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Case Report: Drug Related Supraventricular Tachycardia in A 58-Year-Old Male with Lung Tuberculosis

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Abstract *— Background:* Arrhythmia in tuberculosis is a rare and life-threatening condition. It can occur as an adverse effect of drugs included in the regiment. *Case presentation:* This study reported a case of arrhythmia in a 58-year-old male who experienced sudden onset of palpitations, several days after his tuberculosis regiment was changed from the fixed drug combination to a non-hepatotoxic regiment. Based on the clinical presentation, electrocardiogram, laboratory finding, echocardiogram, and analysis of the adverse effects of all given drugs, a drug was suspected as the cause of his arrhythmia. The patient survived after undergoing acute phase management for arrhythmia and discontinuation of the culprit drug. *Discussion:* One of the serious adverse effects of fluoroquinolone is prolonged QT intervals that could lead to deadly arrhythmia. Therefore, close monitoring of the clinical, vital, and physical is crucial, especially during the first month of therapy. *Conclusion:* Conscientious consideration should be made before starting therapy for tuberculosis, and acute phase management is pivotal in termination of arrhythmia.

Keywords — Supraventricular Tachycardia; Arrhythmia; Lung Tuberculosis; Adverse Effect

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INTRODUCTION

Tuberculosis (TB) is an infectious disease caused by the bacterium Mycobacterium tuberculosis, known as Acid-Fast Bacillus (AFB). The main symptom in pulmonary TB patients is a productive cough lasting for two weeks or more [1]. TB ranks among the top 10 causes of death worldwide. According to the World Health Organization, in 2017, 10 million people suffered from TB, and 1.6 million people died from the disease. The incidence of TB in Indonesia in 2017 was 319 per 100,000 population, with a mortality rate of 40 per 100,000 population. A total of 54 million people were cured of TB with effective diagnosis and therapy from 2000 to 2017 [2]. TB can be cured with treatment using Anti-Tuberculosis Drugs (ATDs). The use of multiple medications in combination can lead to drug interactions [1]. One of the serious adverse effects of TB treatment is the prolongation of the QT interval, which can lead to supraventricular tachycardia (SVT) [3]. This case review will discuss a male TB patient undergoing TB treatment who experienced supraventricular tachycardia, suspected to be due to side effects or drug interactions.

CASE PRESENTATION

A 58-year-old man presented to the emergency department with sudden onset of palpitations, dyspnea, sweating, fatigue and nausea. One month ago, the patient had a history of fixed drug combination (FDC) for his lung tuberculosis, consisted of 150 mg of rifampicin, 75 mg of isoniazide, 400 mg of pyrazinamide, and 275 mg of ethambutol. Sixteen days after started his medication, he experienced jaundice, severe nausea and fatigue. The laboratory confirmed that he had drug induced hepatitis. At that time, to preserved his liver function, the physician in the previous community health center decided to change his medication to 500 mg intramuscular streptomycin, 250 mg ethambutol, and 750 mg levofloxacin. The patient had taken the new regimen for

seven days before the palpitation occurred. Patient had no history of heart disease, alcohol, smoking or exercise, and there was no history of previous medication.

Physical examination revealed weak appearance, Glasgow Coma Scale (GCS) of 15, blood pressure of 90/70 mmHg, rapid and weak heart rate (190x/min), respiratory rate of 24x/min, afebrile, oxygen saturation of 98%. Coarse ronchi was found on left hemithorax, no abnormalities of the heart sound. A 12 - lead electrocardiogram (ECG) was obtained demonstrating supraventricular tachycardia (SVT) as showed on **Fig. 1**.

