

Rifampicin-Induced Acute Liver Failure with Acute Kidney Injury and Thrombocytopenia in a Young Patient: A Rare Case Report.

Vishakha Prajapati¹, Niti Contractor¹, Dr. Bhavin A Vyas²

¹Pharm D. Intern, Department of Pharmacy Practice, Maliba Pharmacy College, Surat, Gujarat, India

²Professor, Department of pharmacology and pharmacy practice, Maliba Pharmacy College, Surat, Gujarat, India

Date of Submission: 15-04-2026

Date of Acceptance: 25-04-2026

ABSTRACT: Rifampicin is a commonly used antimicrobial agent but rarely may cause severe adverse drug reactions involving multiple organs. We report the case of an 18-year-old male who developed severe vomiting (~30 episodes), hematuria, fever, breathlessness, and bluish discoloration of extremities one day after initiating Rifampicin 450 mg and Doxycycline 100 mg for facial acne. Investigations revealed marked hyperbilirubinemia, elevated liver enzymes, coagulopathy, thrombocytopenia, and acute kidney injury, consistent with acute liver failure with AKI, along with hemolysis and metabolic acidosis. The patient subsequently developed sepsis with septic shock, while stool multiplex PCR detected enteropathogenic *Escherichia coli*. He required intensive care management, including vasopressor support, blood product transfusions, broad-spectrum antibiotics such as Meropenem, and renal replacement therapy (SLED followed by hemodialysis). Following withdrawal of rifampicin and aggressive supportive treatment, the patient gradually improved with stabilization of liver parameters and recovery of urine output and was discharged in stable condition. This case highlights a rare but serious presentation of rifampicin-induced acute liver failure with acute kidney injury, sepsis, and thrombocytopenia, emphasizing the importance of early recognition and prompt management of severe drug-induced adverse reactions.

KEYWORDS: Rifampicin, Drug-induced acute liver failure, Acute kidney injury, Sepsis, Thrombocytopenia, Hemolysis, Adverse drug reaction, Hemodialysis.

I. INTRODUCTION

Rifampicin is a broad-spectrum bactericidal antibiotic belonging to the rifamycin class and is widely used in the treatment of infections such as Tuberculosis, leprosy, and other serious bacterial

diseases.¹ It acts by inhibiting bacterial DNA-dependent RNA polymerase, thereby suppressing RNA synthesis and preventing bacterial replication.¹ Although rifampicin is generally well tolerated, commonly reported adverse effects include hepatotoxicity, gastrointestinal disturbances, hypersensitivity reactions, and hematological abnormalities.² Rare but severe complications such as acute liver failure, hemolytic anemia, thrombocytopenia, and acute kidney injury have also been reported and may occasionally progress to multiorgan dysfunction and shock.^{2,3}

Reporting such rare adverse drug reactions is important for clinical awareness and pharmacovigilance, as early recognition and prompt withdrawal of the offending drug can significantly improve patient outcomes.² Here, we report a rare case of rifampicin-induced acute liver failure with acute kidney injury, sepsis, and thrombocytopenia in an 18-year-old patient shortly after initiation of therapy, highlighting the importance of recognizing severe drug-induced reactions even after short-term exposure.³

II. CASE PRESENTATION

An 18-year-old male presented with complaints of severe vomiting (~30 episodes), hematuria, generalized weakness, fever, breathlessness, and bluish discoloration of distal extremities and tongue. One day prior to admission, he had started treatment for facial acne with Rifampicin 450 mg and Doxycycline 100 mg. On arrival, the patient was conscious but appeared markedly weak and was admitted to the intensive care unit for further management.

Initial investigations revealed leukocytosis (TLC 25,900 cells/mm³), thrombocytopenia (1.6 lakh/mm³), and acute kidney injury with serum creatinine 3.78 mg/dL. Liver function tests showed marked hyperbilirubinemia (total bilirubin 11.1

mg/dL) with elevated AST (634 IU/L) and coagulopathy (INR 3.33), suggestive of acute liver failure. Additional findings included markedly elevated LDH (5394 U/L), metabolic acidosis on ABG, methemoglobinemia (3.9%), and D-dimer >1000 ng/mL. HRCT thorax was unremarkable, and 2D echocardiography demonstrated normal cardiac function (LVEF 65%).

