Abdominal Tuberculosis: Diagnostic and Management Problems

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ABSTRACT

Abdominal tuberculosis, as one of the manifestations of extrapulmonary tuberculosis, may involve gastrointestinal tract, peritoneum, mesenteric lymph nodes, liver, and also spleen. Most patients have constitutional symptoms of fever, pain, diarrhea, constipation, alternating constipation and diarrhea, weight loss, anorexia and malaise. It also has an insidious course without any specific laboratory, radiological or clinical findings and makes it difficult to diagnosis. Anti tuberculosis treatments with initiation phase for 2 months and continue with continuation phase for 7 months is effective. Steroids may be used to reduce acute inflammation and limit delayed fibrotic complications.

We report a male patient with abdominal tuberculosis involving peritoneum, liver, colon, paraaorta lymph nodes, and spleen, which at first suggested as a malignancy. Drug induced hepatitis due to anti tuberculosis drug during treatment was emerged and substituted with other regimen. After given anti tuberculosis treatment and steroid as adjunctive treatment, the clinical condition of patient was improved.

Keywords: *abdominal tuberculosis, anti tuberculosis treatment, drug induced hepatitis*

INTRODUCTION

Tuberculosis is currently the leading infectious cause of morbidity and mortality worldwide. The World Health Organization (WHO) estimates that approximately 9 million individuals who developed activetuberculosis disease and more than 2 million deaths due to TB in year 2005. More than 3.8 million new cases of tuberculosis - all forms (pulmonary and extrapulmonary), 90% of them from developing countries - were reported to the WHO in 2001. Due to delayed, inadequate, or unavailable therapy, 2 to 3 million persons die annually, indeed, WHO estimates that 26% of preventable deaths in the developing nations are attributable to tuberculosis.[1,2,3]

Tuberculosis occurs all over the world, but its epidemiology varies in different regions. In Indonesia, based on Survey of housekeeping healthy, Ministry of Heallthy, Republic of Indonesia 1995, tuberculosis is 3rd leading cause of death and 3rd most frequent cause of infectious disease. Death due to tuberculosis infection is estimated about 175,000 people annually.[4]

Tuberculosis infection can affect all organs of the body. Extrapulmonary tuberculosis usually affects the pleura, lymph nodes, peritoneal, gastrointestinal tract, genitourinary tract and vertebrae. Overall, excluding the influence of HIV infection, about 85% of adults present with pulmonary parenchymal disease, 15% with disease at extrapulmonary sites, and approximately 4% with simultaneously active disease at intra and extrathoracic locations.[1,2]

Abdominal tuberculosis is one of the manifestations of extrapulmonary tuberculosis. It may involve

gastrointestinal tract, peritoneum, mesenteric lymph nodes, liver, and also spleen. Intestines and peritoneum can be infected from mesenteric glands, from infected fallopian tubes, direct spread from an adjacent organ and through hematogenous spread. Direct infection of the intestinal wall is possible after ingestion of unpasteurized milk or swallowing large quantities of bacilli from cavitary lung lesion. Endogenous reactivation occurring years after hematogenous spread is common.[5,6,7]

Abdominal tuberculosis is predominantly a disease of young adults. Two-thirds of the patients are 21-40 years old and the sex ratio is equal. Most patients have constitutional symptoms of fever (40-70%), pain (80-95%), diarrhea (11-20%), constipation, alternating constipation and diarrhea, weight loss(40-90%), anorexia and malaise. Pain can be either colicky due to luminal compromise or dull and continues when the mesenteric lymph nodes are involved.[7]

The abdominal form of tuberculosis has an insidious course like any other chronic infectious disease without any specific laboratory, radiological or clinical findings. Due to this non specificity, there are great difficulties in making the diagnosis. As a result, the diagnosis of abdominal tuberculosis still a challenge.

Treatment of standard regimen for abdominal tuberculosis is effective. It includes initiation phase for 2 months and continue with continuation phase for 7 months. Steroid as an adjunctive treatment still controversies. Steroids may be used to reduce acute inflammation and limit delayed fibrotic complications.

CASE ILLUSTRATION

Mr. A, 31 years old, was admitted in our hospital with chief complain of fatigue which worsens since 1 week prior to admission. From his present illness, since 1 week prior to admission, he complained of fatigue. He had fever, nausea, vomiting, and lost of appetite. There was diarrhea, 1 to 5 times daily, yellowish, no bloody stool nor mucus. Diarrhea was accompanied with abdominal pain. His abdomen was also enlargent.

