CHAPTER 10
COAGULATION AND HEMATOLOGICAL DISORDERS IN PREGNANCY

Learning Objectives
By the end of this chapter, the participant will:

1. Define the clinical features of disseminated intravascular coagulation in obstetrics.
2. List three possible causes of disseminated intravascular coagulation.
3. Summarize the management of a woman with disseminated intravascular coagulation.

Definition

Disseminated intravascular coagulation (DIC) is a syndrome of abnormal coagulation and fibrinolysis. Consumption coagulopathy is a disorder marked by reductions in blood concentrations of platelets due to exhaustion of the coagulation factors in the peripheral blood as a result of DIC. It occurs in response to certain obstetric complications that are listed below.

Etiology

- Eclampsia or preeclampsia
- Postpartum hemorrhage
- Sepsis
- Abruptio placentae
- Missed septic abortion
- Sickle cell crisis
- Ruptured uterus
- Trophoblastic disease (choriocarcinoma)
- Hypovolemic shock or massive blood transfusion
- Amniotic fluid embolism
- Intrauterine death

In obstetrics, DIC is always secondary to another health condition of the woman. The method of activation of the clotting system may be:

- Release of thromboplastins into the maternal circulation from placental and decidual tissue: This may happen suddenly in cases of abruptio placentae, amniotic fluid embolism, ruptured uterus, etc., and much more insidiously in the case of intrauterine death and missed abortion. In pregnancies complicated by abruptio placentae with enough severity to cause fetal death, DIC will supervene in about 25% of women. In women with intrauterine death or missed abortion, approximately 25% will develop DIC 5 to 6 weeks after fetal demise, with laboratory changes that, in some cases, become apparent from the start. With early ultrasound diagnosis and the use of prostaglandins to produce cervical changes and facilitate evacuation of the uterus, DIC caused by intrauterine death is likely to decrease.
- Injury to endothelial cells exposing the underlying collagen to the plasma and coagulation factors: This may be the initiating factor in some cases of eclampsia or preeclampsia and sepsis.
- Red blood cell or platelet injury leading to release of phospholipids: This may occur in blood transfusion reactions.

Pathophysiology

Normal hemostasis is a dynamic balance between coagulation, leading to fibrin formation, and the fibrinolytic system, which acts to dispose of fibrin when its hemostatic function has been fulfilled. In DIC, there is excessive and widespread coagulation due to the release of thromboplastins into the maternal circulation. This leads to consumption and depletion of the coagulation factors resulting in a hemorrhagic diathesis. In response to the widespread coagulation and fibrin deposition in the microvasculature, the fibrinolytic system is secondarily activated. This involves conversion of plasminogen to plasmin, which breaks down fibrin to form fibrin degradation products (FDP). FDP have anticoagulant properties, inhibiting both platelet function and the action of thrombin, thus further aggravating the coagulation defect.

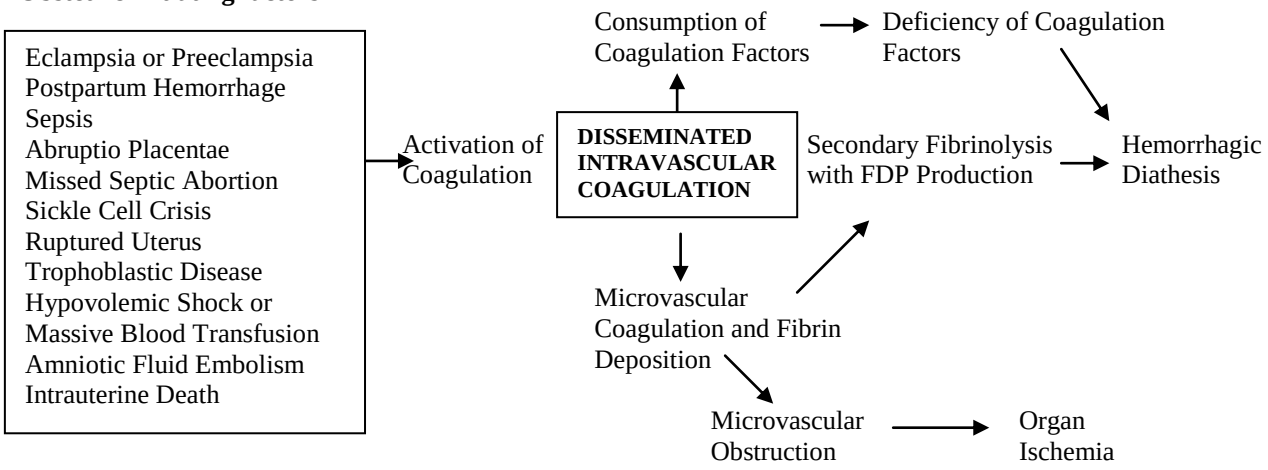
Hemorrhage diathesis is the main problem in most cases, but in some cases, widespread microvascular thrombosis can cause organ ischemia and infarction. This may be an accessory factor in the genesis of renal cortical necrosis in cases with severe abruptio placentae.

