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An Old Enemy Who Is a Master of Deception

[1]Dr M.G.Jayan, [2]Dr Vijy Paul Thomas
[1]*Associate Professor of Gastroenterology M.O.S.CMedical College Kolenchery Ernakulam Kerala. 682311*
[2] *Professor, Department of Surgery,M.O.S.C Medical College,Kolenchery.*

Abstract:
Introduction: *We wish to present an interesting case of a male patient who consumes alcohol occasionally and with history of diabetes since 4 years. He came to our OPD with complaints of gradually increasing abdominal distension since two months.*
Methods: *The patient was admitted for evaluation. Clinical examination revealed moderate ascites. There was no other significant findings. All routine investigations were done. Ascitic fluid cell count was 8640 cells/cu.mm with 60% lymphocytes. Ascitic fluid Adenosine Deaminase (ADA) was 34.5U/L (cut off 30U/L). US abdomen was suggestive of gross ascites and mild coarsening of liver echotexture. MRI abdomen was suggestive of chronic liver disease with moderate ascites and omental thickening.*
Results: *Endoscopy and Colonoscopy being normal, patient was advised to undergo a diagnostic laparoscopy which showed multiple greyish coloured nodular lesions all over the peritoneum, omentum and mesentery with ascites. Peritoneal and omental biopsy was taken. This was suggestive of granulomatous inflammation with occasional Langhan's giant cells. Patient was started on Anti tubercular therapy which resulted in rapid resolution of ascites and clinical improvement of the patient.*
Conclusion: *Our old foe of yester years "tuberculosis" still remains an important cause of morbidity and should always be considered in any patient with cirrhosis presenting with ascites.*
Key words: *Chronic liver disease, ascites, tuberculosis, diagnostic laproscopy.*

I. Introduction

While chronic liver disease continues to challenge and tax both the patient and physician with regards to its complications, at times the physician would get to see certain diseases which complicate the course of this chronic disease. One such disease which was seen in a 47 year old male patient and is presented here.

In January 2015, a male patient aged 47 came to our OPD with history of gradual onset of distention of abdomen. This was not associated with any other symptoms like fever, pain abdomen or vomiting. Patient was on anti-diabetic medications since 4 years. He would consume alcohol occasionally. Prior to coming to our institution he was being treated for the same elsewhere. He was on diuretic therapy for the distension of abdomen. Patient noted that despite taking medicines he did not feel relieved of his symptoms and hence came to our centre for a second opinion.

On examination patient appeared a little dehydrated. His vital statistics were normal. General examination was almost normal except for a moderately distended abdomen. He was admitted and evaluated. There was no history of either exposure to or having suffered from tuberculosis in the past.

II. Investigations

As noted in table1, there was slight prolongation of PT/INR. The ascitic fluid analysis was suggestive of lymphocytic predominance with high protein content. The ADA level was higher than normal. Mantoux test was non–reactive with no significant induration even after 72 hours. The C-Xray was normal.

A screening ultrasound done in the department was suggestive of mildly altered liver echo texture, normal portal vein and normal sized spleen. In view of the findings of ascitic fluid, patient was advised to undergo MRI abdomen. This was suggestive of chronic liver disease, moderate ascites with peritoneal and omental thickening. There was no evidence of bowel thickening. A focal intersphicteric abscess was seen at 6 "0" clock position. In view of these findings on imaging patient was advised to undergo both upper gastrointestinal endoscopy and colonoscopy. Both these were normal and terminal ileum was normal on colonoscopy.

In view of high lymphocyte count in the ascitic fluid, and the MRI findings, patient was advised to undergo a diagnostic laparoscopy, to which he agreed. This was suggestive of gross ascites with greyish white lesions all over the peritoneum, omentum and mesentery (fig 1 and fig 2). Biopsy was taken from peritoneum and omentum. This was suggestive of granulomatous inflammation with occasional Langhan's giant cells.

Fig 1showing ascites **fig 2** peritoneal tubercles

Armed with these findings, a strong possibility of liver cirrhosis with Peritoneal Tuberculosis (PTB) was considered and after due counselling of the patient and family members regarding the risks of anti-tubercular therapy in liver cirrhosis, body weight matched dose of 4 drugs was started.

Patient was followed up on a monthly basis in our department. He continued to do well. He did not suffer any side effects of ATT. A repeat MRI was done in July to document the resolution of disease. There was complete resolution of ascites.The omental/peritoneal tissue thickening had disappeared. The liver was reported as cirrhotic without any mass or nodule.

