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Rifampicin Induced Idiopathic Thrombocytopenic Purpura

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Abstract:- A rare case presentation of rifampin induced idiopathic thrombocytopenia purpura The most common cause of acute onset of thrombocytopenia in an otherwise well child is (autoimmune) idiopathic thrombocytopenic purpura (ITP). In a small number of children, estimated at 1 in 20,000, 1-4 wk after exposure to a common viral infection, an autoantibody directed against the platelet surface develops with resultant sudden onset of thrombocytopenia. A recent history of viral illness is described in 50–65% of children with ITP. The peak age is 1-4 yr, although the age ranges from early in infancy to elderly. In childhood, males and females are equally affected. ITP seems to occur more often in late winter and spring after the peak season of viral respiratory illness. The exact antigenic target for most such antibodies in most cases of childhood acute ITP remains undetermined, although in chronic ITP many patients demonstrate antibodies against αIIb-β3 and GPIb. After binding of the antibody to the platelet surface, circulating antibody-coated platelets are recognized by the Fc receptor on splenic macrophages, ingested, and destroyed. Most common viruses have been described in association with ITP, including Epstein-Barr virus (EBV; see Chapter 281) and HIV (see Chapter 302). EBV-related ITP is usually of short duration and follows the course of infectious mononucleosis. HIV-associated ITP is usually chronic. In some patients, ITP appears to arise in children infected with Helicobacter pylori or rarely following vaccines and after some drugs include valproic acid, phenytoin, carbamazepine, sulfonamides, vancomycin, and trimethoprim-sulfamethoxazole and in rare cases rifampicin.

I. INTRODUCTION

Drug induced immune mediated thrombocytopenia (DITP) can be triggered by wide range of medications. Although many cases of DITP are mild, some are characterised by life threatening bleeding symptoms. In the treatment of tuberculosis there are special therapeutic problem related to adverse effects of drug compliance to treatment and microbial resistance. Thrombocytopenia is uncommon but potentially adverse effect of certain antituberculosis drugs when the incriminating drug is taken by a susceptible individual. Here we report a case of rifampicin induced thrombocytopenic purpura in 9-year-old child.

II. CASE

9 years old female child, 4th birth by order, born of 3rd degree consanguineous marriage, presented with fever and rash. On examination heart rate-94/min, respiratory rate –20/ min, peripheral pulses well felt, blood pressure –104/64 (within centiles) with lower limb rash present. Systemic examination was within normal limit. One month back child was started on drug sensitive tuberculosis treatment in veiw of abdominal lymph node biopsy gene expert, rif ultra suggestive of mtb detected low rif resistance intermediate.

One month child was alright, tolerated antituberculosis treatment well then suddenly she developed fever and rash. She was investigated as follows;

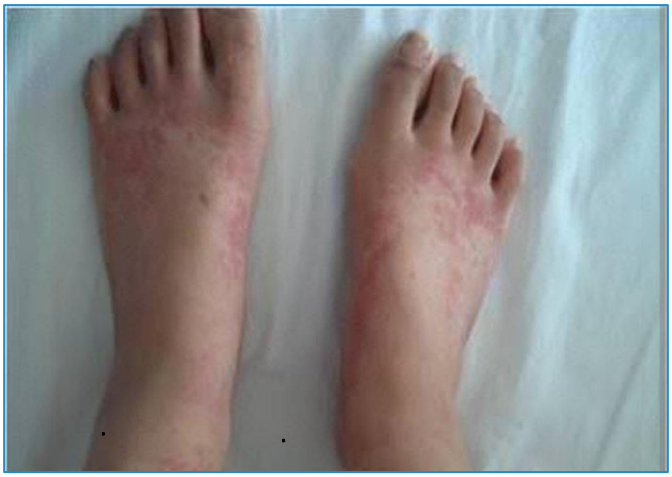

Fig 1 Purpuric rash over lower limbs

III. INVESTIGATION

Complete blood count done suggestive of hb-10.6gm%, wbc-4500/cumm, platelets-11000/microlitre. Peripheral smear examination normocytic normochromic anemia, mild Aniso poikilocytosis, platelets decreased on peripheral smear.

Bone marrow examination was done for this child it was suggestive of normal erythropoiesis, leukopoiesis and megakaryocytes

IV. TREATMENT

For this condition no therapy is needed only observation. Offending drug rifampicin has to be stopped. Treatment with IVIG particularly in children who are mucocutaneous Ly bleeding IVIG@0.8-1 g/kg for 1-2 days induces rapid rise in platelets count (20*10^9)in 95% of patients in 48hours. Another treatment can be given is corticosteroid therapy, prednisolone @ 1-4 mg/kg/day appears to induce quick rise in platelet counts short course to be given until a rise in platelet count > 20*10^9 has been achieved to avoid long term side effects of corticosteroid therapy.

We can transfuse platelets in active bleeding. Child to be evaluated further for drug resistant tuberculosis.

V. DISCUSSION

A few drugs are associated with immune thrombocytopenia thus interfering with clotting mechanism of blood and causing purpura or other abnormal bleeding such as excessive bruising or nose bleeding.

If such symptoms occur the patient should be stop taking rifampicin immediately and never be given again. If possible, platelets count should be done once rifampicin administration has been stopped, the platelet count returns to normal with two days.

No permanent damage to platelets production or function caused. Glycoprotein Ib/IX complex is the target in rifampicin induced ITP.

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