

Mis C

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THE STORY IN ONE SENTENCE

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“MIS-C is the body's immune system misfiring weeks after COVID, causing fever, shock, and heart trouble in children—looks a lot like Kawasaki disease.”

Normal anatomy — the body's job

Normally after any virus—flu, cold, or COVID—the immune system ramps up, kills the invader, then gets the signal to stand down and go quiet again. The blood vessels stay strong and sealed, the heart pumps steadily, and inflammation fades within days.

BODY'S JOB

The immune system's job is to recognize a germ, send white blood cells and chemical messengers called cytokines to destroy it, then switch off once the threat is gone. Blood vessels are lined with a smooth layer of cells (the endothelium) that keeps blood flowing smoothly and doesn't let fluid leak out. The heart muscle (myocardium) squeezes rhythmically to push oxygen-rich blood to every organ. After the virus clears, cytokine levels drop, the blood-vessel lining heals, and everything returns to baseline—no swelling, no leaky pipes, no strain on the heart.

1. Virus enters the body (in MIS-C, SARS-CoV-2, the COVID virus).
2. Immune cells (T cells, B cells, macrophages) recognize viral proteins and release cytokines—chemical alarm signals like interleukin-6 (IL-6) and tumor tissue death (necrosis) factor (TNF)—to fight the infection.
3. The virus is cleared from the body, usually within one to two weeks.
4. Cytokine production drops, the alarm shuts off, and regulatory T cells (the immune system's brakes) calm everything down.
5. Blood vessels repair, inflammation fades, fever stops, and the child feels normal again.

What breaks

In MIS-C, the immune off-switch fails. Instead of calming down after COVID is gone, the immune system keeps making alarm chemicals and starts attacking the child's own blood-vessel lining and heart. Certain genes and the way the virus looked to the immune system seem to trip this misfire, though doctors are still learning exactly why.

CLINICAL TERM

Post-infectious immune dysregulation leading to cytokine-mediated endothelial injury, myocarditis, and systemic vasculitis.

How the body compensates

The body tries to defend blood pressure and oxygen delivery by speeding the heart, tightening vessels, and holding onto fluid—but these moves can look reassuring on the monitor even as the child tips toward full shock and organ failure.

1. {'what': 'A fast heart rate (Tachycardia) (fast heart rate)', 'why': 'The heart beats faster to push more blood per minute when each squeeze is weaker, trying to keep oxygen flowing to the brain and kidneys; heart rates above 130–160 in a resting child are a red flag.'}
2. {'what': 'Peripheral vasoconstriction (narrowed outer vessels)', 'why': 'The sympathetic nervous system (the body's fight-or-flight wiring) clamps down blood vessels in the skin, hands, and feet to shunt blood toward the core organs; the child's hands and feet feel cold and look pale or mottled.'}
3. {'what': 'Increased respiratory rate', 'why': 'The child breathes faster to blow off carbon dioxide and try to fix the acid building up in the blood (lactic too much acid in the blood (acidosis)) from poor oxygen delivery; this can look like the child is working hard to breathe even without lung disease.'}
4. {'what': 'Low urine output (Oliguria) (less urine output)', 'why': 'The kidneys get less blood flow, so they make less urine and hold onto salt and water to try to keep the tank full; urine output dropping below 0.5–1 mL/kg/hour signals the kidneys are in trouble.'}
5. {'what': 'Blood pressure may stay normal initially (compensated shock)', 'why': 'The heart racing and vessels squeezing can keep the top number (systolic pressure) in range for hours, hiding how sick the child really is—this is the dangerous window where teams can be falsely reassured.'}
6. {'what': 'Decompensation and low blood pressure (hypotension) (the crash)', 'why': 'Once the heart is too tired and the vessels too leaky, compensation fails suddenly: blood pressure drops, the child becomes unresponsive or limp, and multi-organ failure accelerates—this is the cliff every provider must see coming and prevent.'}

