

## A Case of Tuberculous Peritonitis Accompanied By Tuberculous Pleuritis

Larasati A.Wahyu<sup>1\*</sup>, Pramarta Y. Dwiputra<sup>2</sup>

Department of Internal Medicine Buleleng Regional General Hospital Bali, Indonesian

Email: [langgira@gmail.com](mailto:langgira@gmail.com)

\*Correspondence

### ABSTRACT

**Keywords:** TB Tuberculous peritonitis is a form of extrapulmonary Peritonitis, TB Pleurisy, tuberculosis, a peritoneal or visceral inflammation caused by Ascites, Effusion, Body Mycobacterium tuberculosis. The disease is rarely Fluid Analysis. independent but is usually a continuation of the tuberculosis process elsewhere, especially pulmonary tuberculosis. We report a case of TB peritonitis accompanied by TB pleurisy, a 29-year-old female patient with complaints of an enlarged abdomen, heartburn, fever, diarrhea, and decreased appetite. Treatment history was Acitral, Zinc, and Metronidazole. On physical examination, it was found that the general condition was weak, and the axilla temperature was 39.5°C. Thorax examination: decreased vesicular sound on the left chest. Abdominal examination found distension, epigastric tenderness, undulation, shifting dullness, checkerboard phenomenon, and increased bowel noise. Laboratory examination of complete blood within normal limits. Complete stool; yellow color, mucus (+), leukocytes 4-6/LPB, bacteria (+). The thorax photo showed left pleural effusion, BOF 3 position: ascites. Abdominal ultrasound results: thickening of the peritoneum, ascites, suspected TB peritonitis. Results of ascites and pleural fluid analysis: rivalta (+) and Adenosine Deaminase (ADA) increased. From anamnesis, physical examination and supporting examination can be established to diagnose TB peritonitis and TB pleuritis, followed by OAT therapy and FDC for 12 months. From this case, it can be concluded that clinical and supporting examinations (radiology) are needed to diagnose correctly, and body fluid analysis examinations can help confirm the diagnosis.



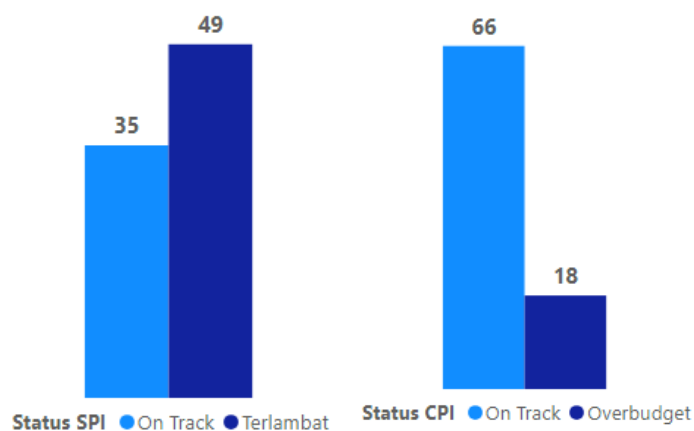
### Introduction

The successful completion of a project is the goal of carrying out a project, where the project is a unique activity and has a period (end); then, the success of the completion of a project will be determined at the end of the project (Vaid & C. Kane, 2017). The classic criterion of practice is the measure of a project's direct performance against its main design parameters, schedule (time), budget (cost), scope, and quality, which the

literature tends to refer to as measures of project management success. This definition was already established in the earliest discussions of projects in the management literature (De Saram & Friedland, 2019).

Delays in construction projects refer to situations where the project is not completed according to the planned schedule. This means construction work took longer than expected in the initial planning (Dahale et al., 2021). Delays in construction projects can occur for various reasons, including those previously mentioned, such as scope changes, lousy weather, obstacles in obtaining permits, delays in shipping materials, and other issues. A budget for a construction project means that the project's cost exceeds the budget set or planned in the initial planning (Atzori, Vidili, & Delitala, 2012). In this situation, the actual expenditure on a construction project exceeds a predetermined estimated cost.

