Photothermal treatment (PCT) with the psoralen S-59 and long wavelength ultraviolet light (UV A) inactivates contaminating viruses and bacteria in high titre as well as leucocytes. In the reported study, the authors evaluated the efficacy of PCT in preventing transfusion-associated graft-versus-host disease (TA-GVHD) in vivo using a well-characterized parent to F1 murine transfusion model. Recipient mice in four groups were transfused with 10⁶ splenic leucocytes. Group 1 were control mice which received syngeneic cells; group 2 received untreated allogeneic cells; group 3 γ-irradiated (2,500 cGv) allogeneic cells and group 4 allogeneic cells treated with 150 μmol/l psoralen S-59 and 2.1 J/cm² UVA. The control mice remained healthy whereas the mice which received untreated allogeneic cells developed clinical and histological lesions of TA-GVHD. The mice in the other two groups remained healthy and developed no signs of TA-GVHD. Thus, in addition to inactivating high levels of pathogenic viruses and bacteria, photochemical treatment is also an alternative to γ-irradiation for the prevention of TA-GVHD. Moreover, since it inactivates leucocytes, it probably also prevents alloimmunization against HLA class 1 antigens.

J.A. Grass, T. Wafa, A. Reames et al.

Prevention of Transfusion-Associated Graft-Versus-Host Disease by Photochemical Treatment

Blood 1999;93:3140–3147

In this excellent review, the authors discuss the following aspects of transfusion-related acute lung injury (TRALI): the signs and symptoms of the syndrome and the blood products which have induced it as well as the percentage (5%) of fatal cases; the antibodies (antigranulocyte and anti-HLA) which cause TRALI, the antibodies present in the donor blood in the vast majority of cases, although a few cases in which the antibodies were present in the recipient and interdonor reactions have also been described; the differential diagnosis, i.e. distinction from cardiogenic and other forms of pulmonary oedema; prevention of TRALI, the necessity of excluding donors whose blood has caused injury; the pathophysiology. Although a reaction of granulocytes with antibodies, subsequent sequestration in the lungs and complement activation are involved in the reaction, there are several pathogenetic aspects that still need an explanation.

P.M. Kopke, P.V. Holland

Transfusion-Related Acute Lung Injury


In this excellent review, the authors discuss the following aspects of transfusion-related acute lung injury (TRALI): the signs and symptoms of the syndrome and the blood products which have induced it as well as the percentage (5%) of fatal cases; the antibodies (antigranulocyte and anti-HLA) which cause TRALI, the antibodies present in the donor blood in the vast majority of cases, although a few cases in which the antibodies were present in the recipient and interdonor reactions have also been described; the differential diagnosis, i.e. distinction from cardiogenic and other forms of pulmonary oedema; prevention of TRALI, the necessity of excluding donors whose blood has caused injury; the pathophysiology. Although a reaction of granulocytes with antibodies, subsequent sequestration in the lungs and complement activation are involved in the reaction, there are several pathogenetic aspects that still need an explanation.


Serious Hazards of Transfusion (SHOT)


In the United Kingdom and Ireland, haematologists were invited to confidentially report deaths and major complications after blood transfusion from October 1996 to September 1998. Over the 24 months, 366 cases were reported, of which 191 (i.e. 52%) concerned ‘the wrong blood to the patient’. Analysis of these cases revealed multiple errors of identification, often beginning when blood was collected from the blood bank. In total there were 22 deaths; 3 due to ABO incompatibility, 1 to an acute haemolytic transfusion reaction, 3 to delayed transfusion reactions, 1 to posttransfusion purpura, 8 to GVHD, 4 to transfusion-related acute lung injury and 2 to transmitted infection (1 bacterial, 1 malaria). In addition there were 84 cases with major morbidity. A ‘nil to report’ form was submitted by 164 of the 424 participating hospitals. The conclusions are that, although blood transfusion is now very safe, vigilance is needed to ensure correct identification of blood and patient. Staff education and awareness of ABO incompatibility and bacterial contamination as causes of fatal or life-threatening reactions to blood.

