11. Haemolytic Transfusion Reactions (HTR)

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 positive DAT or evidence of haemolysis) are excluded.

DATA SUMMARY

Total number of cases	**55**

Implicated Components	
Red cells	**51**
FFP	**0**
Platelets	**4**
Other *(specify)*	**0**
Unknown	

Mortality / morbidity	
Deaths due to transfusion	**0**
Deaths in which reaction was implicated	**1**
Major morbidity	**6**

Gender	
Male	**24**
Female	**31**
Unknown	

Age	
16 years+ to 18 years	**1**
1 year+ to 16 years	**3**
28 days+ to 1 year	**0**
Birth to 28 days	**0**
Total	**4**

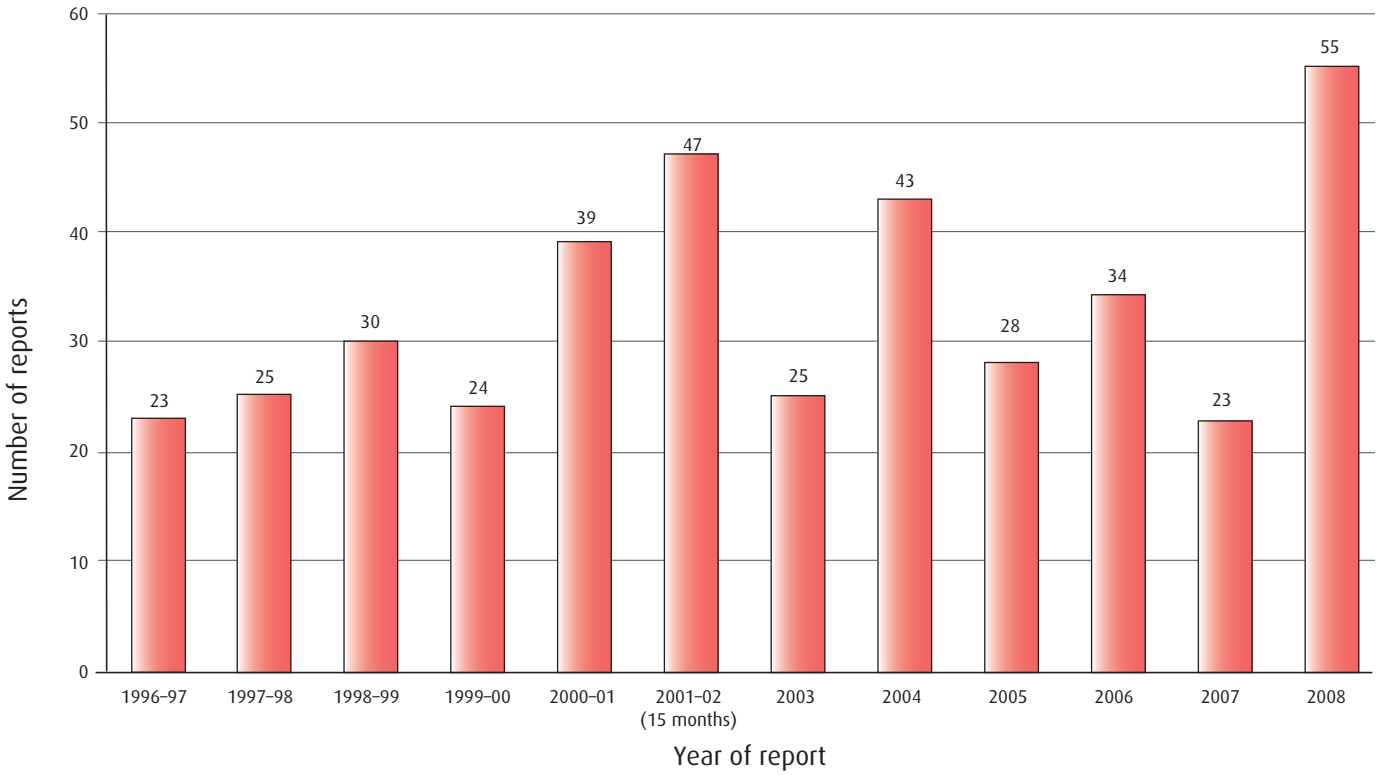
Emergency vs. routine and core hours vs. out of core hours	
Emergency	**10**
Routine	**41**
Not known	**4**
In core hours	
Out of core hours	
Not known/applicable	**55**

Where transfusion took place	
ED	
Theatre	
ITU/NNU/HDU/Recovery	
Wards	
Community	
Other	
Not known	**55**

In total, 63 questionnaires were received; on review 2 were found to be duplicate reports from 2 different hospitals, 3 were transferred out to the IBCT section, 2 were transferred out to the ATR section, 2 were withdrawn altogether, and 1 was transferred in from the ATR section.

