

THE AUTHORISATION OF BLOOD COMPONENTS



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1. INTRODUCTION

This workbook has been developed with the aim of aiding nurses to become proficient in the authorisation of blood and blood components within the realms of their specialty. It is acknowledged that there is significant variation in the use of transfusions in the medical and surgical arena.

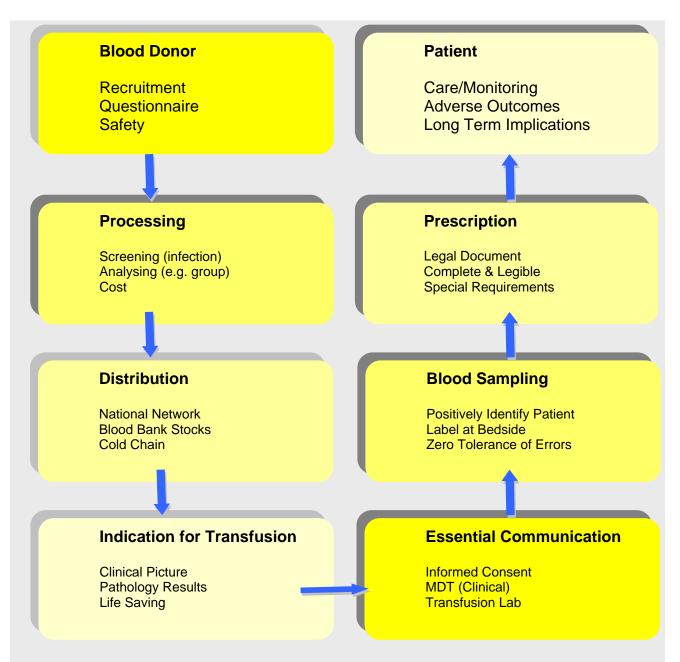
There are two clear advantages of only prescribing blood components when they are absolutely necessary:

- Donated blood is a limited resource and blood supplies may be reduced due to the impact of vCJD.
- There are clear and potentially fatal risks to patients receiving transfusions.

Given the complex nature of differences between individual patients and their particular clinical condition, no guidelines can be absolute. This workbook highlights the main aspects and considerations for safely prescribing blood components, which are generally applicable. This workbook does not cover specific transfusion requirements or thresholds and triggers for every circumstance; the authors acknowledge that there are specific conditions which require precise management.



2. OVERVIEW OF ENTIRE TRANSFUSION PROCESS: SALIENT POINTS





3. ESSENTIAL COMMUNICATION

In order to transfuse patients safely, there are a number of key stakeholders with whom the decision to transfuse must be communicated and where an effective working relationship must be nurtured.

3.1. The Patient

Following a recent consultation on consent for transfusion by the Advisory Committee on the Safety of Blood, Tissues and Organs (SaBTO), there is now a recommendation by the Department of Health (2011) for all NHS Trusts to ensure patients provide their informed consent wherever possible prior to the transfusion of blood components. These DoH recommendations stipulate that there must be an unequivocal clinical indication for transfusion, that information of sufficient quality must be provided to allow patients to give fully informed consent and that retrospective information on transfusion must be given to those patients transfused unknowingly (during their current admission). DoH recommendations also talk about consent being standardised (with a checklist of key points to discuss) and that this is documented. Also particularly pertinent to haematology patients, that transfusion consent is modified i.e. repeated 'regularly' - to cover aspects of increased risks in those multiple transfused patients i.e. iron overload, increased risk of TTI, platelet refractoriness etc and to establish if the patient is still willing to receive transfusion.



Confusion has arisen because written consent is not legally required, but this does not detract from the underlying principle. It is strongly recommended that verbal consent be recorded in the clinical notes (along with the indication) not least because in an increasingly litigious society, how can you prove two years or more down the line that you fulfilled your responsibilities Gaining informed consent is of course a skill in itself, which requires practice, reflection on past experience, and a balance should be struck. The process can be aided by the use of patient information leaflets, these are produced free of charge by NHS Blood and Transplant.

Consent should be taken by the consultant reviewing the patient and recorded in the relevant section of the patient assessment tool. It is the responsibility of the prescribing medical practitioner/nurse authoriser or registered health care professional with the knowledge to do so, to discuss with the patient the need for transfusion and the potential benefits and risks. The patient may not see a medical practitioner during the transfusion episode therefore it is recommended that nursing staff pay particular attention to NMC guidance on consent.



