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CARDIOVASCULAR CHANGES DURING MENOPAUSE TRANSITION

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ABSTRACT

Beginning with the commencement of irregular menstruation or missing periods and ending 12 months after the last menstrual period, this phase marks the shift from active reproduction to the cessation of considerable estrogen release due to the depletion of functioning ovarian follicles. Menopause is connected with alterations typical of cardiovascular aging. Cardiac disease has a wide range of impacts, including alterations in endothelial function, coronary artery physiology, and metabolic dysfunction, all of which lead to structural abnormalities in coronary morphology. Atherosclerosis is the leading cause of death. In earlier cross-sectional investigations, we discovered a significant incidence of metabolic cardiovascular risk factors in women before and after menopause, and we identified the menopausal transition as a key period for atherosclerosis acceleration. In the current longitudinal study, we evaluated changes in the primary cardiovascular risk variables in women after the transition to menopause.

INTRODUCTION

Menopause, which usually occurs between the ages of 45 and 55, is a normal biological process that signals the end of a woman's reproductive years. It is distinguished by the end of the menstrual cycle and a drop in hormone levels, especially progesterone and estrogen. The term "cardiovascular ageing" describes how the cardiovascular system ages, both structurally and functionally. These modifications may involve adjustments to the heart's operation, blood arteries, and general cardiovascular health. For women, the menopause is a crucial time in life that is marked by a number of physiological and hormonal changes. An increased risk of cardiovascular conditions such as heart failure, stroke, and coronary artery disease has been connected to these alterations. To effectively prevent, identify, and treat cardiovascular problems in menopausal women, it is imperative to comprehend how the menopause affects cardiovascular health. (1)

Creating a Connection Between the trouble of CVD and Menopause.

The majority of studies that have established a connection between the risk of cardiovascular disease and menopause have used the age at which women attain the ultimate menopause (p) as a proxy for menopause. Previous research collected menopause age retrospectively since a lengthy follow-up period was required.Although, this strategy is convenient, recollection bias has been a big worry . If characterizing changes as related to time relative to the date of the final menstrual period is the aim, then this bias presents a challenge. Nonetheless, meta-analyses produced reliable findings in research aimed at proving a connection between menopausal age and cardiovascular disease. (2)

