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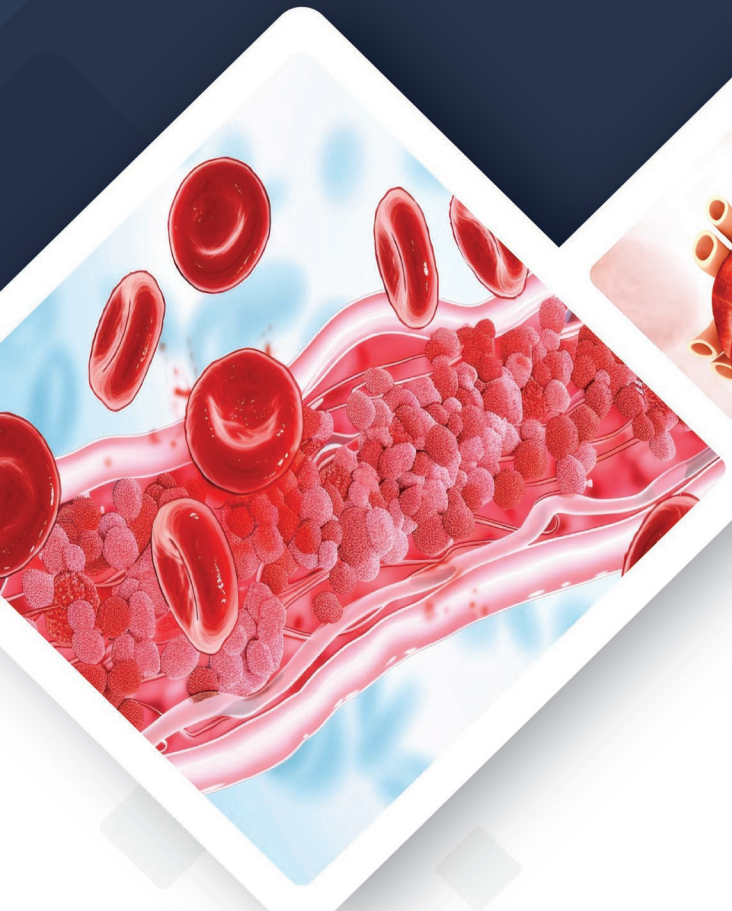





Women In Cardiology And Related Sciences

Inspire, enlighten, excel

Women and Heart Diseases

Editors
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Foreword
Alaide Chieffo

Preface
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CHAPTER 1

Nontraditional Risk Factors for Coronary Artery Disease in Women

Shibba Takkar Chhabra, Prerna Goyal, Shubham Garg, Ankita Goyal

ABSTRACT

Cardiovascular (CV) preventive health care has come a long way after decades of research in knowing the various pathophysiological risk factors and subsequent incorporation into different risk scoring calculators, but sex-specific CV risk stratification has remained underrecognized for a long time. As cardiovascular diseases (CVDs) still remain the leading cause of mortality and morbidity in women, a large body of scientific data has unveiled sex-specific risk factors and sexual dimorphism in response to well-known common risk factors. These risk factors can be divided into traditional and nontraditional risk factors, which can further be grouped into women-specific and women-predominant risk factors. Apart from this, there are some novel and emerging risk factors that can close the existing treatment gaps in CVDs in the women's population. The present review will delve into nontraditional CV risk factors in details pertinent to the women population and also provide a comprehensive overview of various risk scoring models. The knowledge of these risk factors is critical in fostering preventive and therapeutic strategic plans and bringing gender equity in health care.

