

POST-TRANSFUSION PURPURA

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Abstract: Post-transfusion purpura (PTP) presents as thrombocytopenia 7 to 10 days after first blood transfusion ; in these subjects a platelet iso-antibody is found in the plasma. A 40 year old female developed features of typical thrombocytopenic purpura following first blood transfusion for hysterectomy; anti platelet antibodies could not be done; she improved with steroids.

INTRODUCTION

Adverse reactions to transfused blood components occur despite multiple tests, injections and checks. Transfusion reactions may result from immune and non-immune mechanisms. Immune mediated reactions are often due to preformed donor or recipient antibody¹. Post-transfusion purpura predominantly involves women especially multiparous ones². Transfusion therapy even under ideal conditions, carries a significant risk of an adverse reaction³. Various forms of therapy are immunoglobulins, corticosteroids and plasmapheresis. The relevance of reporting this case is to enlighten this rare complication of blood transfusion which may sometimes be fatal.

CASE REPORT

A 40 years old female having 3 children coming from poor socio-economic status, was admitted to Rajindra Hospital; Patiala (Punjab). She had multiple fibroids of varying sizes 15-20 cm for which she had undergone hysterectomy seven days prior to admission. She was non-diabetic and non-hypertensive. There was no history of photosensitivity, joint involvement, haematuria, menorrhagia, gum bleeding, epistaxis, snake bite, bee sting, spider bite and fever especially suggestive of dengue fever. Post-operatively, two units of blood were transfused to her. Each of these was followed by fever and generalised purpuric rash. Past history in the form of trauma, dental extraction, child birth, chemotherapy or drug intake especially NSAIDs were unremarkable. On examination, she was thin pale looking having severe anaemia and facial puffiness. There was no edema, cyanosis, jaundice or lymphadenopathy. Her BP was 110/70 mmHg, respiratory rate 16/minute and pulse rate 115/minute. Chest, CVS, CNS and other systems were unremarkable. Liver and spleen were not palpable. There was diffuse purpuric rash over the lower limbs; upper limbs, chest and abdomen were spared. Her laboratory data revealed Hb 3.2 gm%; TLC 16800/mm³, DLC 66P; 34L, 0E, 0B, ESR 30 mm/hr, urine M/E NAD, B.urea 30mg%, S.creatinine 1.2 mg%, MP slide-negative; IgG and IgM antibodies for dengue, LE cell phenomenon and ANA were negative. Packed cell volume was 12; the platelet count 19000/cmm; the prothrombin time was 18 seconds (control 13 seconds); the bleeding time was 2 minutes 40 seconds. Liver function profile was normal. PBF showed microcytic hypochromic anaemia with normal reticulocyte count. Bone marrow biopsy from the iliac crest was hypercellular. X-ray chest and ultrasound abdomen were normal. Antiplatelet antibodies could not be performed due to financial constraint of the patient. She was treated with intravenous immunoglobulins, 5 grams daily for five days (the recommended dose could not be given again because of financial

constraints). Additionally, intravenous betamethasone 4 mg every 8 hourly alongwith supportive treatment in the form of haematinics was given. The general condition of the patient improved remarkably, her repeat platelet count increased to 1,25,000/c.mm.

DISCUSSION

In view of the clinical features such as age, multiparity and temporal relationship of blood transfusion and onset of purpura, a diagnosis of post-transfusion purpura was made. PTP presents as thrombocytopenia 7 to 10 days after blood transfusion. It has been reported predominantly in women as revealed by Kroll H et al² in their study of 38 patients where the mean age was 60.7 years in 80% of cases, range being 35 to 78 years and the interval between the transfusion and purpura was 2 to 14 days with a mean of 7 days. In our index case, the time interval was 7 days. The postulated mechanism of PTP is the development of platelet specific antibodies in the recipient's serum and the most frequently recognized antigen is HPA-1a found on platelet glycoprotein IIIa receptor. The delayed thrombocytopenia is due to the production of antibodies that react to both donor and recipient platelets. Additional platelet transfusions can worsen the thrombocytopenia and should be avoided^{1,4}. Post-transfusion purpura has been reported in the post-operative period⁵. Transfusion therapy even under ideal conditions, carries a significant risk of an adverse reaction³. In majority of carefully prepared and properly supervised transfusions, there are no untoward effects. However, adverse reactions occur in 5-6% of transfusions⁶ or 20% of all transfusions⁷, which may be immediate or late⁸. Thus blood transfusion carries a slight but definite risk and is not a procedure to be undertaken lightly. Current treatment is with intravenous immunoglobulins (2 gm/kg/day) for two days⁶. Other forms of therapy include corticosteroids and plasmapheresis.

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