This section describes the main findings from 55 completed questionnaires: 9 acute and 46 delayed reactions.

Figure 12
Number of cases of HTR reviewed since 1996



Patients

There were 24 male and 31 female patients, with an age range from 14 months to 94 years.

Of these, 4 patients were under 18 years of age: a 14-month-old and a 7-year-old suffered acute reactions to group O platelets, an 8-year-old patient with sickle cell disease suffered an episode of hyperhaemolysis, and a 16-year-old with sickle cell disease suffered a delayed reaction due to anti-S.

Mortality, morbidity, and imputability

Acute haemolytic transfusion reactions (AHTR) *n* = 9

There were no deaths caused, or contributed to, by these transfusion reactions. The patients in cases A8 and A9 suffered reactions with imputabilities of 2 and 3 respectively. Both patients died subsequently of their underlying disease.

There were 2 cases of major morbidity from AHTR. Case A2 (imputability 2) related to an incompatible red cell transfusion and Case A7 (imputability 1) to an ABO-incompatible platelet transfusion. Both patients required ITU admission but made a full recovery.

The remaining 5 cases of AHTR suffered minor or no morbidity. Two were reported as definitely related to the transfusion (imputability 3) and 3 probably related (imputability 2).

Delayed haemolytic transfusion reactions (DHTR) *n* = 46

There was one death in this group (Case D33) in which the reaction (imputability 2) possibly contributed to death. This patient suffered from CCF, hepatorenal failure and sepsis, was already extremely unwell when the reaction occurred, and died 2 weeks later. The reporter thought that the DHTR possibly contributed to the death.

There were 4 cases of major morbidity. The patient in Case D23 has required long-term dialysis since the DHTR; although no antibodies have been detected, this was reported as probably related to the transfusion (imputability 2). There were 2 other cases of major morbidity in which the patient showed signs of deteriorating renal function, but did not require dialysis (cases D9 and D45); 1 was reported as definitely related (imputability 3) and 1 probably related (imputability 2) to

the transfusion. In Case D16, the patient suffered a dramatic drop in Hb 5–6 days post transfusion, requiring emergency admission and a 4 unit transfusion.

Of the remaining 41 cases, 26 suffered minor morbidity and 15 had no clinical signs or symptoms, with the only laboratory signs being a positive DAT and development of antibody.