In a construction project, delays and overbudgets will affect the project's performance, which also affects the performance of the construction company working on the project (Wibowo, 2023). In this study, the research object used is PT WER, where PT. WER is a company engaged in construction and PT. WER has applied project management knowledge to its construction projects (Pramugaria et al., 2017). At PT. WER currently has a problem where in the July 2023 period, there are 84 ongoing projects, of which 18 projects are overbudgeted according to CPI calculations, and there are 49 projects experiencing delays according to SPI calculations as illustrated in Figure 1 below:



**Figure 1 The Project is late and over budget at PT. WER**

Of the 18 (eighteen) projects that experienced overbudget, including four road and bridge type projects, five water building type projects, three building type projects, one transmission and distribution type project, 3 EPC type projects, and two railway type projects, as illustrated in Figure 2 TB peritonitis is a form of extrapulmonary tuberculosis, a peritoneal or visceral inflammation caused by Mycobacterium tuberculosis<sup>1</sup>. The most common sites of extrapulmonary TB in the body are the lymph nodes, bones, joints, pleura, spinal cord, brain, and abdominal cavity (Chen et al., 2021). The disease rarely stands alone but usually continues the tuberculosis process elsewhere, especially pulmonary tuberculosis<sup>2</sup>.

The World Health Organization (WHO) estimates that there are 8.6 million cases of TB, 80% in 22 countries (Zirta, Uyainah, & PN, 2015). Extra-pulmonary tuberculosis occurs in about 20% of tuberculosis, while abdominal tuberculosis accounts for about 10% of extra-pulmonary tuberculosis. The incidence and severity of abdominal TB have been reported to increase with the rising incidence of TB and HIV infection<sup>2</sup>. TB peritonitis is a rare type of abdominal TB, reported to occur in less than 5% of all TB patients (Sudirman, 2018). TB peritonitis cases are often found in individuals aged 25-45 years, with a female-to-male ratio of 1.5:1<sup>3</sup>.

TB peritonitis infection develops slowly and is characterized by nonspecific symptoms, so delayed diagnosis can increase morbidity and mortality (Murlistyarini, Prawitasari, & Setyowatie, 2018). Patients with TB peritonitis usually present with symptoms lasting 1-12 months and present with symptoms of abdominal pain, weight loss, loss of appetite, fever, diarrhea, constipation, rectal bleeding, edema, and ascites, which require specific investigations to diagnose<sup>4</sup>.

The following case report will report a patient with TB peritonitis accompanied by TB pleurisy.

## Research Methods

A 29-year-old woman came to the emergency room of the Buleleng Regional General Hospital with complaints of an enlarged abdomen and fever; the complaints had appeared for  $\pm$  19 days SMRS, and complaints accompanied by heartburn complaints such as nausea and vomiting were denied. Other complaints are liquid stools with a frequency of 2-3 times a day accompanied by mucus, without blood since  $\pm$  19 days ago, urination within normal limits, and decreased appetite. Complaints, such as coughing, tightness, and weight loss, are denied. Menstruation is within normal limits. The patient said he had no previous history of illness. Treatment history: The patient took Acitral 3x1, Zinc 1x1, and Metronidazole 3x500 mg, obtained from a general practitioner.

The physical examination revealed a weak general condition, compos mentis consciousness, blood pressure 120/80, pulse 90x, respiration 20x, axilla temperature 39.5°C, Spo<sub>2</sub> 99% on room air. Body weight is 50kg, height is 160 cm, and BMI is 19.5 kg/m<sup>2</sup> (average weight).

On examination of the head and neck, there was no anemia, icterus, cyanosis, oral candidiasis on the tongue, enlarged lymph nodes, or increased JVP. On thorax examination and chest inspection, there was a symmetrical movement of the chest wall, no retraction, and normal fremitus; on auscultation, typical vesicular sound in the right chest, and decreased vesicular sound in the left chest, no rhonchi and wheezing. Cardiac examination was within normal limits with no murmurs, gallops, or extrasystoles.