Domino effect

1. SARS-CoV-2 infection triggers initial immune response. — The child catches COVID-19 (often mild or asymptomatic); the immune system learns the virus's spike protein and other pieces, then clears the infection over one to two weeks.
2. Two to six weeks later, the immune system misfires and launches a delayed hyperinflammatory attack. — In genetically susceptible children, antibodies and T cells that were made against the virus start cross-reacting with the child's own tissues (molecular mimicry) or the immune off-switch (regulatory T cells) fails, so cytokines keep flooding out even though the virus is long gone—triggering systemic inflammation.
3. Widespread endothelial injury and vasculitis (inflamed blood vessels throughout the body). — High cytokine levels—especially IL-6, IL-1, and TNF-alpha—damage the endothelium in vessels everywhere: heart, gut, skin, kidneys, brain; the lining swells, gaps open, and fluid leaks into surrounding tissues, causing swelling (edema) (swelling), rash, red eyes, and swollen hands and feet.
4. Myocarditis weakens the heart and coronary arteries develop aneurysms. — Immune cells infiltrate the myocardium, making it thick and stiff so it cannot pump well (reduced ejection fraction, sometimes below 50–55 percent); inflammation also hits the coronary arteries, weakening their walls so they balloon into aneurysms that can clot or burst, risking heart attack even in a young child.
5. Shock develops: distributive (leaky vessels, low resistance) plus cardiogenic (weak pump). — Leaky endothelium drops vascular resistance and lets fluid escape into tissues, while the inflamed heart cannot generate enough pressure; together, blood pressure falls, organs are starved of oxygen, lactic acid builds up, and the child shows cold extremities, confusion, fast breathing, and low urine output.
6. Gastrointestinal and mucocutaneous inflammation appears. — The gut lining is rich in blood vessels and immune cells, so it swells and becomes painful, causing severe belly pain, vomiting, and diarrhea that can mimic appendicitis; mucous membranes (lips, tongue, conjunctiva) turn red and crack, and a polymorphous rash (many shapes—macular, maculopapular) spreads over the trunk and limbs.
7. Impaired blood clotting (Coagulopathy) (abnormal clotting) and clot formation (thrombosis) risk rise. — Inflamed endothelium activates clotting factors and platelets stick to damaged vessel walls; D-dimer and fibrinogen climb, sometimes triggering clots in coronary aneurysms, deep veins, or even pulmonary arteries if inflammation is not controlled quickly.

8. Without immune suppression, the cascade progresses to multi-organ failure. — Untreated cytokine storm and shock damage kidneys (acute kidney injury), liver (elevated transaminases), lungs (acute respiratory distress syndrome), and brain (encephalopathy); mortality climbs if IVIG (intravenous immunoglobulin, which mops up rogue antibodies and cytokines) and corticosteroids (which directly shut down inflammation) are not given promptly to break the cycle.

Symptoms · what the patient feels, and why

- Fever lasting 24 hours or longer (100.4°F / 38°C or higher): The immune system (the body's defense army) stays fired up for days after the coronavirus infection, releasing waves of messenger proteins called cytokines (the body's alarm signals) that reset the brain's temperature thermostat higher and keep it there.
- Belly pain, diarrhea, or throwing up: Inflammation (swelling and irritation) spreads to the gut lining and the blood vessels feeding the intestines, making the belly cramp and the gut lose its normal rhythm so food and water move through too fast or trigger nausea.
- Red, bloodshot eyes without pus (conjunctivitis): Tiny blood vessels in the white part of the eye swell and leak as part of the body-wide inflammatory storm, making the eyes look pink or red—similar to what happens in Kawasaki disease—but no infection is actively growing there.
- Skin rash or bright red, cracked lips and tongue (strawberry tongue): Inflammation in the small blood vessels of the skin and mucous membranes (the moist inner lining of the mouth) causes a blotchy red rash and makes the tongue swell with red bumps that look like a strawberry's surface.
- Swollen hands and feet: Fluid leaks out of inflamed blood vessels in the hands and feet, pooling in the tissue under the skin and causing puffiness—like a sponge soaking up water—because the vessels are no longer sealing tightly.
- Fast heart rate (tachycardia) or chest pain: The heart muscle (myocardium) or the sac around it (pericardium) becomes inflamed, making the heart work harder and beat faster to keep up; chest pain comes from the inflamed tissue rubbing or from the heart struggling to pump against swollen, leaky vessels.
- Swollen lymph nodes in the neck: Lymph nodes (tiny bean-shaped filters that trap germs and alert the immune system) in the neck swell as white blood cells flood in to fight what they think is an active threat, even though the coronavirus is mostly gone and the real problem is the immune system overreacting.
- Trouble breathing or very low blood pressure (shock): Inflammation damages the heart's pumping power and causes blood vessels all over the body to leak and widen, so blood pressure drops dangerously low (shock) and oxygen-rich blood cannot reach the lungs and tissues fast enough, making breathing hard and the body starve for oxygen.