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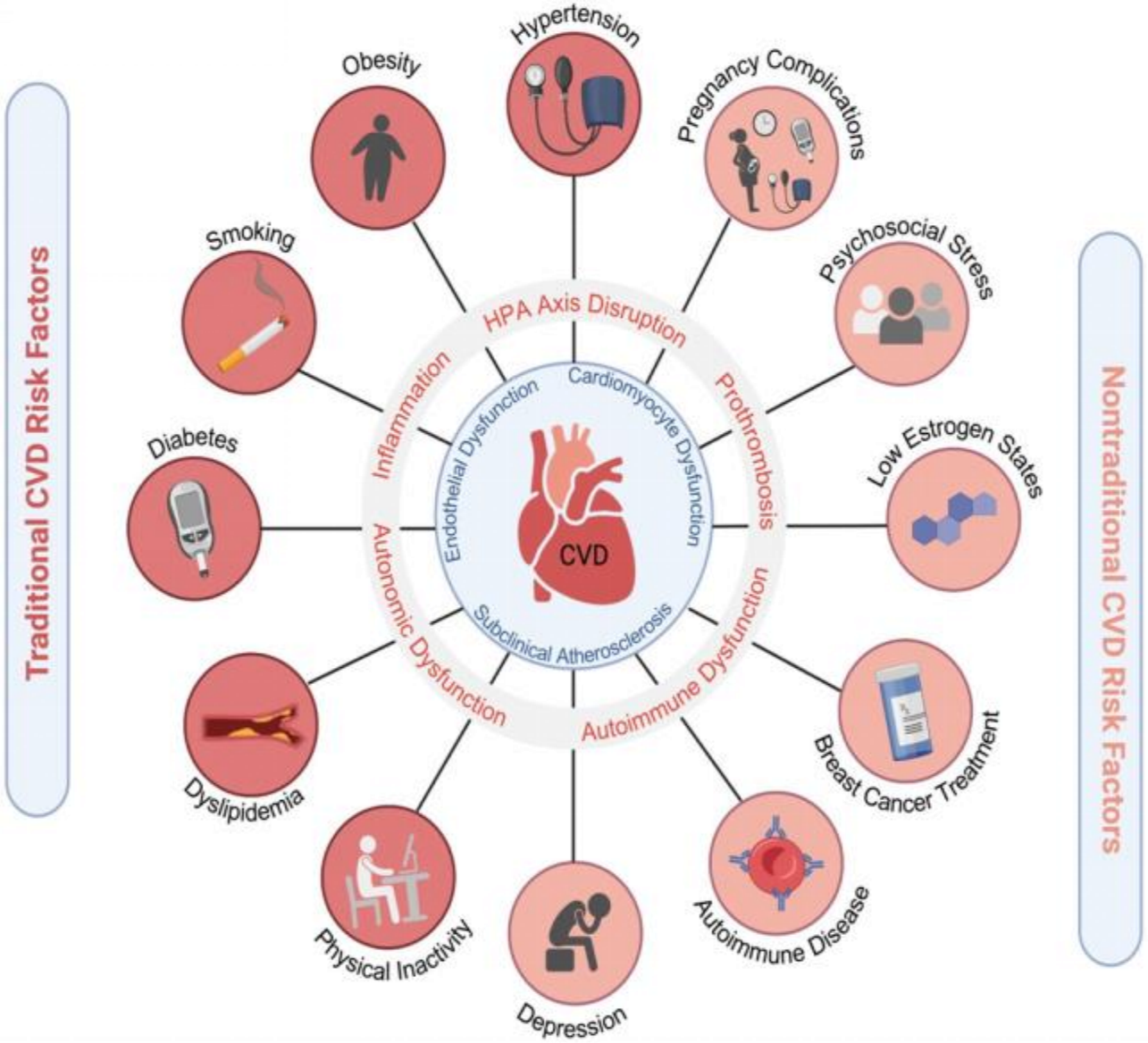


Fig.1 Cardiovascular trouble factors in women. There is a correlation between established and new trouble factors and increased atherosclerotic, thrombotic, and inflammatory stateswomen, which in turn increases the morbidity and mortality from CVD. Hypertensive diseases during gravidity, enhance diabetes, and preterm birth are samples of adverse pregnancy outcomes. Fig referred by. (3)

CORONARY ENDOTHELIAL FUNCTION AND VASCULAR AGING: is the term used to describe the adding hardening of the arteries together with a drop in the vessels' capacity to dilate. It develops in men and women in distinct ways. Unlike the progressive loss of vascular function associated with chronological aging, MP is characterized by rapid-fire vascular aging. Endothelial dysfunction and vascular aging are factors in the development of cardiovascular disease with menopause. Atherosclerosis and hypertension are two conditions that are eased by vascular aging. (4)

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CHANGES IN LIPID PROFILE: Women's lipid biographies are known to start changing during the perimeters, with increases in triglycerides (TD), LDL cholesterol, and total cholesterol (TC). A prospective disquisition of MP transition in Caucasian and non age women who weren't receiving hormone therapy was conducted as part of the disquisition of Women's Health Across the Nation (SWAN) design. It offered evidence that inimical lipid biographies and MP transition are related. It shown that, anyhow of the age at which the final menstrual period occurs, TC, LDL, and lipoprotein- B all increased in the time that followed. These are all associated with endothelial dysfunction and the development of atherosclerosis. Post-MP Carotid pillars are associated with elevated LDL levels during the peri-MP phase. These variations differ from the chronological aging 'linear differences. Over the MP transition, the line of HDL cholesterol or its suggested cardio protective effect isn't harmonious. Elevated HDL cholesterol has Its own independent cardio protective effect in youthful women. This could be as a result of the HDL patches' capability to stimulate cholesterol efflux, which is how HDL extracts cholesterol from supplemental cells. High HDL cholesterol in peri- and post-MP women may be associated with an increased threat of CVD. A measure of the health and remodeling of the highways is carotid India- media consistence, or IMT. The cIMT is higher in post-MP women with advanced HDL situations. Changes in the HDL particle quality throughout the MP transition could be the cause of this. (5)