Abdominal examination; inspection found distension, on palpation; muscular defans (-) epigastric tenderness (+), undulation (+), on percussion; shifting dullness (+), checkerboard phenomenon (+), on auscultation found increased bowel noise. Examination of extremities: warm (+) on all four extremities, edema (-), CRT < 2 seconds.

From the results of the complete blood laboratory examination; WBC: 5.58  $10^3/\mu\text{L}$ , HGB: 11.2 g/dL, MCV: 81.0 fL, MCH, 26.5 pg, PLT 381  $10^3/\mu\text{L}$ , GDA: 90 mg/dL, Ureum: 14.0 mg/dL, Serum Creatinine: 0.56 mg/dL, SGOT; 25.3, SGPT: 21.4, Sodium: 134.2 mmol/L, Potassium: 4.06 mmol/L, Chloride: 98.9 mmol/L, Albumin: 4.25 g/dl. Upon complete urine examination, the results were within normal limits. Complete stool; yellow color, mucus (+), blood (-) leukocytes 4-6 / LPB, bacteria (+), worms (-), fungi (-), amoeba (-). On the thorax photo was an impression of left pleural effusion, ECG: within normal limits, BOF 3 position: there were ascites, no ileus, and pneumoperitoneum. Abdominal ultrasound results: thickening of the peritoneum, ascites, suspected TB peritonitis, Liver, gallbladder, pancreas, spleen, kidneys, buli-buli, and uterus within normal limits. The rapid molecular test (TCM) and HIV test were negative.

The patient was diagnosed with the observation of ascites ec suspected TB, pleural effusion ec suspected TB, and Acute Gastroenteritis. The next plan is to ascite pleural puncture and perform fluid analysis. Therapy given: Nacl 0.9% 20 tpm, Paracetamol 1gr IV (if fever), Ceftriaxone 1x2gr IV, Lanzoprasole 1x30 mg IV, Ondancentron 3x4 mg IV, Furosemide 40 mg PO, Spironolactone 100 mg (PO).

Ascitic fluid analysis results; rival (+), Adenosine Deaminase (ADA): 56.51 U/L, on pleural fluid analysis; Leukocyte cell count: 1584 cells/uL, protein 5.9 g/dL, rivals (+), Adenosine Deaminase (ADA): 50.28 U/L, Pleural LDH: 680, histopathologic examination of the fluid; there were no malignant cells. Diagnosis: TB peritonitis and TB pleurisy, followed by OAT therapy; FDC 1x3 tab. The patient was hospitalized for five days with an improved condition, then the Anti Tuberculosis Drug therapy program for 12 months.

## Results and Discussion

TB peritonitis has nonspecific clinical symptoms. The most common complaints are abdominal pain (73%) and ascites (93%), followed by loss of appetite and weight, nausea, vomiting, cough, fever (58%), diarrhea, constipation, and night sweats; ascites can be caused by peritoneal tuberculosis or can originate from liver disease, malignancy, heart, kidney and other infectious diseases<sup>5</sup> (Febrianto, 2019). Peritoneal TB with ascites may have less tenderness than pyogenic peritonitis with perforation<sup>5</sup>. Peritoneal TB has been classified as the more common "wet type," characterized by ascites, and the rarer "plastic or fibroadhesive type," which manifests as an abdominal mass of adherent bowel loops<sup>6</sup>.

TB can reach the peritoneum hematogenous via the lymphatic system, from ingestion of contaminated sputum from pulmonary TB, contaminated food (especially unpasteurized milk in the case of *Mycobacterium bovis*), or through direct contact from adjacent foci of infection<sup>7</sup> (JUWITA, 2013).

In tuberculous peritonitis, clinical symptoms are non-specific or variable. Complaints and symptoms occur slowly over months, so patients are often unaware of their condition. Complaints range from 2 weeks to 2 years, with an average of more than 16 weeks<sup>7</sup>.