3.2. The Clinical Team

It is vital that having correctly and appropriately prescribed a blood transfusion, the clinical team looking after the patient are made aware of the decision, and able to carry through the process in as safe and efficient a manner as possible. Transfusion Practitioners strive to train nursing/midwifery staff to question the indication for a transfusion, not least because they will have to address subsequent anxiety in patients (and/or their relatives) long after the authoriser has left the arena. It is also vital that there is a sensible prioritisation of the transfusion along with other essential care, in particular effective communication should avoid unnecessary overnight transfusions which audit has shown are all too common and SHOT data has proved are inherently less safe.

3.3. Nurse Authoriser

The Registered Nurse must understand that by agreeing to act as an approved authoriser of blood transfusion under the framework documents they are extending their role and job description. They must perform this role in accordance to the NMC code of Conduct, performance and ethics standards. The protocol must be followed to ensure vicarious liability from their employing Trust. See appendix 4 of the 'Nurse Authorisation' framework document.

3.4. Transfusion Laboratory

Although less visible, the transfusion laboratory Biomedical Scientists (BMS) are integral to safe transfusions and have expert knowledge that is an invaluable resource available to you. For less clear cut cases speaking to a BMS over the phone is essential so that the true clinical picture can be understood by the laboratory. The Chief Medical Officer (HSC 2007) has stated that blood transfusion laboratory staff should be empowered to question the indicative parameters for transfusion, which at the very least should be clear on the request form.

You have a responsibility to inform the laboratory on every request form of:

- Special requirements e.g. CMV Negative or Irradiated
- Previous transfusions
- Previous transfusion reactions
- Previous pregnancies
- Any known antibodies

The BMS can in turn advise you when to repeat blood samples for compatibility testing because recent transfusions can produce new antibodies which if undetected could lead to a haemolytic transfusion reaction.

In summary, safe transfusion is a collaborative process between all of these key stakeholders.



4. TRANSFUSION CHART

There is wide variation between different healthcare organisations as to where blood components are authorised, for example:

- Integrated care pathways
- Separate blood prescription charts
- Medicines prescription chart / fluid chart

There are essential factors required in all of these approaches including:

- Patient minimum dataset (which has been verified)
- Clear legible handwriting
- Type and quantity of components required
- Duration
- Special Requirements
- Additional medications e.g. diuretics / antihistamines
- Sign and PRINT your name, to provide a clear audit trail

NB. Red cell transfusion must be completed within 4 hours of leaving the blood fridge. If it takes 30 minutes for the collection and bedside checking, that only leaves 3½ hours for transfusion time. Patients who have a compromised cardio-vascular system can be prescribed diuretics, commonly 20mg Furosemide P.O. given with the second unit of a two unit transfusion, dose and route can vary depending on the indication. Patients who have previously had mild allergic reactions (more common with platelets) may be prescribed an antihistamine prior to transfusion.

Blood transfusion can be described as a "liquid transplant" it is a human tissue transplant that must be treated in practice with the appropriate gravitas.

5. RISKS OF TRANSFUSION



Since 1996 evidence for the risks of transfusion have been illustrated by the data from Serious Hazards of Transfusion (SHOT) scheme. SHOT is highly respected and valued in transfusion medicine worldwide and is recognized as one of the first haemovigilance schemes. Consistently the data has demonstrated by some considerable margin that human error is the biggest risk in transfusion.

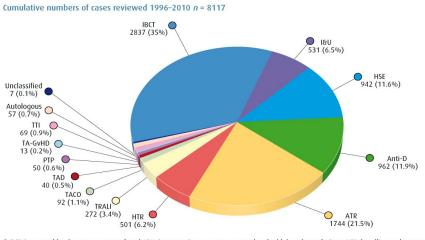
The overview of the transfusion process (<u>section 2</u>) highlights just how many steps there are between blood donor and recipient and that many disciplines are involved. A chain is only as strong as its weakest link, errors can occur at any point. The Blood Safety and Quality Regulations 2005 (SI/50/2005) have strictly addressed the matter of the production and distribution of blood components along the principles of "Good Manufacturing Practice", which are effectively quality assurance mechanisms.



It is now a statutory requirement to investigate and report all errors occurring within the blood transfusion services and hospital laboratories, including "Corrective and Preventative Actions (CAPA)" taken. Although there has been a steep learning curve for both national transfusion services and hospital laboratories, these must be seen as positive steps to improve patient safety.

