
Case Reports

Post Transfusion Purpura in a Thalassemic Child

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Post transfusion purpura (PTP) is a rare condition and only 200 cases have been reported so far. It is commonly seen in women, with a preponderance in the sixth and seventh decade. Majority of these cases have been observed in whites and the condition is rare among Asian patients(1). It has not yet been reported in children(2). PTP is characterized by the development of thrombocytopenia associated with variable bleeding manifestations following a blood transfusion. It occurs in patients whose platelets are PL^{A1} negative and who have been sensitized to the platelet antigens following multiple blood transfusions and in women during pregnancy. We report a case of PTP in a thalassemic child.

Case Report

An 8-year-old thalassemic child on reg-

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ular blood transfusion developed petechiae, ecchymosis and epistaxis two days after receiving 2 units of packed RBCs. The physical examination revealed moderate pallor, generalized petechiae, ecchymosis and absence of jaundice. Systemic examination revealed a liver enlargement of 6 cm and splenomegaly (4 cm). Investigations showed a Hb of 7.2 g/dl, total leucocyte count of $2.8 \times 10^9/L$, platelet count of $40 \times 10^9/L$ and reticulocyte count of 3.5%. The direct and indirect Coomb's test were negative. Plasma Hb was 4 mg%. The liver and renal function tests were normal. Antiplatelet antibodies were positive (2+) by semi-immunofluorescence technique(3). The bone marrow examination revealed moderately cellular marrow with dyserythropoiesis, normal myeloid series and an increased number of megakaryocytes. A diagnosis of post transfusion purpura was made and the patient was treated with platelet transfusion and oral prednisolone (2 mg/kg/day). The platelet count increased to $100 \times 10^9/L$ over the next 2 weeks with amelioration of clinical symptoms. Prednisolone was administered for 3 weeks and then tapered over the next one week. The patient was advised to continue blood transfusion using platelet filters. Subsequent complete blood counts (Hb, TLC, platelets) of the patient on the follow up have been within the normal range.

Discussion

The bleeding episodes in PTP usually occur 5-10 days after blood transfusion and in untreated patients the period of thrombocytopenia may vary from 1 to 4 weeks to as long as 2 months. The mechanism by which PTP is produced is not yet known. The hypotheses that have been for-

warded for the destruction of autologous platelets include the production of an auto antibody of broad specificity that recognizes both transfused (PL^{A1} positive) and autologous (PL^{A1} negative) platelets, temporary production of a platelet autoantibody and immune complex mediated platelet destruction(4,5). It needs to be differentiated from hypersplenism, immune thrombocytopenic purpura and thrombotic thrombocytopenic purpura. The occurrence of thrombocytopenia following transfusions and the presence of anti-platelet antibodies with a normal or increased megakaryocytes in the bone marrow is diagnostic of PTP(4). The risk of severe systemic bleeds in these patients is high. In a review of 71 patients, 5 died from cerebral hemorrhage within the first 10 days of onset of symptoms and hence early institution of therapy has been advocated(5). Prednisolone often results in a rise in platelet counts within 7 days of therapy(6). Plasmapheresis or exchange transfusion have been found to be effective in first instances; recurrence of thrombocytopenia may require repeated exchanges. High dose intravenous immunoglobulin has been shown to be effective in patients with PTP, the mechanism of action being similar to that in autoimmune

thrombo-cytopenia(5). However, the high cost of therapy is a major draw back.

REFERENCES

1. George JN, Et-Harake MA, Aster RH. Post transfusion purpura. *In: Williams Hematology*, 5th edn. Buetler E, Lichtman MA, Collier BS, Kipps TJ New York, McGraw Hill 1995, pp 1340-1341.
2. Beardsley DS. Platelet abnormalities in infancy and childhood. *In: Hematology of Infancy and Childhood*, 4th edn. Eds. Nathan DG, Oski FA. Philadelphia, W.B. Saunders Company, 1993, p 1583.
3. von dem Borne AEG, Verheugh EWA, Oosterhof E, von Riesz E, de la Rivere AB, Engelfriet CP. A simple immunofluorescence test for detection of platelet antibodies. *Br J Hematol* 1978, 39:195-207.
4. Me Farland JG, Aster RH. Clinical significance of platelet specific antigens. *In: Immunologic Aspects of Platelet Transfusion*. Eds. McCarthy LJ, Menitove JE. Arlington VA, American Association of Blood Banks, 1985, pp 63-66.
5. Muller-Eckhardt C. Post transfusion purpura. *Br J Hematol* 1986, 64: 419-424.
6. Vogelsang G, Kickler TS, Bill WR. Post transfusion purpura. A report of five patients and a review of pathogenesis and management. *Am J Hematol* 1986, 21: 259-267.