Cardiovascular risk: Why men die earlier, women live sicker

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Cardiovascular disease remains the leading cause of morbidity and mortality globally, but its expression and impact on lifespan diverge significantly between sexes. Understanding these differential effects is critical for tailoring preventive strategies and clinical management. The prevailing narrative often overlooks how distinct biological and social factors shape cardiovascular trajectories in men and women.

KEY TAKEAWAYS

- The Pivot: Sex-specific differences in cardiovascular risk factor accumulation and their impact on longevity are more pronounced than previously acknowledged.
- The Data: Men typically experience cardiovascular events a decade earlier than women, while women live longer with a higher burden of chronic cardiovascular conditions.
- The Action: Clinicians should adopt sex-specific approaches to risk assessment and intervention, particularly regarding hypertension, diabetes, and dyslipidemia, to address these divergent outcomes.

Cardiovascular disease (CVD) represents a complex interplay of genetic predispositions, lifestyle choices, and environmental exposures. While the major risk factors, including hypertension, dyslipidemia, diabetes mellitus, obesity, and smoking, are well-established, their cumulative effect on longevity and quality of life varies substantially between men and women. This divergence is not merely a matter of timing but reflects fundamental differences in disease pathophysiology, presentation, and response to interventions. For general practitioners and specialists alike, recognising these distinctions is paramount for effective patient care.

Men generally develop CVD earlier in life, often experiencing acute events such as myocardial infarction or sudden cardiac death a full decade before women. This earlier onset contributes to a shorter overall lifespan for men compared to women, despite women often living with a higher burden of chronic cardiovascular conditions for a longer duration. The average age of a first myocardial infarction is approximately 65 years for men, compared to 72 years for women. This seven-year difference in presentation age has profound implications for both individual patient management and public health strategies.

The Early Onset in Men

The accelerated cardiovascular risk in men stems from a confluence of factors. Testosterone, while essential for male development, may contribute to adverse lipid profiles and increased visceral adiposity, particularly when imbalanced. Men also tend to accumulate traditional risk factors, such as smoking and unhealthy dietary patterns, earlier and at higher rates than women in many populations. The prevalence of hypertension, for instance, begins to rise earlier in men, often

in their 30s and 40s, whereas in women, it typically accelerates after menopause. This earlier exposure to elevated blood pressure contributes directly to accelerated arterial stiffening and endothelial dysfunction.

Furthermore, men often present with more severe and extensive coronary artery disease at the time of their first event. This is reflected in studies showing higher rates of multi-vessel disease in men undergoing revascularisation procedures. The typical presentation of angina in men is also more classic, involving substernal chest pain radiating to the arm or jaw, which often leads to earlier diagnosis and intervention. This contrasts with women, who frequently experience atypical symptoms, delaying diagnosis and potentially worsening outcomes.

The Chronic Burden in Women

Women, while experiencing CVD later, often live with a greater burden of chronic cardiovascular conditions, including heart failure with preserved ejection fraction (HFpEF), microvascular dysfunction, and arrhythmias. They also face a higher prevalence of autoimmune diseases, which are independent risk factors for CVD and disproportionately affect women. Conditions like systemic lupus erythematosus and rheumatoid arthritis significantly increase the risk of premature atherosclerosis and cardiac events in women, often years before traditional risk factors would predict.

Hypertension in women, particularly post-menopause, presents distinct challenges. Women tend to have a higher prevalence of isolated systolic hypertension and a greater sensitivity to salt, which can complicate management. The Omron M3 Comfort Blood Pressure Monitor, a clinically validated device, can be a useful tool for monitoring these fluctuations at home, providing valuable data for clinicians. Diabetes also exerts a more detrimental effect on cardiovascular health in women than in men. Women with type 2 diabetes have a higher relative risk of developing CVD and experience worse outcomes post-myocardial infarction compared to diabetic men. This 'sex-diabetes interaction' suggests that the metabolic milieu in women may be particularly vulnerable to the pro-atherogenic effects of hyperglycemia.

Disparate Impact of Specific Risk Factors

The impact of individual risk factors also varies by sex. Smoking, for example, confers a disproportionately higher risk of myocardial infarction in women than in men, even at lower exposure levels. Women who smoke have a 25% higher risk of coronary heart disease compared to men who smoke the same number of cigarettes. This heightened susceptibility underscores the critical importance of smoking cessation efforts tailored to women.

