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A Patient With Ruptured Bullae Caused By Intrapulmonary Complications Of Tuberculosis : A Case Report

Debora Ruth Octavia Simanjuntak[1*], Suzette Gloria I. Pantow[2], Sherly Darto[3] , Lilia Dewiyanti[4], Cipta Pramana[5]

[1]Department of Pediatrics, K.R.M.T. Wongsonegoro General Hospital Semarang, Faculty of Medicine Tarumanagara University, Jakarta, Indonesia; deb.oct4@gmail.com
[2]Department of Pediatrics, K.R.M.T. Wongsonegoro General Hospital Semarang, Faculty of Medicine Tarumanagara University, Jakarta, Indonesia; suzettepantow@gmail.com
[3]Department of Pediatrics, K.R.M.T. Wongsonegoro General Hospital Semarang, Faculty of Medicine Tarumanagara University, Jakarta, Indonesia; Sherlydarto15@gmail.com
[4]Department of Pediatrics, K.R.M.T. Wongsonegoro General Hospital Semarang, Faculty of Medicine Tarumanagara University, Jakarta, Indonesia; dewiyantililia@yahoo.com
[5] Department of Obstetrics and Gynecology, K.R.M.T. Wongsonegoro General Hospital Semarang, Faculty of Medicine Tarumanagara University, Jakarta, Indonesia; pramanacipta@yahoo.com

*(Korespondensi e-mail: deb.oct4@gmail.com)

ABSTRAK

Penyakit menular serius yang dikenal sebagai tuberkulosis (TB) disebabkan oleh basil Mycobacterium tuberculosis (MTB), yang biasanya menyerang paru-paru (TB paru) tetapi juga dapat menyerang tempat lain (TB ekstra paru). Jika tuberkulosis paru tidak diobati dengan benar, komplikasi dapat terjadi. Salah satu konsekuensi TB yang terkenal adalah pneumotoraks spontan. Sekitar 1,5% penderita TB paru dan pleura mengalami pneumotoraks sebagai komplikasi. SSP yang tidak mendapat intervensi segera dapat berkembang menjadi tension pneumotoraks. Kami dengan ini menyajikan kasus seorang wanita berusia 17 tahun dengan riwayat pengobatan TB yang tidak lengkap dengan komplikasi paru yang muncul sebagai pneumotoraks persisten.

Kata kunci: Bullae Pecah, Intrapulmoner, Tuberkulosis

Abstract

A serious infectious disease known as tuberculosis (TB) is caused by the bacillus Mycobacterium tuberculosis (MTB), which typically affects the lungs (pulmonary TB) but can also affect other places (extra pulmonary TB). If pulmonary tuberculosis is not appropriately treated, complications may result. One well-known consequence of TB is spontaneous pneumothorax. About 1.5% of people with pulmonary and pleural TB experience pneumothorax as a complication. SSP that does not receive prompt intervention may progress to tension pneumothorax. We hereby present a case of a 17-year-old female with history of uncompleted TB treatment with pulmonary complications presenting as persistent pneumothorax..

Keywords: *Ruptured Bullae, Intrapulmonary, Tuberculosis*

INTRODUCTION

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis. Although they mostly target the lungs (pulmonary TB), the TB bacilli can also affect other body organs (extra pulmonary TB). Indonesia has an incidence of 460,000 new cases each year, according to the World Health Organization's (WHO) 2014 Global Tuberculosis Report. But in

a related 2015 research, the number was increased to 1 million additional cases annually based on survey data collected since 2013 (Chaturvedi et al., 2016). The percentage of cases in Indonesia reaches 10 % of all cases in the world, which makes Indonesia that Indonesia the second-highest number of tuberculosis cases after India (Carolia & Mardhiyyah, 2016).

The short course chemotherapy, which uses first-line anti-TB medications, is foung to be the most effective anti-tuberculosis therapy. Patient mortality and morbidity are signifacntly affected by TB complications. Complications might occur in any area of the body as a result of presentation delays and poor treatment compliance (Heo et al., 2021). Haemoptysis, pleural effusions, aspergillosis, bronchiectasis, post-tuberculosis obstructive syndrome, and spontaneous pneumothorax are common consequences (Sebayang, 2020). Rare complications include tuberculous laryngitis, chronic cor pulmonale, pulmonary artery hypertension, tracheitis, ATT-induced hepatitis, acute respiratory failure linked with tuberculosis, paradoxical response, and tuberculous vasculitis (Desai, 2022).