About one year prior to admission, he got difficulties in breathing, chronic productive cough with serous sputum, no hemoptysis. There was fever, night sweat, body weight loss, and fatigue. Urination and stool were normal. In other hospital, the patient was diagnosed to have lung infection and must take several drugs including rifampicin. He was told to have regular visit at outpatient clinic and the drugs must be taken for minimum 6 months of period, but after 3 months he felt his condition better, thus he stopped the drugs by himself.

About 8 months prior to admission, his abdomen got swelling and got pain in all quadrant. He also got diarrhea, 1 - 5 times daily, no bloody stool. He had nausea and vomiting and low grade fever. He also had cough with serous sputum. Hemoptysis was denied. In other hospital, he was told that his spleen was enlarged. He then was referred to our hospital for further management.

As out patient, at Internal Medicine Department in our hospital, he was taken ascites fluid for examination. Upper and lower gastrointestinal tracts endoscopies were performed. In pulmonology division, his chest X-ray showed specific process, and he was planned to take sputum examination before giving treatment. Unfortunately, the patient didn't have regular visit to his doctor. He was also referred tosurgery department for lymph node biopsy.

From his previous history of illness and family history, there was no history of bloody cough, chronic cough, liver disorder, malignancy, diabetes mellitus, and hypertension. He underwent appendices operation in 2003. He has smoking habit, 1 to 2 packs daily since 10 years ago, alcohol drinking habit about 1 bottle every week since 1 year ago, and sniffing drug user. Intravenous drug user was denied. There is also history of free sexual habit.

The physical examination found general condition was general weakness, moderately ill, body weight 47 kg, height 173 cm, body mass index 16. Blood pressure 110/70 mmHg, heart rate of 110 times/minute, respiratory rate of 28 times/minute, body temperature of 36.8^{o}C. Conjunctiva was pale, sclera was no icteric. Oral thrus was positive. JVP 5 - 2 cm H_2O. Lung auscultation found breath sounds vesicular, wet rales, no wheezing. Heart examination was within normal limit. Abdominal examination found distended, tense, pain in all quadrant, liver not palpable, spleen Schuffner 1, dull in middle, lower and middle right, no chest board phenomenon, no mass, and protrude of umbilicalis. Extremities examination found no edema and palmar eritema. There was enlargement of supraclavicula, right and left inguinal lymph nodes, diameter 1.5 cm, tender, mobile, no sign of inflammation. Digital rectal examination found no mass, yellow feces without any blood.

Laboratory examination found anemia microcytic hypochrome (Hb 7.1 g/dL, MCV 68 fl, MCH 22 pg, MCHC 32 g/dL) with normal leukocyte and platelet count. There was increase in erythrocyte sedimentation rate (ESR 110 mm/hour), increase in ALT (ALT 56 U/L) and hypoalbuminemia (albumin 2.4 g/dL).

From previous examination as out patient, abdominal CT scan showed ascites, splenomegaly with multiple hypodens lesion, and paraaorta lymph nodes enlargement. Abdominal ultrasound showed splenomegaly with nodul, chronic hepatitis, and ascites.

Cytology ascites showed reactive mesotel. Colonoscopy revealed hemorrhoid interna gr II and colitis infective with differential diagnosis colitis TB, Crohn's disease, and ulcerative colitis. Histopatology from colon and ileocaecal biopsy revealed colitis infective, no sign of malignancy and chronic active ileitis, and no sign of malignancy. Three consecutive acid fast bacilli sputum were negative. Chest X-ray found infiltrates in both apex lungs and parahilar. Top lordotic chest X-ray showed fibroinfiltrates and calcification in the apex of right lung.

Endoscopy revealed mild gastritis, mild esophagitis and biliary reflux. Histopatology of biopsy of gaster showed non active chronic gastritis, atrophic with intestinal metaplasia. *Helicobacter pylori* not found. Cytology of supraclavicula lymph node showed possibility of specific process.

The problems at admission were chronic diarrhea, pulmonary tuberculosis with secondary infection, peritonitis, splenomegali with nodul, lymphadenopathy, candidosis oral, hypoalbuminemia, anemia microcytic hypochrome, suspecting of HIV infection/AIDS, and malnutrition.

