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Tuberculous Peritonitis

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ABSTRACT Tuberculous peritonitis is rare in the United States but continues to be reported to occur in certain high-risk populations, which include patients with AIDS or cirrhosis, patients on continuous ambulatory peritoneal dialysis, recent immigrants from areas of high endemicity, and those who are immunosuppressed. The diagnosis of this disease requires a high clinical index of suspicion and should be considered in the differential of ascites with a lymphocyte predominance and serum-ascitic albumin gradient of <1.1 mg/dl. Microbiological or pathological confirmation remains the gold standard for diagnosis. Ascitic fluid cultures have low yield, but peritoneoscopy with biopsy or cultures frequently confirms the diagnosis. Newer techniques with future application include determination of adenosine deaminase and interferon gamma levels in ascitic fluid. Ultrasound and computed tomography are frequently used to guide fluid aspiration and biopsies. Six months of treatment with antituberculosis therapy is adequate except in cases of drug-resistant tuberculosis. The role of steroids remains controversial. Surgical approaches may be required to deal with complications including bowel perforation, intestinal obstruction from adhesions, fistula formation, or bleeding.

INTRODUCTION

Abdominal tuberculosis (TB), most common in the developing world (1–3), is not entirely uncommon in the United States and Europe. Patients with AIDS, immigrants from areas where TB is endemic, Native Americans on reservations, the urban poor, and the elderly are at particular risk (4, 5). TB rates have decreased from 52.6 cases per 100,000 population in 1953 to 4.2 per 100,000 population in 2008 to 3.0 per 100,000 in 2014 (6). While the United States experienced a temporary resurgence in the late 1980s and early 1990s, this has clearly abated and recent rates appear to be plateauing (6). The case rate among those born outside the United States is 13 times higher than for those born in the United States, for whom the rate is exceedingly low, at 1.2 cases per 100,000 (6). Interestingly, the proportion of extrapulmonary TB cases has increased (from 16% in 1993 to 20% in 2008). Peritoneal TB, the principal but not the only form of intra-abdominal TB, accounts for 6.1% of all extrapulmonary TB cases (6). Symptoms and signs of peritoneal TB are nonspecific, and a high index of suspicion needs to be maintained to make the diagnosis in a timely manner. Here, we review the epidemiology, pathogenesis, clinical features, available diagnostic techniques, and therapy of tuberculous peritonitis.

EPIDEMIOLOGY

Of all sites affected by extrapulmonary TB, the abdomen is the sixth most common after lymphatic, genitourinary, bone and joint, miliary, and meningeal involvement (5). TB was on the decline until it made a resurgence as a result of the AIDS epidemic between 1985 and 1992 (6). Despite this, the incidence of extrapulmonary TB has been stable as a proportion of all TB cases (5). One hundred thirty-two cases of peritoneal TB were reported between 1963 and 1986, which represented 3.3% of all extrapulmonary TB cases (4). Since then, the numbers have been rising in developed (7)

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and developing (3) countries. More recently, peritoneal TB was found to represent 6.1% of all extrapulmonary TB cases in the United States from data collected between 1993 and 2008 (6). Tuberculous peritonitis is predominantly a disease affecting young adults in the third and fourth decades of life but can occur at any age (8–11). Several case series of peritoneal TB in children report a low incidence in children. One retrospective study found 26 cases between 1980 and 1993 in three teaching hospitals in San Diego, CA (12). Interestingly, 80% of mycobacterial isolates from this study were identified as *Mycobacterium bovis* and the rest were *Mycobacterium tuberculosis*. In four large case series from the developing world, women were affected more frequently than men, accounting for 57% to 67% of reported cases (1–3, 13). More recently in developed countries, this trend has reversed, with men accounting for an equal number of or more cases than women (8, 9, 14).