He was advised to complete full duration of ATT. Necessary counselling regarding the underlying liver disease was given.

III. Literature Review-:

Cirrhosis, which can be the final stage of any chronic liver disease, is a diffuse process characterized by fibrosis and conversion of normal architecture to structurally abnormal nodules. These "regenerative" nodules lack normal lobular organization and are surrounded by fibrous tissue. The process involves the whole liver and is essentially irreversible. (1)

Tuberculosis (TB), which is one of the oldest diseases known to affect humans. This disease is caused by bacteria of the *Mycobacterium tuberculosis* complex and usually affects the lungs, although other organs are involved in up to one-third of cases. Transmission usually takes place through the airborne spread of droplet nuclei produced by patients with infectious pulmonary TB. (2)

M. tuberculosis is a rod-shaped, non-spore-forming, thin aerobic bacterium measuring 0.5micro m by 3micro m. Mycobacteria, including *M. tuberculosis*, are often neutral on Gram's staining. However, once stained, the bacilli cannot be decolorized by acid alcohol; this characteristic justifies their classification as acid-fast bacilli. (2)

More than 5.8 million new cases of TB (all forms, both pulmonary and extra pulmonary) were reported to the World Health Organization (WHO) in 2009; 95% of cases were reported from developing countries. (2)

In India, 40-50% of the adult population is infected, although only 5% of these may develop active disease during first year or two and another 2-5% will develop disease later on in life. The estimated incidence of tuberculosis in India is 1.96 million new cases annually and the estimated prevalence is 3.8 million cases. The disease is common in those with an immune deficient state.(3)

Primary pulmonary TB occurs soon after the initial infection with tubercle bacilli. It may be asymptomatic or present with fever and occasionally pleuritic chest pain. The lesion forming after initial infection (the Ghon focus) is usually peripheral and accompanied by transient hilar or paratracheal lymphadenopathy, which may not be visible on standard chest radiography. The lesion heals spontaneously and is seen only as a small calcified nodule. The Ghon focus, with or without overlying pleural reaction, thickening, and regional lymphadenopathy, is referred to as the *Ghon complex*.

Adult tuberculosis is known as Post -Primary tuberculosis. It is also referred to as reactivation or secondary TB.Being localized to the apical and posterior segments of the upper lobes,the extent of lung parenchymal involvement varies greatly, from small infiltrates to extensive cavitary disease.(2)

IV. Peritoneal Tuberculosis

Definition-:

Peritoneal tuberculosis(PTB) is a form of abdominal TB that involves the omentum, intestinal tract, liver, spleen, or female genital tract in addition to the parietal and visceral peritoneum. It accounts for about 1-2% of all cases of tuberculosis**. (**4)PTB follows either the direct spread of tubercle bacilli from ruptured lymph nodes and intraabdominal organs or haematogenous seeding.

An excellent review on peritoneal tuberculosis (PTB) was published in Alimentary Pharmacology and Therapeutics by F. M. Sanai and K. I. Bzeizi in 2005 (5).The first documented case of ancient 'peritoneal tuberculosis (PTB) was described in humans in 1843. (6)

A large series on abdominal tuberculosis was published by Bhansali S K in 1977 in American J Gastro. The problems related to the management of abdominal tuberculosis were discussed with reference to 300 surgically verified cases. At operation, the disease was found to involve the alimentary canal in 196 cases; in the remaining 104, only the lymph nodes and/or the peritoneum were affected. Intestinal resection was carried out in 100 cases. One hundred and seventy-nine cases showed evidence of caseation.

A very comprehensive review on Gastrointestinal Tuberculosis (GI TB) was published in Indian J Med Res 120, in October 2004, by M.P. Sharma & Vikram Bhatia from Department of Gastroenterology, All India Institute of Medical Sciences, New Delhi, India.(7). Pimparkar found evidence of abdominal tuberculosis (bowel, peritoneum and liver) in 3.72 per cent of 11,746 autopsies carriedout in K.E.M. Hospital, Mumbai between 1964 to 1970. (8)

In the article by Rajesh Upadhyay and Aesha Singh published in Medicine Update 2012 volume 22 ,the authors comprehensively covered all the aspects of tuberculosis in patients with liver cirrhosis.(3)

One of the largest contemporary series included 60 patients who were identified during a 12-year period at a center in Hong Kong. The mean age at presentation was 55 with an approximately equal gender distribution. Risk factors (in descending order of frequency) were cirrhosis, CAPD, diabetes mellitus, underlying malignancy, use of systemic corticosteroids, and AIDS. However, 20% of patients had no risk factors. (9)

What is the prevalence of PTB?

