

Post-Transfusion Purpura (PTP) n=2

25

Author: Tom Latham

Definition:

Post-transfusion purpura is defined as thrombocytopenia arising 5–12 days following transfusion of cellular blood components (red cells or platelets) associated with the presence in the patient of antibodies directed against the HPA (human platelet antigen) systems.

Two cases of confirmed PTP were reported this year. Five cases were initially reported but three were withdrawn because HPA alloantibodies had not been found. This compares with 1 confirmed case last year.

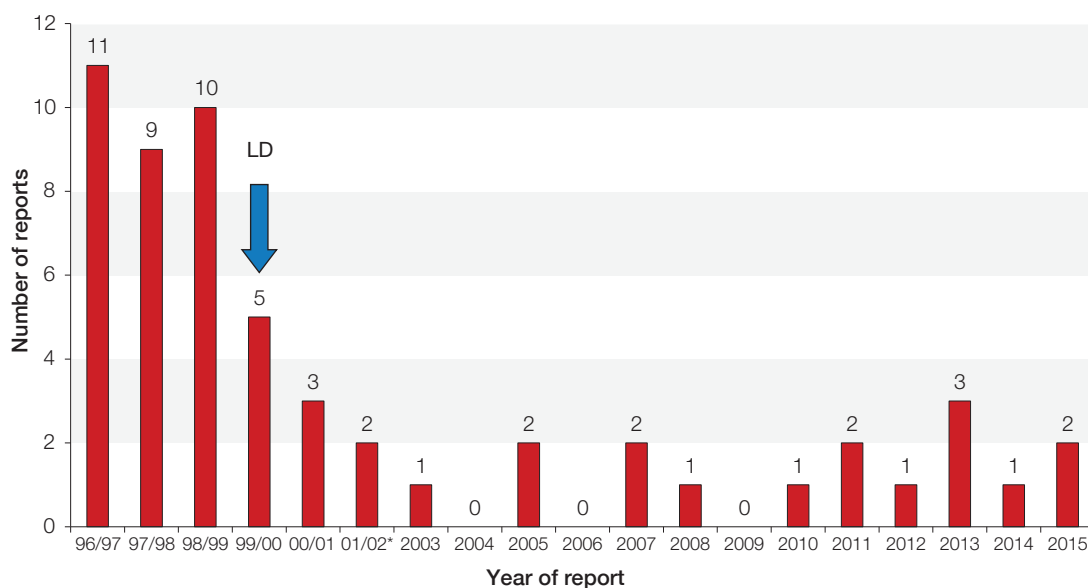


Figure 25.1:
The number of cases of PTP with confirmed HPA alloantibodies reported annually to SHOT since 1996, a total of 56 reports. Cumulative data 1996 to 2015

LD indicates the introduction of leucodepletion in 1999

Analysis of cumulative data since 1996 has shown that there have been 56 cases of serologically confirmed PTP. Almost all, 52/56 (92.9%), of these patients have been female. Alloantibodies with specificity for HPA-1a remain the most frequent cause of PTP found either alone or in combination with other antibodies in 75.0% of cases. The annual number of reported cases has decreased since the introduction of universal leucodepletion of cellular components during 1999.

Causative antibody specificity	Number of cases
HPA-1a alone	37
HPA-1a with other HPA antibodies	5
Other HPA antibodies (HPA-1b,-2b, -3a, -3b, -5a, -5b and -15a)	14
Total	56

Table 25.1:
Cumulative causative antibody specificity 1996–2015

Case History: PTP followed by immune thrombocytopenia

A 61 year old multiparous female was admitted with multiple injuries following a road traffic accident. She required several surgical interventions and a total of 5 units of red cells. Her platelet count was $195 \times 10^9/L$ on admission, $12 \times 10^9/L$ on day 10 and $5 \times 10^9/L$ on day 15. She had petechiae, bruising, wound oozing and oral blood blisters. Platelet transfusions were given without increment. Serological investigation confirmed the presence of HPA-1a alloantibodies.

She received 2g/kg of intravenous immunoglobulin (IVIg) in divided doses (day 17–21). Her platelet count remained $<10 \times 10^9/L$. Plasma exchange was performed on 4 alternate days (day 32–39) without effect. IVIg 2g/kg in divided doses (day 43–46) was repeated. Three days later her platelet count was $448 \times 10^9/L$ and she was discharged.

One month later she attended a preoperative assessment clinic. Further neurosurgery was required but deferred as her platelet count was $43 \times 10^9/L$. No further blood transfusions had been given. At this point a diagnosis of immune thrombocytopenia (ITP) was made. She commenced prednisolone 60mg/day and her platelet count recovered to $127 \times 10^9/L$ allowing surgery to be performed. HPA-1a-negative red cells were made available but were not required.

COMMENTARY

Two cases were reported this year. The first case was unusual as it was a male patient with anti-HPA-5a. This was in a chronically anaemic patient who developed purpura 6 days after transfusion.

The second case (described above) is interesting on two points (Burney et al. 2016). The majority of cases of PTP respond to IVIg but plasma exchange has been used as a second-line treatment in refractory cases. The subsequent development of steroid-responsive thrombocytopenia suggests autoimmune thrombocytopenia. It has been proposed that an autoimmune mechanism is responsible for bystander destruction of the recipients own HPA-1a-negative platelets, and the history in this case would be consistent with such a mechanism.

Advice on management of PTP is available in Practical Transfusion Medicine (Murphy et al. 2009)

Recommendations from previous years can be found in the supplementary information on the SHOT website www.shotuk.org.

References

- Murphy M. (2009) **Post-transfusion purpura**. In Murphy M, Pamphilon D, editors. Practical Transfusion Medicine. 3rd ed: Wiley-Blackwell 2009:117–21
- Burney C, Wolf J, Lucas G, et al. (2016) **Post transfusion purpura followed by immune thrombocytopenia**. Br J Haematology 173 (Suppl 1), 157