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Case Report

Rifampicin induced acute thrombocytopenia with skin rashes: a case report

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ABSTRACT

Rifampicin is an essential component of the treatment regimen for tuberculosis. Extensive clinical experience has shown that the drug has mild adverse effect, but on rare occasions, it can cause life threatening adverse reactions like thrombocytopenia. Blajchman et al first described rifampicin-induced thrombocytopenia in 1970. Since then, almost 40 cases have been documented in the literature and a recent review by Cooper and Ghanima included rifampicin in the group of not uncommon drugs that cause immune-mediated thrombocytopenia. Diagnosis of drug-induced immune thrombocytopenia is by identifying clinical symptoms and a careful evaluation of the suspected causative drug. The physician treating tuberculosis patients must be aware of this rare life-threatening complication, when we detected early, is completely reversible.

Keywords: Tuberculosis, Thrombocytopenia, Rifampicin, Drug induced, Skin rashes, Anti-tuberculous therapy

INTRODUCTION

Among first line anti tuberculous drugs, one of the most potent drug is rifampicin. Most common adverse reactions with rifampicin include gastrointestinal effects, cutaneous reactions, hepatotoxicity and flu-like syndrome. Immunological reactions, such as haemolytic anaemia, agranulocytosis and thrombocytopenia are rare complications.[1] Thrombocytopenia is a potential life-threatening adverse effect, which is reversible on early diagnosis and treatment.[2] Immune-mediated thrombocytopenia induced by rifampicin was first reported in the year 1970.[3] An estimated incidence of approximately 10 cases per 1,000,000 population per year has been reported.[4] Since being a rare case, we are reporting a case of rifampicin induced acute thrombocytopenia with skin rashes.

CASE REPORT

A 42-year-old female diagnosed case of smear positive pulmonary tuberculosis on anti - tubercular drugs (4 FDC-3 pills/day) for past one week presented with chief complaints of fever, hyper pigmented rash all over the body for 3 days for which she stopped ATT 3 days back. Patient was diagnosed as ATT induced skin rash and admitted in our hospital. Dermatologist opinion was obtained for hyper pigmented rashes (Figure 1) and diagnosed as lichenoid dermatitis due to ATT. Meanwhile routine blood investigations were done. Reports showed platelets of 10000 cu.mm. In view of very low platelet counts and fever, dengue fever was suspected and dengue serology IgM positive. Thrombocytopenia was presumed to be due to dengue fever. So, patient was managed with whole blood/platelet transfusion.

After two to three days of conservative management, patient improved symptomatically. Her serial blood investigation showed gradual improvement in platelets counts from 10000 to 6 lakhs/cu.mm. Then we re-challenged ATT one by one, starting with isoniazid 150 mg 1st day followed by 300 mg 2nd day. Patient did not show any worsening of symptoms and skin rashes. On 3rd day we started 150 mg of rifampicin, within 12 hours patient developed symptoms of fever, breathlessness, chest pain and new hyper pigmented rashes (Figure 2). From 6

lakhs platelets dropped to 11,000 cu.mm. So, we concluded that skin rash and thrombocytopenia was due to rifampicin and not dengue fever where sudden precipitous fall in platelets will not occur. Later we re-challenged with other drugs except rifampicin, patient did not show any worsening of symptoms and skin rashes and her platelets improved on conservative management.

