



Disseminated Intravascular Coagulation (DIC)

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Course Description:

This course focuses on participants gaining a better understanding of Disseminated Intravascular Coagulation (DIC), the issues it brings to health care organizations, while providing participants with a practice setting to examine and develop their own skills. Education is empowering. DIC is a detrimental disease process that is life threatening for the women it effects.

Approximate Time to Complete: 100 minutes



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In this course you will:

- Develop sound critical judgment in the delivery of health care in a labor and delivery unit when (DIC) occurs.
- Discover learning theories and instructional implications regarding health care delivery in a labor and delivery unit when DIC occurs.
- Develop, implement, and evaluate health care delivery in a practice setting prior to an actual event. This will allow for early recognition of an actual event.
- Gain knowledge into active health care delivery. This will allow for rapid implementation of the necessary steps needed when DIC is suspected.
- Address issues and implement changes in the health care unit as necessary to ensure a safe environment. Equipment and supplies needed when DIC occurs will be in every labor and delivery room.
- Convert proven learning into actual health care delivery.



- Background Information
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Disseminated Intravascular Coagulation (DIC)

A pathologic disruption of the finely-coordinated process of hemostasis

- Massive activation of the clotting cascade results in widespread thrombosis, which leads to depletion of platelets and coagulation factors and excessive thrombolysis.
- This can result in hemorrhage, thrombosis, and/or multi-organ failure.
- A major medical challenge occurs when a woman presents with DIC and is further challenging when she is carrying a viable fetus..
- In the interest of the pregnant woman with DIC and heavy bleeding, performing an emergency cesarean delivery may not be appropriate. However, a category fetal heart rate (FHR) tracing and delaying delivery to transfuse the woman may not be in the best interest of the fetus.
- Labor and delivery of a fetal demise in a woman with DIC has the possibility for disastrous hemorrhage.



Occurrence of Disseminated Intravascular Coagulation (DIC)

- DIC in pregnancy has a prevalence of less than 0.5% [1, 2, & 3].
- Several large population - based studies illustrate the prevalence.



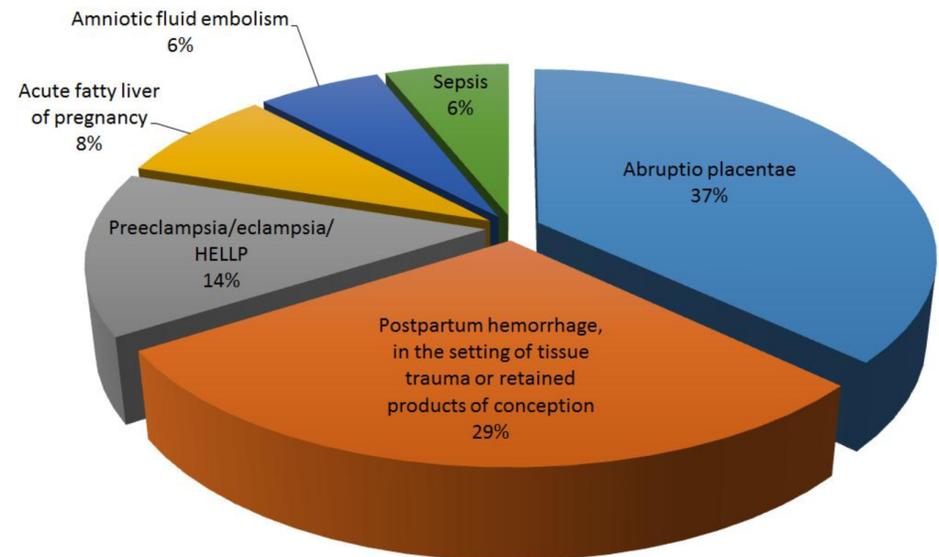
Rollover each marker to learn about studies in different countries.



- DIC does not occur in isolation.
- Pregnancy complications that may trigger and propagate DIC were evaluated in a review of 49 cases of DIC [2].
- Antecedent conditions included the following:
- Abruptio placentae – 18 cases (37 percent)
- Postpartum hemorrhage, in the setting of tissue trauma or retained products of conception – 14 cases (29 percent)
- Preeclampsia/eclampsia/HELLP – 7 cases (14 percent)
- Acute fatty liver of pregnancy – 4 cases (8 percent)
- Amniotic fluid embolism – 3 cases (6 percent)
- Sepsis – 3 cases (6 percent)
- The fetus died in one-quarter of these cases



Click the chart to enlarge it.



