

DIC · Disseminated Intravascular Coagulation

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

THE STORY IN ONE SENTENCE

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

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

Normal anatomy — the body's job

Your blood clots only where you need it—like plugging a cut. A clot forms, seals the leak, then your body breaks it down when healing is done. Special brake proteins (protein C and antithrombin) keep clotting from spreading beyond that one spot. Keep this picture in your head, because every weird finding in DIC is a change from this normal job.

BODY'S JOB

Normally, when a blood vessel tears, platelets (tiny sticky cells) rush to the spot and stick together. At the same time, clotting proteins in your blood form long strands of fibrin (like a net) that hold the platelet plug in place. Once the vessel heals, your body releases enzymes that break down the clot into small pieces called D-dimer, which get cleared away. Brake proteins (protein C, antithrombin, and tissue factor pathway inhibitor) constantly patrol your blood to make sure clotting stays local and does not spread. This balance—clotting where you need it, stopping it everywhere else—keeps blood flowing smoothly while still sealing leaks fast.

1. A blood vessel tears (cut, puncture, or internal injury).
2. Platelets stick to the tear and clump together to form a soft plug.
3. Clotting proteins activate in a cascade (one triggers the next) and weave fibrin strands through the platelet plug to make it strong.
4. The clot seals the leak and stops bleeding.

5. Brake proteins (protein C, antithrombin) stop the clotting signal from spreading beyond the injury site.
6. Once the vessel heals, enzymes break down the clot into small fragments (D-dimer) that wash away in the blood.
7. Platelets and clotting proteins return to normal levels, ready for the next injury.

What breaks

Something big—like a severe infection, major trauma, cancer, or pregnancy emergency—triggers clotting everywhere at once. The brake proteins cannot keep up. Tiny clots form in small blood vessels throughout your organs, using up all your clotting supplies and leaving nothing for normal bleeding control.

CLINICAL TERM

Widespread activation of the coagulation cascade (clotting system) with simultaneous consumption of platelets, fibrinogen, and clotting factors, leading to microvascular clot formation (thrombosis) (tiny clots in small vessels) and bleeding from depletion of clotting components.

How the body compensates

The body tries to keep blood pressure and oxygen delivery going by forming clots and pushing blood harder. Vital signs can look stable for a while, hiding how close the system is to collapse. The crash point—bleeding to death, organ failure, or both—can come suddenly once clotting supplies run out.

1. {'what': 'Heart rate rises', 'why': 'The heart beats faster to pump more blood and make up for low oxygen delivery to organs (from clots blocking small vessels).'}
2. {'what': 'Blood pressure may stay normal at first', 'why': 'Blood vessels squeeze (vasoconstriction) to keep pressure up, masking how much the system is struggling.'}
3. {'what': 'Liver works overtime to make new clotting proteins', 'why': 'The liver tries to replace consumed fibrinogen and clotting factors, but cannot keep up with the rate they are being used in clots.'}
4. {'what': 'Bone marrow releases young platelets', 'why': 'The marrow pushes out immature platelets to replace those trapped in clots, but production is too slow.'}
5. {'what': 'The body breaks down clots to reopen vessels', 'why': 'Enzymes (plasmin) dissolve fibrin clots to restore blood flow, releasing D-dimer fragments—but this also worsens bleeding because it removes any clots that were helping.'}

6. {'what': 'Vital signs crash suddenly', 'why': 'Once clotting supplies are fully depleted, the body cannot compensate anymore. Blood pressure drops (shock), organs fail (kidneys stop making urine, lungs need a ventilator, brain function declines), and bleeding becomes uncontrollable.'}
7. {'what': 'A stable-looking patient can tip fast', 'why': 'Compensation hides the true severity. By the time vital signs crash, the window to reverse DIC is narrow—you must treat the trigger early, before supplies run out.'}

Domino effect

1. A big trigger activates clotting everywhere — 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 — 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 — 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 — 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 — 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 — 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 — 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 — 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.

