8. Haemolytic Transfusion Reactions

Definition

Haemolytic transfusion reactions are split into two categories: acute and delayed. Acute reactions are defined as fever and other symptoms/signs of haemolysis within 24 hours of transfusion, confirmed by a fall in Hb, rise in LDH, positive DAT and positive crossmatch. Delayed reactions are defined as fever and other symptoms/signs of haemolysis more than 24 hours after transfusion, confirmed by one or more of: a fall in Hb or failure of increment, rise in bilirubin, positive DAT and positive crossmatch not detectable pre-transfusion. Simple serological reactions (development of antibody without pos DAT or evidence of haemolysis) are excluded.

Thirty-six questionnaires were received: 1 was transferred to the Acute Transfusion Reaction (ATR) section and another to the IBCT section; 1 was transferred from the ATR section, and 1 was submitted in duplicate.

This section describes the main findings from 34 completed questionnaires: 11 acute and 23 delayed reactions.

Patients

11 males (5 acute, 6 delayed) and 23 females (6 acute, 17 delayed)

Age range 11 months – 92 years

Four reports relate to patients <18 years, and 1 to a patient <12 months. Two, aged 11 months and 5 years, suffered AHTRs following ABO incompatible platelet transfusions (cases A9 and A10). Two, aged 9 and 10, both with sickle cell disease, suffered from DHTRs (cases D4 and D9).

Mortality, morbidity, and imputability

Acute reactions (11)

There was 1 death, in an infant already on ITU, thought to be unrelated to the transfusion reaction. There were no cases of serious morbidity, although 2 patients suffered haemolysis due to ABO antibodies from group O platelets. One required admission from outpatients to the ward.

One reaction was reported as possibly related, 5 as probably related and 5 as definitely related to the transfusion. In 2 cases, where an antibody to a low frequency antigen was found retrospectively, there were definite signs of an acute reaction, but no conclusive evidence of haemolysis. These cases should be interpreted with caution.

Delayed reactions (23)

There were no reports of mortality or major morbidity associated with the DHTRs, and 7 patients were reported to show no clinical signs or symptoms. Two patients were already on ITU, 4 required admission to the ward, and at least 1 required a further transfusion.

Laboratory signs of haemolysis

Many patients showed laboratory signs of haemolysis without any clinical signs being noted. The laboratory signs are often complicated by the underlying disease, and are defined as follows:

- Group 1 with Positive DAT only
- Group 2 Falling haemoglobin(↓Hb)/positive DAT/spherocytes (2 of these parameters)
- Group 3 Elevated bilirubin ± ↓Hb ±positive DAT ±spherocytes
- Group 4 As group 3 + renal impairment

4 patients were in Group 1
6 patients were in Group 2
13 patients were in Group 3