On physical examination of patients with tuberculous peritonitis, the most common symptoms are fever, ascites, abdominal swelling, abdominal pain, pallor and fatigue, pleural effusion, hepatomegaly, splenomegaly, intra-abdominal tumor, checkerboard phenomenon, lymphadenopathy, and lung & pleural involvement (based on chest photograph)<sup>7</sup>. Infection of the adjacent pleura may reach the peritoneum, resulting in peritoneal tuberculosis. Pleural effusion is observed in 22 to 32% of patients with peritoneal tuberculosis and pulmonary source in 15 to 20% of cases<sup>8</sup>.

No single test can effectively rule out the diagnosis of peritoneal TB. TB, and a combination of socio-epidemiologic history (e.g., travel, homelessness, incarceration, sick contacts, drug use) and immunologic risk assessment is essential. Classic symptoms such as fever, weight loss, and night sweats may be absent<sup>8</sup>.

The patient was directed to the diagnosis of tuberculous peritonitis and pleural tuberculosis based on several symptoms such as an enlarged abdomen, fever, gastrointestinal complaints such as diarrhea, and typical signs of tuberculous peritonitis, namely the checkerboard phenomenon and unilateral pleural effusion (often, pleural effusion is unilateral, mild to moderate in volume, which is 25 to 75% of patients)<sup>8</sup>. Furthermore, evaluation with other supporting examinations should be done to confirm the diagnosis since most of the patients' complaints are not typical symptoms<sup>9</sup>.

Patients were evaluated for etiology according to standard protocols. Complete blood count, liver function tests, renal function tests, chest X-ray, abdominal ultrasonography, and ascitic fluid analysis, including cell count, albumin, protein, and Adenosine Deaminase Activity (ADA)<sup>9</sup>.

Changes in hematological indices, including white blood cell count and erythrocyte sedimentation rate, are nonspecific. Mild to moderate normochromic, normocytic anemia and thrombocytosis are frequent findings. The white blood cell (WBC) count is usually average, but lymphomonocytosis is uncommon<sup>10</sup>. The erythrocyte sedimentation rate is almost always elevated in at least 50% of cases. Usually, in TB peritonitis, the ascitic fluid is straw-colored with protein  $>30\text{g/L}$  and a total cell count of  $500\text{-}1500/\text{il}$ , predominantly lymphocytes ( $>70\%$ ). A low serum-ascites albumin gradient ( $<11\text{ g/L}$ ) is seen in 100% of patients with TB peritonitis, but the specificity remains low. Due to its low accuracy, ascites LDH measurement is not routinely used<sup>10</sup>. In TB pleurisy, biochemical features include elevated protein levels of more than  $4.5\text{ g/dL}$  and slightly elevated DHL<sup>10</sup>. The diagnosis of TB pleurisy is usually made through a combination of clinical history, pleural fluid analysis (predominantly lymphocytic cell count, protein concentration  $>3.0\text{ g/dL}$ , elevated lactate dehydrogenase often  $>500\text{ IU/L}$ , and glucose level  $<60\text{ mg/dL}$ ), positive culture in sputum or pleural fluid, and positive ADA level  $>40\text{ U/L}$ <sup>10</sup>.

Ultrasound is an essential tool for diagnosing peritoneal tuberculosis due to its accessibility, low cost, and ease of performance. Ultrasound is diagnosis-oriented and should be the first diagnostic investigation if peritoneal tuberculosis is suspected, especially in high-risk populations<sup>11,12</sup>.

In TB peritonitis, the most common ultrasound result is ascites (84.2%); ascites are easy to recognize and appear to echo if it is free fluid without debris. The presence of internal echoes is characteristic of exudative ascites. Peritoneal thickening (89.4%), omental thickening (73.6%), sonography is more sensitive than CT in detecting diffuse peritoneal thickening, especially in the presence of ascites, which is usually found in chronic inflammation. Mesentery involvement is joint and can be found in the early stages. It is characterized by wall thickening and is associated with increased echogenicity and multiple lymph nodes. Other abdominal lesions are as follows: Splenic nodules, ileocaecal involvement, and hepatic nodules; in advanced stages of the disease, lymph nodes can be visualized sonographically as hypoechoic areas with irregular borders due to ossified conglomerates of necrosis<sup>12</sup>.

