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Current Prospective of Hypertension Disease and their Future Aspects in Different Stage of Female Health

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ABSTRACT

Cardiovascular disease is the primary cause of mortality in women. Due to age and worsening of risk factors over the menopausal transition, risk of coronary heart disease events increases in postmenopausal women with diabetes. Randomized studies have conflicted regarding the beneficial impact of estrogen therapy upon intermediate cardiovascular disease markers and events. Therefore, estrogen therapy is not currently recommended for indications other than symptom management. However, for women at low risk of adverse events, estrogen therapy can be used to minimize menopausal symptoms. The risk of adverse events can be estimated using risk engines for the calculation of cardiovascular risk and breast cancer risk in conjunction with screening tools such as mammography. Use of estrogen therapy, statins, and anti-platelet agents can be guided by such calculators particularly for younger women with diabetes. Risk management remains focused upon lifestyle behaviors and achieving optimal levels of cardiovascular risk factors, including lipids, glucose, and blood pressure. Use of pharmacologic therapies to address these risk factors, particularly specific hypoglycemic agents, may provide some additional benefit for risk prevention. The minimal benefit for women with limited life expectancy and risk of complications with intensive therapy should also be considered.

Keywords- Hypertension, Blood Pressure, Female, Gestational, Pregnancy, Menopause.

I. INTRODUCTION

Hypertension is a leading modifiable risk factor for cardiovascular disease and is highly prevalent worldwide.(1) The prevalence of hypertension is known to vary by sex and age, and the impact of increased blood pressure is different for men and women. (2) For a comparable 10mmHg increase in systolic blood pressure, women experience a 25% increase in CVD risk while men risk is only 15% higher.(3) Sex-specific differences in blood pressure (BP) have been noted since the early 1900's when women were first observed to have lower BP compared to men of a similar age.(4) BP, and consequently hypertension prevalence, is lower in women from adolescence until menopause or the fifth decade of life,(5–7) following which the prevalence of hypertension increases steeply in women.(8) Despite the higher prevalence of hypertension in men, a recent study of 32,833 individuals (17,733 women [54%]) followed for over four decades, demonstrated that women actually have a steeper increase in BP as early as the third decade that continues throughout the life course.(7) These differences persisted even after adjustment for multiple cardiovascular risk factors. Taken together, these sex differences in BP across the life course may have important implications for the diagnosis and treatment of hypertension in men and women, though currently there are no sex-specific guidelines for the diagnosis or treatment of hypertension. In this review, we highlight a variety of sex-specific biological processes across a

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women's lifespan from young adulthood through menopause and older age that impact BP and hypertension (Figure 1). We will also emphasize areas where future studies are needed to further understand these differences in hypertension mechanisms and treatment, highlighting the importance of prespecified analyses by sex in clinical trials. Although beyond the scope of this review in which we focus on biological sex and hypertension, gender - a social construct – also influences blood pressure and hypertension. Gender inequities and different socialization patterns across genders may affect cardiovascular risk.(9)

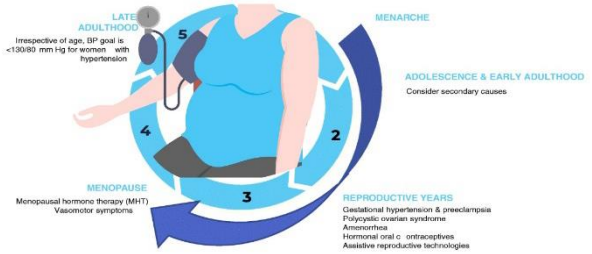

Fig: 1 Issues unique to hypertension and blood pressure in women across the lifespan.

II. APPROACH TO HYPERTENSION IN ADOLESCENTS AND YOUNG ADULTS

Among the general population, the age-standardized prevalence of HT in 2023 was reported to be 34% in men and 32% in women [14]. In Korea, data from the Korea National Health and Nutrition Examination Survey (KNHANES) reported that the age-standardized prevalence of HT among adults aged ≥ 20 years decreased from 26.0% in 1998 to 22.2% in 2019 [3]. However, with the rapid aging of the population, the absolute number of people with HT has steadily increased with more than 12 million at 2023.

The prevalence of HT among the younger generation is much lower than that in the overall population. The prevalence among young adults was estimated to be 9.5%, resulting in 1.27 million young adults being affected. The prevalence of HT in young adults differ depending on the sex. Although such disparity among sex is a common phenomenon, the pattern of difference differs compared to the older population. In middle aged population, the prevalence of HT is similar between different sexes and the HT prevalence rises higher in women compared to men when they get older. In younger generations under 40 years of age, the prevalence of HT in men is approximately 15%, but that in women is less than 5% [3]. Similarly, in the United States, according to the National Health and Nutrition Examination Survey (NHANES) 2022-23 data, the prevalence among young adults aged 18 to 39 years was 7.5%, which was subdivided into 9.2% in men and 5.6% in women [14]. In Japan, an analysis of data from the National Health and Nutrition Survey reported that the prevalence of HT ranged from 10.2% to 18.1% in young men and 4.1% to 4.5% in young women in 2023 [15].