HEART'S FAT DEPOTS: Directly covering the heart between the myocardium and visceral pericardium is called pericardia adipose tissue (EAT). EAT and PAT are now known to be novel risk factors for coronary heart disease. Pericardial adipose tissue (PAT) is situated out side the parietal pericardium and anterior to the EAT. Because these fat depots are so close to the heart, they might be more harmful than visceral fat. Compared to menopausal women, late primp/postmenopausal women had 20.7% higher PAT and 9.9%higher Eat in the SWAN cardiovascular fat ancillary trials. PAT might therefore be particular MP-specific CHD risk sign. (6)

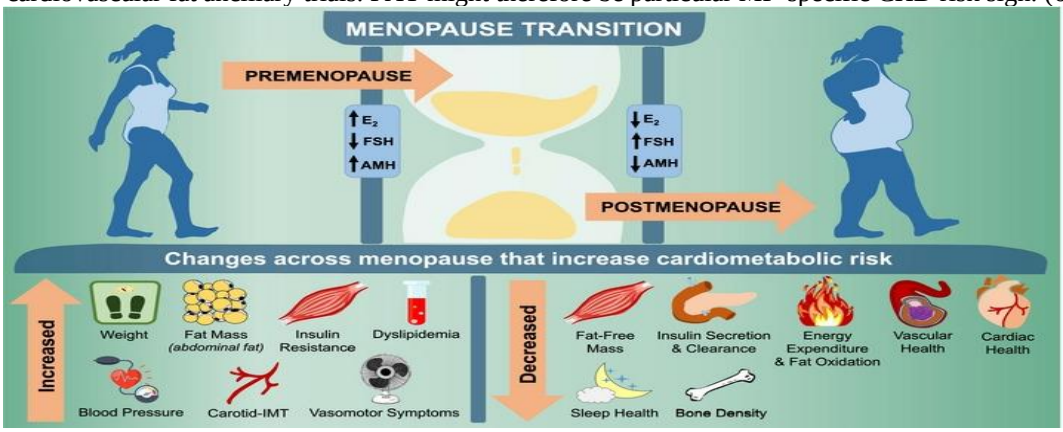


Fig.2 Correlated alterations in cardiometabolic risk with the transition to menopause. (7)

CVD SUBCLINICAL: Sub-clinical CVD may be indicated by markers such as IMT, coronary artery calcification (CAC), an indicator of atherosclerotic plaques, aortic calcification, and measurements of vascular stiffness such as aortic pulse wave velocity or flow-mediated dilation (a sign of endothelial function). These have the ability forecasts occurrences. A rise in IMT is a characteristic of late primp in women when their dyslipdemia and metabolic syndrome deteriorate. Additionally, there appears a connection between MP transition and the likely hood endothelial dysfunction. (6)