Timing of reaction in relation to transfusion

AHTR

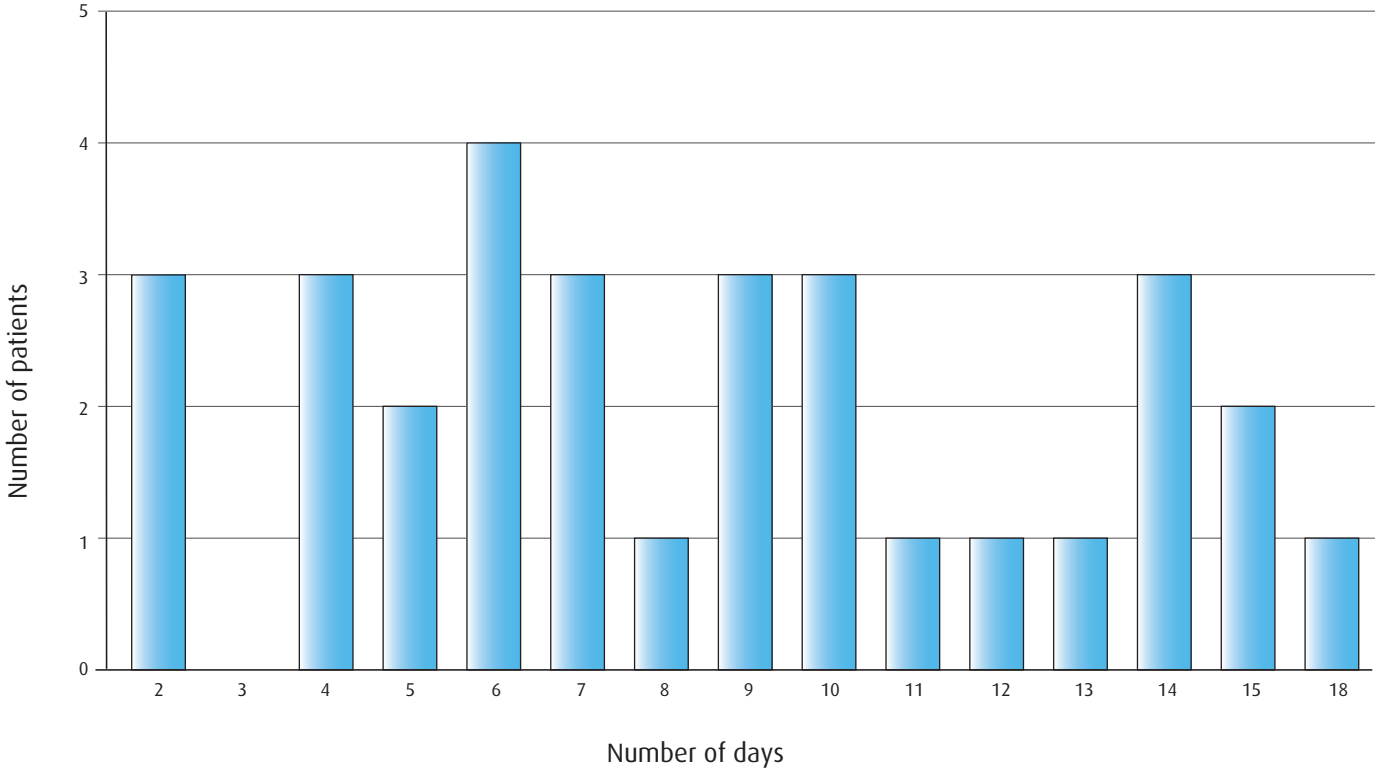
In all, 6 reactions occurred during the transfusion and 3 within 24 hours of transfusion (2 were reported as 'next day').

DHTR

Figure 13 shows the reported interval in days between the implicated transfusion and clinical signs or symptoms of a DHTR. The median is 8 days, and the range 2–18 days.

New antibodies were found between 5 and 38 days after transfusion in the 15 asymptomatic cases.

Figure 13
Number of days between transfusion and reaction



Serological findings

AHTR *n* = 9

There were 4 reactions due to anti-A from mismatched platelets. These were 3 apheresis donations, of which 2 were HLA matched, and 1 derived from pooled buffy coats. All platelets were labelled negative for high-titre anti-A and anti-B. However, the pooled buffy coat donation was retrospectively found positive for a high-titre IgG anti-A.

In 2 cases, patients were transfused with incompatible blood before antibody identification was complete: 1 had a serious reaction requiring ITU admission (Case 2) caused by anti-Jk^{a}+Fy^{b} and 1 had a milder reaction due to anti-Fy^{a}.

There was 1 reaction in a patient where anti-Jk^{a} was, retrospectively, weakly detectable in the pre-transfusion sample using a different IAT technique (A1).

In 2 cases (A3 and A4) there were laboratory signs of haemolysis although no antibody was identified until 2 and 6 days after the transfusion respectively. In the latter case the Jk(a+) unit was weakly incompatible on retrospective testing using a different IAT technique.

