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RIFAMPICIN INDUCED ACUTE THROMBOCYTOPENIA AND EXTENSIVE PURPURA: A CASE REPORT

Dermatology

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ABSTRACT

Rifampicin is one of the first-line antitubercular drug. While generally well-tolerated, its associated adverse effects can include hepatitis, purpura, cutaneous flushing, nausea, emesis, pyrexia, and pruritus. This report discusses the case of a 49-year-old female patient undergoing treatment for tuberculosis who presented with severe thrombocytopenia accompanied by a widespread purpuric rash across her entire body. Following clinical evaluation, the thrombocytopenia and purpura was attributed to rifampicin administration. The patient was managed with a daily regimen of prednisolone, resulting in a 50% reduction of the rash within 24 hours and absolute resolution within 72 hours.

KEYWORDS

Rifampicin, Thrombocytopenia, Purpuric Rash

INTRODUCTION

Tuberculosis is a chronic granulomatous infection caused by Mycobacterium tuberculosis. It remains the leading cause of mortality globally from a single infectious pathogen.[1] Data from the World Health Organization's Global Tuberculosis Report 2025 indicates that approximately 10.7 million individuals contracted tuberculosis in 2024. The highest burden of disease was concentrated in South-East Asia (34%), followed by the Western Pacific (27%) and Africa (25%). Furthermore, two-thirds of global tuberculosis cases were from just eight nations: India, Indonesia, the Philippines, China, Pakistan, Nigeria, the Democratic Republic of the Congo, and Bangladesh. Notably, India alone accounted for 25% of the global tuberculosis in 2024.[2] On a global scale, more than 96% of newly diagnosed pulmonary tuberculosis cases respond effectively to the standard four-drug regimen utilized since the 1980s.[3] These first-line drugs comprise of Isoniazid, Rifampicin, Pyrazinamide, Ethambutul, and Streptomycin. Although severe or fatal adverse drug events from these first-line agents are uncommon, they do occur. Rifampicin an effective antitubercular drug can be prescribed either daily or intermittently. Its recognized side effects encompass hepatotoxicity, cutaneous eruptions, gastrointestinal distress, and flu-like symptoms. Immunological and autoimmune complications, including thrombocytopenia, are predominantly associated with high-dose, intermittent dosing schedules.[4] The first documentation of rifampicin-induced thrombocytopenia dates back to the 1970s by Blajchman et al.[5] Thrombocytopenia is occasionally caused by rifampicin, when high-doses are given twice-weekly. This complication is rare when the medication is given in small daily doses, or following a prolonged interruption of therapy.[6]

In this paper, we present the case of a 49-year-old female tuberculosis patient, on antitubercular therapy, who developed thrombocytopenia and an extensive purpuric rash throughout her body, which was subsequently diagnosed as an adverse reaction to rifampicin therapy.

CASE HISTORY

A 49 year old female patient presented with extensive purpuric rash covering her entire body sparing only her face. The patient appeared severely ill. The laboratory findings revealed significant thrombocytopenia with platelet count of 30,000/µL. The patient's medical history revealed that she had a cardiac pacemaker implantation two years ago. She had developed pleural effusion which had been tapped by a chest specialist. She was diagnosed of pulmonary tuberculosis. Consequently she was started on antitubercular regimen consisting of Rifampicin 600 mg + INH 300 mg and ethambutol 800 mg. Twenty five days after initiation of antitubercular therapy patient developed extensive purpuric rash accompanied by severe stinging sensation all over the body. An empirical diagnosis of drug induced purpura due to Rifampicin was made. Management was initiated immediately by discontinuing all antitubercular medications from day one and administering prednisolone 60mg daily in the morning along with Omeprazole for gastric protection. Within 20 hours of treatment initiation, the patient showed clinical improvement. There was fifty percent clearance of purpuric rash. Corticosteroid therapy was continued for three days. The patients liver function tests remained within normal physiological limits. Ongoing cardiac monitoring revealed that her prior cardiovascular history were unrelated to development of purpura. Due to severe nature of the adverse drug reaction, Rifampicin was discontinued. Although some case reports indicate rechallenge with Rifampicin under close observation but this can lead to fall in platelet levels.

DISCUSSION

Tuberculosis remains highly prevalent in India, with its disease burden significantly intensified by comorbid factors such as undernutrition, smoking and poverty. Standard first-line pharmacological management for tuberculosis involves a combination of isoniazid, rifampicin, pyrazinamide, ethambutol and streptomycin. Antitubercular therapy constitutes a rare but important cause of thrombocytopenia. Tuberculosis itself may cause immune-mediated thrombocytopenia.[7] Among the standard first-line agents, rifampicin is most frequently implicated in thrombocytopenia, but certain case reports have highlighted that isoniazid can also cause thrombocytopenia.[8][9]

In our study patient reported development of extensive purpura along with thrombocytopenia. Administration of corticosteroid and stopping further administration of antitubercular therapy helped the patient to recover. Our findings are somewhat similar to case reported by Nawahirsha et al where the patient with rifampicin-induced thrombocytopenia presented with purpuric rash. Maintaining a high index of suspicion for these uncommon adverse drug reactions is vital for optimizing patient outcomes.[10]

A case report by Nakane et al presents a case of severe immune thrombocytopenia. Their case report detailed a 74-year-old male treated for tuberculous pleurisy who experienced profound thrombocytopenia following his initial exposure to Rifampicin. The patients platelet levels plummeted within just seven days of starting a combination regimen.[11] In our study patient reported development of extensive purpura along with thrombocytopenia. Administration of corticosteroid and stopping further administration of antitubercular therapy helped the patient to recover.

CONCLUSION

From this case report we want to emphasize that we should be aware of the rare adverse drug reactions associated with antitubercular drugs.

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