In Indonesia, early treatment termination (drop-out) is the main cause of treatment failure for TB patients, which accounts for 50% of cases. Patients who have received treatment but have stopped receiving it for two months or more with a positive smear of acid fast bacilli are considered drop-outs (Andani & Savitri, 2022). Drug resistance, namely the formation of drug-resistant strains during chemotherapy, is a problem brought on by drop-out patients.4 These patients are also at risk of devastating complications due to the untreated TB and ongoing destruction of the lungs (Briones-Claudett et al., 2020). We hereby present a case of a 17-year-old female with history of uncompleted TB treatment with pulmonary complications (Haryati et al., 2022).

CASE ILLUSTRATION

A 17-year-old female patient was brought to the emergency department of Wongsonegoro District Hospital with complaints of cough, breathlessness, and productive cough. The patient was diagnosed with typhoid fever at first when she was admitted to Citarum PW Hospital. During treatments, the patient suddenly developed respiratory symptoms which alert the medical staffs at Citarum PW Hospital to refer the patient for adequate care. The patient was then referred to our hospital. Other complaints include dizziness and blurry vision. The patient had a history of chronic cough and hemoptysis a month before seeking medical help. Past medical history of the patient include uncomplete tuberculosis (TB) treatment (classified as a drop-out patient). She was given a TB diagnosis ago and has been off of her medications on her own for months since, as she acknowledged. She has commuting issues and the distance from the nearest primary health care facility to her Pesantren (a live-in religious educational facility) is quite far (Mirzai et al., 2020).

On presentation, the patient was alert (GCS E4, V5, M6), tachypneic (24 breaths per minute) and hypoxemic (pulse oximetry 92% room air), also tachycardic (117 beats per minute), with blood pressure of 111/68 mmHg and axillary temperature of 36.6 C. On physical examination, shifting to the right of the trachea was seen, chest movement was asymmetrical on inspection, breath sounds was absent on the left hemithorax on auscultation and hypersonor on percussion of the upper thorax. Examination of other systems were within normal limits. Initial chest x-ray showed tension hydropneumothorax differential diagnosed with bullae on the left lung. Emergency needle decompression was done in the emergency room with 14G Abbocath on the 7th intercostal space (ICS). The patient was then abdmitted to the pediatric intensive care unit (PICU) for further medical assistance. A chest tube (water sealed drainage, WSD) was inserted to the left hemithorax of the patient. Laboratory examination results during admission were as follow : Haemoglobin (Hb) 7.7 g/dL, Hematrocrite (Ht) 22.3 %, Leucocyte count 12800/ uL, thrombocyte count 583000/uL, sodium 120.0mmol/L, INR 1.05, aPTT 27.2

s, PT 11.7 s. The patient was found HIV-negative. Because the patient was anemic, blood transfusions were given. Primary medications include levofloxacin 750 mg, methylprednisolone 62.5 mg, ceftriaxone 1 gr, and interval nebulizations with ipratropium bromide and salbutamol. Other medications were given as indicated. CT-scan of the patient after stabilization and admission was done to further evaluate the conditions of the lungs. CT-scan revealed a destroyed left lung with extensive bronchiectasis and also left pneumothorax with multiple bronchopleural fistula. After chest tube insertion for 5 days, undulation was still seen in from the tube with productive production of drainage. Due to this finding, the patient was diagnosed with persistent pneumothorax with left destroyed lung (Figure 3). On the fifth day of admission, the patient developed severe breathlessness and altered consciousness (Suri et al., 2019). She was then intubated and placed on mechanic ventilation.

