

# TTP and HUS

Tiny clots form throughout small blood vessels, shredding red blood cells and using up platelets.

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## THE STORY IN ONE SENTENCE

Tiny clots form throughout small blood vessels, shredding red blood cells and using up platelets.

“TTP and HUS both cause broken red blood cells and low platelets from tiny clots in small vessels—TTP needs plasma exchange immediately.”

## Normal anatomy — the body's job

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Normally, the enzyme ADAMTS13 cuts von Willebrand factor (vWF)—a large, sticky clotting protein—into small, safe pieces so platelets can clot injuries without clogging healthy vessels.

## BODY'S JOB

vWF is made in the lining of blood vessels and floats in the blood as huge, sticky strings. ADAMTS13 trims those strings down to a safe size. Platelets stick to the smaller vWF pieces only when a blood vessel is cut, forming a clot to stop bleeding. This keeps clotting in the right place at the right time.

1. Blood vessel lining releases large vWF into the bloodstream.
2. ADAMTS13 enzyme cuts vWF into smaller, less sticky pieces.
3. Platelets stick to small vWF only at injury sites to form clots.
4. Red blood cells flow smoothly through open, healthy vessels.
5. Kidneys, brain, and other organs get steady blood flow and oxygen.

## What breaks

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The enzyme that cuts a sticky clotting protein stops working, or the lining of blood vessels gets damaged and triggers clotting where it shouldn't happen.

**CLINICAL TERM**

TTP: severe deficiency of ADAMTS13 (<10% activity) from autoantibodies (acquired TTP) or genetic mutations (congenital TTP). Without ADAMTS13, ultra-large vWF multimers persist and spontaneously bind platelets, forming microthrombi. HUS: Shiga toxin (from STEC, e.g. *E. coli* O157:H7) or dysregulated complement activation (atypical HUS) injures vascular endothelium, especially in glomerular capillaries, triggering platelet aggregation and microvascular clot formation (thrombosis).

## How the body compensates

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The body tries to maintain blood pressure and oxygen delivery by speeding the heart and breathing, but organ damage (especially brain and kidneys) can progress silently until sudden decompensation.

1. The bone marrow releases young red cells (reticulocytes) and makes more platelets to replace those lost, but production cannot keep up with destruction.
2. The heart pumps faster (tachycardia) to move the remaining oxygen-carrying red cells through the body more quickly.
3. Breathing rate may increase to bring in more oxygen and compensate for anemia.
4. Blood pressure may stay normal early on, masking the severity of microvascular clotting and organ starved blood flow (ischemia) underneath.
5. The spleen and liver try to clear damaged red cells and clot debris, but the flood of broken cells overwhelms them, causing yellowing of the skin and eyes (jaundice) and elevated LDH.
6. This compensation can hide the crisis—vital signs may look stable even as brain, kidneys, or heart are starved of oxygen by microthrombi.
7. Watch for subtle signs of decompensation: confusion, decreased urine output, chest pain, or worsening anemia and a low platelet count (thrombocytopenia)—these mean the body's reserves are failing.

## Domino effect

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1. ADAMTS13 enzyme is absent or blocked (TTP), or vessel lining is injured by toxin or complement (HUS). — In TTP, autoantibodies destroy ADAMTS13 or a person is born without the gene to make it. In typical HUS, Shiga toxin from *E. coli* binds to and kills endothelial cells in the kidneys. In atypical HUS, mutations let complement proteins attack the body's own vessel walls. This is the single upstream break.