- Feeling very tired, confused, or extremely irritable: The brain receives less oxygen and glucose (its main fuel) when blood pressure drops and inflammation spreads; cytokines (inflammatory messenger chemicals) also cross into the brain and disrupt normal signaling, clouding thinking and mood.
- Recent COVID-19 infection or exposure (usually 2–6 weeks before symptoms start): MIS-C is not the virus itself attacking—it is the immune system launching a delayed, misdirected counterattack weeks after the body has mostly cleared the coronavirus, mistakenly targeting the child's own tissues instead of the germ.

Focused exam

- Fever $\geq 38.0^{\circ}\text{C}$ (100.4°F) lasting more than 24 hours, starting 2–6 weeks after COVID-19 infection or exposure: MIS-C always starts with persistent high fever — this is the immune system flooding the body with inflammatory signals (cytokines) after the original virus is gone, like a smoke alarm that won't shut off even after the fire is out
- Strawberry tongue, cracked red lips, rash on palms/soles, or peeling fingertips: The immune system is attacking the lining of small blood vessels (endothelium) everywhere — the skin and mouth show on the outside what is happening inside the heart and gut
- Muffled or distant heart sounds, a fast heart rate (tachycardia) out of proportion to fever, cold extremities, or capillary refill >2 seconds: The heart muscle (myocardium) and the sac around it (pericardium) are swelling, and the heart is pumping weakly — this is the most dangerous part of MIS-C because it can lead to shock
- Severe belly pain, vomiting, or diarrhea without another explanation: Inflammation is hitting the intestines and the lining of the abdomen (peritoneum) just like it hits blood vessels everywhere else — this is not gastroenteritis, it is part of the systemic inflammatory storm
- Red, bloodshot eyes (conjunctivitis) without pus or discharge: Non-pus-filled (purulent) conjunctivitis is a hallmark mucocutaneous sign — tiny blood vessels in the eyes are inflamed, matching the Kawasaki-like picture seen in MIS-C
- Low blood pressure (Hypotension) (systolic BP <5 th percentile for age), weak pulses, or altered mental status: Shock means the heart can no longer push enough blood to the brain, kidneys, and other organs — this is a medical emergency requiring ICU-level support immediately

Diagnostics · every test answers a question

- Complete blood count (CBC) with differential → Is the immune system activated and are the blood cells responding abnormally?

- C-reactive protein (CRP) and red blood cell (erythrocyte) sedimentation rate (ESR) → How much inflammation is flooding the bloodstream right now?
- Troponin I or T and brain natriuretic peptide (BNP or NT-proBNP) → Is the heart muscle injured or straining to pump?
- D-dimer → Is the blood clotting system activated and forming tiny clots?
- Ferritin → Is there extreme immune activation that could signal cytokine storm?
- SARS-CoV-2 PCR (nasal swab) and SARS-CoV-2 antibody (IgG) → Was there recent or past COVID-19 infection triggering this response?
- Echocardiogram (echo) → Are the coronary arteries swollen (aneurysms) or is the heart muscle too weak to pump?
- Albumin and liver enzymes (ALT, AST) → Are the liver and blood vessels leaking protein because of widespread inflammation?

