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Free Communications: Transfusion 1

1
Haemolytic transfusion reactions – how many are preventable?

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Of 2087 adverse events analysed by the Serious Hazards of Transfusion (SHOT) scheme (1996–2003), 556 (27%) were haemolytic transfusion reactions (HTRs). 303 were due to incorrect blood component transfused (IBCT): 226/303 were ABO incompatible and 77/303 caused by other red cell antibodies. A further 40 cases were reported as acute HTRs (AHTRs; i.e. occurring within 24 hours of transfusion) whilst 213 were recognised more than 24 hours after transfusion and reported as delayed HTRs (DHTR). 21/556 (4%) patients died and 58 suffered major morbidity. HTRs associated with IBCT result from clinical or laboratory errors and are all preventable. It has been assumed that other HTRs are unavoidable. Closer scrutiny reveals that this may not always be the case, though review is hampered by incomplete investigations. 9/40 AHTRs occurred in group A (8/9) or group B (1/9) patients given group O platelets. Of the 31 AHTRs related to red cell transfusion, 4 were due to errors, 9 were in patients with auto-antibodies, in only 4 of whom allo-antibodies had been adequately excuded/identified. At least 24/213 DHTRs were potentially avoidable; in 18 cases the antibody was detectable retrospectively in the pre-transfusion sample, in 6 cases the presence of a previous antibody was not communicated to the laboratory. Two patient deaths related to DHTR might have been avoided by earlier diagnosis and clinical involvement. 14% HTRs reported to SHOT would have been avoided by compliance with pre-transfusion testing guidelines and provision of group A platelets for all group A recipients.

HTRs can be clinically overlooked and inadequately investigated. National guidelines are needed for the investigation and management of HTR, with focus on the identification of underlying causes to guide the choice of future component therapy. Reference laboratories can provide valuable support in elucidating complex serological problems.

2
Effect of storage age of transfused blood on 48 hour Hb increment and recovery of 2,3 DPG in haematology patients

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Post-transfusion viability and quality of red cells is reduced by prolonged storage at 4C. The clinical and ecconomic benefit of transfusion may therefore be affected by the storage age of red cells at transfusion. We undertook a randomised, blinded, crossover study.10 patients with anaemia due to marrow failure received four consecutive top up transfusions. Two transfusions were of 'old' blood (>24 days storage) and two transfusion were of 'new' blood (<10 days storage). All units were in optimal additve solution and leucodepleted. Hb increments was measured at 48 hours. Red cell 2,3dpg was measured pre-transfusion, immediately post-transfusion, at 48 hours and 14 days. Quality of life measures were taken.

48 hour Hb increments per unit for 'old' blood (0.95 g/dl) and 'new' blood (0.96 g/dl) were not different ($p = 0.8$). 2,3DPG levels were equal pre-transfusion (17.1 vs 17.1 umol/g Hb, $p = 0.94$) and at 14 days post-transfusion (17.1 vs 16.5 umol/g Hb, $p = 0.312$) but significantly lower for 'old' blood immediately post transfusion (14.0 vs 15.9 umol/g Hb, $p = 0.0124$), and at 48 hours post transfusion (16.0 vs 17.0 umol/g Hb, $p = 0.048$). Assuming 2,3 DPG of pre-transfusion cells remained unchanged, then at 48 hours 'old' blood showed approximately 75% recovery of 2,3 DPG compared to 100% for 'new' blood. QoL measures showed no differences between old and new blood.

In summary we found no evidence of a difference in 48 hour survival between old and new blood.We found slower recovery, even at 48 hours, of 2,3 DPG in blood stored for >24 days. This may be of clinical significance especially in patients receiving massive transfusion for hemorrhage where oxygen delivery from transfused blood may be critical.

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3
Transfusion transmitted malaria – a slip through the net

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An estimated 0.5–2% western European blood donors are at risk of exposure to malaria. Blood services develop guidelines to defer potentially infected donors. We report a case of transfusion transmitted malaria where strict donor selection guidelines failed to prevent transmission.

A 50-year-old male with sickle cell disease was transfused 7 units of blood between June–September 2003. In September 2003 he was admitted with fever. Blood films revealed *P. falciparum* infection. Treatment with anti-malarials eradicated parasites but he ultimately succumbed to multi-organ failure. Blood films made 2 weeks prior to admission showed malaria, indicating that an earlier donation was the source. The patient had not left UK since immigrating from Jamaica in 1957!

All implicated donations were identified and archive samples tested for malarial antibody. 1 of the 7 samples was positive by both screening assays. The donor was a 38-year-old female, born and raised in Ghana, with no history of malaria. She visited Ghana in 1996, and had