Table 45
AHTR cases

Case number	Antibody (ies) in plasma	Clinical symptoms	Laboratory evidence	Comments
1	Jk^a	Fever, tachycardia, abdominal pain	0.5 g/dL↓ in Hb; pos DAT; anti-Jk^a eluted	Antibody only detectable pre-transfusion by LISS tube, not DiaMed.
2	Jk^a + Fy^b	Fever, rigors	bilirubin↑; Hb↓; pos DAT	Blood given urgently before IBGRL confirmed Ab ID. Initially thought to be anti-JMH; ITU admission required.
3	Jk^a	Dyspnoea, chest and lumbar pain	Pos DAT; anti-Jk^a eluted 6 days later	Implicated unit Jk(a+), but antibody not identified until 6 days post Rx. Unit retrospectively weakly positive by DiaMed but negative by LISS tube.
4	c+E + non-specific	Fever, rigors	Bilirubin↑ neg DAT; no antibodies detected for 2 days	Implicated unit was c positive.
5	Fy^a	Fever; dyspnoea, jaundice	Bilirubin↑; Hb↓; pos DAT; no eluate performed	Antibody screen weakly positive but blood required urgently.
6	A	Fever, rigors, dyspnoea, hypotension, jaundice	Bilirubin↑; no Hb increment; LDH↑	Required ITU admission.
7	A	None reported	Bilirubin↑; Hb↓; pos DAT; anti-A eluted	High-titre IgG anti-A in buffy coat group O platelets; death unrelated.
8	A	None reported	Bilirubin↑; Hb↓; pos DAT; anti-A eluted	HLA matched apheresis plts were not IgG anti-A negative. 14-month-old died, death unrelated.
9	A	Rigors, pyrexia	pos DAT; anti-A eluted	Platelets negative for high-titre anti-A.

AHTR cases

Group A patients receiving Group O platelets

Case A6

A 71-year-old male with Hodgkin's disease received 2 units of group A red cells and 1 unit of group O apheresis platelets. On completion of the platelets he became dyspnoeic and wheezy, then hypotensive, pyrexial and jaundiced. The Hb remained at 8.2g/dL despite a 2 unit red cell transfusion, and the bilirubin increased from 28 to 237 µmol/L. The patient became increasingly unwell, and was admitted to ITU. In-house testing showed the DAT to be positive pre and post transfusion, but this was not confirmed by the reference centre and the eluate was non-reactive. The donor was confirmed as negative for high-titre anti-A. Although no anti-A was evident in the plasma or eluate, there is clear evidence of a haemolytic episode.

Case A7

A 7-year-old boy on PICU who was group A received a pool of group O buffy coat derived platelets in an emergency. The platelets were labelled as high-titre negative. There were no immediate signs of an acute transfusion reaction, but the following day the bilirubin rose from 23 to 88 µmol/L and the DAT was positive. Anti-A was present in the plasma and was eluted from the patient's red cells, necessitating the use of group O red cells for future transfusion. The patient died from his underlying disease.

Case A8

A 14-month-old baby with leukaemia, group A received 2 apheresis doses of group O HLA matched platelets. The next day the baby suffered a sharp and unexpected fall in Hb and his bilirubin showed a marked rise. The DAT was positive and anti-A was detected in both plasma and eluate. The donor was retrospectively shown to have a high-titre IgG anti-A1.

Case A9

A 76-year-old female patient with MDS who was group A received 1 donation of group O HLA matched platelets followed by 2 units of group A red cells. During the second unit of red cells the patient developed a fever and rigors and the transfusion was discontinued. The DAT became positive and anti-A was detected in the plasma and eluate. The platelets were negative for high-titre anti-A and anti-B.

Learning point

- Group O platelets can cause acute haemolytic reactions even when tested and labelled negative for high-titre haemolysins. They should not be kept by hospitals as stock, and should only be used for non-group O patients as a last resort. This applies especially to paediatric patients.

Severe acute reaction due to misidentified antibody

Case A2

A 62-year-old male patient with diabetes, renal impairment, dehydration and sepsis, required blood to cover a below knee amputation. All panel cells were positive by IAT and surgery was postponed while samples were referred to the red cell reference laboratory. The DAT was strongly positive (IgG coating), as were all cells tested, even after alloadsorption. A panel treated with 2-aminoethylisothiouronium bromide (AET) was negative, and a specificity of anti-JMH was tentatively reported, which is unlikely to be of clinical significance. Four units were issued by the reference laboratory, 2 of which were transfused. Eight days later a further 4 units were issued and samples were requested for referral to IBGRL and for molecular genotyping. During transfusion of the first unit, the patient developed rigors and pyrexia and the transfusion was stopped. The Hb fell from 7.3 to 4.3g/dL and there was a rise in bilirubin. The patient was admitted to ITU. Further investigation revealed anti-Jk^a and anti-Fy^b, in addition to autoantibody, all confirmed by IBGRL. The eluate was positive but gave non-specific reactions. The implicated unit was Jk(a+). An additional 8 units of Jk(a-) blood were transfused and the patient made a full recovery.