A number of studies have been conducted worldwide to evaluate the prevalence of tuberculosis in patients with liver cirrhosis. Evidence suggests a higher prevalence of tuberculosis in cirrhotics as compared to the general population. In a cohort study of patients with liver cirrhosis done in Denmark (1977 to 1993), the incidence rate of tuberculosis was found to be 168.6 per 1, 00,000 and it was highest in men over 65 years of age, with an incidence rate of 246 per 1, 00,000.(10)

Another study conducted in Western India showed the prevalence rate of tuberculosis to be 15 times higher than in the general population andwas significantly higher in alcoholics. (11)

TB of the gastrointestinal tract is the sixth most frequent form of extra-pulmonary site, after lymphatic, genitourinary, bone and joint, miliary andmeningeal tuberculosis**.(**12)Peritoneum and its reflections are common sites of tuberculous involvement of the abdomen. (13)

Both the incidence and the severity of abdominal tuberculosis are expected to increase with increasing incidence of HIV infection in India. About 0.4 million people in India are coinfected with HIV and tuberculosis. In a study from Mumbai, HIV seroprevalence was found in 16.6 per cent in patientswith abdominal tuberculosis as compared to 1.4 per cent in voluntary blood donors. (14)

In the case of luminal tuberculosis the most common site of involvement is the ileocaecal region, due to physiological stasis, and an abundance of lymphoid tissue at this site. In Bhansali'sseries, including 196 patients with gastrointestinal tuberculosis, ileum was involved in 102 and caecum in 100 patients. Of the 300 patients in a studyileocaecal involvement was present in 1628. The frequency of bowel involvement declines as one proceeds both proximally and distally from theileocaecal region. (7)

It is very important to note that abdominal lymph node and PTB may occur without gastrointestinal involvement in about one third of thecases**.(**15)

Approximately 70 percent of patients have symptoms for more than four months before the diagnosis is established.Peritoneal Tuberculosis (PTB) peritonitis should be added to the differential diagnosis of any patient presenting with several weeks of abdominal pain, fever, and weight loss. (16)

V. What are the types of PTB?

Peritoneal tuberculosis occurs in 3 forms: (*i*) Wet type with ascites; (*ii*) Encysted (loculated) type with a localized abdominal swelling; and (*iii*) Fibrotic type with abdominal masses composed of mesenteric and omental thickening, with matted bowel loops felt as lump(s) in the abdomen. A combination of these types are also common. (7)

The spectrum of disease in children is different from adults, in whom adhesive peritoneal and lymph nodal involvement is more common than gastrointestinal disease.(17)

Being a disease which can cause profound immune suppression, cirrhotics are at higher risk for PTB. This was well illustrated in a Korean study wherein, 31% cirrhotics had extrapulmonary tuberculosis as compared to 12% in the non-cirrhotic control group with predominance of peritoneal tuberculosis. (18)

Ascites due to peritoneal tuberculosis may be difficult to diagnose in the setting of liver cirrhosis where portal hypertensive ascites is common. (19).

Alcoholic liver disease is a significant risk factor of developing PTB, as was well documented in the study by Shakil et al. who found that alcohol was the underlying cause in 90% of patients with cirrhosis who developed PTB.**(**20,21, 22).The mechanism behind the increased susceptibility of ALD patients to this disease remains unknown and may be related to malnutrition.

PTB is a subacute disease and its symptoms evolve over a period of several weeks to months. Presence of comorbid conditions such as cirrhosis result in atypical presentations that may lead to delayed diagnosis.Elderly patients with TBP may manifest minimal constitutional symptoms, which again might cause a delay in the diagnosis.

Cumulative data of clinical features compiled from 35 studies of Peritoneal Tuberculous peritonitis (PTB) was published in the article by F. M. Sanai and K. I. Bzeizi in Alimentary Pharmacology Therapeutics in 2005. The data revealed that abdominal pain, weight loss and fever were the most common symptoms being 64.5%, 61% and 59% respectively. The most common finding was ascites followed by abdominal tenderness

being seen in 73% and 47.7% respectively.Abdominal symptoms such as vomiting, diarrhoea and constipation were uncommon. It is also interesting to note that PTB rarely occurs simultaneously with tuberculous enteritis.