INTRODUCTION

For decades, global studies have implicated cardiovascular diseases (CVDs), particularly coronary artery disease (CAD), as the leading cause of morbidity and mortality for both men and women. In parallel with the rise in obesity and diabetes, the prevalence of CVDs among Indians has reached epidemic proportions. The reported annual death rate from CAD is quite high in women and is responsible for one in three deaths in women, regardless of color or ethnicity. The global INTERHEART research, involving more than 52,000 individuals suffering from myocardial infarction (MI), has reported that women typically present with CAD a decade later than men, primarily in the postmenopausal age group.[1] However, women's outcomes and survival are poorer than men. The gender disparities in CVD outcomes are primarily caused by poor adherence to guideline-directed therapies, limited access to health care, delayed or atypical clinical presentation, and a slow treatment approach. Apart from this, a few pathological entities, such as MI with no obstructive CAD (MINOCA), ischemia with nonobstructive CAD (INOCA), spontaneous coronary artery dissection (SCAD), heart failure with preserved ejection fraction, and Takotsubo syndrome, are specifically seen in the female population. Understanding the key gender-specific differences in various risk factors, anatomy, and pathophysiology of CAD is crucial for directing appropriate preventive strategies at an early stage. The manuscript will delve into women-specific CV risk factors in detail, focusing especially on nontraditional or emerging factors that are unique to the women's population because of their distinctive biological and reproductive traits.[2] Numerous pathways involving disruptions in the autonomic, hypothalamic–pituitary, and inflammatory balances have shown these factors to culminate in a prothrombotic or proatherogenic state. The recent 2019 American College of Cardiology/American Heart Association (ACC/AHA) guidelines and 2020 ACC review on primary prevention of CVD in women have included these emerging risk enhancers, offering an opportunity for early recognition and initiation of preventive measures to reduce the burden of CVD in our communities.

The well-known established risk factors that are common to both genders include age, obesity, family history, dyslipidemia, metabolic syndrome, diabetes, and hypertension **(Fig. 1)**.

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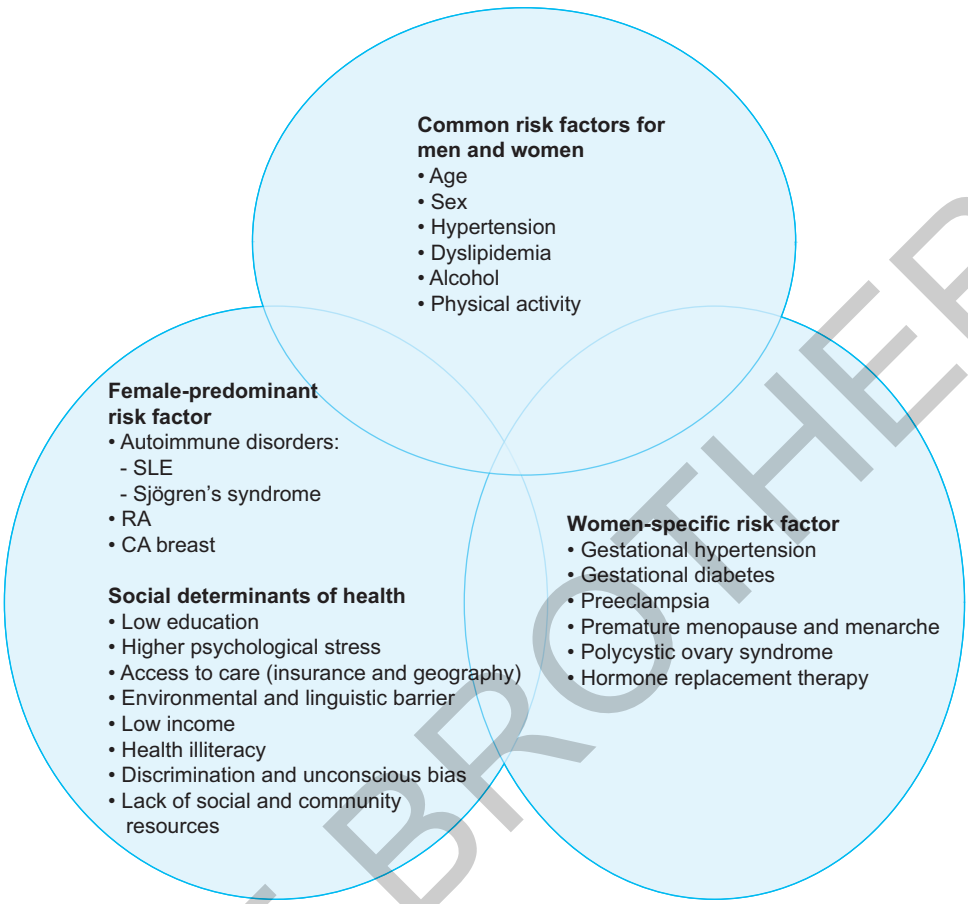

