Healthy Her

A Call to Reframe Menopause in Clinical Practice

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Menopause Is Not a Symptom Problem. It is a Systems Problem We Must Acknowledge. The gap in care is not accidental. It is structural.

Women have historically been underrepresented in cardiovascular trials.
Cardiology has largely focused on obstructive, male-pattern disease.
Menopause has remained siloed within gynecology, disconnected from cardiovascular risk assessment.

As a result, we have failed to integrate one of the most important physiologic transitions in a woman's life into our prevention models.
And patients are paying the price.

For decades, menopause has been framed as a quality-of-life transition defined by hot flashes, night sweats, mood changes, and sleep disruption. But that framing is not only incomplete, but for many women, outright dangerous.

Menopause is not simply a nuisance. It is a biologic transition with profound cardiovascular, metabolic, and inflammatory consequences, occurring at a time when prevention matters most.

Think about this. Women will spend nearly half of their lives in the postmenopausal state. Yet, we continue to treat menopause as a peripheral concern rather than what it truly represents:
a critical inflection point in cardiovascular risk.

Estrogen is not simply a reproductive hormone. It is a vascular and metabolic regulator in women. It supports endothelial function through nitric oxide production, promotes favorable lipid profiles, enhances insulin sensitivity, and helps regulate fat distribution toward subcutaneous rather than visceral stores. It also exerts anti-inflammatory effects at the vascular level. This is the physiology we were never taught to prioritize in medical school nor in our training programs.

With the loss of estrogen, this physiologic balance shifts. What follows is not subtle:

- Increased vascular stiffness
- Rising LDL and ApoB
- Worsening insulin resistance
- Redistribution of fat to visceral depots
- Escalation of systemic inflammation

Further, the transition through perimenopause into menopause is marked by measurable and clinically meaningful changes:

- Blood pressure rises
- Lipid profiles worsen
- Glucose regulation becomes impaired
- Visceral adiposity increases despite stable weight

These changes converge to accelerate atherosclerosis and contribute to conditions disproportionately seen in women, including:

- Heart failure with preserved ejection fraction (HFpEF)
- Ischemia with non-obstructive coronary arteries (INOCA)
- Coronary microvascular dysfunction

This is not cosmetic. This is cardiometabolic remodeling. Yet, this risk shift is rarely treated with urgency. Instead, symptoms are often normalized, minimized, or attributed solely to "aging." Moreover, there is a diagnostic gap when normal is not normal, as women are more likely than men to present with cardiovascular disease that is not easily detected with our traditional diagnostic tools. This diagnostic gap contributes to delayed care, undertreatment, and persistent disparities in outcomes for women.

Menopause should trigger a systematic reassessment of cardiovascular risk. Instead, this very rarely happens. At a minimum, physicians should be evaluating:

- Lipid profiles (with consideration of ApoB and Lp(a))
- Glucose metabolism (A1c, and when appropriate, insulin resistance)
- Blood pressure trends, ideally including home monitoring
- Changes in body composition, particularly visceral adiposity
- Sleep quality and duration

When appropriate, additional tools such as coronary artery calcium (CAC) scoring or coronary CT imaging can refine risk assessment further.

Therapies should be assessed including MHT (menopausal hormone therapy). We must move beyond binary thinking on hormones, even while it remains one of the most misunderstood aspects of menopause care. To be clear, it is not a universal solution, but, neither should it be dismissed outright.

The clinical reality is nuanced:

- Timing matters
- Route of administration matters
- Patient selection matters

To be clear, hormone therapy is currently not primarily a cardiovascular prevention strategy. However, it favorably influences multiple cardiometabolic pathways, including lipid metabolism, vascular function, and insulin sensitivity.

It should certainly be considered within the context of an individualized, comprehensive risk strategy. It is time to move beyond isolation, and outdated, oversimplified narratives.

Of particular importance are aggressive lifestyle interventions. This is not a nice to have, it is foundational to the menopausal transition.

Particularly important are:

- Resistance training to preserve muscle mass and metabolic health
- Adequate protein intake
- Sleep optimization
- Reduction in alcohol intake
- Stress regulation and cortisol management

These interventions carry amplified benefit during the menopausal transition, when metabolic vulnerability is heightened. As such, menopause should not be viewed as an endpoint. It is a **signal**. An opportunity to:

- Reassess risk
- Initiate prevention strategies
- Identify early disease
- Engage patients at a moment of heightened awareness and motivation

If we begin to treat menopause as a cardiovascular transition, not just a hormonal one, we can fundamentally alter the trajectory of disease in women.

Menopause is not a symptom problem.

It is a **cardiovascular event in slow motion**.

And if we continue to treat it otherwise, we will continue to miss one of the most powerful prevention opportunities in all of medicine. Menopause is not the time to wait.

It is the time to look more closely.