Ascites was the predominant finding and it is present in about 73% of the patients. A smaller percentage of patients (5–13%) presented with the classical 'doughy'abdomen. This is described as the dry or plastic type of TBP and the patients have very little ascites, which can only be detected by ultrasonography or during laparoscopy.(23, 24)

The insidious nature of this disease makes the diagnosis a clinical challenge.The indolent nature of this disease was illustrated by the recent case series, which consistently reported a prolonged period of symptoms before the diagnosis was established**.(**25 26 27 28)

As we are well aware there are many causes for ascites. It is important to note that the reported incidence of PTB as a cause for ascites is only 2%. **(**29)

Diagnosis-:

Mild to moderate normochromic, normocytic anemia and thrombocytosis are frequent findings. (20,30, 31). The white cell count (WBC) is usually normal but, lympho-monocytosis is not uncommon(30,32).

Ascitic fluid analysis is routinely performed in evaluating all patients presenting with ascites. The WBC in TBP varies widely ranging from counts of <100 cells/mm3 to as high as 5000 cells/ mm3.**(**20, 25). Most patients however, have cell counts between 500 and 1500 cells/mm3. The cells are predominantly lymphocytes with the possible exception of patients with underlying renal failure where, for unknown reasons, the cells are mostly neutrophils. Ref (25, 33)

Shakil et al. showed that raised LDH above 90 U/L carries a sensitivity of 90% and a specificity of 14% for TBP.Ascitic fluid total protein levels >25 g/L is seen in almost 100% of patients with isolated TBP. However, the sensitivity of this test is significantly reduced (42–70%) when PTB complicates cirrhosis. (21, 22, 34, 35)

The serum-ascites albumin gradient (SAAG) is a simple but important test to diagnose ascites due to portal hypertension. This is calculated by taking the gradient of serum and ascitic fluid albumin. SAAG is more than 11g/L in ascites due to portal hypentension. (36). A low SAAG (<11 g/L) is seen in 100% of patients with TBP, although the specificity remains low **(**34, 37). Elevation of CA-125 has been documented in the majority of patients with TBP and created considerable confusion by mimicking advanced ovarian carcinoma. (38)

Unlike in pulmonary tuberculosis where Ziehl–Neelsen (ZN) staining of the sputum is a very important test to diagnose tuberculosis, staining of ascitic fluid for mycobacteria will be positive in only about 3% of cases with proven PTB(5)

The current gold standard for the diagnosis of TB is culturing the mycobacteria from clinical specimens. Culture methods based on a combination of liquid or biphasic media, together with solid media, are used. Usually culturing 10–50 mL of ascitic fluid is done.Centrifuging 1L of ascitic fluid may increase the concentration of bacilli thereby improving culture results.

BACTEC radiometric system is a rapid method for detecting mycobacteria in clinical specimens, with a mean time to detection of 14 days, and can be used to complement conventional methods. (39). Adenosine deaminase (ADA) is an aminohydrolase that converts adenosine to inosine and is thus involved in the catabolism of purine bases. ADA is increased in tuberculous ascitic fluid due to stimulation of T-cells by mycobacterial antigens.ADA levels were determined in the ascitic fluid of 49 patients by Dwivedi et al. and in their study the level of ADA in tuberculous ascites was significantly higher than those in cirrhotic or malignant ascites. (40)

Molecular techniquesare providing a new approach to the rapid diagnosis oftuberculosis by nucleic acid probes and PCR. The insertionsequence IS6110 has been successfully used as atarget for PCR amplification in clinical samples by manyinvestigators. (41)

Imaging-:

Ultrasound abdomen can be used to assess the liver disease and is the most important initial imaging modality. Ascites can be documented. When the protein content is high, strands may be seen in the fluid. Ultrasound also helps us to do diagnostic paracentesis.

B M Epstein and J H Mann published an article in American JRadiology on the various features of GI TB on CT.Tubercular ascitic fluid is of high attenuation value (25-45HU) due to its high protein content. Strands, fine septae and debris within the fluid are characteristic. Thickenedperitoneum and enhancing peritoneal nodules may be seen. (42)

Mesenteric disease on CT scan is seen as a patchy or diffuse increase in density, strands within the mesentery, and a stellate appearance. Lymph nodes may be interspersed. Omental thickening can be appreciated as an "omental cake" appearance. Caseating lymph nodes with hypodense centers and peripheral rim enhancement may be seen, and these may get calcified. Mesenteric, mesenteric root, celiac, porta hepatis and

peripancreatic nodes are characteristically involved. The retroperitoneal nodes are relatively spared, and are almost never seen in isolation, unlike lymphoma.(43)

The presence of a smooth peritoneum with minimal thickening and pronounced enhancement on CT suggests TBP, whereas nodular implants and irregular peritoneal thickening suggests carcinomatosis. (5)