Another remarkable finding is the high prevalence of prehypertension among young adults. According to the Korean Hypertension Face Sheet 2023, nearly one-third of young adults over 20 years of age (3,387,000 in 10,536,000) have prehypertension with systolic BP (SBP) 130 to 139 mmHg and diastolic BP (DBP) 80 to 89 mmHg [16]. If they are not adequately controlled, a considerable portion of the prehypertensive population will progress to the hypertensive range with aging.

The most important problem in the younger hypertensive population is that more than 90% of them are not aware that they have HT. The awareness rates of HT have improved little in younger adults from 10% in 1998 to 15% in 2023, which is in sharp contrast to older adults, whose awareness rates have steadily increased from 30 to 80% in the same period in Korea [3]. As a result, only 24% of young hypertensive patients seek medical care at least once and among them, only one-third are regularly treated [2]. Similarly, the US NHANES 2022-23 data reported an awareness rate of 74.7%, a treatment rate of 50%, and a control rate of 40.2% among young adults with HT, which was lower than that of hypertensive patients in the all-age group (84.4%, 74.7%, and 53. 9%, respectively) [17]. Conversely, young hypertensive patients showed better control rate than older adults when treated.

HT is an important cardiovascular risk factor even in young adults. Recently, the association between office BP levels and major cardiovascular events in 98,000 young hypertensive patients (< 50 years old) was evaluated, using the Korean National Health Insurance Service database [29]. During a mean follow-up of 9.5 ± 2.8 years, 4,918 (5%) major adverse cardiac events (MACEs) were documented in the young HT cohort. Elevated BP levels (< 120/ < 70 mmHg) were significantly correlated with an increased risk of MACE in younger Korean hypertensive patients. Similarly, Son et al. [30] reported that young men aged 20 to 39 years old with prehypertensive BP range (130–139/80–89 mmHg) had a higher risk of cardiovascular disease when compared with those having normal BP (< 120/ < 80 mmHg; incidence, 215 vs. 164 per 100,000 person-years; adjusted hazard ratio, 1.25; 95% confidence interval [CI], 1.21–1.28), coronary heart disease (incidence, 134 vs. 103 per 100,000 person-years; adjusted hazard ratio, 1.23; 95% CI, 1.19–1.27), and stroke (incidence, 90 vs. 67 per 100,000 person-years; adjusted hazard ratio, 1.30; 95% CI, 1.25–1.36). Women with the same BP range also had an increased risk of cardiovascular disease (incidence, 131 vs. 91 per 100,000 person-years; adjusted hazard ratio, 1.27; 95% CI, 1.21–1.34), coronary heart disease (incidence, 56 vs. 42 per 100,000 person-years; adjusted hazard ratio, 1.16; 95% CI, 1.08–1.25), and stroke (incidence, 79 vs. 51 per

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100,000 person-years; adjusted hazard ratio, 1.37; 95% CI, 1.29–1.46).

Moreover, a longer HT morbidity duration leads to a higher lifetime risk of HT-mediated organ damage. The onset of HT before the age of 35 years was significantly associated with increased risk of left ventricular hypertrophy, coronary calcification and diastolic dysfunction compared to the onset of HT over the age of 45 years, in the follow-up period of 25 years [31]. Also, in a prospective study of 3,381 adults (age, 18–30 years at baseline) with 25 years of follow-up, higher cumulative SBP and DBP and fasting blood glucose were consistently associated with cognitive impairment in middle age [32].

BP patterns tend to change with age. As DBP tends to decrease with age, it is usually considered as an important risk factor of cardiovascular events in younger adults, whereas systolic BP is considered more important in older age [33]. However, isolated systolic HT (defined as SBP > 140 mmHg and DBP < 90 mmHg) which is especially common in young males [34], was associated with future development of sustained HT as well [35]. With such perplexing results, guidelines have not yet reached consensus on the treatment policy for isolated systolic HT in young patients [36]. However, a scoping review in 2021, encompassing 20 studies, identified important predictors of cardiovascular risk of isolated systolic HT in young males, suggesting drug treatment in high-risk young adult patients [34].

