



Disseminated Intravascular Coagulation

DIC · Always secondary — find the trigger, support the patient

TREAT THE CAUSE
nothing else is definitive

WHAT IT IS *consumption and clotting at the same time*

THE MECHANISM

Systemic activation of coagulation causes **widespread microvascular thrombosis**, which **consumes platelets and clotting factors**, producing **bleeding**. Secondary fibrinolysis adds to it. The patient can be clotting and bleeding simultaneously.

THE TRIGGERS — ALWAYS LOOK

Sepsis (commonest) · **major trauma** and burns · **malignancy**, especially **acute promyelocytic leukemia** · **obstetric** emergencies (abruption, amniotic fluid embolism, HELLP) · severe pancreatitis · transfusion reaction · snake envenomation · heat stroke.

THE LABS

Falling platelets · **prolonged PT and aPTT** · **low fibrinogen** · **markedly raised D-dimer** · schistocytes on the film. **Trend them** — the direction of travel matters more than any single value.

SCORE IT *ISTH overt DIC score — a score of 5 or more is compatible with overt DIC*

Parameter	Value	Points
Platelet count	$\geq 100 \times 10^9/L$ · 50–100 · < 50	0 · 1 · 2
D-dimer / fibrin marker	No rise · moderate rise · strong rise	0 · 2 · 3
Prolongation of PT	< 3 s · 3–6 s · > 6 s	0 · 1 · 2
Fibrinogen	≥ 100 mg/dL · < 100 mg/dL	0 · 1

Only apply the score when there is an underlying condition known to be associated with DIC. If the score is under 5, it may be non-overt DIC — repeat the panel in 24–48 hours rather than dismissing it. **Fibrinogen is an acute-phase reactant**, so a "normal" value in a septic patient may already represent a substantial fall.

MANAGE IT *support is guided by phenotype and bleeding, not by numbers alone*

- 1** **Treat the underlying cause aggressively — this is the only definitive therapy.** Source control and antibiotics for sepsis, delivery for obstetric DIC, ATRA for APL. Blood products buy time; they do not treat DIC.
- 2** **Do not transfuse to correct numbers in a non-bleeding patient.** Product support is for **active bleeding** or before an invasive procedure. Reflex correction of an abnormal panel adds volume and risk without benefit.
- 3** **If bleeding: platelets** for a count under about $50 \times 10^9/L$ (under 20 if not bleeding but at high risk), **fresh frozen plasma** for significantly prolonged PT/aPTT, and **cryoprecipitate or fibrinogen concentrate** for **fibrinogen under 150 mg/dL**.
- 4** **If the phenotype is thrombotic** — purpura fulminans, digital ischemia, venous thromboembolism — use **therapeutic or prophylactic heparin**. All immobilized patients with DIC should have **VTE prophylaxis** unless actively bleeding.
- 5** **Repeat the panel every 6–12 hours** while unstable. DIC is a moving target, and the trend tells you whether the underlying treatment is working.

TWO SPECIAL CASES *where the usual rules change*

ACUTE PROMYELOCYTIC LEUKEMIA

A **hematological emergency**. Severe DIC with **hyperfibrinolysis** and a high early death rate from hemorrhage, especially intracranial. **Start ATRA immediately on clinical suspicion**, before cytogenetic confirmation. Support aggressively: **platelets above 30–50** and **fibrinogen above 150 mg/dL**. Avoid invasive procedures until corrected.

OBSTETRIC DIC

Onset is often abrupt and severe — abruption, amniotic fluid embolism, retained products, HELLP. **Definitive treatment is delivery and uterine evacuation**. Fibrinogen rises in pregnancy, so a value in the "normal" range may be dangerously low; use pregnancy-adjusted thresholds and involve obstetric hematology early.

DISTINGUISH IT FROM THE MIMICS *several conditions share the picture but need opposite treatment*

Condition	What separates it from DIC
TTP	Coagulation studies are normal — normal PT, aPTT and fibrinogen with schistocytes and profound thrombocytopenia. Treatment is plasma exchange , and plasma or platelet transfusion is not the answer.
Liver failure	Prolonged PT and low platelets, but factor VIII is normal or high in liver disease and low in DIC (it is consumed). D-dimer rises less dramatically. Splenomegaly and stigmata of chronic liver disease point away from DIC.
Dilutional coagulopathy	After massive transfusion or large-volume crystalloid. D-dimer is not markedly raised , and the history makes it obvious. Treat with targeted product replacement.
HIT	Thrombosis with a normal fibrinogen and a platelet fall 5–10 days into heparin exposure. Bleeding is unusual. Score with the 4Ts and stop all heparin.
Primary hyperfibrinolysis	Low fibrinogen and bleeding but platelets relatively preserved . Seen in prostate disease and some malignancies, and it is one of the few settings where antifibrinolytics are appropriate.

WHAT TO ORDER AND WHEN *a practical panel*

THE PANEL

Complete blood count with film · PT, aPTT, **fibrinogen**, **D-dimer** · renal and liver function · lactate and blood gas. Add **factor VIII** if liver disease is the competing diagnosis, and a **blood film for schistocytes** if a microangiopathy is possible.

THE RHYTHM

Repeat every 6–12 hours while unstable, and after each significant transfusion. A rising platelet count and fibrinogen with a falling D-dimer means the underlying cause is being controlled — that trend is the best bedside marker that your treatment is working.

PEARLS & PITFALLS

- DIC is never a primary diagnosis.** If you have made it and cannot name the trigger, you have not finished the workup.
- A normal fibrinogen does not exclude DIC** in sepsis or pregnancy — it is an acute-phase reactant and should be high.
- Do not chase the numbers** in a non-bleeding patient. Transfuse for bleeding or for procedures.
- Suspected APL means ATRA now**, not after the cytogenetics return. Delay costs lives to intracranial hemorrhage.
- Antifibrinolytics are generally avoided** in DIC, since they can worsen microvascular thrombosis, except in defined hyperfibrinolytic states.
- Give VTE prophylaxis** to the non-bleeding DIC patient — the thrombotic risk is real and frequently forgotten.

