Low platelets or ITP

Find what caused it — drug, infection, bone marrow disease, big spleen, or immune system attack.

THE STORY IN ONE SENTENCE

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“Low platelets or ITP — check if a drug caused it, bone marrow problem, or infection. ITP means low platelets only, nothing else wrong. Give steroids or IVIG if bleeding or platelets below 30,000.”

Normal anatomy — the body's job

Platelets are tiny cell fragments made in the bone marrow (the soft factory inside your bones) that travel through your blood and plug holes in blood vessels to stop bleeding. Normal count is 150,000 to 450,000 per microliter, and each platelet lives about 10 days before the body breaks it down and makes a new one.

BODY'S JOB

Platelets stick to tears or holes in blood vessel walls and clump together to form a plug, like a cork in a leak. They also release chemicals that help build a stronger clot on top of the plug. Without enough platelets, even tiny leaks cannot be sealed, so blood seeps under the skin (bruises) or leaks out (bleeding).

1. Bone marrow makes new platelets every day from big parent cells called megakaryocytes, which break apart into hundreds of tiny platelets.
2. Platelets flow through your bloodstream, waiting and watching for any damage to a blood vessel wall.
3. When a vessel tears or gets scratched, platelets rush to the spot, stick to the rough edge, and clump together to seal the hole.
4. After about 10 days, old platelets are filtered out by the spleen and liver, and the bone marrow makes new ones to replace them — a constant cycle that keeps the count steady.

What breaks

Low platelets happen when the bone marrow does not make enough, the immune system destroys them too fast, or the spleen traps too many and keeps them out of circulation. Sometimes several causes combine.

> **CLINICAL TERM**
> A low platelet count (Thrombocytopenia) (low platelet count). The main mechanisms are: decreased production (bone marrow failure from leukemia, aplastic anemia, chemotherapy, or toxins), increased destruction (immune a low platelet count [ITP], drug-induced immune destruction, or thrombotic microangiopathy like TTP or HUS), or sequestration (an enlarged spleen holding platelets inside instead of releasing them into the blood).

How the body compensates

The bone marrow tries to make platelets faster to keep up with destruction, and the body tries to clot faster at injury sites, but if the count drops too low or too fast, bleeding wins and the body cannot keep up. You can look stable until the count hits the danger zone and bleeding suddenly starts.

1. The bone marrow ramps up platelet production, making more megakaryocytes and releasing younger, larger platelets into the blood — you may see these on a blood smear.
2. If the cause is immune destruction, the spleen works overtime breaking down tagged platelets, but the marrow cannot keep pace if antibodies are strong.
3. At counts above 50,000, most people do not bleed from everyday bumps or cuts — the body still has enough platelets to seal small injuries.
4. Between 20,000 and 50,000, bruising becomes easier and nosebleeds or gum bleeding may start, but serious bleeding is still rare.
5. Below 10,000, the risk of spontaneous bleeding (especially brain bleeding (hemorrhage)) climbs sharply because there are not enough platelets to plug even tiny leaks in fragile vessels.
6. The body cannot warn you with pain or other symptoms until bleeding actually starts — so platelet count is the main early warning signal.

Domino effect

1. Platelets drop below normal (less than 150,000). — The bone marrow is not making enough, the immune system is destroying them, the spleen is trapping them, or clotting is using them up — so fewer platelets are available in the blood to stop leaks.