It is possible that the patient was suffering a DHTR from the first transfusion in addition to the AHTR, perhaps explaining the fall in Hb to below pretransfusion levels.

Learning point

- If there is any doubt about the specificity of an alloantibody in the presence of a positive DAT (or transfused cells), referral for genotyping will allow for the selection of antigen matched blood, while further serological testing is performed.

DHTR *n* = 46

Kidd (Jk) antibodies were the most common, implicated in 32/46 cases (70%) either singly or in combination with other specificities; 48% of all new antibodies had Kidd specificity; and 69% of all new sole antibodies were Kidd.

Table 46 shows details of the serology, laboratory signs and time interval by case, and Table 47 shows the specificity of new antibodies detected post transfusion, by blood group system.

Table 46
Serology, laboratory signs and timing of reaction

Case number	New antibody (ies) in plasma	Antibodies in eluate	Comments	Days post transfusion
1	c+E+Jk^{a}	E	Anti-K previously identified; No signs or symptoms	8-16
2	c+E+Jk^{a}	Jk^{a}	Anti-Jk^{a} not identified in plasma; Hb↓	14
3	Jk^{a}	Jk^{a}	Anti-E pre-transfusion; Hb↓ bilirubin↑	9
4	Jk^{a}	Jk^{a}	Hb↓ bilirubin↑	14
5	Jk^{b}	No eluate done	Hb↓ bilirubin↑	5
6	S	No eluate done	Hb↓↓ bilirubin↑↑	15
7	C+?K	One cell pos – specificity not determined	Hb↓ bilirubin↑	10
8	Jk^{b}+Lu^{a}	Jk^{b}	Hb↓ bilirubin↑, but liver disease and bleeding	10 days
9	Jk^{a}	Jk^{a}	Anti-D and anti-K previously identified; Hb↓ bilirubin↑ creatinine↑ haemoglobinuria	13
10	k	k	Hb↓ bilirubin↑	10
11	M	No eluate done	Hb↓ bilirubin↑	12
12	Fy^{a}	Fy^{a}	No signs or symptoms	14
13	None		Post-transfusion hyperhaemolysis in patient with sickle cell disease bilirubin↑ Hb↓ haemoglobinuria	7
14	E	No specificity	Hb↓	15
15	Jk^{a} + cold autoantibody	No eluate done	Anti-D+C pre-transfusion Hb↓ bilirubin↑	2
16	Jk^{a}+M+E	Jk^{a}, M, E	Hb↓↓ bilirubin↑↑	5
17	Jk^{a}	Jk^{a}	No signs or symptoms	13
18	Jk^{b}	No eluate done	No signs or symptoms	10
19	E+Jk^{b}	Negative	Antibodies only detected with polybrene; Hb↓ bilirubin↑	14
20	c+E	c	Hb↓	2-6
21	C+S	Eluate negative	Hb↓ bilirubin↑	9
22	E+Fy^{a}+Jk^{b}	Fy^{a}	No signs or symptoms	11
23	None	Eluate negative	DAT pos pre & post C3 only; Hb↓ bilirubin↑	6
24	Jk^{b}	Not stated	Hb↓ bilirubin↑	2
25	E	E + wk reactions ?anti-c	No signs or symptoms	38
26	Jk^{a}	No eluate done	Hb↓ bilirubin↑↑	4
27	Jk^{b}	Jk^{b}	Hb↓ bilirubin↑	4
28	K	No eluate done	No signs or symptoms	38
29	K+Jk^{a}	Jk^{a}	Hb↓ bilirubin↑↑	2
30	Jk^{a}	Jk^{a}	Hb↓ bilirubin↑	6
31	Jk^{a}	No eluate done	Hb↓ bilirubin↑	6