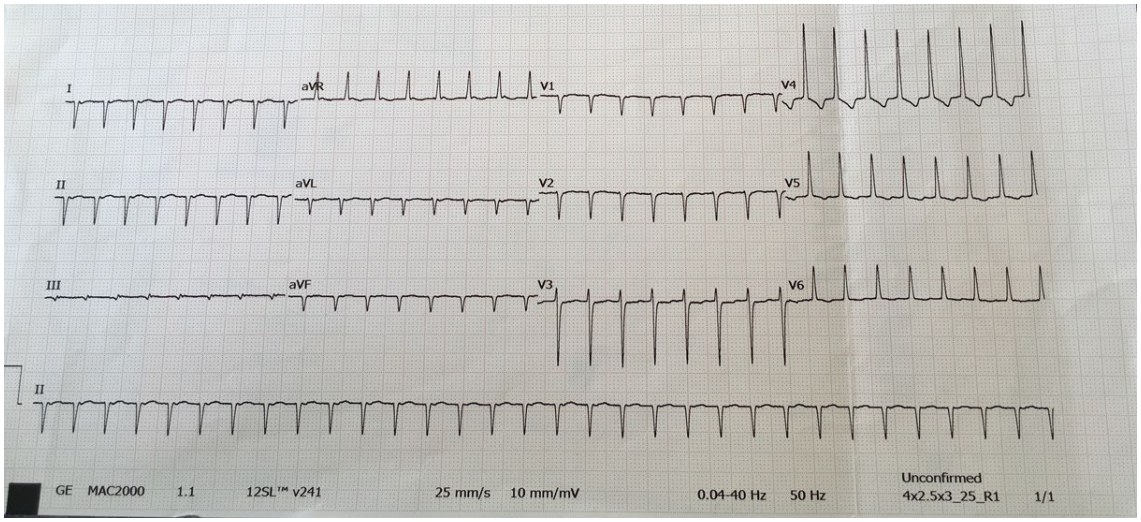

Fig. 1. ECG result of the patient showed narrow complex supraventricular tachycardia 189 times/minute, prolonged QTc 498 milliseconds.

Laboratory result show hemoglobin (10.5 g/dL), leucocyte (6,800/mm3), platelets (459.000/mm3), AST (22 U/L), ALT (26 U/L), normal albumin serum (3.0 g/dL), normal BUN (13 mg/dL) and creatinine (0.86 mg/dL). Serum electrolyte was normal, natrium (134 mmol/L), kalium (4.1 mmol/L), chloride (101 mmol/L), calcium (9.1 mg/dL), and magnesium (2.1 mg/dL). HIV and HbsAg were non reactive. Chest radiography showed lung tuberculosis, upper left schwarte and organized pleural effusion. The patient was diagnosed as lung tuberculosis and supraventricular tachycardia.

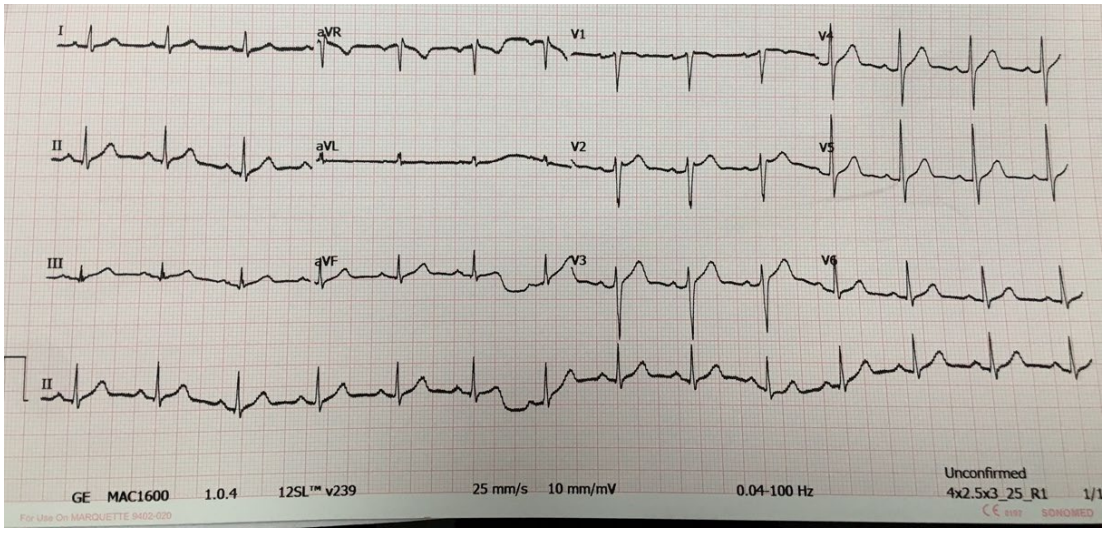

Fig. 2. Post diltiazem administration showed the heart rhythm returning to sinus 82 times/minute

