Blood Product Support for the Oncology Patient

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Cover Illustration by Christie Grams
I. INTRODUCTION

A. The ability to provide adequate blood product support has made most of modern, dose-intensive therapy in oncology possible. For example, leukemia induction and consolidation therapy cannot be safely done without the ability to provide platelet and, to a lesser extent, packed red blood cell (PRBC) transfusions. This review will focus on the indications for blood component therapy, complications of therapy, and special blood product manipulations. Although the principles are broadly applicable to all patients, emphasis will be placed on issues relevant to patients with hematological malignancies and solid tumors. The topics of stem cell transplantation and donor lymphocyte transfusion are beyond the scope of this review.

B. Allogeneic (homologous) transfusions will be reviewed; autologous donation has limited applicability in oncology.

C. Although the introduction of recombinant hematopoietic growth factors and peripheral stem cell (as opposed to bone marrow) transplantation has decreased the need for blood products, these innovations have not eliminated the necessity for blood component therapy.

D. The indications for blood component therapy have evolved during the last 2 decades, and new complications have been recognized. Thus, the potential benefits of transfusion must be weighed against the attendant risks.

II. INDICATIONS FOR BLOOD COMPONENT THERAPY

A. Whole blood therapy is not indicated, and most transfusion medicine services no longer offer the product.

B. Packed erythrocyte transfusion

1. The most basic indication for PRBC transfusion is symptomatic anemia. The classic transfusion threshold of “10/30” (which is used to signify a hemoglobin level of 10 g/dL and a hematocrit of 30%) is not justified by available data. Although the use of a numerical trigger is not encouraged, a transfusion trigger of either 8.0 g/dL (proposed by the National Institutes of Health) or 7.0 g/dL (the American Association of Blood Banks) has been proposed.1–5

2. The decision to transfuse PRBC should be based on how well the patient tolerates the anemia. For example, an older person with angina and/or chronic obstructive pulmonary disease may not tolerate the same low level of hemoglobin as a younger, healthier person.

3. In a patient with bleeding or a significant bleeding risk (eg, in uremia), a hematocrit of 27% or more may improve primary hemostatic function.6,7

4. Some data suggest that a higher hemoglobin level is associated with improved outcome for patients with certain malignancies (eg, cervix cancer undergoing combined radiation and chemotherapy).8,9 If these data are confirmed, then PRBC transfusion may be indicated in these patients if the hemoglobin level cannot be raised to adequate levels with erythropoietin therapy. However, there are also controversial studies10–14 showing an increased mortality after transfusion with colon cancer.

5. Some studies show an improved quality of life with a higher hemoglobin level for patients with malignancies. Most of these studies incorporated the use of erythropoietin rather than PRBC transfusions.15,16

C. Platelet transfusions

1. Platelet transfusions are indicated in symptomatic thrombocytopenia, as prophylaxis against bleeding for patients with thrombocytopenia,
and in bleeding associated with platelet deficiency or impaired function.

2. During the previous 4 decades, prophylactic platelet transfusions were typically given to patients whose platelet counts had substantially decreased (< 20,000/µL) after chemotherapy. However, recent prospective, randomized trials have shown that platelet transfusion in patients with leukemia is not necessary until the platelet count is less than 10,000/µL.17–20

3. Concurrent risk factors—such as disruption of gastrointestinal or urogenital mucosa by chemotherapy and/or radiation, coagulation dysfunction from liver disease or disseminated intravascular coagulation, and direct tumor invasion—modify the bleeding risk. Thus, some patients with hemorrhagic cystitis may have significant bleeding if their platelet count is greater than 20,000/µL and platelet transfusion might be indicated in these patients.

4. In some bone marrow failure states, such as aplastic anemia or myelodysplasia, the risk of serious bleeding is often less and patients occasionally tolerate platelet counts of less than 10,000/µL without bleeding.

