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A Case Report On Rifampicin- Induced Thrombocytopenia

Hannah Joseph, Sreehari. R, Layana Roy
Pharm D. Department of Pharmacy Practice. Nazareth college of Pharmcy, Othera, Thiruvalla

***Corresponding Author:**
Hannah Joseph
Pharm D. Department of Pharmacy Practice. Nazareth college of Pharmcy, Othera, Thiruvalla

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Abstract

Rifampicin is one of the most widely used and effective antituberculosis drugs. Thrombocytopenia is an uncommon but potentially life threatening complication of Rifampicin and is characterized by rapid destruction of platelets. This adverse effect is relatively more common with intermittent therapy than with daily regimen. Here we report a case of rifampicin-induced thrombocytopenia, which although rare, needs attention.

Keywords: Rifampicin, Thrombocytopenia, Complication, Platelets

Introduction

Treatment for Tuberculosis (TB) has been a therapeutic struggle for a long time. Although the majority of Antitubercular medications are generally safe, severe reactions can occasionally occur [1]. Rifampicin is an essential drug in the treatment regimen for TB [2]. Thrombocytopenia is an infrequent but possibly a deadly side effect of several antitubercular medications including Rifampicin [3]. Rifampicin induced Thrombocytopenia was first reported in 1970 by Blajchman and co-workers [4,1]. It is typically reversible if detected early and treated properly. Other drugs known to cause Thrombocytopenia are Isoniazid, Pyrazinamide, Quinine, Quinidine, Sulfonamides, Tolbutamide, Penicillamine, Chloroquine, Digoxin, Chlorthiazide, Amphotericin B, Sedatives, Anticonvulsants, Methyldopa, Aspirin etc [1,3]. We are reporting a case of Rifampicin-induced thrombocytopenia which was being treated for abdominal tuberculosis. The physician treating tuberculosis patients must be aware of this uncommon, potentially fatal consequence of Thrombocytopenia associated with Rifampicin.

Case Report

A 40- year- old male, presented to the Emergency Department of Believers Church Medical College Hospital, Thiruvalla with chief complaints of generalized tiredness for 1 week and bleeding per rectum for 1 month. He also reported a history of fever 3 days back. He is a known case of Chronic kidney disease on maintenance hemodialysis since 2017. He had recently diagnosed abdominal tuberculosis for which he was on ATT from 21/05/2022. Patient was taking 4 tablets of fixed dose combination of Rifampicin 150mg, Isoniazid 750mg, Pyrazinamide 400mg and Ethambutol 275 mg.

On examination his vital parameters were normal. His blood investigations showed severe anemia with Hemoglobin level of 3.7mg/dl and severe thrombocytopenia with platelet value of 0.01 Lakhs/microlitre. His serum Creatinine value was 4.76mg/dl, and the PT, INR values were 17.9 and 1.33 respectively. He was admitted under the Nephrology department of Believers church medical college and hospital for further treatment. An emergency Gastroenterology consultation was sought in view of bleeding per rectum and grade III Hemorrhoids and they advised conservative management for the same. In view of low Hb count and elevated serum creatinine level, he was initiated

on an emergency session of Hemodialysis and 2 units of PRBC was transfused during Dialysis.

In view of high suspicion of Rifampicin induced thrombocytopenia, ATT was withheld and he was treated with a short course of steroid therapy with IV methylprednisolone for 4 days. A total of 12 units of platelets were transfused during the entire hospital stay.

On further investigations, the bone marrow aspiration and biopsy showed normocellular marrow with increased megakaryocytes. Over the next 4 days of treatment, his bleeding subsided, platelet improved and he became symptomatically better. He was discharged on 14/06/2022 with a platelet count of 0.66 Lakhs/microlitre and decided to restart ATT without Rifampicin on follow up visits.

During follow- up visits, his platelets count became normal to 2.31 Lacks/cumm. ATT was restarted after subtracting Rifampicin from the regimen. He was prescribed with the following regimen:

T. Isoniazid 300mg daily

T. Ethambutol 800mg post Hemodialysis- only on HD days.

T. Pyrazinamide 1500mg post Hemodialysis- only on HD days

T. Levofloxacin 250mg OD.

There were no similar complaints on subsequent follow up. He has been warned against taking rifampicin for the rest of his life.

Discussion

Thrombocytopenia can arise with all primary anti-tubercular drugs. The Isoniazid causes it to manifest as a hematological reaction [5]. There are instances of thrombocytopenia being caused by Ethambutol and Pyrazinamide, perhaps through an immunological mechanism [6,7].

Adverse reactions to rifampicin are uncommon on daily regimen but are relatively common with intermittent regimen [8]. These include cutaneous syndrome, abdominal syndrome, a flu like syndrome, respiratory syndrome, purpura and elevated serum transaminase levels [1,3].

Anti-rifampicin antibodies have been demonstrated to be the cause of rifampicin-induced thrombocytopenia [9]. Even though we were unable to measure the rifampicin dependent antibodies in our patient, the occurrence of thrombocytopenia is attributed to rifampicin dependent antibodies. In the presence of rifampicin, these antibodies cause a complement to be fixed to the platelets, which causes platelet disintegration [10]. The incidence of thrombocytopenia usually occurs with intermittent therapy with rifampicin and less common with daily regimen [11]. It has been suggested that continuous treatment with rifampicin results in neutralization of any of the antibodies formed, the antigen--antibody complex being continuously removed without causing allergic reaction. Discontinuation of treatment allows a sufficient quantity of antibody to be built up during the drug-free interval so that when rifampicin is re-administered, an intense reaction ensues [1]. Though Rifampicin induced thrombocytopenia is rare with daily regimen, it has been reported by some authors. The mechanism hypothesized is that in the presence of the drug, the immune complexes nonspecifically adsorb to the platelet membrane, causing platelet damage and rapid removal from the circulation. The binding epitope of the Immunoglobulin G (IgG) antibody was found in the glycoprotein Ib/IX complex which is the target in rifampicin-induced immune thrombocytopenia [3]. As we were unaware of the rhythm of drug intake in our patient it is uncertain whether the thrombocytopenia was brought on by daily or irregular rifampicin consumption.

Thrombocytopenia induced by Rifampicin is often treatable and reversible if caught early. Our patient recovered completely after discontinuing the medicine and receiving supportive steroid treatment.

Conclusion

Healthcare professionals need to be aware of the potentially fatal hazards associated with thrombocytopenia, despite the fact that it is an uncommon side effect of Rifampicin. Both minor bleeding complications such as bruising or epistaxis, as well as more serious ones, including Gastrointestinal and abrupt subdural hemorrhage, can worsen thrombocytopenia. It is typically reversible if detected early and treated properly.

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Volume 5, Issue 4; July-August 2022; Page No 912-914

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Volume 5, Issue 4; July-August 2022; Page No 912-914