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A Case Report of Rifampicin Induced Thrombocytopenia.

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Case Report

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ABSTRACT

Rifampicin is an important drug in the treatment of tuberculosis (mycobacterial disease) under RNTCP (which is given intermittently according to weight). With clinical experience it has been shown that drug is well tolerated with minor side effects. But it can also cause some life threatening complications like hepatitis, hypersensitivity reaction leading to hemolysis, haemoglobinuria, hematuria, renal insufficiency and ARF, thrombocytopenia and bleeding disorder. These adverse effects are common with intermittent therapy than with daily regimen. We are reporting a case of rifampicin induced Acute thrombocytopenia in a patient who is on AKT CAT-I regimen under RNTCP

INTRODUCTION

Tuberculosis is a disease of past but still treatment of which has been a therapeutic challenge since long. Most of the antituberculosis drugs are relatively safe but serious reaction are uncommon. Thrombocytopenia is an uncommon but potentially fatal advese effect seen with certain antitubercular drugs including rifampicin [1]. Discontinuation of suspected drug leading to resolution of thrombocytopenia provides a strong evidence of drug induced thrombocytopenia. Rifampicin induced thrombocytopenia was 1st reported in 1970 [2,3]. It is usually reversible condition if detected early with high index of suspicion and appropriately. Other drugs causing thrombocytopenia are quinine, quinidine, chloroquine, sulfonamide, tolbutamide, chlorthiazide, digoxin, penicillamine, Amphoterecin B, sedatives, anticonvulsant, methyldopa and aspirin etc [4].

Patient History

25 years old male patient who is laborer by occupation came with the complaints of epistaxis and hemoptysis since 15 days. Patient is not complaining of headache, fever, joint pains, bleeding from other sites, abdominal pain and other symptoms suggestive of viral infection and malaria.

Patient was started on AKT CAT I (HRZE) under RNTCP before one and half month for sputum positive pulmonary tuberculosis. After starting of AKT patient was apparently normal but after 25 days of starting AKT, he developed epistaxis and hemoptysis but not taking any treatment for it for 15 days and taking home remedies himself for it as he suspecting these symptoms are due to heat. On examination patient was hemodynamically stable and ENT examination showed no local pathology which may lead to epistaxis. There was no history suggestive of drug intake which causes thrombocytopenia. Repeat X- ray chest PA showed no worsening compare to previous one. Lab investigations done on the day of admission is suggestive of Thrombocytopenia and are as follow : Hb - 12.3 gm/dl; TC – 6500; DC - 74/24/1/1; Platelet count -33000 per dl; ESR - 106 at the end of one hour.Prothrombin time and aPTT was normal. But his bleeding time was prolonged and clotting time was within normal limits. To rule out other causes of thrombocytopenia Dengue antibodies and ANA profile was done which were negative. We have modified the AKT Regimen and started DOTS therapy CAT I without rifampicin. On the next day Heamoptysis and

epistaxis stopped. Later after 6 days we got done his complete hemogram HB - 11.7 gm/dl TC - 7200/dl, Platelet Count - 4.19 lac /dl

There is considerable improvement in the platelet count after modifying AKT without rifampicin. So we continued AKT CAT I without rifampicin and substituted rifampicin by Inj Streptomycin.

DISCUSSION

Thrombocytopenia can occur with any of the primary antitubercular drugs. In case of Isoniazid it occurs as a hematological reaction [5]. Ethambutol [6] and Pyrozinamide also causes thrombocytopenia probably by an immunological reaction.Thrombocytopenia is a major adverse effect of several drug treatment. rifampicin has been recognized as cause of immune thrombocytopenia during intermittent high dose therapy. We characterized the antibody of a patient who presented with purpura and thrombocytopenia during treatment of TB with rifampicin. Drug dependent binding of the antibody to platelets was demonstrated by flow cytometry. In a glycoprotein specific immunoassay, the binding epitope of the IgG antibody was found in the glycoprotein Ib/Ix complex as well as monoclonal antibodies against GPIIb/IIIa , GPIa/IIa and GP IV . These findings clearly demonstrate that rifampicin induces the formation of drug dependent antibodies capable of causing thrombocytopenia. The binding site of rifampicin dependent antibody, located in the glycoprotein Ib/Ix complex seems to be a favored target for antibodies induced by different drugs [7]. Adverse reactions are uncommon on daily regimens but are relatively common with intermittent regimens [4]. These include Cutaneous Syndrome, Abdominal Syndrome, Flu like Syndrome, Respiratory syndrome, Purpura and increased transaminases. The drugs causing thrombocytopenia lead to either suppression of platelet production or immunological platelet destruction, most drugs induced thrombocytopenia by latter mechanism , the platelets are damaged by complement activation following the formation of drug antibody complex . The best proof of drug induced etiology is the prompt rise in the platelet count when suspected drug is discontinued. [8] Incidence of thrombocytopenia occurred from 1st to 14th month of therapy of rifampicin . Most workers agree that continuous treatment with rifampicin results in neutralization of any of the antibodies found , antigen-antibody complex is continuously removed without causing allergic reaction. Discontinuation of treatment allowed the sufficient quantity of platelets to built up during drug free interval so that when rifampicin is readministered an intense reaction ensues [9].

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