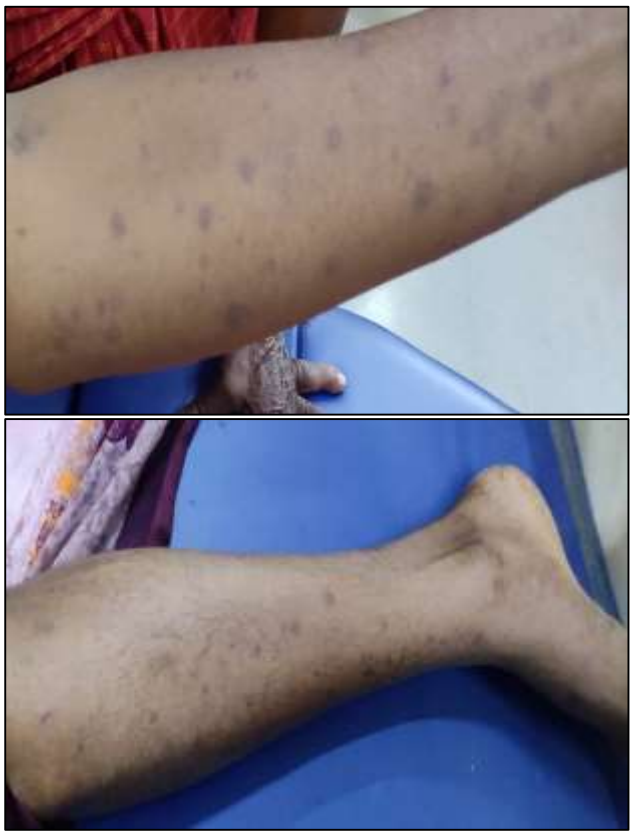

Figure 1: Multiple, hyperpigmented macules over the upper and lower limbs.

Figure 2: Hyperpigmented macules over the face.

DISCUSSION

Prevalence of adverse reactions with first line anti-tuberculous drugs varies from 8.0 to 85%. Most frequent adverse drug reactions are usually mild.[5] Rifampicin is one of the most common antitubercular drug for the causation of thrombocytopenia by antibody-mediated platelet destruction mechanism.[7-9]

Thrombocytopenia can occur with all first line antitu-bercular drugs (isoniazid, pyrazinamide, ethambutol, and rifampicin). Isoniazid causes thrombocytopenia in the form of hematological reaction where else other first line drugs like ethambutol and pyrazinamide by immunological mechanism.

other drugs apart from anti-tuberculous drugs associated with isolated thrombocytopenia are quinidine, quinine, heparin, sulfonamides, anticonvulsants (phenytoin, carbamazepine, ethosuximide, valproic acid), antibiotics (penicillins, cephalosporins, piperacillin, vancomycin, trimethoprim, levofloxacin, aztreonam, linezolid), nonsteroidal anti-inflammatory drugs (ibuprofen, aceclofenac, naproxen), abciximab, amiodarone, dexamethasone, furosemide, and gold compounds.

Diagnosis of drug-induced immune thrombocytopenia is by identifying clinical symptoms (bruising, petechiae, bleeding) and a careful evaluation of the suspected causative drug.

General laboratory investigation, such as total blood count, peripheral blood smear (to rule out pseudo thrombocytopenia) and platelet serology tests can be done. Isolated thrombocytopenia in a patient who is taking several medications is a challenging problem.

Laboratory confirmation of drug induced thrombocytopenia at time of initial presentation is usually not possible because tests for drug dependent antiplatelet antibodies facilities are not available in most clinical laboratories. So, the diagnosis of drug induced thrombocytopenia can be confirmed only by resolution of thrombocytopenia after discontinuation of suspected drug. Till now only isolated cases are reported due to rifampicin induced thrombocytopenic purpura.[6]

In our case, we diagnosed rifampicin induced thrombocytopenia clinically as we discussed above, we were not able to do antibody testing because of non-availability in our hospital.

Treatment of rifampicin-induced thrombocytopenia includes drug discontinuation, platelet transfusion, intravenous immunoglobulins, corticosteroids administration initially when drug-induced thrombocytopenia is indistinguishable from primary idiopathic thrombocytopenic purpura. Offending drug should not be reused since even a small quantity of drug can lead to immune reaction.[10]

CONCLUSION

Diagnosis of drug-induced immune thrombocytopenia is by identifying clinical symptoms and a careful evaluation

of the suspected causative drug. The physician treating tuberculosis patients must be aware of this rare life threatening complication, when we detected early, is completely reversible.

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