Treatment logic

- Intravenous immunoglobulin (IVIG) 2 g/kg as a single dose over 8–12 hours → IVIG floods the bloodstream with pooled antibodies from thousands of donors, which blocks the overactive immune cells (B cells and macrophages) from releasing more inflammatory signals (cytokines) — this calms the immune storm attacking the heart, blood vessels, and other organs
- Methylprednisolone 1–2 mg/kg/day IV (or equivalent corticosteroid) → Steroids turn down the genes inside immune cells that make inflammatory proteins (cytokines like IL-6 and TNF-alpha) — this stops the immune system from dumping more fuel on the fire and reduces swelling in the heart, blood vessels, and gut
- Aspirin: high-dose (30–50 mg/kg/day in 4 divided doses) during acute phase if Kawasaki-like features, then low-dose (3–5 mg/kg/day once daily) once fever resolves → Aspirin blocks an enzyme (COX-1) that makes platelets sticky — this prevents clots from forming inside inflamed coronary arteries where blood flow is turbulent and vessel walls are damaged
- Echocardiogram at diagnosis, 1–2 weeks, and 4–6 weeks (cardiology-guided schedule) → Repeated echo imaging shows whether coronary arteries are dilating into aneurysms (z-score ≥ 2.5) and whether heart muscle function (ejection fraction) is recovering — this guides how long to continue aspirin, whether to add anticoagulation, and whether the child can return to normal activity
- IV fluids (cautious boluses, 10–20 mL/kg) and inotropes (epinephrine, milrinone) if shock or low blood pressure develops → Fluid boluses expand the blood volume so there is more liquid to push through the circulation; inotropes make the heart muscle squeeze harder and blood vessels relax — this keeps oxygen-rich blood flowing to the brain, kidneys, and other vital organs while the immune storm is still raging

- Anakinra (IL-1 receptor antagonist, 2–10 mg/kg/day) or infliximab (TNF-alpha blocker, 5–10 mg/kg) if no improvement after 24–48 hours of IVIG and steroids → Anakinra blocks interleukin-1 (IL-1), a key inflammatory messenger that keeps the cytokine storm going; infliximab blocks tumor tissue death (necrosis) factor-alpha (TNF-alpha), another major driver of inflammation — these target specific molecules in the immune cascade when first-line treatment is not enough
- Anticoagulation (enoxaparin, warfarin, or aspirin dose escalation) if coronary artery z-score ≥ 2.5 or giant aneurysm (z-score ≥ 10 or absolute diameter ≥ 8 mm) → Anticoagulants thin the blood more than aspirin alone, preventing clots from forming in large or giant coronary aneurysms where slow, turbulent blood flow creates high clot risk — this reduces the chance of a heart attack (myocardial tissue death from blocked blood flow (infarction)) from a clot blocking the artery

Medications

Intravenous Immunoglobulin (IVIG) · Pooled antibody therapy

IVIG is a mix of antibodies (germ-fighting proteins) collected from thousands of healthy donors. When given through an IV, these antibodies flood the bloodstream and calm down the immune system that has gone haywire. They block the harmful antibodies the child's own body is making, they soak up inflammatory signals like sponges, and they tell white blood cells to stop attacking the body's own tissues. This cools the fierce swelling in blood vessels, the heart, and other organs that MIS-C causes.

1. IVIG antibodies enter the bloodstream and spread to every tissue.
2. They bind to and block the child's own harmful antibodies that are mistakenly attacking the body.
3. They attach to receptors on immune cells (like macrophages and T cells) and send a 'stand down' signal.
4. They soak up cytokines (inflammatory messenger chemicals such as IL-6 and TNF-alpha) that are causing fever, swelling, and organ damage.
5. Inflammation in the heart, blood vessels, gut, and other organs begins to drop within hours to days.
6. Fever usually breaks within 24 to 48 hours if IVIG works well.