Chronic diarrhea based on history of diarrhea since 8 months, 1 to 5 times daily, liquid stool, no bloody diarrhea, sometimes with mucus, body weight loss, loss appetite, nausea, vomiting. Abdominal was found distended, tense, dull in middle, right middle, lower right, no chest board phenomenon, and no mass. Laboratory examination showed anemia microcytic hypochrome, no leukocytosis, segment elevation. Colonoscopy and histopatology of colon biopsy revealed colitis infective. Chronic diarrhea was suggested due to colitis infective with differential diagnosis of colitis TB, Crohn's disease, and ulcerative colitis. Plan of diagnosis was stool analysis. Treatment was intravenous fluid Ringer lactate 1,500 cc daily, anti diarrhea, and ceftriaxone 2 g once daily.

Pulmonary tuberculosis with secondary infection based on chronic cough with sputum, fever, body weight loss, night sweat, history of having tuberculosis treatment, tachypneu, wet rales in both lungs, palpable lymph nodes. No leukocytosis, there are segment and erithrocyte sedimentation rate elevation. Acid fast bacilli sputum was negative. Chest X-ray showed specific process. Plan of diagnosis was acid fast bacilli sputum and culture, microorganism sputum and culture, consultation with Ophthalmology Department, biopsy for histopathologic from lymph nodes. Treatment was INH 1 x 300 mg, rifampisin 1 x 450 mg, pirazinamid 2 x 500 mg, etambutol 2 x 500 mg and ceftriaxone 1 x 2 g.

Peritonitis based on abdominal pain and tenderness with abdominal distended, tense, pain in all quadrant, dullness in middle, right middle and lower right and middle right dullness, no chest board phenomenon. Digital rectal examination found no mass, yellow faeces, no blood. Abdominal ultrasound and CT scan showed ascites, lymph nodes paraaorta enlargement, and multiple nodules in spleen. Peritonitis was suggested due to peritonitis tuberculosis with differential diagnosis peritonitis carcinomatosis. Diagnostic plan was abdominal ultrasound for second times, paracentesis with ultrasound guided.

Splenomegaly with nodul was based on spleen Schuffner 1, lymphadenopathy, previous abdominal ultrasound and CT scan showed ascites, lymph nodes paraaorta enlargement, and splenomegali with multiple nodules. Splenomegaly with nodul was suggested due to malignancy with differential diagnosis tuberculosis of the spleen. Diagnostic plan was LDH, abdominal ultrasound and spleen biopsy.

Candidosis oral based on history of free sexual habit, oral thrush positive. Candidosis oral suggested due to HIV infection with differential diagnosis of immunocompromise person due to malnutrition and chronic infection. Diagnostic plan was anti HIV and CD4. Treatment of candidosis was mycostatin.

Hypoalbuminemia based on loss appetite, chronic diarrhea, albumin 2.4 g/dl. Hypoalbuminemia suggested due to malnutrition with differential diagnosis malabsorption and liver function disorder. Diagnostic plan was complete liver function test, albumin serial. Treatment was high calories and protein diet 2,700 calorie, extra protein from egg and milk.

Anemia microcytic hypochrome based on fatigue, loss appetite, chronic diarrhea, no bloody stool, tacypneu, tachycardia, and colonoscopy showed colitis infective. Laboratory found anemia microcytic hypochrome. Anemia microcytic hypochrome suggested due to iron deficiency with differential diagnosis anemia chronic disease and anemia of chronic bleeding. Diagnostic plan was serial peripheral blood count, blood count morphologic, reticulocyte, serum iron, TIBC, feritin, fecal analysis, benzidine test. Treatment was packed red cell transfusion, treatment for underlying disease, and high calories high protein diet.

Suspecting of HIV/AIDS based on history of free sexual habit, prolonged fever, chronic diarrhea, body weight loss, and candidosis oral. Diagnostic plan was anti HIV and CD 4.

Malnutrition based on loss of appetite, nausea, vomiting, chronic diarrhea, pulmonary tuberculosis. Body weight 47 kg, height 173 cm, with body mass index 16. Upper arm circumferential was 23. Malnutrition suggested due to difficult intake and chronic infection with differential diagnosis HIV infection with wasting syndrome. Diagnostic plan was serial body weight and upper arm circumferential measurement, consult to nutritionist for nutrition

analysis. Treatment was high calories and high protein diet 2,700 calories, with extra milk.