32	Jk^a	No eluate done	No signs or symptoms	8
33	Jk^b	No eluate done	Anti-c pre-transfusion Hb↓ bilirubin↑ creatinine↑	4
34	Jk^a	No eluate done	No signs or symptoms	5
35	$S+Fy^a+Fy^3+Jk^b$	Non-reactive	Antibodies known elsewhere but not detectable pre-transfusion Hb↓↓	14
36	Anti-N + autoantibody	Negative	Autoantibody likely responsible for haemolysis Hb↓ bilirubin↑	8
37	Jk^a	No eluate done	No signs or symptoms	7
38	$c+Jk^a+Fy^a$	c, Fy^a	Hb↓↓ bilirubin↑↑	11
39	Jk^a+f	Jk^a	Hb↓↓ bilirubin↑	9
40	Jk^a+C^w	No eluate done	No signs or symptoms. Antibodies known elsewhere but not detectable pre-transfusion	3
41	E	E	No signs or symptoms	14
42	Jk^a	No eluate done	No signs or symptoms	10
43	Jk^a	Jk^a	No signs or symptoms	Not stated
44	Jk^a	Jk^a	Hb↓	7
45	Jk^b+E	Eluate negative	Hb↓ bilirubin↑↑ creatinine↑	7
46	Jk^a	Jk^a	No signs or symptoms	Not stated

Table 47
DHTRs – new specificities by blood group system

Antibody specificity by blood group system	Number of cases	Sole *new* antibody
Kidd		
Jk^a	22	15
Jk^b	10	5
Rh		
C	2	0
E	10	3
c	4	0
f	1	0
C^w	1	0
Kell		
k	3	1
k	1	1
Duffy		
Fy^a	4	1
Fy^3	1	
MNSs		
S	3	1
M	2	1
N*	1	1
Other		
Lu^a	1	0

** Patient also had autoantibodies*

Serological Techniques Used – DHTRs only

Table 48
IAT technology used for antibody screening

IAT screening technology	Number of cases*	By automation
DiaMed	22	19
BioVue	10	10
CRRS	3	3

** 11 reports had no answer in this section; in 9 of the 11 cases, there were pre-existing antibodies*

- This spread of IAT technology matches that used overall by UK laboratories (data from UK NEQAS questionnaire September 2008).
- In 35 cases plasma was stated as being used for pre-transfusion testing, while 11 reports had no answer to the question.
- 24 undertook an IAT crossmatch (8 of these had a positive antibody screen), 2 an immediate spin, and 16 electronic issue; 4 reports had no answer to the question.

Use of eluates

In 29/46 (63%) of cases an eluate was made from the patient's post-transfusion red cells and tested for antibody. This has increased from 60% in 2007 and 35% in 2006. Of these 29 eluates, 23 were performed in reference laboratories and 6 in-house. In 21 cases a specific antibody(ies) was identified.

Retrospective testing findings

Retrospective testing of the pre-transfusion sample was undertaken in house in 18 cases (39%): the same results were obtained in all 18 cases. However, in only 8 of these cases was repeat testing confirmed by a reference centre or by a different technique

DHTR cases

DHTR possibly contributed to death

Case D33

73-year-old male patient with CCF, peripheral vascular disease and hepatorenal dysfunction was transfused 2 units of red cells for anaemia. Six days later anti-c was identified and 2 units of c negative blood were transfused. After 4 days the patient became jaundiced with laboratory signs of haemolysis: raised bilirubin, rising creatinine (already high), falling Hb and reduced haptoglobins. The DAT was positive, anti-Jk^{b} was identified in addition to anti-c, but no eluate was performed. The patient died 2 weeks later. Although already very sick, it was thought that the DHTR might have contributed to this man's death.

Long term morbidity but no detectable antibody

Case D23

A 41-year-old female patient with SLE and factor XI deficiency received a 3 unit transfusion for anaemia. She presented 6 days later in the ED with aches and pains, dark urine and jaundice. Bilirubin was raised, and Hb had dropped from 11.3 to 9.0 g/dL. The creatinine was significantly raised and the patient is now continuing dialysis awaiting a renal transplant. The DAT was positive pre and post transfusion (complement coating only), but the eluate was non-reactive. Despite testing by the hospital and the reference laboratory with a variety of techniques on plasma and serum (with the addition of fresh complement), including enzyme IAT and PEG IAT, no antibodies were detected. Although the serology was all negative, there was no other explanation for the haemolytic episode, which was thought probably to be due to the transfusion. The patient has been fully phenotyped and if further transfusion is required Jk(a-), S- K- blood will be provided.