The development of peritoneal TB has been associated with several comorbidities. Fifty percent of patients with HIV/AIDS have extrapulmonary manifestations of TB. In comparison, only 10 to 15% of non-HIV-infected patients develop extrapulmonary disease (15). Tuberculous peritonitis as the initial manifestation of HIV infection was reported first in 1992 (16). Alcoholic liver disease has been shown to be associated with peritoneal TB, though not causally, by Shakil et al. (17). In this study, 62% of patients had underlying alcoholic liver disease. Nearly three-quarters of Native Americans were believed to be heavy alcohol consumers in one series (18). Another high-risk group is patients with end-stage renal disease on continuous ambulatory peritoneal dialysis (CAPD) (19). In one series, 14 of 790 patients on CAPD were diagnosed with peritoneal TB between 1994 and 2000 (20). Other risk factors include diabetes mellitus, underlying malignancy, and the use of corticosteroids and/or other immunosuppressants (4, 15, 21, 22).

PATHOGENESIS

Peritoneal TB is thought to be the result of reactivation of latent foci of infection established in the peritoneum via hematogenous spread to the mesenteric lymph nodes from previous pulmonary infection. Ingestion of bacilli with subsequent passage through Peyer's patches in intestinal mucosa to mesenteric lymph nodes is another possible route of infection, as is contiguous spread from infected lymph nodes or ileocecal TB (4, 23, 24). Less frequently, direct spread from genitourinary sites (fallopian tubes) or hematogenous spread from active pulmonary disease or miliary TB can occur (11, 25). A significant proportion (15 to 20%) of patients with abdominal TB also have active pulmonary disease (26, 27). The causative organism is principally *Mycobacterium tuberculosis*, but *Mycobacterium bovis* has been reported to cause abdominal TB via ingestion of unpasteurized milk (12, 28).

CLINICAL FEATURES

Peritoneal TB is an insidious disease with a subacute presentation and can be challenging to diagnose if unsuspected. The average duration of symptoms prior to diagnosis extends from weeks to months (3). Several case series report the two commonest symptoms to be abdominal pain (31% to 94%) and fever (45% to 100%) (8, 9, 11, 14, 29). Other systemic symptoms—weight loss, fatigue, malaise, and anorexia—are more prominent with peritoneal TB than with other forms of abdominal TB (24). Diarrhea is unusual but may be present in one-fifth of patients (11). A minority have symptoms of coexistent pulmonary TB, including cough and hemoptysis.

Physical examination often reveals ascites (73%) and abdominal tenderness (47.7%) (11, 29). The classic "doughy" abdomen is rare (5 to 13%). Peritoneal TB has been classified as the more common "wet type," which is characterized by ascites, and the less common "plastic or fibroadhesive type," which manifests as abdominal masses comprised of adherent bowel loops (25). The absence of signs of chronic liver disease, including palmar erythema, spider angiomata, and dilated abdominal wall veins, should increase clinical suspicion for TB peritonitis.

The tuberculin skin test may be positive in about 50% of patients (14, 17, 21, 27, 30). TB blood tests such as the Quantiferon Gold, which was approved in the United States in 2005, are not affected by *M. bovis* BCG status. Like the TB skin test, TB blood tests do not differentiate between active and latent disease, and the added clinical utility of these tests in abdominal TB has been postulated but not yet established (31, 32). Mild to moderate normocytic, normochromic anemia is common, and the erythrocyte sedimentation rate is universally elevated (11). Chest roentgenograms may be abnormal for anywhere from 19% to 83% of patients. Chow et al. report chest roentgenogram findings of active or healed TB for one-third of patients (14).

Ascitic fluid typically is a straw-colored lymphocytic exudate with a serum-ascitic albumin gradient less than

1.1 and protein level of >2.5 to 3 g/dl (11, 25). Cell counts range from 500 to 1,500 per mm^3, and cells are predominantly lymphocytes (40 to 92%) (11, 14) except in patients with renal failure, in whom neutrophils predominate (20, 33). Ascitic fluid that is bloody, chylous, or purulent and with leukocyte counts as low as 10 cells has also been reported (7, 34). Table 1 summarizes the classical clinical and laboratory features of TB peritonitis.

