Disseminated Intravascular Coagulation

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Synonyms and related keywords: disseminated intravascular coagulation, DIC, thrombohemorrhagic disorder, sepsis, major trauma, abruptio placenta, fibrinolytic activation, endothelial injury, cytokines, tissue factors, thrombin, plasmin, coagulation cascade, acute DIC, chronic DIC, localized DIC, idiopathic purpura fulminans, septic abortion, deep venous thrombosis, DVT, hematemesis, hematochezia, azotemia, renal failure, hematuria, petechiae, purpura, hemorrhagic bullae, acral cyanosis, acute myelocytic leukemia, mucin-secreting adenocarcinomas, amniotic fluid embolism, eclampsia, retained dead fetus syndrome, myeloproliferative syndromes, paroxysmal nocturnal hemoglobinuria, Raynaud disease, giant hemangiomas, Kasabach-Merritt syndrome, hemolytic uremic syndrome, systemic DIC, procoagulant activation, inhibitor consumption, end-organ damage, end-organ failure, decreased platelet count, thrombosis, microvascular thrombosis, spontaneous hemorrhage, subacute bleeding, gram-negative sepsis, gram-positive infections, rickettsial, cytomegalovirus, CMV, varicella, hepatitis, histoplasma, malaria, mucin-secreting adenocarcinoma, placental abruption, acute fatty liver of pregnancy, transfusions, snake envenomation, liver disease, acute hepatic failure, leukemia, rheumatoid arthritis, Raynaud's disease, ulcerative colitis, Crohn disease, Crohn's disease, sarcoidosis, aortic aneurysms, acute renal allograft rejection

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INTRODUCTION

Background: Disseminated intravascular coagulation (DIC) is a complex systemic thrombohemorrhagic disorder involving the generation of intravascular fibrin and the consumption of procoagulants and platelets. The subcommittee on DIC of the International Society on Thrombosis and Hemostasis has suggested the following definition for DIC: "An acquired syndrome characterized by the intravascular activation of coagulation with loss of localization arising from different causes. It can originate from and cause damage to the microvasculature, which if sufficiently severe, can produce organ dysfunction".

DIC is seen in association with a number of well-defined clinical situations, including sepsis, major trauma, and abruptio placenta, and with laboratory evidence of the following:

- Procoagulant activation
- Fibrinolytic activation
- Inhibitor consumption
- Biochemical evidence of end-organ damage or failure

DIC is a pathophysiologic term describing a continuum of events that occur in the coagulation pathway in association with a variety of disease states. DIC occurs in acute and chronic forms.

Consider DIC in patients with one of the underlying disorders listed above, with evidence of decreased or decreasing platelet counts, and with any of the laboratory findings listed above.

As the sequelae of DIC can be devastating, early clinical suspicion and laboratory diagnosis are essential. This article provides essential guidelines for the appropriate diagnosis and clinical treatment of patients with DIC.

Pathophysiology: The pathophysiology of DIC involves the initiation of coagulation via endothelial injury or tissue injury and the subsequent release of procoagulant material in the form of cytokines and tissue factors. Interleukin-6 and tumor necrosis factor may be the most influential cytokines involved in coagulation activation (via tissue factor) and may be responsible for the end-organ damage that occurs. Further, in the setting of sepsis, neutrophils and their secretory products may promote platelet-mediated fibrin formation.

Two proteolytic enzymes, thrombin and plasmin, are activated and circulate systemically. Their balance determines a bleeding or thrombotic tendency. Thrombin cleaves fibrinogen to form fibrin monomers. Thrombin ultimately potentiates the coagulation cascade and leads to small- and large-vessel thrombosis, with resultant organ ischemia and organ failure. Regulatory mechanisms of the coagulation cascade, such as tissue factor pathway inhibitor (TFPI), antithrombin III, and activated protein C, are largely defective. Plasmin, a component of the fibrinolytic system, is capable of degrading fibrin into measurable degradation products. Plasmin also activates complement. Plasmin and thrombin induce qualitative and quantitative platelet abnormalities.