2. Giant vWF tangles trap platelets (TTP) or damaged vessel walls activate platelets (HUS), forming microthrombi in small vessels throughout the body. — Without ADAMTS13 cutting vWF, ultra-large multimers stay sticky and spontaneously bind platelets. In HUS, injured endothelium exposes clotting triggers. Either way, platelets clump into tiny clots that clog capillaries.
3. Platelet count drops (thrombocytopenia) and red blood cells are shredded (microangiopathic destroying red blood cells (hemolytic) anemia with schistocytes). — Platelets are consumed faster than the marrow can make them. Red cells are physically torn apart as they are forced through narrowed, clot-filled vessels, releasing hemoglobin and leaving schistocyte fragments visible on the blood smear.
4. Hemoglobin spills into the blood; LDH, indirect bilirubin, and free hemoglobin rise while haptoglobin drops to zero. — Broken red cells dump their contents—hemoglobin and LDH—into the plasma. The liver cannot make haptoglobin (the hemoglobin-binding protein) fast enough to clear it. Bilirubin rises as the liver processes dead red cell debris.
5. Brain and kidneys are hit hardest in TTP; kidneys are the main target in HUS. — TTP microthrombi clog small vessels everywhere, but the brain (causing confusion, seizures, stroke) and kidneys (causing acute kidney injury) are especially vulnerable because their capillaries are dense and sensitive to low oxygen. HUS targets the glomeruli (kidney filters) preferentially because Shiga toxin or complement binds most strongly there, leading to acute renal failure, proteinuria, and blood in the urine (hematuria).
6. Organs starved of oxygen begin to fail: confusion, stroke, or coma (brain); acute kidney injury with rising creatinine and low urine output (kidneys); chest pain or arrhythmia (heart). — Microthrombi block blood flow, so tissues downstream get too little oxygen (ischemia). Brain cells die or misfire, causing neurologic symptoms. Kidney tubules and glomeruli cannot filter waste or regulate fluid. Heart muscle may become blood-starved (ischemic), triggering arrhythmias or tissue death from blocked blood flow (infarction).
7. Without treatment, multi-organ failure, irreversible brain damage, or death occurs; with prompt plasma exchange (TTP) or supportive care and complement blockade (atypical HUS), most patients survive. — TTP is >90% fatal without plasma exchange, which removes autoantibodies and replaces ADAMTS13. Plasma exchange reverses the clotting cascade within days. Typical HUS (Shiga toxin) is managed with fluids and dialysis; most children recover. Atypical HUS requires eculizumab (a complement blocker) to stop vessel attack. Early recognition and treatment prevent permanent organ damage.

## Symptoms · what the patient feels, and why

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- Broken red blood cells (schistocytes) on the blood smear: Tiny blood clots made of sticky platelets form throughout small vessels, and red blood cells slam into these clots at high speed and shatter into pieces (like a bug hitting a windshield). These broken fragments are called schistocytes.
- Very low platelet count (thrombocytopenia): Platelets clump together abnormally inside small blood vessels all over the body, using them up faster than the bone marrow can replace them, so the number in the bloodstream drops sharply.
- Low haptoglobin and high LDH in the blood: Haptoglobin is a protein that mops up free hemoglobin (the oxygen-carrying part of red blood cells). When lots of red cells break open, haptoglobin gets used up and drops low. LDH (an enzyme normally inside cells) spills out when cells break, so it rises high in the blood.
- Dark or tea-colored urine: When red blood cells break apart, hemoglobin is released into the bloodstream; the kidneys filter it out and dump it into the urine, turning the urine dark brown or red (hemoglobinuria).
- Sudden confusion, trouble speaking, or weakness on one side of the body: Tiny clots block blood vessels in the brain, starving patches of brain tissue of oxygen (ischemia). This can cause stroke-like symptoms or altered mental status as brain cells stop working properly.
- Sharp drop in urine output or swelling in the legs and face: Clots jam the tiny filters (glomeruli) in the kidneys, damaging them so they cannot clean the blood or make urine normally. Fluid and waste build up in the body (acute kidney injury).
- Fever (more common in TTP): In TTP, widespread blood vessel damage and clotting can trigger inflammation throughout the body, raising the body's thermostat (hypothalamus) and causing fever.
- Severe abdominal pain or bloody diarrhea (more common in typical HUS): Most typical HUS starts with an infection by Shiga toxin-producing *E. coli* (often from undercooked meat or contaminated water). The toxin damages the lining of the intestines, causing cramping, pain, and bloody stools before it enters the bloodstream and attacks the kidneys.