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The initial therapy was performed to the patient, nasal oxygen of 3 litres per minute, intravenous fluid of NaCl 0.9%, and valsava maneuver. Slow intravenous bolus of 0.25 mg/kgBB diltiazem in 10 cc aquadest was administered following the initial therapy, and to alleviate the symptom of nausea, 8 mg of ondancentron and sucralfate syrup were given. The post diltiazem ECG showed the cardiac rhythm had back to normal as showed on **Fig. 2**.

The following day, echocardiography was performed and we found no abnormalities of all four chambers of heart, valves, systolic and diastolic function. GeneXpert was also being tested and came out negative (resistance to rifampicin not detected). The patient was treated with 2,5 mg oral bisoprolol twice a day, low desensitization dose of rifampicin was started at 150 mg, 100 mg of isoniazid, and 750 mg of ethambutol. Patient was monitored closely for clinical complaints, vital and ECG.

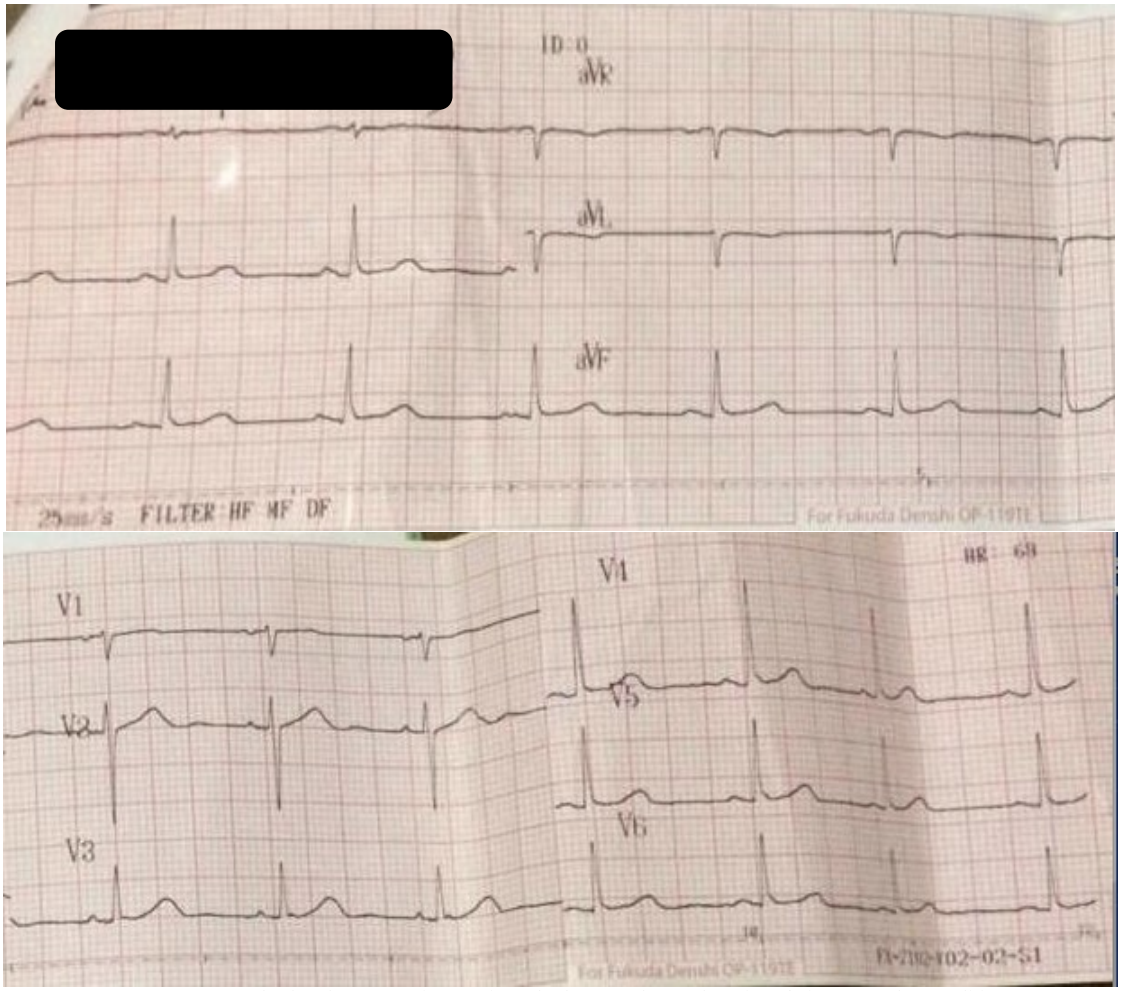

Fig. 3. One month after the new regiment, sinus rhythm was detected 75 times/minute, no abnormalities.

On the third day of treatment, the patient had no complaints, vital and physical examinations were normal. ECG result was normal (sinus rhyhtm, 88 times/min, normal axis, no abnormalities on p-wave, QRS-wave and ST-segment), laboratory result was also normal, AST (21 U/L) and ALT (18 U/L). The patient was discharged from the hospital and the therapy for lung tuberculosis was continued as follows : 450 mg of rifampicin, 100 mg of isoniazid, and 750 mg of ethambutol.

One month after, the patient had no symptoms, normal vital sign and physical examination, laboratory finding of liver function was normal, AST (24 U/L), ALT (29 U/L). ECG examination was performed and found to be normal, as showed on **Fig. 3**. No adverse effects detected regarding the new regiment given. The regiment was continued in the community health center.

Table 1. Patient's ECG interpretation

Assessment	First Admitted	Post Diltiazem Administration	One month Follow-up
Heart rate (times/minute)	189	82	75
Rhythm	Regular	Regular	Regular
Axis	Extreme axis deviation	Normal	Normal