Acute DIC is characterized by generalized bleeding, which ranges from petechiae to exsanguinating hemorrhage or microcirculatory and macrocirculatory thrombosis. This leads to hypoperfusion, infarction, and end-organ damage. In severe cases, patients may develop fever and a shocklike picture with tachycardia, tachypnea, and hypotension. Chronic DIC is characterized by subacute bleeding and diffuse thrombosis. Localized DIC is characterized by bleeding or thrombosis confined to a specific anatomic location. It has been associated with aortic aneurysms, giant hemangiomas, and hyperacute renal allograft rejection.

Frequency:

- **In the US:** Approximately 18,000 cases of DIC occurred in 1994. DIC may occur in 30-50% of patients with sepsis.

Mortality/Morbidity: Morbidity and mortality depend on both the underlying disease and the severity of coagulopathy. Assigning a numerical figure for DIC-specific morbidity and mortality is difficult. Below are examples of mortality rates in diseases complicated by DIC:

- Idiopathic purpura fulminans associated with DIC has a mortality rate of 18%.

- Septic abortion with clostridial infection and shock associated with severe DIC has a mortality rate of 50%.
- In the setting of major trauma, the presence of DIC approximately doubles the mortality rate.

Sex: Incidence is equal in males and females.

Age: No age predilection is known.

History: In addition to the symptoms related to the underlying disease process, ascertain history of blood loss and hypovolemia, such as gastrointestinal bleeding. Look for symptoms and signs of thrombosis in large vessels, such as deep venous thrombosis (DVT), and of microvascular thrombosis, such as renal failure. Bleeding from at least 3 unrelated sites is particularly suggestive of DIC.

- Epistaxis
- Gingival bleeding
- Mucosal bleeding
- Cough
- Dyspnea
- Confusion, disorientation
- Fever

Physical:

- Circulation
  - Signs of spontaneous and life-threatening hemorrhage
  - Signs of subacute bleeding
  - Signs of diffuse or localized thrombosis
- Central nervous system
  - Nonspecific altered consciousness/stupor
  - Focal deficits not usually present
- Cardiovascular system
  - Hypotension
  - Tachycardia
  - Circulatory collapse
- Respiratory system
  - Pleural friction rub

- Signs of adult respiratory distress syndrome (ARDS)

- Gastrointestinal system
  - Hematemesis
  - Hematochezia
- Genitourinary system
  - Signs of azotemia and renal failure
  - Acidosis
  - Hematuria
  - Oliguria
  - Metrorrhagia
  - Uterine hemorrhage
- Dermatologic system
  - Petechiae
  - Purpura
  - Hemorrhagic bullae
  - Acral cyanosis
  - Skin necrosis of lower limbs (purpura fulminans)
  - Localized infarction and gangrene
  - Wound bleeding and deep subcutaneous hematomas
  - Thrombosis

Causes: Causes of DIC can be classified as acute or chronic, systemic or localized. DIC may be the result of a single or multiple conditions.

- Acute DIC
  - Infectious
    - Bacterial (eg, gram-negative sepsis, gram-positive infections, rickettsial)
    - Viral (eg, HIV, cytomegalovirus [CMV], varicella, hepatitis)
    - Fungal (eg, histoplasma)
    - Parasitic (eg, malaria)
  - Malignancy
    - Hematologic (eg, acute myelocytic leukemias)
    - Metastatic (eg, mucin-secreting adenocarcinomas)

- Obstetric
  - Placental abruption
  - Amniotic fluid embolism
  - Acute fatty liver of pregnancy
  - Eclampsia
- Trauma
- Burns
- Motor vehicle accidents (MVAs)
- Snake envenomation
- Transfusion
- Hemolytic reactions
- Massive transfusion
- Liver disease - Acute hepatic failure
- Prosthetic devices
- Shunts (Denver, LeVeen)
- Ventricular assist devices

- Chronic DIC
  - Malignancies
    - Solid tumors
    - Leukemia
  - Obstetric
    - Retained dead fetus syndrome
    - Retained products of conception
  - Hematologic
    - Myeloproliferative syndromes
    - Paroxysmal nocturnal hemoglobinuria
  - Vascular
    - Rheumatoid arthritis
    - Raynaud disease
  - Cardiovascular - Myocardial infarction
  - Inflammatory