Clinical Features and Diagnosis

Clinical features

1. The main symptoms and signs are those of the obstetric complications causing the DIC.
2. Hemorrhagic manifestations may be relatively subtle with bruising, purpuric rash, epistaxis, and venipuncture oozing, or more dramatic with profuse bleeding from operative sites and postpartum hemorrhage.
3. Thrombotic sequelae rarely present in acute DIC as they are overshadowed by the hemorrhagic diathesis. The most common thrombotic manifestations are renal, hepatic, and pulmonary dysfunction**.**

Figure 1 - Causes and pathophysiology of disseminated intravascular coagulation



Diagnosis

Awareness of the obstetric conditions that may trigger DIC and the presenting clinical features is essential. Often the urgency of the situation and the lack of sophisticated laboratory facilities prevent definitive hematological tests. Interpretation of test results may also be difficult because the DIC process is so dynamic that results, when available, will often not reflect the current status of the woman. In severe cases of DIC, virtually all of the tests of coagulation and fibrinolysis will be abnormal, but in milder cases, the results are variable.

Bedside clot (observation) test

This is the most available and simplest test. An abnormal test indicates gross abnormality of the coagulation system. It is done by taking 5 ml of blood in a glass tube (or syringe), holding it in your fist to keep it warm, while inverting or tipping the tube three or four times and observing the following:

- Clotting time is prolonged if it takes greater than 7–8 minutes for a clot to form.
- Clot consolidation and retraction: The clot should be able to withstand inversion of the tube after 30 minutes and should not lyse or breakdown within 1 hour. The clot should occupy at least half of the total blood sample.

Where laboratory facilities are available, additional testing will reveal the following results:

- Platelet levels (platelet count) may be low or progressively fall.
- Partial thromboplastin time (PTT) is variable and may only be prolonged later in the process when the clotting factors are severely depleted.
- Prothrombin time (PT) or International Normalized Ratio (INR) will become prolonged.
- Thrombin time (TT) is usually prolonged.
- Fibrinogen levels (fibrinogen assay) are normally increased in pregnancy to 400-650mg/dl. In DIC, the level falls but may be in the normal non-pregnant range. With severe DIC, the fibrinogen levels usually fall below 150mg/dl.
- FDP: Levels of 80λ/ml confirm a DIC diagnosis. However, these elevated levels will remain for 24–48 hours after the DIC has been controlled.
- A blood film or blood smear may show abnormally shaped ("helmet" or "tear" shaped) and fragmented red blood cells (schistocytes). These are formed by the alteration of normal red blood cells as they are forced through the fibrin mesh in the obstructed capillary bed.

Management and Treatment

In most obstetric situations, DIC develops rapidly. Treatment must be prompt. Often, both time and facilities do not permit the luxury of thoroughly delineating the deficient clotting mechanisms. The process and progress of DIC is so dynamic that laboratory results may not reflect the current situation. This does not mean that one should not try and follow the laboratory aspects of the coagulopathy and enlist the aid of a hematologist if available. It does mean, however, that even without detailed hematological evaluation, one must have a rational management plan that will cover most of the problems encountered in this potentially disastrous complication.