5. The decision to use either single-donor apheresis (SDP) collected platelets or pooled random donor units (usually from 5 to 6 donors) remains controversial, and practice varies widely. Although SDP are theoretically advantageous because of a presumed decreased risk of infection and alloimmunization, prospective studies have not demonstrated clear superiority of SDP units over pooled platelets.21

D. Fresh frozen plasma (FFP)
1. The use of FFP should be limited to patients with coagulation factor deficiencies when specific factor replacement product is not available (eg, factor VIII) or as replacement fluid for patients with thrombotic thrombocytopenic purpura (TTP) who are undergoing plasma exchange.

2. FFP should not be used as a volume expander in the absence of symptomatic coagulopathy and should not be used as a source of albumin.

E. Cryoprecipitate
1. Cryoprecipitate is made from FFP that has been thawed at a temperature between 1 to 6°C. The precipitate that forms in the cold is rich in fibrinogen, factor VIII, and von Willebrand factor (vWF).

2. With the availability of specific products for factor VIII and vWF, the use of cryoprecipitate is now limited. Perhaps the most common use in oncology is in the setting of profound disseminated intravascular coagulation and acute leukemia with attendant hypofibrinogenemia.

F. Granulocyte transfusions
1. Although uncommonly used, granulocyte transfusions are indicated in the setting of serious bacterial and fungal infection when recovery of endogenous neutrophils is anticipated within 3 to 7 days and the patient is refractory to appropriate antimicrobials. The data regarding efficacy are mixed and may be related to suboptimal numbers of neutrophils that could be transfused.

2. The number of granulocytes that can be collected for transfusion has significantly increased because of the use of granulocyte–colony-stimulating factor (G-CSF) to stimulate release from donors and recent advances in apheresis techniques.22 Thus, there is a resurgence of interest in granulocyte transfusions in the appropriate setting.23,24

III. DOSING OF BLOOD COMPONENT THERAPY

A. The guiding principle is to deliver the minimum dose required for the desired clinical effect and not necessarily to achieve a predetermined number.25 The amount of blood product that is given also depends on the ability of the patient to handle the attendant fluid volume challenge.

B. Packed erythrocytes
1. An often-quoted adage is that “there is no indication for transfusion of a single unit of PRBC;” however, this statement is not valid. The number of units that should be given is the amount that will achieve relief of symptoms.

2. Each unit of PRBC has a volume of between 250 to 330 mL, depending on the amount of plasma and additive solution present.

3. Each unit of PRBC will raise the hemoglobin level by 1 g/dL and the hematocrit by 3% in an average-sized adult without splenomegaly or ongoing bleeding.

C. Platelet transfusion
1. Each random donor platelet concentrate contains at least 5.5 × 10¹⁰ platelets in a volume of approximately 50 mL of platelets
and plasma; a single-donor apheresis unit contains at least $3 \times 10^{11}$ platelets in approximately 300 mL of plasma.

2. The expected increase in platelet count will be affected by factors such as ongoing platelet consumption, splenomegaly (and thus pooling of transfused platelets), and alloimmunization (see section V, subsection G). Various formulas have been proposed to assess the efficacy of platelet transfusion response, including the percentage of platelet recovery and the corrected count increment. Although useful for clinical studies, these formulas require knowing the number of platelets transfused, body surface area, or the estimated blood volume of a patient. In practice, most clinicians find this method cumbersome. As a rule of thumb, a random SDP concentrate would be expected to increase the platelet count by 2000/µL (12,000/µL for a SDP unit) after transfusion.26

3. The platelet count increment can be measured immediately after transfusion (10 minutes) and will not differ significantly from that taken at 1 hour,27 making it more convenient to obtain the blood sample for measurement of the post-transfusion platelet count when the platelet bag is being disconnected.

D. Fresh frozen plasma
1. Each unit of FFP is approximately 220 mL. In some blood centers, plasma collected by apheresis is available as so-called “jumbo-packs” and has volumes of 500 to 600 mL.