- Severe hemorrhage, itself, does not cause DIC but severe postpartum hemorrhage can be associated with DIC.
- The loss of clotting factors, platelets plus the generation of large amounts of fibrinogen products interfere with fibrin clot formation and platelet aggregation causing the bleeding in DIC.
- When severe postpartum hemorrhage occurs rapidly the depletion of clotting factors and platelets leads to consumptive coagulopathy; this is not DIC.
- When large amounts of tissue factor are released during severe postpartum hemorrhage it can be accompanied by true DIC [6].
- Following separation of the membranes and placenta, uterine decidual-derived tissue factor is normally released into the maternal circulation, activates the coagulation cascade, and generates thrombin [7,8].
- There are various causes (i.e. large laceration, placenta accreta) of postpartum hemorrhage that are associated with large release of tissue factor, resulting in intense physiologic intravascular coagulation process initiated by placental separation occasionally leading to DIC.
- Approximately 1-5% of all DIC cases are attributed to obstetric hemostatic emergencies in high - resource countries and even higher percent in low - resource countries [9].
- The remaining cases are due to nonobstetric causes.
- Causes of DIC not specific to pregnancy should be considered, especially when an obvious pregnancy-associated cause is absent [10,11].
- The most common events that initiate DIC in the general population are sepsis, tissue trauma/destruction, and cancer (Table 1).



*Click on the image
to view Table 1.*



- Levels of some coagulation factors increase to prevent excessive peri-partum bleeding during pregnancy.
- In addition to systemic changes in coagulation factors, decidual cells lining the vascular bed of the placenta strongly express tissue factor, similar to other vascular endothelial cells [12,13].
- At the site of decidual trauma the tissue factor is released to initiate the coagulation cascade which generates thrombin and thus crosslinked fibrin.
- Physiologic inhibitors of coagulation serve to prevent excessive fibrin generation.
- When DIC ensues the excessive production of thrombin leads to widespread intravascular fibrin deposition and widespread fibrinolysis.
- The result is a depletion of coagulation factors and platelets along with the production of fibrin degradation products leading to profound bleeding diathesis ([Figure 1](#)).
- These changes overwhelm and incapacitate the physiologic regulatory mechanisms and lead to thrombin not being contained.
- The uncontrolled and ongoing fibrin deposition may lead to thrombosis, end organ damage and failure.