Symptoms · what the patient feels, 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.
- Belly pain or signs of organ damage (liver, intestines): Clots block blood flow to the intestines or liver, starving those tissues of oxygen, or bleeding happens inside them and causes swelling and pain.
- Blood oozing slowly and continuously from wounds or surgical cuts that won't stop: The body has run out of enough clotting factors and platelets to form a stable clot, like trying to build a wall with too few bricks.

Focused exam

- Bleeding from multiple sites at the same time — gums, IV sites, surgical wounds, urinary catheter, nose, or under the skin as bruises: In DIC, the body uses up all its clotting pieces (platelets and fibrinogen) trying to stop tiny clots everywhere, so there are none left to stop real bleeding. When you see bleeding from more than one place at once, DIC is happening right now.
- Purple or black patches on fingers, toes, nose, or ears that do not turn white when you press on them (purpura fulminans or acral starved blood flow (ischemia)): Tiny clots plug up small blood vessels and choke off blood flow to the skin and tissue. When tissue turns purple or black, it means those areas are dying because no blood can reach them. This proves DIC is blocking vessels in real time.
- Low blood pressure (systolic under 90 mmHg), fast heart rate (over 110), confusion, extreme sleepiness, or no urine output: These signs mean the body is in shock — not enough blood is reaching the brain, kidneys, and other organs. In DIC, shock happens because bleeding steals blood volume AND tiny clots block blood flow at the same time.
- Belly pain, vomiting blood (bright red or coffee-ground color), or black tarry stool: Tiny clots in the blood vessels feeding the stomach and intestines damage the lining, causing bleeding from the gut. When you see belly pain plus blood coming out either end, DIC is attacking the digestive system.
- Trouble breathing, oxygen saturation under 90% on room air, or coughing up blood: Tiny clots in the lung blood vessels block oxygen from reaching the bloodstream. When the lungs cannot get oxygen into the body, organs start failing. This is life-threatening organ damage from DIC.
- New confusion, trouble speaking, or weakness on one side of the body: Tiny clots can block blood vessels in the brain, causing a stroke. New brain symptoms in a patient with bleeding elsewhere mean DIC is hitting the brain right now.

Diagnostics · every test answers a question

- Platelet count → Are there enough platelets left to form clots and stop bleeding?
- Fibrinogen level → Is there enough fibrinogen (the glue that holds clots together) left in the blood?
- Prothrombin time (PT) and International Normalized Ratio (INR) → How long does it take for the blood to start clotting?
- D-dimer → Is the body breaking down clots as fast as it is making them?
- Peripheral blood smear → Are red blood cells getting shredded as they squeeze past tiny clots in small vessels?

- ISTH DIC score (International Society on Clot formation (Thrombosis) and Haemostasis scoring system) → Does this patient meet the official criteria for DIC?
- Basic metabolic panel (BMP) and creatinine → Are the kidneys still filtering blood and making urine?
- Liver function tests (ALT, AST, bilirubin) → Is the liver being damaged by tiny clots or shock?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.

- Support blood pressure with IV fluids (crystalloid like normal saline or lactated Ringer's) and vasopressors (norepinephrine) if fluids alone do not bring pressure up → IV fluids replace lost blood volume from bleeding. Vasopressors squeeze blood vessels to push blood pressure up when fluids are not enough. Keeping blood pressure up keeps blood flowing to the brain, kidneys, and heart.
- Monitor platelet count, fibrinogen, PT/INR, D-dimer, ISTH DIC score, urine output, creatinine, and liver enzymes every 6–12 hours in the ICU → Tracking these numbers tells you if the trigger is under control, if transfusions are working, and if organs are starting to recover or getting worse. Trends (going up or down) guide whether to give more blood products, escalate treatment, or keep the same plan.

Medications

Platelet transfusion · Blood component replacement

Gives the body fresh platelets (tiny cell pieces that plug holes in blood vessels) when your own platelets have been used up. In DIC, clots form everywhere at once and burn through all your platelets, so bleeding starts. Fresh platelets let wounds close again.