Fig. 1: Venn diagram of common female-predominant and women-specific risk factors for CAD.
(CAD: coronary artery disease; CA breast: breast cancer; RA: rheumatoid arthritis; SLE: systemic lupus erythematosus)

WOMEN-SPECIFIC CARDIOVASCULAR RISK FACTORS

- *Pregnancy-associated disorders*: A woman in her gestation witnesses enormous changes in hemodynamics, glucose metabolism, and insulin sensitivity, which provide a potential window of opportunity for healthcare providers to identify high-risk mothers who may develop CVDs later in life. In addition, a few of these disorders are proposed to cause in utero programming of the fetus via epigenetic modifications and increase the risk of CVDs and metabolic abnormalities in children at an early age. A number of pregnancy-related disorders have been recognized as CV risk enhancers by imposing a state of chronic inflammation, endothelium dysfunction, and early subclinical atherosclerosis, as discussed below:[3]
  - *Hypertensive disorders of pregnancy (HDPs)*: One of the most prevalent and troublesome pregnancy-associated problems is hypertensive disorders that include preeclampsia, eclampsia, and gestational hypertension. The condition associated with the highest risk of ischemic heart disease (IHD) is preeclampsia, which affects 3–5% of pregnancies. There is a 2.6-fold increased risk of CVDs and a fourfold increased risk of heart failure in preeclamptic mothers. HDPs have been associated with the future development of chronic systemic hypertension, diabetes, and metabolic syndrome in various meta-analyses, even after a decade of preeclamptic pregnancy.[4] The likelihood of recurrent CV events is elevated by the severity, parity, and recurrence of hypertensive pregnancy illnesses. The underlying pathophysiological mechanisms include placental ischemia leading to endothelial and vascular disruptions and subsequent hypertension and increased CV risk.[3]