Adenosine Deaminase Activity (ADA) has been studied in body fluids, including ascitic fluid, to diagnose TB peritonitis and has been shown to have high sensitivity and specificity<sup>12</sup>. Tuberculous peritonitis shows increased ascitic fluid adenosine deaminase (ADA) levels to more than 36 U/L. While serum ADA levels also increased to more than 54 U/L, the ratio of ascitic fluid ADA to serum ADA was above 0.98. The presence of all these findings indicates the presence of tuberculosis. ADA acts as a catalytic enzyme in the deamination of adenosine nucleosidase into inosine nucleosidase. ADA is found in lymphocytes, and stimulation of lymphocytes increases ADA activity in body fluids. This lymphocyte stimulation is caused by the tuberculosis bacteria, which activates the cellular immune response and, in turn, increases ADA levels. In studies using meta-analysis and systematic review, the results of data calculated from 20 studies, including studies that had a total of 2,291 patients, showed a pooled sensitivity of 0.90 (95% CI: 0.85 -0.94), a pooled specificity of 0.94 (95% CI: 0.92 - 0.95), and a DOR of 149 (95% CI: 86–255). The pooled analysis results suggested a clinically significant diagnostic value of ascitic fluid ADA for tuberculous peritonitis<sup>12,13</sup>. In patients with pleural tuberculosis, where ADA testing can also establish the diagnosis, ADA quantification is an enzyme produced by macrophages and activated T lymphocytes. This quantification is usually elevated, i.e., levels higher than 40 U/L. It is necessary to consider differential diagnosis with other pathologies such as rheumatoid arthritis, systemic lupus erythematosus, lymphoma, some adenocarcinomas, and empyema. The sensitivity of this method varies from 90 to 100%, and its specificity ranges from 89 to 100%. This method of diagnosis is more sensitive for pleural tuberculosis than pleural histopathologic examination and bacteriologic tests<sup>12,13</sup>.

Many doctors prefer a combination of clinical examination with other methods, such as laparoscopic biopsy and histopathological examination of peritoneal tissue (which can show caseation necrosis), acid-fast bacilli (AFB) staining, as well as radio-imaging techniques such as abdominal computed tomography (CT) scanning for diagnosis. However, all these methods are time-consuming, expensive, insensitive, invasive, or non-specific, making them ineffective in daily practice. While CT scans show non-specific findings, both cultures and smears fail to yield positive results. No more than 3% of cases show positive AFB smears, while only 20% show positive cultures<sup>13</sup>. Complications of

laparoscopy include bowel perforation, bleeding, infection, and death, but these are rare and seen in <3% of cases. Complications may be more common in fibroadhesive types<sup>13</sup>.

Treatment of tuberculous peritonitis is the same as for pulmonary tuberculosis, i.e., patients should receive at least six months of therapy. Patients who have not received treatment and are not resistant to oral antituberculosis drugs are treated with a first-line regimen consisting of an initial phase including Isoniazid 5 mg/kgBW, 15 mg/kgBW Etambutol, Rifampicin 10 mg/kgBW, and Pyrazinamide 25 mg/kgBW given daily for two months, then an advanced phase including a combination of Isoniazid (10 mg/kg BW) and Rifampicin (30mg/kg BW) 3x a week for four months. This therapy guideline gives good results after two months. Tuberculous peritonitis can be treated for 9-12 months (2HRZE/710RH). The treatment guideline can use a Fixed Drug Combination (FDC), which consists of a combination of 2 or 4 types of drugs in one tablet with a dose according to the patient's weight packaged in a package for one patient consisting of an intensive phase every day for 56 days RHZE (150/75/400/275), and an advanced stage of 7 to 10 months RH (150/150) 3x a week. This guidance may improve patient compliance<sup>13</sup>.