## Focused exam

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- Schistocytes (broken red blood cells shaped like helmets or triangles) on the blood smear slide: This is the fingerprint of TTP and HUS. Red blood cells are getting sliced apart as they squeeze through tiny blood vessel clots built from sticky von Willebrand factor protein clumps. You must look at the slide yourself under the microscope to see them.

- A low platelet count (Thrombocytopenia) (platelet count below 150,000 per microliter, often much lower): In TTP and HUS, platelets stick to the von Willebrand factor clumps and disappear from the bloodstream. This is why you do NOT give platelet transfusions — adding more platelets feeds the clotting fire and can make things worse.
- Low haptoglobin (less than 25 mg/dL) and high LDH (lactate dehydrogenase over 250 U/L): Haptoglobin is a protein that catches free hemoglobin when red cells break open. When it drops low, red cells are dying fast. High LDH means cells all over the body are getting wrecked. Together these prove red blood cell destruction (hemolysis) (red cell destruction) is happening right now.
- Fever, confusion, slurred speech, weakness on one side of the body, or other stroke signs: TTP can attack the brain by plugging tiny blood vessels in the brain tissue. These signs mean TTP is severe and life-threatening. Start plasma exchange (blood filtering) immediately — do not wait for the ADAMTS13 test result to come back.
- Bloody diarrhea or watery diarrhea for 3 to 7 days BEFORE the blood problems started: In HUS, Shiga toxin from bad bacteria (most often E. coli O157:H7 from undercooked meat or contaminated water) starts by attacking the gut lining. TTP comes out of nowhere with no diarrhea. Asking about diarrhea tells you which disease you are dealing with.
- Rising creatinine (above 1.2 mg/dL in adults, higher values show worse kidney damage) or decreasing urine output: Both TTP and HUS can wreck the kidneys by clogging the tiny filtering vessels (glomeruli). In HUS, kidney failure is the main problem. In TTP, brain trouble is usually worse. Tracking which organ is failing hardest guides your treatment focus.

## Diagnostics · every test answers a question

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- Peripheral blood smear (looked at under the microscope by a trained person) → Are red blood cells being chopped apart inside the blood vessels right now?
- Complete blood count (CBC) with platelet count → How low are the platelets, and is the patient anemic?
- Lactate dehydrogenase (LDH) → Are cells breaking apart and spilling their insides into the blood?
- Haptoglobin → Is hemoglobin leaking out of red cells faster than the body can catch it?
- Indirect (unconjugated) bilirubin → Is the liver seeing a flood of broken-down red blood cell parts?
- Reticulocyte count → Is the bone marrow pumping out baby red blood cells to replace the ones being destroyed?
- ADAMTS13 activity level and ADAMTS13 antibody → Is the enzyme that cuts up von Willebrand factor missing, and is the immune system attacking it?

- Basic metabolic panel (BMP) with creatinine and blood urea nitrogen (BUN) → Are the kidneys failing because tiny clots are clogging the filtering units?
- Urinalysis → Is there blood or protein spilling into the urine because the kidney filters are damaged?
- Stool culture and Shiga toxin test (STEC test, or PCR for Shiga toxin genes) → Did bad bacteria (*E. coli* O157:H7 or other Shiga-toxin-producing *E. coli*) cause this by releasing toxin in the gut?
- Coagulation panel (PT, aPTT, fibrinogen, D-dimer) → Is this disseminated intravascular coagulation (DIC) instead of TTP or HUS?