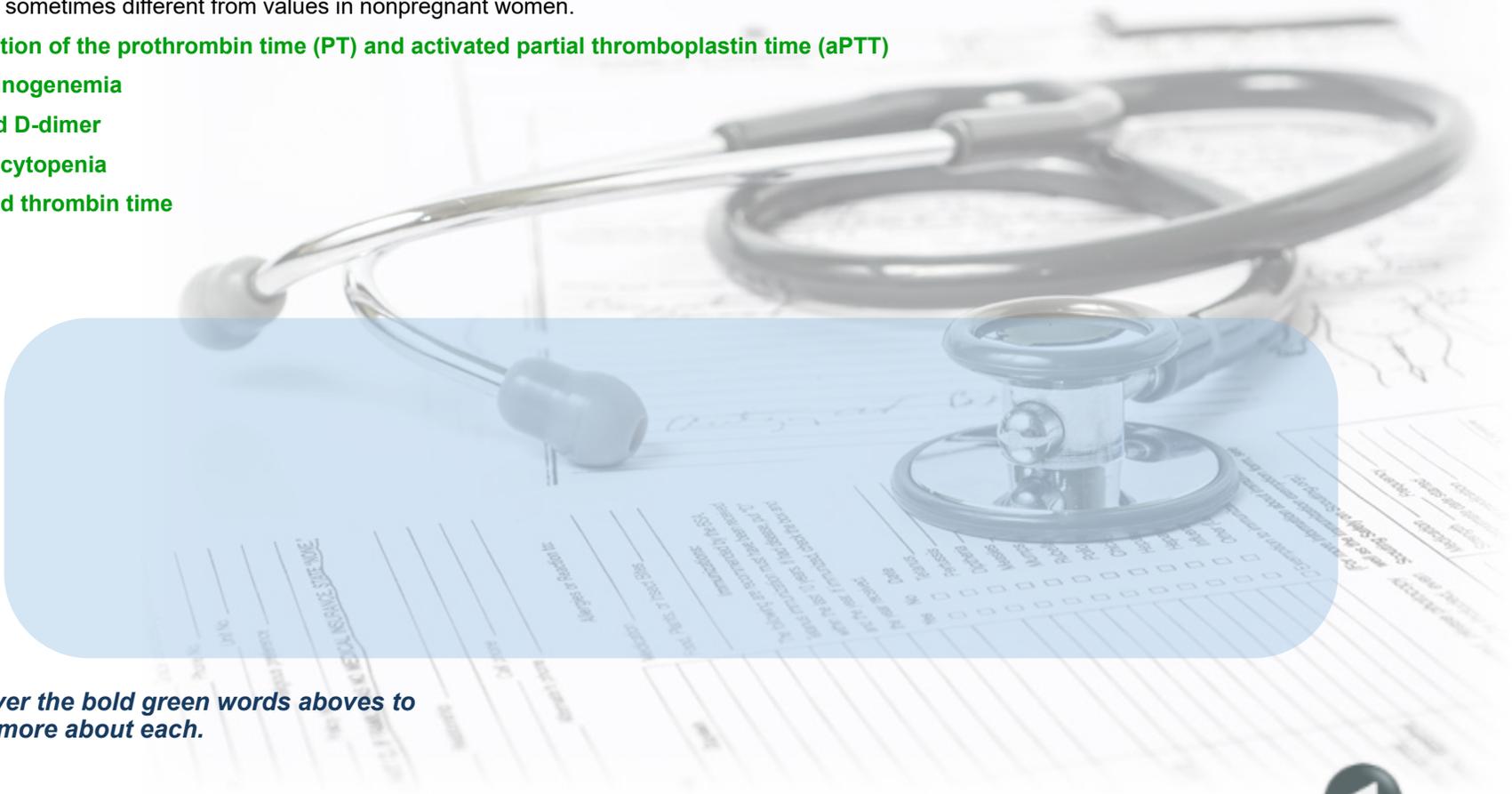
- DIC can be exacerbated by additional pregnancy complications and worsen hemostatic defects, although the mechanisms are not clear.
- Events occurring in pregnancy such as preeclampsia, eclampsia and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome may contribute to endothelial damage.
- Acute fatty liver may impair the production of coagulation factors produced by the liver and impair clearance of fibrin degradation products and shock may reduce tissue perfusion.
- When sepsis occurs the interaction of DIC with systemic inflammatory response syndrome plays a role in the pathogenesis of DIC [15].
- Hemorrhage alone does not cause DIC.
- In the setting of shock, severe tissue hypoxemia has been proposed to result in the release of tissue factor from the damaged cells [14].
- When significant injury or necrosis of fetoplacental tissue, as in abruptio placenta and retained fetal demise, occurs this cascade may be initiated by release of procoagulant substances leading to fulminant DIC.
- Amniotic fluid is also rich in procoagulants and anticoagulants [14].



- One of the following pregnancy complications may be present with DIC:
 - Abruptio placentae
 - Severe preeclampsia/eclampsia/HELLP syndrome
 - Amniotic fluid embolism (AFE)
 - Acute fatty liver of pregnancy
 - Septic abortion
 - Retained dead fetus
 - Massive hemorrhage
- Patients may present with severe bleeding (i.e. vaginal, intrauterine, intraabdominal) and/or diffuse oozing of blood from skin (i.e. at intravenous sites) or mucosa (i.e. from a bladder catheter).
- Some patients have signs of shock
 - tachycardia
 - hypotension
 - weak peripheral pulses
 - altered mental status
 - cool extremities
 - narrow pulse pressure (< 25 mmHg)
 - organ dysfunction
 - acute renal failure
 - hepatic dysfunction
 - acute lung injury
 - neurologic dysfunction



- Laboratory findings of DIC generally include prolongation of coagulation times and thrombocytopenia.
- These laboratory findings are interpreted for the pregnant woman which can be different from the nonpregnant woman ([Table 2](#)), which are sometimes different from values in nonpregnant women.
- **Prolongation of the prothrombin time (PT) and activated partial thromboplastin time (aPTT)**
- **Hypofibrinogenemia**
- **Increased D-dimer**
- **Thrombocytopenia**
- **Prolonged thrombin time**