2. Tiny red or purple dots appear on the skin, especially on the legs (called pinpoint bleeding spots (petechiae)). — With fewer platelets, tiny capillaries under the skin leak a little bit of blood, and because there are not enough platelets to plug the holes, the blood stays under the skin as small dots.
3. Bruises form more easily and spread larger than normal (called purple bleeding blotches (purpura) or bruises (ecchymoses)). — When you bump into something, blood vessels tear and leak — normally platelets rush in and seal it fast, but with low platelets the leak keeps going and blood spreads under the skin, making a big bruise.
4. Bleeding starts from the gums, nose, or digestive tract; periods become very heavy. — Mucous membranes (the wet lining of the mouth, nose, gut, and uterus) have fragile vessels that break easily — without enough platelets, even normal wear and tear causes bleeding that does not stop on its own.
5. If platelets fall very low (below 10,000), bleeding can happen inside the brain, eyes, or other organs without any injury. — At extremely low counts, even the normal pressure of blood flow can push through vessel walls that have tiny weak spots, and there are not enough platelets to plug them — brain bleed is the most dangerous because it can cause stroke or death.
6. The team checks a blood smear, asks about medicines and infections, and may test bone marrow to find the cause. — Treatment depends on the cause: if a drug is destroying platelets, stop it; if it is ITP, give steroids or IVIG to calm the immune system; if bone marrow is failing, treat the underlying disease — so the diagnosis must be accurate.
7. If ITP is confirmed and platelets are very low or bleeding is active, steroids (prednisone or dexamethasone) or IVIG (intravenous immunoglobulin) are given to raise the count quickly. — Steroids quiet the immune attack on platelets, and IVIG floods the system with antibodies that block the spleen from destroying platelets — both buy time for the count to recover and bleeding to stop.
8. If the cause is not fixed or platelets stay low despite treatment, the spleen may be removed (splenectomy) or other immune drugs (rituximab, thrombopoietin agonists) are used. — The spleen is where most platelet destruction happens in ITP — removing it stops the destruction and lets platelets circulate normally; other drugs either block the immune attack or tell the bone marrow to make more platelets.

Symptoms · what the patient feels, and why

- Pinpoint red or purple dots on the skin (petechiae): Platelets are the cell pieces that plug tiny leaks in blood vessels. When there are too few, tiny blood vessels leak under the skin and leave small red or purple dots you can see.

- Easy bruising from small bumps: Without enough platelets to seal breaks in blood vessels, even a light bump lets blood spread under the skin, making a bruise that is bigger or appears more easily than normal.
- Bleeding gums or nosebleeds that are hard to stop: The mouth and nose have many delicate blood vessels close to the surface. When platelet counts are low, these vessels cannot seal quickly, so bleeding lasts longer than it should.
- Heavy or prolonged menstrual periods: The inner lining of the uterus sheds each month and needs platelets to stop the bleeding. Fewer platelets mean the body takes longer to clot and seal off the bleeding vessels.
- Blood in the urine or stool: Small blood vessels in the bladder or intestines can leak when platelet counts drop very low, releasing blood into urine or stool because the body cannot patch the tiny holes fast enough.
- Sudden, severe headache or confusion: Very low platelets can let bleeding happen inside the skull (intracranial bleeding (hemorrhage)). Blood pressing on brain tissue causes headache and confusion, and this is a medical emergency.
- No symptoms at all (found only on routine blood test): Many people with mild to moderate low platelets feel completely fine. The condition is discovered when a complete blood count (CBC) is done for another reason and shows fewer platelets than normal.

Focused exam

- Pinpoint bleeding spots (Petechiae) (tiny red or purple dots) on the skin, especially legs and lower body: Pinpoint bleeding spots (Petechiae) appear when platelets drop below about 50,000 per microliter and cannot plug tiny leaks in the smallest blood vessels (capillaries). These dots are the signature mark of very low platelets and tell you clotting is failing at the smallest level.
- Easy bruising (ecchymoses) with light bumps or no clear injury: Bruises form when blood leaks out of damaged vessels and there are not enough platelets to seal the break. Large bruises from minor touch mean platelet count is likely below 30,000 and the body cannot form normal clots.
- Bleeding from mucous membranes: gums, nose, heavy menstrual bleeding, or blood in urine or stool: Bleeding from body openings signals platelets below 20,000 and severe clotting failure. This warns that dangerous bleeding into the brain or gut could start soon, especially if platelets drop below 10,000.
- Normal-sized spleen on abdominal exam (no an enlarged spleen (splenomegaly)): ITP does not enlarge the spleen. A big spleen suggests the spleen is trapping and destroying platelets from liver disease, infection (like malaria or HIV), or lymphoma—not immune destruction. A normal spleen supports the diagnosis of ITP.