Currently the practice in clinical areas is less controlled and both national audits, and clinical incidents, demonstrate areas of weakness. The biggest risk to patients is healthcare professionals' complacency. A good example of this is patient identification and labeling of blood samples. The task itself is not complicated and as a result, dare we say, practitioners do not perform this process with the necessary respect for safe protocol. Simple mislabeling of blood samples has led to patients being transfused the wrong ABO blood group with devastating consequences.

6. SHOT CASES



^a IBCT, incorrect blood component transfused; I&U, inappropriate, unnecessary and under/delayed transfusions; HSE, handling and storage errors; ATR, acute transfusion reactions; HTR, haemolytic transfusion reactions; TRALI, transfusion-related acute lung injury; TACO, transfusion-associated direutalory overlaad; TAD, transfusion-associated dyspneoa; PTP, post-transfusion purpura; TA-GvHD, transfusionassociated graft versus host disease; TTI, transfusion-transmitted infection.

In broad figures in 2010 over 99% of all blood components transfused occurred without incident although we cannot be sure that every event has been reported. The overall numbers of adverse events are low, and so the chances of these types of reactions equally are low however, we can never know which patients will suffer these outcomes. There is additionally the unknown element of undiscovered pathogens for which transfusion can be the medium of entry. These arguments combine to form the principle that patients should only be transfused when it is absolutely necessary. Furthermore, during and after a transfusion, patients should be closely monitored.

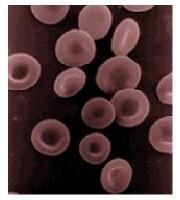


7. RED CELLS

7.1. Basic facts

Red cells are also known as erythrocytes and are the most common type of blood cell in the body, they make up a quarter of the cells in the body. They contain Haemoglobin molecules which transport oxygen. See section 7.4

Red cells are made in the bone marrow, 2.4 million are produced per second and they circulate for about 100–120 days in the body before their components are recycled by macrophages. Each circulation takes about 20 seconds.



In humans, mature red blood cells are flexible biconcave

disks that lack a cell nucleus. This aids transport of oxygen through the microcirculation and tiny arterioles.

7.2. Erythropoiesis

This is the process by which red blood cells are produced. It is stimulated by decreased O_2 in circulation, which is detected by the kidneys, which then secrete the hormone erythropoietin. This hormone activates increased erythropoiesis ultimately producing red blood cells. This usually occurs within the bone marrow; however, in humans with certain diseases, erythropoiesis also occurs outside the bone marrow, within the spleen or liver. This is termed extra medullary erythropoiesis.

7.3. Why transfuse red cells?

The aim of transfusing red cells is to maintain sufficient oxygen delivery to the tissues - the oxygen delivery must exceed oxygen consumption. Red cells then complete the cycle of respiration by transporting carbon dioxide to the lungs for expiration. Red cells must **never** be used for volume replacement.

7.4. How is oxygen (O_2) carried in the blood?

Oxygen is carried in the blood in two forms:



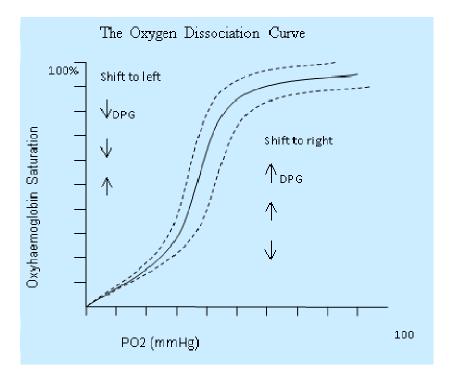
- 1. Dissolved in plasma in a very small amount which could never sustain tissues and another more effective method of carriage is needed.
- 2. The Haemoglobin (Hb) molecule found in red cells has 4 binding sites for O_2 , the Hb is usually 97-98% saturated, hence the vast majority of O_2 is transported by this mechanism.

In health there is vast excess in capacity to deliver O₂ to tissues.



7.5. The oxygen dissociation curve and oxygen delivery

The oxygen dissociation curve shows the saturation of Hb at various partial pressures of oxygen in the body. At high pO_2 (i.e. in the lungs), oxygen binds to Hb (to form oxyhaemoglobin). If we follow the curve, when the blood passes through the heart and arteries the pO_2 drops but the Hb does not lose much oxygen. However as the blood reaches the deoxygenated tissues there is a large change in the % saturation of Hb, consequently the oxyhaemoglobin releases the oxygen.



When the Hb molecule is fully saturated with O_2 it does not easily give it up, however as each O_2 molecule breaks free from the Hb molecule binding site the next O_2 molecule is released more willingly.