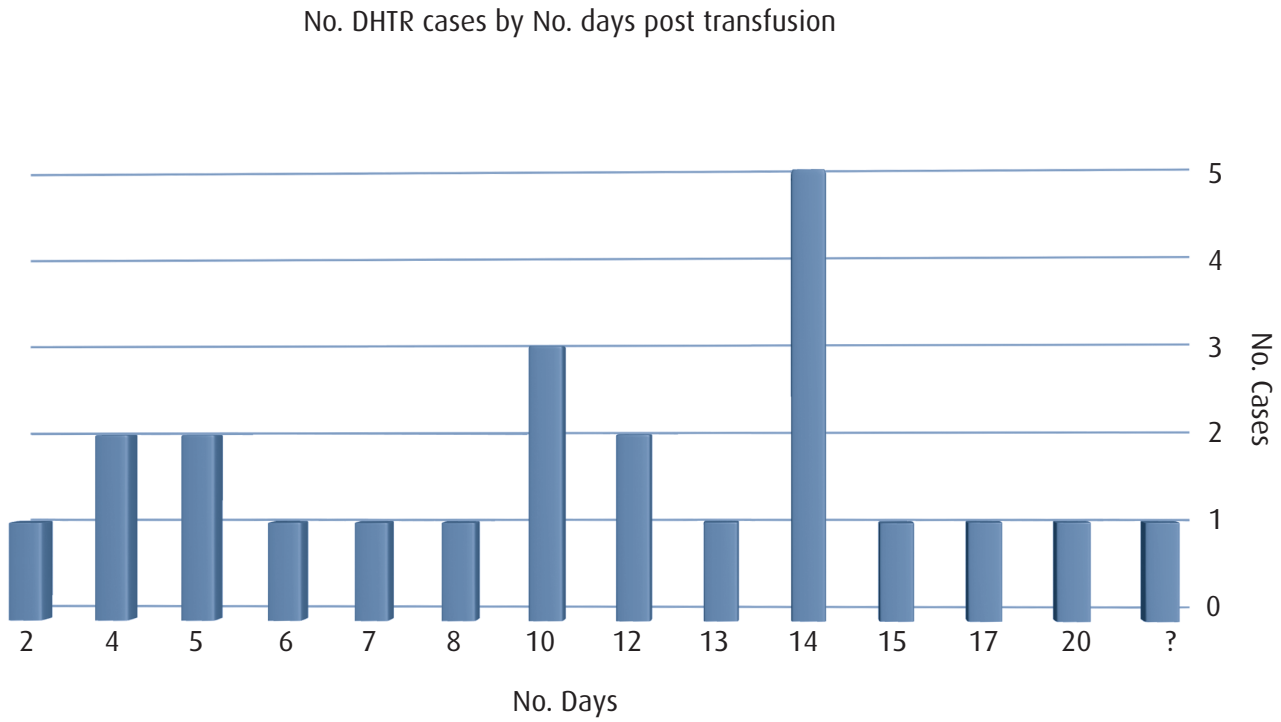
Timing of reaction in relation to transfusion

Acute

Eight reactions occurred during the transfusion, and 3 within 24 hours of the transfusion. One reaction due to a platelet transfusion was reported as occurring 4 days post-transfusion, however the patient received 2 mismatched platelet transfusions on days 1 and 3 and the bilirubin was noted to be raised on day 4 (see vignettes).

Delayed

Figure 7
Time relationship to transfusion



Median = 10 days
Range = 2 to 20 days

Figure 7 shows the reported interval in days between the implicated transfusion and signs or symptoms of a DHTR. The intervals given are not necessarily those when the signs or symptoms were first noted; in asymptomatic cases this relates to the number of days that elapsed before a repeat sample happened to be tested. There was one case where symptoms were noted within 48 hours of transfusion; however, this patient also received a previous transfusion 7 to 8 days before the onset of laboratory signs, which was more likely to have been implicated in the DHTR than the reported transfusion (case D13 – see vignette).

Serological findings

Acute reactions

Two reactions were due to anti-A from mismatched apheresis platelets, despite these being tested and found negative for high-titre anti-A and anti-B. Three occurred in patients where antibodies to low frequency antigens were found retrospectively, following a negative antibody screen and electronic issue of blood: in 1 case the antibody was identified as anti-Wr^a, but in the other 2 cases no specificity was identified, even after referral to IBGRL; in these latter 2 cases the evidence for haemolysis is low or absent. In 2 cases no antibody was detected until several days post transfusion (anti-Jk^b and anti-S+K) and it is unclear whether these were the cause of the reactions. In 1 case a cold agglutinin appeared to be responsible for the acute reaction, although further red cells antibodies developed over the next few days. In 1 case anti-Vel was apparently misidentified as a cold antibody. In 1 case several new antibodies developed, which may have been detectable pre-transfusion had a fresher sample been used. One reaction was caused by an enzyme-only anti-C. Table 28 shows details.

Table 28
Acute reactions – serology and symptoms and laboratory signs (11 cases)