SAFETY WATCH

Allergic reaction during the infusion: watch for hives, trouble breathing, or chest tightness; slow or stop the drip if this happens. · Headache, chills, or fever during or after the infusion (common and usually mild). · Rarely, blood clots in children who already have very thick blood or severe inflammation—this is why some children also get aspirin. · Rarely, kidney injury in children who are dehydrated or have pre-existing kidney problems—ensure good hydration before the infusion. · Rarely, aseptic meningitis (sterile inflammation of the brain lining) causing severe headache and stiff neck hours to days later. · Very rarely, red blood cell destruction (hemolysis) (red blood cell breakdown) in children with certain blood types—monitor blood counts after the dose.

Methylprednisolone · Corticosteroid (immune suppressor)

Methylprednisolone is a strong steroid medicine that mimics cortisol, the body's own 'calm-down' hormone. It shuts off the genes inside immune cells that make inflammatory proteins (cytokines), and it tells those cells to stop multiplying and releasing their attack chemicals. This cools the runaway inflammation in MIS-C, especially when IVIG alone is not enough or when the child has very severe disease with shock or heart problems.

1. Methylprednisolone enters immune cells and binds to a receptor inside the cell.
2. The drug-receptor pair moves into the nucleus (the cell's control center).
3. It turns OFF genes that code for inflammatory cytokines (such as IL-1, IL-6, and TNF-alpha) and turns ON genes that make anti-inflammatory proteins.
4. Fewer cytokines are released, so the fever, swelling, and organ damage slow down.
5. Blood vessels become less leaky, blood pressure stabilizes, and the heart's workload decreases.
6. Effect builds over hours to days; children often improve faster when steroids are added to IVIG.

SAFETY WATCH

High blood sugar—steroids make the body resist insulin, so check glucose levels regularly, especially in children getting continuous infusions. · High blood pressure—steroids can cause the body to hold onto salt and water; monitor blood pressure closely. · Mood changes, trouble sleeping, or hyperactivity (common even with short courses). · Increased risk of infection if used for a long time, because steroids suppress the immune system. · Stomach irritation or rarely bleeding—give with food or a stomach-protecting medicine if needed. · Rarely, avascular tissue death (necrosis) (bone tissue death) with prolonged high doses, though MIS-C courses are usually short. · Do not stop abruptly after several days of high doses—taper the dose slowly to let the adrenal glands (which make natural cortisol) wake back up.

Aspirin (low-dose) · Antiplatelet agent (clot preventer)

Low-dose aspirin (typically 3 to 5 mg per kg per day) stops platelets (tiny blood cells that form clots) from sticking together. It does this by blocking an enzyme inside platelets called COX-1, which normally makes a chemical (thromboxane A2) that tells platelets to clump. By preventing clumping, aspirin reduces the risk of dangerous clots forming in inflamed blood vessels, especially the coronary arteries that feed the heart. In MIS-C, inflammation can damage the lining of blood vessels and make clots more likely, so this protection is critical.

1. Aspirin is absorbed in the stomach and enters the bloodstream.
2. It permanently blocks the COX-1 enzyme inside platelets.
3. Without working COX-1, platelets cannot make thromboxane A2, the signal that makes them sticky.
4. Platelets become less able to clump together and form clots.
5. Blood flows more smoothly through inflamed or damaged vessels, reducing the chance of a heart attack or stroke.
6. The effect lasts for the lifetime of each platelet (about 7 to 10 days), so even a single daily dose keeps protection steady.

SAFETY WATCH

Bleeding risk—aspirin makes it harder for blood to clot, so watch for unusual bruising, nosebleeds, blood in urine or stool, or prolonged bleeding from cuts. · Stomach irritation or pain—aspirin can damage the stomach lining; give with food to reduce this. · Very rarely, Reye syndrome (a serious liver and brain condition) if aspirin is given during or right after a viral illness in young children—this is why aspirin is avoided in most childhood fevers, BUT in MIS-C the benefit outweighs this small risk, and most children are past the acute viral phase. · Allergic reaction (rare)—rash, wheezing, or swelling. · Do not give aspirin with other NSAIDs (like ibuprofen) without checking with the care team, as they can interfere with each other.