At 7 days care, patient complaint of become more nausea. No more diarrhea. On physical examination we found sclera was slightly icteric. Oral thrus was disappeared. ALT and AST increase more than 3 times normal limit (ALT 214 U/L and AST 104 U/L), direct and indirect bilirubin increase (direct bilirubin 1.4 mg/dL and indirect bilirubin 0.4 mg/dL), and more hypoalbuminemia (albumin 1.8 g/dl). Anti HIV screening was non reactive. CD 4 was low (CD4 154 uL). Fecal analysis showed positive occult blood test and food maldigestion. Fecal culture full with Candida colonies and B. hominis infection. Fluconazole 150 mg once daily and metronidazol 500 mg three times daily were given.

The new problem was drug induced hepatitis due to antituberculosis treatment. The antituberculosis drugs was stopped and substituted to second regimen which consisted of ethambutol 500 mg twice daily, Streptomycin 750 mg once daily, and levofloxacin 500 mg once daily. Intravenous albumin 20% 100 cc, 3 times was also given. There was no choroiditis tuberculosis. ICT tuberculosis was positive and tuberculin test was negative.

At 10 days care, the patient no more cough. There were no more rales. The three consecutive acid fast bacilli sputum were negative. The sputum culture showed streptococcus viridans which sensitive to ceftriaxone.

The ascitic fluid analysis revealed conclusion of chronic infection with differential diagnosis of lymphoproliferative disease. The specimen for PCR TB was not send due to financial problem. The cytologic of ascitic fluid showed reactive mesotel, no sign of malignancy. The histopatology results from liver biopsy showed liver tuberculosis. The cytologic results from spleen biopsy showed no sign of malignancy. Dexamethason injection 10 mg three times daily was added as the therapy regimen for 2 weeks.

On 21st days care, patient complained of having severe abdominal pain in all quadrants. He couldn't have defecation and flatus. There was decreasing in bowel sound. The three positional abdominal X-ray showed partial obstruction ileus suspected in small bowel. Suggestion from Surgery Department was perform nasogastric tube. He was told to fasting and was given total parenteral nutrition Triofusin E 1,000 per 12 hours and dextrose 5%: NaCl 0.9% per 8 hours was given. On the third day of fasting, patient could have flatus, no defecation, and the bowel sound became normal. He then was given food step by step and the response of the treatment was good.

On 27thdays care, patient complained for the second times of having severe abdomen pain, he couldn't have defecation and flatus. There was increasing in bowel sound in right quadrant and decreasing in left quadrant abdomen. The three positional abdominal X-ray showed partial obstruction in distal small bowel suspected in terminal ileum. Suggestion from Surgery Department was perform nasogastric tube. On the next day of fasting, patient could have flatus and defecation, and the bowel sound became normal. Food was given step by step and the response of the treatment was good.

The peritoneoscopic was planned twice but due to minimal ascites, it was canceled. The third abdomen ultrasound still showed multiple nodules and enlargement of paraaorta lymph nodes but the ascites was very minimal.

The patient was discharged on the 30th days care. There was no abdominal pain, his appetite increased and his body weight tended not gaining. The size of lymph nodes was decreased to 0.8 cm. The pulmonary and extrapulmonary tuberculosis including abdominal tuberculosis and lymph node tuberculosis was established. Isoniazid was planned to give with titration also with oral prednisone in polyclinic. Anti HIV treatment was planned to be given in the next three months.

Two months after taking tuberculosis treatment, patient's body weight increased 10 kg. There was no abdominal pain. The lymph nodes were not palpable. The abdominal ultrasound was performed and showed normal liver, no ascites, no enlargement of paraaorta lymph nodes, and no splenomegaly. The nodul in spleen was decrease in size and only one nodul is found. The colonoscopy was performed for evaluation and showed some improvement.