Case No.	Antibody (ies) in plasma	Clinical Symptoms	Laboratory Evidence	Comments
A1	Anti-Jk^{a} + S + ?K	Fever and dark urine	Bilirubin 18 to 64	Pre-existing anti-Fy^{b} + E. Samples taken 2 days later showed additional antibodies.
A2	Anti-Wr^{a}	Fever, chills, rigors, dark urine, jaundice	Poor/absent increment in Hb following transfusion	Electronic issue.
A3	Anti-Jk^{b}	Fever, chills, rigors	Bilirubin 11 to 47 (3 days post-tx)	Anti-Jk^{b} not detectable until 7 days post reaction.
A4	Anti-Vel + Kn^{a}	Chills, rigors, dark urine, hypertension, hypothermia, increased pulse rate	Poor/absent increment in Hb	Thought to be cold antibody though DAT neg. Ab disappeared on prewarming. Anti-Vel identified retrospectively.
A5	Cold agglutinin reactive at 30^{o}C	Fever, rigors, dark urine, jaundice	DAT pos, no other lab results reported	Subsequent txs tolerated through blood warmer. Anti-C^{w} developed 6 days later, followed by anti-E.
A6	Probable unidentified antibody to low frequency antigen	Fever, rigors, back pain, hypotension, tachycardia	DAT neg pre- and post-tx, bilirubin 18 to 36	Screen negative, electronic issue, but unit found to be incompatible retrospectively by IAT.
A7	Anti-Jk^{a} + S + Kp^{a} + Lu^{a} + HI	Dyspnoea/difficulty breathing, anxiety, hypertension & drop in O_2 sats	Bilirubin 9 to 63	Pre-existing anti-E + Rg1. No retrospective testing. Pre-tx sample taken 72 hours instead of 24 hours.
A8	Anti-C (enzyme only)	Fever, rigors	Poor/absent increment in Hb, bilirubin 7 to 95	
A9	Anti-A from group O platelets	Jaundice	Bilirubin16 to 94 to 210	Platelets tested and labeled as 'high titre negative'.
A10	Anti-A from group O platelets	Fever, rigors, headache	Bilirubin 11 to 62, pos DAT	Platelets tested and labeled as 'high-titre negative'.
A11	Unidentified antibody to low frequency antigen	Fever, chills, chest pain/discomfort, rigors	DAT negative, no rise in bilirubin	Screen negative, electronic issue, but unit found to be incompatible retrospectively by IAT.

Delayed reactions

Kidd antibodies were the most commonly implicated, in 15/23 (65%) of cases, either singly or in conjunction with other specificities. Table 29 shows the specificity of new antibodies detected post-transfusion, by blood group system.

Table 29
Delayed reactions – serology and time after transfusion

Case No.	New antibody (ies) in plasma	Antibodies in Eluate	Comments	No. days post-tx
D1	Anti-Fy^{b}	No eluate performed		**20**
D2	Anti-Fy^{a} +M+?	Anti-Fy^{a}	Pre-existing anti-E.	**10**
D3	Anti-D + C	No eluate performed	Retrospective testing detected anti-D in pre-tx sample by solid phase technique only. Cold antibodies detected pre-tx.	**7**
D4	Anti-Fy^{a}+Jk^{b}+M	Anti-Fy^{a}	Pre-existing anti-C+e+K. Required admission.	**12**
D5	Anti-Fy^{a} + Jk^{a}	Anti-Fy^{a} + Jk^{a}	Pre-existing anti-E. Required further transfusion.	**10**
D6	Anti-Jk^{b}	No eluate performed	Pre-existing anti-Fy^{b}. Already on ITU.	**4**
D7	Anti-Jk^{a}	Anti-Jk^{a}		**8**
D8	Anti-Jk^{a}	No eluate performed		**10**
D9	Anti-Jk^{b}	No eluate performed	Required admission.	**14**
D10	Anti-Jk^{a} + enz-only anti-E	No eluate performed	Patient already on ITU.	**5**
D11	Anti-Jk^{b}	No eluate performed	Pre-existing anti-E. Required admission.	**13**
D12	Anti-Fy^{a} + E	Anti-Fy^{a}	Pre-existing anti-K.	**4**
D13	Anti-Jk^{a} + f + M + s	No eluate performed	Pre-existing anti-K.	**2 – 9**
D14	Anti-E	No eluate performed		**14**
D15	Anti-Jk^{a}	No eluate performed		**15**
D16	Anti-Jk^{a}	Anti-Jk^{a}		**17**
D17	Anti-Jk^{a}	No eluate performed	Pre-existing anti-Fy^{b}.	**14**
D18	Anti-S+Jk^{a}+Fy^{a}+E		Known to NBS.	
D19	Anti-Fy^{b} + c	Anti-Fy^{b} + c		**12**
D20	Anti-E	No eluate performed		**14**
D21	Anti-Jk^{a}	No antibodies detected		**6**
D22	Anti-Jk^{a}	No eluate performed	Pre-existing anti-e. Admission required.	**8**
D23	Anti-C	No eluate performed	AIHA.	**14**