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- *Gestational diabetes mellitus (GDM)*: GDM is characterized by hyperglycemia and is secondary to the physiological development of insulin resistance from the second trimester and onward. The high-risk groups include obese females, advanced age at first pregnancy, stress, a sedentary lifestyle, and poor dietary habits characterized by a high-calorie, highly processed diet. Future risk of diabetes mellitus and CVD is increased eight- and 2.3-fold in GDM mothers as early as one decade postdelivery. Research has demonstrated alterations in atherosclerosis markers in GDM mothers, e.g., a decrease in apelin, a marker of endothelial dysfunction, and pentraxin 3 (PTX3), a cardioprotective marker, whereas osteoprotegerin (OPG), a soluble member of the superfamily of tumor necrosis factor (TNF) receptors, is found to be elevated in GDM. Not only GDM, blood glucose levels even below that of glycemic thresholds of GDM can enhance the future risk of obesity, diabetes, and dyslipidemia, all of which can culminate in increased CV risk.[3,5]
- *Assisted reproductive technology (ART)*: Blood pressure and peripheral vascular resistance first rise and then fall as a result of changes in the hormonal milieu experienced by ART patients. Also, there is a change in the thrombotic–thrombolytic equilibrium, favoring coagulation, activation of the renin–angiotensin–aldosterone (RAA) system, and vascular damage secondary to ovarian hyperstimulation. Additionally, pregnancies produced by ART show a greater rate of adverse events for both the mother and fetus. These events include an increased risk of hypertensive disorders and an increase in thromboembolic events early in the first trimester. Nevertheless, even failure of these techniques can substantially increase CV risk in the first year follow-up postfailure.[6]
- *Preterm delivery*: Delivery occurring before 37 weeks is defined as preterm delivery. Preterm delivery has been associated with future risk of diabetes, dyslipidemia, and chronic hypertension and, therefore, enhanced CV risk in various meta-analyses. CV risk has been reported to increase 1.4-fold in preterm births, more in very premature (<32 weeks of gestation) deliveries. The risk increases in direct proportion to the number of preterm births. Not only this, CVD risk is also increased in mothers giving birth to small for gestational age babies.[3] A higher degree of maternal weight gain and persistence of maternal obesity postpartum are other key contributors to pregnancy-related enhanced CV risk.
- *Polycystic ovary syndrome (PCOS)*: It is an endocrine disorder characterized by hyperandrogenemia, ovulatory dysfunction, and insulin resistance. These women are at risk of developing diabetes and other CV predisposing conditions such as dyslipidemia, metabolic syndrome, and hypertension.[7] Evidence of premature atherosclerosis and increased coronary artery calcium has been reported in PCOS women compared to women with normal ovulatory cycles.
- *Age at menarche and menopause*: Menarche and menopause are two major milestones in a woman's life cycle that can govern CV risk by modifying both the biological and physiological balances. Both early-onset menarche (<10 years of age) and late-onset menarche (>15 years) have been identified as CV risk enhancers. Obesity, behavioral factors, or lifestyle could be playing a mechanistic role for menarche-related CV events. The role of estradiol (E2) in providing cardioprotection in premenopausal women is of paramount importance. E2 has anti-inflammatory, antioxidant, antiapoptotic, lipid-friendly, and antifibrotic properties, thereby maintaining vasodilatation and endothelial stability in a healthy premenopausal state. Following menopause, lower E2 levels are linked to hyperinflammation, vascular dysfunction, an increase in atherogenic lipoproteins, and activation of the sympathetic nervous system and RAA system. In addition, menopausal age is marked by the development of comorbidities such as diabetes, hypertension, and obesity. Therefore, CV risk is increased remarkably in postmenopausal women. Women who develop early menopause (<40 years), no matter whether spontaneous or surgically induced, have higher odds of developing CV events later on. Bilateral salpingo-oophorectomy (BSO) is a condition that induces surgical menopause and, when performed in a female <45 years of age, can also increase her CV risk, as reported in the Nurses' Health Study. In another cohort, even hysterectomy without BSO in women <35 years of age conferred an increased risk of CAD and heart failure, along with hypertension, obesity, and dyslipidemia.[8]
- *Hormone replacement therapy (HRT)*: Contrary to the above speculation, E2 replacement therapy in

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postmenopausal women given for cardioprotection has shown disappointment with mixed findings and has been discouraged due to thrombosis-enhancing adverse effects. The benefits of HRT are primarily determined by the timing of the initiation of therapy. The current recommendations for HRT are control of vasomotor symptoms in postmenopausal females <60 years of age or <10 years postmenopause or early-onset menopause. But HRT in late menopause should be avoided and should be given in a shared decision between clinician and patient. Apart from this, the formulation of HRT, whether oral or transdermal, and their estrogenic or progestin proportions can have varying impacts on CV outcomes. Presently, HRT for primary prevention of CVDs is not recommended. Furthermore, transgender hormone therapy (THT) in transgender cohorts has shown varying results that need redressal and more scientific evidence before initiation in this specific group of the population. Transgender persons acquire a unique CV risk profile. Transwomen receiving estrogenic therapy witnessed greater thrombotic events and strokes, whereas transmen receiving testosterone therapy had greater rates of MI, hypertension, weight gain, and lipid alterations.

WOMEN-PREDOMINANT RISK FACTORS (TABLE 1)