1. Donor platelets are separated from whole blood and stored in a bag.
2. Platelets are transfused through an IV line into your bloodstream.
3. New platelets flow to bleeding spots and clump together to form a plug.
4. The plug stops small-vessel bleeding while you treat the underlying cause of DIC.

SAFETY WATCH

Usually given only if platelet count drops below 20,000, or below 50,000 and the person is actively bleeding or needs surgery — otherwise you may be feeding the clotting fire. · Watch for allergic reactions: hives, fever, chills during the transfusion. · Risk of fluid overload (too much volume in the blood) if given too fast, especially in people with weak hearts or kidneys. · Small infection risk from any blood product, though screened carefully.

Fresh frozen plasma (FFP) · Blood component replacement

Replaces clotting factors (proteins the liver makes that work in a chain reaction to form clots). In DIC these factors are consumed by constant clotting, so bleeding can't stop. FFP refills all the factors at once so the clotting steps can work again.

1. Plasma (the liquid part of blood with all clotting factors) is frozen right after donation to preserve the proteins.
2. The plasma is thawed and transfused through an IV.

3. Clotting factors (II, V, VII, VIII, IX, X, XI, XIII and others) enter your bloodstream immediately.
4. These factors join the clotting cascade so clots can form where needed and bleeding slows.

SAFETY WATCH

Only give if there is active bleeding AND clotting tests show the factors are low (prolonged PT/aPTT) — do not give 'just in case,' because extra clotting factors can worsen the clotting problem. · Risk of fluid overload — each unit adds volume, so watch for shortness of breath and leg swelling. · Allergic reactions possible (itching, rash, fever). · Rare risk of lung injury (TRALI — fluid leaks into the lungs within 6 hours of transfusion, causing sudden breathing trouble). · Small infection risk, though screening is rigorous.

Cryoprecipitate · Blood component replacement

Delivers concentrated fibrinogen (a sticky protein that forms the mesh of a clot) plus Factor VIII and von Willebrand factor. In DIC, fibrinogen is consumed faster than the liver can make it, so clots fall apart. Cryoprecipitate quickly raises fibrinogen so clots can hold together.

1. Plasma is slowly thawed; proteins that precipitate out (settle at the bottom) are collected — this is cryoprecipitate.
2. The concentrate is transfused through an IV.
3. Fibrinogen levels in your blood rise quickly.
4. When platelets plug a hole, fibrinogen is converted to fibrin, forming a strong net that stops bleeding.

SAFETY WATCH

Give only if fibrinogen is very low (usually below 100 mg/dL) and bleeding is active — do not give routinely. · Allergic reactions can occur (hives, itching, fever). · Fluid overload is less likely than with FFP because the volume is smaller, but still watch in people with heart or kidney problems. · Small infection risk from any blood product.

Unfractionated heparin (low dose) · Anticoagulant (clot blocker)

Blocks a clotting protein called thrombin so new clots stop forming. Used ONLY in certain DIC cases (mainly some cancers) where clotting is the main problem and bleeding is mild or absent. It tries to interrupt the clotting storm without making bleeding worse.

1. Heparin is given as a continuous IV drip or injections under the skin.
2. Heparin sticks to antithrombin, a protein in your blood.
3. The heparin-antithrombin complex inactivates thrombin and Factor Xa (two key clotting enzymes).

4. New clot formation slows, reducing the consumption of platelets and clotting factors.

SAFETY WATCH

Use ONLY in select cases, typically cancer-related DIC with more clotting than bleeding, after talking with a blood specialist (hematologist). · Can cause or worsen bleeding — watch for blood in urine or stool, new bruises, bleeding gums. · Check platelet count regularly; rarely, heparin triggers the immune system to destroy platelets (HIT — heparin-induced a low platelet count (thrombocytopenia)), causing dangerous clots. · NOT used routinely in most DIC; standard teaching is to treat the cause and replace what is missing, not to give blood thinners.