DIAGNOSIS

The diagnosis of peritoneal TB requires a high index of clinical suspicion. Microbiological or pathological confirmation is usually required for definitive diagnosis. The gold standard diagnostic procedure remains laparoscopy and peritoneal biopsy (11, 13, 35).

Ultrasonographic appearances of peritoneal TB include ascites (either free or loculated seen in 30 to 100%) (36, 37), echogenic debris with multiple fine strands of fibrin (38), and/or peritoneal thickening (39). Computed tomography (CT) is more sensitive in the detection of bowel thickening and abdominal lymphadenopathy (40, 41). Both imaging modalities can be used to guide fine-needle aspiration of ascitic fluid or peritoneal biopsies.

TABLE 1 Clinical and laboratory features of tuberculous peritonitis[a]

Symptom, sign, or laboratory finding	Frequency (%)	Sensitivity (%)
Symptoms		
Systemic		
Fever	59	
Weight loss	61	
Abdominal		
Abdominal pain	64.5	
Diarrhea	Up to 21	
Signs		
Abdominal tenderness	47.7	
Ascites	73	
Abdominal mass	6–40	
Laboratory findings		
Positive purified protein derivative skin test		38
Abnormal chest radiograph		19–83
Ascitic fluid		
Protein > 3 g%		84–100
Lymphocyte predominance		68
ADA		Up to 100
AFB smear		3
Culture		35
Interferon gamma assay		93

[a]Data from references 1, 2, 11, 17, 21, 26, 27, 30, 34, 35, 47, 48, and 63.

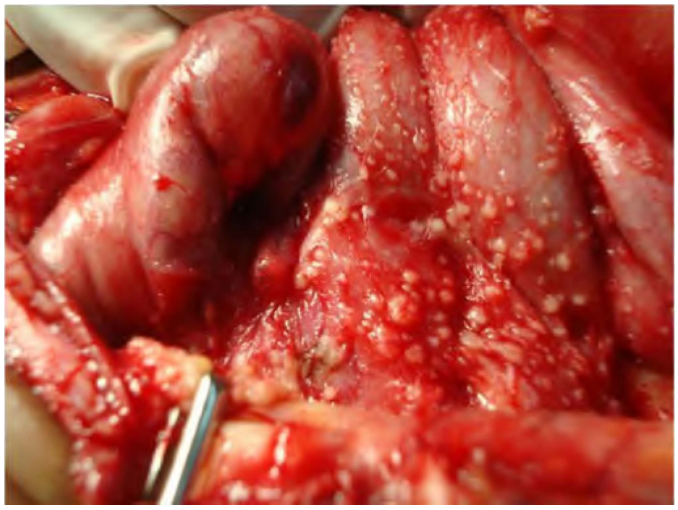

FIGURE 1 Miliary seedlings on peritoneum and serosal surface of bowel with dense adhesions. Reproduced from reference 64, per CC BY 2.0 (https://creativecommons.org/licenses/by/2.0/).

Features on CT when used in combination (mesenteric macronodules, smooth peritoneal thickening, lymph nodal masses with hypodense centers, splenic lesions, and calcifications) may help distinguish peritoneal TB from peritoneal carcinomatosis (42, 43).

Aspiration of ascitic fluid with subsequent microbiological examination with staining for acid-fast bacilli (AFB) and cultures is frequently a step in the diagnosis of peritoneal TB. AFB stains and cultures are notoriously insensitive in identifying the organism, with reported sensitivities for stains being 3% (11, 21) and that for cultures being 35% (11). The yield may increase when larger volumes of ascitic fluid are cultured. Singh et al. reported an 83% positivity rate for culture of 1 liter of peritoneal fluid (1). The diagnosis may be delayed further because *M. tuberculosis* requires 4 to 8 weeks to grow on traditional media. Fortunately, the use of the Bactec radiometric system has reduced this time to 2 weeks.

