Role of blood transfusion in obstetrics

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ABSTRACT
Blood transfusion in obstetrics should be administered to save the patient’s life. Severe blood loss could lead to hypovolemic shock which requires an immediate blood transfusion to prevent organ failure and death. Transfusion of blood and blood components must be approached like the use of medicines. They must be used only after weighing the benefits against the risks as well as in the right dose, right indication, and schedule. This review article deals with preparation, indications, administration of blood components, and its complications in obstetrics.

Key words: Blood components; coagulation factor deficiency; FFP; obstetric hemorrhage; platelets.

Introduction
It is believed that the first human blood transfusion was performed in 1667 by Dr. Jean Baptiste Denys in France. He transfused the blood of a sheep in a 15-year-old boy who survived the transfusion. In 1818, Dr. James Blundell, a British obstetrician performed the first successful blood transfusion of human blood for the treatment of postpartum hemorrhage using the patient’s husband as a donor.[1] Since then blood transfusion is in clinical practice. After Austrian Karl Landsteiner’s discovery of blood groups in 1901, blood transfusion methods became very safe without transfusion reactions.[1]

Blood is fluid in the vascular system whose function is to transport gases, substances, and heat in the systemic and pulmonary circulation which consists of water containing electrolytes, nutrients, vitamins, hormonal messenger substances, and gases in dissolved form together with various cell populations and proteins [cell population consists of erythrocytes, leukocytes, thrombocytes, and immune precursors like reticulocytes].[2] The main function of erythrocytes, basically hemoglobin is to transport blood gases. Oxygen is carried to the tissues by hemoglobin of RBC to which it is bound. So, anemia has the potential to reduce oxygen delivery.[1]

One common indication for blood transfusion in women is severe anemia. Prevalence of which is high in south Asian countries wherein half of the global maternal deaths due to anemia are reported from India.[3] Another indication is obstetric hemorrhage.

Although the need for transfusion can be reduced by preventing/treating anemia and blood loss; there are limitations to prevention and BT becomes essential.

Guidelines for a blood transfusion by WHO and RCOG in obstetrics are available but not mandatory.[4]

Improper use of blood components has led to significant morbidity and mortality. Therefore, the right decision for transfusion is the use of the right product, for the right...
Preparation for Transfusion

Preparing for an obstetric hemorrhage requires the drawing of a blood sample from the patient to obtain cross-matched blood. The first step in the process of preparing blood is determining the ABO type and the presence or absence of the Rh factor. Following typing, blood is screened for common antibodies. Screening involves mixing the recipient’s blood with commercially available antigens. If red blood cell agglutination or hemolysis occurs, antibodies are present and must be characterized. The initial type and screen takes approximately 45 min and is best for patients at low risk for requiring blood transfusion.

The most recent American Society of Anesthesiologists Practice Guidelines for Obstetric anesthesia state that routine blood crossmatch is not necessary.[6]

Red Cell Transfusion

Generally, red cell transfusions are given to increase the hemoglobin level in patients with anemia to replace losses after acute bleeding episodes. It is generally indicated if the value is less than 7 g/dL.

Whole blood should be considered as the choice only when treating an adult who has bled acutely and massively where volume replacement is necessary. It is collected in 350–450 mL and is separated into three components packed cells, FFP, and platelets. It has a shelf life of 40 days. It replaces many coagulation factors, which are important in obstetrics, especially fibrinogen, and its plasma treats hypovolemia.[7]

Banked blood should not be used unless there is a clear line of demarcation between sedimented cells and supernatant plasma, which should be straw-colored and free from visible signs of hemolytic. Hemolytic may be indicated by a reddish-purple discoloration in the supernatant plasma spreading upwards. The blood should have been stored at 4°C and should not be time expired. At no time should the blood have been allowed to freeze, as frozen and thawed blood may be lethal and blood which has been out of refrigerator for more than 30 min should not be used.[8]

Packed red cells should be transfused in patients with chronic anemia as volume replacement is not required. Packed cells will improve the patient’s oxygen-carrying capacity. Packed red, made by discarding plasma and adding cells of one or more other bottles should only be prepared from blood less than a fortnight old and should be used within 12 h of preparation.