P-wave	Negative	Positive (sinus rhythm)	Positive (sinus rhythm)
PR interval, segment	Could not be measured	Normal (160 ms)	Normal (120 ms)
QRS complex	Narrow (<120 ms)	Narrow (<120 ms)	Narrow (<120 ms)
ST segment	ST depression of lead II, aVR, V4, V5, V6	Normal (isoelectric)	Normal (isoelectric)
QTc (milliseconds/ms)	498	374	358
Conslusion	Supraventricular tachycardia	Normal ECG	Normal ECG

DISCUSSION

The incidence of arrhythmias in TB patients is very rare. The first reported case linking arrhythmia and TB was by Schnitzer in 1947 in a patient who suffered sudden cardiac death due to arrhythmia. Post-mortem examination revealed TB myocarditis and miliary TB[4]. By 2009, there were seven reported cases of arrhythmia in TB myocarditis[5]. TB myocarditis can cause superior vena cava obstruction, right ventricular obstruction, ventricular tachycardia, heart block, valvular dysfunction, and heart failure [4,6-9].

Supraventricular tachycardia (SVT) is a term used to describe tachycardia (atrial and/or ventricular) with a rate of more than 100 beats per minute at rest, involving tissue originating from His bundle or above [10].

QT interval prolongation could lead to deadly arrhythmia. It is classified into two categories: (1) Long QT syndrome (LQTS) and (2) acquired LQTS (usually related to medication) [11]. Data from the Food and Drug Administration Adverse Event Reporting System (FDA AERS) collected over five years from 1,743,234 reports of drug side effects revealed 374 cases of torsades de pointes (TdP), with 230 cases attributed to antibiotics. The most common were levofloxacin (55 cases), moxifloxacin (37 cases), ciprofloxacin (35 cases), clarithromycin (22 cases), and azithromycin (16 cases) [12]. Levofloxacin has been prescribed over 200 million times, with 15 cases of ventricular arrhythmia or cardiac arrest per 10 million prescriptions. Data from R.W. Johnson Pharmaceutical Research Institute and Ortho-McNeil Pharmaceuticals in the UK also reveals that there was an incidence of TdP at a frequency of 1 in every 1 million prescriptions. In vivo analysis to marmot isolates, levofloxacin prolonged the QT interval by 0.6-3.3% (milliseconds) [13].