## Treatment logic

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- Plasma exchange (plasmapheresis or therapeutic plasma exchange, TPE) started within hours → Removes the giant sticky von Willebrand factor clumps (ultra-large vWF multimers) and the antibodies attacking ADAMTS13 from the blood. Replaces them with fresh frozen plasma that contains working ADAMTS13 enzyme. This stops platelets from sticking together, breaks the clotting cycle, and saves the brain and kidneys. Without plasma exchange, TTP kills over 90 percent of patients; with it, survival jumps to over 80 percent.
- High-dose corticosteroids (methylprednisolone 1 to 2 mg per kg per day IV, or prednisone 1 mg per kg per day by mouth) → Quiets the immune system so it stops making antibodies that attack ADAMTS13 in acquired TTP. Reduces inflammation in the blood vessel lining in atypical HUS. This slows the destruction of red cells, platelets, and blood vessel walls, and helps the body stop the disease process.
- Rituximab (anti-CD20 monoclonal antibody, 375 mg per square meter IV once a week for 4 weeks) → Kills the B cells (a type of white blood cell) that are making antibodies against ADAMTS13. This stops the source of the immune attack in acquired TTP. Studies show rituximab cuts the relapse rate in half and helps more patients go into remission. It is now standard first-line treatment along with plasma exchange and steroids.
- Caplacizumab (anti-von Willebrand factor nanobody, 10 mg IV once, then 10 mg subcutaneous daily) → Blocks the sticky part of von Willebrand factor so it cannot grab platelets and build clots, even while the giant clumps are still in the blood. This speeds up platelet recovery, reduces the number of plasma exchange sessions needed, and cuts the risk of death and major complications during the acute phase. FDA approved in 2019 for TTP.

- Eculizumab (complement C5 inhibitor, 900 mg IV weekly for 4 weeks, then 1,200 mg every 2 weeks) for atypical HUS → Blocks the complement cascade (part of the immune system) at the C5 step. In atypical (non-Shiga-toxin) HUS, a broken complement system attacks the lining of blood vessels. Eculizumab stops this attack, protects the kidney filters (glomeruli), and prevents further damage. Studies show it restores kidney function and prevents relapse in most patients with atypical HUS.
- Supportive care: IV fluids, careful blood pressure control, avoid platelet transfusions, monitor kidney function and start dialysis if needed → Keeps blood volume up and organs (especially kidneys) perfused while the specific treatments work. Avoids triggering more clots — platelet transfusions in TTP add fuel to the fire and can cause stroke or death, so they are only given if there is life-threatening bleeding. Dialysis replaces kidney filtering when the glomeruli are too damaged to clean the blood.
- DO NOT give platelet transfusions unless life-threatening bleeding is happening → Prevents making TTP worse. Adding platelets when von Willebrand factor clumps are still in the blood gives the clumps more platelets to grab, which builds more clots in the brain, kidneys, and other organs. Studies show platelet transfusions in TTP increase the risk of stroke and death.

## Medications

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### **Plasma exchange (plasmapheresis) · Blood-filtering procedure (removes harmful antibodies and replaces missing ADAMTS13 enzyme)**

In TTP, the body attacks ADAMTS13 (the enzyme that cuts up big sticky von Willebrand factor proteins). Without it, huge sticky clumps form in the blood and trap platelets, blocking tiny vessels. Plasma exchange pulls out the patient's plasma (the liquid part of blood containing the attacking antibodies) and swaps in fresh donor plasma that has working ADAMTS13. This stops new clots from forming and lets the body recover.

1. Patient's blood is drawn through a machine that spins it to separate plasma from cells
2. The plasma (containing anti-ADAMTS13 antibodies and huge von Willebrand factor clumps) is thrown away
3. Fresh frozen plasma from healthy donors (full of working ADAMTS13) is mixed back with the patient's own red and white cells
4. The cleaned blood goes back into the patient
5. ADAMTS13 now cuts the big sticky proteins into safe small pieces, so platelets stop getting trapped

**SAFETY WATCH**

Large-bore IV line needed (usually central line in neck or chest) · Risk of allergic reaction to donor plasma (hives, trouble breathing) · Low calcium from the citrate preservative in donor plasma (can cause tingling or muscle cramps) · Infection risk from the catheter · Bleeding if blood thinner (used to keep the line open) dose is too high

**Corticosteroids (prednisone or methylprednisolone) · Immunosuppressant (broad dampener of immune system)**

Steroids quiet down the overactive immune cells that are making antibodies against ADAMTS13. They block the inflammatory signals (like NF-kappa-B inside immune cells) so fewer antibodies are produced and the attack on the enzyme slows.