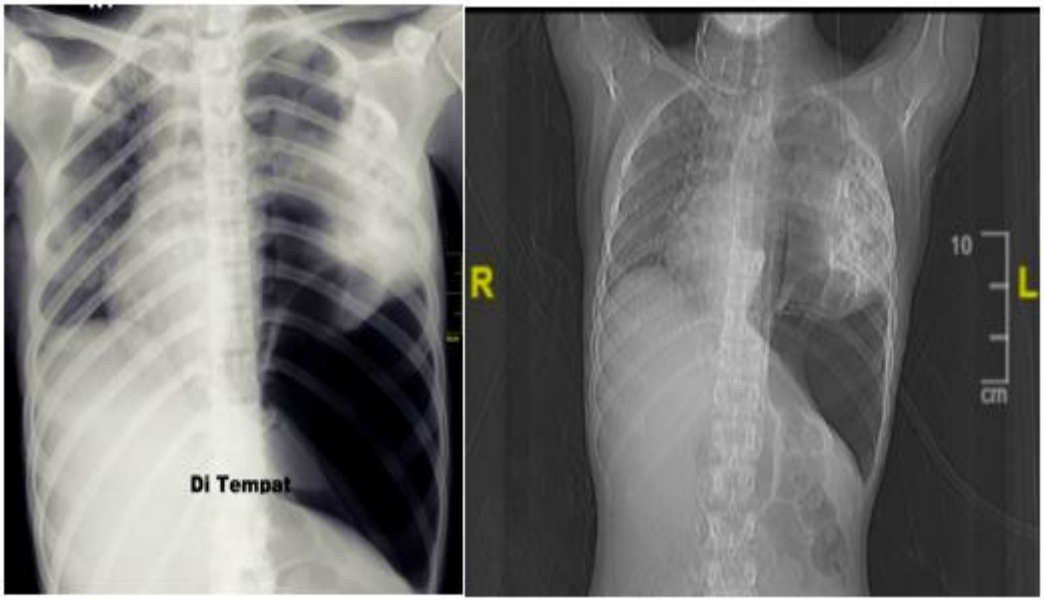

Figure 1. Thorax x-ray of the patient, before (left) and after (right) chest tube insertion