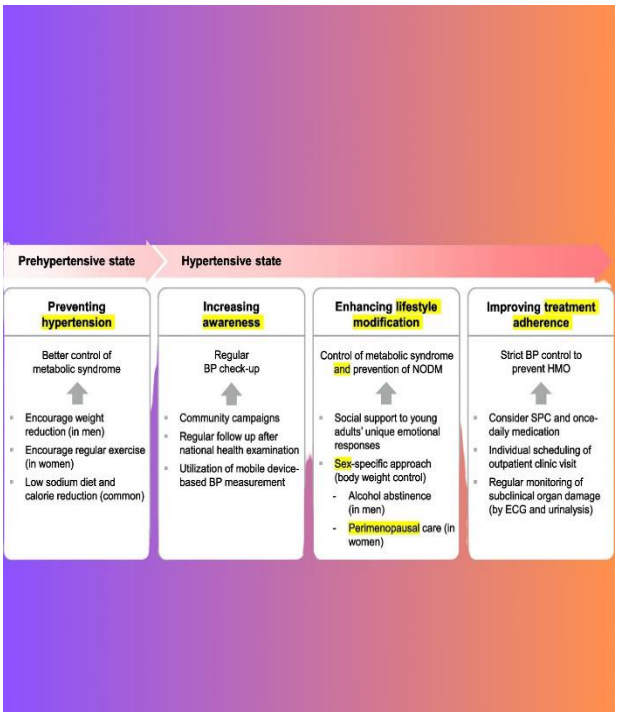

Fig: 2 Strategies to enhance current management status of young population with high blood pressure (BP). NODM, new onset diabetes mellitus; HMO, hypertension-mediated organ damage; SPC, single pill combination; ECG, electrocardiogram

III. HYPERTENSION DURING PREGNANCY

Gestational hypertension is the development of hypertension at or after 20 weeks gestation, in the absence of other features of pre-eclampsia. Gestational hypertension is associated with an increased risk of developing pre-eclampsia (up to 25%, depending on the gestation at presentation), as well as the future development of cardiovascular disease. Fetal growth restriction is not typically a feature of gestational hypertension. Regular blood pressure monitoring is necessary to ensure the blood pressure remains at 110–140/80–90 mmHg. There should be regular assessment for the development of pre-eclampsia and close surveillance of fetal growth and wellbeing. Once the blood pressure is controlled, gestational hypertension may continue to be managed with outpatient care, under close and regular review. Pre-eclampsia is a complex multisystem disorder of pregnancy arising from abnormal placentation, resulting in an imbalance of angiogenic and anti-angiogenic factors, oxidative stress and immunological involvement. The maternal response to this is thought to involve systemic vascular endothelial dysfunction. Pre-eclampsia may be superimposed on chronic hypertension, or present as new onset hypertension, arising at or after 20 weeks gestation, with the presence of one or more of the typical clinical features.

Risk factors for pre-eclampsia include maternal age, primiparity, previous pre-eclampsia, multiple gestation, prolonged interpregnancy interval and assisted reproduction therapies. Other factors are underlying renal disease or hypertension, antiphospholipid syndrome, systemic lupus erythematosus, diabetes and a maternal body mass index (BMI) above 30 kg/m^2.

To accommodate a growing fetus, maternal hemodynamics undergoes significant changes. The upregulation of the renin–angiotensin–aldosterone system, which begins in the luteal phase, is compounded by hormonal surges after fertilization.[3] It results in volume expansion *via* salt and water retention. Despite having high levels of renin (up to eight times normal) and aldosterone (up to 20 times normal), there is no rise in BP in normal pregnancy.[4] This is made possible by pregnancy-related vasodilation and decreased responsiveness of maternal vasculature to vasoconstrictors.[3] In the early first trimester, estrogen, progesterone, and relaxin surge lead to nitric oxide release, resulting in systemic vasodilation. The vasodilatory action of prostacyclins compounds this effect. Volume expansion and increased ventricular mass cause an increase in the stroke volume. There is physiologic anemia due to volume expansion, and the heart rate rises to compensate for anemia and vasodilatation. Increased stroke volume and heart rate lead to high cardiac output.[5] Beginning in the early first trimester, the mean arterial pressure drops by approximately 8–10 mm Hg (10% from baseline).[5] This

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decline reaches its nadir between the 16th and 20th weeks of gestation, after which it trends toward prepregnancy levels at approximately 40 weeks of gestation. Diastolic pressure shows a more significant decline as compared with systolic pressure. The rise in arterial compliance and venous capacitance in a healthy pregnancy leads to decreased effective plasma volume, resulting in a pregnancy-related decline in BP. Earlier studies suggested that women with preeclampsia have reduced plasma volume.[6] However, recent evidence is consistent that the suppressed plasma renin activity, higher BP, and subsequent decrease in GFR seen in preeclampsia are consistent with vasoconstriction and overfilled circulation rather than true hypovolemia.[7]

In normal pregnancy, kidneys enlarge, renal blood flow increases by 60%, and the GFR increases by up to 50% in midgestation.[6] The GFR is approximately 30% lower in women with preeclampsia than in normal pregnancy.[8] Women with preeclampsia have exaggerated hypercoagulability, dyslipidemia, and insulin resistance compared with normal pregnancy, which puts them at a higher cardiometabolic risk.[9,10] Women with preexisting vascular diseases, such as hypertension, diabetes, and CKD, are at higher risk of developing preeclampsia. This may be due to preexisting endothelial dysfunction.

