

Microangiopathic Hemolytic Anemia (MAHA)

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, red blood cells glide smoothly through blood vessels of all sizes, delivering oxygen to every organ. In microangiopathic destroying red blood cells (hemolytic) anemia, something damages the smallest blood vessels or causes tiny clots to form inside them. When red blood cells try to squeeze through these damaged or narrowed vessels, they get sliced apart like a balloon scraping over sharp rocks. The broken cells spill their contents into the blood, leading to anemia, dark urine, and yellowing of the skin. Many organs can be harmed because they do not get enough oxygen and the small vessels get clogged with clot fragments.

What you might feel

- Dark or tea-colored urine
- Pale skin and mucous membranes
- Fatigue and weakness
- Shortness of breath with mild activity
- Yellowing of skin or eyes (jaundice)
- Pinpoint bleeding spots (Petechiae) or purple bleeding blotches (purpura) (small red or purple spots on skin)
- Confusion or altered mental status
- Decreased urine output

Call the doctor right away if...

- Platelet count below 30,000/microL with confusion or neurologic changes suggests TTP requiring immediate plasma exchange
- Hemoglobin below 7 g/dL with chest pain or severe shortness of breath needs urgent transfusion

- Creatinine rising despite fluids with decreased urine output indicates acute kidney injury needing nephrology consultation
- New pinpoint bleeding spots (petechiae) or purple bleeding blotches (purpura) with any mucosal bleeding requires platelet count check and hematology evaluation within hours

What the care team will do

- Monitor for signs of organ damage from microthrombi (tiny blood clots)
- Assess for bleeding tendencies despite low platelet count
- Evaluate neurologic status every 2-4 hours
- Protect patient from falls and injury
- Monitor urine output and color hourly
- Prepare for urgent plasmapheresis (plasma exchange) in TTP
- Hold all antiplatelet and anticoagulant medications until provider review
- Educate family about disease severity and need for ICU monitoring

Medicines you may be given — and why

caplacizumab

Caplacizumab is a tiny antibody piece that blocks von Willebrand factor (a sticky protein) from attaching to platelets. When von Willebrand factor cannot grab platelets, it stops forming the abnormal clumps in small blood vessels that destroy red blood cells. This drug works immediately while plasma exchange removes the autoantibodies causing TTP.

rituximab

Rituximab finds and kills the B-cells (white blood cells) that make autoantibodies against the ADAMTS13 enzyme in acquired TTP. By removing these antibody-producing cells, the drug stops new autoantibodies from forming and allows normal ADAMTS13 levels to return. Effects begin in days but peak benefit takes weeks as existing antibodies clear.

prednisone or methylprednisolone

Corticosteroids reduce production of autoantibodies against ADAMTS13 by suppressing B-cell and T-cell activity. They also decrease inflammation in small blood vessels damaged by microthrombi. Steroids are added to plasma exchange and rituximab to speed remission and reduce relapse risk in acquired TTP.

eculizumab or ravulizumab

These drugs block the C5 protein in the complement system (part of immune defense). Atypical HUS happens when complement attacks the body's own blood vessel lining. By stopping C5 from splitting into C5a and C5b, the drug prevents the membrane attack complex from punching holes in endothelial cells that trigger microthrombi formation.

folic acid supplementation

Folic acid is needed to make new red blood cells in bone marrow. When red blood cell destruction (hemolysis) destroys red blood cells rapidly, the body uses up folate stores trying to replace them. Supplementing 1 mg daily prevents folate deficiency that would make anemia worse by impairing red blood cell production.

renal replacement therapy (hemodialysis or peritoneal dialysis)

When microthrombi destroy enough kidney glomeruli, waste products (creatinine, urea) and excess fluid accumulate because kidneys cannot filter blood. Dialysis temporarily replaces this filtering function by removing toxins and fluid across an artificial membrane. In HUS, 50-70% of children need temporary dialysis, though most recover kidney function within weeks.

Where this information comes from

- American Society of Hematology (ASH) 2020 Guidelines for Management of Thrombotic Thrombocytopenic Purpura
- American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin No. 222: Gestational Hypertension and Preeclampsia (2020)
- Scully M et al. A phase 2 study of the safety and efficacy of rituximab with plasma exchange in acute acquired thrombotic thrombocytopenic purpura. *Blood*. 2011;118(7):1746-1753
- George JN, Nester CM. Syndromes of thrombotic microangiopathy. *New England Journal of Medicine*. 2014;371(7):654-666

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