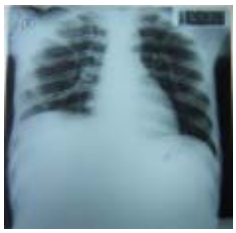

Figure 1. Chest X-ray showed specific process with fibro infiltrat in apex right lung and infiltrate in right pericardia

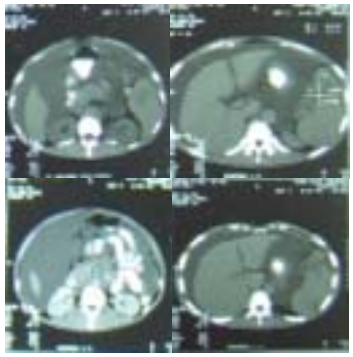

Figure 2. Abdominal CT scan before admission showed ascites, paraaorta lymph nodes enlargement, and splenomegaly with multiple hypodens lesion

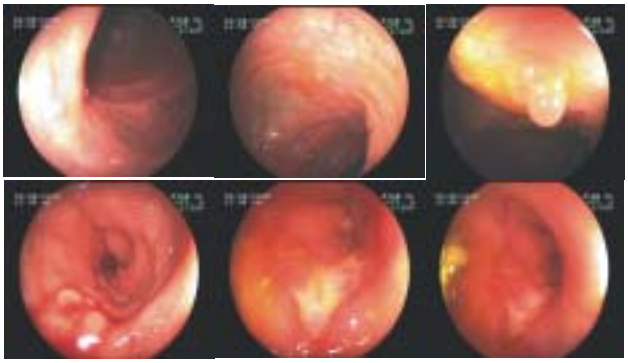

Figure 3. Colonoscopy before admission showed colitis infective with ulceration, polyp and polipoid lesion, and lymphoid follicle hyperplasia

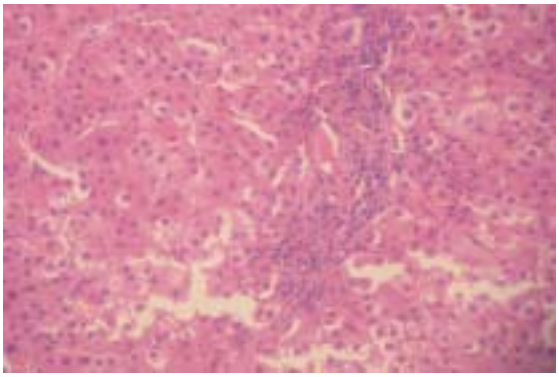

Figure 4. Histopathology of liver biopsy found several tubercles consists of epiteloid cells and lymphocyte with the conclusion was liver tuberculosis

DISSCUSION

The main problem in abdominal tuberculosis is diagnostic problem and in the management of tuberculosis in this case, the problem of drug induced hepatitis due to anti tuberculosis drugs was emerged.[8] The signs and symptoms of abdominal tuberculosis usually not specific and can mimic other diseases. In this case, the patient had symptoms and signs that mimic malignancy including fever, weight loss, lost of appetite, ascites, multiple lymphadenopathies and splenomegaly with multiple nodules. After careful examinations, the diagnostic of abdominal tuberculosis including peritonitis tuberculosis, intestinal tuberculosis, liver tuberculosis and suspected of spleen tuberculosis was established.

Abdominal tuberculosis in this patient was established based on history, physical examination, laboratory and other supporting examination and good response after anti tuberculosis treatment. This abdominal tuberculosis was also found with pulmonary tuberculosis. History revealed symptoms of abdominal pain, night sweat, weigh loss, loss appetite, nausea, vomiting, abdominal distended and abdominal pain, and chronic diarrhea. As in references, fever and malaise, abdominal pain and weight loss are the most common described symptoms, being found in 72%, 60% and 58% of cases. Abdominal distension, usually due to ascites, is described varying from 10-65%. The proportion of cases with coexisting respiratory tuberculosis has varied from less than 30-40%. Simadibrata et al, in a retrospective study in Cipto Mangunkusumo hospital in year 2001 - 2003, found 59 patients with abdominal tuberculosis. This abdominal tuberculosis was coincided with pulmonary tuberculosis in 25 (42.3%) cases. Diarrhea was found in 32.4% patients. Fever was found in 59.5% patients. Night sweating was found in 40.5%. Body weight loss and loss of appetite was found in 78.4%.[9-10]