- Normal red blood cells and white blood cells on peripheral blood smear, with only platelets reduced: ITP destroys only platelets. If red cells look broken (schistocytes) or white cells are also low, think TTP (thrombotic thrombocytopenic purple bleeding blotches (purpura)), HUS (hemolytic uremic syndrome), or bone marrow failure instead. Isolated low platelets point toward ITP.
- No fever, no yellowing of the skin and eyes (jaundice) (yellow skin), no confusion or neurologic changes: ITP itself does not cause fever, broken red cells (jaundice), or brain dysfunction. If these signs appear with low platelets, suspect TTP or HUS—both need urgent plasma exchange. Their absence supports ITP.

Diagnostics · every test answers a question

- Complete blood count (CBC) with platelet count → How low are the platelets, and are red and white cells normal?
- Peripheral blood smear (manual review under microscope) → Are platelets truly low, or are they clumping? Are red cells broken? Are there cancer cells?
- HIV, hepatitis C, and hepatitis B serology → Is a chronic viral infection triggering the immune attack on platelets?
- Direct antiglobulin test (DAT or Coombs test) → Is the immune system also attacking red blood cells along with platelets?
- Quantitative immunoglobulins (IgG, IgA, IgM levels) → Does the patient have an immune deficiency that can cause low platelets?
- Bone marrow biopsy and aspirate → Is the bone marrow making platelets normally, or is something blocking production?
- Lactate dehydrogenase (LDH), haptoglobin, indirect bilirubin, and reticulocyte count → Are red blood cells being destroyed along with platelets, suggesting TTP or HUS?
- ADAMTS13 activity level (if TTP is suspected) → Is the enzyme that prevents clots in small vessels severely low, confirming TTP?

Treatment logic

- Stop any drug that can cause immune platelet destruction (heparin, quinine, sulfonamides, vancomycin, certain anticonvulsants, NSAIDs) → Removes the trigger that turned on the immune attack against platelets. Once the drug is cleared (usually within days), the antibodies fade and the bone marrow can restore platelet counts.

- Corticosteroids: prednisone 1 mg/kg/day orally for 2–4 weeks, or dexamethasone 40 mg/day for 4 days → Suppresses the immune system—especially B cells and macrophages in the spleen that make and use antibodies to tag platelets for destruction. Platelet count usually rises within 3–7 days. Current ASH and international guidelines recommend corticosteroids as first-line for adults and children with ITP and bleeding or platelet count below 20,000–30,000.
- Intravenous immunoglobulin (IVIG) 1 gram/kg as a single dose, or 0.4 gram/kg/day for up to 5 days → Floods the immune system with normal antibodies that block the receptors (Fc receptors) on macrophages in the spleen, so the macrophages cannot grab and destroy antibody-coated platelets. Also may neutralize the anti-platelet antibodies. Platelet count often jumps within 24–48 hours. Used for urgent situations, active bleeding, or to boost response to steroids.
- Platelet transfusion (only if platelet count below 10,000 with severe bleeding, or below 50,000 before urgent surgery or during CNS bleeding (hemorrhage)) → Immediately adds platelets to the blood to plug holes in bleeding vessels and prevent or treat life-threatening bleeding (hemorrhage), especially in the brain. Buys time while other treatments (steroids, IVIG) take effect.
- Tranexamic acid (TXA) 1–1.5 grams orally or IV three times daily for mucosal bleeding (nosebleeds, gum bleeding, heavy periods) → Blocks plasmin, the enzyme that breaks down clots. This stabilizes the fragile clots that low platelets can barely form, stopping oozing from mucous membranes.
- Thrombopoietin receptor agonists (TPO-RAs): eltrombopag (starting 50 mg/day orally) or romiplostim (starting 1 mcg/kg subcutaneous weekly) → Mimics thrombopoietin, the hormone that signals bone marrow megakaryocytes to make more platelets. Drives platelet production higher to overcome the immune destruction. Platelet count rises in 1–2 weeks. Used for chronic ITP not responding to steroids or IVIG, or to avoid splenectomy. ASH guidelines endorse TPO-RAs as second-line in adults.
- Rituximab 375 mg/m² IV weekly for 4 weeks, or 1,000 mg IV on days 1 and 15 → Kills the B cells (a type of white blood cell) that make the antibodies attacking platelets. Stops the immune attack at its source. Response takes 4–8 weeks. About 60% of patients respond, and half maintain remission for a year or more. ASH guidelines list rituximab as an option for adults with persistent or chronic ITP.
- Splenectomy (surgical removal of the spleen) → Removes the spleen, which is the main organ where macrophages trap and destroy antibody-coated platelets. Also removes a major site of antibody production. About 70–80% of patients achieve long-term remission. Considered definitive second-line treatment for chronic ITP not controlled by medicines.