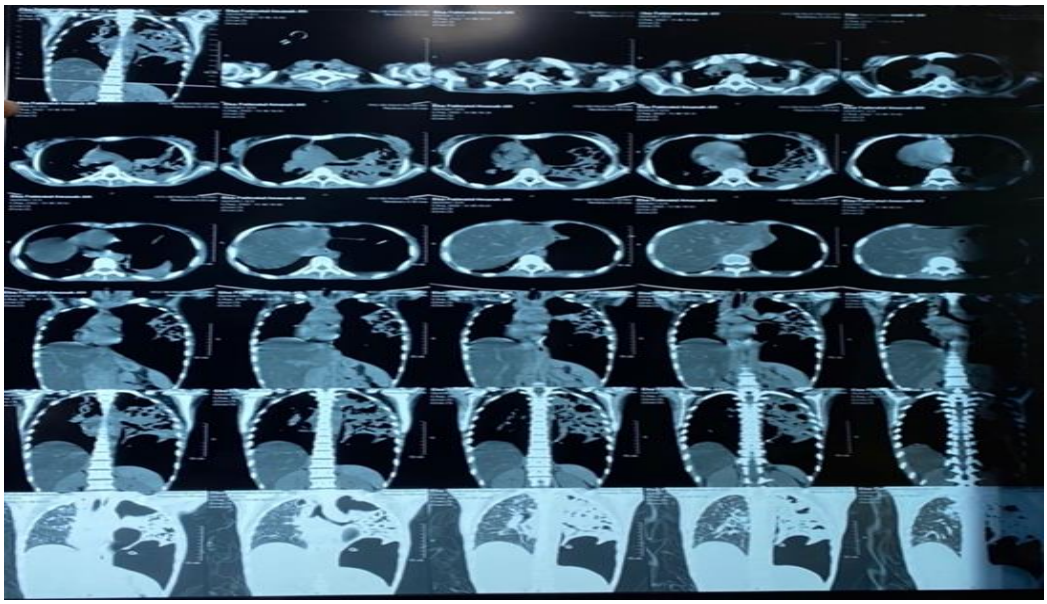

Figure 2. Thorax CT-scan of the patient

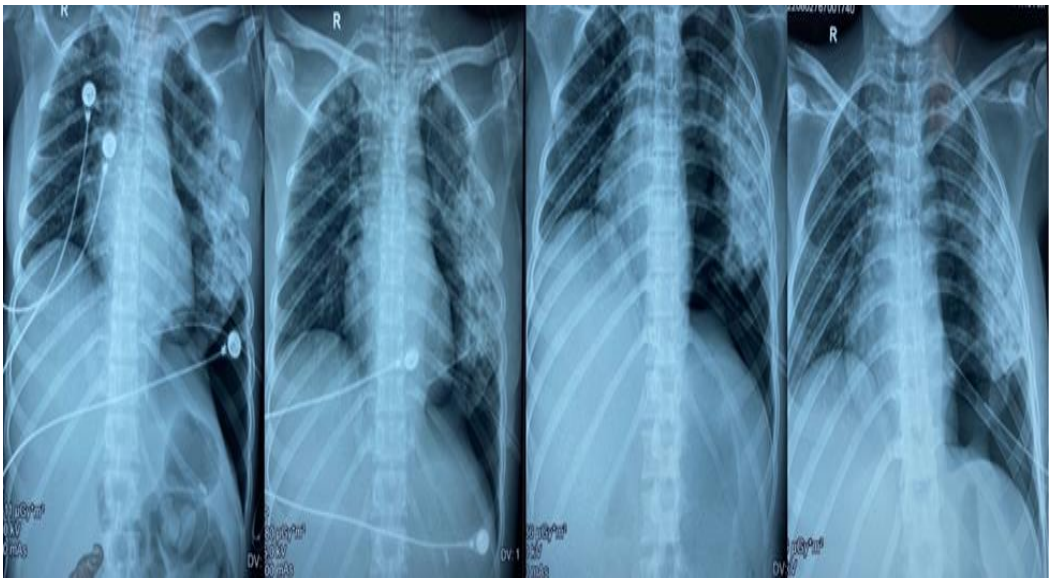

Figure 3. Serial thorax X-ray of the patient on the 2nd, 5th, 8th and 11th day of admission showing persistent hydropneumothorax with infiltrates and consolidations on the left lung

DISCUSSION

A serious infectious disease known as tuberculosis (TB) is caused by the bacillus Mycobacterium tuberculosis (MTB), which typically affects the lungs (pulmonary TB) but can also affect other places (extra pulmonary TB) (Alhakeem et al., 2020). If pulmonary tuberculosis is not appropriately treated, complications may result. Pleuritis, pleural effusion, empyema, and tuberculous laryngitis are a few early problems that can develop. Massive hemoptysis, lobe collapse brought on by duct obstruction, bronchiectasis, spontaneous pneumothorax, fibrothorax, and hydropneumothorax are examples of advanced-stage complications that may occur (Chen et al., 2021). In this patient, tension pneumothorax on the left hemithorax caused the patient to seek medical help. Due to the history of incomplete TB treatment, this could be the cause of her current disease.

One well-known consequence of TB is spontaneous pneumothorax. Air in the pleural space is what is defined as a pneumothorax (Tiberi et al., 2019). Pneumothorax may be primary or secondary. Secondary pneumothorax occurs in persons with significant underlying pulmonary disease. Pneumothorax complicating pulmonary tuberculosis can occur at any age, however patients under 30 years old experience it most frequently (Thakkar et al., 2021).

About 1.5% of people with pulmonary and pleural TB experience pneumothorax as a complication. 6,8,9 Secondary spontaneous pneumothorax (SSP) is most frequently caused by pulmonary tuberculosis in nations where TB is a widespread issue (Nitsch et al., 2023). In people with underlying lung disease, a pneumothorax has far more severe symptoms, and treatment may be more complex. Pneumothorax secondary to TB, in contrast to the benign nature of primary spontaneous pneumothorax, can be life-threatening due to the patient's underlying respiratory illness and the impaired cardiopulmonary reserve. Pneumothorax secondary to TB usually occurs after extensive TB involvement of the lung. The most frequent causes of pneumothorax in TB are the rupture of a cavity and the subpleural focus (Li et al., 2022). Bronchopleural fistulization (a communication between a main stem, lobar, or segmental bronchus and the pleural space) 11 and empyema with severe cavitary formations

or occasionally miliary TB also can cause SSP. The TB organism invades the pleura and causes liquifactive necrosis, then pleural rupture (Xiang & Wu, 2020).