The patient received a fixed-dose combination (FDC) therapy containing four drugs: 150 mg of rifampicin (R), 75 mg of isoniazid (H), 400 mg of pyrazinamide (Z), and 275 mg of ethambutol (E). The FDC tablets were taken three times daily. On day 16, the patient reported nausea and weakness, leading to hospitalization and laboratory tests showing an increase in AST < 3 times the upper limit of normal and ALT 3 times the upper limit of normal, resulting in a diagnosis of Drug-Induced Hepatitis.

Drug-Induced Hepatitis (DIH) typically occurs around 20 days after therapy and lasts for approximately 14 days. The most common symptoms are nausea, vomiting, and anorexia [14]. Management of DIH based on the national guideline of tuberculosis control by the Ministry of Health of the Republic of Indonesia includes [15] :

- If liver dysfunction is suspected to be caused by ATD, all hepatotoxic ATDs must be discontinued. Treatment may include streptomycin (S) and ethambutol (E) while waiting for liver function to improve. If liver function returns to normal or near-normal, rifampicin (R) can be reintroduced gradually, followed by isoniazid (I).

- In cases of severe TB where discontinuing treatment would be harmful, a non-hepatotoxic regimen may be administered, including S, E, and one of the fluoroquinolone ATDs.
- Treatment with ATDs should be halted until liver function tests normalize and symptoms (nausea, abdominal pain, etc.) resolve before resuming treatment.
- If liver function tests cannot be performed, it is recommended to wait two weeks after jaundice or nausea and weakness, and until liver palpation is no longer palpable, before restarting treatment.
- If symptoms and signs do not resolve and there is severe liver dysfunction, a non-hepatotoxic regimen consisting of S, E, and one fluoroquinolone can be given (or continued) for 18-24 months.
- Once liver function issues are resolved, the original ATD regimen can be resumed one at a time. If symptoms of liver dysfunction reappear or liver function tests return to abnormal, the last added ATD should be discontinued. Recommendations for resuming treatment with Rifampicin suggest starting it first, and after 3-7 days, Isoniazid can be added. In patients with a history of jaundice who can tolerate H and R, it is strongly advised to avoid Pyrazinamide.
- Substitute regimens depend on which ATD caused the liver dysfunction. If R was the cause, a regimen of 2HES/10HE is recommended. If H was the cause, a regimen of 6-9 RZE can be provided. If Z is discontinued before the patient completes the initial treatment phase, the total duration of treatment with H and R can extend to 9 months. If neither H nor R can be given, a non-hepatotoxic ATD regimen of S, E, and one fluoroquinolone should be continued for 18-24 months.
- If liver dysfunction and jaundice occur during the initial treatment phase with H, R, Z, and E (Category 1 regimen), once liver function is restored, the same regimen can be resumed but Z should be replaced with S to complete the initial two-month phase, followed by H and R for an additional six-month continuation phase.
- If liver dysfunction and jaundice occur during the continuation phase (Category 1 regimen), once liver function is restored, H and R should be resumed for a complete four-month continuation phase.

According to the national guideline, after being diagnosed with Drug-Induced Hepatitis, the patient was given intramuscular injections of 500 mg of streptomycin, 250 mg of ethambutol, and 750 mg of levofloxacin.

Rifampicin is administered for all forms of tuberculosis at a dose of 10 mg/kg/day (not exceeding 600 mg/day), once daily by mouth in adults. Taking it with food reduces absorption by 30% [16]. This drug is bactericidal against Mycobacterium tuberculosis by inhibiting the activity of DNA-dependent RNA polymerase, preventing the bacteria from synthesizing RNA and proteins, leading to cell death. The biological half-life of rifampicin at a dose of 450 mg is 3.47 hours, eliminated in urine and feces after 96 hours [17].

Drug interactions may occur if rifampicin is given together with isoniazid, pyrazinamide, and streptomycin. Rifampicin increases the hepatotoxicity of isoniazid. Combination with pyrazinamide is also synergistically increases toxicity to the liver. Rifampicin used with streptomycin reduces the effects of streptomycin because rifampicin induces the efflux transporter P-glycoprotein (MDR1), increasing the elimination of streptomycin. When used with fluoroquinolone antibiotics, rifampicin accelerates the metabolism of those drugs [18].