- *Autoimmune disorders*: Women are more likely to experience autoimmune disorders than men, carrying >80% of the burden of chronic autoimmune illnesses globally. The highest sex predilection is shown in systemic lupus erythematosus (SLE) and Sjögren's syndrome, where the female-to-male ratio ranged from 7:1 to 10:1, whereas the corresponding figures are 2:1 and 3:1 in rheumatoid arthritis, multiple sclerosis, and psoriatic disorders. In particular, women's endocrinologic transition from puberty to pregnancy and menopause and its impact on the immune system plays an instrumental role in increasing the susceptibility to autoimmune diseases.[9] This is pertinent as young women who suffer from autoimmune illnesses are especially vulnerable to develop CVDs, especially CAD, pericardial diseases, arrhythmias, and valvular heart disease. Compared to healthy controls, young women with SLE were found to have a 50 times higher relative risk of MI compared to their age-matched counterparts. In another study, the risk of MI was up to four times higher in postmenopausal women with SLE. Similarly, studies on RA patients have shown that women are more likely to develop left ventricular hypertrophy than men. The increased CV risk has been attributed to immune dysregulation and systemic inflammation-mediated endothelial and microvascular dysfunction.[8] Besides this, usage of corticosteroids for the management of autoimmune conditions may expedite the onset of metabolic dysfunction, obesity, and atherosclerosis at an early age, which may enhance the risk of CVDs in women with autoimmune diseases.
- *Cancer and anticancer therapy-related cardiotoxicity*: CVD and breast cancer share numerous risk factors, including obesity and smoking. Anticancer therapies are lifesaving, but their propensity to cause cardiotoxicity and CV events during therapy or in postcancer survivors has now been addressed by the International Cardio-Oncology Society and European Society of Cardiology. Anthracycline-based therapies and tyrosine kinase inhibitors have been associated with cardiomyopathy, left ventricular dysfunction, and hypertension subsequent to alterations in cardiac structure and functions. Other chemotherapeutic agents that can cause cardiac morbidity are alkylating agents, antimicrotubule agents, and monoclonal antibodies. Radiation therapy to the chest wall and mediastinum for malignancies is another important cause of anticancer-related cardiotoxicity. Therefore, baseline CV assessment in cancer patients before initiating anticancer treatment and periodically thereafter is critical to mitigate both short- and long-term cardiac effects. Advances in early detection and breast cancer treatment have resulted in a growing population of survivors, many of whom face a heightened risk of long-term cardiac complications due to their treatments. In older women, the mortality risk from CVD often surpasses that of breast cancer itself.[10]

TABLE 1: Emerging risk factors.

Environmental factors	*Social factors*	*Novel factors*
• Air pollution • Heavy metal exposure	• Discrimination • Racism	Gut microbiome and associated intestinal dysbiosis

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- *Environmental factors*: Environmental factors such as air pollution, long-term arsenic exposure, and cadmium and lead exposure have been linked to CVDs that includes congenital heart disease, cerebrovascular accident, and peripheral artery disease. Asian communities have higher exposure to air pollution compared with non-Hispanic White communities in the United States. Air pollution exposure is linked with carotid intima-media thickness that is a predictor of CVD. Higher exposure to lead among non-Hispanic Black women in the United States is of major concern because blood lead levels increase during periods of increased bone metabolism such as pregnancy and menopause and have been related to preeclampsia and increased hypertension risk.[11]
- *Social factors*: Women who perceive discrimination and racism are at an increased risk of CVDs due to upregulation of the sympathetic nervous system and hypothalamic axis and inflammatory dysregulation, which cause CV health disparities among women of underrepresented races and ethnicities. Non-Hispanic Black women living in socially disabled communities are more likely to have hypertension. Researchers suggest that the cumulative impact of racism and sexism contributes to a higher allostatic load, which measures the physiological effects of stress on the body. This also leads to early decline in health experienced by non-Hispanic Black women facing chronic stress, danger, and social marginalization.[11] Having an intimate partner is associated with reduced risk of incident CHD and a better prognosis among patients, but strain (e.g., conflict) and disruption (i.e., separation, divorce) in these relationships are associated with increased risk and poor outcomes.[12]

