Inflammation plays a key role in the pathogenesis of several cardiovascular diseases such as atherosclerosis, heart failure, and hypertension. Many studies have reported that clinical and experimental hypertension is associated with enhanced inflammatory mediators such as C-reactive protein (CRP) and cytokines at both tissue and circulating levels. This inflammatory situation could be primarily related with the elevated mechanical forces exerted against the arterial wall. In vitro studies have shown that elevated shear stress and circumferential strain are associated with overexpression of adhesion molecules, cytokines, and transcription factors such as NFκB and AP-1 in the arterial wall. In addition, circulating and paracrine factors associated with hypertension such as the renin–angiotensin system (RAS) could participate in this systemic inflammatory response. Angiotensin II plays an important role in functional and vascular alterations associated with hypertension through its proliferative, fibrotic, oxidative, and inflammatory actions. The inflammatory effect of angiotensin II is mediated, at least in part, through the activation of NFκB and the subsequent production of inflammatory mediators including interleukin (IL)-6 and IL-1β. We have previously demonstrated that treatment with an AT1 receptor antagonist reduced elevated IL-6 and IL-1β in spontaneously hypertensive rats, where this effect was associated with a reduction of NFκB and an enhancement of IkB vascular expressions.

The interplay between inflammation, angiotensin II, and hypertension has been extensively examined in preclinical studies, but information in humans is limited. In the present issue of *American Journal of Hypertension*, Chamarthi et al. further demonstrated a relationship between CRP, IL-6, and hypertension by comparing hypertensives to normotensives. The study also showed a significant rise in IL-6 in response to angiotensin II infusion in both hypertensives and normotensives. However, activation of circulating RAS through low salt diet (reflected by a higher plasma renin activity) was not associated with IL-6 enhancement. Perhaps, RAS activation through a low salt diet was not potent enough to cause a stimulatory effect on inflammatory mediators similar to the one caused by angiotensin II infusion.

Thus, inter-relationships between elevated blood pressure, inflammation, and angiotensin II appear to play an important role in the vascular alterations associated with hypertension. Considering that inflammation plays a key role in the development of atherosclerosis, and that hypertension is a primary risk factor for the development of this process, it could be proposed that the interplay between elevated blood pressure, inflammation, and angiotensin II could be of relevance in the complex mechanisms by which hypertension leads to atherosclerosis.

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