Major morbidity with full recovery

Case D9

A 67-year-old female patient with known anti-D+K was transfused 3 units post THR and discharged with a Hb of 11.4 g/dL. She was readmitted 13 days later with chills, nausea, vomiting, jaundice and dark urine. Hb had fallen to 8.7g/dL, bilirubin and LDH were raised and the urea and creatinine were both elevated, indicating a serious haemolytic reaction. Anti-Jk^{a} was identified in the plasma and eluate.

Case D45

A 94-year-old male patient with acute coronary syndrome received 4 units of red cells for anaemia. Seven days later the patient was admitted with jaundice and haematuria. Bilirubin and creatinine were raised and the Hb dropped to its pre-transfusion level. Anti-Jk^{b} + E were identified, and although the DAT was positive (complement coating only) the eluate was non-reactive.

Case D16

A 21-year-old female patient received 8 units of red cells over a 2 day period for postoperative bleeding, and was discharged with a Hb of 14.3 g/dL. Five days later she required emergency admission with severe jaundice, dark urine and a Hb of 9.5g/dL. By the following day the Hb had fallen to 6.3g/dL, necessitating an urgent 4 unit transfusion. The DAT was positive and anti-Jk^{a}+M+E were detected in both the plasma and the eluate. On investigation there was documentation in the notes of anti-Jk^{a} from previous testing at a different hospital. It is not known whether the patient produced an antibody card.

Antibody only detectable using non-standard technique

Case D19

A 27-year-old male patient with NHL and an Hb of 7.5g/dL received 2 units of red cells and was discharged with a Hb of 9.5g/dL. He was readmitted 18 days later weak and lethargic with difficulty breathing. His Hb had dropped to 5.7g/dL and the bilirubin was raised. The DAT was positive (IgG coating) but the eluate was non-reactive. No antibodies were detected, so samples were referred to the NBS reference centre, where anti-Jk^{b}+E were identified by polybrene technique only.

There is clearly some evidence of haemolysis in this case; however, the Hb dropped to lower than the pre-transfusion Hb, and it is not clear how much of the picture is due to DHTR and how much to the underlying disease. There was no pre-transfusion sample available to confirm the presence or absence of these weak antibodies or a positive DAT prior to the transfusion.

Known antibodies cause severe delayed reaction

Case D35

A 35-year-old female patient with sickle cell disease received a 7 unit exchange transfusion of Rh and K matched blood in preparation for surgery. Two weeks later the patient presented to another hospital with an apparent sickle cell crisis, urinary tract infection and fever. The initial Hb was 7.9 g/dL but this dropped to 3.7g/dL over the next 4 days. The antibody screen was positive and this hospital had a record of previous multiple antibodies for the patient. Investigation by the reference laboratory revealed anti-S+Fy^{a}+Fy3+Jk^{b}, with a positive DAT (both IgG and complement) but a non-reactive eluate. Despite the massive drop in Hb the patient remained reasonably well and was treated with IVIg as no compatible blood was available. The HbS level was at 89% on readmission, indicating haemolysis of all the transfused red cells, but additional hyperhaemolysis cannot be excluded.

Learning points

- If used appropriately, antibody cards can prevent DHTRs. However, patients need to understand the importance of this information, and need to be encouraged to show them to hospital staff on admission, and certainly if a transfusion is required.
- Evidence in the notes of previous antibodies should be made available to the transfusion laboratory.
- 'New' patients with sickle cell disease are likely to have been tested and possibly transfused elsewhere. They are at higher than average risk of developing red cell antibodies and where possible hospitals should actively seek a transfusion and antibody history.

COMMENTARY

Group O platelets, both apheresis and pooled, that tested negative for high-titre haemolysins have once again caused haemolytic reactions. In 1 case retrospective testing showed that one of the donors was in fact high-titre positive for IgG anti-A. There have been 13 previous reports to SHOT (at least 8/13 in children under 18) of group O platelets causing ATRs in group A or B recipients, with a further 4 (2 in children) this year. The majority have therefore been in paediatric patients.

