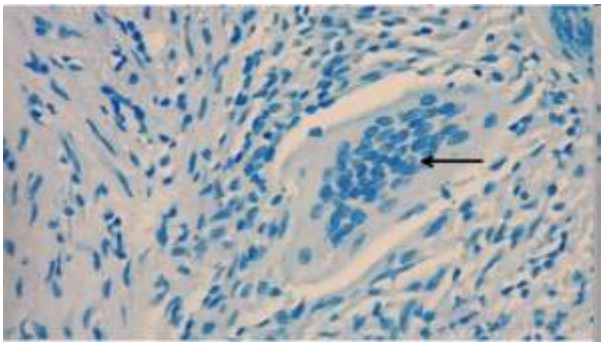


Figure 4.

A) A hematoxylin and eosin stain of a biopsied omental nodule is notable for granulomatous inflammation



B) ZiehlNeelsen stain is positive for acid fast bacilli (black arrow)



DISCUSSION

Peritoneal tuberculosis specifically accounts for 0.1% to 0.7% of all tuberculosis cases worldwide.[5] In underdeveloped countries, it accounts for 1-2% of all tuberculosis infections.[6] Peritoneal tuberculosis can result from hematogenous spread from pulmonary infection, swallowed sputum from active pulmonary infection, direct spread from other organs, or ingesting contaminated food or milk.[7] Risk factors include HIV, malnutrition, diabetes mellitus, and cirrhosis. Due to the nonspecific clinical signs and insidious nature of peritoneal tuberculosis, it can be extremely difficult to diagnose, oftentimes mimicking peritoneal carcinomatosis or other gastrointestinal diseases.[8] Delays in diagnosis and treatment due to the lack of specific presentation can lead to higher morbidity and mortality rates.[9] Undiagnosed and therefore untreated peritoneal tuberculosis can lead to severe sepsis, advanced tuberculosis symptoms, intestinal perforation, and death.[10]

According to prior studies, primary pulmonary tuberculosis on CXR imaging is only seen in 15-20% of patients with peritoneal tuberculosis.[11] Peritoneal tuberculosis also developed in 20-33% of patients who had no risk factors at all.[10] A negative PPD test does not exclude peritoneal tuberculosis and only 70% of cases present with positive PPD test.[12] The CA 125 tumor marker is found during fetal development as a soluble glycoprotein antigen and is typically elevated in patients with ovarian cancer.[13] This tumor marker elevation may also be associated with irritation and inflammation of the pleural and peritoneal linings, so this can also be seen in peritoneal tuberculosis cases.[12] Abdominal CT imaging can detect peritoneal carcinomatosis and mimics, however specific diagnosis is not possible. The gold standard for diagnosis of peritoneal tuberculosis is peritoneal biopsy and histological evidence of acid-fast bacilli. Since this can be challenging due to the lower bacterial load found in extrapulmonaryM. tuberculosis infections, there are complementary diagnostic methods to increase confidence in diagnosis.[10] Other tests that can be performed include RT-PCR (polymerase chain reaction), ELISPOT, antibody search by ELISA, Western Blot, and adenosine deaminase (ADA) in ascites.[10]

We described a case in which a 13-year-old female presented with peritoneal tuberculosis. She manifested with severe abdominal pain, absence of appetite, weakness, loss of weight, non-productive cough, and ascites. After thorough investigation trying to rule out other gastrointestinal diseases and peritoneal carcinomatosis, laparoscopic peritoneal biopsy confirmed the presence of positive acid-fast

bacilli and foreign body cell granulomas. Despite a negative CXR, her cough could be explained by possible diaphragmatic irritation. The patient was placed on 4-FDC tuberculosis treatment (rifampicin, isoniazid, pyrazinamide, and ethambutol) for six months per standard tuberculosis treatment protocol. After placement on treatment the patient began to feel better with a significant decrease in abdominal pain and other symptoms. This case illustrates that differential diagnosis of abdominal pain and ascites should include peritoneal tuberculosis despite history or risk factors.

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