MERCOLA
Take Control of Your Health

Over 50% of Heart Attacks in Younger Women Aren't from Clogged Arteries

Analysis by Dr. Joseph Mercola | April 08, 2026

STORY AT-A-GLANCE

- More than half of heart attacks in younger women are not caused by blocked arteries, which means conventional screening focused on cholesterol and plaque often misses real risk
- Oxygen supply imbalance — frequently triggered by anemia, illness, or physical stress — is a major driver of heart attacks in women, making underlying health conditions direct cardiovascular risk factors
- Artery tears and other non-plaque causes are often misclassified, which leads to treatment strategies that fail to address the true mechanism behind the event
- Iron imbalance adds another hidden layer because excess iron inside heart cells drives oxidative damage that weakens cardiac tissue and worsens injury during heart stress
- Testing ferritin and transferrin saturation (TSAT) together — and keeping iron within an optimal range — supports cellular energy, improves prevention strategies, and reduces long-term heart risk

Heart attack — medically called myocardial infarction — occurs when blood flow to your heart muscle drops or stops, meaning heart tissue begins to suffer injury or die. It's characterized by chest pressure, shortness of breath, fatigue, nausea, dizziness, and pain that spreads to your jaw, arm, or back. Left untreated, it leads to permanent heart damage, heart failure, rhythm disturbances, and death.

For decades, the standard explanation has centered on blocked arteries — plaque builds up, a clot forms, and blood flow cuts off. That explanation holds for most men, but research reveals it fails to account for the majority of heart attacks in younger women, where entirely different mechanisms drive the event.

At the same time, separate research into iron regulation inside heart cells exposes a hidden layer of cardiovascular damage that standard screening overlooks. Together, these findings force a rethink of how heart attacks develop, why younger women face unique risks, and which overlooked biological factors shape outcomes — starting with what a large population study actually found when investigators classified each event by its true cause.

Heart Attacks in Women Often Start Differently

For a study published in the Journal of the American College of Cardiology, researchers examined heart injury events in adults age 65 and younger to determine what actually caused each heart attack, rather than assuming plaque blockage as the default explanation.[1] Instead of relying on broad diagnostic labels, they reviewed individual medical records, imaging, and lab data to classify each event by its biological cause.

Findings showed plaque-related heart attacks **dominated in men**, yet represented less than half of events in women, where alternative causes formed the majority. This means risk assessment based on **cholesterol** or artery blockage leaves major blind spots, especially if you're female or younger than typical heart disease populations.

- **More than half of women's heart attacks had non-plaque causes —** Atherothrombosis — the classic plaque-and-clot scenario — accounted for 75% of heart attacks in men but only 47% in women. In other words, heart attacks fall into multiple biological categories. The classic plaque-and-clot event is only one pathway.

Others occur when oxygen delivery drops despite open arteries, when the artery wall tears, when small vessels malfunction, or when a clot travels from elsewhere. Recognizing these pathways explains why many women experience heart injury even when imaging doesn't show major blockages. This shifts prevention away from a single pathway and toward a broader understanding of stress, inflammation, blood flow, and cellular health.

- **Supply-demand mismatch emerged as a dominant driver —** Researchers identified secondary myocardial infarction caused by oxygen supply and demand imbalance as a major contributor, representing 34% of events in women compared with 19% in men.[2] Supply-demand mismatch occurs when the heart's oxygen needs rise faster than the body can supply them.

  The artery may remain open, yet the muscle still becomes oxygen-deprived — similar to an engine that stalls under heavy load despite an unobstructed fuel line. This often occurs during illness, anemia, infection, or physical stress. This highlights why fatigue, illness, or metabolic strain play a direct role in heart risk rather than acting as background factors.

- **Artery tears were far more common than previously recognized —** Spontaneous coronary artery dissection, meaning a tear inside the artery wall that traps blood and blocks flow, occurred far more often in women and frequently went misclassified at the time of diagnosis. In spontaneous coronary artery dissection, blood enters the artery wall and creates a pocket that compresses the channel carrying blood forward.

  The blockage comes from within the wall itself rather than from plaque inside the artery. The study reported many of these cases were initially labeled as plaque events even though the mechanism differed entirely. Misclassification matters because treatments that target plaque don't address artery wall injury, which affects recurrence risk and recovery.