  Socioeconomic status (SES) has a measurable and considerable influence on CV health. Women from lower SES backgrounds, particularly those in poverty, are more likely to be affected by inequities in education, income, and access to healthcare services. These disparities contribute to a heightened risk of ischemic heart disease and higher rehospitalization rates due to CVDs when compared to women in higher SES groups.[13]
- *Novel risk factors*: Gut microbiome and associated intestinal dysbiosis are the upcoming novel markers for CVDs. Gender differences in gut microbiota are a potential area of active research that can disclose novel therapeutics in CV primary prevention. A study had shown a correlation between diversity in gut microbiota and arterial stiffness in the women population. One such newly discovered marker is marinobufagenin (MBG) that has been found to be elevated in patients with unstable hemodynamics and hypertension.
- *Psychological factors*: Psychological risk factors such as anxiety, work and marital stress, depression, low SES, and loneliness are associated with CVD.[14]

CARDIOVASCULAR RISK SCORES APPLICABLE TO WOMEN

Framingham Risk Score

This basic algorithm for predicting coronary heart disease (CHD) takes into account variables such as age, sex, blood pressure (BP), high-density lipoprotein cholesterol (HDL-C) level, total cholesterol, and diabetes. Despite being a valuable resource for risk assessment, Framingham risk score (FRS) has many drawbacks, such as its inability to forecast the onset of other vascular illnesses other than CHD. FRS is an unreliable tool for young patients because young patients were underrepresented in the FHS cohort and experienced few events in the research. Diabetes, which is regarded as an equivalent to coronary disease, is not given the importance it deserves in FHS because the tool was not designed with the diabetes population in mind. Although it was found to be a useful risk assessment tool in White males and females, it did not meet the expectations of Japanese American men and women in the subsequent studies. Another possible drawback is that FHS neglected to consider a family history of CAD as a risk factor.[9]

Reynolds Risk Score

Data from around 25,000 Americans were used to generate the Reynolds risk score. An increasing number of additional inflammatory markers were added to prediction algorithms as our understanding of the underlying biological processes improved; the most well validated of them was the Reynolds risk score. The risk assessment score—possibly the only one—was examined in two distinct cohorts of men and women. It took into account family history and high-sensitivity C-reactive protein (hsCRP), but it did not include diabetes because it views diabetes as a high-risk factor for CVD. Due to its function as an acute phase reactant, hsCRP can become increased in insignificant circumstances, rendering it a less accurate diagnostic in low- and middle-income nations with a high

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rate of subclinical infections. However, the Reynolds risk score can be a useful tool for women, particularly if they have atherosclerosis, a chronic inflammatory condition.[15]

Differences in risk factors for CVD events between men and women have been recognized and are often incorporated into risk prediction tools, which are typically developed separately for each sex. Additionally, several female-specific reproductive and pregnancy-related factors have been linked to either increased or decreased CV risk. However, incorporating these female-specific factors into models with already established CV risk factors has shown minimal or no significant improvement in predicting CV events. Previous studies examining the contribution of these factors have primarily focused on women aged 40 years and above. For pregnancy-related factors in particular, it may be essential to explore their added value beyond established risk factors in younger women.[16]

NEW RISK ELEMENTS

Numerous studies, including the Rotterdam study group, the Multi-Ethnic Study of Atherosclerosis (MESA), the German Heinz Nixdorf Recall (HNR) (risk factors, evaluation of coronary calcium, lifestyle), and the CARDIA studies, demonstrated a strong correlation between the presence (extent of atherosclerotic plaque burden) of CAD by angiogram and the coronary artery calcium (CAC) measured by CT scan without contrast. However, the degree of luminal narrowing was not correlated with CAC. People with diabetes, smokers, and a wide range of asymptomatic groups of all ages demonstrated this effect. Research conducted as part of the Women's Health Initiative focused exclusively on postmenopausal women. Despite recent research raising doubts about the usefulness of calcium score as a CAD risk predictor, the MESA trial found that calcium score was included in the CAD risk model to predict CAD risk at 10 years. Nevertheless, the stroke and all types of atherosclerotic cardiovascular disease (ASCVD) are not taken into account in this risk score. The rate and degree of the progression are not significant risk factors for the computation of CAD risk.[17]

