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Arterial hypertension throughout the life cycle of women: what factors influence it?

Hipertensión a través del ciclo de vida de la mujer: ¿qué factores influyen?

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INTRODUCTION

Detection and timely managing cardiovascular risk factors (CVRF) reduce cardiovascular (CV) morbidity and mortality. Systemic arterial hypertension (SAH) is the main modifiable CVRF contributing to the global cardiovascular disease (CVD) burden due to ischemic heart disease being a leading cause of disability. Women represent ~51% of the hypertensive population and are at greater risk than men for acute myocardial infarction (AMI) and stroke.[1]

During the life cycle, women experience sex-linked changes, which increase the risk of SAH. They manifest from adolescence, early adulthood, reproductive stage, and menopause until late adulthood *(Figure 1)*. The prevalence of SAH is low in women during adolescence, youth, and young adulthood (20%), it increases towards the third decade of life and in perimenopause (40%). This increase is more notable at menopause or in the fifth decade of life (75%), when it exceeds men, being even more frequent after 75 years (85%). The impact on CVD is greater in women, since an increase of 10 mmHg in SBP increases the risk of CVD by 25%, compared to 15% in men.[2,3]

ADOLESCENCE AND EARLY ADULTHOOD

Women have lower blood pressure (BP) levels than men because of the effect of female sex hormones. Estrogens regulate the renin-angiotensin-aldosterone system (RAAS), reduce the activity of the angiotensin-converting enzyme (ACE), levels of angiotensin-2, endothelin, and the expression of its A and B types of receptors; 17β-estradiol increases nitric oxide synthase and decreases angiotensin 2 production, which leads to vasodilatation and decreased BP. The maximum estrogen peak in the luteal phase of the menstrual cycle coincides with a decrease in BP and should be considered in BP measurements.[2] Polycystic ovarian syndrome (PCOS) is a common cause of SAH in young women. Metabolic alterations and SAH, are related to a state of insulin resistance and overstimulation of the RAAS. Elevated levels of testosterone and hypothalamic hypoestrogenism, decrease vasodilation and increase BP levels. The prevalence of SAH in women with PCOS is 65%.[4,5] SAH in adolescents (12-18 years) and young adults (19-39 years) can be secondary to other etiologies such as fibromuscular dysplasia, rheumatological diseases (rheumatoid arthritis, vasculitis, Takayasu, systemic lupus erythematosus) and to the use of combined oral contraceptives (COC). The use of COC, mainly those containing medroxyprogesterone acetate or ethinylestradiol), can increase BP levels and may condition SAH by 5-10%; their use is not recommended in patients with uncontrolled BP (> 140/90 mmHg). Once therapy is installed, ambulatory BP measurements at least four times per week are recommended, with follow-up visits with their physician every four to six months. If there is an increase in BP, COC should be suspended and continued

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with non-hormonal contraception (condoms, spermicide, intrauterine device (IUD). The relative risk of SAH (RR 1.5) with COC has a reversible cumulative effect, wich increases by 13% for every five years of use; in most cases, BP returns to previous values three months after cessation. The increase in BP is related to a rise in hepatic production of angiotensinogen, activation of the RAAS, an increase in vascular resistance, and a decrease in circulating nitric oxide (NO).[2,6]

REPRODUCTIVE AGE

Hypertensive disorders of pregnancy (HDP) are characterized by systolic blood pressure (SBP) ≥ 140 mmHg and diastolic blood pressure (DBP) ≥ 90 mmHg and include chronic SAH, gestational SAH, preeclampsia/eclampsia, and chronic SAH with superimposed preeclampsia/eclampsia. They have an annual incidence of 5-10% and cause complications in the mother and the child. Endothelial dysfunction may persist after delivery. Women with a history of HDP, especially preeclampsia, have a higher risk of CV events and mortality (RR 2.0) during the subsequent ten years. BP generally subsides within two weeks of delivery and resolves in 12 weeks. If hypertension persists, the diagnosis of chronic hypertension should be considered.[7,8]

Women with assisted reproductive therapy have a greater susceptibility to presenting HDP (30.6%); the probability increases with twin or multiple pregnancies (13%) vs single pregnancy (6%). Compared with spontaneous conception, they have a higher risk of HDP and preeclampsia (RR 1.7 and 1.34, respectively), so, they must be followed up throughout their lives and estimated their CV risk.[2,9,10]