2. For patients with coagulation protein deficits, enough FFP should be given to achieve adequate hemostatic levels of factors (typically at least 50% factor activity). By definition, 1 unit of activity for any factor is that found in 1 mL of plasma. Adults have approximately 40 mL/kg of plasma volume. For example, using a conservative estimate of 0% factor activity to start, 1400 mL of FFP would need to be given to a 70 kg person in order to achieve 50% activity (70 kg \times 40 mL/kg \times 1%/mL). However, this is a rough approximation because the plasma distribution varies for different factors. For instance, the recovery for factor VIII is 50% and the recovery for factor IX is 80%. In the example, 2800 mL of FFP would need to be given to raise the factor VIII level to 50% activity, which is a considerable volume load. In practice, one can measure coagulation parameters (eg, prothrombin time or international normalized ratio [INR] and partial thromboplastin times [PTT]) to adjust dosing.

3. In the setting of TTP, the volume exchanged is generally between 1.0 and 1.5 multiplied by the calculated plasma volume of the patient.

E. Cryoprecipitate
1. Each unit of cryoprecipitate is approximately 15 mL. These units are typically pooled into a larger unit (comprising 10 smaller units) for clinical use, resulting in 150 mL of volume delivered.

2. In the setting of acute disseminated intravascular coagulation (DIC), most clinicians give enough cryoprecipitate to increase the fibrinogen level to 100 to 150 mg/dL or more. At these levels, the INR and PTT should be normal if hypofibrinogenemia is the major cause.

F. Granulocyte transfusions
1. Granulocytes are collected by apheresis from a single donor, and thus the number of cells will differ greatly. Donors given steroids, and especially G-CSF, before collection yield significantly more neutrophils. Most granulocyte collections contain a significant number of platelets and erythrocytes; therefore, patients and donors should be erythrocyte compatible to avoid transfusion reactions.

2. A granulocyte dose of 1 to $2 \times 10^{10}$ has traditionally been given. This amount generally did not yield an appreciable increase in neutrophil count of the recipient. Using G-CSF and steroid “primed” collection, 4 to $8 \times 10^{10}$ cells can be obtained that lead to measurable, but transient, increases in recipient neutrophil counts.22,23 Transfusions are generally given daily or twice daily until endogenous recovery of neutrophils.

IV. MODIFICATION OF BLOOD PRODUCTS

A. Oncology patients often require repeated blood component therapy, are immunocompromised, or have transfusion reactions. Modifications of the basic blood products are done in attempts to address or prevent these problems.

B. Leukocyte depletion (Table 1)
1. Leukocytes in blood components are thought to mediate febrile non-hemolytic transfusion
reactions; induce alloimmunization to human leukocyte antigens (HLAs) and thus cause platelet refractoriness; and transmit intracellular organisms, such as cytomegalovirus (CMV). Thus, leukodepletion is indicated to:

a. Decrease the incidence of febrile non-hemolytic transfusion reactions
b. Decrease alloimmunization to HLAs and thus decrease platelet refractoriness
c. Decrease the transmission of CMV

2. Prospective studies have shown that leukodepletion does, in fact, achieve these aims. However, some physicians doing stem cell transplants still do not feel comfortable using leukocyte-reduced products instead of CMV-seronegative blood products.

3. Leukodepletion can be done using bedside filters that remove 99.9\% of contaminating leukocytes (post-storage leukodepletion). Leukocytes can also be removed before storage of the blood component (pre-storage). Pre-storage leukodepletion reduces the amount of immunomodulatory cytokines that form during storage. The resultant blood product should have \(5 \times 10^6\) leukocytes per unit or less. The Food and Drug Administration may soon require that all cellular blood products be leukocyte reduced before storage.

C. \(\gamma\)-Irradiation (Table 2)

1. Transfusion-associated graft versus host disease (TA-GVHD) is an almost uniformly fatal complication of allogeneic blood transfusion caused by proliferation and differentiation of donor T-lymphocytes in a recipient. Although generally occurring in immunocompromised recipients, it can also occur in immunocompetent hosts when there is a significant degree of HLA homology between donor and recipient.

2. \(\gamma\)-Irradiation effectively eliminates the risk of TA-GVHD.