Treat the initiating cause

Until the obstetric complication leading to the DIC occurs, most women who develop coagulopathy are healthy young women, although some may already suffer from anemia or malnourishment. These women have a great ability to recover rapidly and completely once the initiating cause is removed. In most cases, this entails emptying the uterus.

Maintain organ perfusion

In women, in whom the cause, or result, of DIC is hemorrhage, maintaining organ perfusion is the most urgent and important principle to follow. This is best accomplished by:

- Rapid infusion of Ringer's lactate or normal saline
- Rapid replacement with whole blood (Fresh blood is best, if available, because of its higher concentration of clotting factors and functional platelets.)

In most women, swift treatment of the initiating cause and maintenance of organ perfusion are all that is required for successful treatment. Once the cause of DIC has been removed, the liver will replenish adequate levels of most coagulation factors within 24 hours. The platelet count may take 5 to 6 days to return to normal, but will probably reach adequate levels for hemostasis within 24 hours.

Where additional resources are available
Where resources, in addition to those already mentioned, are available:

- Administer oxygen by face mask or by endotracheal intubation and intermittent positive pressure ventilation, if necessary, to achieve satisfactory arterial oxygenation.
- Monitor the above by a central venous pressure line if possible.
- Monitor the urinary output. Aim to keep the urinary output at least 30–60ml/hour.
- Monitor the complete blood count. Aim to keep the hematocrit >30%.

Replacement of procoagulants

The use of these is best guided by a hematologist. Knowing that the initiating cause of DIC is being treated, it is logical to think that critical and low procoagulant levels should be replaced to facilitate hemostasis and the adage "adding fuel to the fire" does not apply.

- Fresh frozen plasma replaces most clotting factors and has the least risk of transmitting hepatitis. As a working rule, give 1 unit after the initial 4 to 6 units of whole blood and thereafter 1 unit for every 2 units of whole blood required.
- Cryoprecipitates may be necessary if fibrinogen levels are low.
- Platelets can be transfused in severe cases of thrombocytopenia. One unit of platelets can raise the number of platelets to about 5,000 to 10,000.

Inhibition of the disseminated intravascular coagulation and fibrinolysis

The use of heparin has been advocated as a method of blocking DIC. It is recommended for cases of chronic DIC such as intrauterine death syndrome. Chronic DIC is considered a less severe form of DIC that produces no or only mild symptoms, such as bleeding from the skin or mouth. It is not recommended if the woman is bleeding profusely.

Epsilon aminocaproic acid inhibits the conversion of plasminogen to plasmin; its use has been suggested as a means to counteract secondary fibrinolysis. It is not recommended in these cases.

It is doubtful whether either of these two agents is ever justified in obstetric women with acute DIC. Their use is to a large extent theoretical, and wide practical experience with them is lacking. Their potential for worsening the hemorrhagic diathesis is very real. An exception may possibly be justified in the woman with intrauterine death who is not bleeding, but has strong laboratory evidence of DIC and coagulation factor deficiency. In these rare cases, under the guidance of an expert hematologist, one may consider an intravenous infusion of 1,000 units of heparin per hour until the clotting factors are restored to normal levels. Steps can then be taken to empty the uterus.

Walking Blood Bank

Obtaining blood in a developing country is a problem because blood banks are rarely available. This is complicated if some of the adult population has HIV or other blood-borne infections. When an obstetric emergency requires a transfusion, relatives are asked to donate their blood. It is important that bottles and tubing required for drawing and transfusing the blood be available. This does not allow for testing the blood for HIV, hepatitis, or other blood-borne diseases. To make safer blood available, the concept of a walking blood bank has been developed. Potential donors are identified and their blood is grouped. They are then called to give a blood donation once a woman's blood group has been determined. Blood compatibility is assured before the blood is drawn. Potential donors can be tested for HIV and hepatitis.

Key Messages

1. DIC is a life-threatening condition. Survival depends on prompt diagnosis and treatment, including rapid fluid replacement with fresh blood.
2. Health care providers need to work to improve the availability of access to blood in their health care facility.

