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Marie Briet

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Career Development Abstracts

ADVANCES IN ARTERIAL STIFFNESS ASSESSMENT

Evelien Hermeling

Please see Career Development Extended Review Paper section.

LARGE ARTERY REMODELLING AND STIFFENING IN MODERATE CHRONIC KIDNEY DISEASE

Marie Briet

Vascular and Hypertension Research Unit, Lady Davis Institute for Medical Research, Division of Nephrology, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montréal, Québec, Canada

Chronic kidney disease (CKD) is characterized by a very high cardiovascular risk even at moderate stage. Patients with CKD are exposed to traditional cardiovascular factors and to uremia-associated CV factors such as inflammation, oxidative stress, anemia, mineral metabolism disturbance which could affect the structure of large and small arteries. In moderate CKD, large artery damage is mainly characterized by an increase in aortic and carotid stiffness compared to hypertensive and healthy subjects. Interestingly, we recently demonstrated that aortic stiffness did not increase during CKD progression and was not associated with CKD progression. On the contrary, aortic stiffness has a clear impact on the cardiovascular prognostic in end stage renal disease. A maladaptive remodeling of large arteries is the second main abnormality in moderate CKD and is characterized by dilation and a thinning of the arterial wall leading to an increase in circumferential wall stress. The longitudinal measurements of carotid remodeling parameters have shown that intima-media thickness decreased with a slope comparable to the one measured in high cardiovascular risk patients but in an opposite way. We recently demonstrated that, in opposition to aortic stiffness, circumferential wall stress measured at the level of the carotid artery was associated with CKD progression and end stage renal disease.

In conclusion, arterial damage in moderate CKD is characterized by an increase in arterial stiffness, associated with a poor cardiovascular outcome

at end stage but with debated impact on CKD progression, and a paradoxical thinning of the arterial wall, associated with CKD progression.

INFLAMMATION AND LARGE ARTERIES

Dr. Kaisa Maki-Petaja

Rheumatoid arthritis (RA) provides an interesting model to explore the relationship between inflammation and large artery function and stiffness

Firstly, we demonstrated that RA is associated with increased aortic stiffness compared with healthy controls, and that aortic stiffness correlates with current, but not historical measures of inflammation. We also established that patients with RA have reduced endothelial function compared with matched control subjects and that endothelial function is independently associated with inflammatory markers.

Having shown that inflammation is a key mediator in aortic stiffening and endothelial dysfunction, we investigated the effect of anti-inflammatory treatments. We demonstrated that anti-TNF- α therapy reduces aortic stiffness and improves endothelial function to a level comparable with healthy individuals. Also, we showed that endothelial function and aortic stiffness can be improved with cholesterol reduction therapies, simvastatin and ezetimibe.

The mechanism by which inflammation leads to aortic stiffening remains unclear. One possible explanation is endothelial dysfunction. We tested this hypothesis by determining whether an improvement of endothelial function with tetrahydrobiopterin (BH₄), leads also to a reduction of aortic stiffness. We showed that BH₄ improves endothelial function, but had no effect on aortic PWV in patients with active RA. This suggests that endothelial dysfunction is not the driving force behind aortic stiffening in RA and that these conditions may just exist in parallel, both influenced by inflammation. The future work will involve exploring other possible mechanisms, which could explain the inflammation-induced aortic stiffening, such as changes in the composition of extracellular matrix and inflammatory cell infiltrations within the aortic wall.