P. Sort, M. Navasa, V. Arroyo et al.

Effects of Intravenous Albumin on Renal Impairment and Mortality in Patients with Cirrhosis and Spontaneous Bacterial Peritonitis


In patients with cirrhosis and spontaneous bacterial peritonitis, renal function frequently deteriorates. The impairment is probably related to a reduction in effective arterial blood volume and is associated with a high mortality rate. The authors conducted a study to determine whether plasma volume expansion with intravenous albumin would prevent renal impairment and reduce mortality in such patients. Only cefotaxime was given to 63 control patients and cefotaxime as well as albumin at a dose of 1.5 g/kg body weight to 62 patients. Renal impairment was defined as a nonreversible deterioration of renal function during hospitalisation. The infection resolved in 59 patients in the cefotaxime group (94%) and in 62 in the cefotaxime + albumin group (98%). Renal impairment developed in 33% of the patients in the cefotaxime group and in 10% in the test group (p = 0.002). In the cefotaxime group 29% of the patients died but only 10% in the other group (p = 0.01). At 3 months, the mortality rates were 41% and 22%, respectively (p = 0.03). The authors conclude that in patients with cirrhosis and spontaneous bacterial peritonitis, treatment with albumin in addition to an antibiotic reduces the incidence of renal impairment and death as compared to treatment with an antibiotic alone.
A Combination of Megakaryocytes Growth and Development Factor and Interleukin-1 Is Sufficient to Culture Large Numbers of Megakaryocytes Progenitors and Megakaryocytes for Transfusion Purposes

Br J Haematol 1999;106:553–563

Thrombocytopenia induced by chemotherapy is a major risk in the treatment of cancer. Although haematopoietic recovery is hastened by stem cell transplantation, a considerable number of platelet transfusions are generally still needed with the danger of alloimmunisation and the development of refractoriness. The administration of recombinant thrombopoietin has not resulted in a satisfactory reduction of the duration of chemotherapy-induced thrombocytopenia. The use of ex-vivo-expanded autologous progenitors of megakaryocytes might contribute to enhanced platelet recovery in such patients. The authors investigated the effect of various combinations of cytokines on the proliferation in a liquid culture system and different combinations for megakaryocyte expansion. It was found that optimal ex vivo expansion of megakaryocytes is achieved by the combination of peglated megakaryocyte growth and development factor and IL-1. The numbers of megakaryocyte progenitors and megakaryocytes obtained in the liquid culture system were such that they are suitable for transfusion purposes.

Clinical Consequences of Alterations in Platelet Dose: A Prospective, Randomised, Double-Blind Trial

Transfusion 1999;39:674–681

Little is known about the effect of platelet dose on the magnitude or duration of the post-transfusion increment, particularly when platelets are transfused prophylactically. Financial constraints and an increasing demand for platelets make it important to evaluate the clinical consequences of lowering the platelet dose.

The authors administered 158 apheresis platelet concentrates prophylactically to 46 patients undergoing high-dose chemotherapy followed by haematopoietic progenitor cell transplantation in a prospective, randomised, double-blind, multiple-crossover study. Transfusions were given in pairs, differing only in platelet count: a lower-dose platelet concentrate (LDP) which contained a mean of 3.1×10¹¹ platelets (range 2.3–3.5×10¹¹) and a higher-dose platelets concentrate (HDP) containing 5.0×10¹¹ platelets (range 4.6–6.1×10¹¹). Patients who were bleeding or were refractory to platelet transfusions were excluded. The mean post-transfusion increment after LDP was 17,010 per microlitre and that after HDP 31,057 (p<0.0001). Only 37% of LDPs resulted in an increment of at least 20,000/μl versus 81% of HDPs (p<0.0001). The mean transfusion-free interval after LDPs was 2.16 days but 3.03 days after HDPs (p<0.01). Transfusion LDPs was associated with an increase of 39–82% in the relative risk per day of requiring subsequent transfusions (p<0.0001).

In conclusion, the use of LDPs for prophylactic platelet transfusions in such patients results in a lower increment, a lower likelihood of obtaining a post-transfusion platelet increment of 20,000/μl, a shorter transfusion interval and a greater risk per day of requiring additional platelet transfusions.

Provision of HPA-1a-Negative Platelets for Neonatal Alloimmune Thrombocytopenia: Screening, Testing and Transfusion Protocol

Immunohematology 1999;15:71–74

The vast majority of cases of alloimmune thrombocytopenia of the fetus or newborn are due to maternal anti-HPA-1a. The therapy of choice in cases of severe thrombocytopenia is transfusion of HPA-1a-negative platelets. These are not usually routinely available.

Platelets from 10–30 apheresis products are screened several times a week with a solid-phase red cell adherence assay. If HPA-1a negativity is confirmed in the PSIFT using three anti-HPA-1a sera, the donor is asked to reschedule. On the subsequent donation, the platelets are retested. Once confirmed as HPA-1a-negative, the donors are entered in the computer database. Each time these donors donate, the products are listed on the daily inventory sheet with a code indicating the HPA-1a-negative status. The apheresis recruitment department tries to schedule at least two HPA-1a-negative donors weekly. As a result, HPA-1a-negative platelets are available most of the time. Assuming that donors donate four times a year, 36 HPA-1a-negative apheresis donors are sufficient for the above schedule. A protocol for the diagnosis of alloimmune thrombocytopenia due to anti-HPA-1a is also described in the paper.