Endoscopy-:

Colonoscopy is used to diagnose ileal and cecal disease. Mucosal nodules of variable sizes (2 to 6 mm) and ulcers in a discrete segment of colon, 4 to 8 cm in length are the classical findings. The nodules have a pink surface with no friability and are most often found in the caecum especially near the ileocaecal valve. Large (10 to 20 mm) or small (3 to 5 mm) ulcers are commonly located between the nodules. The intervening mucosa may be hyperemic or normal. (44) It is preferable to take multiple biopsies at least 8 to 10 in number. Biopsies should be taken from the edge of the ulcers. Granulomas have been reported in 8-48 per cent of patients and caseation in a third (33-38%) of positive cases. (45)

A combination of histology and culture of the biopsy material can beexpected to establish the diagnosis in over 60 per cent of cases. (7)

Laparoscopy-:

Laparoscopy is the diagnostic tool of choice in patientswith suspected TBP as it allows for inspection and biopsy. Bhargava et al studied 87 patients with high proteinascites, of which 38 were diagnosed as having tuberculosis. (46)

The laparoscopic findings in peritoneal tuberculosisare grouped into three categories being-:

(1) Thickened peritoneum with tubercles. Other abdominal organs may be studded with tubercles(66% cases)

(2) Thickened peritoneum without tubercles.(21% cases)

(3) Fibro-adhesive peritonitis with markedly thickenedperitoneum and multiple thick adhesions fixing theviscera.(13% cases)

The diagnostic yield of laparoscopic examination is very high with a sensitivity macroscopic appearances approaching 93%. Other diseases like peritoneal carcinomatosis, sarcoidosis, starch peritonitis and Crohn's disease may occasionally mimic thelaparoscopic features of TBP.

Complications of laparoscopy are rare, and mortality is up to 0.04 %.(47, 48,and 49)

How is the treatment of tuberculosis different from that in patients without liver disease?

Making the diagnosis of TBP in the setting of liver cirrhosis is challenging since both diseases can present with ascites.Tubercular ascites should be suspected when a patient with compensated disease develops ascites without any other aggravating factor that could have decompensated the liver or where increasing or resistant ascites occurs despite diuretic treatment in cirrhotic ascites.(3). TBP is treated with the same 4 drugs used to treat pulmonary tuberculosis. All patients should receive conventional anti-tubercular therapy for at least 6 months including initial 2 months of rifampicin(R), isoniazid (INH), pyrazinamide (PZA) and ethambutol (E).

What should be the approach to a patient with liver cirrhosis?

Patients in Childs A Cirrhosis may be treated with standard 4 drug regimefor 2 months followed by 2 drugs for remaining 4 months (total 6 month treatment). PZA is potentially the most hepatotoxic drug and it may be completely avoided and a 3 drug regimen can be given for 9 months.

Patients in Child class B & C treat according to the 2010 WHO guidelines, depending on the severity of the disease and degree of decompensation.(50)

One or two hepatotoxic drugs may be used in moderately severe disease and hepatotoxic drugs should be completely avoided in decompensated Child C cirrhosis.

9 months of INH/ R/ and E or 2 months of INH, R, E and Streptomycin followed by 6 months of INH and Rwould be ideal.

Alternately 2 months of INH, E and Streptomycinfollowed by 10 months of INH and E can be done. All hepatotoxic drugs van be avoided and treatment with streptomycin and ethambutol and quinolones for 18 months can be done.

Regular LFT monitoring should be done in all cirrhotic patients receiving anti-tubercular treatment and drug therapy may be stopped /altered as per the LFT reports. Hepatotoxicity due to anti-tubercular treatment is more commonly observed in patients with hepatic cirrhosis and patients do not fare well once hepatotoxicity occurs unless prompt action is taken.

In patients with a normal liver, one would consider modification in treatment if the liver enzymes are elevated above upper limit of normal by more than 3 times. There are no clear cut guidelines for patients with cirrhosis, but extreme caution is warranted.Any rise in serum bilirubin should be treated with great caution and

hepatotoxic drug treatment stopped immediately. Treatment should be stopped and re-started after serum bilirubin and transaminase return to near normal. Drugs are re-started in a sequential fashion starting with rifampin first.

VI. Conclusion-:

The index case was presented to highlight the importance of suspecting unusual causes for ascites in a patient with liver cirrhosis. All routine investigations being done, one should proceed to an ultrasound of the abdomen. Ascitic fluid should be analysed. A CT of the abdomen may be done to rule out HCC. Both upper and lower endoscopy must be done for varices and to rule out luminal tuberculosis. If the diagnosis is still in doubt, one may proceed to do a diagnostic laproscopy with biopsy to confirm the diagnosis.

