

Successful Treatment of Tuberculosis Combined With Rifampicin Induced Interstitial Nephritis: A Case Report

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Abstract

A 61-year-old man was admitted to our hospital on September 2021 because of a three-month history of fatigue, night sweats, and weight loss. The patient had been diagnosed with organized pneumonia and was treated with methyl prednisolone at a local hospital. One week before presentation to our hospital, he had developed fever, cough, sputum and shortness of breath. Upon admission to our hospital, sputum acid-fast resistant bacteria (ARB) and urine ARB were positive and mycobacterium tuberculosis was cultured. With the diagnosis of drug-sensitive pulmonary and urinary tuberculosis, treatment consisting of isoniazid, rifampicin, ethambutol and pyrazinamide was started. After two months, his symptoms had not improved and he developed nausea and vomiting. At the next visit to our hospital, his renal function tests were further increased. He was taken for emergency dialysis. Diagnosis of interstitial nephritis was made by kidney biopsy, which was considered to have developed due to the use of rifampicin. Rifampicin was discontinued and methyl prednisolone was added. The new drug regimen was arranged as isoniazid, moxifloxacin, ethambutol, pyrazinamide and cycloserine. Cycloserine was discontinued due to tremors and prothionamide was started. When drug-induced hepatitis developed, his subsequent treatment was changed to isoniazid, levofloxacin, ethambutol, and pyrazinamide. The patient was treated with methyl prednisolone for two months. Currently, he has been taking the treatment stated above for 4 months without any problems and it is scheduled to be completed in 12 months.

Keywords: Tuberculosis; Rifampicin; Interstitial Nephritis; Tremor

Abbreviations: ARF: Acute Renal Failure; AIN: Acute Interstitial Nephritis; DI-AIN: Drug-Induced Acute Interstitial Nephritis

Introduction

One of the main causes of acute renal failure (ARF) is acute interstitial nephritis (AIN). AIN can develop due to drugs, infections, or autoimmune diseases. In many countries, the leading cause is drug-induced acute interstitial nephritis (DI-AIN) [1]. Almost all drugs are associated with the risk of DI-AIN. It is most commonly caused by non-steroidal anti-inflammatory drugs and antibiotics [2]. Kidney biopsy is required for diagnosis and once confirmed; culprit drugs must be discontinued [1]. Although the exact mechanism is not known, T cell-mediated delayed type hypersensitivity is mentioned [3]. Finding and removing the suspect drug from treatment prevents kidney damage [2].

In drug-sensitive tuberculosis patients, treatment consists of isoniazid, rifampicin, ethambutol, and pyrazinamide. The inclusion of rifampicin in the regimen shortens the treatment interval with a higher treatment success [4]. There are studies examining nephritis that develops with continuous or intermittent use of rifampicin [5,6]. We describe a case of pulmonary and urinary tuberculosis patient who was treated with continuous rifampicin and developed drug-induced interstitial nephritis.

Case Presentation

A 61-year-old man was admitted to our hospital on September 2021 because of three-month history of fatigue, night sweats, and weight loss. Chest radiography demonstrated bilateral apical infiltrates with cavitation and diffuse pneumonic infiltration in all lung segments (Figure 1). The patient had been diagnosed with organized pneumonia and was treated with methyl prednisolone at a local hospital. The patient, whose complaints gradually increased, was admitted to the emergency department of our hospital with fever, cough, sputum and shortness of breath. Sputum and urine samples were positive for Mycobacterium tuberculosis and culture later grew M. Tuberculosis. With the diagnosis of drug-sensitive pulmonary and urinary tuberculosis, he began therapy with daily isoniazid (300 mg), rifampicin (600 mg), ethambutol (1500 mg) and pyrazinamide (2000 mg). Results of initial laboratory tests (blood count, liver and kidney function tests) were in normal limits. After two months, his symptoms had not improved and he developed nausea and vomiting. Upon his next visit to our hospital, his renal function tests were increased. Creatinine: 11.6 mg/dL, HCO₃:12.4 and pH was 7.22. He was taken for emergency dialysis. There was proteinuria; no bacteria were seen