Subsequent investigations showed worsening inflammatory markers with TLC 34,300 cells/mm³, ferritin 16,300 ng/mL, and procalcitonin >100 ng/mL, indicating sepsis with septic shock. Evidence of hemolysis was present with positive direct Coombs test, hemoglobinuria, elevated LDH, and increased reticulocyte count. Stool multiplex PCR detected enteropathogenic *Escherichia coli*. Ultrasound abdomen revealed mild hepatomegaly, grade I renal parenchymal disease, cystitis, and minimal ascites. The patient developed hypotension requiring norepinephrine infusion, and antimicrobial therapy was escalated to Meropenem. Coagulopathy was managed with fresh frozen plasma and cryoprecipitate transfusions. Progressive oliguria necessitated heparin-free sustained low-efficiency dialysis (SLED) followed by intermittent hemodialysis.

Over the subsequent days, liver enzymes, bilirubin levels, and inflammatory markers gradually improved, although renal dysfunction persisted, requiring repeated dialysis sessions. Supportive management included hydrocortisone, N-acetylcysteine, vasopressor support, and broad-spectrum antibiotics. Blood cultures remained sterile during the hospital stay.

By day 10 of hospitalization, the patient showed clinical improvement with resolution of fever and stabilization of hemodynamic status, although intermittent dialysis was still required. Episodes of hypertension were managed with Nicardipine, Amlodipine, and Clonidine, while symptomatic therapy including Metoclopramide and Pantoprazole was provided.

By day 14, the patient demonstrated clinical recovery with adequate urine output and stable vital parameters. Renal Doppler showed grade II renal parenchymal disease, and the dialysis catheter was removed. At discharge, serum creatinine had improved to 6.72 mg/dL, and the patient was hemodynamically stable with satisfactory urine output.

Considering the temporal association between initiation of rifampicin and the onset of severe hepatic injury, acute kidney injury, hemolysis, thrombocytopenia, and septic shock, the final diagnosis was rifampicin-induced acute liver

failure with acute kidney injury, sepsis, and thrombocytopenia. The patient was discharged on oral supportive medications including Pantoprazole and Nicardipine, with advice for adequate hydration and close nephrology follow-up.

III. DISCUSSION

Rifampicin is widely used for the treatment of infections such as Tuberculosis, but rare severe adverse reactions including acute liver failure (ALF) and acute kidney injury (AKI) have been documented.¹ Drug-induced hepatotoxicity is one of the most recognized complications of rifampicin therapy and may range from mild asymptomatic elevation of liver enzymes to fulminant hepatic failure.² Several case reports have also described rifampicin-associated AKI characterized by gastrointestinal symptoms, hemolysis, thrombocytopenia, and rapid deterioration in renal function.³ The pathogenesis of rifampicin-induced renal injury is believed to be immune mediated, involving the formation of rifampicin-dependent antibodies that target erythrocytes and renal tubular cells, leading to complement activation and cellular injury.⁴ This immune mechanism may result in acute tubular necrosis or acute interstitial nephritis, the most commonly reported pathological patterns in such cases.⁵ Hemolysis and hemoglobinuria can further aggravate renal damage through pigment-induced tubular toxicity.⁶ In addition, rifampicin has been associated with thrombocytopenia and thrombotic microangiopathy, likely due to immune-mediated platelet destruction.⁷ In previously reported studies, many patients required temporary renal replacement therapy, although most recovered renal function following prompt discontinuation of the drug and supportive treatment.⁸

The present case demonstrates a similar clinical pattern with the rapid onset of hepatic dysfunction, AKI, hemolysis, thrombocytopenia, and septic shock shortly after exposure to rifampicin. Early recognition of this severe adverse drug reaction is crucial because immediate withdrawal of the suspected drug is the most important step in management.⁹ Supportive care including hemodynamic stabilization, blood product transfusion, treatment of sepsis, and renal replacement therapy may be required in severe cases.¹⁰ Clinicians should maintain a high index of suspicion when patients receiving rifampicin present with symptoms such as vomiting, hematuria, jaundice, or reduced urine output, particularly within a short period after drug initiation.¹¹