Medications

Prednisone · Corticosteroid (immune-calming steroid)

Prednisone calms down the immune system's attack on platelets. In ITP, the immune system mistakes platelets for invaders and tags them for destruction. Prednisone tells the immune cells (especially B cells and T cells in the spleen and bloodstream) to back off, so fewer platelets get destroyed and platelet counts can climb back up.

1. You swallow the pill; it enters the bloodstream and spreads throughout the body.
2. Prednisone binds to receptors inside immune cells (B cells, T cells, and macrophages).
3. It tells those cells to quiet down: they make fewer antibodies against platelets and destroy fewer platelets in the spleen.
4. With less destruction, platelet counts rise over days to weeks.

> **SAFETY WATCH**
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> High blood sugar (can unmask or worsen diabetes) · Weight gain and increased appetite · Mood swings, trouble sleeping, or feeling anxious · Stomach irritation or ulcers (take with food) · Higher risk of infections (the immune system is damped down) · Bone thinning (osteoporosis) and muscle weakness with long use · Never stop prednisone suddenly after weeks of use—taper slowly under a doctor's watch to avoid adrenal crisis

Dexamethasone (high-dose pulse) · Corticosteroid (immune-calming steroid)

Dexamethasone is a stronger, longer-lasting steroid than prednisone. Given in short, high-dose pulses (usually 40 mg daily for 4 days), it hits the immune system hard and fast to stop the attack on platelets. It quiets the same immune cells (B cells, T cells, macrophages) but with a bigger punch in a shorter time, often bringing platelet counts up quickly.

1. You take a high dose (40 mg) by mouth once a day for 4 days.
2. Dexamethasone floods the bloodstream and enters immune cells.
3. It binds to receptors inside those cells and shuts down antibody production and platelet destruction.
4. Platelet counts often climb within days; the effect can last weeks to months.

SAFETY WATCH

Same as prednisone: high blood sugar, weight gain, mood changes, trouble sleeping · Stomach upset (take with food) · Increased infection risk while immune system is quieted · Because it is a short pulse, long-term bone and muscle problems are less common than with daily prednisone · Do NOT take if you have an active serious infection unless treating ITP is urgent

IVIG (Intravenous Immunoglobulin) · Immune globulin (antibody infusion)

IVIG is a bag of pooled antibodies from thousands of healthy donors. When infused, it floods the bloodstream with normal antibodies that confuse and distract the immune system. The spleen's garbage-disposal cells (macrophages) get so busy clearing the infused antibodies that they stop destroying the patient's own platelets. IVIG also blocks some of the harmful antibodies stuck to platelets. Platelet counts usually rise within 1–3 days.

1. IVIG is infused slowly into a vein over several hours (total dose usually 1 gram per kilogram of body weight, sometimes split over 1–2 days).
2. The flood of donor antibodies saturates the receptors on macrophages in the spleen.
3. Macrophages shift from destroying platelets to clearing the infused antibodies.
4. Platelet counts climb quickly, often peaking in 2–7 days, then may fall again after weeks as the IVIG wears off.

SAFETY WATCH

Headache (common, sometimes severe) · Fever, chills, or flu-like aching during or after infusion · Rare but serious: blood clots (stroke, heart attack, deep vein clot), especially in older adults or those with clot risk · Rare: kidney injury (more likely if you are dehydrated or have kidney disease) · Rare: allergic reactions or aseptic meningitis (severe headache, stiff neck) · Infuse slowly and drink plenty of fluids before and after

Rituximab · Monoclonal antibody (B-cell–targeting biologic)

Rituximab is a lab-made antibody that locks onto a marker (CD20) found on B cells, the immune cells that make antibodies. Once rituximab binds, the immune system tags those B cells for destruction. With fewer B cells around, fewer harmful anti-platelet antibodies are made, and platelet counts can recover. It takes weeks to work because old antibodies must fade and new B cells take months to grow back.