In peritonitis tuberculosis, there are three forms i.e. wet type with ascites, dry type with adhesions, and fibrotic type with omental thickening and loculated ascites.[10] Based one physical examination and abdominal ultrasound, this patient is included in fibrotic type with omental thickening and loculated ascites. The USG guided paracentesis results showed ascitic fluid asexudates, the polymorphonuclear cells greater than the mononuclear cells, ascitic LDH 143 U/L. The serum ascites albumin gradient (SAAG) was less than 1.1 mg/dL. There was no acid fast bacillus on smear and culture. From reference, ascitic fluid in tuberculous peritonitis is exudative and contains between 50 and 10,000 leukocytes per uL, the majority of them being lymphocytes, although polymorphonuclear leukocytes occasionally predominate. The ascitic fluid protein exceeds 2.5 g/dL in more than 85% of cases, and the glucose concentration usually is less than 30 mg/dL. LDH usually more than 90 U/L. Acid fast organisms are rarely seen on smears of the fluid. Staining for acid fast bacilli is positive in less than 3% of cases. A positive culture is obtained in less than 20-80% of cases and it takes 6 - 8 weeks for the mycobacterial colonies to appear. Singh et al, in an earlier study cultured 1 liter of ascitic fluid after centrifugation and obtained 83% culture positive. Serum ascites albumin gradient (SAAG) will usually be less than 1.1 mg/dL and indicating that the cause of ascites is not portal hypertension. Polymerase chain reaction (PCR) for detection compare with culture in specimen which negative acid fast bacilli, the sensitivity and specificity were 80% and 97%. Ascitic adenosine deaminase levels are useful as diagnostic tools. Taking cut off of 33 U/l, the sensitivity and specificity were 100% and 97%.[10-13]

Peritoneal biopsy guided by laparoscopic or peritoneoscopic can be diagnostic in more than 95% of patients, up to 100% will be positive and should be strongly considered to confirm diagnosis. This patient was planned to have a peritoneoscopy but due to minimal ascites and the harm risk to do it, this procedure was canceled.

In gastrointestinal tuberculosis, endoscopic findings are not patognomonic. Tuberculosis may occur in any location of gastrointestinal tract, although lesions proximal to the terminal ileum are unusual. The most common sites of involvement are the terminal ileum and the caecum. In the terminal ileum or caecum, the most common manifestations are pain, which may be misdiagnosed as appendicitis, and intestinal obstruction. A palpable mass may be noted and together with the appearance of the abnormality on barium enema or small bowel film, may easily be mistaken as carcinoma.[12]

The intestinal tuberculosis in this patient based on history of chronic diarrhea, fever, loss appetite, weight loss and abdominal pain. The colonoscopy found hyperemic mucosa with erosion, edema, ulceration, small multiple polyp, polipoid lesion and lymphoid follicle hyperplasia. Misra et al, reported the clinical features and colonoscopic appearance in 50 patients with colonic tuberculosis. Abdominal pain, fever, anorexia, weight loss and diarrhea were the common symptoms. The colonoscopic features consisted of ulcers (92%), nodules (88%), deformed caecum and ileocaecal valve (42%), strictures (25%), multiple fibrous bands (8%) and polypoid lesion (6%). The histologic features of colonic biopsy in this patient found colitis infective and chronic active ileitis. Misra et al, reported histologic examination of the colonic biopsy specimens of the tuberculosis in 50 patients showed well-formed, non caseating granulomas (18%), collection of loosely arranged epitheloid cells (40%) and chronic non-specific inflammatory changes (42%).[13] The histopatology of colon biopsy only found colitis and ileitis. The granulation or caseation feature wasn't found. Granulomas may not be seen in mucosal biopsies of nodules, ulcers or other lesions because they are mostly located in the submucosa of the tissue. Caseation may be absent or be present only in the lymph nodes, and may not even be found in patients who have received anti-tuberculous treatment in the past.[13]

Chronic hepatitis with early cirrhosis in this patient was first suggested due to hepatitis viral and alcoholic liver disease with possibility of liver tuberculosis. Later, the Hbs Ag and anti HCV results were negative. The liver tuberculosis was still in suspicion. The histopatology of the liver biopsy was done and the liver tuberculosis was established. The spleen ultrasound and spleen CT scanning showed multiple nodules. At first the malignancy was suggested besides the spleen tuberculosis. But the cytologic found no sign of malignancy and no sign of tuberculosis. In this case, based on the tuberculosis at other site in abdominal, the spleen tuberculosis is sill in a high index of suspicion. From reference, the spleen and the liver are much less commonly affect in tuberculosis due to the low oxygen tension in these organs. The liver and spleen is usually involved as part of miliary disease. It is usually a diffuse infiltrative process. The miliary involvement of the liver can produce a 'bright' appearance on ultrasound, which, however, is non specific. Nodular forms of liver involvement are described which can mimic either cirrhosis or carcinoma. From several studies, the spleen tuberculosis was found from CT scan as hypoechogenic and hypodense nodules.[6,14,15,16]