Awareness of such rare reactions is essential to prevent delays in diagnosis and to improve patient outcomes.¹² This case also highlights the importance of pharmacovigilance and reporting of rare adverse drug reactions, which helps expand the current understanding of drug safety and guides clinicians in safer prescribing practices.¹³

IV. CONCLUSION

This case highlights a rare but severe adverse drug reaction associated with Rifampicin, presenting as acute liver failure, acute kidney injury, hemolysis, thrombocytopenia, and septic shock shortly after initiation of therapy. Although rifampicin is widely used for infections such as Tuberculosis, clinicians should remain vigilant for uncommon but life-threatening complications. The rapid onset of multiorgan dysfunction in this patient emphasizes the importance of early recognition of drug-induced toxicity, prompt withdrawal of the suspected agent, and aggressive supportive management, including renal replacement therapy and hemodynamic stabilization. Timely intervention in this case contributed to gradual clinical recovery and stabilization of organ functions. Reporting such rare adverse drug reactions is essential for strengthening pharmacovigilance systems, improving clinician awareness, and promoting safer prescribing practices, particularly when drugs with known hepatotoxic and nephrotoxic potential are used.

Clinical Significance

The clinical importance of this case is that it shows the rare but serious risk of multi-organ failure, especially acute liver failure, acute kidney injury, and blood-related issues. These can quickly develop after starting Rifampicin.

This report reminds doctors that even common and usually safe antibiotics can cause severe, unexpected reactions. They need to be alert when patients show sudden, multi-system symptoms like jaundice, blood in urine, or low urine output soon after treatment begins. It highlights that quick diagnosis and stopping the problematic drug are key to patient recovery. By documenting such rare side effects, this case supports important drug safety efforts and underscores the need for careful monitoring and evidence-based care to prevent complications and improve results in urgent medical situations.

REFERENCES

- [1]. Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's The Pharmacological Basis of Therapeutics. 13th ed. New York: McGraw-Hill; 2018.
- [2]. Tostmann A, Boeree MJ, Aarnoutse RE, de Lange WC, van der Ven AJ, Dekhuijzen R. Antituberculosis drug-induced hepatotoxicity: concise up-to-date review. *J Gastroenterol Hepatol.* 2008;23(2):192-202.
- [3]. Schaberg T, Rebhan K, Lode H. Risk factors for side-effects of rifampin in patients with pulmonary tuberculosis. *Eur Respir J.* 1996;9(10):2026-30.
- [4]. Muthukumar T, Jayakumar M, Fernando EM, et al. Acute renal failure due to rifampicin. *Nephrol Dial Transplant.* 2002;17(4):633-6.
- [5]. Covic A, Goldsmith DJ, Segall L, et al. Rifampicin-induced acute renal failure: a series of 60 patients. *Nephrol Dial Transplant.* 1998;13(4):924-9.
- [6]. De Vriese AS, Robbrecht DL, Vanholder RC, et al. Rifampicin-associated acute renal failure. *Am J Kidney Dis.* 1998;31(1):108-15.
- [7]. Pereira J, Hidalgo P, Ocqueteau M, et al. Rifampicin-induced immune thrombocytopenia. *Br J Haematol.* 1996;95(2):403-5.
- [8]. Covic A, et al. Rifampicin-induced acute renal failure requiring dialysis. *Nephrol Dial Transplant.* 1998;13:924-9.
- [9]. Andrade RJ, Chalasani N, Björnsson ES, et al. Drug-induced liver injury. *Nat Rev Dis Primers.* 2019;5:58.
- [10]. Ostermann M, Joannidis M. Acute kidney injury management in ICU. *Lancet.* 2016;388:261-75.
- [11]. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl.* 2012;2:1-138.
- [12]. Navarro VJ, Senior JR. Drug-related hepatotoxicity. *N Engl J Med.* 2006;354:731-9.
- [13]. Edwards IR, Aronson JK. Adverse drug reactions: definitions and classification. *Lancet.* 2000;356:1255-9.