Suggestion for Applying the Sexual and Reproductive Rights Approach to this Chapter

Information about illnesses and/or complex concepts, such as the ones described in this chapter, need to be communicated to women and their families in plain language. Be careful about the words you use. Provide time for the women, her spouse, and her family to ask questions so they understand what is happening.

APPENDIX 1

ANEMIA

An Overview
Anemia is one of the most common blood disorders. It occurs when the number of healthy red blood cells or the concentration of hemoglobin decreases in the body. The red blood cells (RBCs) contain **hemoglobin**, the molecule that carries oxygen to the body's tissues.

Anemia occurs for different reasons, these include:
- Increased destruction (break down) of RBCs
- Excessive blood loss (i.e. hemorrhage)
- Inadequate production of RBCs by the bone marrow

In some cases, anemia results from an inherited disorder, and in other cases the condition is caused by something in a person's environment such as a nutritional problem, malaria, infection, or exposure to a drug or toxin. It frequently occurs during pregnancy. Delivery poses a significant risk of further blood loss.

It is known that half the people in the developing world are anemic. In some areas, 80% of mothers are anemic. Severe anemia is dangerous for a mother and her baby. It makes the mother feels weak and tired, and may prevent her from taking care of her family during the pregnancy. She is more likely to become infected by disease. Surgical operations are more risky, and if she bleeds, she is more likely to die. Her child is at an increased risk to be born early, to be small for gestational age, and is also at risk for infections (antenatal and neonatal infections). There may also be poor development or growth of the infant, including growth of the baby's brain.

During a normal pregnancy, the hemoglobin falls. This is a dilutional effect because the body makes relatively more plasma than red cells This effect can be more apparent in women who have an underlying disorder causing anemia due to inadequate nutrition, inherited disorders of abnormal blood production and breakdown, and infections such as malaria and HIV/AIDS, both of which can cause anemia.

Types and causes of anemia

1. Anemia caused by the destruction of red blood cells

Hemolytic ("hemo" means blood, "lytic" means destroying) anemia occurs when RBCs are being destroyed prematurely, and the bone marrow (the soft, spongy tissue inside bones that produces new blood cells) simply can not keep up with the body's demand for new cells. This can happen for a variety of reasons. The common cause is malaria. Sometimes, infections or certain medications, such as antibiotics or antiseizure medications, are responsible

In **autoimmune hemolytic anemia**, the immune system mistakes RBCs for foreign invaders and begins destroying them.

Other causes are inherited defects in the RBCs that may involve their structure, or the production of hemoglobin or RBC enzymes. Common forms of inherited hemolytic anemia include (a) sickle cell anemia, (b) thalassemia, and (c) glucose-6-phosphate dehydrogenase deficiency.

a) Sickle cell anemia is a severe form of anemia found almost exclusively in people of African heritage, although it may also affect those of Caucasian, Saudi Arabian, Indian, and Mediterranean descent. In this condition, the hemoglobin forms long rods when it gives up its oxygen, stretching RBCs into abnormal sickle shapes. This results in premature destruction of RBCs, chronically low levels of hemoglobin, and recurring episodes of pain. About one out of every 500 African-American children is born with this form of anemia.

b) **Thalassemia**, which usually affects people of Mediterranean, African, and Southeast Asian descent, is marked by abnormal and short-lived RBCs. Thalassemia major, also called Cooley's anemia, is a severe form of anemia in which RBCs are rapidly destroyed and iron is deposited in the skin and vital organs. Thalassemia minor involves only mild anemia and minimal RBC changes. People with thalassemia minor do not absorb iron easily, and may have low iron stores if their diet is poor.

c) **Glucose-6-phosphate dehydrogenase (G6PD) deficiency** most commonly affects men of African heritage, although it has been found in many groups of people. The RBCs of people with this condition either do not make enough of the enzyme G6PD or the enzyme that is produced is abnormal and does not work efficiently. When someone born with the deficiency has an infection, takes certain medicines, or is exposed to specific substances, the body's RBCs suffer extra stress. Without adequate G6PD to protect them, many RBCs are destroyed prematurely.