- **Traditional risk scoring failed to identify many patients —** Separate analysis within the research showed that 45% of individuals who experienced a first heart attack would have been categorized as low or borderline risk shortly before the event using standard cardiovascular scoring systems. This explains why many first heart attacks appear unexpected — risk tools focused on plaque overlook mechanisms that develop through stress, illness, vascular injury, and oxygen imbalance.

- **Mortality varied by cause rather than by heart attack label —** Five-year outcomes differed significantly across mechanisms, with supply-demand mismatch showing the highest overall mortality at 33% compared with 8% for plaque-related events and near-zero cardiovascular mortality after artery tear events. This reveals that the underlying trigger — not simply the presence of a heart attack — determines long-term prognosis.

- **Heart injury develops through multiple pathways —** This includes plaque blockage, artery wall disruption, oxygen imbalance, clot migration, and vessel spasm. Each pathway requires a different prevention strategy, medication approach, and follow-up plan.

Iron Inside Heart Cells Drives Hidden Damage

The first study identified oxygen supply-demand mismatch as the leading non-plaque cause of heart attacks in women, with anemia ranking among the key triggers. The logical response seems straightforward — restore iron, fix the anemia, protect your heart. But a second body of research reveals that equation is far more complicated than it appears.

Excess iron itself, when it accumulates inside heart cells, becomes a direct source of the kind of damage that drives heart failure and worsens injury during cardiac events.

For a study published in Circulation Research, researchers explored how iron balance at the cellular and mitochondrial level influences cardiovascular disease, heart failure, and injury during reduced blood flow events.[3]

The paper focused on how iron functions as a required mineral for enzyme activity and energy production while also acting as a catalyst for chemical reactions that damage cells when levels rise beyond control. Blood tests can suggest iron deficiency while heart tissue simultaneously accumulates iron. In practical terms, your bloodstream can appear depleted even as cells store excess iron that drives oxidative damage, which complicates treatment decisions.

- **Cellular iron accumulation linked to structural heart damage —** The paper described how excess iron inside heart cell mitochondria — the structures that generate energy — increases formation of highly reactive molecules that damage DNA, proteins, and cell membranes.

  These molecules, known as reactive oxygen species, accelerate tissue injury and contribute to the development of heart failure and cardiomyopathy. In practical terms, this means iron overload drives wear and tear at the level where heart energy is produced.

- **Iron-driven chemical reactions amplify oxidative stress —** The paper detailed how iron participates in a reaction where iron converts hydrogen peroxide into hydroxyl radicals — the most damaging form of reactive oxygen species.

  These reactions trigger lipid peroxidation, meaning cell membrane fats degrade, which weakens heart cells and disrupts function. This mechanism helps explain how excess iron accelerates structural damage within heart tissue and worsens injury during cardiac stress.

- **Evidence showed mitochondrial iron increases during cardiac injury events —** Researchers reported that mitochondrial iron rises during ischemia-reperfusion injury — meaning tissue damage that occurs when blood supply returns after a period of restriction — and that adjusting baseline iron levels reduces injury severity in experimental models.

- **Excess iron links to broader chronic disease risk —** The paper also noted that increased tissue iron appears across multiple chronic conditions, including neurological diseases, kidney disease, and cancer, where abnormal iron distribution contributes to cellular injury and disease progression.

  Iron accumulation has been observed in aging tissues and in neurodegenerative disorders such as Parkinson's and Alzheimer's disease, highlighting that the impact extends beyond the heart. Iron balance influences whole-body health, not just cardiovascular outcomes, which makes testing and monitoring a central long-term strategy.

Address Hidden Drivers Behind Heart Attacks

Many heart attacks begin long before plaque blocks an artery. The real issue often involves oxygen delivery, vascular stress, and cellular energy strain. A practical approach starts with recognizing that standard screening misses many of these triggers — and that the actions you take before and after a cardiac event shape outcomes as much as emergency treatment does.

1. **Know that heart attack symptoms in women often look different —** Pressure, jaw pain, nausea, extreme fatigue, and shortness of breath without classic crushing chest pain still warrant emergency evaluation. Delayed recognition is one reason non-plaque heart attacks in women go misclassified.