1. Steroid enters the immune cell and binds to receptors inside
2. The receptor moves into the cell's control center (nucleus)
3. It turns off genes that make inflammatory proteins and antibodies
4. Fewer anti-ADAMTS13 antibodies are released into the blood
5. The remaining ADAMTS13 (from plasma exchange) can work without being attacked as much

**SAFETY WATCH**

High blood sugar (can trigger or worsen diabetes) · Increased infection risk (immune system is dampened) · Mood swings, trouble sleeping, increased appetite · Bone thinning (osteoporosis) and muscle weakness with long-term use · Stomach ulcers (give with food or add a stomach protector) · Never stop suddenly after weeks of use (must taper down slowly to let the adrenal glands wake back up)

**Rituximab · Monoclonal antibody (targeted immune suppressant)**

Rituximab is a lab-made antibody that locks onto CD20 (a marker on the surface of B cells, the immune cells that make antibodies). Once rituximab sticks to CD20, it marks those B cells for destruction. Killing the B cells stops them from churning out the anti-ADAMTS13 antibodies, so the attack on the enzyme ends and TTP is less likely to come back.

1. Rituximab is given through an IV and travels through the bloodstream
2. It finds B cells and attaches to the CD20 protein on their outer shell
3. The immune system's cleanup crew (macrophages and natural killer cells) see the marked B cells and destroy them
4. B-cell numbers drop sharply within days
5. Without B cells, production of anti-ADAMTS13 antibodies falls and stays low for months

## 6. ADAMTS13 levels recover and stay normal, preventing relapses

### SAFETY WATCH

Infusion reaction during the first dose (fever, chills, low blood pressure; give acetaminophen and antihistamine beforehand and run the drip slowly) · Increased infection risk (B cells help fight bacteria; risk of pneumonia and sinus infections) · Rare reactivation of hepatitis B virus (check hepatitis status before starting) · Very rare progressive brain infection (PML) in people with weak immune systems · Low immunoglobulin levels long-term (may need vaccine boosters or antibody replacement)

## Caplacizumab · Anti-von Willebrand factor nanobody (blocks platelet clumping)

Caplacizumab is a tiny engineered antibody piece that grabs onto von Willebrand factor (the sticky blood protein) and covers the spot where platelets normally attach. Even though the big sticky clumps are still floating around (because ADAMTS13 is missing), platelets can't stick to them anymore, so the dangerous clots in tiny vessels stop forming right away.

1. Caplacizumab is injected under the skin (subcutaneous) daily
2. It circulates in the blood and finds von Willebrand factor molecules
3. It covers the sticky patch (A1 domain) where platelets would normally grab on
4. Platelets float past the big clumps without sticking
5. Microthrombi (tiny clots in brain, kidneys, heart) stop forming immediately
6. Platelet count rises and organ damage halts while plasma exchange and immunosuppression work on the root cause

### SAFETY WATCH

Bleeding risk (gums, nose, heavier periods) because von Willebrand factor also helps clotting when you're injured · Must monitor for signs of serious bleeding (headache, black stool, vomiting blood) · Usually continued for 30 days after stopping plasma exchange, then tapered carefully · Injection-site reactions (redness, itch)

## Supportive care for STEC-HUS (fluids, dialysis if needed) · Supportive therapy (no specific drug neutralizes Shiga toxin)

In typical HUS (caused by Shiga toxin from E. coli O157 or other STEC bacteria), the toxin damages the cells lining blood vessels in the kidneys and gut. The best treatment is careful IV fluid to keep blood flowing through the injured kidneys and dialysis (a kidney machine) if the

kidneys stop making urine or toxins build up. Antibiotics are avoided during the diarrhea phase because killing the bacteria can make them dump more toxin all at once and worsen HUS.