There are several factors that can move the curve to the left or right (denoted by the dashed line in the diagram)

As tissues become more active the rate of respiration increases, more carbon dioxide is released the dissociation curve shifts to the right and Hb becomes more efficient at releasing oxygen.

2, 3-Diphosphoglycerate (2, 3-DPG)

2, 3-DPG is present in the red blood cell and is an important adaptive mechanism, because the production increases for several conditions in the presence of diminished peripheral tissue O_2 availability, such as hypoxaemia, chronic lung disease, anaemia, and congestive heart failure, among others. It will improve oxygen delivery but this takes approximately 50 days to occur.



It is an important adaptation because it means that chronic anaemia can be well tolerated because a 50% decrease in oxygen carrying capacity is accompanied by only a 25% decrease in oxygen availability. And that the reserve of oxygen-carrying capacity is such that cardiac output at rest does not usually increase until the Hb falls below 7 g/dl.

High levels of 2, 3-DPG shift the oxygen dissociation curve to the right, while low levels of 2, 3-DPG cause a leftward shift, seen in states such as septic shock.

There are many other mechanisms to compensate for low Hb levels and to maintain oxygen delivery. Tissues may increase blood flow by recruiting more capillaries or vasodilatation. Tissues may also increase oxygen extraction ratios. In acute anaemia clinicians may underestimate the effectiveness of such adaptive mechanisms, leading them to towards over-reliance on Hb levels and transfusions.

The cardiac output is probably a bigger player in the delivery of O_2 to the tissues than the O_2 content. This is because the cardiac output can almost instantaneously respond to a fall in PaO₂ saturation of Hb. Moderate hypoxaemia leads to an increase in the cardiac output. On the other hand, compensation for a fall in cardiac output is slow and weak (it takes time to increase Hb production and the O_2 dissociation curve is flat – it can't become anymore saturated). Nevertheless, in the clinical setting, it is often easier to increase the Hb or the fraction of inspired oxygen than to increase the cardiac output.

8. CHRONIC ANAEMIA

There are a number of differences between acute and chronic anaemia and it is worth considering these when deciding whether or not to transfuse a patient.

8.1. Hypovolaemia

Hypovolaemia is at least initially the major problem in acute blood loss; however the patient with chronic anaemia is normovolaemic or even hypervolaemic.

8.2. Rate of change and ability to compensate

In cases of chronic anaemia, the fact that the changes have happened more slowly brings two advantages; firstly there is time to consider the risks and benefits and involve the patient in the decision to transfuse; secondly, an increase in red cell 2, 3-DPG leads to a shift in the oxygen dissociation curve and improved delivery of oxygen to tissues. Consider transfusing one unit at a time with assessment after each unit to avoid unnecessary transfusion and donor exposure.



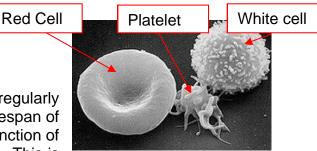
8.3. The alternatives (dependent on the reason for anaemia)

Management will differ depending on the cause. Transfusions should not be given where there are effective alternatives, e.g. treatment of iron, folate or B12 deficiency, unless the anaemia is life threatening. This will have been considered by the senior medical staff managing the patients care. If there is evidence that the patient would benefit from alternative treatments please refer to the patient's medical team prior to authorising a transfusion.

9. PLATELETS

9.1. Basic facts

Platelets, or thrombocytes, are small, irregularly shaped clear cell fragments. The average lifespan of a platelet is normally just 5 to 9 days. The function of platelets is the maintenance of haemostasis. This is



achieved primarily by the formation of thrombi when damage to the endothelium of blood vessels occurs.

9.2. Maintenance of haemostasis

When the endothelial layer is injured, collagen, von Willebrand Factor and tissue factor from the sub-endothelium is exposed to the bloodstream. When the platelets contact these, they are activated to become aggregated (e.g. to clump together). The blood clot is only a temporary solution to stop bleeding; vessel repair is therefore needed. The aggregated platelets help this process by secreting chemicals that promote the invasion of fibroblasts from surrounding connective tissue into the wounded area to completely heal the wound or form a scar. The obstructing clot is slowly dissolved by the fibrinolytic enzyme, plasmin, and the platelets are cleared by phagocytosis.

9.3. Indications for platelet transfusion

Platelet transfusions are indicated for the prevention and treatment of haemorrhage in patients with thrombocytopenia or platelet function defects. Platelets are not indicated in all causes of thrombocytopenia and may indeed be contraindicated in certain conditions (BCSH 2003).