3. Oncology patients who should have their cellular blood products \(\gamma\)-irradiated include the following:
   a. Patients with stem cell transplants or those potentially eligible for stem cell transplantation
   b. Patients who have received purine analogues, such as fludarabine or cladribine.
   c. Patients with Hodgkin’s disease and possibly those with non-Hodgkin’s lymphoma
d. Patients receiving blood products from blood relatives (recent cases involving directed donors who did not identify themselves as blood relatives have suggested that all directed donor units should be irradiated) or those who are HLA-matched.
   e. Patients with congenital cellular immunodeficiencies
   f. Patients with acute leukemia (controversial)
g. There is no evidence that \(\gamma\)-irradiated blood products are necessary for patients with solid tumors and HIV infection who do not fit into any of the previous categories.

4. Preliminary data suggest that an adequate number of clonogenic T-cells may exist in FFP to mediate TA-GVHD. The need to irradiate FFP in the appropriate setting has not been shown to be necessary but seems prudent.

D. Plasma depletion (Table 3)

1. Urticarial reactions to blood products are generally thought to be mediated by allergies to plasma protein. Anaphylactic reactions rarely occur when sensitized IgA-deficient individuals are transfused with IgA-containing plasma.

2. For patients with 2 or more urticarial reactions that were not preventable by premedication with antihistamines, plasma depletion should be tried to prevent further reactions.
   a. Platelets can be washed and resuspended
in normal saline and/or albumin; however, this process will activate the platelets and could make them less functional when transfused and could result in a smaller platelet increment. Washed products must also be used shortly after the washing procedure because of bacteriologic concerns.

b. "Washed" PRBC may be used when erythrocyte transfusions are indicated.

E. Cytomegalovirus-seronegative blood products

1. CMV infection and disease are complications that can occur in an immunocompromised patient. For example, CMV pneumonitis is a life-threatening complication of allogeneic stem cell transplantation. CMV colitis can also occur in this setting. HIV patients may also develop CMV retinitis and colitis. These infections are primarily from activation of latent infection in these patients.

2. Latent CMV resides in mononuclear cells; thus, leukocyte reduction can effectively decrease the incidence of CMV transmission.\(^{28,31}\) However, not all authorities agree that a leukocyte-reduced product is equivalent to a blood product from a CMV-seronegative donor. The risk of CMV infection in a CMV-seronegative stem cell transplant recipient, even with components from CMV-seronegative donors, is about 2%. In addition, the importance of infection with a second serotype is uncertain.

3. In the oncology setting, the indications for CMV-seronegative products are:
   a. A patient who has received a CMV-seronegative allogeneic stem cell transplant. Although patients who have received an autologous stem cell transplant can also get CMV infection, it is rare for them to develop disease (e.g., pneumonitis). However, it is still the practice at many transplant programs to give only CMV-seronegative products to all CMV-seronegative patients whether they are receiving an allogeneic or autologous transplant.
   b. Patients with HIV who are seronegative for CMV.

4. Other indications in the non-oncology setting include intrauterine and neonatal transfusion and transfusion to a CMV-seronegative pregnant woman.

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viable T lymphocytes can proliferate and differentiate in the recipient, leading to graft versus host disease, which is nearly always fatal</td>
<td>Irradiate cellular blood products, although some evidence suggests that FFP should also be irradiated</td>
</tr>
</tbody>
</table>

Indications:
- Patients with stem cell transplants
- Patients with leukemia
- Patients with Hodgkin’s disease
- Patients with CLL who have been treated with fludarabine or other purine analogues
- Patients who are recipients of blood products from a directed donor (often a relative)
- Patients with solid organ transplants who are receiving immunosuppressive medications

CLL = chronic lymphocytic leukemia; FFP = fresh frozen plasma.

V. COMPLICATIONS OF BLOOD COMPONENT THERAPY

A. The complications that can occur in patients with malignancies are similar to those in patients without cancer. Complications, such as alloimmunization and TA-GVHD, are of greater concern in cancer patients because they are often immunocompromised and receive multiple transfusions.