Table 30
DHTRs – New specificities by blood group system

Antibody specificity by blood group system	Number of cases	Sole *new* antibody
Kidd		
Jk[a]	11	7
Jk[b]	4	3
Rh		
D	1	0
C	2	1
E	5 (1 enz only)	2
c	1	0
f	1	0
Duffy		
Fy[a]	5	0
Fy[b]	2	1
MNSs		
S	1	0
s	1	0
M	3	0

Serological Techniques Used – DHTRs only

Table 31 shows the technology used for antibody screening by IAT.

Table 31
IAT technology used for antibody screening

IAT screening technology	Number of cases	By automation
DiaMed	10	9
BioVue	2	2
Solid phase	2	2
No answer	10	10

In 13 cases plasma was used for pre-transfusion testing, and in 1 case serum (9 not stated).

Fifteen undertook an IAT crossmatch, and 6 electronic issue; 2 did not answer the question.

Use of eluates

Eight out of 23 (35%) stated that an eluate made from the patient's post-transfusion red cells was tested for antibody; 5/8 were performed in reference labs and 3 in-house. In 7 cases a specific antibody(ies) was identified. In 1 case the eluate was negative.

Retrospective testing findings

Retrospective testing of the pre-transfusion sample was undertaken in 11/23 (48%) cases; the same result was obtained in 10 of these. In 1 case (case 3), the causative antibodies were detectable retrospectively but only by a reference laboratory with a solid phase technique (Capture RRS).

Clinical management and review

Thirty-two (94%) of cases were referred to the HTC, and 22 (63%) to the Transfusion Centre Reference Laboratory. Twenty of these were reported to both.

Vignettes AHTRs

Case A1

An 82-year-old female patient with MDS required a routine top-up transfusion. The patient was found to have anti-Fy^{b}+E antibodies. Compatible antigen negative blood was issued on Thursday evening. On Friday morning the SHO reported the patient as having pyrexia and 'blood' in the urine. Transfusion reaction investigations were carried out using the pre- and post-transfusion samples. No additional antibodies/incompatibilities were observed, but the bilirubin rose from 16 to 64 mol/litre. A further request for blood and a new sample was sent on Saturday and compatible E-, Fy(b-) units were issued. The patient once again developed pyrexia and blood in the urine. New samples were obtained on Monday and referred to the BTS. Anti-Fy^{b} + E + Jk^{a} + S +??K were reported as being present in the plasma. The Hb increased from 6.9 to 9.9 following the first transfusion on Thursday but dropped back down to 6.8 by Monday. Antigen negative blood was transfused with no further problems.

Case A2

A 58-year-old male patient with MDS and no detectable red cell antibodies, received a routine top-up transfusion, issued electronically. The patient experienced fever, chills & rigors after 200 mL had been transfused. Transfusion was stopped and the unit was returned to the blood bank with fresh blood samples. Investigation revealed that although the antibody screen was negative, the implicated unit was incompatible with both pre- and post-transfusion samples, and the post-transfusion sample showed a positive DAT. Anti-Wr^{a} was identified by the BTS reference laboratory. The bilirubin rose from 18 pre-transfusion to 110 post-transfusion, and the Hb fell to below pre-transfusion levels. The patient also suffered from haemoglobinuria, but had no prolonged ill effects.

Case A3

An 86-year-old female patient with no detectable red cell antibodies received a routine 3 unit red cell transfusion for anaemia. The first 2 units were transfused uneventfully, but after 150 mL of the third the patient suffered developed a fever, chill and rigors. The transfusion was discontinued and returned to the laboratory. Retrospective testing on the pre-transfusion sample and tests on the post-transfusion samples still revealed no red cell antibodies, and the DAT was negative; however the bilirubin rose from 11 pre-transfusion to 47, 3 days post transfusion. A sample taken 7 days post transfusion revealed the presence of anti-Jk^{b}. The units transfused were not typed for Jk^{b}. The only sign of haemolysis was an increase in bilirubin 3 days post-transfusion, and this was reported as being possibly related to the transfusion.