1. Child (usually age <5) eats food contaminated with Shiga toxin-producing *E. coli*
2. Bacteria release Shiga toxin in the gut, which enters the bloodstream
3. Toxin attaches to Gb3 receptors on endothelial cells (lining of small vessels) in kidneys and brain
4. Cells die, vessel walls crack, and platelets clump to patch the damage (causing low platelet count and red blood cell destruction (hemolysis) as red cells shatter going through the clots)
5. Kidneys swell and stop filtering; toxic waste builds up
6. Give IV normal saline or lactated Ringer's carefully to avoid overload
7. Start dialysis if potassium is dangerously high, fluid backs up into lungs, or waste (creatinine, urea) climbs
8. Most children recover kidney function in 1–2 weeks as the toxin clears

#### **SAFETY WATCH**

Monitor fluid balance closely (too much causes lung swelling; too little worsens kidney injury) · Watch electrolytes daily (high potassium can stop the heart) · Dialysis catheter infection risk · Seizures if brain swelling occurs (treat with anti-seizure medicine and careful fluid management) · Rarely, long-term kidney damage or high blood pressure

### **Eculizumab (for atypical HUS) · Monoclonal antibody (complement inhibitor)**

Atypical HUS happens when the complement system (a cascade of immune proteins that normally punches holes in bacteria) is stuck in the 'on' position and attacks the body's own blood-vessel linings, especially in the kidneys. Eculizumab is a lab-made antibody that blocks C5 (one of the key complement proteins). By stopping C5, it halts the formation of the membrane attack complex (the final hole-punching weapon), so vessel damage stops and the kidneys can heal.

1. Patient has a genetic mutation or autoantibody that prevents normal brakes on the complement system
2. Complement proteins (C3, C5) pile onto kidney vessel walls
3. C5 normally splits and forms the membrane attack complex (MAC), which drills holes in cells
4. Eculizumab binds tightly to C5 and blocks the split
5. No MAC forms, so endothelial cells stay intact
6. Platelet clumping and red-cell breakage stop; kidney function stabilizes or improves

## 7. Given by IV infusion every 1–2 weeks, often lifelong

### SAFETY WATCH

HIGH risk of meningococcal infection (*Neisseria meningitidis*) because C5 is needed to kill that bacteria—patient **MUST** get meningococcal vaccine (all serogroups A, B, C, W, Y) at least 2 weeks before starting, or get antibiotics (penicillin or macrolide) for the first 2 weeks if vaccine can't wait · Watch for sudden fever, severe headache, stiff neck, rash (signs of meningitis)—seek ER immediately · Increased risk of other encapsulated bacteria (pneumococcus, *Haemophilus*)—give vaccines · Infusion reactions (rare) · Very expensive; requires lifelong therapy in most cases of atypical HUS

## Red blood cell transfusion (as needed for severe anemia) · Supportive blood product

Both TTP and HUS cause destroying red blood cells (hemolytic) anemia (red cells break apart as they slam through tangled clots in tiny vessels). If hemoglobin drops so low that the patient is dizzy, short of breath, or the heart is racing dangerously, packed red blood cells are transfused to carry oxygen while the underlying disease is treated.