One unit of packed erythrocytes is derived from one unit of whole blood to have a hematocrit of 55 to 80 volume percent, depending on the length of gentle centrifugation. The volume of one unit of RBCs with citrate phosphate dextrose adenine (CPD-A1) anticoagulant is between 225 mL to 350 mL. Stored at 2–6°C for 35 days. One unit contains the same volume of erythrocytes as one unit of whole blood. It will increase the hematocrit by 3 to 4 volume percent depending on patient size. Packed red blood cell and crystalloid infusion are the mainstays of transfusion therapy for most cases of obstetrical hemorrhage.[7]

One unit of RBC will raise the Hb of an average size adult by 1 g/dL (or raise Hct by 3%–4%).[7] In a previously healthy patient in urgent need for blood following hemorrhage a transfusion rate of 100 mL/min until the systolic blood pressure reaches 100 mmHg is well-tolerated but the rate should thereafter be cutback according to state of the blood pressure. In, correcting chronic anemia and in patients suffering chronic debilitating disorders, especially cases of cardiac disease, the rate of administration should not exceed 20–40 drops per min for fear of embarrassing the heart. Signs of overload are jugular-vein filling and moist sounds from pulmonary edema at lung bases.[8]

The clinician administering the blood is responsible for ensuring that grouping and compatibility testing have been adequately carried out, preferably under proper laboratory conditions. Only in the gravest emergency should Group O Rh-negative blood be used blindly since even this may contain anti-A or anti B antibodies and uncommon immunization against even Rh-negative antigens is possible.[8]

Complications of blood transfusion[6]

1. Febrile reaction: Grade 1: temperature to 37.8°C. Grade 2: temperature above 37.8°C with the sensation of chill but no shivering. Grade 3: Rigor
2. Circulatory failure and pulmonary edema

3. Hemolytic reactions: This follows giving of incompatible blood or blood already partly hemolyzed by freezing, heating, infection, and prolonged storage

The symptoms are of rapid onset and include fever, headache, the constricting sensation of chest, and lumbar pain. Hemoglobinuria and jaundice develop subsequently, the former in a few hours and latter in a few days. Death when it occurs is usually due to acute cardiac failure and suppression of urine. A transfusion reaction is characterized by fever, hypotension, tachycardia, dyspnea, chest or back pain, flushing, severe anxiety, and hemoglobinuria. Immediate supportive measures include stopping the transfusion, treating hypotension and hyperkalemia, provoking diuresis, and alkalinizing the urine. Assays for urine and plasma hemoglobin concentration and an antibody screen help confirm the diagnosis

4. Allergic reactions such as urticaria, rashes, angioneurotic edema

5. Air embolism: Another dangerous complication usually due to connecting a syringe to the transfusion bottle to speed up the drip, can also occur from faulty apparatus, even after puncturing the tubing to inject substances into the blood being given. This last hazard is reduced if the puncture is made at the distal end of the tube nearest the needle. Air embolism should be promptly treated by clamping off the source, turning the patient on to her left side for 2 h and giving oxygen

6. TRALI: The syndrome of transfusion-related acute lung injury (TRALI) can be a life-threatening complication. It is characterized by severe dyspnea, hypoxia, and noncardiogenic pulmonary edema that develop within 6 h of transfusion. It is estimated to complicate at least 1 in 5000 transfusions. Although the pathogenesis is incompletely understood, injury to the pulmonary capillaries may arise from anti-human leukocyte antigen (HLA) antibodies in donor plasma. These antibodies bind to leukocytes that aggregate in pulmonary capillaries and release inflammatory mediators. A delayed form of TRALI syndrome has been reported to have an onset of 6 to 72 h following transfusion. Management is with supportive therapy that may include mechanical ventilation

7. Bacterial infection: Bacterial infection from transfusion of a contaminated blood component is unusual because bacterial growth is discouraged by refrigeration. The most often implicated contaminant of red cells includes Yersinia, Pseudomonas, Serratia, Acinetobacter, and Escherichia species. The more important risk is from bacterial contamination of platelets, which are stored at room temperature. Current estimates are that 1 in 1000 to 2000 platelet units are contaminated. Death from transfusion-related sepsis is 1 per 17,000 for single-donor platelets and 1 per 61,000 for apheresis-donor packs

8. Viral infection: Risks from many transfusion-related viral infections have been curtailed. Fortunately, the most feared infection, HIV, is the least common. With current screening methods using nucleic acid amplification, the risk of HIV or hepatitis C virus infection in screened blood is estimated to be 1 case per 1 to 2 million units transfused. The risk of HIV-2 infection is less. Other viral infections include hepatitis B transmission, which is estimated to be <1 per 100,000 transfused units. Choosing donors who have been vaccinated will lower this incidence.