Other causes are rupture of a bulla which has developed adjacent to the lung fibrosis. 10 Bullae are defined as air spaces in the lungs, measuring more than 1 cm in diameter when distended (Petreanu et al., 2023). In many cases, it can be asymptomatic until it ruptures and cause SSP. The disease process in this patient was very much not evaluated and not treated adequately. Many publications only consider SSP in cases of active TB, but due to the concomitant lung destruction that would be the cause of SSP, as was obvious in our patient, SSP can develop at any stage of the disease, including at the stage of sequela (Denning et al., 2018). We suspect a ruptured bullae as the cause of SSP in this patient, but we cannot fully rule out the possibility of other causes such as rupture of a cavity or a subpleural focus.

The most common symptoms associated with SSP are chest pain and dyspnea. The clinical signs are dependent on the degree of collapse of the underlying lung. Another typical symptom is a cough. Untreated pneumothorax can progress into tension pneumothorax, which is charactized by progressive and severe chest discomfort and dyspnea that is usually acute (Quincho-Lopez et al., 2020). Studies showed that chest pain, cough, expectoration and fever were more frequent in patients with active TB than in non-TB patients with pneumothorax (Wu et al., 2020). Fever and constitutional symptoms such as weight loss and anorexia were also commonly seen probably due to TB being the major etiology. The patient in our case appeared underweight, with chest pain and productive cough as her main symptoms.

On examination, the affected side showed decreased chest movements and vocal fremitus. Trachea deviates away from the affected side. Additionally, the affected side may have decreased or no breath sounds and be hypersonor to percussion. There may be hypoxia, tachycardia, hypotension, and jugular venous distension (JVD). Although hypotension and JVD weren't present in our patient, other signs were positive on the left hemithorax and indicated pneumothorax. On the affected side of the chest radiograph, there is a region of hypertranslucence without bronchovascular signs, and the lung is compressed toward the hilum. SSP that does not receive prompt intervention may progress to tension pneumothorax. 8 The mediastinum will appear shifted to the opposing side of the pneumothorax. SSP due to TB that develops into tension pneumothorax can be fatal (Jamil & Kasi, 2022). Significant risk factors for the development of tension pneumothorax in pulmonary TB are co-infection, advanced TB (i.e., fibrotic adhesion and the size of bullae), and smoking.

Pneumothorax management includes both a conservative strategy and active management. If the patient has few symptoms (is clinically stable) and the pneumothorax is relatively small—less than 20% of the hemithoracic volume—conservative treatment is advised. 10,16 Active case management is necessary for patients with tension pneumothorax who are clinically stable but have a big pneumothorax or who are clinically unstable (Kapoor et al., 2018). Patients with underlying lung disease usually appear clinically unstable, for which aggressive management is also recommended. Simple aspiration or intercostal chest tube drainage constitute active management. Patients with SSP have a lower success rate with aspiration. In any other case, WSD chest tube drainage enables gradual lung re-expansion. As seen in our patient, the treatment of tension pneumothorax definitely necessitates urgent needle decompression followed by WSD chest tube placement. The use of a 14-G needle in emergency treatment in tension pneumothorax is supported by the Advanced Trauma Life Support (ATLS) guideline (Mizushima et al., 2022).

Hydropneumothorax is the abnormal presence of air and fluid in the pleural space. Though non-traumatic hydropneumothorax is rare, tuberculosis (TB) was found to be the etiolog in 80.7% of the cases. Most patients presented with symptoms of acute respiratory compromise that is breathlessness due to ventilation perfusion mismatch and cough due to

pleural involvemen (Zhang et al., 2019)t. Hydropneumothorax is frequently associated with pneumothorax secondary to tuberculosis with untreated empyema, when it is called as pyopneumothorax. 10 Light's criteria with the goal to identify all exudates correctly. On applying Light's criteria, tuberculosis patients have exudative pleural effusion. ADA test may also be performed to confirm tuberculosis. ADA levels has high accuracy in the diagnosis of the pleural TB and should be used as a routine test in its investigation (Dreuning et al., 2019).

Chest tube insertion with antimicrobial chemotherapy is the management of choice. Chest tube is usually needed for longer duration due to the underlying TB and most of these patients had bronchopleural fistula as evident by prolonged air leak.Multiple loculations and adhesions might also contributes for prolonged chest tube insertion. This could be the cause of our patient's extended chest tube insertion and the persistent pneumothorax that was shown to have productive drainage (Grech & Shoukry, 2022).