Case A4

A 78-year-old male patient with a peri-prosthetic femoral fracture and a history of pernicious anaemia required blood for theatre. The antibody screen was positive and 2+ reactions were noted with all panel cells tested by IAT and stronger reaction by enzyme IAT; the auto was negative. The operation was postponed and samples sent to the BTS reference laboratory. A cold agglutinin was reported and blood compatible when tested strictly at 37°C was issued. The patient received a total of 6 units pre- and during surgery, with no signs of a reaction. Ten days later the patient's Hb was 6.8 and 3 more units were issued as suitable (compatible at 37°C), although the strength of reaction of the antibody had increased to 4+ by IAT. The patient suffered from chills and rigors, and a pyrexia developed during the first unit. The haematologist recommended giving the other 2 units through a blood warmer, after the patient had stabilised. The next day, unit 2 was transfused through a blood warmer, but the patient started shivering after 10 minutes; the transfusion was stopped and the bag returned to the laboratory; dark urine was also noted. Samples were referred to IBGRL and anti-Vel + anti-Kn^a were identified; the DAT was still negative. Laboratory tests showed a small rise in bilirubin, a significant increase in creatinine and a fall in haptoglobins, indicating haemolysis and deteriorating renal function.

Vel antibodies are predominantly IgM (but may also be IgG or have an IgG component) and bind complement; they are well known to cause often severe haemolytic transfusion reactions, may have a wide thermal range and may be reactive by direct agglutination. There has been a previous report of anti-Vel disappearing in vitro on pre-warming[27].

Case A5

A transfusion-dependent, 60-year-old male patient receiving a Stem Cell Transplant for AML received a routine top-up transfusion of red cells. Pre-transfusion testing showed the presence of cold agglutinins with an upper thermal range of 30°C; no antibodies were detected when the testing was undertaken at 37°C. The blood was transfused cold and he developed fever and rigors after 200 mL of the first unit, when the transfusion was immediately stopped. Dark urine and jaundice were also noted. The DAT was weakly positive (both IgG and C3 coating), and the eluate gave non-specific reactions. Subsequent transfusions were given through a blood warmer and were tolerated. Six days after the first transfusion anti-C^w developed, and this was followed by anti-E.

Case A6

A 37-year-old female patient with upper GI bleeding had 3 units of red cells issued electronically following a negative antibody screen. After 100 mL of the third unit the patient suffered from pyrexia, rigors, lumbar pain, hypotension and tachycardia. The transfusion was stopped and the bag returned to the laboratory. The antibody screen and DAT were negative on both pre- and post-transfusion samples, but the unit was incompatible by IAT on both. The bilirubin increased from 18 to 36, but no other laboratory signs of haemolysis were noted. Despite testing against several low frequency antigens no specificity was determined by the reference laboratory.

Case A7

A 44-year-old male patient with haemorrhagic effusion of his right lung, required transfusion. Anti-E plus anti-Rg^a were identified by the BTS reference laboratory and 2 units of compatible blood were transfused. Seven days later 2 further crossmatch compatible units were transfused, but the patient complained of SOB and tachycardia following completion of the second unit, and became jaundiced the next day. A 3-day-old sample was used for the second set of serology and no retrospective testing was possible, since there was no sample left. A post-transfusion sample revealed the presence of anti-S + Jk^a +Kp^a +Lu^a + HI, in addition to the anti-E and –Rg^a. The DAT was positive (C3 coating only), but no antibodies were detected in an eluate.

This reaction might have been prevented had a fresher sample been used as recommended in the BCSH guidelines[28].

Case A8

A multiply transfused 68-year-old female patient developed fever and rigors during a routine transfusion for anaemia. Laboratory testing showed a rise in bilirubin from 7 to 95, and haemoglobinuria was noted. Pre- and post-transfusion samples gave a negative antibody screen, but an enzyme only anti-C was identified by the reference laboratory, in the post-, but not the pre-transfusion sample.