Transfusion of already infected blood may produce symptoms indistinguishable from those of a hemolytic reaction, except that there is extreme hypotension with warm extremities. There may also be vomiting, diarrhea, and abdominal pain. Antibiotics and vasopressor agents may be required.

Because of its high prevalence, cytomegalovirus-infected leukocytes are necessarily often transfused. Thus, precautions are taken for immunosuppressed recipients, keeping in mind that this includes the fetus. Finally, there are slight risks for transmitting West Nile virus, human T-lymphotropic virus Type 1, and parvovirus B19.

PLATELETS - Platelets are called thrombocytes, are non-nucleated, disk-shaped cells of 2–3 µm in size that is found in blood. The normal platelet count in adults is usually between 150000/µL to 450000/µL. They play an important role in maintaining hemostasis. They are activated when exposed to a disrupted endothelium leading to platelet aggregation and formation of hemostatic plug.

Platelet membranes act as a binding surface for initiation and continuation of coagulation cascade. There is increased risk of bleeding that can be potentially life-threatening because of platelet disorder whether it is qualitative or quantitative. Platelets are very precious and its transfusion can be life-saving, but should not be used when not indicated, as in some cases transfusion can lead to worsening of primary disease like TTP/HUS.

INDICATIONS: Platelet transfusion could be done prophylactically, to prevent spontaneous bleeding in high-risk cases or in preparation for invasive procedure that would otherwise cause bleeding if no platelet transfusion would have been given and thirdly therapeutically (i.e., to treat active bleeding).
When the invasive procedure is performed, platelet transfusion is required to keep the platelet count above 50,000/µL. Platelets should be transfused quickly in patients with thrombocytopenia and active bleeding to keep the platelet above 50,000/µL. Patients with platelet consumption (ITP, DIC) or platelet function disorders, platelets are transfused only for bleeding or in some invasive procedures.\(^9\)

**GENERAL CONSIDERATIONS:** Cause of thrombocytopenia should be established prior to platelet transfusion as platelet transfusions are not indicated in all cases of thrombocytopenia and may be contraindicated in certain conditions such as 1. Heparin-induced thrombocytopenia, 2. Thrombotic thrombocytopenic purpura 3. Hemolytic uremic syndrome, 4. Idiopathic thrombocytopenic purpura 5. Congenital IgA deficiency, 6. Post transfusion purpura.

After establishing the cause, the decision to transfuse the platelets should not be based solely on the patient’s count but should be supported by the need either to prevent or treat bleeding. Informed consent is required as in case of any other blood component transfusion. Transfusion should be given only after the risks associated with transfusion have been considered and only when the benefits outweigh the risks. Risks such as bacterial contamination (1:10000) and alloimmunization to platelets increased with platelet transfusion.

Ensure that all other coagulation parameters are checked as well. As all the patients of thrombocytopenia may not respond to platelet transfusion if other defects in hemostatic factors are present.

### Platelet Collection

Platelet can be collected in two ways:

As random donor platelet (RDP) concentrates from whole blood-derived platelets or apheresis from a single donor in blood bank.

RDP: For the preparation of whole blood-derived platelets every unit of donated whole blood is centrifuged with slow spin and platelet-rich plasma is collected in a separate bag. This PRP is centrifuged to separate platelets and plasma in different bags. The number of platelets per unit varies according to platelet count of the donor. Each unit of RDP is 50–70 mL plasma with platelets between 5.5*10^10 to 7*10^10. Usually 4–6 units of RDP are pooled together to allow transfusion of 3–4 * 10^11 platelets per transfusion. These are called RDP or whole blood-derived platelets.