Dyslipidemia also shows sex-specific patterns. While elevated LDL-C is a universal risk factor, women often present with higher HDL-C levels, which historically offered some protective effect. However, this protection diminishes with age and the onset of menopause. Triglyceride levels also appear to have a stronger predictive value for CVD in women than in men, particularly in the context of metabolic syndrome. The Oxford Handbook of Cardiology provides a concise overview of these and other complex risk factor interactions.

Obesity, especially central adiposity, is a significant risk factor for both sexes, but its metabolic consequences can differ. Obese women are at a higher risk for developing diabetes and hypertension, which then synergistically increase their CVD risk. Men with central obesity, while also at high risk, may exhibit different patterns of insulin resistance and dyslipidemia. These distinctions highlight the need for sex-specific guidelines in obesity management and cardiovascular prevention.

The Role of Hormones and Genetics

Endogenous sex hormones play a crucial role in these observed differences. Estrogen, particularly estradiol, has cardioprotective effects, including favourable impacts on lipid profiles, endothelial function, and vascular tone. The decline in estrogen levels during menopause contributes significantly to the acceleration of CVD risk in women, leading to increased rates of hypertension, dyslipidemia, and metabolic syndrome. This hormonal shift explains why the incidence of CVD in women begins to approach that of men approximately 10 years after menopause.

Genetic factors also contribute to sex-specific CVD risk. While many genetic variants associated with CVD are common to both sexes, some exhibit sex-specific penetrance or expression. For instance, certain genetic polymorphisms related to lipid metabolism or inflammatory pathways may confer different levels of risk in men versus women, interacting with hormonal environments to modulate disease susceptibility. Research continues to unravel these complex gene-environment-hormone interactions.

Clinical Implications and Unanswered Questions

These sex-specific differences necessitate a re-evaluation of current risk assessment tools and treatment algorithms. Many existing risk calculators, such as the Framingham Risk Score or the SCORE system, were developed predominantly using male cohorts or did not adequately account for sex-specific risk factor weighting. This can lead to underestimation of risk in women, particularly younger women, and potentially delayed initiation of preventive therapies. Clinicians must consider these nuances when evaluating individual patients.

The open-label design of many observational studies on sex differences is an obvious caveat. While large cohort studies provide robust epidemiological data, they cannot establish causality. Furthermore, confounding factors, such as socioeconomic status, access to healthcare, and health-seeking behaviours, often correlate with sex and can influence observed outcomes. Disentangling these variables requires sophisticated statistical modelling and, ideally, prospective interventional trials designed with sex as a primary stratification factor.

The optimal timing and intensity of primary prevention strategies also remain an area of ongoing debate. Should men, given their earlier onset of disease, receive more aggressive lipid-lowering or blood pressure management earlier in life? Should women, particularly around the perimenopausal transition, be targeted with more intensive screening for emerging cardiovascular risk factors? These questions underscore the need for further research into sex-specific guidelines and personalised medicine approaches.

> **CLINICAL IMPLICATIONS**
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> The persistent gap in cardiovascular outcomes between men and women demands a more nuanced clinical approach. Relying on a one-size-fits-all model for risk assessment and prevention is clearly insufficient, given the distinct biological and social trajectories of cardiovascular disease in each sex. We must move beyond simply acknowledging differences to actively integrating them into our diagnostic and therapeutic algorithms. For instance, the earlier and more aggressive accumulation of risk factors in men suggests that primary prevention efforts, including lifestyle modifications and pharmacotherapy for hypertension or dyslipidemia, should commence earlier and be more rigorously pursued in male patients. Conversely, the later onset but higher burden of chronic conditions in women, particularly post-menopause, calls for heightened vigilance for conditions like HFpEF and microvascular dysfunction, which are often underdiagnosed in this population. Guideline bodies, such as the European Society of Cardiology, need to refine their recommendations to

> explicitly address these sex-specific considerations. This includes developing risk calculators that more accurately predict individual risk for both men and women, and providing clearer guidance on when to initiate specific therapies based on sex and age. Without such tailored approaches, we will continue to see avoidable disparities in cardiovascular health outcomes.

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