Tuberculin test in this patient has negative result. It could happen because of inadequate immunological response due to chronic infection and malnutrition as in this patient. Tuberculin test can be negative in HIV positive persons, in the undernourished, those immunosuppressed by medication and those with advanced disease.[9] Unreactive tests may occur in up to 20% of patients with active tuberculosis if sensitized T cells are depleted or nonfunctional due to old age or an immunodeficiency state.[6] This patient are HIV negative. The CD4 result was low suggested the patient had an immunodeficiency state probably due to chronic infection and malnutrition. But the possibility of having HIV infection is still in high index of suspicion so that the anti HIV was planned to be checked at 3 months after the first result.

In this case, the ICT TB was positive. ICT (rapid immunochromatographic assay) based on detection of IgG antibody directly to antigen which is secreted by M. tuberculosis. This test has sensitivity and specificity of 56.7 % and 90.4%. For extrapulmonary TB, its sensitivity is 16.7%. Sensitivity is higher in positive acid fast bacilli and in pulmonary TB.[17,18,19]

In patient with colonic tuberculosis, minor rectal bleeding is known to occur frequently, major bleeding has only occasionally been reported.[13] In this case, patient has microcytic hypochrome anemia. The patient didn't have any complain of bloody stool and the digital rectal examination didn't find any abnormality. The laboratory found increase of reticulocyte and the fecal analysis showed positive occult blood test. The bleeding was suggested due to intestinal tuberculosis.

The most common complication of intestinal tuberculosis is obstruction. In this case, the patient was having partial obstruction ileus. Those were suggested due to several reasons. From reference, the obstruction of the small or large bowel occurs in 10-60% of patients and is classically chronic and intermittent. Factors responsible for obstruction include contraction of collagenous tissue following healing of the circumferential intestinal tubercular ulcer, hypertrophy of the bowel wall leading to narrowing of the lumen, kinking of intestines due to intraperitoneal

adhesions and retraction of mesentery and shortening of the right colon by scar tissue. The obstruction was also due to narrowing of the lumen by hyperplastic caecal tuberculosis, by strictures of the small intestine, which are commonly multiple, or by adhesions. Adjacent lymph nodes involvement can lead to traction, narrowing and fixating of bowel loops. In India, around 3 - 20% of all cases of bowel obstruction are due to tuberculosis. In a large series of 348 cases of intestinal obstruction, Bhansali and Sethna found tuberculosis to be responsible for 54 (15.5%) cases; 33 cases were small bowel and 21 large bowel obstruction.[10,20]

Treatment for tuberculosis infection in this patient was categorized as first category tuberculosis treatment of WHO which is including in severe extrapulmonary tuberculosis i.e. Isoniazid, rifampisin, pirazinamid, and etambutol. But in the seven days of treatment, the patient suffered from drug induced hepatitis. The ALT and AST increased more than three times than normal limit. The tuberculosis drugs then stopped and substituted to other regimen i.e. etambutol, streptomycin and levofloxacin. The rifampisin than given with titration starting from low doses to normal dose and after liver function test become normalize, patient was planned to be given isoniazid with titration. Tuberculosis drugs particularly isoniazid and pirazinamid have a direct effect of hepatotoxicity, otherwise rifampisin can have direct toxicity usually in combination with isoniazid. From references, regular monitoring of liver function test is indicated for all patients receiving tuberculosis therapy. When patients experience serious hepatitis, all potential hepatotoxic drugs should be held until liver function test and symptoms normalize, then the drugs can be reintroduced one with titration at 3 to 4 day intervals, monitoring liver function tests and symptoms to identify the offending agent.[3,21]

This patient was given the injection steroid dexametason for 2 weeks and then substituted to prednisone until 6 weeks and then tapering off. Steroids may be used to reduce acute inflammation and limit delayed fibrotic complications. Commonly, prednisone 1 mg per kilogram of body weight is given.[3]

Microcytic hypochrome anemia in this patient is due to gastrointestinal tract bleeding and chronic infectious disease. Blood peripheral showed microcytic hypochrome anemia with high reticulocyte and low ferrous level. Fecal analysis showed positive occult blood test. The characteristic of serum iron studies show typical pattern of low serum iron, low TIBC and elevated serum feritin level.[22] The treatment was directed to treat underlying disease and supported with high calories high protein diet and ferrous supplement.

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