Case A9

An 11-month-old male A D negative infant undergoing cardiac surgery required a platelet transfusion. The patient was given group B negative apheresis platelets on day 0, and group O apheresis platelets on days 1 and 3 (tested negative for high-titre haemolysins), as group A platelets were unavailable. On day 4, laboratory tests showed evidence of haemolysis – raised bilirubin (16 pre, 94 day 1 and 210 day 4); the DAT became positive and anti-A was eluted from the red cells. The patient was already on ITU and died from underlying disease.

Case A10

A 5-year-old female, group AB D positive child with ALL required platelet and red cell transfusion. One unit of group O apheresis (tested negative for high-titre haemolysins) was transfused followed by 1 unit of group A red cells. One hour (68 mL) into the red cell transfusion, the patient developed a fever, rigors and headache. The DAT was positive, the bilirubin rose from 11 to 62, and the Hb fell from 6.7 to 6.1.

Case A11

Following a post-partum haemorrhage, a 36-year-old patient required 4 units of red cells, which were issued electronically. On transfusing the first unit, the patient experienced pyrexia, headaches, shivering, flushing, rigors and tachycardia. The transfusion was stopped. By this point the patient had received all of the unit. The pack, transfusion set, first sample of urine and a new sample for antibody testing were sent to the transfusion department. On retesting of samples, the pre- and post-antibody screens were still negative but the implicated unit was incompatible by IAT on both pre and post samples. This was confirmed by IBGRL to be an antibody to a low frequency antigen, but no specificity was determined. There were no clinical signs or laboratory evidence of haemolysis – the DAT was negative and the bilirubin level remained normal.

Learning points

- If used inappropriately, prewarming techniques can reduce or remove the activity of clinically significant antibodies, and should only be used where cold autoantibodies or specific cold alloantibodies have been positively identified.
- Blood warmers should be used where high-thermal range cold agglutinins are present.
- Some transfusion reactions may be prevented in recently transfused patients, by using fresher blood samples, taken in line with BCSH recommendations[28].
- Group O apheresis platelets can cause acute haemolytic reactions even when tested and found negative for high-titre haemolysins. They should only be used for non-group O patients (particularly paediatric patients) as a last resort, and should not be kept by hospitals as stock.

Vignettes DHTRs

Case D3

An 88-year-old female patient with a GI bleed and no known history of transfusion required red cells during on-call hours. The patient was grouped as O D negative with a positive antibody screen. Cold agglutinins were suspected and the sample was referred to the reference laboratory, where the presence of cold agglutinins only was confirmed. The patient was transfused with 2 units of O D negative blood, followed by 5 units of O D positive blood. Seven days later a further transfusion was required and anti- D+C was identified in the plasma. The DAT was negative and tests indicated that no D positive cells remained in the circulation; the bilirubin was slightly elevated (39) and the Hb dropped from 13.0 immediately post transfusion, to 7.3, initially attributed entirely to continued bleeding. Retrospective testing of the pre-transfusion sample confirmed no alloantibodies by routine techniques (including enzymes), but anti-D+C were identified using a Capture R solid-phase technique.

Case D13

A 68-year-old female patient with known anti-K was transfused with 9 units of K- blood for a massive GI bleed. Six days later, a further 3 units of K- blood were transfused, using a fresh sample for crossmatching. Another 2 days later the patient was noted to be jaundiced with a bilirubin of 130 (pre-transfusion bilirubin not known). The Hb fell to 6.2, from a pre-transfusion Hb of 8.2. Anti-K+Jk^a+s+f+M were identified by the reference laboratory; the DAT was positive with both IgG and complement coating but elution was not performed.

This was reported as a DHTR to the second transfusion 2 days previously. However, it is more likely due to the first transfusion 8 days earlier or to both, since the Hb dropped to below pre-transfusion levels.

Case D18

An 87-year-old female patient with unknown transfusion history required transfusion for anaemia. Anti-K was identified in the pre-transfusion sample and K- blood was transfused. An unspecified number of days later a further sample was tested and multiple antibodies were found. Samples were sent to the BTS reference laboratory where the patient was previously known to have anti-S+Jk^a+Fy^a+E. Fortunately, although this patient had a positive DAT, there were no apparent signs of a DHTR.