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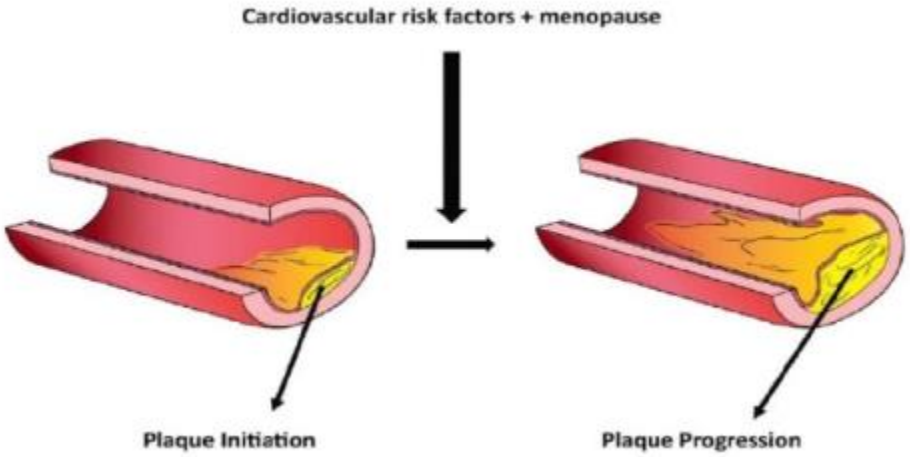


Fig. 3: Demonstrates how menopause and cardiovascular risk factors may have an effect on the development of atherosclerosis. (6)

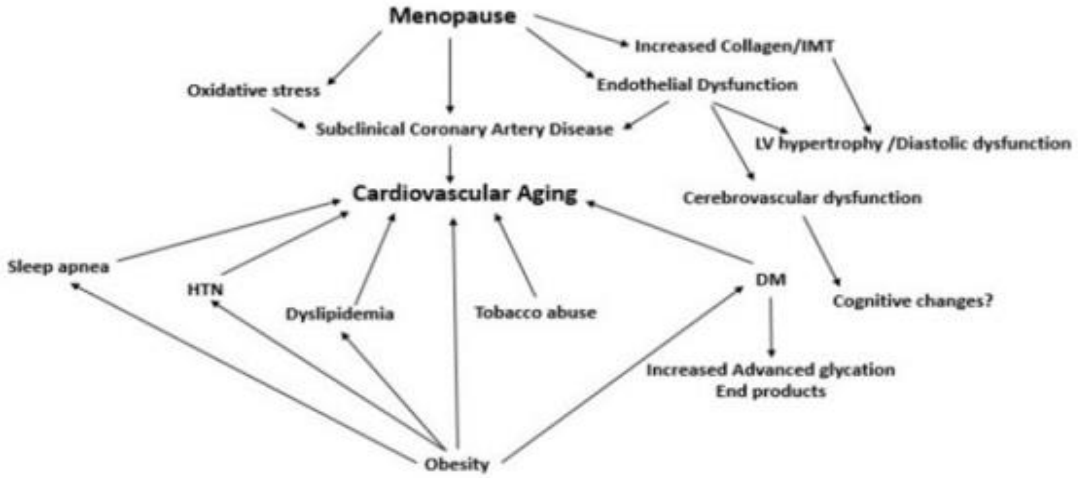


Fig.4: Demonstrates how cardiovascular risk factors are highlighted in the context of menopause. (6)

THE METABOLIC SYNDROME PREVALENCE: The co-existence of many metabolic risk factors, such as central obesity, dyslipidemia, hypertension, and impaired glucose tolerance, is known as metabolic syndrome.
The distribution and storage of fat are significantly influenced by estrogen. The accumulation of fat occurs in the thighs, buttocks, and hips prior to menopause. Because of chronologic aging, women tend to gain weight (total body fat) after midlife and beyond. However, there is a shift in the distribution of fat as well as the body composition (fat:lean body mass) in women who undergo the MP transition. (6), (8), (9).