Major side effects of rifampicin, experienced by 20-30% of patients, include flu - like symptoms, abdominal pain, respiratory symptoms, shock, kidney failure, purpura, and thrombocytopenia. In the first two months of treatment, temporary disturbances in liver function (increased serum transaminases) are common. Rifampicin can cause red discoloration in urine, sweat, tears, and saliva. This red discoloration results from the drug's metabolism and is not harmful [16]. Cardiovascular side

effects include hypotension, sinus tachycardia, ventricular arrhythmia, seizures, and cardiac arrest, which have been reported in cases of fatal rifampicin overdose [19].

Isoniazid is administered at a dose of 5 mg/kg body weight, with a maximum of 300 mg orally. It acts as a bactericidal agent by inhibiting the biosynthesis of mycolic acid, the largest component of the cell wall of Mycobacterium tuberculosis [20]. The biological half-life of isoniazid in fast acetylators is less than 1 hour, and in slow acetylators, it is 3 hours. A large amount is excreted in urine within 24 hours [21]. Drug interactions can occur with pyrazinamide, increasing hepatotoxicity [20].

Common side effects involve the nervous system and liver [22]. Signs of toxicity in the peripheral nervous system include tingling, burning sensations in the feet, and muscle pain. These effects can be reduced by administering pyridoxine at a dose of 100 mg per day or with vitamin B complex. In such cases, treatment can continue. Hepatitis can occur in approximately 0.5% of patients [16]. Increases in serum transaminase levels occur in 10-20% of patients, along with bilirubinemia, bilirubinuria, jaundice, and severe hepatitis [22].

In the vascular system, a rare side effect is vasculitis related to hypersensitivity reactions [23]. A study found that the use of isoniazid together with rifampicin increases the incidence of hepatitis by 2.7%. The older the user, the higher the risk of drug-induced hepatitis. This risk increases with alcohol consumption, chronic liver disease, and in postpartum women. Warning symptoms related to hepatotoxic effect include anorexia, nausea, vomiting, dark-colored urine, jaundice, red spots on the body, weakness, and fever lasting more than three days accompanied by discomfort in the upper right abdomen [22].

Pyrazinamide is given at a dose of 25 mg/kg body weight orally [16]. This drug works as a bactericidal [24]. Its active metabolite, pyrazinoic acid, inhibits the membrane transport function of Mycobacterium tuberculosis [25]. Pyrazinamide can also eliminate persistent bacilli that multiply sporadically and have the potential for relapse [26]. The biological half-life of pyrazinamide is 10 hours, and it is excreted in large amounts through urine [21].

Interactions have been found when the drug is administered together with isoniazid and rifampicin. Pyrazinamide increases liver toxicity when used in conjunction with isoniazid and rifampicin [20]. Severe liver injury can occur with the use of pyrazinamide alongside rifampicin for 2 months, necessitating close monitoring [27]. The main side effects of pyrazinamide include drug-induced hepatitis, joint pain, and occasionally gout attacks, as pyrazinamide metabolites inhibit uric acid secretion in the renal tubules [16]. In the vascular system, pyrazinamide affects blood coagulation mechanisms and the integrity of blood [28].

Ethambutol is given at a dose of 15 mg/kg body weight orally [16]. This drug works bacteriostatically by inhibiting the synthesis of bacterial metabolites, which disrupts cell metabolism, cell multiplication, and leads to cell death [29]. The biological half-life is 3-4 hours, and it is excreted in urine (50-80%) and feces (20%).

Interactions with isoniazid increase the risk of nerve damage, leading to muscle weakness, pain, burning sensations, numbness, or tingling in the hands or feet [30]. Side effects of ethambutol usually appear at a dose of 15 mg/kg body weight and include retrobulbar neuritis, which can be reversible if detected early and treatment is stopped [31]. Vision disturbances include reduced sharpness and red-green color blindness [16]. Cardiovascular side effects may include myocarditis and pericarditis [26].