2. **Ask what caused the event, not just whether one occurred —** If you or someone you know experiences a heart attack, push for classification beyond the default plaque assumption. The study showed misclassification led to ineffective or harmful treatment — particularly for artery tears initially labeled as plaque events. The specific cause determines which treatment, follow-up, and prevention strategies actually help.

3. **Don't rely solely on standard risk scores —** Forty-five percent of first heart attacks in the study occurred in people rated low or borderline risk. If you have unexplained fatigue, episodes of chest tightness during illness or stress, or a family history that doesn't fit the typical plaque profile, advocate for deeper evaluation rather than accepting a clean bill of health from a standard screening.

4. **Treat underlying stressors that create oxygen imbalance —** Since supply-demand mismatch drove 34% of women's heart attacks — often triggered by anemia, infection, or physical stress — managing these conditions directly improves oxygen delivery to your heart and reduces vulnerability during stress. Resolving anemia matters, but so does addressing infection, chronic illness, and recovery from acute stress rather than treating them as separate from heart risk.

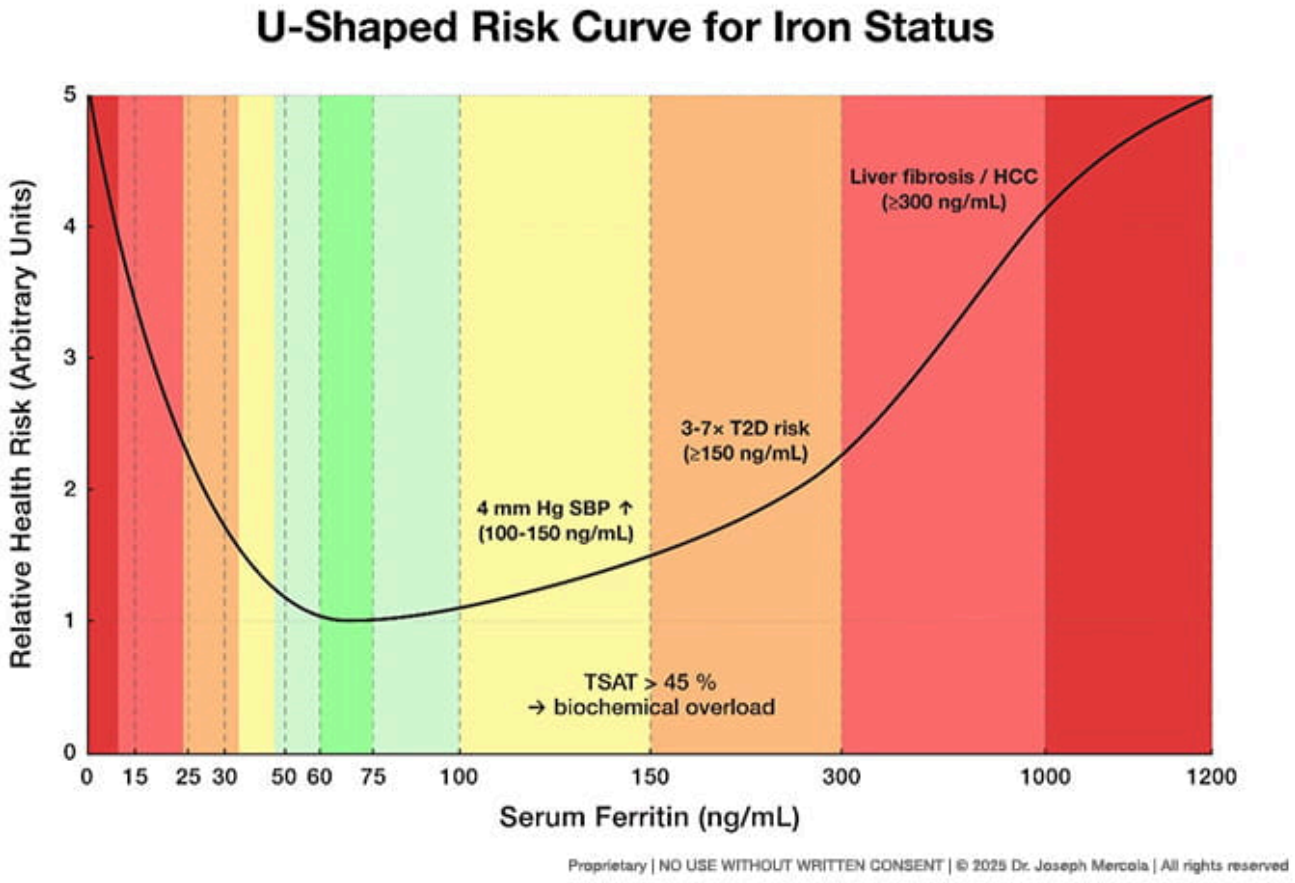
5. **Test your iron status before adding iron — and always read two markers together —** Iron supports heart function only within a narrow physiological range, where both deficiency and excess influence risk. Too little reduces oxygen delivery and energy production, while excess iron inside cells accelerates oxidative injury. Heart risk emerges at both extremes. Ferritin and transferrin saturation (TSAT) work as a pair to reveal if your iron levels are where they should be.