It is therefore important that the cause of the thrombocytopenia is known before transfusion. As with all transfusions the decision to transfuse must involve assessment of the risks versus expected benefits.



10. TRANSFUSION REACTIONS

10.1. Acute Haemolytic Transfusion Reaction (AHTR)

AHTRs can be immune-mediated or non-immune-mediated. Immune-mediated haemolytic transfusion reactions happen when transfused red cells of the incorrect ABO group react with the patient's anti-A or anti-B immunoglobulin M (IgM) causing an acute severe clinical reaction (symptoms may occur after just a few mls of blood have been transfused).

AHTR symptoms are produced due to destruction of red cells within the bloodstream (intravascular haemolysis). This occurs because the antigen antibody reaction activates the entire complement cascade, which results in holes punched in the red cells causing them to rupture.

Transfusion of a small volume of ABO-incompatible plasma is unlikely to cause haemolysis in the recipient. However, infusing a unit of plasma (or cryoprecipitate or platelet concentrate) containing a potent anti A or anti B antibody may haemolyse the recipient's red cells. Group O plasma and platelet components should only be given to group O recipients (Transfusion Medicine 2007).

Administration of ABO incompatible blood may start with the patient feeling anxious commonly referred to as a "sense of impending doom".

N.B. You must be aware that an AHTR can present very subtly with little more than a fever. Refer to your trust guidelines in treating transfusion reactions.

These reactions are usually due to human error at some point in the transfusion process. It is therefore imperative to take steps immediately to protect any patients that may be involved in the error.

10.2. Bacterial Contamination

Common signs and symptoms of a bacterial contamination are fever, chills, rigors, vomiting tachycardia and hyper or hypotension, and collapse, usually during the transfusion but can occur a few hours later. The shock that occurs is likely to be due to bacterial toxins, although immune reactions that take place between naturally occurring antibodies and the bacteria are also a factor. The signs and symptoms may be similar to acute haemolytic transfusion reactions or severe acute allergic reactions.

These patients should be managed initially as for an acute haemolytic reaction, but also take blood cultures and seek expert microbiology advice and give combination of antibiotics that will be active against the range of bacteria that may be involved.



10.3. Transfusion Related Acute Lung Injury (TRALI)

The mechanism by which TRALI occurs is not yet fully understood. In most cases leucocyte antibodies have been found in the plasma of the donor that are thought to act against antigens on the recipient's leucocytes. It is thought that these antibodies activate the leucocytes, which are then sequestered in the lungs where they cause pulmonary capillary endothelial cell damage that allows fluid to leak into the alveoli (Dry 1999). However, in some cases, no antibody is found in the donor so TRALI may occur as part of a "two-hit" process. The first hit is a serious illness in the patient. The second hit is the transfusion of a blood component containing either:

- Leucocyte antibody directed against the patient's leucocytes
- Biologically active lipids that develop in stored blood, capable of causing the release of inflammatory mediators that damage the pulmonary vascular endothelium
- Or both leucocyte antibody and biologically active lipids



Symptoms include fever and chills, hypotension, breathlessness and non-productive cough, usually occurring within a few hours of the transfusion. The chest X-ray characteristically shows bilateral nodular infiltrates in a batwing pattern, typical of acute respiratory distress syndrome.

The management will involve seeking urgent critical care and haematology advice. Treatment is the same as adult respiratory distress syndrome from any cause. Diuretics should be avoided.

All cases of TRALI must be reported to MHRA and SHOT. The blood service will contact the donor and, if appropriate, remove them from the donor panel.

10.4. Transfusion Associated Circulatory Overload (TACO)

TACO is a condition that is probably under diagnosed, which may cause acute left ventricular failure (LVF) with dyspnoea, tachypnoea, non-productive cough, raised JVP, basal lung crackles, frothy pink sputum, hypertension and tachycardia. Patients are at risk if over-transfused or transfused too quickly.

10.5. Allergic Reactions

10.5.1. Anaphylaxis

Anaphylactic reactions are IgE mediated. They are likely to occur in IgA deficient patients with anti-IgA. These patients can have severe anaphylaxis when exposed to donor plasma containing IgA.

Signs and symptoms include hypotension, bronchospasm, periorbital and laryngeal oedema, vomiting, erythema, urticaria and conjunctivitis, dyspnoea, chest pain, abdominal pain and nausea.