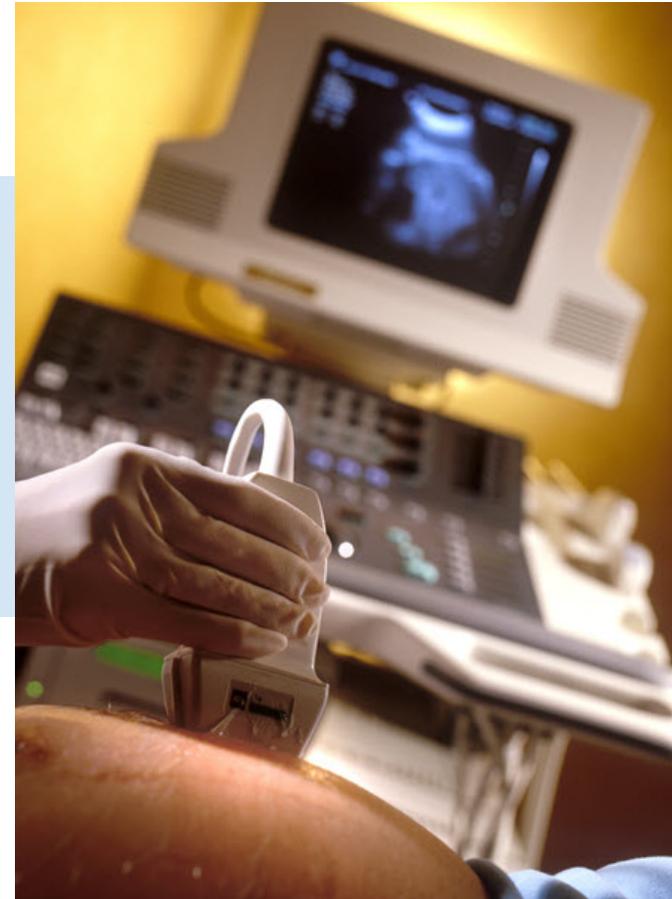


Rollover the bold green words above to learn more about each.



Clinical Evaluation

- When there is ongoing hemorrhage, shock or fetal distress the evaluation for DIC may need to occur concurrently with initial management of the specific disorder(s).
- Many pregnancy-associated causes of DIC are obvious from the history and physical examination.
- Additional findings of sepsis, malignancy, and liver failure should be sought, especially if the cause is not obviously apparent.
- Maternal vital signs are monitored closely.
- Fetal assessment as with every pregnant women.



- Laboratory testing includes the following:
 - Complete blood count with platelet count
 - Coagulation studies including prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen level, and D-dimer.
 - BUN and creatinine
 - Liver function tests (LFT)
 - Urine output and blood loss should be monitored closely.
- Prior to the return of the first set of laboratory studies, a red top tube (ie, no additives) containing 5 mL of blood can be observed for clotting (Lee and White test).
 - At room temperature, if the blood in the tube clots within 8 to 10 minutes and the clot remains intact, the patient likely has adequate fibrinogen stores.
 - If the blood in the tube does not clot or an initial clot dissolves, it is likely that the patient is markedly deficient in key clotting factors.
- Although rarely necessary in the obstetric setting where DIC is typically fulminant, serial laboratory assessments over a few hours showing progressively prolonged coagulation times, decreasing platelet counts, increasing values for D-dimer and/or fibrin-degradation products, and falling fibrinogen levels can help distinguish mild DIC from normal pregnancy-related changes in these laboratory values.
- Blood and urine cultures should be performed in patients with suspected sepsis.
- In cases where intrauterine infection is suspected, amniotic fluid culture is appropriate.

Criteria for Diagnosis

- DIC is a clinical diagnosis
- There is no single highly sensitive or specific test.
- The diagnosis of acute DIC is made in a pregnant woman when the clinical setting is appropriate such as placenta abruption, AFE, or sepsis, and there is thrombocytopenia, prolonged PT, aPTT, low fibrinogen, and fibrinolysis (increased D-dimer) when another cause is not evident.
- When DIC is suspected, collaborating and consulting with specialists is recommended to confirm the diagnosis and to eliminate other possibly life-threatening causes of the findings such as thrombotic thrombocytopenic purpura (TTP).

Scoring Systems

- Scoring systems have been developed and have been used in research studies to diagnose DIC in nonpregnant women as there is no single diagnostic test to confirm or reject this diagnosis.
 - The scoring system usefulness in pregnancy is unknown.
- An important limitation for any DIC scoring system is that these systems are only intended to be used in the appropriate clinical setting.

Differential Diagnosis

- When considering the differential diagnosis, other causes of bleeding, thrombosis, and organ damage must be consider in the pregnant woman.
 - Bleeding, thrombosis, and/or organ damage can accompany DIC or contribute to DIC pathogenesis
- Resolution of DIC may result with treatment for the underlying cause.

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There are numerous factors with the management of pregnant women with vaginal bleeding in the second and third trimesters including gestational age, the cause of bleeding, the severity and fetal status.



Management - Quick Overview

Notify staff and services that will or may be needed:

- Anesthesia
- Neonatology
- Blood bank
- Surgery
- Obstetrics
- Pelvic Surgery
- Maternal Fetal Medicine
- Gynecologic Oncology
- Interventional Radiology
- General Surgery



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