

CASE REPORT

A 28-year old female presented to the surgery OPD of Rajindra hospital, Patiala, with pain and lump in the right lower abdomen of 15 days duration. Pain was of moderate intensity, off and on and aggravated after meals. Patient's examination revealed a mass in the right iliac fossa of approximate size of 10 X 10cm. it was nontender, mobile, firm. Non ballotable, with irregular surface and was dull on percussion. Routine blood investigations and abdominal X-rays (erect and supine) were unremarkable. An abdominal/pelvic ultrasounds showed around 180 ml fluid collection in the right iliac fossa, and reported this as appendicular abscess. Ct scan showed a neoplastic mass 8X7.7 cm in size, irregularly marginated, involving ileocaecal junction, cecum and the proximal ascending colon causing luminal narrowing, along with some small areas of necrosis within this. In ultrasound and CT liver was normal; there were no secondaries in liver. Barium enema showed a large filling defect in proximal ascending colon and caecum with mucosal irregularities and this was also reported as growth in the cecum. Operative findings demonstrated a big mass in the cecum. A standard right hemicolectomy was done with removal of 15 cm distal ileum, and an ileotransverse was done. Postoperative period was uneventful.

The histopathology reported carcinoid tumor in the ileum. There was no penetration of the muscular or serosal layer and the resection margins were free of tumor. On immunohistochemistry, the tumor cells were positive for neuron specific enolase and focally positive for chromogranin, confirming the diagnosis of carcinoid tumor of ileum. The patient was planned to be regularly reviewed in the surgical clinic every 6 months for 3 years with urinary 5-HIAA assays.

DISCUSSION

In the present case there was no feature suggesting carcinoid syndrome. The presentation was with a palpable mass in the right iliac fossa along with CT (Fig-1) and barium finding suggesting the same; it was considered to be like a classical case of carcinoma cecum. Accordingly a right hemicolectomy was carried out. Even the gross specimen of Rt. Hemicolectomy (Fig.2) suggested a cecal tumor. Only on cut section (fig.3) it was found that there was no tumor in the lumens of



Fig.1 Gross specimen of Fig.2 Cut section of Fig.3 CT abdomen of the right hemicolectomy of specimen showing ileal pt showing cecal mass patient showing big mass carcinoid encroaching in cecum the cecal lumen

cecum although the lumen was being encroached upon by the big tumour which was actually in the terminal ileum. Histopathology and immunohistochemistry subsequently confirmed this to be carcinoid tumor.

On an exhaustive search of literature about ileal carcinoid clinically mimicking a carcinoma of the cecum, we could not find a single such reference. Hence it was considered fit for this case to be reported for its rare presentation. To conclude it is suggested that ileal carcinoid should also be kept in mind while evaluating a right iliac fossa mass, and should be considered in the differential diagnosis of cecal carcinoma as management is entirely different for both.

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Case Report

Rifampicin Induced Thrombocytopenia: A Case Report.

N. S. Neki

Department of Medicine, Govt. Medical College and Guru Nanak Dev Hospital, Amritsar, India

Abstract: Rifampicin is an essential drug in the treatment regimen for tuberculosis. It is generally well tolerated. But very rarely it can cause serious adverse reactions in the form of acute renal failure and thrombocytopenia. A case of acute thrombocytopenia occurring in a patient on Rifampicin for the treatment of pulmonary tuberculosis is being reported here.

INTRODUCTION

Rifampicin is a crucial drug as well as essential component of the treatment regimen for tuberculosis. Apart from minor adverse effects

in the form of nausea, vomiting and rash, very rarely it may cause life threatening side effects like acute renal failure and thrombocytopenia^{1, 2}.

The first case of Rifampicin induced thrombocytopenia was reported

Correspondence: Prof. N. S. Neki, Professor of Medicine, Govt. Medical College and Guru Nanak Dev Hospital, Amritsar, India
e-mail: dr nsneki_123@ yahoo.com

by Blajchman et al (1970)³. It is properly reversible if detected early.