2. Anemia caused by blood loss

Blood loss can also cause anemia—whether it's because of excessive bleeding due to injury, surgery, or severe bleeding in ante partum and in post partum period, or a problem with the blood's clotting mechanism. Slower, long-term or acute blood loss, such as intestinal bleeding due to inflammatory bowel disease, or in cancer polyps, can also cause anemia. Anemia can also result from heavy menstrual disorders in adolescent girls and women. Any of these factors will also increase the body's need for iron because it is needed to make new RBCs. It takes at least three months to replenish the RBC supply back to normal levels; the length of this time is extended with pre-existing iron and other nutrient deficiencies.

Infection with hookworms may cause so much blood loss that a woman can become anemic**.** Hookworm larvae enter the body through the skin or through contaminated food or water. They migrate to the small intestine where, as adults, they attach to the mucosa and ingest blood. Symptoms may include abdominal pain, diarrhea, colic, or nausea. The blood loss in the stools is occult blood loss, i.e. not visible. The primary method of diagnosis is to detect the worm eggs upon microscopic examination of the stools.

3. Anemia caused by inadequate red blood cell production

Infants are born with high levels of hemoglobin and RBCs in their blood. High levels in the fetus help the fetal blood carry enough oxygen while the developing baby is in the relatively oxygen-poor environment inside the uterus. After birth, when more oxygen is available, the baby's hemoglobin level normally drops to a low point at about 2 months of age, a condition known as physiologic anemia of infancy. After this occurs, the infant's body gets the signal to increase RBC production. This temporary and expected drop in the blood count is considered normal, and no treatment is needed.

Anemia also occurs when the body is not able to produce enough healthy RBCs. This can happen because of a deficiency of iron or certain other substances in the body or from inherited defects or diseases that interfere with the production of RBCs.

Iron is essential for the production of hemoglobin in RBCs. Poor dietary iron intake (or excessive loss of iron from the body) leads to **iron-deficiency anemia**, the most common cause of anemia in children. Iron-deficiency anemia can affect children at any age, but it is most commonly seen in children less than 2 years of age, and in teens, particularly in adolescent girls who have started menstruating.

During the first 6 months of life, babies are usually protected against developing iron deficiency due to the stores of iron built up in their bodies while they are in the uterus. However, by the second half of the first year of life, as infants continue to undergo significant growth, often they do not take in enough iron through breast milk alone or regular cow's milk (which contains less iron than fortified infant formula) to meet their iron needs. The growth spurt that occurs during puberty is also associated with an increased risk of iron-deficiency anemia. Girls are at particularly high risk because of the onset of menstruation; the monthly blood loss increases the need for dietary iron.

Anemia can be caused by deficiency in the nutrients folic acid and vitamin B12, both of which are necessary for normal blood production. Folate deficiency results in megaloblastic anemia. Folate deficiency is generally found in malnourished individuals, especially alcoholics, infants fed solely on cow's milk, pregnant women, and adults over 60. Malabsorption syndromes often produce folate deficiency. Certain drugs such as phenytoin, phenobarbital, primidone, isoniazid, and cycloserine are associated with the reduction of folate absorption and metabolism. Oral supplementation with folic acid and vitamin B12 is a common treatment of folic acid deficiency. Folic acid is found in many foods, especially asparagus, broccoli, endive, spinach, and lima beans Folic acid deficiency responds quickly to supplementation. **Pernicious anemia** is a type of anemia that occurs when a person lacks a substance that is necessary to absorb and process vitamin B12. However, these forms anemia are rarely found in babies and young children.

