Case Report

Acute Kidney Injury Due to Anti-Tuberculosis Drugs: How Much Are We Aware of It?

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ABSTRACT

The global scenario of tuberculosis (TB) infection varies from one country to another; according to the World Health Organization (WHO), Bangladesh is one of the world's 30 high TB burden countries and near about 73,000 people die annually due to tuberculosis. Several drugs are used in Category-1 and Category-2 regimen for TB treatment. Despite several side effects of these drugs, we have to use them invariably following the WHO guideline in our country. Although isoniazid and ethambutol have been associated with acute kidney injury (AKI), recent evidence showed that rifampin is the most common anti-TB drug responsible for AKI. Rifampicin toxicity may appear at both the initial administration and readministration. A 50-year old male pulmonary TB patient developed severe renal impairment after taking Category-1 anti-TB drugs. When severe renal impairment developed, anti-TB therapy was stopped and five sessions of hemodialysis were given. When renal function came back to normal, treatment was resumed with oral ethambutol and levofloxacin in full dose (excluding rifampicin). Later, oral isoniazid was added with low dose and increased dose was given based on monitoring of renal function. Even after adding injection streptomycin no deterioration of renal function was observed. We herein report this rare case and review of literature for academic interest and creating more awareness among the physicians.

Keywords: *Acute kidney injury, anti-tuberculosis drugs, pulmonary TB*

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INTRODUCTION

Tuberculosis (TB) is a communicable disease affecting one-third of the global population. According to the World Health Organization (WHO), most people who developed TB in 2019 were in the South-East Asia region (44%).[1] Bangladesh is one of the world's 30 high TB burden countries and near about 73,000 people die annually due to tuberculosis.[1] Prompt anti-tuberculosis treatment remains the most important and effective intervention for controlling spread, but adverse events from first-line anti-TB drugs are not uncommon.[1,2] Several drugs are used in Category-1 and Category-2 regimen for TB treatment. Despite several side effects of these drugs, we have to use them invariably following the WHO guideline in our country. Acute kidney injury (AKI) is a rare and severe complication that can interrupt treatment and cause permanent kidney damage.[3] Although isoniazid and ethambutol have been associated with AKI,[2-4] in recent years, evidence showed that rifampin is the most common anti-TB drug responsible for AKI.[5-10] Reviewing literature of rifampin-induced AKI, the mean age of reported cases was around 40–45 years and the recovery rate ranged between 40% and 96%.[5-10] However, the incidence to rifampin-induced AKI is uncertain because the definitions of AKI used in previous studies had much variations.[2] Recently, a male TB patient was admitted in a tertiary level specialized hospital in Dhaka, Bangladesh with acute kidney injury due to anti-tuberculosis drugs.