Case D23

A 69-year-old male patient with Waldenstrom's macroglobulinaemia and warm AIHA was admitted with an Hb of 7.3g and 2 units of red cells were requested. No underlying alloantibodies were demonstrated and 2 units of blood compatible with the absorbed plasma were transfused. Seventeen days later the patient presented with a Hb of 5.3 and a raised bilirubin of 63 (no pre-transfusion bilirubin available). Allo-anti-C was identified by a reference centre, but no elution was performed.

It is not clear whether the haemolysis was due to the transfusion or the underlying AIHA. However, the laboratory has since implemented a policy to give Rh matched red cells to patients with AIHA.

Learning points

- It is advisable to provide Rh and K matched blood to patients with AIHA, in line with BCSH guidelines[28].

COMMENTARY

- An example of anti-Vel was mistaken for a cold antibody and removed by pre-warming[27].
- Three cases of acute reaction occurred where antibodies to low frequency antigens were undetected in pre-transfusion testing (antigens absent from the screening cells and blood issued electronically). Although reported as AHTRs, only the case involving anti-Wr^a demonstrated a clear haemolytic reaction. Patients may have symptoms of an acute reaction following transfusion of a unit to which they have an antibody, but unless there is evidence of haemolysis, these two facts are not necessarily related.
- Anti-Wr^a is a relatively common antibody and may be naturally occurring; although this antibody can cause severe HTRs and HDN, many examples are of no clinical significance.
- Group O apheresis platelets, which test negative for high-titre haemolysins may cause haemolytic reactions particularly in paediatric patients.
- In all cases but one (where an answer was given) plasma rather than serum was used for both pre- and post-transfusion investigations. It is known that weak complement binding antibodies, e.g. some examples of anti-Kidd, may be missed when using plasma, unless more sensitive techniques are used, e.g. enzyme IAT.
- Only 35% of investigations included testing an eluate made from the patient's red cells. Where a mixture of antibodies is present, an eluate may help to distinguish which specificity(ies) is more likely to be implicated in a haemolytic reaction. Furthermore, the implicated antibody may only be present in an eluate. Identification of all specificities present is essential if further haemolytic reactions are to be prevented.
- As in previous years, communication problems have contributed to DHTRs, where information about previously known antibodies has not been available at the time of a subsequent transfusion.

RECOMMENDATIONS

- Group identical platelets should be selected whenever possible, with group O being the last choice for non group O recipients. Where children are concerned the Amendments and Corrections to the BCSH guidelines 'Transfusion Guidelines for Neonates and Older Children', should be followed[29,30].

  Action: Hospital transfusion laboratories

- Investigation of a suspected HTR should include retesting of the pre-transfusion sample (where still available) by different or more sensitive techniques. Consideration should also be given to requesting clotted samples for investigation of suspected HTRs and using polyspecific AHG. Where hospital resources are limited, this will require referral to a reference centre.

  Action: Hospital transfusion laboratories

Carried over from 2005:

- All cases of suspected AHTR and DHTR should be appropriately investigated, and ideally referred to a reference laboratory. Referring hospitals should make it clear to reference laboratories that they are investigating a DHTR to ensure that timely, appropriate tests are undertaken. Clinical details should be completed on the request forms and the donation numbers of the units transfused should be included, so that their phenotype can be determined.

  Action: Hospital transfusion laboratories and blood services reference laboratories

- Reference laboratories should ensure that investigation of DHTRs includes testing an eluate made from the patient's red cells when the DAT is positive.

  Action: Blood services reference laboratories

- In line with recommendations made in the BCSH Guidelines consideration should be given to issuing antibody cards or similar information to all patients with clinically significant red cell antibodies[28]. These should be accompanied by patient information leaflets, explaining the significance of the antibody and impressing that the card should be shown in the event of a hospital admission or being crossmatched for surgery. Laboratories should be informed when patients carrying antibody cards are admitted.

  Action: The CMO's NBTC and its counterparts in Scotland, Wales, and Northern Ireland

- There is a need for a review, co-ordinated by a professional national body, of how long specimens should be kept post-transfusion. The review needs to consider the relative risks and benefits of storing specimens beyond the time that they are suitable for use in further crossmatching tests.

  Action: BBTS and BCSH