    - Ulcerative colitis
    - Crohn disease
    - Sarcoidosis
- Localized DIC
    - Aortic aneurysms
    - Giant hemangiomas (Kasabach-Merritt syndrome)
    - Acute renal allograft rejection
    - Hemolytic uremic syndrome

DIFFERENTIALS

Hemolytic Uremic Syndrome
Thrombocytopenic Purpura

Other Problems to be Considered:

Severe liver failure (most common differential disorder)
Idiopathic purpura fulminans
Primary fibrinolysis
Vitamin K deficiency
Hemolysis, elevated liver function, and low platelets (HELLP) syndrome in pregnancy

WORKUP

Lab Studies:

- Prothrombin fragment 1 and 2
    - Enzyme-linked immunosorbent assay (ELISA) quantitates levels of prothrombin fragment (PF) 1 and 2 in the circulations. This provides evidence of factor Xa generation and is easily performed.
    - Levels are abnormal in 90% of patients with DIC.
- D-dimer test
    - D-dimer is an antigen formed as a result of plasmin lysis of cross-linked fibrin clots.
    - The presence of this fragment documents the presence of thrombin (cross-linking) and plasmin (fibrinolysis).
    - This monoclonal antibody test has the greatest specificity and is a highly reliable test for diagnosis of DIC.
- Antithrombin III level
    - Functional antithrombin III levels decrease in DIC.
    - This synthetic substrate assay is a reliable and useful test for diagnosis and therapeutic monitoring.

- Fibrin and fibrinogen degradation products
    - Latex particle agglutination test is used to detect fibrinogen and fibrin degradation products (FDPs).
    - Degradation products increase as plasmin biodegrades fibrinogen and fibrin.
    - This test is not diagnostic of DIC, yet levels are elevated in 85-100% of patients.
- Fibrinopeptide A
    - ELISA or radioimmunoassay is used to measure fibrinopeptide A (FPA).
    - FPA is a breakdown product of fibrinogen, indicative of thrombin activity.
    - Levels are abnormal in 88% of patients with DIC.
- Platelet count
    - Platelet counts are invariably decreased. This is usually evident in the peripheral smear.
    - Functional deficits in platelets are often present, and further studies are not indicated.
- Fibrinogen
    - Thrombin-time–based assay is used to measure fibrinogen levels.
    - Levels usually are decreased in DIC.
    - Fibrinogen is an acute-phase reactant and initially may be elevated secondary to the primary disease.
- Prothrombin time
    - Prothrombin time (PT) tests the extrinsic and common pathways.
    - PT may be normal, prolonged, or shortened in DIC.
    - It is generally an unreliable test for diagnosis of DIC, and 50-75% of patients have prolonged values.
- Activated partial thromboplastin time
    - Activated partial thromboplastin time (aPTT) tests the intrinsic and common pathways.
    - Values are unpredictable in DIC.
    - It is an unreliable test for diagnosis of DIC, and 50-60% of patients will have prolonged values.
- Thrombin time
    - Thrombin time measures the conversion of fibrinogen to fibrin.
    - It should be prolonged in DIC.
- Protamine test
    - The protamine test is a paracoagulation test that detects fibrin monomers in plasma.
    - Fibrin web formation indicates a positive result.
    - This test should be positive in patients with DIC.
- Anemia

- Schizocytosis
- Decreased coagulation factors
    - Factor V
    - Factor VIII
    - Factor X
    - Factor XIII
    - Protein C
- Hemoglobinuria
- Hematuria
- Hematochezia
- No single diagnostic test exists for DIC. DIC is initially suggested by the following combination: a clinical condition consistent with DIC, thrombocytopenia (< 100 X 10^9/L), prolonged PT and aPTT, and presence of FDP/D-dimer. Other tests listed here also may help to exclude DIC.

Imaging Studies:

- Base diagnostic imaging on the underlying pathologic process as well as suspicion for areas of thrombosis and hemorrhage.
- Perform a bilateral perihilar soft-density chest radiograph if pulmonary injury is present.

Other Tests:

- Base other tests on the underlying pathologic process as well as suspicion for areas of thrombosis and hemorrhage.