Aspirin (high-dose) · Nonsteroidal anti-inflammatory drug (NSAID) and antiplatelet agent

High-dose aspirin (typically 30 to 50 mg per kg per day, divided into four doses) works in two ways. First, it blocks the COX enzymes in many cells, which cuts down production of prostaglandins (chemicals that cause fever, pain, and swelling). This cools inflammation throughout the body. Second, it still prevents platelets from clumping, just as low-dose aspirin does. High-dose aspirin is used when MIS-C looks very much like Kawasaki disease (another condition with severe blood vessel inflammation and risk of coronary artery aneurysms) or when inflammation is especially fierce.

1. High-dose aspirin is absorbed and spreads through the bloodstream to inflamed tissues.
2. It blocks both COX-1 and COX-2 enzymes in many cell types.
3. With less COX activity, cells make fewer prostaglandins, so fever drops, pain eases, and swelling decreases.
4. At the same time, aspirin permanently shuts off COX-1 in platelets, preventing them from making the clumping signal thromboxane A₂.
5. Inflammation and clot risk both fall, protecting the heart and other organs.
6. Once fever and inflammation are controlled (usually after a few days to a week), the dose is lowered to the low antiplatelet dose to continue clot prevention.

SAFETY WATCH

Stomach upset, pain, or rarely bleeding or ulcers—give with food and consider a stomach-protecting medicine (like a proton pump inhibitor) during high-dose treatment. · Bleeding risk—watch for bruising, nosebleeds, blood in stool or urine, or prolonged bleeding from cuts. · Ringing in the ears (Tinnitus) (ringing in the ears) or hearing changes can signal the dose is too high—tell the care team right away. · Rarely, Reye syndrome if given during or just after a viral illness in young children—risk is low in MIS-C because most children are past the acute viral phase, but the team monitors closely. · Allergic reaction (rare)—rash, wheezing, or swelling. · Liver enzyme elevation—checked with blood tests during treatment. · Do not stop high-dose aspirin abruptly; taper to low-dose aspirin as the inflammation improves, then continue low-dose for weeks to months (depending on whether coronary artery changes are present).

Enoxaparin · Low molecular weight heparin (anticoagulant, blood thinner)

Enoxaparin is a blood thinner given by injection under the skin. It works by boosting a natural protein in the blood called antithrombin, which then blocks several clotting factors (especially Factor Xa) in the clotting cascade. This stops the blood from forming large, dangerous clots. In MIS-C, if a child has very severe inflammation, thick blood, or already has clots in blood vessels or the heart, enoxaparin provides stronger clot prevention than aspirin alone. It is often used in the ICU when a child has poor heart function or evidence of clots on imaging.

1. Enoxaparin is injected under the skin and absorbed into the bloodstream.
2. It binds to antithrombin, a natural clotting inhibitor in the blood.
3. The enoxaparin-antithrombin complex rapidly inactivates Factor Xa, a key enzyme in the clotting cascade.
4. With Factor Xa blocked, the cascade cannot proceed to form thrombin (the enzyme that makes fibrin clots).
5. Clot formation slows or stops, and blood flows more freely through inflamed or narrowed vessels.
6. Doses are adjusted based on blood tests (anti-Xa levels) to keep clot prevention in the safe range.

SAFETY WATCH

Bleeding risk—enoxaparin can cause serious bleeding, so watch for unusual bruising, blood in urine or stool, nosebleeds, or bleeding at injection sites. · Injection site reactions—redness, pain, or small bruises where the medicine is given. · Rarely, heparin-induced a low platelet count (thrombocytopenia) (HIT), a condition where platelet counts drop and clotting actually increases—monitor platelet counts regularly. · Rarely, allergic reaction. · Spinal or epidural a pooled collection of blood (hematoma) (bleeding around the spine) if a spinal procedure is done while the child is on enoxaparin—avoid spinal taps or epidurals unless absolutely necessary and the drug has been held for the right amount of time. · Dose must be adjusted for kidney function, because enoxaparin is cleared by the kidneys. · Do not stop abruptly if the child has high clot risk; transition to aspirin or another agent as the care team directs.