B. Febrile non-hemolytic transfusion reactions (FNHTR)

1. FNHTR are perhaps the most common acute complication of blood component therapy in oncology patients. Recipient antibodies to leukocyte antigens mediate these reactions. The incidence has been reported to be up to 30%,\(^{32}\) especially with platelet transfusions. However, with the routine use of leukocyte-reduced products, the incidence is now approximately 10% to 15%.\(^ {32} \) More recent studies suggest that this rate can be decreased even further with the use of platelets that are 3 days old or less.\(^ {33} \)

2. Fever usually occurs within 4 hours of transfusion and is not accompanied by symptoms that generally accompany hemolysis, such as back pain, hypotension, or dark urine. Occasionally,
fever can occur during the transfusion. In this instance, it is prudent to stop the transfusion and evaluate for hemolysis or sepsis.

3. Fever during or after a transfusion can also be caused by bacterial contamination. The incidence of bacterial contamination is up to 1 in 1000 in platelet units because they are stored at room temperature to preserve viability. However, septic transfusion reactions as a consequence of this contamination are rare.

C. Acute and delayed hemolytic transfusion reactions

1. Acute hemolytic reactions caused by blood type mismatch are rare and usually are the result of clerical error.
   a. A special situation where this blood type mismatch may arise is in the setting of allogeneic stem cell transplant when engraftment occurs because of an ABO-incompatible donor. Donor isoagglutinins directed against antigens present on circulating recipient erythrocytes then cause hemolysis of these cells.
   b. This reaction can be prevented by doing erythrocyte exchange of the recipient and by giving blood products typed for the donor.

2. Delayed hemolytic reactions result from minor erythrocyte antigen mismatches not detected by initial crossmatching. Onset is usually 5 to 10 days after transfusion. The onset may be sooner in patients with prior transfusions or pregnancies.

3. The therapy and workup for hemolytic transfusion reactions are beyond the scope of this review, but they involve stopping the transfusion, treating hypotension or respiratory distress, obtaining blood samples to document hemolysis, repeating crossmatching, and ensuring adequate urine flow.

D. Transfusion-associated acute lung injury

1. This acute complication of transfusion is caused by neutrophil agglutinins in donor plasma to recipient leukocytes and leads to neutrophil aggregation and activation as well as a non-cardiogenic pulmonary edema.

2. Diagnosis lies in recognition of the problem and demonstration of anti-leukocyte antibodies.

3. Treatment is supportive with supplemental oxygen or ventilatory support as necessary.

Table 3. Washed Blood Products (Plasma Depletion)

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solution</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma proteins mediate allergic or febrile reactions not eliminated by leukocyte reduction</td>
<td>Washing removes plasma and also removes leukocytes but not as efficiently as leukocyte filters or pre-storage leukocyte reduction</td>
<td>Patients with a history of urticarial/allergic reactions or with non-hemolytic febrile reactions not eliminated by leukocyte reduction</td>
</tr>
</tbody>
</table>

E. Transmission of infectious disease (Table 4)

1. The transmission of infectious agents remains a major concern for patients receiving blood component therapy despite the fact that the blood supply is safer than ever. It remains possible to transmit infectious agents via transfusion even with aggressive donor screening, volunteer donors, and very sensitive tests for transmissible agents.

2. The risk of infectious disease transmission is very low when compared with the risks inherent in oncology treatment (Table 4). This risk is likely to decrease even further with the introduction of nucleic acid testing of all donations of blood and blood components.

F. Immunosuppression

1. Case-control studies suggest that cancer patients receiving more transfusions have a higher mortality rate, even when controlled for known prognostic factors that might predict for mortality.

2. Some laboratory data suggest that multiple transfusions are associated with immunosuppression. However, whether this transfusion-associated immunomodulation is clinically relevant is uncertain and controversial.

G. Alloimmunization

1. Repeated exposure to cellular blood products can lead to alloimmunization against HLAs, leukocyte-specific antigens, or platelet-specific antigens.

2. These antibodies can mediate febrile non-hemolytic transfusion reactions, transfusion-related acute lung injury, or platelet refractoriness.