SDP-Apheresis (single donor) platelets:

These are obtained by doing one or 2-hour apheresis procedures in blood bank on volunteer donors. During this procedure, large volumes of whole blood are processed into extracorporeal circuits and centrifuged to separate components. The red blood cells and certain amounts of plasma are returned to the donor and platelets are collected. A typical single donor platelet unit provides the equivalent of six or more units of platelets from whole blood (i.e, 3 to 6 * 10^11 platelets) and is approximately 200–400 mL in quantity.

Advantages of RDP:

Lower cost, ease of collection, and no separate apheresis unit is required.

Disadvantages of RDP:

Recipient exposure to multiple donors in a single transfusion and logistic issues related to bacterial testing.

Advantages of SDP:

Exposure of the recipient to a single donor rather than multiple donors, which potentially reduces the possibility of infection and alloimmunization.

Disadvantages of SDP:

Cost and need for special equipment.

### PLATELET STORAGE AND DOSAGE

Platelets have a shelf life of 5 days and are stored at 20°C–24°C with continuous gentle agitation to facilitate gas exchange and prevent platelet aggregations. Platelets are stored at room temperature because cold induces clustering of von Willebrand factor receptors on the platelet surface and morphological changes of the platelets, leading to enhanced clearance by hepatic macrophages and reduced platelet survival in the recipient. Platelets are metabolically more active at room temperature than other cells, so they are stored in bags that allow oxygen and carbon dioxide exchange. Dextrose is added as energy source and citrate is added to maintain pH and to prevent clotting. Platelet transfusion should be completed within 4 h of spiking the pack.

### Complications of Platelet Transfusion\(^9\)

1. **Febrile nonhemolytic transfusion reactions (FNHTR):**

These reactions are mediated by various inflammatory
mediators like IL-6 release during the manufacturing and storage of platelets may present as fevers, chills, and rigors.

2. **Allergic and anaphylactic reactions**: Allergic reactions to platelet transfusion are really common. They are usually due to IgE antibodies in recipients directed against proteins in the donor plasma rather than platelets themselves. Common symptoms include urticaria and pruritis in mild cases, and wheezing, shortness of breath, and hypotension in more severe cases.

3. **Infection**: Platelet transfusion is considered as the largest overall infectious risk involved in blood supply. Donor screening procedures and pathogen inactivation do not completely eliminate the risk of bacterial and other blood-borne infections, and infection by bacterially contaminated platelets represents a serious hazard of platelet transfusion. Platelets are stored at room temperature where bacteria can proliferate rapidly. The incidence of bacterial contamination is higher for platelets than RBCs (1 in 2000 for platelets vs 1 in 30000 for RBC). The risk of infection increases with increased duration of storage and in RDP as compared with SDP. Anaphylactic reactions (i.e., severe allergic reactions) are very rare complications of platelet transfusion. Associated with rapid onset of shock, angioedema, and respiratory distress.

**Alloimmunization and Post-transfusion purpura**: Expression of class 1 HLA on donor platelets are recognized by the donor immune system as foreign matter. Production of HLA antibodies leads to platelet transfusion refractoriness and rare complications of post-transfusion purpura.

**Transfusion-related acute lung injury**: TRALI is more common with platelet transfusion than with RBC transfusion. It is associated with antileukocyte antibodies in donor plasma. Patients usually present as dyspnea or nonproductive cough within 6 h of transfusion.

**Ta GVHD**: Transfusion-associated graft vs host disease in invariably fatal; it occurs because presence of T lymphocyte in donor transfused component in a patient with profound immune deficiency. Prevention is done by irradiation of transfusion product with 25 Gy gamma radiations, as platelets are not affected by this irradiation dose.

**FRESH FROZEN PLASMA**: This component is prepared by separating plasma from whole blood and then freezing it at -18 to -30°C within 8 h of collection.[7] The standard FFP unit collected from a single unit has a volume of approximately 200–250 mL and contains 0.4 g of fibrinogen and all clotting factors.

Approximately 30 min are required for frozen plasma to thaw. Plasma is not appropriate for use as a volume expander in the absence of specific clotting factor deficiencies. It is a source of all stable and labile clotting factors, including fibrinogen.