   Ferritin measures what's in the warehouse, while TSAT tells you what percentage of delivery trucks are actually carrying cargo right now. Reading ferritin alone leads to bad decisions — TSAT provides the context that makes the number meaningful. If ferritin is low and TSAT falls below 25%, true iron deficiency is likely — stores are depleted and delivery is poor.

   If both climb high — ferritin above 100 ng/mL and TSAT above 45% — iron overload becomes the concern. A trickier pattern appears when ferritin runs high but TSAT stays low or normal, which often signals that inflammation is trapping iron in storage and masking how little is actually available for use.

For both men and women, the ideal zone is typically ferritin between 50 and 100 ng/mL and TSAT between 25% and 35%. Below 15 ng/mL, ferritin signals depleted reserves. Above 150 ng/mL, excess iron feeds the oxidative damage described earlier.

Because fatigue shows up at both extremes, supplementing based on how you feel rather than what your labs show risks pushing levels in the wrong direction. Annual testing provides a baseline, while more frequent testing may be appropriate during pregnancy, heavy training, chronic illness, or after iron supplementation.



6. **Lower excess iron when levels are too high —** When iron levels are elevated, periodic blood donation gradually reduces stored iron and lowers oxidative burden. Individuals unable to donate may require therapeutic phlebotomy under medical supervision. Donation frequency should be guided by repeat testing to avoid shifting from overload into deficiency.

Training intensity, menstrual status, pregnancy, illness, and genetics all influence iron needs and safe ranges. Periodic testing is important to ensure your iron levels stay optimized. Iron works best within a narrow range — viewing it as a dial rather than a default supplement creates a clear path toward protecting cardiovascular health over time.

FAQs About Heart Attacks in Younger Women and Iron Balance

Q: Why aren't many heart attacks in younger women caused by clogged arteries?

A: Research shows a large share of heart attacks in younger women result from causes other than plaque buildup, including oxygen supply imbalance, artery tears, vessel spasm, and clot movement from elsewhere in the body. This means conventional screening focused mainly on cholesterol and plaque misses important risk pathways.

Q: What is supply-demand mismatch and why does it matter for heart risk?

A: Supply-demand mismatch happens when your heart needs more oxygen than your body delivers. Illness, anemia, infection, and intense physical or metabolic stress create this imbalance. When oxygen demand exceeds supply, heart tissue becomes vulnerable even without artery blockage.

Q: Why can standard heart risk scores miss people who later have heart attacks?

A: Common risk tools focus heavily on plaque-related factors such as cholesterol, blood pressure, and age. The research found many individuals who experienced a first heart attack were categorized as low or borderline risk shortly beforehand, highlighting the limits of plaque-focused screening.

Q: How does iron influence heart health beyond anemia?

A: Iron plays a dual role. It supports oxygen transport and cellular energy production, yet excess iron inside heart cells drives oxidative stress that damages DNA, proteins, and cell membranes. Blood tests can show low circulating iron while tissue iron remains elevated, which complicates treatment decisions.

Q: What is the safest way to manage iron levels for heart protection?

A: Testing ferritin and TSAT together provides the clearest picture because ferritin reflects stored iron and TSAT shows how much iron circulates and is usable. Keeping iron within an optimal range — rather than assuming more iron is beneficial — helps reduce oxidative stress and supports long-term cardiovascular health.

Sources and References

- [1] Journal of the American College of Cardiology September 15, 2025, Volume 86, Number 12
- [2] Science Alert December 28, 2025
- [3] Circ Res. 2016 Nov 11;119(11):1164–1166