Procedures:

- Base procedures on the underlying pathologic process as well as suspicion for areas of thrombosis and hemorrhage.
- Conduct invasive procedures with care because of bleeding complications. Procedures should follow the administration of clotting factor and platelet repletion.

Prehospital Care: Monitor vital signs, assess and document extent of hemorrhage and thrombosis, correct hypovolemia, and administer basic hemostatic procedures when indicated.

Emergency Department Care: The cornerstone of DIC management is treatment of the underlying disorder. The following supportive measures are essential:

- Continue prehospital measures.
- Attend to life-threatening issues such as airway compromise or severe hemorrhage.
- Determine the underlying cause of the patient's DIC and initiate therapy. Obtain appropriate imaging studies if necessary.
- Draw specimens for appropriate coagulation studies and other diagnostic laboratory tests.
- Begin anticoagulant therapy if indicated (see Medication for indications).

- Replace blood products as indicated (see Medication).
  - RBC transfusion (ie, packed RBCs [PRBCs])
  - Platelet concentrates
  - Fresh frozen plasma (FFP)
  - Cryoprecipitate
- Antithrombin III concentrate

Consultations:

- Consult a hematologist for assistance with diagnosis and management.
- Consult a transfusion specialist or a blood bank; determine the availability of general and specialized blood products that may be necessary for the acute management of fulminant DIC.
- Consult a critical care specialist if multiple organ failure is present.
- Early consultation is indicated for this complicated, life-threatening condition. Obtain other subspecialty consultations as indicated by the patient's primary diagnosis.

Therapy should be based on etiology and aimed at eliminating the underlying disease. Therapy should be appropriately aggressive for the patient's age, disease, and the severity and location of hemorrhage/thrombosis. Treatment for acute DIC includes anticoagulants, blood components, and antifibrinolytics.

Hemostatic and coagulation parameters should be monitored continuously during treatment. Base therapeutic decisions on clinical and laboratory evaluation of hemostasis. In cases of low-grade DIC, therapy other than supportive care may not be warranted or may include antiplatelet agents or subcutaneous heparin; treatment decisions should be based on clinical and laboratory evaluation of hemostasis. Activated human protein C has been shown to reduce the rate of mortality in the setting of severe sepsis for patients at high risk for death; this should be used cautiously and

appropriately, following guidelines for administration.

Drug Category: *Anticoagulant agents* -- These agents are used in the treatment of clinically evident intravascular thrombosis when the patient continues to bleed or clot 4-6 h after initiation of primary and supportive therapy. Thrombosis can present as purpura fulminans or acral ischemia. Take special precaution in obstetric emergencies or massive liver failure. The anti-inflammatory properties of antithrombin III may be particularly useful in DIC secondary to sepsis.

Drug Name	Heparin -- Use and dose of heparin is based on severity of DIC, underlying cause, and extent of thrombosis. Monitoring results of therapy is mandatory. Heparin augments antithrombin III activity and prevents conversion of fibrinogen to fibrin. Does not actively lyse but inhibits further thrombogenesis. Prevents reaccumulation of a clot after spontaneous fibrinolysis.
Adult Dose	80-100 U/kg SC q4-6h or 20,000-30,000 U/d IV continuous infusion
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; subacute bacterial endocarditis; active bleeding; history of heparin-induced thrombocytopenia
Interactions	Digoxin, nicotine, tetracycline, and antihistamines may decrease effects; NSAIDs, aspirin, dextran, dipyridamole, and hydroxychloroquine may increase toxicity
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Monitor for localized bleeding or hematoma; may aggravate hemorrhage; in neonates, preservative-free heparin is recommended to avoid possible toxicity (gasping syndrome) by benzyl alcohol, which is used as preservative; caution in severe hypotension and shock

Drug Name	Antithrombin III (ATnativ, Thrombate III) -- Used for moderately severe to severe DIC or when levels are depressed markedly. Alpha 2-globulin that inactivates thrombin, plasmin, and other serine proteases of coagulation, including factors IXa, Xa, XIa, XIIa, and VIIa. These effects inhibit coagulation.
Adult Dose	Total units = (desired level - initial level) (0.6 X total body weight kg) IV q8h with a desired level >125% or loading dose of 100 U/kg IV over 3 h; followed by continuous infusion of 100 U/kg/d
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	Increases anticoagulation effects of heparin
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in hypotension; despite measures taken to delete infectious agents from human product, potentially still can transmit disease or contain unknown infectious agents

Drug Category: *Recombinant Human Activated Protein C* -- These agents inhibit factors Va and VIIIa of the coagulation cascade. They may also inhibit plasminogen activator inhibitor-1 (PAI-1).