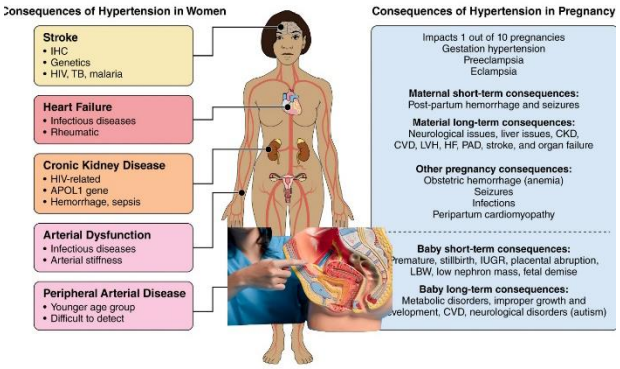

Fig: 3 Chronic hypertension during Pregnancy Hypertension & Menopausal

Studies have confirmed that hypertension is twice as likely post menopause than in premenopausal females [14]. There may be multiple aetiologies and triggers for hypertension in post-menopausal females, with renin-angiotensin-aldosterone system dysregulation, sympathetic activation and declining oestrogen levels being key factors. Animal studies suggest that multiple mechanisms are likely involved in post-menopausal hypertension [57].

During the menopausal transition, females are subjected to age-related changes in vascular function and sex hormone production due to declining ovarian function, which increases susceptibility to hypertension more so than age-matched males (Fig. 4) [58]. Ageing alters the structure and function of the vascular system, resulting in progressive arterial stiffening and decreased vasodilatory capacity [58, 59]. During ageing, lifestyle and biological factors modulate vascular function, increasing vascular oxidative stress and reducing antioxidant defences [58, 60]. Increased vascular oxidative stress leads to progressive dysfunction of the endothelial cell layer of the vascular wall. Dysfunction of the endothelial cell layer results in an imbalance in the secretion of endothelium-derived substances [58]. This imbalance impairs arterial endothelium-dependent vasorelaxation, enhancing constriction and vascular remodelling, which increases peripheral vascular resistance and is involved in the development of hypertension [61].

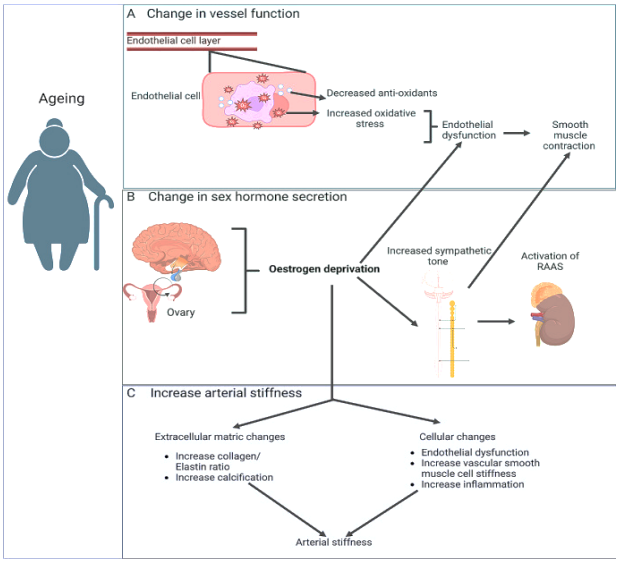

Fig: 4 Pathophysiological changes associated with ageing that predispose to increased blood pressure in post-menopausal women

IV. CONCLUSION

Hypertension is a key risk factor for cardiovascular disease in females, who appear to develop cardiovascular disease at lower BP thresholds and be more vulnerable to treatment-related adverse effects than men. Sex-specific risk factors, including pregnancy and menopause, make females more likely to have more adverse cardiovascular outcomes than age-matched men. Despite scientific advances, gaps in management outcomes persist between the two sexes. Current high BP treatment guidelines and recommendations are similar for both sexes, without addressing sex-specific factors. BP trials continue to have an inadequate representation of females. Therefore, future investigations into ideal diagnostic thresholds, BP control targets and treatment regimens are needed in females.

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