By definition, a persistent pneumothorax constitutes ongoing bubbling of air from an in situ chest drain, 48 h after its insertion. This definition corresponds to our case. Persistent pneumothorax may be chest tube related (kinks or malposition), lung parenchymal disease, bronchopleural fistula. Chest tube related causes were ruled out from the follow-up imaging we performed. Lung parenchymal disease and bronchopleural fistula may cause this to happen. There are differences in the management guidelines proposed by the American College of Chest Physicians (ACCP) and British Thoracic Society (BTS). The BTS recommends a thoracic surgery consult if the air leaks persist beyond 2 days or if the lung does not re-expand, while the ACCP recommends intervention for air leaks persisting beyond 4 days in primary spontaneous pneumothorax and over 5 days in secondary pneumothorax. Surgical reseaction such as blebectomy with pleurectomy is indicated in patients with a prolonged air leak (more than 72 hours). More frequently utilized in recent years in the treatment of pneumothorax is less invasive video-assisted thoracic surgery (VATS) (Shehata et al., 2018).

Radiological signs of destroyed lung are very frequent in patients with SSP, with a study found a percentage as high as 53.2%. This is also seen in our patient. Chronic damage can range in severity from hardly noticeable scarring to severe bronchiectasis and remarkable fibrocavitary destruction. Chronic restrictive or obstructive deterioration in pulmonary function may result from TB illness. When parenchymal damage is advanced, a lung lobe or the entire lung may be destroyed. 3 It is known that TB can cause unilateral lung destruction. In their analysis of individuals with unilateral lung loss, Rajasekaran et al. discovered that pulmonary tuberculosis was the root cause in 83.3% of cases. 8 Reduced lung volume, cavities, bronchiectasis and fibrosis are the predominant findings in destroyed lungs. 10

SSP as a potential intrahospital fatal evolution risk. Our patient was transferred to the ICU for management of respiratory distress and pneumothorax drainage. Poor general health, severe respiratory impairment, patients' ages, and HIV infection are all risk factors. The most frequent causes of ICU admission are acute respiratory failure (> 90%), septic shock (between 20 - 34%), and multiorgan failure (between 34 - 44%) in these patients. Given the disease's frequent complexity and poor prognosis, managing tuberculosis in the ICU is challenging. According to WHO recommendations, patients who have drug-susceptible strains of M. tuberculosis should get standard quadruple therapy, which consists of rifampin, isoniazid, ethambutol, and pyrazinamide (Shalaby et al., 2018). Additionally, drug resistance must be suspected, especially in our situation as the patient was a drop-out. It is also important to take into account any co-infections. Administration of antituberculosis medication in the ICU is mostly dependent on intestinal absorption, which can be slowed down or altered by gastroparesis, intestinal paralysis, enteral feeding, edema from hypoalbuminemia, and gut flora changes brought on by critical illness. Additionally, critical illness can affect the pharmacokinetics of antituberculosis medications. Because first-line medications are rarely

offered in an intravenous formulation, second-line medications like fluoroquinolones and aminoglycosides are frequently used. In our patient, levofloxacin was given as an alternative. In our patient, methylprednisolone was given. Corticosteroids are routinely given concurrently in the ICU (Petreanu et al., 2023). In a recent meta-analysis, corticosteroids were found to have a greater impact on individuals with a severe form of the disease, reducing mortality in all kinds of tuberculosis. 2-

The patient's history of incomplete TB treatment may be the primary cause of all these agonizing conditions. Most people do not have a thorough understanding of tuberculosis treatments and problems, especially when it comes to how crucial it is to finish treatment in order to avoid unintended complications (Wu et al., 2020). Studies indicated that inability to obtain health services was a contributing factor to the issue. Other causes include unpleasant experience of the drug's adverse effects of the drugs and false belief in traditional medicines. The key to treating tuberculosis is figuring out how to inspire patients to follow the prescribed course of therapy in accordance with the established regiment. To do so, it is necessary to improve certain pulmonary TB control programs in order to support pulmonary TB patients (Jamil & Kasi, 2022).

CONCLUSION

The intrapulmonary complications of TB seen in our patients were persistent pneumothorax with destroyed lung. Complications of pulmonary TB is devastating and has poor prognostic outcomes. Effort should be exerted into educating patients regarding the importance of TB treatment completement in order to avoid unwanted complications.

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