Thus, it is often used for the treatment of women with consumptive or dilutional coagulopathy. It is used as a component in massive transfusion protocols. APTT and prothrombin time (PT) of more than 1.5 times the reference value for the laboratory and a fibrinogen concentration of 0.8 g/L or less are indications for transfusion of FFP. It is used to replace single inherited clotting factor deficiency, mostly factor V for which no virus-safe fractionated product is available.

**PF24**: Plasma that is separated and frozen at -18° to -30°C within 24 h of collection is called PF24. It can be stored for 1 year. Both FFP and PF24 contain all the requisite plasma coagulation factors in a slightly reduced quantity than plasma. As the clotting factors are in a concentrated form, FFP should not be used as a source of specific clotting factors.

Thawed plasma is plasma that was frozen (FFP and PF24), thawed and kept in refrigerator temperature for up to 5 days.

**Liquid plasma**: Liquid plasma is the plasma that is never been frozen. It is stored at 1 to 6°C for up to 26 days, and in vitro, it appears to be superior to thawed plasma.

**S/D plasma**: S/D plasma is plasma treated with viral inactivating agents prior to freezing.

**DOSE AND INFUSION RATE** - The usual dose of plasma is approximately 10–15 mL/kg (approx 3–5 units) in most adults. This dose (750–1250 mL) represents a significant volume challenge.

The infusion rate is: for healthy adults 2–3 mL/kg/h (one unit in 1.5 h). For patients with volume overload or heart failure (approximately one unit in 4 h). The plasma used must be ABO compatible with the recipient. It does not need to be Rh incompatible; anti-D prophylaxis is not necessary for Rh-D negative recipients of Rh D positive FFP.
Each unit of cryoprecipitate is prepared from one unit of fresh-frozen plasma. It is reduced from an initial volume of 250 mL to a final volume of 10 mL. Each 10 to 15 mL unit contains at least 200 mg of fibrinogen, factor VIII, Von Willebrand factor, factor XIII, and fibronectin. Deficiency of fibrinogen specifically may be corrected by the administration of fibrinogen concentrate or cryoprecipitate. A dose of 3 g of fibrinogen concentrate or 10 units of cryoprecipitate increases plasma fibrinogen levels by 1 g/L. Alternatively, 4 units of FFP will provide the same amount of fibrinogen. Cryoprecipitate is an ideal source of fibrinogen when levels are dangerously low and there is oozing from surgical incisions. Because cryoprecipitate has only a small amount of plasma, ABO compatibility is not necessary.

Another alternative is virus-inactivated fibrinogen concentrate. Each gram of this raises the plasma fibrinogen level approximately 40 mg/dL. Either is used to replace fibrinogen. However, there are no advantages to these compared with fresh-frozen plasma for general clotting factor replacement. Exceptions are general factor deficiency replacement for women in whom volume overload may be a problem, an unusual situation in obstetrics, and for those with a specific factor deficiency.

**Recombinant Activated Factor VII (rFVIIa)** - This synthetic vitamin K-dependent protein is available as NovoSeven. It binds to exposed tissue factor at the site of injury to generate thrombin that activates platelets and the coagulation cascade. Since its introduction, rFVIIa has been used to help control hemorrhage from surgery, trauma, and many other causes. More than three-fourths of level I trauma centers include it in their massive transfusion protocols.

One major concern with rFVIIa use is arterial—and to a lesser degree venous—thrombosis. Also been used to control severe hemorrhage in women with and without hemophilia. It has been used with uterine atony, lacerations, and placental abruption or previa. In approximately one-third of cases, hysterectomy was required. Importantly, rFVIIa will not be effective if the plasma fibrinogen level is <50 mg/dL or the platelet count is <30,000/μL. An effective dose is 50–100 μg/kg intravenously every 2 h until hemostasis is achieved.

**MASSIVE TRANSFUSION**: The need for massive blood transfusion in obstetrics (10 U or more in 24 h) occurs in patients with placenta previa, accreta, abruption placenta, and severe postpartum bleeding. Coagulation defects is a common experience when a patient requires massive transfusion. This is because the banked blood is deficient in viable platelets and to some extent factor V and VIII. These have to be replaced either by FFP or fresh blood. If patient becomes deficient in clotting factors, 1 U of fresh frozen plasma should be administered for every 4 units packed RBCs. Citrate intoxication is also to be feared in truly massive transfusion as several liters of citrated blood is given rapidly. This can be countered by 10 mL of a 10% solution calcium gluconate per liter of citrated blood transfused in excess of 2 L. Fortunately, a healthy liver is able to detoxicate citrate rapidly.