Once diagnosed, ATT can be given based on body weight. The liver function must be monitored periodically. Any variation from baseline should be regarded with caution and patient should be well counselled regarding the potential side effect of drugs. Periodic visits coupled with emergency visits as needed may be scheduled.

Monitored carefully, these patients have a very good response to ATT and is very gratifying to the physician to see the improvement in these patients.

Bibliography

[1]. Lee Goldman, Andrew I Schafer, Guadalupe Garcia-Tsao: Cirrhosis and its sequelae;Cecil text book of medicine;chapter 156:999-1006 25th edition, Saunders An Imprint of Elsevier.

[2]. Longo, Fauci ,Kasper, et al., editors. Harrison's principles of internal medicine 18th ed. New York McGraw Hill 2012.

[3]. Tuberculosis In Liver Cirrhosis:Rajesh Upadhyay, Aesha Singh, Delhi Medicine Update 2012 volume 22 pages 476-478

[4]. Peritoneal tuberculosis K. Mimidis, K. Ritis, G. Kartalis Annals Of Gastroenterology, 2005, 18(3):325-329

[5]. F. M. Sanai & K. I. Bzeizi Systematic review: tuberculous peritonitis – presenting features, diagnostic strategies and treatment Division of Hepatology, Department of Internal Medicine, Riyadh, Saudi Arabia Aliment Pharmacol Ther 2005; 22: 6

[6]. Dineen P, Homan WP, Grafe WR. Tuberculous peritonitis:43 years' experience in diagnosis and treatment. Ann Surg 1976; 184: 717–22.

[7]. M.P. Sharma & Vikram Bhatia Department of Gastroenterology, All India Institute of Medical Sciences, New Delhi, IndiaIndian J Med Res 120, October 2004, pp 305-315

[8]. Pimparkar BD. Abdominal tuberculosis. J Assoc Physicians India 1977; 25 : 801-11

[9]. Chow KM, Chow VC, Hung LC, et al. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic fluid samples. Clin Infect Dis 2002; 35:409.

[10]. Thulstrup A.M,Molle I, Svendsen N and Sorensen H.T. Incidence and prognosis of tuberculosis in patients with cirrhosis of liver, A Danish population based study. Epidemiology and infection 2000;124:221-225.

[11]. Baijal R, Praveenkumar H.R, Amrapurkar, et al. Prevalence of tuberculosis in patients with cirrhosis of liver in western India. Trop doct 2010;40:3163-164.

[12]. Paustian FF. Tuberculosis of the intestine. In: Bockus HL,editor. Gastroenterology, .11, vol 11 2nd ed. Philadelphia :W.B. Saunders Co.; 1964 p. 311.

[13]. Dineen P, Homan WP, Grafe WR. Tuberculous peritonitis:43 years' experience in diagnosis and treatment. Ann Surg 1976; 184: 717–22.

[14]. Rathi PM, Amarapurakar DN, Parikh SS, Joshi J, Koppikar GV, Amarapurkar AD, et al. Impact of human immunodeficiency virus infection on abdominal tuberculosis in western India. J Clin Gastroenterol 1997;24 : 43-8.

[15]. Hoon JR, Dockerty MB, Pemberton J. Ileocaecal tuberculosis including a comparison of this disease with non-specific regional enterocolitis and noncaseous tuberculated enterocolitis. Int Abstr Surg 1950; 91 : 417-40.

[16]. Tuberculous peritonitis AuthorsValerie Byrnes, MRCPI, MD, MMScSanjiv Chopra, MD, MACPSection EditorBruce A Runyon, MDDeputy EditorAnne C Travis, MD, MSc, FACG, AGAF in Uptodate 2015

[17]. Sharma AK, Agarwal LD, Sharma CS, Sarin YK.Abdominal tuberculosis in children : experience over a decade. Indian Peadiatr 1993; 30 : 1149-53.

[18]. Cho YZ, Lee SM, Yoo CG, et al. Clinical characteristics of tuberculosis in patients with liver cirrhosis. Respirology 2007;12:401-5.

[19]. 20th European Congress of clinical microbiology tuberculosis complicated with chronic liver and infectious disease, Vienna, Austria, 2010;10-13

[20]. Shakil AO, Korula J, Kanel GC, et al. Diagnostic features of tuberculous peritonitis in the absence and presence of chronic liver disease: a case control study. Am J Med 1996; 100: 179–85

[21]. Aguado JM, Pons F, Casafont F, et al. Tuberculous peritonitis: a study comparing cirrhotic and noncirrhotic patients. J Clin Gastroenterol 1990; 12: 550–4.