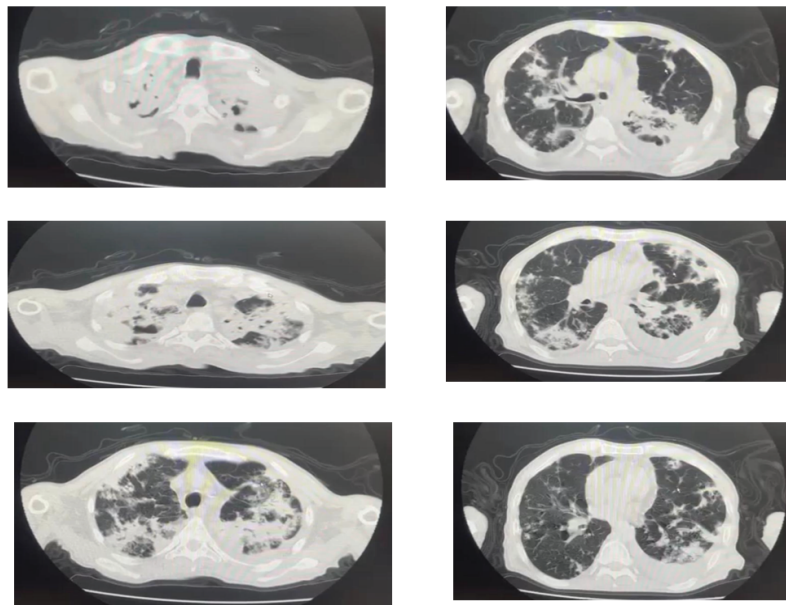


Figure 1: Pulmonary computed tomography of the patient at the first diagnosis of tuberculosis.

in the urine culture. The diagnosis of interstitial nephritis, possibly due to the use of rifampicin, was made by kidney biopsy. Rifampicin was discontinued and methyl prednisolone was added to the therapy. Methyl prednisolone treatment was started at 0.5 mg/kg with a plan to be discontinued in 2 months. The new drug regimen included isoniazid, moxifloxacin, ethambutol, pyrazinamide and cycloserine. At the fourth day of new regime, cycloserine was discontinued due to tremors and prothionamide was started. After 40 days, the patient was seen at an outpatient clinic due to nausea and abdominal pain. It was observed that liver enzymes increased more than 5 times. The enzymes eventually returned to the normal range, the patient's complaints also improved, and the treatment of isoniazid, levofloxacin, ethambutol, and pyrazinamide was resumed. He has been taking this last regimen for 3 months without any problems and is scheduled completion in 12 months.

Discussion

A patient who developed AIN due to rifampicin while receiving antituberculosis treatment is shared. AIN can develop due to drugs, infections, or autoimmune diseases. In many countries, the leading cause is DI-AIN [1]. The most important step in treatment is to find the responsible drug and discontinue it. Intravenous or oral steroids are recommended for treatment [7]. Although acute tubular necrosis due to rifampicin is more common, cases of AIN are also reported. Up to now, there are 22 patients diagnosed with acute renal failure by kidney biopsy due to the use of rifampicin. Most of the patients mentioned were male like our patient [8]. The presence of antibodies linked to rifampicin is mentioned in the etiology. However, it is not always possible to determine their presence [9]. We could not examine rifampicin-related antibodies in our patient. In a study evaluating 8 patients who developed AIN due to rifampicin in Japan, rifampicin was discontinued in all patients; some of them were given dialysis and some of them were only given steroids [10]. In our patient, rifampicin treatment was discontinued and both dialysis and steroids were administered. After rifampicin was discontinued, our patient recovered with a short-term dialysis program and steroid treatment.

Re-administration of the drug may be fatal in patients who develop AIN due to rifampicin. Cross-reactivity with rifabutin is unclear [11]. In such a life-threatening situation, we think that it is better for patient safety to start an alternative drug regimen instead of giving rifampicin to the patient. We were able to achieve treatment success with alternative drugs in the patient.

Complications that may develop can be recognized with early follow up in patients receiving tuberculosis treatment. Discontinuation of the culprit drug is important in assessing a patient's prognosis.

Ethics Approval and Consent to Participate

The patient file was reviewed retrospectively, and ethics committee approval was not obtained.

Consent for Publication

Written informed consent to participate and publish was obtained from all individual participants included in the study.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are not publicly available due to privacy of the patient's file and archive but are available from the corresponding author on reasonable request.

Competing Interests

Data available on request from the authors.

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Authors' Contributions

Zeynep Yegin Katran, Ali Burkan Akyıldız and Aylin Babalık take part of Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Validation; Visualization; Writing-original draft.

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