1. Rituximab is infused into a vein, usually once a week for 4 weeks.
2. It travels through the blood and sticks to CD20 markers on B cells.
3. The immune system (complement proteins and killer cells) destroys the marked B cells.

4. Over weeks to months, anti-platelet antibody levels drop and platelet counts rise.
5. B cells slowly regrow over 6–12 months.

SAFETY WATCH

Infusion reactions (fever, chills, itching, low blood pressure) during or right after the IV—pre-meds (acetaminophen, antihistamine) help · Increased infection risk (especially respiratory infections) while B cells are low · Rare but serious: reactivation of hepatitis B (screened before treatment) or progressive multifocal leukoencephalopathy (PML, a rare brain infection) · Low immunoglobulin levels over time, raising infection risk · Do NOT take live vaccines while on rituximab or for months after

Eltrombopag · Thrombopoietin receptor agonist (TPO-RA, platelet-growth signal)

Eltrombopag mimics thrombopoietin, the natural hormone that tells the bone marrow to make more platelets. It binds to receptors (TPO receptors) on megakaryocytes (the large bone-marrow cells that break into platelets) and switches on their growth and platelet production. Over days to weeks, platelet counts climb. It does NOT calm the immune attack; it simply out-produces the destruction.

1. You swallow the pill once a day on an empty stomach (do not take with dairy, calcium, iron, or antacids—they block absorption).
2. Eltrombopag enters the bloodstream and reaches the bone marrow.
3. It binds to TPO receptors on megakaryocytes and tells them to grow and mature faster.
4. More platelets are released into the blood; counts usually start to rise within 1–2 weeks.

SAFETY WATCH

Liver enzyme elevation (check liver tests monthly)—can rarely cause liver injury · Risk of blood clots (deep vein clots, pulmonary embolism (a clot lodging in a lung artery)) if platelet count climbs too high · Cataracts (clouding of the eye lens)—get eye exams · Bone-marrow fibrosis (scarring) with long-term use—rare · Rebound drop in platelets if stopped suddenly · Do NOT take with dairy, calcium, iron, or antacids within 4 hours (they block the drug)

Romiplostim · Thrombopoietin receptor agonist (TPO-RA, platelet-growth signal)

Romiplostim is an injected protein that acts like thrombopoietin, the hormone that signals the bone marrow to make platelets. It binds and activates TPO receptors on megakaryocytes (platelet-making cells), ramping up platelet production. Unlike eltrombopag (a pill),

romiplostim is a shot given under the skin once a week. Platelet counts typically rise within 1–2 weeks.

1. You (or a nurse) inject romiplostim under the skin (abdomen, thigh, or arm) once a week.
2. The protein enters the bloodstream and travels to the bone marrow.
3. It locks onto TPO receptors on megakaryocytes and flips the growth switch.
4. Megakaryocytes mature faster and shed more platelets into the blood.
5. Platelet counts rise over days; the dose is adjusted weekly to keep counts in a safe range.

SAFETY WATCH

Risk of blood clots if platelet count goes too high · Bone-marrow fibrosis (scarring) with long-term use—rare, monitored with blood smears · Rebound a low platelet count (thrombocytopenia) (counts drop lower than before) if stopped suddenly · Headache, muscle or joint pain, dizziness (common but usually mild) · Antibody formation (very rare) that can block natural thrombopoietin and worsen counts · Dose is adjusted weekly based on platelet count—close monitoring required

Platelet transfusion · Blood product (donor platelets)

Platelet transfusion delivers a bag of donor platelets directly into the bloodstream. It is a fast but temporary fix for life-threatening bleeding or very low counts (usually <10,000 or <20,000 with infection or planned surgery). In ITP the transfused platelets are often destroyed quickly by the same immune attack, so the benefit is short-lived (hours to a day or two). Transfusion buys time while other treatments (steroids, IVIG) take effect.