[22]. Burack WR, Hollister RM. Tuberculous peritonitis: a study of 47 proved cases encountered by a general medical unit in twenty-five years. Am J Med 1960; 28: 510–23.

[23]. Bhargava DK, Shriniwas, Chopra P, et al. Peritoneal tuberculosis:laparoscopic patterns a and its diagnostic accuracy. Am J Gastroenterol 1992; 87: 109–12.

[24]. Nafeh MA, Medhat A, Abdul-Hameed AG, et al. Tuberculous peritonitis in Egypt: value of laparoscopy in diagnosis. Am J Trop Med Hyg 1992; 47: 470–7.

[25]. Lui SL, Tang S, Li FK, et al. Tuberculosis infection in Chinese patients undergoing continuous ambulatory peritoneal dialysis. Am J Kidney Dis 2001; 38: 1055–60.

[26]. Wang HK, Hsueh PR, Hung CC, et al. Tuberculous peritonitis: analysis of 35 cases. J Microbiol Immunol Infect 1998; 31: 113–8.

[27]. Thoreau N, Fain O, Babinet P, et al. Peritoneal tuberculosis:27 cases in the suburbs of northeastern Paris. Int J Tuberc Lung Dis 2002; 6: 253–8.

[28]. Chow KM, Chow VCY, Hung LCT, et al. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial culture of ascitic fluid samples. Clin Infect Dis 2002; 35: 409–13.

[29]. Runyon BA, Reynolds TB. Approach to the patient with ascites. In: Yamada, T, Alpers, D, Owyang, C, Powell, D, Silverstein, F, eds. Textbook of Gastroenterology. New York, USA: J.B. Lippincott, 1991: 846–64.
[30]. Manohar A, Simjee AE, Haffejee AA, Pettengell KE. Symptoms and investigative findings in 145 patients with tuberculous peritonitis diagnosed by peritoneoscopy and biopsy over a five year period. Gut 1990; 31: 1130–2.
[31]. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. Am J Gastroenterol 1993; 88: 989–99.
[32]. Aguado JM, Pons F, Casafont F, et al. Tuberculous peritonitis:a study comparing cirrhotic and noncirrhotic patients. J Clin Gastroenterol 1990; 12: 550–4
[33]. Lui SL, Lo CY, Choy BY, et al. Optimal treatment and long term outcome of tuberculous peritonitis complicating continuous ambulatory peritoneal dialysis. Am J Kidney Dis 1996; 28: 747–51.
[34]. Demir K, Okten A, Kaymakoglu S, et al. Tuberculous peritonitis-reports of 26 cases, detailing diagnostic and therapeutic problems. Eur J Gastroenterol Hepatol 2001; 13: 581–5.
[35]. Vardareli E, Kebapci M, Saricam T, et al. Tuberculous peritonitis of the wet ascitic type: clinical features and diagnostic value of image-guided peritoneal biopsy. Dig Liver Dis 2004; 36: 199–204.
[36]. Boyer TD. Diagnosis and management of cirrhotic ascites. In: Zakim, D, Boyer, TD, eds. Hepatology: A Textbook of Liver Disease. 4th edn. Philadelphia, USA: W.B. Saunders, 2003:631–58.
[37]. Han SH, Reynolds TB, Fong TL. Nephrogenic ascites. Analysis of 16 cases and review of the literature. Medicine (Baltimore) 1998; 77: 233–45.
[38]. Bilgin T, Karabay A, Dolari E, Peritoneal tuberculosis with pelvic abdominal mass, ascites and elevated CA 125 mimicking advanced ovarian carcinoma: a series of 10 cases. Int J Gynecol Cancer 2001; 11: 290–4
[39]. Chow KM, Chow VCY, Hung LCT, et al. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial culture of ascetic fluid samples. Clin Infect Dis 2002; 35: 409–13.
[40]. Dwivendi M, Misra SP, Misra V, et al. Value of adenosine deaminase estimation in the diagnosis of tuberculous ascites. Am J Gastroenterol 1990; 85: 1123-1125.
[41]. K. Mimidis, K. Ritis, G. Kartalis: Peritoneal tuberculosis Annals of Gastroenterology 2005, 18(3):325-329
[42]. Gulati MS, Sarma D, Paul SB. CT appearances in abdominal tuberculosis. A pictorial assay. Clin Imaging 1999;23: 51-59
[43]. Ha HK, Jung JI, Lee MS, et al. CT differentiation of tuberculosis peritonitis and peritoneal carcinomatosis. Am J Roentgenol 1996; 167: 743-748.
[44]. Bhargava DK, Tandon HD, Chawla TC, Shriniwas, Tandon BN, Kapur BM. Diagnosis of ileocecal and colonic tuberculosis by colonoscopy. Gastrointest Endosc 1985; 31 : 68-70
[45]. Singh V, Kumar P, Kamal J, Prakash V, Vaiphei K, Singh K. Clinico colonoscopic profile of colonic tuberculosis. Am J Gastroenterol 1996; 91 : 565-8.
[46]. Bhargawa DK, Shriniwas S, Chopra P, et al. Peritoneal tuberculosis: Laparoscopic patterns and its diagnostic accuracy. Am J Gastroenterol 1992; 87: 109-112.
[47]. Barry RE, Brown P, Read AE. Physicians use of laparoscopy.BMJ 1978; 2: 1276–8.
[48]. Lewis A, Archer RJ. Laparoscopy in general surgery. Br J Surg 1981; 68: 778–80.
[49]. Loffer FD, Pent D. Indications, contraindications and complications of laparoscopy. Obstet Gynecol Surv 1975;30: 407–23
[50]. World Health Organisation. Treatment of tuberculosis; guidelines for national programme. 2010;97-98.