MENOPAUSE

In post-menopause, women have a higher prevalence of hypertension than same-age men. In addition to the hormonal factor (decrease in estrogen), genetics and other CVRF influence the elevation of BP. Likewise, there is overstimulation of the RAAS, vasoconstriction, and increased sensitivity to salt. Androgen production conditions increased arterial stiffness, vascular inflammation, and endothelial dysfunction. All these factors are related to increasing BP levels during this stage.[11] The testosterone/estradiol ratio increase is associated with an increased incidence of CVD (RR 2.0) and cardiovascular events.[12] Women with early menopause (< 45 years), whether natural or induced, have a higher risk of SAH compared to those with menopause at an expected age (> 45 years, RR 1.20, p = 0.03).[13]

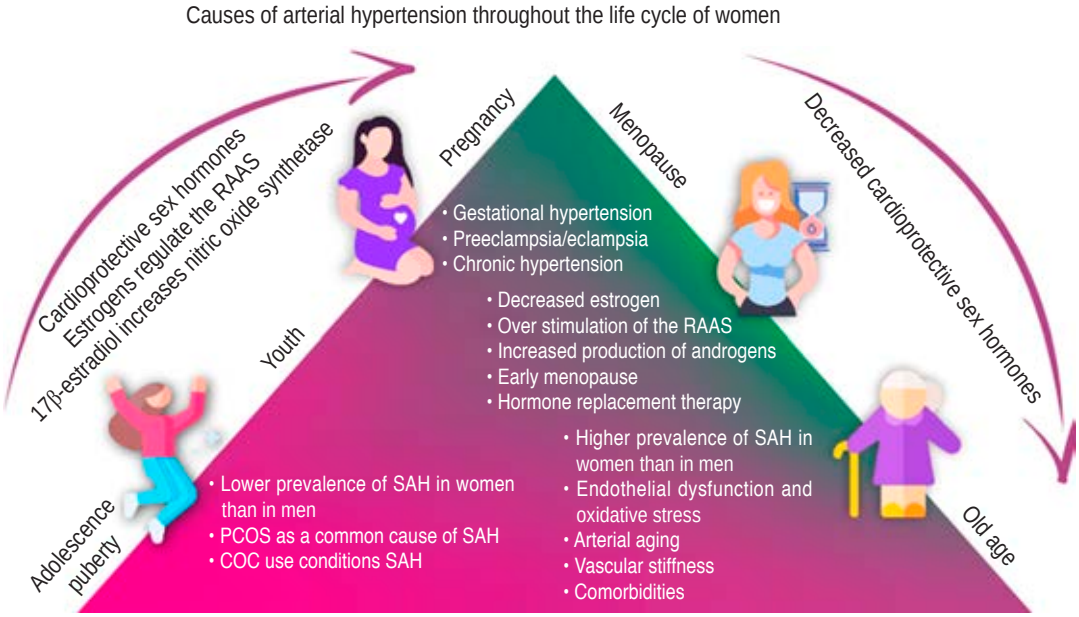

Figure 1:

Causes of arterial hypertension throughout the life cycle of women. Different conditions influence the increase in blood pressure at different stages of life. SAH = systemic arterial hypertension. PCOS = polycystic ovary syndrome. COC = conjugated oral contraceptives. RAAS = renin-angiotensin-aldosterone system.

The effect of hormone replacement therapy (HRT) on BP elevation is controversial. There is evidence reporting an increase in SBP levels with the use of combined therapies (estrogens/progestins); however, other authors refer that the use of HRT does not modify BP.[12,14]

OLDER ADULTHOOD

The prevalence of SAH after age 65 is higher in women than men, 80-85% of women > 75 years are hypertensive. The associated factors are multiple: endothelial dysfunction, oxidative stress, vascular age, arterial stiffness, and other comorbidities. BP elevation is more severe and difficult to control, so management must be strict to avoid CV complications.[2,11]

CONCLUSIONS

SAH can be present throughout the different stages of a woman's life. The increase in BP is conditioned by sex-specific risk factors and other comorbidities, which vary according to age. These must be recognized and considered for proper management and control of BP to reduce CV complications and mortality.

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