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CASE SUMMARY

A 50-year-old man hailing from a sub-urban area with a history of receiving five sessions of hemodialysis following severe renal impairment was admitted to the Gonoshasthaya Nagar Hospital, Dhaka, Bangladesh, in the beginning of 2024. He stated that he was diagnosed with pulmonary TB in 2011, and he received category-1 anti-TB therapy for 6 months and got recovery. However, he was diagnosed once again in the end of 2023 and started category-1 anti-TB therapy. After a week of treatment, he suddenly developed severe nausea and vomiting. He got admitted in a local hospital; he had severe renal impairment with serum creatinine 13.30 mg/dL at that time. Then he was referred to a private clinic where anti-TB therapy was stopped and five sessions of hemodialysis given. After that he was shifted to this hospital for better management. After admission, he was clinically improved and urine output was adequate. On investigation, his serum creatinine was 6.5 mg/dL, while blood urea was 90mg/dL and urine analysis revealed no abnormalities. Moreover, serum electrolytes and sonography of the urinary system revealed no abnormalities. To find out the cause of renal impairment, patient's history and pathological investigations done earlier were revisited (including urine analysis, sonography of urinary system, serum HBsAg, Anti-HCV, C-ANCA, P-ANCA, ANA, and chest x-ray P/A view). However, no abnormalities were detected except in the chest x-ray, which revealed a cavitary lesion in the upper zone of the right lung. It was assumed that the AKI was due to ingestion of anti-TB drugs. After few days, hemodialysis was stopped and the patient was managed conservatively with close monitoring. After a week, his condition further improved with adequate urine output and investigations revealed serum creatinine 4.19 mg/dL and blood urea 80 mg/dL. The patient was discharged from the hospital. After one and a half month, his condition further improved and serum creatinine came back to the baseline (1.27 mg/dL). He was advised for further investigations, e.g., sputum for acid-fast bacilli (AFB) and Gene Xpert; both reports were suggestive of active TB infection. Chest radiograph also revealed the same cavitary lesion in the lung. Then alternate regimen of anti-TB medication was prescribed, i.e., with oral ethambutol 400 mg 3 tablets daily, levofloxacin 500 mg once daily (for 2 months) and later added with oral isoniazid 300 mg ¼ tablet for 2 days, then ½ tablet another 2 days, followed by 1 tablet once daily. After one month of regular treatment, there was mild clinical improvement; however, chest x-ray still showed that cavitary lesion of the lung. Then intramuscular injection of streptomycin 1 gm daily was added for 2 months along with previous drugs. After 2 months, patient's chest x-ray showed significant change by healing of the cavitary lesion. Then injection streptomycin was withdrawn from the list and continued with oral ethambutol and isoniazide. After 6 months of complete anti-TB treatment, his serum creatinine was found 1.06 mg/dL and chest x-ray showed few fibrotic areas. The patient was advised to continue anti-TB therapy for another 3 months. During our treatment with alternate anti-TB regimen, patient's renal function was stable and serum creatinine level was around 1.10 mg/dL.

DISCUSSION

Acute kidney injury (AKI) is usually a rare complication in patients on anti-TB therapy.[11] However, several reports addressed this rare event and most revealed rifampicin as the most common responsible drug.[5-10] The mechanism of rifampicin-induced AKI is not well established. Evidence suggests that it is either a type II or type III hypersensitivity reaction induced by rifampicin antigens in which anti-rifampicin antibodies form immune complexes that are deposited in renal vessels, the glomerular endothelium, and the interstitial area. These reactions cause two different pathologic changes in the kidneys. The deposition of immune complexes in the vessels causes vascular constriction and tubular ischemia, leading to acute tubular necrosis, Whereas the deposition of immune complexes in the interstitial area leads to acute interstitial nephritis.[5,8,11] Renal biopsies may reveal either tubulo-interstitial or glomerular lesions. Oedema resulting from proteinuria is also relatively common.[8,10,11] In our case, the cause of AKI was not confirmed because renal biopsy was not performed. However, the results of previous studies suggest that even without histology studies, the diagnosis of rifampin-induced AKI can be made based on the typical time course and by excluding other etiologies.[5] In this case, after one week of taking anti-TB therapy, the patient developed severe renal impairment. To exclude other cause and previous renal impairment, we tried our best to be informed about patient's demographic, family and clinical history

meticulously and did all relevant investigations. Since rifampicin is commonly associated with AKI, we started alternate regimen of anti-TB therapy by excluding rifampicin. Firstly, ethambutol and levofloxacin were started in full doses, then isoniazid was added with low dose and the dose was increased based on monitoring of renal function. Even after adding injection streptomycin no deterioration of renal function was observed. Rifampicin toxicity may appear at both the initial administration and readministration. However, with treatment, the clinical course of rifampicin-induced acute kidney injury is considered favourable in most patients.[10,11] We herein report this rare case and review of literature for academic interest and creating more awareness among the physicians.

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Ethical Approval: Written informed consent was taken from the patient and permission was obtained from the hospital authority.

Authors' Contribution: AM Kaiser was involved in patient selection, data collection and clinical management; AM Kaiser, RN Islam, ME Jalil and ASM Morshed were equally engaged in patient's data analysis, literature search and review as well as manuscript writing, editing and final submission.

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