Breast Artery Calcification

Women with breast artery calcification (BAC) have nonclinically significant higher HDL-C, a higher prevalence of prediabetes and diabetes, higher systolic BP, and a higher prevalence of hypertension. Over the past several decades, there has been growing interest in BAC, a type of medial calcification that can be seen incidentally on mammography and may correlate with coronary calcification. Identification of CV risk by demonstrating an association between BAC and CAD may enable an instrumental, sex-specific methodology to identify women at increased risk from CAD. A meta-analysis by Abi Rafe et al. of five studies, including nearly 1,000 women, demonstrated that patients with BAC had a 1.59-fold increased risk of CAD by angiography. There is a 3.5-fold increased risk of CAD in women with BAC compared with those without. The general utility of coronary artery calcium score (CACS) has been risk prognostication by directly visualizing CAC as a marker of subclinical heart disease.[18]

Epicardial Adipose Tissue

Epicardial adipose tissue (EAT) is the fat depot located between the myocardium and the visceral pericardium. Rather than being passive tissue, EAT secretes a variety of bioactive molecules known as adipokines, which exert significant exocrine and paracrine effects. Surrounding the coronary arteries, EAT engages in a complex, bidirectional interaction with the vascular wall. Under normal conditions, this interaction supports the homeostasis of the vascular wall. However, when pericoronary adipose tissue (PCAT) becomes dysfunctional, it releases harmful proinflammatory adipokines that contribute to atherogenesis. Inflammation of the PCAT has been linked to CAD and major CV events. Cardiac computed tomography (CCT) allows for the visualization and precise quantification of EAT while also assessing both coronary arteries and PCAT **(Fig. 2)**. This noninvasive imaging tool can be utilized to monitor patient responses to new anti-inflammatory therapies aimed at treating CAD.[19]

CURRENT CHALLENGES

The knowledge gaps in the optimal calculation of CV risk in women population and timely start of appropriate preventive strategies are still under preliminary phase. Answers to many of questions are still mystical, e.g., whether its beneficial to start antiplatelets and statins in women with rheumatological diseases or risk scoring for CV risk stratification in women with adverse pregnancy outcomes.

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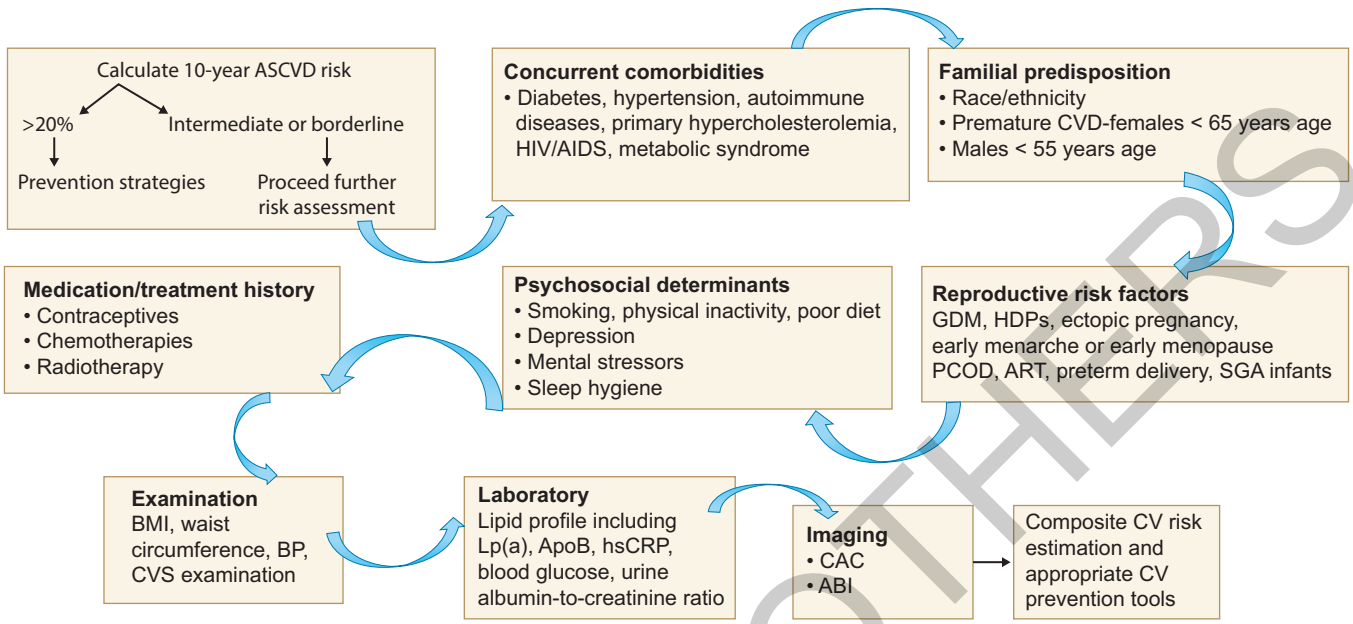