Drug Name	Drotrecogin alfa-activated (Xigris) -- Indicated for reduction of mortality in patients with severe sepsis associated with acute organ dysfunction and at high risk of death. Recombinant form of human activated protein C that exerts antithrombotic effect by inhibiting factors Va and VIIIa. Has indirect profibrinolytic activity by inhibiting PAI-1 and limiting formation of activated thrombin-activatable-fibrinolysis-inhibitor. May exert anti-inflammatory effect by inhibiting human tumor necrosis factor (TNF) production by monocytes, blocking leukocyte adhesion to selectins, and limiting thrombin-induced inflammatory responses within microvascular endothelium.
Adult Dose	24 mcg/kg/h IV by continuous infusion over 96 h
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; increased risk of bleeding (eg, active internal bleeding, recent hemorrhagic stroke, recent intraspinal or intracranial surgery, recent or current trauma, presence of epidural catheter, intracranial neoplasm, cerebral herniation, severe head trauma)
Interactions	None reported; coadministration with drugs that affect hemostasis may increase risk of bleeding (eg, warfarin, heparin, thrombolytics, glycoprotein IIb/IIIa inhibitors)

Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Bleeding is most common serious adverse effect; caution with conditions that increase risk of bleeding including INR >3, concurrent therapeutic heparin (>15 U/kg/h), within 6 wk of GI bleeding episode, within 3 d of thrombolytic therapy, within 7 d of platelet inhibitors administration, within 3 mo of ischemic stroke, intracranial arteriovenous malformation or aneurysm, known bleeding diathesis, chronic severe hepatic disease; stop infusion if clinically significant bleeding occurs; caution with thrombocytopenia (<50 X 10^9/L); chronic severe hepatic disease and known bleeding diathesis not associated with the acute coagulopathy related to sepsis

Drug Category: *Blood components* -- Blood components are used to correct abnormal hemostatic parameters. These products should be considered only after initial supportive and anticoagulant therapy. Washed PRBCs and platelet concentrates are considered safe in uncontrolled DIC. Specialized blood components (cryoprecipitate, FFP) may interfere with or improve DIC.

Drug Name	PRBCs (washed) -- Preferred to whole blood since they limit volume, immune, and storage complications. Obtain PRBCs after centrifugation of whole blood. Use washed or frozen PRBCs in individuals with hypersensitivity transfusion reactions.
Adult Dose	1 unit of PRBCs should raise hemoglobin by 1 g/dL or raise hematocrit by 3%
Pediatric Dose	Not established
Contraindications	Competent adult or legal guardian may refuse blood product; immediate consultation with hospital ethical and legal staff is mandated
Interactions	None reported
Pregnancy	A - Safe in pregnancy
Precautions	Use CMV-negative units or filtered ones; transfusion reactions and transmission of blood-borne pathogens are a concern; benefits should outweigh risks associated with such products

Drug Name	Platelet concentrates (Random or single donor, pheresis units) -- Considered safe for use in acute DIC.
Adult Dose	Based on platelet count and clinical situation
Pediatric Dose	Not established
Contraindications	Competent adult or legal guardian may refuse blood product; immediate consultation with hospital ethical and legal staff mandated
Interactions	None reported
Pregnancy	A - Safe in pregnancy
Precautions	Platelets should be CMV-negative or the pheresis units from single donors filtered; benefits should outweigh risks associated with such products