1. A bag of donor platelets is infused into a vein over 15–30 minutes.
2. The platelets enter circulation and immediately help form clots at bleeding sites.
3. In ITP, immune antibodies and the spleen destroy many transfused platelets within hours.
4. The count bumps up briefly, then falls again—repeat transfusions may be needed for ongoing severe bleeding.

SAFETY WATCH

Allergic or feverish (febrile) (fever) reactions during transfusion · Risk of infection (bacterial, viral) from donor blood—screened but not zero · Transfusion-related acute lung injury (TRALI)—rare, causes sudden breathing trouble · Fluid overload (too much volume) in patients with heart or kidney problems · In TTP or HIT, transfusion can worsen clotting —use only if bleeding is life-threatening · Blood-type matching (ABO) is less critical than for red cells, but Rh matching is preferred in young women

Tranexamic acid (TXA) · Antifibrinolytic (clot-protecting drug)

Tranexamic acid blocks plasmin, the enzyme that breaks down clots. Normally, after a clot forms, the body slowly dissolves it to clean up. TXA puts the brakes on that breakdown, so small clots in the mouth, nose, or bladder last longer and stop bleeding more effectively. It does NOT raise platelet counts—it just helps the few platelets you have hold a clot together longer.

1. You swallow a pill or rinse your mouth with liquid TXA (for mouth bleeds) or receive it by IV.
2. TXA enters the bloodstream and sticks to plasminogen (the inactive form of plasmin).
3. It prevents plasminogen from turning into plasmin, the enzyme that chews up clots.
4. Clots stay intact longer, reducing oozing from mucous membranes (gums, nose) or small cuts.

> **SAFETY WATCH**
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> Do NOT use in DIC, TTP, or active clotting (it can worsen clots) · Nausea or stomach upset (take with food) · Rare: seizures at very high IV doses · Rare: blood clots (deep vein, pulmonary, stroke) if used in high-risk patients · Safe for short-term use (days to weeks) for mucosal bleeding; less data for long-term daily use

Splenectomy (surgical removal of the spleen) · Surgery (not a medication, but a key treatment option)

The spleen is the main organ where antibody-tagged platelets are destroyed in ITP. Removing it takes away the chief destruction site. After splenectomy, even though harmful antibodies may still circulate, there is no spleen to filter out and destroy platelets, so counts usually rise and stay higher. About two-thirds of people with ITP go into long-term remission after splenectomy.

1. The spleen is removed by surgery (laparoscopic or open).
2. Without the spleen, tagged platelets are no longer trapped and destroyed in that organ.
3. Platelet counts usually climb within days to weeks and often stay up.
4. The liver and lymph nodes can still clear some antibody-coated platelets, but far less than the spleen did.

SAFETY WATCH

Lifelong increased risk of serious infections, especially from encapsulated bacteria (pneumococcus, meningococcus, Haemophilus)—vaccinate at least 2 weeks before surgery (pneumococcal, meningococcal, Hib) and keep boosters current · Surgical risks: bleeding, infection, injury to nearby organs · Post-splenectomy sepsis (overwhelming bloodstream infection)—rare but can be fatal; seek care immediately for fever · Slightly higher long-term risk of blood clots · Consider splenectomy only after trying medications, because it is irreversible

Stop heparin and switch to a non-heparin anticoagulant (for HIT) · Management strategy for heparin-induced a low platelet count (thrombocytopenia) (HIT)

In HIT, heparin triggers antibodies that activate platelets, causing dangerous clots and a drop in platelet count. Stopping all heparin (even flushes and line coatings) removes the trigger. You must switch immediately to a different blood thinner that does NOT cross-react—such as argatroban (a direct thrombin inhibitor) or fondaparinux (a synthetic factor Xa blocker)—to prevent new clots while platelet counts recover.

1. All heparin (unfractionated and low-molecular-weight) is stopped immediately.
2. A non-heparin anticoagulant (argatroban, fondaparinux, or bivalirudin) is started by IV or injection.
3. The new drug blocks clotting without activating platelets or triggering HIT antibodies.
4. Platelet counts usually start to recover within days to a week once heparin is gone.
5. After counts normalize and clot risk drops, the patient may transition to warfarin (with overlap) or a direct oral anticoagulant for long-term prevention.