Streptomycin is given 15 mg/kgBB intramuscularly. This drug binds the Mycobacterium tuberculosis 30S ribosomal unit irreversibly, thus inhibits the protein synthesis of the cell membrane [32]. The interaction with rifampicin will cause the increase of elimination through glycoprotein-P transporter, resulting in lower drug concentration and effect [18]. The high dose accumulation along with other risk factors such as old age, and long duration of therapy impacts the vestibulocochlear nerve, causing irreversible hearing and vestibular impairment (vertigo, ataxia, and nystagmus). In the renal tubules, it causes oliguria, urine cast, proteinuria, decrease of creatinine clearance and increase of ureum and serum creatinine [33]. It can also cause neuromuscular inhibition lead to respiratory failure, anaphylaxis, syncope and cardiac arrest [34]. One scientific research found

that when the drug is given above the therapeutic dose, it will decrease the stretching effect of the myocardia during ventricular tachycardia by inhibit stretch-sensitive channel [35].

Levofloxacin is one of the fluoroquinolone drug administered for tuberculosis therapy. It is given orally between 750 - 1000 mg/day [14]. It has bactericidal effect by inhibit topoisomerase IV and DNA gyrase, enzymes for replication, transcription, repair, and recombination of Mycobacterium tuberculosis [36]. Interaction with rifampicin will enhance the metabolism of levoflaxacin. Common side effects including skin reaction and allergy, eryhtema, pruritus, and rash, rarely urticaria, angioedema, anaphylactic and vasculitis. arthropathy, tendinopathy, achilles rupture, and cartilage erosion develop at long-term and high-dose use. On cardiovascular system, the prolongation of QT interval often occurs, resulting in ventricular tachycardia and polymorphic tachycardia (torsades de pointes) [37].

To assess which drug is responsible for any side effect, the Naranjo Adverse Drug Reaction (ADR) Probability Scale is usually used to help standardize assessment of causality. This scale contains ten questions [38], such as :

1. Are there previous conclusive reports on this reaction ? Yes (+1)
2. Did an adverse event appear after the suspected drug was given ? Yes (+2)
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was given ? Yes (+1)
4. Did the adverse reaction appear when the drug was readministered ? Not known or not done
5. Are there alternative causes that could have caused the reaction ? No (+2)
6. Did the reaction reappear when a placebo was given ? Not known or not done
7. Was the drug detected in any body fluid in toxic concentrations ? Not known or not done
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased ? Not known or not done
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure ? Not known or not done
10. Was the adverse event confirmed by any objective evidence ? Yes (+1)

The algorithm score is divided into doubtful (≤ 0), possible (1 to 4), probable (5 to 8), and definite (≥9). As for this patient, there are previous conclusive reports from various research on how levofloxacin, or fluoroquinolone, can cause arrhythmia. The arrhythmia appeared after levofloxacin was given and improved when it was discontinued. We did not readminister levofloxacin since the adverse event was life threatening. Heart disease and abnormal levels of electrolytes are the common cause of arrhythmia. Other causes, such as alcohol or recreational drugs, tobacco and exercise. Considering the normal result of echocardiography and the value of electrolyte serum, the patient also had no history of other arrhythmia causes, we concluded that there causes no other causes of arrhythmia, except for the drug. In this case, we did not administer placebo since it could possibly interfere with the ongoing tuberculosis therapy. We did not check the level of drug in the system, and we did not increase or decrease the dose to prevent the reappearance of arrhythmia. The patient was not a regular patient of our hospital, therefore, the previous drug reports were not available in our medical records, and there was no history of previous drug exposure from anamnesis. The adverse event was confirmed by electrocardiography results. The score for this patient is 7, indicating that levofloxacin is probably the cause of the adverse event.

Build upon the narration above, the most likely drug that cause supraventricular tachycardia in this patient is levofloxacin, therefore this drug is no longer being administrated to the patient. Levofloxacin molecules acts as inhibitor of the human ether-a-go-go related gene (hERG) channel codes rapid components of potassium channel, thus causing accumulation of potassium in the heart myocytes. Inhibition of the hERG is causing delayed repolarization, increase the duration of action potential, and early after depolarization, reflected as prolonged QT interval on the electrocardiogram. Furthermore, it could lead

to torsades de pointes, a deadly form of arrhythmia [39]. The internal and external risk factors are also take part in the incident of arrhythmia such as genetic, female gender, age, underlying disease, and co-administration with class III antiarrhythmic drugs. Some modifiable risk factors, including hypokalemia, hypomagnesemia, drug interaction, and bradycardia should be corrected to prevent arrhythmia [40].