1. Patient's hemoglobin is checked (normal ~12–16 g/dL; in severe TMA may drop to <7)
2. Donor red cells (type-matched and screened) are given slowly through an IV
3. New red cells boost oxygen delivery to brain, heart, and kidneys
4. Symptoms of anemia (fatigue, chest pain, confusion) improve
5. Transfused cells are also at risk of breaking if clotting is still active, so definitive treatment (plasma exchange, eculizumab, supportive care) must continue

### SAFETY WATCH

Transfusion reaction (fever, chills, back pain—stop transfusion immediately) · Fluid overload (especially if kidneys are failing)—give diuretic if needed · Rare severe reactions (hemolytic reaction if wrong blood type, TRALI lung injury, allergic shock) · Infection risk (screened but not zero) · Iron overload if many transfusions over time

## Red flags

- New confusion, slurred speech, or one-sided weakness (signs of stroke or brain bleeding)
- Urine output drops sharply or stops completely (acute kidney failure)
- Platelet count falls dangerously low (often below 20,000) with active bleeding (nosebleeds, bleeding gums, or bruises that spread quickly)
- Severe chest pain or trouble breathing (possible heart or lung clots)

- Seizures or sudden loss of consciousness

## Nursing priorities

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1. Monitor neurological status every 2 hours using Glasgow Coma Scale and check for new confusion, headache, vision changes, or seizure activity — Tiny clots in brain blood vessels cause stroke-like symptoms and seizures in TTP. Brain injury can progress rapidly, and any change in mental status signals worsening disease that needs immediate plasma exchange or increased treatment intensity.
2. Measure strict intake and output every hour and notify provider immediately if urine output drops below 0.5 mL per kg per hour — The kidneys are damaged by broken red blood cell pieces clogging tiny kidney filters (glomeruli). Dropping urine output signals acute kidney injury that may require urgent dialysis to remove waste products and prevent dangerous potassium levels.
3. Hold all platelet transfusions unless approved by hematology and the patient has life-threatening bleeding — Giving platelets when the patient is making tiny clots everywhere provides more fuel for clotting and can worsen organ damage. Platelets are being consumed in the disease process, not truly low from bone marrow failure, so transfusion is dangerous.
4. Coordinate daily plasma exchange treatments through a large-bore central line and monitor vital signs every 15 minutes during the procedure — Plasma exchange removes the antibodies blocking ADAMTS13 enzyme in TTP and supplies new enzyme. The procedure removes 1 to 1.5 times the patient's plasma volume and replaces it with fresh frozen plasma, which can cause fluid shifts, allergic reactions, and citrate toxicity (low calcium causing numbness and heart rhythm problems).
5. Monitor hemoglobin, platelet count, LDH, and haptoglobin levels daily before morning rounds — These lab values track disease activity. Rising platelets and falling LDH (lactate dehydrogenase, an enzyme released from broken cells) mean treatment is working. Haptoglobin (a protein that binds free hemoglobin) stays low during active red blood cell destruction (hemolysis). Trending these numbers guides how long to continue plasma exchange.
6. Assess for signs of bleeding including checking all IV sites, gums, stool, and urine for blood and applying pressure for 10 minutes to any puncture sites — Low platelet counts increase bleeding risk. Even though we avoid platelet transfusions, we must prevent trauma and catch bleeding early. Intracranial bleeding (hemorrhage) (bleeding in the brain) is a life-threatening complication when platelets drop very low.

7. Avoid all unnecessary needle sticks, use electric razors instead of blades, and keep fall precautions in place at all times — A low platelet count (Thrombocytopenia) makes even small injuries cause significant bleeding. Preventing trauma reduces risk of dangerous bleeding (hemorrhage) while the platelet count recovers with treatment.
8. Monitor for signs of fluid overload including lung sounds, weight, and peripheral swelling (edema), especially if kidney function is declining — Acute kidney injury prevents fluid removal, and plasma exchange adds large volumes of fluid. Pulmonary edema (fluid flooding the lungs) (fluid in lungs) can develop quickly and cause respiratory failure requiring intubation and mechanical ventilation.

## Sources

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- ASH (American Society of Hematology) 2020 TTP guideline
- KDIGO (Kidney Disease: Improving Global Outcomes) clinical practice guideline for acute kidney injury
- CDC information on hemolytic uremic syndrome
- NEJM review: Thrombotic Microangiopathies

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