Fig. 2: Stepwise approach to cardiovascular risk assessment in women.
(ABI: ankle–brachial index; AIDS: acquired immunodeficiency syndrome; ApoB: apolipoprotein B; ART: assisted reproductive technology; ASCVD: atherosclerotic cardiovascular disease; BMI: body mass index; BP: blood pressure; CAC: coronary artery calcium; CVD: cardiovascular disease; CVS: cardiovascular system; GDM: gestational diabetes mellitus; HDPs: hypertensive disorders of pregnancy; HIV: human immunodeficiency virus; hsCRP: high-sensitivity C-reactive protein; PCOD: polycystic ovary disease; SGA: small for gestational age)

CONCLUSION

Though our primary prevention tools for CVD are elaborate and extensive, they still lack important gender-specific nontraditional CV risk factors that are unique to the female population. A female, as a separate biological identity, inherits a different hormonal, anatomical, and hemodynamic milieu, which can modify her CV risk differently from a male. The incorporation of these risk factors in existing screening risk calculators can lower the CVD prevalence, and associated disability in various global communities needs further validation. Therefore, it is imperative that a multidisciplinary task force, including experts from diverse fields of cardiology, obstetrics, pediatrics, rheumatology, endocrinology, oncology, and primary care, can partner together in designing a concrete evidence-based screening model specifically for women that can acknowledge these undervalued and pertinent risk factors.

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Women and Heart Diseases

Women and Heart Diseases is a comprehensive academic resource dedicated to understanding cardiovascular disease (CVD) in women. With CVD being the leading cause of mortality among women globally, this book highlights the unique risk factors that women face, including pregnancy-related conditions, autoimmune disorders, and social determinants of health.

The book, comprising 21 chapters, covers non-traditional risk factors, disparities in treatment access, and the intersection between CVD and other conditions like breast cancer. It also delves into critical areas such as coronary artery disease, spontaneous coronary artery dissection, and heart failure. Women and Heart Diseases serves as an essential reference for physicians and researchers, advocating for better prevention, management, and equitable healthcare for women. The initiative is supported by WINCARS, a pioneering association aimed at improving cardiovascular health outcomes for women in India and beyond.

Sarita Rao is a leading Interventional Cardiologist and the Director of the Cath Lab at Apollo Hospitals, Indore, Madhya Pradesh, India, and President of WINCARS Association. With over 23 years of experience, she has performed more than 30,000 cardiac procedures, pioneering advanced interventions in India. She was the first female cardiologist in the country, and the central region to perform the various complex interventions. She is a mentor, researcher, and advocate for women in cardiology. Dr Rao is the Co-Chair, EAPCI International Affairs, Fellowship Director of WIN-APSIC and has received numerous prestigious awards, and published numerous publications in international and national journals.

Jyotsna Maddury is a distinguished Interventional Cardiologist, retired as Head and Professor of Cardiology at Nizam's Institute of Medical Sciences, Hyderabad, Telangana, India, and the founder of the WINCARS Association. She has passion for women's heart health and made remarkable contributions to research in cardiovascular diseases, especially in stem cell therapy in AMI. Dr Maddury has published more than 200 original research papers and reviews in national and international journals and serving on the editorial boards of multiple medical journals. She trained many general and interventional cardiologists as guide and teacher. Her numerous accolades include the Women Leadership Award and the International Scientist Award, etc.

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