Aplastic anemia occurs when the bone marrow is unable to produce sufficient numbers of blood cells. More often, aplastic anemia is caused by a virus infection or exposure to certain toxic chemicals, radiation, or medications, such as antibiotics, antiseizure medications, or cancer medications. Some childhood cancers can cause anemia of this type, such as with certain types of leukemia in which abnormal cells crowd out the bone marrow cells needed to produce blood cells. Chronic diseases of other organs can result in anemia. For example, the kidneys and thyroid gland make hormones that are needed by the bone marrow to produce blood cells. HIV/AIDS is an important factor for women with anemia. Women infected with the HIV virus, but without symptoms tend to maintain adequate RBC production. However, women with advanced disease become anemic (>50%) for a variety of reasons. This is due to the normal progression of HIV disease. The virus can infect parts of the bone marrow responsible for manufacturing RBCs. There is also deficiency of the hormone erythropoietin that is required to stimulate RBC production. Opportunistic infections such as mycobacterium disease or fungal diseases can worsen anemia. Nutritional deficiencies due to malabsorption and decreased intake are common. This is a major source of anemia vitamin B12 deficiency.

Anemia can also be caused by short birth intervals or twins, mainly because the woman's body allocates the nutrients to the baby(ies) first and this may occur at her expense.

Signs and symptoms of anemia

The most common sign of iron deficiency and other types of nutritional anemia is mild paleness of the skin, along with decreased pinkness of the lips, the lining of the eyelids, and the nail beds. Other common signs of anemia may include irritability, fatigue, dizziness, lightheadedness, and a rapid heartbeat.

Depending on the condition causing the anemia, other signs and symptoms may occur, such as jaundice (yellow-tinged skin), dark tea-colored urine, easy bruising or bleeding, and enlargement of the spleen or liver. Anemia in pregnant women may result in intrauterine growth retardation, prematurity, and low birth weight among newborns.

In infants and preschool children, iron deficiency anemia can result in developmental delays and behavioral disturbances, such as decreased motor activity and problems with social interaction and attention to tasks. Recent research studies indicate that behavioral problems may persist into and beyond school age if the iron deficiency is not properly treated.

Diagnosing anemia

In many cases, health care providers do not discover anemia until blood tests are done as part of a routine physical examination. A complete blood count may indicate that there are fewer RBCs than normal. Hemoglobin and hematocrit are also of value. Other diagnostic tests may include blood-smear examination, iron tests, hemoglobin electrophoresis, bone-marrow aspiration and biopsy.

Treating anemia

Treatment for anemia depends on the cause of the condition. It is important not to assume that any symptoms are due to iron deficiency.

If the person has iron deficiency anemia, the health care provider may prescribe iron supplements in the form of drops (for infants) or a liquid or tablet form, and may also recommend adding certain iron-rich foods to the diet.

If a teenage girl is anemic and has heavy or irregular menstrual periods, in some cases, it may be appropriate to prescribe a birth control pill to help regulate her bleeding. The hormones in oral contraceptives, also known as the birth control pill, will decrease her blood loss and regulate her menstrual cycle.

Folic acid and vitamin B12 supplements may be prescribed if the anemia is traced back to a deficiency of these nutrients.

If a certain medication appears to be the cause, the health care provider may discontinue it or replace it with something else, unless the benefit of taking the drug outweighs this side effect.

If an infection is the cause, the anemia will usually get better when the infection passes on its own or it is cured by treatment. Sometimes the drugs used to treat the infection will cause the woman to develop anemia or make it worse. For example, pregnant women living with HIV may develop anemia associated with the long-term use of AZT. Prevention of maternal-to-child transmission of HIV regimens containing AZT are generally well tolerated by women living with HIV and their infants. However, the World Health Organization (WHO) makes these recommendations for the treatment of pregnant women living with HIV:

- Women with indications for ART who have severe anemia (Hb<7g/dl) should be started on a non-AZT-containing regimen and receive treatment for anemia. The alternatives to AZT are d4T or ABC (abacavir).
- Women without indications for ART should only take AZT-containing prophylactic regimens after severe anemia has been corrected (Hb>7g/dl). Alternatively the antenatal component of prophylaxis could be avoided and women receive only ARV prophylaxis, beginning in labour. This will not be as effective in preventing mother-to-child transmission.

The prevention and case management of malaria is important for reducing anemia among pregnant women living in malaria endemic areas. Women who are living with HIV may also become infected with malaria, and develop anemia. Co-infection may exacerbate the effects of the individual diseases or conditions.