Anakinra · Interleukin-1 receptor antagonist (targeted immune blocker)

Anakinra is a synthetic copy of a natural protein that blocks the receptor for interleukin-1 (IL-1), a powerful inflammatory messenger chemical (cytokine). In MIS-C, immune cells are churning out huge amounts of IL-1, which drives fever, swelling, and tissue damage. Anakinra acts like a 'decoy key' that fills up the IL-1 receptors on cells so the real IL-1 cannot plug in. With IL-1 blocked, the inflammatory cascade cools down. Anakinra is used when IVIG and steroids are not enough or when the child is still very sick despite initial treatment.

1. Anakinra is given by injection under the skin or through an IV.
2. It enters the bloodstream and travels to inflamed tissues.
3. Anakinra binds tightly to IL-1 receptors on the surface of cells.
4. With the receptor occupied, IL-1 (the inflammatory cytokine) cannot attach and send its 'keep attacking' signal.
5. Cells stop releasing more inflammatory chemicals, fever drops, and swelling decreases.
6. Heart function and blood pressure often stabilize within 24 to 48 hours if the child responds well.

SAFETY WATCH

Increased risk of infection, because blocking IL-1 weakens part of the immune defense—watch for new fever, cough, or other signs of infection. · Injection site reactions—redness, swelling, or pain where the shot is given (common and usually mild). · Rarely, low white blood cell count—monitor blood counts during treatment. · Rarely, allergic reaction. · Rarely, liver enzyme elevation—checked with blood tests. · Do not give live vaccines while on anakinra, because the immune system is partly suppressed.

Infliximab · Tumor tissue death (necrosis) factor-alpha (TNF-alpha) inhibitor (targeted immune blocker)

Infliximab is a laboratory-made antibody that binds to and neutralizes tumor tissue death (necrosis) factor-alpha (TNF-alpha), a key inflammatory messenger chemical (cytokine) that drives swelling, fever, and tissue damage. In MIS-C, immune cells release floods of TNF-alpha, which tells blood vessels to leak, organs to swell, and the heart to struggle. Infliximab acts like a sponge that soaks up TNF-alpha before it can plug into receptors on cells and trigger inflammation. It is used when IVIG and steroids are not enough or when the child is still critically ill despite other treatments.

1. Infliximab is given through an IV infusion.
2. It circulates in the bloodstream and binds tightly to TNF-alpha molecules.
3. With TNF-alpha neutralized, it cannot attach to receptors on immune cells and blood vessel walls.
4. Cells stop releasing more inflammatory chemicals, and blood vessels become less leaky.
5. Fever drops, blood pressure stabilizes, and organ function (especially heart and lung) often improves within 24 to 72 hours.
6. Infliximab has a long half-life, so a single dose can have effects for days to weeks.

SAFETY WATCH

Increased risk of serious infections (bacterial, viral, fungal, and tuberculosis reactivation), because TNF-alpha is important for fighting certain germs—screen for tuberculosis before starting if possible, and watch closely for new infections. · Infusion reactions—fever, chills, rash, chest tightness, or drop in blood pressure during or shortly after the infusion; slow the infusion and give premedication (like antihistamines or steroids) to reduce risk. · Rarely, new or worsening heart failure—monitor heart function closely, especially in children who already have poor heart function. · Rarely, allergic reaction or formation of antibodies against infliximab, which can reduce its effect or cause delayed reactions. · Rarely, autoimmune reactions (like lupus-like syndrome) or liver injury—monitor liver enzymes and watch for new rashes or joint pain. · Do not give live vaccines while on infliximab or for several months after, because the immune system is partly suppressed. · Very rarely, increased risk of certain cancers (like lymphoma) with long-term use, though MIS-C treatment is usually short.