## **Conclusion**

Early diagnosis, immediate treatment, and follow-up of TB peritonitis are essential to reduce mortality from the disease. From this case, it can be concluded that clinical and supporting examinations (radiology) are needed to make the correct diagnosis, and body fluid analysis examination can help confirm the diagnosis, with the advantages of being non-invasive, easy to perform, cost-effective, and primarily available in health facilities.

## Bibliography

- Atzori, Sebastiana, Vidili, Gianpaolo, & Delitala, Giuseppe. (2012). The usefulness of ultrasound in the diagnosis of peritoneal tuberculosis. *The Journal of Infection in Developing Countries*, 6(12), 886–890. <https://doi.org/10.3855/jidc.2654>
- Chen, I. Hui, Torng, Pao Ling, Lee, Chia Yi, Lee, Kuang Han, Hsu, Heng Cheng, & Cheng, Wen Fang. (2021). Diagnosis of peritoneal tuberculosis from primary peritoneal cancer. *International Journal of Environmental Research and Public Health*, 18(19), 10407.
- Dahale, Amol Sonyabapu, Puri, Amarinder Singh, Sachdeva, Sanjeev, Agarwal, Anil K., Kumar, Ajay, Dalal, Ashok, & Saxena, Pritul D. (2021). Reappraisal of ascitic fluid adenosine deaminase's role in diagnosing peritoneal tuberculosis in cirrhosis. *The Korean Journal of Gastroenterology*, 78(3), 168–176.
- De Saram, Sophia, & Friedland, Jon S. (2019). Gastrointestinal and peritoneal tuberculosis. *Extrapulmonary Tuberculosis*, 25–42.
- Febrianto, Heru. (2019). *Asuhan Keperawatan Klien Tuberkulosis Paru Dengan Masalah Ke Tidak Efektifan Bersihan Jalan Nafas Di Ruang Teratai Rsud Bangil Pasuruan*. STIKes Insan Cendekia Medika Jombang.
- JUWITA, SARTIKA. (2013). *Analisis Distribusi Infeksi Mycobacterium Bovis Dengan Teknik Konvensional, Polymerase Chain Reaction (Pcr) Dan Geographical Information System (Gis) Pada Ternak Sapi Perah Di Kabupaten Enrekang*. Universitas Hasanuddin.
- Murlistyarini, Sinta, Prawitasari, Suci, & Setyowatie, Lita. (2018). *Intisari Ilmu Kesehatan Kulit dan Kelamin*. Universitas Brawijaya Press.
- Pramugaria, Elieza L., Nusi, Iswan Abbas, Setiawan, Poernomo Boedi, Purbayu, Herry, Sugihartono, Titong, Maimunah, Umami, Kholili, Ulfa, Widodo, Budi, Thamrin, Husin, & Vidyani, Amie. (2017). *Problematic Diagnosis of a Patient with Tuberculosis Peritonitis*.
- Sudirman, Taufik. (2018). Abses Psoas Tuberkulosis. *Medicinus*, 5(2).
- Vaid, Urvashi, & C. Kane, Gregory. (2017). Tuberculous peritonitis. *Tuberculosis and Nontuberculous Mycobacterial Infections*, 433–438.
- Wibowo, Gunawan Ari. (2023). Problema Diagnosis Peritoneal Tuberkulosis Pada Wanita 15 Tahun. *Jurnal Ilmiah Kesehatan Media Husada*, 12(2), 172–179.
- Zirta, Novie R., Uyainah, Anna, & PN, Evi Yuniastuti. (2015). Karakteristik klinis tuberkulosis ekstraparu pada pasien dengan dan tanpa infeksi human immunodeficiency virus di rumah sakit cipto mangunkusumo jakarta. *Indones J Chest Crit Emerg Med*, 2(2), 67–74.