**AUTOLOGOUS TRANSFUSION**: Patient phlebotomy and autologous blood storage for transfusion have been disappointing. Exceptions are women with a rare blood type or with unusual antibodies. In one report, three-fourths of women who began such a program in the third trimester donated only one unit. This is further complicated in that the need for transfusion cannot be predicted. For these and other reasons, most have concluded that autologous transfusions are not cost-effective.

**CELL SALVAGE**: To accomplish auto transfusion, the blood lost intraoperatively into the surgical field is aspirated and filtered. The red cells are then collected into containers with concentrations similar to packed red cells and are infused as such. Intraoperative blood salvage with reinfusion is considered to be safe in obstetrics.

**GRANULOCYTE TRANSFUSION**: Granulocyte transfusion may not be necessary for obstetrics but overall as a blood component, we should know. There is a renewed interest and application of granulocyte transfusion. Granulocytes are harvested from properly selected donors by apheresis after they are stimulated by dexamethasone and G-CSF. Usually it is transfused within few hours after the collection though it can be stored for 24 h at room temperature. The criteria for transfusions are absolute neutrophil count <500 cells/μL, evidence of bacterial or fungal infections and unresponsiveness to antimicrobial treatment for at least 48 h. The main indications are neutropenia from chemotherapy or transplantation, aplastic anemia, chronic granulomatous disease, and neonatal sepsis. Complications are pulmonary adverse reaction, transfusion-associated GVHD, alloimmunization, and infection.

**BOMBAY BLOOD GROUP** - The Bombay blood group is a rare blood group that is characterized by the absence of A, B, and H antigens in the red blood cell surfaces. It is known as the “Hh” blood group or the “Oh” blood group. It was first discovered by Dr. Y.M. Bhende C. K. Deshpande, and H.M Bhaia of the Seth Gordhandas Sunderdas Medical College in Bombay (Mumbai) in 1952 who first spelled it as Bombay.

(1) Thus, it is called the Bombay Blood group.
THE ‘H’ ANTIGEN: Karl Landsteiner in his discovery of the famous ABO blood types identified that the red blood cells have an “H” antigen on their cell surfaces.

This H antigen is the precursor of A and B antigens which is modified into “A” or “B” antigen likewise and the individual gets either “A”, “B”, or “AB” blood group. This modification occurs in the presence of a transferase enzyme. If this enzyme is lacking, then the “H” antigen is not modified and these individuals have the “O” blood group.

But if an individual does not have the “H” antigen but has the recessive “h” antigen on its cell surface; then such individuals though they may have the transferase enzyme the antigen would not be converted into “A, B, AB, or O” blood groups. Such individual will rather develop antibodies against “A or B” antigens to protect themselves. The probable reason for this exception is due to the production of an inactivated enzyme that is incapable of producing H antigen.

Epidemiology

When the Bombay blood group is misdiagnosed, fatal hemolytic transfusion reactions occur. It is one of the rarest blood groups, present in about 0.0004% (0.4 million) in the human population. In India, the incidence is 1 in every 7,600 population but, while latest statistics by Suraci et al. stated that this type of blood group is found in southeast Asia. The incidence in this region is 1/10,000 and in Caucasians it is 1/250,000 cases.

A high level of consanguinity presents among the parents of the Bombay phenotype.

This rare blood group is commonly seen in the tribal population of India; most commonly, in Maharashtra, Odisha, Karnataka, Andhra Pradesh, and Tamil Nadu.

Individuals with “O” blood group and the “Bombay Blood” group do look similar phenotypically as both of them do not have any antigen of A or B on their cell surface. This allows the health professionals to miss the Bombay blood group cases considering them as “O” blood groups. The difference can only be identified when we assess these individuals based on their genotype where the “O” blood group has the dominant “H” antigen while the “Bombay Blood” group has the recessive, “h” antigen.

CLINICAL MANIFESTATIONS: Patients with Bombay blood group are generally asymptomatic. As they do not have any antigen on their cell surface, such individuals can easily donate blood to any other individual of the ABO blood group system unless some other blood factor genes such as the Rhesus factor are incompatible.