MP AND OXIDATIVE STRESS: Aging and oxidative stress are closely related. Atherosclerosis can be caused by an excess of free radicals, such as Reactive Oxygen Species (ROS), and a decrease in antioxidant levels. This decrease, in conjunction with a progressive loss of estrogen in the female reproductive system, is strongly linked to a number of MP sequelae, including non-cardiac consequences like osteoporosis and heart disease and vasomotor abnormalities., (10), (9. Tchernof)

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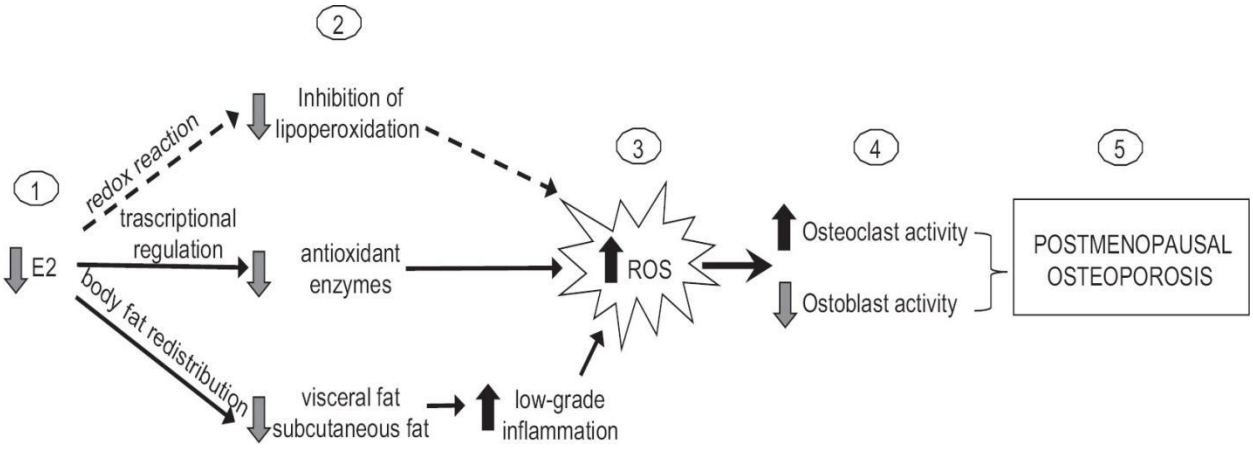


Fig.5 Oestrogen decline, oxidative stress, and postmenopausal osteoporosis: a cause-and-effect link. (11)**. Menopause-related reduction in oestrogens (E2). (1)resulting in a loss in systemic and local (bone) protection against reactive oxygen species assault.(2) This impact is attributed to the ability of 17β-oestradiol to act as a direct antioxidant and, most likely, upregulate the expression of antioxidant enzymes. It also contrasts the growth of pro-inflammatory visceral fat. Oxidative stress occurs when reactive oxygen species (ROS) levels rise uncontrollably.(3)This affects the equilibrium of bone production and resorption.(4) therefore boosting the latter activity and leading to the onset of post-menopausal osteoporosis.(5).**

The Decline of Estrogen and Cerebrovascular Illness: In the cerebrovascular system, estrogens reduce vascular tone and thereby enhance blood flow, whereas androgens increase tone. Another way that estrogens and androgens work is through increasing angiogenesis. Estrogen has the ability to lower inflammation and oxidative stress, which protects neurons by maintaining the blood-brain barrier and lowering oxidative stress. Changes in MP hormone levels may have a detrimental effect on cognition and contribute to cerebrovascular dysfunction in the presence of cardiovascular diseases. (12)

Handling Cardiac Symptoms During the Menopause Transition:- The main symptom of MP is called vasomotor symptom (VMS). reduction of the thermoneutral zone, causing mild variations in body temperature to trigger compensatory flushing and perspiration, ultimately resulting in hot flashes and nocturnal sweats. CV concerns have been connected to VMS. VMS also adds to a lower quality of life overall, irritation, difficulties concentrating, and poor sleep quality. Lifestyle adjustments, non-hormonal medicines and systemic hormone treatment may be indicated for the management of VMS. (13)

The treatment of moderate to severe VMS is the main use case for hormone therapy. The gold standard for treating VMS is hormone treatment. This could involve progesterone and estrogen therapy (EPT) for women who still retain their uterus or the use of estrogen alone (ET) for women who have had hysterectomy. Progestogens or the SERM bazedoxifene are two options for endometrial protection, which women with uteri require to prevent endometrial neoplasia. The use of systemic hormones, which can be administered orally (PO) or transdermally (T/D), is necessary for the management of VMS. Generally speaking, the shortest amount of time spent using the lowest dose of hormones required for symptom alleviation is advised.