Laparoscopy allows peritoneal inspection as well as the option of pathological and microbiological confirmation of the diagnosis. Laparoscopic examination with biopsy confirms tuberculous peritonitis in 85 to 90% of cases (13, 27, 44, 45). The laparoscopic appearances have been classified into three types: thickened peritoneum with scattered whitish miliary nodules and ascites (66%), thickened peritoneum with ascites and adhesions (21%), and the fibroadhesive type where the peritoneum is markedly thickened with yellowish nodules and cheesy material with extensive adhesions (13%) (44) (Fig. 1). Sanai and Bzeizi report a 93%

sensitivity and 98% specificity of laparoscopic examination in making the diagnosis of peritoneal TB from data accumulated from 402 patients in 11 studies (11). Complications of laparoscopy include bowel perforation, bleeding, infection, and death, but these are rare, seen in <3% of cases. Complications may be more common in the fibroadhesive type (11, 46). Laparoscopic biopsy should be performed whenever possible for histological and/or microbiological confirmation. Sensitivities for peritoneal biopsy are similar to those of laparoscopic inspection (25).

More recently, noninvasive tests to detect peritoneal TB have become available. A meta-analysis by Riquelme et al. reported the sensitivity and specificity of adenosine deaminase (ADA) levels in ascitic fluid to be 100% and 97%, respectively, when cutoff values of 36 to 40 IU/liter were used (47). High levels of interferon gamma in ascitic fluid have been shown to be of similar value (48, 49).

The differential diagnosis of tuberculous peritonitis includes the differential diagnosis of ascites as well as the differential diagnosis of granulomatous peritonitis. On initial clinical presentation malignancy, for example, carcinomatosis peritonei or ovarian cancer may be the first concern. Interestingly, increased serum CA-125 levels have been reported in patients with peritoneal TB (50, 51). Also, malignant ascites is frequently a bloody exudate. Another important differential is end-stage liver disease with ascites and spontaneous bacterial peritonitis. The presentation of spontaneous bacterial peritonitis is more acute and can be diagnosed by examination of the ascitic fluid (neutrophil count of >250 or a positive Gram stain or culture).

Granulomatous peritonitis on histopathology may not always be secondary to *M. tuberculosis*. The differential includes starch peritonitis from surgical gloves, peritoneal sarcoid, and nontuberculous mycobacterial peritonitis in patients undergoing CAPD (52–58). In these cases microbiological confirmation becomes imperative.

TREATMENT

The treatment of peritoneal TB is primarily medical. Anti-TB regimens used are identical to those for pulmonary TB (59). The role of corticosteroids is controversial, and empirical data are lacking (60). Singh et al. reported no fibrotic complications in the 23 patients randomized to receive steroids, compared to 4 fibrotic complications in patients not on steroids (1). A delay in the initiation of medical therapy can result in significant morbidity and even mortality (61). More than 80% of patients deteriorated clinically while being evaluated in one series, and the overall mortality rate was reported to be 35% (14). Surgical intervention is reserved for complications arising from adhesions and inflammation, including bowel perforation, intestinal obstruction, fistulae, abscesses, and hemorrhage (25, 62).

CONCLUSIONS

Tuberculous peritonitis is rare in the United States but continues to be reported to occur in certain high-risk populations, including patients with AIDS or cirrhosis, patients on CAPD, recent immigrants from areas of high endemicity, and those who are immunosuppressed. The diagnosis of this disease requires a high clinical index of suspicion and should be considered in the differential of ascites with a lymphocyte predominance and serum-ascitic albumin gradient of <1.1 mg/dl. Microbiological or pathological confirmation remains the gold standard for diagnosis. Ascitic fluid cultures have low yields, but peritoneoscopy with biopsy or cultures frequently confirms the diagnosis. Newer techniques with future application include determination of ADA and interferon gamma levels in ascitic fluid. Ultrasound and CT are frequently used to guide fluid aspiration and biopsies. Six months of treatment with anti-TB therapy is adequate except in cases of drug-resistant TB. The role of steroids remains controversial. Surgical approaches may be required to deal with complications, including bowel perforation, intestinal obstruction from adhesions, fistula formation, or bleeding.

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