There is concern that many second-line drugs used to treat multidrug-resistant tuberculosis (MDR-TB) may cause fatal arrhythmias linked to QT interval prolongation. A QTc interval of more than 500 ms is considered a risk factor for ventricular arrhythmias such as torsades de pointes (TdP) [41]. TdP is a syndrome of polymorphic ventricular arrhythmia occurring in the setting of marked prolongation of the QT interval by ECG. In TdP the QT interval is prolonged in the heartbeats before the sudden onset of rapid and disorganized contractions of the heart. Patients with TdP experience dizziness or loss of consciousness if the arrhythmia is brief. If sustained, TdP can be lethal [42]. As mentioned in endTB observational study, 2 fatal drug-related cardiac events and 1 drug-related cardiac arrhythmia were documented, all associated with hypokalemia and involving the use of bedaquiline, clofazimine, capreomycin, and p-aminosalicylic acid (excluding moxifloxacin or delamanid) [43].

QT interval is measured at the beginning of Q-wave to the end of T-wave, presenting the time needed of ventricle to depolarize and repolarize. The interpretation of QTc according to Bazett formula is shown in **Table 2**.

Table 2. In male adult, normal QTc is less than 430 milliseconds, borderline range between 430 - 450 milliseconds, and prolonged when it is more than 450 milliseconds, whereas in adult female, the normal QTc is less than 430 milliseconds, borderline range between 450-470 milliseconds and prolonged when it is longer than 470 milliseconds [44].

Rating	**1-15 y.o (milliseconds)**	**Adult Male (milliseconds)**	**Adult Female (milliseconds)**
Normal	<440	<430	<450
Borderline	440-460	430-450	450-470
Prolonged	>460	>450	>470

Consider performing an electrocardiogram examination before starting therapy as baseline and repeat every month during therapy. In the case of Multi Drugs Resistant - Tuberculosis (MDR-TB), patients who administer clofazimine or fluoroquinolone along with bedaquiline or delamanid should be monitored every week in the first month, then every month during therapy. Following matters should take special attention when the QT interval prolongation is more than 500 milliseconds [14] :

- Repeat ECG to confirm the prolongation of QT interval.
- Consider the discontinuation of the suspected drugs
- Check the albumin and electrolytes level, especially potassium, calcium, and magnesium.
- Maintain the potassium level above 4 mEq/L and magnesium level above 1,8 mg/dL

The main principal of ventricular tachycardia management divided into acute and continuation phase. Acute phase management is aimed at overcoming hemodinamic instability, arrhythmia conversion, and eliminate clinical symptoms. In the case of narrow complex arrhythmia, initiation of the Valsava maneuver, and cold-water face immersion can be performed to modify atrioventricular conduction and thus, terminate arrhythmia. The effectivity of the Valsava maneuver reaches 19.4 - 54% and is best performed at a supine position [45]. When this maneuver fails and the patient hemodynamically stable, intravenous anti-arrhythmic drug should be administered. Bolus of 6 - 18 mg of adenosine is the drug of first choice [46]. When contraindicated or ineffective, non-dihydropyridine calcium-channel antagonist such as verapamil or diltiazem, or short acting beta-blocker such as esmolol can be administered. Advanced management for the ventricular tachycardia is radiofrequency

ablation or maintenance therapy [47]. As for our patient, the administration of diltiazem followed by bisoprolol to help maintain the heart rate back to normal. The TB treatment was continued using rifampicin, isoniazid, and ethambutol while closely monitoring the liver function.

CONCLUSION

Careful consideration is required when giving fluoroquinolone as one of the drug in tuberculosis regiment regarding the incident of arrhythmia. Baseline electrocardiogram, liver, and renal function tests should be examined before starting the therapy. Acute management of ventricular tachycardia is pivotal in termination of arrhythmia. Close monitoring must be carried out in the first month of therapy in order to achieve the successful and complete elimination of tuberculosis.

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