Rifampicin-induced thrombocytopenia in a patient of disseminated tuberculosis

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ABSTRACT

Thrombocytopenia may be associated with a variety of conditions and risks depending on its severity, ranging from mild epistaxis to life-threatening bleeding. Many drugs or herbal remedies can cause thrombocytopenia by either inhibiting platelet production and/or enhancing their destruction from the peripheral blood-mediated through an immunological mechanism implicating drug-dependent antibodies. Drugs are a common cause of acute immune-mediated thrombocytopenia in adults, the drug etiology is often initially unrecognized. Most cases of drug-induced thrombocytopenia are caused by drug-dependent antibodies that are specific for the drug structure and bind tightly to platelets by their Fab regions but only in the presence of the drug. Thrombocytopenia is an uncommon but life-threatening complication of certain antitubercular drugs. The discovery of isolated thrombocytopenia in a patient taking several medications presents a challenging clinical problem. We report a case of a young immunocompetent female who presented with disseminated tuberculosis and was found to have rifampicin-induced thrombocytopenia.

Key words: Immunocompetent, Miliary tuberculosis, Rifampicin, Thrombocytopenia

to identify clinically significant thrombocytopenia. Adverse reactions due to rifampicin are either dose-related or allergic. Serious reactions to anti-TB drugs are uncommon. Rifampicin-induced thrombocytopenia is an uncommon but potentially life-threatening complication of anti-TB treatment [1]. Rifampicin-induced thrombocytopenia was first reported by Blajchman et al. in 1970 [2]. Most of the described cases were observed in patients with high-dose intermittent therapy with rifampicin (1200 mg twice weekly) [3]. Only a few cases of thrombocytopenia have occurred during daily treatment or after administration of rifampicin following an interruption of therapy [2,4].

CASE REPORT

A 30-year-old female patient was admitted with a history of fever (101°F) mainly during evening hours, non-productive cough, and pain abdomen for the past month.

At presentation, the patient was conscious, oriented with stable vitals, respiratory rate of 18 breaths/min, and SpO₂ of 96% at room air. On palpation, the right supraclavicular lymph node was enlarged (2 cm × 1.5 cm) and firm in consistency. A respiratory system examination showed bilateral air entry with no added sounds. Other systems examination was unremarkable.
Thrombocytopenia attributed to rifampicin, though rare, has been reported in the treatment of pulmonary TB [6-8]. In our patient, we ruled out infection by cultures and collagen vascular disease with antinuclear antibody testing. Our patient was not receiving any of the drugs except rifampicin which is known to cause thrombocytopenia.

TB Research Center, Chennai, reported only a single case of rifampicin-induced thrombocytopenia among over 8000 patients treated for TB over 30 years [9]. In our case, the patient developed thrombocytopenia after interruption of antitubercular treatment for 2 months on a daily regimen. Our patient had tolerated the initial treatment with rifampicin daily without any complaints, but since her liver function test got deranged. Hence, it was withheld for a few days. Severe thrombocytopenia was observed within 2 days after restarting rifampicin. Serious adverse reactions due to rifampicin, which is immune complex-mediated, are mostly encountered during intermittent therapy or when there is a gap in treatment [10].

In patients with clinically DITP, an etiological agent can be identified in only 10% of cases. In the remaining cases, the etiological diagnosis can be pointed out by a prompt rise in the platelet count on withdrawal of the offending drug [2]. If detected early and treated appropriately, thrombocytopenia during rifampicin therapy is usually reversible. Since in our patient, it was detected early, the drug was stopped and supportive steroid therapy was given following which the resolution occurred. It has been recommended that rifampicin-induced thrombocytopenia is an absolute contraindication to further therapy with rifampicin [11]. Rechallenging with the offending drug even in small doses is contraindicated if purpura occurs [12].

**CONCLUSION**

Disseminated TB is associated with high mortality despite the availability of effective treatment. Platelet counts should be monitored after starting antitubercular drugs. The clinician must be aware of these rare complications which are life-threatening, but if detected early is completely reversible.

**REFERENCES**

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