Drug Name	Fresh frozen plasma (FFP) -- This treatment entails removing blood from body, spinning it to separate cells from plasma, and replacing cells suspended in fresh frozen plasma, albumin, or saline. Contains coagulation factors as well as protein C and protein S. Can be performed using either 2 large-bore peripheral IV sites or multiple lumen central line. Recommended with active bleeding and fibrinogen <100 mg/dL.
Adult Dose	15-20 mL/kg IV or based on clinical situation
Pediatric Dose	Administer as in adults
Contraindications	Documented hypersensitivity
Interactions	None reported
Pregnancy	A - Safe in pregnancy
Precautions	Viral contamination and infection are remotely possible but unlikely

Drug Name	Cryoprecipitate or fibrinogen concentrates -- Not commonly recommended except when fibrinogen is needed.
Adult Dose	Each bag contains 80-100 U of factor VIII; base administration on fibrinogen levels, antithrombin III levels, and coagulation parameters
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; uncontrolled DIC with abnormal antithrombin III levels

Interactions	None reported
Pregnancy	A - Safe in pregnancy
Precautions	Benefits should outweigh risks associated with transfusion therapy; viral contamination and infection are remotely possible although unlikely because of prescreening

Drug Category: *Antifibrinolytic agents* -- These agents are used only after all other therapeutic modalities have been tried and deemed unsuccessful. Increase in circulating plasmin and laboratory evidence of decreased plasminogen should be documented. Antifibrinolytics may be useful in cases of DIC secondary to hyperfibrinolysis associated with acute promyelocytic leukemia and other forms of cancer.

Drug Name	Aminocaproic acid (Amicar) -- Inhibits fibrinolysis via inhibition of plasminogen activator substances and, to a lesser degree, through antiplasmin activity. Main problem is that thrombi that form during treatment are not lysed, and clinical significance of reducing bleeding is uncertain.
Adult Dose	Load 5-10 g IV slowly; followed by 2-4 g/h IV; not to exceed 30 g/d
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; evidence of active intravascular clotting process; because aminocaproic acid can be fatal in patients with DIC, important to differentiate between hyperfibrinolysis and DIC
Interactions	Estrogens may cause increase in clotting factors, leading to hypercoagulable state
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Do not administer unless definite diagnosis of hyperfibrinolysis has been made; caution in cardiac, hepatic, or renal disease

Drug Name	Tranexamic acid (Cyklokapron) -- Used as alternative to aminocaproic acid. Inhibits fibrinolysis by displacing plasminogen from fibrin.
Adult Dose	Nonstandardized dosing: 25 mg/kg PO tid/qid; 1-2 g IV q8-12h
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; ongoing DIC and CNS involvement
Interactions	None reported
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Adverse effects include gastrointestinal and visual disturbances and hypotension; caution in renal impairment

Further Inpatient Care:

- Most patients with acute DIC require critical care treatment appropriate for the primary diagnosis, occasionally including emergent surgery.
- Assessment of severity of DIC (DIC score)
  - A DIC scoring system has been proposed by Bick to assess the severity of the coagulopathy as well as the effectiveness of therapeutic modalities.
  - Clinical and laboratory parameters are measured with regularity (every 8 h).

Further Outpatient Care:

- Patients who recover from acute DIC should follow up with their primary physician or a hematologist.
- Patients with low-grade or chronic DIC may be treated by a hematologist on an outpatient basis after initial assessment and stabilization.

In/Out Patient Meds:

- Outpatient medications may include antiplatelet agents for those with low-grade DIC and/or antibiotics appropriate to the primary diagnosis.

Transfer:

- Patients who are stable enough for transfer should be referred expeditiously to centers with appropriate critical care and subspecialty expertise, such as hematology, blood bank, or surgical centers.

Complications:

- Acute renal failure
- Life-threatening thrombosis and hemorrhage (in patients with moderately severe to severe DIC)
- Cardiac tamponade
- Hemothorax
- Intracerebral hematoma
- Gangrene and loss of digits
- Death

Prognosis:

- The prognosis is influenced most by the underlying condition that led to DIC and the severity of the DIC.

MISCELLANEOUS

Medical/Legal Pitfalls:

- Failure to establish early clinical suspicion and make a laboratory diagnosis, as the sequelae of DIC can be devastating
- Failure to focus on treating the underlying cause of DIC when the thromboembolic and bleeding complications of the process seem to be dominating the clinical picture

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