The BCSH guidelines[20] recommend the use of ABO identical platelets as the first choice, with provision of group O platelets to non-group O recipients only as the last choice for paediatric patients.[21] There is evidence that major ABO mismatched platelets (e.g. A to B) have a reduced transfusion efficacy,[22,23] which must be balanced against the risk of AHTR with a minor mismatch (e.g. O to A). When a decision needs to be made regarding the group of platelets to transfuse, liaison between the laboratory and the clinical team looking after the patient is essential, so that conflicting special requirements (e.g. CMV negative, HLA matched) can be carefully prioritised to minimise risk to the patient, and maximise transfusion benefit.

Finally, the risk of AHTR in these unavoidable situations might be reduced if the cut off for 'high-titre' anti-A along with anti-B for identifying high-risk group O donors was set at a lower limit than the current 1/128.

This year 63% of investigations included testing an eluate made from the patient's red cells, compared with 60% last year and only 35% the year before. Where a mixture of antibodies is present an eluate may help to distinguish which specificity(ies) is the 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.

Kidd (Jk) antibodies were implicated in 70% of DHTRs and also in 60% of non-ABO AHTRs. Acute reactions occurred in 2 patients where the Kidd antibody was retrospectively weakly detectable in the pre-transfusion sample by a different technique (one by DiaMed but not LISS tube, and the other vice versa). This demonstrates that no single technique will detect all weak antibodies and highlights the difficulties with detection of Kidd antibodies.

Two serious DHTRs occurred in patients known to have antibodies, but which were undetectable at the time and not known about in the transfusion laboratory providing the blood. In one case the evidence was in the patient's notes, and in the other case a patient with sickle cell disease was well known to another hospital usually treating the patient and to the blood service reference laboratory.

RECOMMENDATIONS

New recommendations this year:

- Blood services should review the critical titre of 1/128 for screening high-titre anti-A and anti-B, and consider whether donations should be screened for IgG in addition to IgM antibodies.

  Action: UK Blood Services

- Prior to transfusion, an antibody history and a transfusion history should be actively sought for previously unknown patients with sickle cell disease. This must include contacting the local blood service reference laboratory as well as any other hospitals the patient has attended.

  Action: Hospital; blood transfusion laboratories

- A national register of patients with antibodies, linked between the red cell reference laboratories, should be considered.

  Action: UK Blood Services

Previous recommendations still relevant

Previous recommendations remain relevant and the first 2 are particularly pertinent to this year's cases.

Year first made	Recommendation	Target	Progress
2000–01	Group identical platelets should be selected whenever possible, with group O being the last choice for non-group O recipients. Blood services should stock higher levels of non-group O platelets.	**Hospital blood transfusion laboratories, Blood Service issue departments and the NBTC Transfusion Laboratory Managers Working Group**	'Amendments and Corrections' to the BCSH guidelines 'Transfusion Guidelines for neonates and older children' clarifies these recommendations.
2001–02	Consideration should be given to issuing antibody cards or similar information to all patients with clinically significant red cell antibodies. 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.	**The CMO's NBTC and its counterparts in Scotland, Wales, and Northern Ireland**	This recommendation was made in the BCSH Guidelines (BCSH, 2004).
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 an HTR 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.	**Hospital blood transfusion laboratories, Blood Service reference laboratories and the NBTC Transfusion Laboratory Managers Working Group**	BCSH guidelines for investigation and management of transfusion reactions are in progress.

2005	Reference laboratories should ensure that investigation of DHTRs includes testing an eluate made from the patient's red cells when the DAT is positive.	**Blood Service reference laboratories**	Eluates were undertaken in 63% of cases this year and 60% last year compared with 35% in 2006 and 50% in 2005. This appears to be progress.
2001–02	Investigation of a suspected HTR should include re-testing 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. These actions may involve referral to a reference centre.	**Hospital blood transfusion laboratories and the NBTC Transfusion Laboratory Managers Working Group**	This will be addressed in the revision of the BCSH Guidelines for Compatibility Testing, which are in progress.
2005	Pre-transfusion testing on patients who have been recently transfused and require further transfusion should be carried out in accordance with BCSH Guidelines relating to the timing of the samples	**Hospital blood transfusion laboratories and the NBTC Transfusion Laboratory Managers Working Group**	
2003	There is a need for a review, coordinated 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.	**BBTS and BCSH**	Revised BCSH guidelines for compatibility procedures in blood transfusion laboratories are in progress.