Tranexamic acid (TXA) · Antifibrinolytic (clot stabilizer)

Stops the body from breaking down clots too quickly by blocking plasmin (an enzyme that dissolves fibrin). In some bleeding conditions TXA is helpful, but in DIC it is usually NOT recommended because it can trap clots in tiny vessels and worsen organ damage.

1. TXA is given by IV or by mouth.
2. It binds to plasminogen (the inactive form of plasmin) and stops it from becoming active.
3. Without plasmin, fibrin clots stay intact longer.
4. In DIC, this can trap clots in organs and block blood flow, so TXA is generally avoided unless bleeding is life-threatening and clotting is not the main problem.

SAFETY WATCH

Generally AVOIDED in DIC with active widespread clotting — it can make clots stick around and block blood flow to kidneys, lungs, and brain. · May be considered only in very specific situations (e.g. severe bleeding with fibrinolysis dominant) under close specialist guidance. · Can cause nausea, diarrhea. · Rarely causes seizures, especially at high doses or in people with kidney problems.

Recombinant activated protein C and antithrombin concentrates · Natural anticoagulant replacement

These are lab-made versions of proteins your body uses to slow clotting. The idea was to refill these natural brakes in DIC. However, large studies showed they do not improve survival and may increase bleeding, so they are NOT recommended for routine use in DIC.

1. The proteins would be given by IV infusion.
2. Activated protein C would inactivate Factors Va and VIIIa, slowing clot formation.
3. Antithrombin would bind and inactivate thrombin and Factor Xa.
4. In theory, this would calm the clotting storm.

SAFETY WATCH

NOT recommended for routine use in DIC — withdrawn or not approved in many countries after trial results. · Bleeding risk is significant. · Very expensive with no proven benefit. · Do not use outside of research settings or very rare, specific cases under expert guidance.

Red flags

- 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)

Nursing priorities

1. Stop any active bleeding by applying direct pressure to bleeding sites and avoid any needle sticks, injections, or procedures unless absolutely life-saving. — 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. — 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. — 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.
4. Watch closely for new bleeding in dangerous places: check pupils and neurological status every hour for brain bleeding, listen to lung sounds for chest bleeding, and palpate the abdomen for swelling or pain from belly bleeding. — Bleeding into the brain causes stroke, seizures, or death. Bleeding into the lungs stops oxygen from getting in. Bleeding into the belly can cause shock. These hidden bleeds kill faster than visible bleeding.

5. Measure urine output every hour and report if less than 30 milliliters per hour or if urine is dark, bloody, or stops completely. — Tiny clots block the small blood vessels in the kidneys, causing acute kidney injury (sudden kidney failure). Catching this early means we can give fluids and medications to protect the kidneys before permanent damage happens.
6. Protect the patient from any new injury: keep bed rails padded, use electric razors only, use soft toothbrushes or mouth swabs, and do not take rectal temperatures or insert rectal tubes. — Even tiny injuries cause uncontrollable bleeding when platelets and clotting factors are used up. A small cut from a regular razor or trauma from a rectal thermometer can bleed for hours.
7. Administer blood products exactly as ordered: fresh frozen plasma for clotting factors, platelet transfusions, cryoprecipitate for fibrinogen (a specific clotting protein), and packed red blood cells for severe anemia from blood loss. — Transfusions replace the clotting factors and platelets that DIC used up. But we must balance this carefully because giving too much can make more clots form and make DIC worse.
8. Monitor oxygen levels continuously with pulse oximetry and watch for trouble breathing, fast breathing, or blue lips and fingers. — Tiny clots can block blood vessels in the lungs causing pulmonary embolism (a clot lodging in a lung artery) (clots in lung arteries) or ARDS (severe lung inflammation and fluid). Low oxygen means organs are dying and the patient may need a breathing tube and ventilator fast.

Sources

- International Society on Thrombosis and Haemostasis (ISTH) DIC guideline
- British Committee for Standards in Haematology (BCSH) 2009 DIC guideline
- American Society of Hematology (ASH)
- New England Journal of Medicine (NEJM) clinical review

Last reviewed: 2026-07-07