3. Antibodies may be transient and do not
preclude the use of appropriate blood component therapy in the future.

H. Transfusion-associated graft versus host disease
1. This relatively rare complication of allogeneic blood transfusion occurs when viable donor lymphocytes are able to proliferate and then react to recipient antigens, usually in the setting of an immunocompromised recipient.
2. TA-GVHD can also occur in an immunocompetent host when significant homology exists between donor and recipient HLAs, which allows the donor lymphocytes to evade detection by the host immune system.
3. The onset is usually 5 to 10 days after transfusion and begins with a skin rash, fever, and elevated liver function tests. Unlike the graft versus host disease (GVHD) associated with allogeneic stem cell transplants, marked pancytopenia also develops.
4. Diagnosis rests on strong clinical suspicion, skin biopsy, and demonstration of a mixed chimeric state using HLA-typing or molecular analysis of polymorphic genes (or conversion to donor HLAs in the peripheral blood).
5. Treatment has been unsatisfactory, and patients generally do not respond to the maneuvers used for transplant-associated GVHD. Transient responses have been reported with high-dose immunosuppression. One patient has survived after high-dose immunosuppression and autologous stem cell transplant.36

VI. SPECIAL PROBLEMS IN BLOOD COMPONENT THERAPY

A. Platelet refractoriness
1. Platelet refractoriness is defined as an inadequate post-transfusion platelet increment after 2 successive platelet transfusions.37
   a. A patient should not be considered refractory after one inadequate increment because this problem may be caused by the platelet unit and not by the patient. Some data suggest that fresher platelets (ie, those stored for the least amount of time) lead to improved post-transfusion increments. Some centers also do not routinely ABO-match platelet transfusion, but data suggest better post-transfusion increments if this is done.37
   b. Platelet refractoriness is not always mediated by alloantibodies; thus, it should not be assumed that the platelet refractory patient has been alloimmunized. Other causes of poor platelet response to transfusion are:
      1) Splenomegaly
      2) Sepsis (with or without DIC)
      3) Tumor-associated DIC
      4) Amphotericin B
      5) Malignancy-associated TTP
      6) Significant bleeding
      7) Veno-occlusive disease of the liver (after high-dose therapy and autologous stem cell transplant)
2. Alloantibody-mediated platelet refractoriness can be directed either against HLAs or against platelet-specific antigens. Platelets express HLA-A and, to a limited extent, HLA-B. Most patients refractory to platelets have anti-HLA antibodies. To a lesser extent, antibodies are directed against platelet glycoproteins.
   a. HLA antibodies can be demonstrated by lymphocytotoxic antibody assays. These demonstrate anti-HLA antibodies.
   b. Antiplatelet antibodies (whether directed at HLA or platelet-specific antigens) can also be demonstrated using various platelet crossmatch systems that use erythrocyte agglutination as indicative of the presence of platelet antibodies.
3. If alloantibodies are thought to be mediat-
ing platelet refractoriness in a patient, then either HLA-compatible platelets or crossmatch-compatible platelets can be used (Table 5).38,39

a. HLA-compatible platelets are identified in 2 ways, depending on the strategy of the particular blood center. In some blood centers, donors with the most compatible (but not necessarily identical) HLA-A and B antigens are chosen. In other centers, the specificity of the anti-HLA antibodies is determined and donors who lack those antigens are then asked to donate. The latter strategy generally yields more donors but may have a greater risk of further alloimmunization.

b. An alternative or complementary strategy is to use crossmatch-compatible platelets. In platelet crossmatching, samples of platelets that are already in storage are incubated with recipient serum and reactions are determined (usually involving agglutination). Donor platelets that do not react with recipient serum can then be used for donation. This method obviates the need to call in donors because the platelets are already available.

c. These 2 methods have not been compared in a randomized study; available studies show that the methods may be complementary and not necessarily redundant.38

d. These strategies do not always improve post-transfusion platelet increments.

e. Alloantibody titers decrease with time; therefore, a patient may not remain refractory with future transfusion.