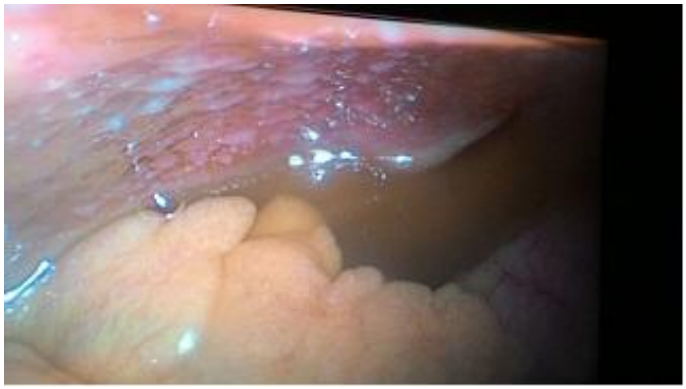

Fig 1 Showing Ascites

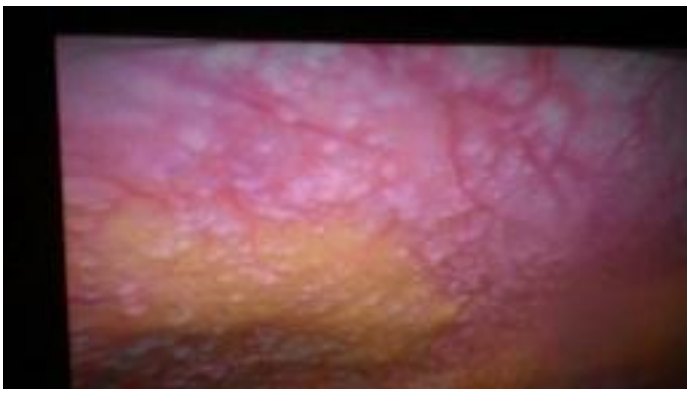

Fig 2 Showing Peritoneal Tubercles

Table 1

Haemoglobin	12.6mg/dl		**Ascitic Fluid**	values
Total WBC Count	11,000/CU.MM		Total cells	8320
neutrophils	70%		Polys	4%
lymphocytes	13%		lympho	96%
monocytes	9%		sugar	146 mg%
ESR	10		protein	7.3 g%
Platelet Count	3,76,000		ADA	34.5 U/L Normal up to 30U/L
PT/INR	16.4 test 11.3 control 1.46		**HBsAg}** **HIV }** **HCV }**	Negative
TSH	1.80		**Alpha Feto Protein**	Normal
Urea **Creatinine** **Amylase** **Lipase**	All within normal limits		**Sodium serum**	132 mmol/L
Uric acid	9.8mg%		**Potassium serum**	4.8 mmol/L
Serum Bilirubin Total	0.6mg%		**SGPT**	23 U/L
Direct Bilirubin	0.4mg%		**Albumin**	4.1 mg/dl
SGOT	26 U/L		**Globulin**	2.8 mg/dl
Alkaline Phosphatase	57 U/L		**Mantoux test**	Not Reactive