Hookworm infection is treated with a 400 mg single dose tablet of albendazole or mebendazole. This treatment is considered safe in pregnancy, although the WHO recommendations state that all drugs should be taken after the first trimester. Children between the ages of 1–2 years receive a single dose of one-half tablet (200mg). One whole tablet may be given to children over 2 years of age.

Treatment varies according the mild, moderate, or severe anemia and the underlying cause. The WHO recommends the routine use of iron and folate supplementation for all pregnant women living in areas with a high prevalence of iron deficiency. Iron supplementation is also recommended during the postpartum period.

Treatment for more severe or chronic forms of anemia may include (depending on the cause) transfusions of normal RBCs taken from a donor; removal of the spleen or treatment, with medications to prevent blood cells from being removed from the circulation or destroyed too rapidly; and medications to fight infection or stimulate the bone marrow to make more RBCs.

Preventing anemia

Whether anemia can be prevented depends on the cause of the condition. Presently, there is no way to prevent anemia that is caused by genetic defects affecting the production of RBCs or hemoglobin.

However, the following steps can be taken to help prevent iron deficiency, which is the most common form of anemia.

1. **Cow's milk consumption**: Cow's milk given before 1 year of age or in too large an amount at any age has been associated with anemia. Parents also tend to associate milk with good nutrition and may not realize that the child needs better sources of iron. It is best ***not*** to introduce cow's milk into the child's diet until the second birthday. In addition, the child should not drink more than 24 to 32 ounces or <1 L of milk each day. If it is difficult get the child eat more iron-rich foods, the child may need an iron supplement.

2. **Iron-fortified cereal and formula**: These products can help ensure that the baby is getting enough iron, especially as the baby makes the transition from breast milk or formula to solid foods and other animal milks. These products should be continued at least until the child's first birthday and ideally until the child is older than 2 years of age.

3. **Well-balanced diet:** Make sure that the child or teen regularly eats foods that contain iron. Good choices include iron-fortified grains and cereals, red meat, egg yolks, leafy green and yellow vegetables, yellow fruits, potato skins, tomatoes, molasses, and raisins. Iron found in meat, poultry, and fish is more easily absorbed than iron found in plant-based and iron-fortified foods. However, it is not essential to eat meat in order to have an iron-rich diet. Most traditional diets contain good quality iron-rich foods or food combinations that support the absorption of iron from foods. Certain food combinations can inhibit or promote absorption of iron; for example, drinking coffee or black or green tea (including iced tea) with a meal can significantly lower the amount of iron absorbed. On the other hand, vitamin C helps the body absorb iron, so eating fruit, for example, with a meal will help the body absorb the iron in other foods.

4. **Side effects of medications:** Some medications can cause anemia. In most cases, the benefit of the drug outweighs the risk, but there is a need to monitor for signs of anemia and treat accordingly as soon as they occur.

5. **Healthy well-nourished mother,** with normal iron stores, can supply half the iron the baby needs from her own body. So the mother only needs to get the other half from her food. Many mothers, especially teenagers start their pregnancies with iron stores that are too small. They easily become anemic because they do not get enough iron in their food. Dietary counseling should be provided encouraging iron-rich foods in combination with quality sources of vitamin C-rich foods, where available. In addition, iron tablets should be provided.

6. **Insecticide-treated bed nets and intermittent preventative treatment of malaria**. See the malaria section in the Chapter 7, Infections.

Resources:

- Baskett, TF. *Essential Management of Obstetric Emergencies*. 4th Ed. Bristol: Clinical Press Ltd., 2004. pp. 90-96. This chapter has been adapted with permission of the author.
- Glen Mola; Jim Thornton; Michael Breen; Colin Bullough; John Guillebaud; Frank Addo, Eds. *Primary Mother Care and Population.* Stamford: The Signal Press; 2003.
- World Health Organization, ANTIRETROVIRAL DRUGS FOR TREATING PREGNANT WOMEN AND PREVENTING HIV INFECTION IN INFANTS: TOWARDS UNIVERSAL ACCESS Recommendations for a public health approach, 2006 available from http://www.who.int/hiv/pub/guidelines/pmtctguidelines2.pdf