B. Patients who refuse blood product support because of religious or other reasons

1. Some patients with malignancies will not accept allogeneic blood products or even plasma derivatives (eg, Jehovah’s Witnesses) because of their religious beliefs. These patients have taught us that some people can tolerate extremely low levels of hemoglobin.

2. With modern growth factor support, it is possible to administer myelosuppressive chemotherapy to these patients.

3. Recombinant human erythropoietin (rHuEpo) may be used in most of these patients to support the hemoglobin level.

   a. The dose of rHuEpo generally starts at 150 IU/kg per day thrice weekly (this can also be given as a weekly dose) and increases to 300 IU/kg per day thrice weekly if the patient does not respond after 4 to 8 weeks.

   b. The rHuEpo is stabilized with human albumin; some patients will refuse this preparation for religious reasons, but most have found this to be acceptable.

4. G-CSF or granulocyte-macrophage colony-stimulating factor can be used to support neutrophils, as in other patients.

5. Interleukin-11 can be used if patients have severe thrombocytopenia.

Table 5. Human Leukocyte Antigen (HLA)– or Crossmatch-Compatible Platelets

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solution</th>
<th>Indications</th>
<th>Not indicated when platelet refractoriness is not immunologically mediated (eg, DIC, hypersplenism)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractoriness to platelet transfusion can be mediated by antibodies directed against HLA or by platelet-specific antigens</td>
<td>Provision of HLA- or crossmatch-compatible units may improve platelet recovery after transfusion</td>
<td>Indicated for patients who have poor platelet recovery after 2 successive platelet transfusions</td>
<td>Indicated for patients who have antibodies to HLA or platelet-specific antigens</td>
</tr>
</tbody>
</table>

DIC = disseminated intravascular coagulation.
include acute reactions, infectious disease transmission, and possibly immunosuppression.

- The proper use of blood product support can lead to improved safety, improved quality of life, and possibly improved outcome for patients with cancer.

**ACKNOWLEDGMENT**

The author would like to thank P.V. Holland, MD, for his helpful suggestions and review of this manuscript.

**BOARD REVIEW QUESTIONS**

Choose the single best answer for each question.

1. A 34-year-old man is undergoing high-dose therapy and allogeneic stem cell transplantation for relapsed acute myelogenous leukemia in second complete remission. He is cytomegalovirus (CMV) seronegative. It is now more than 3 days after his stem cell infusion, and his hemoglobin level is 7.0 g/dL. Two units of packed red blood cells (PRBC) are ordered for transfusion. All of the following would be appropriate EXCEPT:
   A) Irradiating the PRBC
   B) Washing and re-suspending PRBC
   C) Using leukocyte-reduced PRBC
   D) Using CMV-seronegative donor PRBC

2. With current viral screening procedures, the risk of transfusion-transmitted viral infection is greatest for which of the following viruses?
   A) HIV-1/2
   B) Human T cell lymphotrophic virus (HTLV)-1/2
   C) Hepatitis C virus
   D) Hepatitis B virus

3. Prospective randomized studies to date reveal that the minimum safe platelet threshold for prophylactic platelet transfusion in patients with acute myelogenous leukemia undergoing induction chemotherapy is:
   A) 5000/µL
   B) 10,000/µL
   C) 20,000/µL
   D) 30,000/µL

4. Transfusion-associated graft versus host disease (TA-GVHD) differs from that associated with allogeneic stem cell transplant in that TA-GVHD is more likely to affect which organ?
   A) Liver
   B) Skin
   C) Bone marrow
   D) Kidney

5. A 40-year-old man presents with bleeding gums and epistaxis; he is diagnosed with acute promyelocytic leukemia (FAB-M5). His leukocyte count is 1300 cells/µL, hemoglobin is 9.3 g/dL, platelet count is 27,000/µL, INR is 1.64, PTT is 60 seconds, and fibrinogen is 50 mg/dL. The most appropriate blood component for initial transfusion is which of the following?
   A) Single-donor platelets
   B) Cryoprecipitate
   C) Granulocyte transfusions
   D) Fresh frozen plasma

**ANSWERS**


**REFERENCES**