

DIC · Disseminated Intravascular Coagulation

DIC means clotting and bleeding happen at the same time. The underlying trigger is the real problem.

THE ONE-LINE TRUTH

The blood's clotting system turned on everywhere at once.

The chain — how it unfolds

1. A big trigger activates clotting everywhere — why: Sepsis, major trauma, cancer, placental abruption, amniotic fluid a traveling clot lodging in a vessel (embolism), severe pancreatitis, transfusion reaction, or envenomation releases tissue factor or other clotting activators into the bloodstream, turning on the clotting cascade system-wide instead of just at one injury site.
2. Tiny clots form in small blood vessels throughout organs — why: The clotting signal spreads faster than brake proteins (protein C, antithrombin) can stop it, so fibrin clots form in capillaries in kidneys, lungs, brain, liver, and skin.
3. Organs start to fail from blocked blood flow — why: Those tiny clots block oxygen and nutrient delivery in capillaries, so kidneys stop filtering (creatinine rises, urine drops), lungs struggle (low oxygen, need for ventilator), brain function declines (confusion, lethargy), and liver enzymes rise.
4. Clotting supplies get used up — why: Platelets and fibrinogen get trapped in clots faster than the bone marrow and liver can make new ones, so blood levels drop—platelet count falls (often below 50,000), fibrinogen drops (often below 100 mg/dL), and clotting times get longer (PT and PTT rise).
5. The body breaks down clots, releasing D-dimer — why: Enzymes (plasmin) try to dissolve clots to reopen blocked vessels, chopping fibrin into small fragments called D-dimer—so D-dimer levels skyrocket (often above 5,000 ng/mL), signaling that massive clotting and breakdown are happening at once.

6. Bleeding starts at the same time clotting continues inside — why: With platelets and fibrinogen depleted, blood cannot clot normally where it should—so the patient oozes from IV sites, bleeds from the gums, has blood in urine or stool, or develops large bruises (purpura), even though clots keep forming deep in organs.
7. Organs fail and bleeding becomes uncontrollable — why: The trigger keeps driving more clotting, depleting supplies further, so organs shut down (kidneys, lungs, brain) and bleeding worsens—leading to shock, multi-organ failure, and death unless the underlying trigger is stopped.
8. The only way to stop DIC is to treat the trigger — why: DIC is not the disease—it is the body's broken response. You must fix the upstream cause (antibiotics for sepsis, surgery for trauma or placental abruption, chemotherapy for cancer, delivery for pregnancy emergency) or the clotting-and-bleeding spiral continues no matter how much blood or platelets you give.

Spot it — what you see, and why

- Bleeding from many places at once (gums, nose, IV sites, under the skin, in urine or stool): Your clotting factors and platelets are being used up so fast all over your body that your blood can't clot where it needs to anymore, like using up all your glue before you finish the craft project.
- Purple or red bruises and spots on the skin (petechiae and purple bleeding blotches (purpura)): Tiny blood vessels leak because there aren't enough platelets left to plug the holes, so red blood cells escape into the skin and make dark marks.
- Blood clots forming in small blood vessels (causing fingers, toes, or organs to stop working right): The clotting system gets turned on everywhere at once, so clots form inside tiny blood vessels and block blood flow to tissues, like dozens of small traffic jams all over the body.
- Confusion, trouble thinking clearly, or passing out: Either tiny clots are blocking blood flow in the brain or bleeding is happening inside the brain, both stopping oxygen and nutrients from reaching brain cells.
- Trouble breathing or fast breathing: Clots or bleeding in the lungs make it hard for oxygen to pass into the blood, or the lungs are damaged by the clots blocking their tiny blood vessels.
- Very little or no urine coming out: Tiny clots clog the filters in the kidneys so they can't make urine, or bleeding damages the kidney tissue itself.

Red flags — act now

- Bleeding that won't stop from multiple sites at the same time

- Sudden confusion, seizure, or loss of consciousness
- Trouble breathing or very low oxygen levels
- No urine output or very dark urine
- Severe belly pain or signs of shock (fast heart rate, low blood pressure, cold and sweaty skin)

Treat it — in this order

1. Find and fix the underlying trigger — give antibiotics for sepsis, deliver the baby for placental abruption or amniotic fluid a traveling clot lodging in a vessel (embolism), start chemotherapy for acute promyelocytic leukemia, remove dead tissue for severe pancreatitis or trauma, or stop the transfusion for transfusion reaction → The trigger is what turned on the clotting factory and told it to run out of control. Stopping the trigger shuts off the signal, so the body stops making tiny clots everywhere. Without the trigger, the clotting system can reset itself.
2. Transfuse platelets if platelet count drops below 50,000 per microliter AND the patient is actively bleeding or about to have surgery or a procedure. Give platelets if count is below 20,000 even without bleeding. → Platelet transfusion puts new platelets into the bloodstream so the body has enough pieces to form clots at bleeding sites. This buys time for the body to stop the trigger and start making its own platelets again.
3. Transfuse fresh frozen plasma (FFP) — usually 10–15 mL per kilogram of body weight — if the patient has active bleeding AND the PT/INR is prolonged → FFP contains all the clotting proteins (factors) the body used up. Replacing these proteins helps the blood clot at bleeding sites and slows down the bleeding. FFP gives the liver raw materials to keep making new clotting proteins.
4. Transfuse cryoprecipitate — usually 1 unit per 10 kg of body weight — if fibrinogen drops below 100 mg/dL → Cryoprecipitate is concentrated fibrinogen (the glue that holds clots together). Giving cryoprecipitate raises fibrinogen quickly so the blood can form stable clots. Without fibrinogen, clots fall apart and bleeding never stops.
5. Give low-dose heparin (prophylactic or therapeutic dose) ONLY in specific situations — cancer-triggered DIC with more clotting than bleeding, or purple bleeding blotches (purpura) fulminans with limb-threatening clots — and ONLY after discussing with a hematologist → Heparin blocks part of the clotting cascade, so fewer new tiny clots form in small vessels. This gives the body's natural anti-clotting system time to catch up and stop runaway clotting. Heparin is ONLY helpful when clotting is the main problem, not bleeding.

Your top priorities at the bedside

1. Stop any active bleeding by applying direct pressure to bleeding sites and avoid any needle sticks, injections, or procedures unless absolutely life-saving. — why: The patient has almost no clotting factors (proteins that stop bleeding) or platelets (cells that form clots) left. Every needle poke creates a new bleeding site that will not stop on its own.
2. Monitor for signs of shock every 15 minutes: check blood pressure, heart rate, skin temperature and color, capillary refill, and level of consciousness. — why: Patients can bleed internally into the belly, chest, or brain where you cannot see it. Shock signs like low blood pressure, fast heart rate, cold sweaty skin, and confusion mean they are losing too much blood volume and organs are not getting enough oxygen.
3. Measure and record all blood loss from every site including IV sites, gums, nose, urine, stool, and drains; save any blood-soaked dressings or pads for the provider to see. — why: Accurate measurement of total blood loss helps the provider decide how many units of blood products to transfuse. Seeing the actual bleeding helps confirm DIC instead of other bleeding disorders.