Reduced HT dosages are linked to a decreased incidence of breast discomfort, unplanned vaginal bleeding, and venous thromboembolism (VTE). It may take 6–8 weeks for HT at lower dosages to start showing symptom alleviation. Oral conjugated equine estrogen (CEE) 0.3 mg, oral 17 beta-estradiol 0.5 mg, and estradiol patch 0.025 mg are some possible forms of estrogen. Oral progesterone, such as medroxyprogesterone acetate (MPA), may be prescribed if progestogens are necessary for the patient. (14)

Progestogens (natural or synthetic) can be used alone to treat VMS; however, they are less effective than estrogen therapy and have inadequate long-term safety data. Long-term use raises concerns about the potential of breast pathology. Progesterone formulations include oral MPA 10 mg/day, oral megestrol acetate 20mg, and micronized progesterone 300 mg nightly.

Tissue Selective Estrogen Complex (TSEC) is an FDA-approved treatment for VMS in women with a uterus that combines Bazedoxifene, a SERM (Selective Estrogen Receptor Modulator), and (CEE). It has the additional benefit of preventing osteoporosis. Bazoxifene provides endometrial protection. As a result, extra progestin is not recommended.

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The government-approved Bioidentical Hormone Therapy (BHT), which includes estradiol, estrone, and micronized progesterone formulations, is regulated for purity, safety, and efficacy. The FDA has not approved "Compounded BHT" products sold as BHT. There are special considerations here, particularly about safety. These are often manufactured by a pharmacist in a compounding pharmacy using the provider's prescription. The compounded BHT may contain a variety of hormones. As a result, the quality, efficacy, and safety of the components cannot be guaranteed. The concentration of hormones in these formulations is also unknown, as is their bioavailability. As a result, there is a risk of over- or underdosing. The dangers associated with compounded BHT are typically not disclosed.They may contain unapproved combinations of drugs and be administered by experimental means, such as hormone pellets, troches, or subdermal implants.

Compounded BHT should only be considered if patients are unable to tolerate FDA-approved hormones due to concerns such as component allergies or a lack of a dose or formulation. (15)

CONCLUSION

Menopause e is frequently a turning point in women's health around the world. Increasing evidence from experimental and clinical investigations suggests that cardio metabolic changes might occur during the menopausal transition, compounding the influence of aging on the risk of cardiovascular disease. The menopausal transition is associated with increased fat mass (mostly inter nuclear), insulin resistance, dyslipidemia, and endothelial dysfunction. Endogenous estrogen exposure throughout the reproductive years protects women from cardiovascular disease, which is lost approximately ten years following menopause. Women with vasomotor symptoms during menopause appear to have a worse cardio metabolic profile. Early management of the traditional risk factors of cardiovascular disease (i.e., hypertension, obesity, diabetes, dyslipidemia, and smoking) is essential; However, it is important to recognize in the reproductive history the female-specific conditions (i.e., gestational hypertension or diabetes, premature ovarian insufficiency, some gynecological diseases such as functional hypothalamic amenorrhea, and probably others) that could enhance the risk of cardiovascular disease. In this Review, the first of two papers, we provide an overview of the literature for understanding cardio metabolic changes and the management of women at higher risk in midlife (40-65 years), with a focus on identifying factors that can predict the occurrence of cardiovascular disease. We also present research on preventive non- hormonal measures in the context of cardio metabolic health. (6)

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