Editorial

Should we Measure Arterial Stiffness in Hypertensive Women

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Introduction

Hypertension is identified as the world's leading risk factor for cardiovascular morbidity and mortality[1] Hypertension is widely seen in men; however, compared with younger women, it is more prevalent among women than in older men.[2] Hypertension is associated with decreased arterial distensibility or increased arterial stiffness. This is partly attributed to high-pressure blood flow, which stretches the vessel wall, inducing endothelial dysfunction. Nitric oxide (NO) released from healthy endothelium is responsible for maintaining resting distensibility.[3] Age, obesity, acute and chronic smoking, diabetes, hypertension, end-stage renal disease and hypercholesterolemia, labeled as the potential cardiovascular disease risk factors, often remain the same for arterial stiffness.[4] As blood passes through these rigid arteries, higher amplitude pressure waves are produced during forward motion, and these waves return early as reflective waves, causing vasculature damage to heavily perfused organs such as myocardium, kidney, and brain.

Pulse wave velocity (PWV) is the forward pressure wave speed and an arterial stiffness marker. PWV can be obtained from carotid and femoral waveforms[5] or brachial tibial waveforms.[6,7] Studies have shown that cardiovascular predictions and all-cause death can be made even in noncardiovascular subjects based on carotid femoral (Cf) PWV values.[8,9] Arterial stiffness risk can be classified as normal, mild, moderate, and severe, based on the normal values which ranges from 5 to > 15 m/s.[10-13] In 2007[14] ESH/ESC specified the threshold value of Cf-PWV as 12 m/s. Systemic studies focusing on these guidelines have shown that a 1 m/s increase in Cf-PWV is related to 10% cardiovascular accident risk.[15]

In a study, Diabetes Risk Profiler, a noninvasive blood pressure curve device, was used to evaluate PWV in women with hypertension. Increase in PWV was found to correlate with elevated blood pressure.[16,17] Patients have significantly higher blood pressure and PWV compared with the controls. The variations in PWV values across varying degrees of hypertension, however, were not significant. That is due to the fact that most of patients were using vasodilators. Duration of antihypertensive use, location of PWV assessment, blood pressure, heart rate smoking, alcohol, and salty diet intake prior to PWV measurement should be considered as confounding factors when assessing differences in PWV values across these groups.

Hypertension, a major cardiovascular disease risk factor, can contribute to increased oxidative stress, microvascular inflammation, and endothelial damage. During hypertension, elastic fibers become compromised and damaged, resulting in decreased arterial wall stiffness.[18] Hypertension duration is substantially correlated with artery stiffening due to excessive oxidative load and prolonged strain, resulting in gradual vascular remodeling and loss of artery elasticity.

In women under 60 years of age, PWV is smaller than in males, but values increase sharply after menopause.[19] In postmenopausal women, endogenous vasodilatory estrogen[20] depletion leads to increased arterial rigidity.[21] Increased arterial stiffness in women results in poor hypertension control, weaker diastolic activity, impaired cardiac-arterial coupling, and left ventricular remodeling, all related to adverse cardiovascular effects.[22]

Interventions should aim to alter modifiable risk factors and prevent early development of increased arterial rigidity. Aerobic exercise,[23] intake of moderate alcohol,[24] and diet rich in flavonoids[25] and low in salt[26] have been shown to enhance artery compliance despite decreased blood pressure. By lowering blood pressure, vasodilators such as nitroglycerin,[27] drugs that inhibit renin angiotensin,[28-31] and calcium channel blockers[32] have shown increased compliance. Among vasodilators, drugs inhibiting renin-angiotensin-aldosterone pathways tend to have superior effect than others in decreasing arterial stiffness.[33] Statins,[34] by restoring endothelial functionality, demonstrated positive impact on arterial stiffness. Nebivolol, a NO donor β-blocker, has shown to reduce PWV independent of blood pressure and

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heart rate-lowering effects.[35] By its anti-inflammatory effect, endothelin-A antagonists[36] and acetylsalicylic acid (aspirin) improved arterial stiffness. Blood glucose-lowering drugs like glitazones and metformin[37,38] have shown improved arterial rigidity through multiple mechanisms. A research found that hormone therapy (HT) administration reversed arterial stiffness,[39] but exacerbated after HT discontinuation.[40]

Conclusion

Hypertensive women experience higher pulse pressure and stiffness. In diagnosing early cardiovascular disease in hypertension, evaluation of arterial stiffness, in addition to blood pressure, is essential. Treatments to alter cardiovascular risk factors, lower blood pressure, and enhance endothelial reflection may be useful in preventing further vascular disease progression in hypertension.

Conflicts of Interest
None declared.

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