SAFETY WATCH

Bleeding risk with any anticoagulant—monitor closely · Argatroban is cleared by the liver—dose adjustments needed in liver disease · Fondaparinux is cleared by the kidneys—avoid in severe kidney disease · Confirm HIT with lab tests (PF4 ELISA, serotonin-release assay) while treating—do not wait for results to stop heparin if HIT is suspected · Avoid warfarin alone at the start (can cause clots in HIT)—overlap with a non-heparin drug until platelets recover and INR is stable · Never give heparin again in the future; carry a medical alert and inform all providers

Red flags

- Blood in urine or stool, or vomit that looks like coffee grounds

- Sudden severe headache, confusion, vision changes, or weakness on one side of the body (signs of bleeding in the brain)
- Heavy bleeding that will not stop after 10 minutes of firm pressure
- Platelet count below 10,000 per microliter (very high risk of serious bleeding)
- Fever, fast heart rate, confusion, and yellow skin together with low platelets (may be thrombotic thrombocytopenic purple bleeding blotches (purpura) or TTP, not ITP)

Nursing priorities

1. Check the platelet count result and ask the lab if the sample was drawn in an EDTA tube (purple top), then request a recheck in a blue top or direct finger stick smear — EDTA anticoagulant makes platelets clump together in about 1 out of 1,000 people, creating pseudothrombocytopenia (fake low platelets), so the patient does not actually have low platelets and does not need dangerous treatments
2. Assess for active bleeding by checking gums, nose, skin for pinpoint bleeding spots (petechiae) or large bruises (ecchymoses), and ask about blood in urine, stool, or heavy menstrual bleeding — Platelets under 10,000 cannot form clots, so any bleeding can become life-threatening quickly, especially in the brain or gut, and requires emergency platelet transfusion and IVIG
3. Institute bleeding precautions including no rectal temperatures or suppositories, no intramuscular injections, electric razor only, soft toothbrush, and fall precautions with bed alarm and non-slip socks — Any trauma or puncture can cause uncontrolled bleeding when platelets are low because there are not enough platelets to plug damaged blood vessels
4. Review the medication list with the provider and stop any drugs known to lower platelets, especially heparin, aspirin, ibuprofen, naproxen, sulfa antibiotics, and quinine — Drug-induced a low platelet count (thrombocytopenia) (low platelets from medicine) reverses in 7 to 10 days after stopping the drug because the bone marrow can make new platelets once the drug is gone
5. Monitor neurologic status every 2 to 4 hours by checking for new headache, confusion, vision changes, slurred speech, or weakness on one side — Intracranial bleeding (hemorrhage) (brain bleeding) is the leading cause of death in ITP when platelets drop under 10,000, and early signs let you give emergency platelets and stop fatal bleeding
6. Administer corticosteroids exactly as ordered, either prednisone 1 milligram per kilogram by mouth daily or dexamethasone 40 milligrams by mouth for 4 days, and teach the patient to take with food — Steroids stop the immune system from destroying platelets by blocking antibodies that attack platelet surface proteins, raising the count in 2 to 7 days in 70 percent of ITP patients

7. Prepare to give IVIG (intravenous immunoglobulin) 1 gram per kilogram slowly over 6 to 8 hours if platelets are under 10,000 or the patient has active bleeding, and watch for headache or allergic reaction — IVIG blocks the spleen from destroying platelets and raises the platelet count within 24 hours, much faster than steroids, so it saves lives during severe bleeding or prevents bleeding before emergency surgery
8. Teach the patient to avoid contact sports, heavy lifting over 10 pounds, and activities with fall or injury risk until platelet count is above 50,000 — Trauma causes bleeding into muscles and organs when platelet counts are low because clotting cannot happen fast enough, and internal bleeding can be fatal even without external cuts

Sources

- American Society of Hematology 2019 ITP guideline
- UpToDate clinical decision support
- Blood (American Society of Hematology journal)
- Cochrane systematic reviews

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