Red flags

- Chest pain, trouble breathing, or lips/face turning blue—the heart or lungs may be failing.
- Blood pressure dropping (shock): cold hands and feet, confusion, very fast heart rate, or not peeing—the body is not getting enough blood flow.

- Severe belly pain that will not go away—inflammation may be damaging the intestines or other organs.
- New confusion, seizure, or extreme sleepiness—the brain is not getting enough oxygen or is being affected by the inflammation.
- Swelling or bulging of a blood vessel in the heart (coronary artery aneurysm)—a dangerous complication that can lead to a heart attack even in a child.

Nursing priorities

1. Check blood pressure, heart rate, capillary refill (how fast skin color returns after pressing a fingernail), and urine output every 1 to 2 hours — MIS-C causes vasodilation (blood vessels open too wide) and capillary leak (fluid leaving blood vessels into tissues), dropping blood pressure fast into shock, so early catching means we start fluid boluses and pressors before organs fail
2. Place the child on continuous cardiac monitoring (heart monitor showing rhythm on screen) and watch for arrhythmias (abnormal heart beats) or ST-segment changes on the ECG (electrical tracing of heart) — Myocarditis from MIS-C inflammation damages heart muscle cells and electrical pathways, causing dangerous rhythms like ventricular a fast heart rate (tachycardia) (fast rhythm from bottom heart chambers) or heart blocks that can stop the heart
3. Measure strict intake and output, weighing every diaper or using a urometer (urine measuring chamber), and report urine less than 1 milliliter per kilogram per hour — Kidneys need good blood flow to make urine, so dropping urine output is the earliest sign that shock is starting or that we gave too much fluid and the heart cannot pump it, both needing immediate treatment changes
4. Give IVIG slowly over 10 to 12 hours through a dedicated IV line, checking vital signs every 15 minutes for the first hour, and watch for headache, chills, or back pain — IVIG contains immunoglobulins (infection-fighting proteins from donated blood) that calm the overactive immune system, but infusing too fast causes volume overload in an already weak heart or allergic reactions including anaphylaxis (whole-body allergic emergency)
5. Assess for new chest pain, shortness of breath, or decreased oxygen saturation below 92 percent, and notify the provider immediately if any appear — These symptoms mean heart failure is worsening from myocarditis or pericardial a fluid collection (effusion) (fluid around the heart sac) is building up, and the child may need diuretics (water pills), oxygen, or transfer to intensive care for inotropes (medicines that strengthen heart squeeze)
6. Do not give NSAIDs like ibuprofen until after the echocardiogram and cardiology team approves — NSAIDs (non-steroidal anti-inflammatory drugs like Motrin) can worsen heart function and cause more inflammation in damaged heart muscle, and we need the echo first to see how weak the heart is before choosing safe fever medicines

7. Check inflammatory markers (CRP, ESR, ferritin, D-dimer) and cardiac enzymes (troponin, BNP) when ordered, and track if numbers are going down after treatment starts — CRP (C-reactive protein showing body inflammation), ferritin (iron storage protein that rises with inflammation), troponin (protein released when heart cells die), and BNP (brain natriuretic peptide showing heart strain) tell us if IVIG and steroids are working or if we need rescue medicines like anakinra or infliximab at 48 hours
8. Teach family that MIS-C is not contagious now, the child needs 4 to 6 weeks of heart activity restrictions, and cardiology follow-up appointments are required even if the child feels better — Parents fear spreading disease and want the child back to sports fast, but coronary aneurysms (balloon-like bulges in heart arteries) can form weeks later even after inflammation calms, and those aneurysms can cause heart attacks in children if not monitored with repeat echocardiograms

Sources

- CDC Health Alert Network (HAN) on MIS-C
- American Academy of Pediatrics (AAP) interim guidance on MIS-C
- American College of Rheumatology (ACR) 2020–2022 clinical guidance for MIS-C
- New England Journal of Medicine (NEJM) case series and reviews on MIS-C

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