But, the challenge arises in case these individuals require blood transfusions. In such cases, these individuals develop severe hemolytic reactions if they receive blood from other blood group individuals including the O blood group.

These individuals have antibodies against A, B, and H antigens; so they can receive blood ONLY from individuals who have the Bombay blood group which is a rare commodity.

Blood Component Therapy in Obstetrics

SEVERE ANEMIA: Blood transfusion is life-saving for severely anemic patients presenting in the last 4 weeks of pregnancy to combat hemorrhage that may complicate delivery. It is important to ensure that the total blood volume is not increased as this may precipitate cardiac failure/pulmonary edema. These patients who have usually been anemic for a long time have pronounced myocardial ischemia, which may be accompanied by cardiomegaly. To avoid any added strain on the heart, packed cells are transfused very slowly with simultaneous administration of a diuretic such as furosemide, which helps to maintain a negative fluid balance. This patient is kept in propped up position and oxygen inhalation is given if required.[11]

HELLP SYNDROME: Platelets are not given unless the platelet count is below 20,000/mm³ or below 50,000 and the patient shows signs of altered hemostasis. If there is evidence of bleeding or an invasive procedure that needs to be performed, platelets should be transfused, if the platelet count is below 20,000.[11]

ANTEPARTUM HEMORRHAGE: Management would depend on the type and severity of the hemorrhage. Initial management involves rapid assessment of maternal hemodynamic status and the rate of continuing blood loss, followed by a fetal assessment. Patients should have a large bore (14–16 gauge) intravenous line established and depending on the amount of blood loss, fluid replacement should commence. With continued bleeding at least four units of blood should be cross matched. Baseline hemoglobin and hematocrit levels should be obtained.

ABRUPTION: The major maternal risk is that of hypovolemic shock. There is usually a tendency to underestimate the amount of blood loss because of the unknown amount of concealed bleeding. This leads to under transfusion and inadequate correction of hypovolemia. Persistent hypovolemia can lead to acute renal failure. Coagulation failure resulting from DIC is a common complication. Restoration of maternal circulating volume should be a priority treatment. While fresh whole blood is ideal it is usually not available. A crystalloid...
infusion should be started before blood becomes available. If indicated, FFP at the rate of one unit for every four units of PRBC should be transfused to replace labile clotting factors even before laboratory signs of coagulation defects become obvious. Anti-D immunoglobulin prophylaxis should be given to nonimmunized Rh negative mother.

DIC: Seen in obstetric conditions such as abruptio placentae, amniotic fluid embolism, and prolonged fetal death in utero. It seems to result from a massive release of thromboplastin into the circulation causing intravascular formation of fibrin, consumption of coagulation factors, and subsequent activation of fibrinolytic system.

Usually, fibrinogen depletion will have to be corrected, and if there is marked thrombocytopenia, fresh blood containing viable platelets or a platelet transfusion is highly desirable. Cryoprecipitate preparations may be of great value, but in its absence a liter of FFP should be given which will supply 3 g of fibrinogen and factors V and factor VIII, the circulating volume being made up with transfused whole blood or crystalloids. Synthetic plasma expanders may aggravate hemorrhage. Blood transfusion is, of course, the first immediate necessity but stored blood is poor in platelet content and factors V and VIII. Fresh blood is naturally ideal but not available. Platelet concentrate may be given instead.[11]

POSTPARTUM HEMORRHAGE: The incidence of postpartum anemia necessitating red blood cell transfusion is less than 1% after vaginal delivery and 1–7% after cesarean section. Red cell transfusion is indicated when the blood loss exceeds 30% or more of blood volume. A Hb level of at least 8g/dl is recommended after transfusion. It should be remembered that patients with acute hemorrhage can have normal or hematocrit values until the plasma volume is restored. The clinical evaluation in such situations is extremely important.[11]

Conclusion
This review focuses on the importance of the appropriate use of blood and its products in the clinical setting. It is indicated only in case of obstetric hemorrhage with Hb <7 g/dl and Platelet transfusion when platelet count <20,000/cu mm or with platelet count <50,000 with bleeding episodes. They must be used only after weighing the benefits against the risks as well as in the right dose and right dose and schedule.

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