10.5.2. Less severe allergic reactions

These reactions involving urticaria or itching within minutes of starting a transfusion are quite common.

If a patient has suffered an allergic reaction in the past an antihistamine may be given before starting the transfusion. If a reaction occurs in spite of this precaution saline-washed blood components should be considered.

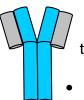
10.6. Febrile Non-Haemolytic Transfusion Reactions (FNHTR)

These reactions are often caused by cytokines from leucocytes in the transfusion. The patient has a fever (> 1.5°C above baseline) usually with shivering and general discomfort during the transfusion or up to two hours after.

N.B. care must be taken in diagnosing a FNHTR as haemolytic and septic reactions can present similarly.

10.7. Delayed complications of transfusion

10.7.1. Delayed Haemolytic Transfusion Reaction (DHTR)



Immune-mediated haemolytic reactions involving IgG typically cause extravascular haemolysis (in this context extravascular means outside the main blood vessels) for example:

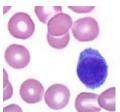
- Rh antibodies (anti-D, anti-C, anti-c, anti-E, anti-e)
- Anti-Kell (anti-K, anti-k, anti-Kp^a, anti-Js^a etc.)
- Anti-Kidd (anti-Jk^a and anti-Jk^b)
- Anti-Duffy (anti -Fy^a and anti-Fy^b)
- Anti-S

Antibodies attach to antigens on the red cell, which are then phagocytosed by macrophages, and also lysed within the hepatic and splenic sinusoids.

The reaction usually occurs within 14 days of transfusion, and may include falling Hb levels, a lower rise in Hb than expected, jaundice, fever and rarely haemoglobinuria or renal failure. The laboratory should be informed immediately and must investigate. Renal function should be closely monitored although specific treatment is rarely required.



10.8. Transfusion Associated Graft-versus Host Disease (TA-GvHD)



TA-GvHD is a life threatening complication of transfusion that is in most cases preventable. Usually a recipient of a transfusion will recognise any foreign lymphocytes and destroy them; however there are 2 conditions that prevent the recipients from protecting themselves in this way:

- 1. If the recipient shares the HLA haplotype of the donor
- 2. Defective cell-mediated immunity in the recipient.

In these patients the donor lymphocytes engraft and proliferate, they then recognise the recipient's cells as foreign and attack them causing inflammation and tissue damage.

Clinical features include fever, skin rash, diarrhoea, hepatic dysfunction, and bone marrow failure typically 1-2 weeks after the transfusion (Handbook of Transfusion



Medicine 2005).

Although all blood components are leucodepleted by filtration, lymphocytes are a similar size to red cells, and some are still present after filtration. Cellular blood components can be made safe by gamma or X-ray radiation that stops lymphocyte proliferation. These components have a label applied to the bag, which turns black on successful irradiation. Patients who require irradiated blood should be given an information leaflet and card, which are available from the local blood centre.

10.9. Post Transfusion Purpura (PTP)

PTP is an extremely rare but serious complication of transfusion; there is a sudden drop in platelet count usually 5-10 days after transfusion. The thrombocytopenia is so severe that haemorrhage occurs and could be fatal. In a patient who has already been sensitised either by pregnancy or a previous transfusion the thrombocytopenia is caused by an antibody-mediated reaction that destroys both donor and (for reasons not completely understood) the patient's own platelets (2011). PTP must be reported to both the MHRA and SHOT. Treatment of choice is IV IgG (85% response rate). Platelet transfusions (random or antigen negative) are ineffective and should be given for life threatening haemorrhage only.



11. COMPLICATIONS OF CHRONIC TRANSFUSION

Some patients require long-term red cell transfusion programmes, e.g. Haemoglobinopathies or marrow failure due to disease. These patients require sensitive care to deal with problems including poor venous access, reduced quality of life and frequent visits to hospital. Additionally, the more donor blood a patient is exposed to the more difficult it can become to find them suitable blood (excepting thalassaemia) and the higher the chances of a transfusion reaction.

Another complication of long-term transfusion therapy is the build-up of iron, which is deposited in major organs (heart, liver and endocrine system). Often when this problem presents itself, the patient is extremely unwell and there is little that can be done. Iron chelating therapy to excrete the excess iron initially was limited to subcutaneous infusions of desferrioxamine, which had varying levels of patient compliance (pain, oedema, inconvenience) similar to other chronic diseases. More recently oral iron chelators have been introduced which have been shown to be effective and with minimal toxicity (Hershko 2006).



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