CASE REPORT

A 45 year old male weighting 52 kg, nondiabetic, non hypertensive, chronic biri smoker since 10 years, non-vegetarian presented with complaints of cough, fever, dyspnoea and loss of appetite since 1^{1/2} mild anemia and pedal edema. There was no jaundice or clubbing. Chest examination revealed crepitations in apices of both lungs. His laboratory profile revealed Hb. 9.8gm%, TLC 16800/33³, DLC P - ₇₀₋₂₈ L₁ E₁ M₁ ESR 90 mm at the end of the first hour, Mantoux test +ve with 15 mm induration. HIV eliza was non reactive, ANA test and antibodies for dengue were also negative. Sputum smears on occasions were positive for tubercle bacilli by fluorescent microscopy, confirmed later on by positive cultures. X-ray chest showed infiltration of both lungs apices. Liver and renal profile was normal. In view of the sputum positivity, clinical findings and x-ray chest, he was put on treatment in the form of Rifampicin 600mg, isoniazid 300 mg, ethambutol 1000 mg, pyrazinamide 1500 mg daily for 2 months along with antianemic and supportive therapy. He was discharged from the hospital and advised to continue rifampicin 600 mg and isoniazid for 4 months more. But after taking the therapy for 6 months, he reported back with hemorrhagic spots on the lower extremities. On examination, there was large sized ecchymosis involving lower extremities. But he was well oriented and afebrile. Investigations revealed a platelet count of 20,000 cells/mm³ and bleeding time 6 minutes and clotting time of 12 minutes. Liver and renal profile were within normal limits. Bone n=marrow biopsy revealed megakaryocytosis. A diagnosis of acute thrombocytopenia was made. The patient was treated with I/V fluids, inj. Hydrocortisone 200 mg I/V thrice daily. Rifampicin was also stopped. The bleeding subsided with improvement in his general condition as well as improvement in the X-ray chest after continuous treatment with isoniazid, ethambutol and pyrazinamide without adverse effects. Rechallenge with 600 mg of rifampicin resulted in fall of platelets from 160000/mm³ to 10200/mm³ within 4 hours with positive antiplatelet IgG and IgM antibodies. Rifampicin was again stopped and patient continued on other antitubercular drugs. The steroid dose was gradually tapered off and shifted to oral prednisolone tablets 20 mg thrice daily which was also stopped after tapering off. He was discharged with a normal platelet count 2,30,000/mm³ and bleeding time of 2 min and clotting time of 3 minutes. However the patient was lost on follow up.

DISCUSSION

The most common causes of thrombocytopenia are vital or bacterial infections, autoimmune disease, collagen vascular disorders including SLE, Myelolymphoproliferative disorder including leukemia, anemia including aplastic anemia, HIV, massive blood transfusion, hypersplenism, alcoholism, surgery, osteopetrosis, bone cancer, pancytopenia, DIC, dengue, leishmaniasis, syphilis, myelodysplastic syndrome, prosthetic heart valve, Gaucher's disease, snake poisoning, blood poisoning, non Hodgkin's lymphoma, thrombotic thrombocytopenic purpura, eclampsia and idiopathic thrombocytopenic purpura.⁴ Important drugs⁴ which may cause thrombocytopenia include chemotherapeutic agents, sulphanamides, penicillins, cephalosporins, heparin, thiazide diuretics, pentamidine, valproic acid, PAS, cotrimoxazole, mercaptopurine,

vancomycin, methyl dopa, indomethacin, ticlopidine, captopril, quinidine, and sirolimus etc.

An extensive review of the literature revealed rarely occurring rifampicin induced thrombocytopenia^{6,9}. It has been postulated that with the daily use of Rifampicin, there is neutralization of any antibody formed and the immune complexes are continuously removed without causing any allergic reactions¹⁰. In our patient, rifampicin induced thrombocytopenia may be due to formation of immune complexes which absorb to the platelet membrane resulting in platelet damage and their rapid removal from the circulation^{3,4}. The binding epitope of the IgG antibody was found in the glycoprotein Ib/IX complex which is the target in Rifampicin induced immune thrombocytopenia^{11,12}. Our patient recovered completely on stopping the drug along with steroid therapy.

Rechallenge and de-challenge of a drug are established tools but not accepted technique for the diagnosis of adverse events.¹³ Bassi et al¹⁴ found Rifampicin dependent antibodies in 10 out of 32 patients three weeks after discounting 600 mg daily. No antibodies were found the day after treatment was stopped. Any delay in re-treatment allows a sufficient amount of antibodies to build up during the antigen free interval. Occasional patients with platelet counts <10000 to 20000/- mm³ have severe hemorrhage and may require plasmapheresis or platelets transfusion while waiting for the platelet count to rise⁴.

CONCLUSION

The presentation of this case report is to enlighten the medical fraternity about the rare occurrence of the life threatening Rifampicin induced thrombocytopenia which if detected early is completely reversible on stopping the drug. These patients must be instructed to avoid the offending drug in the future, since early minute amounts of drug are needed to set up subsequent immune reaction⁴.

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