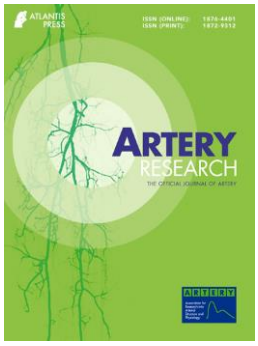

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

ARTERIAL STIFFNESS IN INFLAMMATORY DISEASE

Kaisa Maki-Petaja

To cite this article: Kaisa Maki-Petaja (2016) ARTERIAL STIFFNESS IN INFLAMMATORY DISEASE, Artery Research 16:C, 47–47, DOI: https://doi.org/10.1016/j.artres.2016.10.153

To link to this article: https://doi.org/10.1016/j.artres.2016.10.153

Published online: 7 December 2019

Common genetic forms of NAFLD such as the I148M variant in PNPLA3 ('PNPLA3 NAFLD') are characterized by steatosis but not insulin resistance or an increased risk of CVD or diabetes. The molecular mechanisms underlying this dissociation in the human liver and its implications for CVD will be discussed.

Invited Lecture

CONSTITUENT BASED MODELLING OF ARTERIAL WALL MECHANICS

Lydia Aslanidou [1], Rodrigo Araujo Fraga da Silva [1], Patrick Segers [2], Nikos Stergiopulos [1]

[1]*Institute of Bioengineering, Ecole Polytechnique Federale de Lausanne, Lausanne, Switzerland*

[2]*IBiTech-bioMMeda, Ghent University-iMinds Medical IT, Ghent, Belgium*

Abstract

In the preclinical setting, Angiotensin-II infusion has been the most popular model for mouse aneurysm research in the last 15 years. Nonetheless, little is known about the ascending aortic aneurysm pathobiology of this model and several lingering questions regarding the abdominal aortic aneurysm pathology (AAA) have long remained unaddressed, namely the suprarenal location of the murine AAA, the large morphological variation of the lesions and the presence of intramural thrombus. Technological advancements in both in vivo and ex vivo imaging techniques have significantly enhanced our understanding of the mechanisms driving the Angiotensin-II mouse model pathology. Our implementation of the groundbreaking PCXTM imaging modality has challenged the existing paradigm on this model while yielding unprecedented insight into previous observations on murine dissecting AAA. The detailed 3D PCXTM images have unveiled a previously unknown pivotal role for small, supraceliac and thoracic side branches to the onset of the disease. Mural ruptures in the vicinity of small side branches lead to apparent luminal dilatation and intramural hematoma. The PCXTM-based observations are in line with -seemingly incongruous- previous findings obtained with other imaging techniques, thereby raising a point on the importance of the implemented imaging modality when characterizing this aneurysm model.

Focus Lecture

ARTERIAL STIFFNESS IN INFLAMMATORY DISEASE

Dr Kaisa Maki-Petaja

University of Cambridge, Cambridge, UK

Abstract

Many inflammatory conditions are associated with an increased risk of cardiovascular disease (CVD) and mortality. As well as accelerated atherosclerosis, increased plaque instability and endothelial dysfunction; arterial stiffness has been proposed as one of the potential mechanisms underlying the increased CVD in these patients. Indeed, patients with chronic inflammatory conditions such as rheumatoid arthritis (RA), lupus erythematosus, human immunodeficiency virus, chronic obstructive pulmonary disease (COPD), and inflammatory bowel disease have been shown to have increased arterial stiffness. This appears to correlate with the level of inflammation, suggesting that arterial stiffness may be reversible with anti-inflammatory treatment. Numerous small-scale interventional studies have demonstrated that anti-inflammatory and cholesterol-reduction therapies with pleiotropic effects can reduce arterial stiffness in certain inflammatory conditions.

The association between increased arterial stiffness and inflammation appears obvious, yet the mechanism is poorly understood. One of the proposed mechanisms is arterial inflammation. In FDG PET/CT studies, patients with psoriasis, COPD and RA have been shown to have sub-clinical aortic inflammation. Arterial inflammation can subsequently lead to changes in the hydration state of the arterial wall and the composition of extracellular matrix, such as changes in glycosaminoglycan (GAG) synthesis. Indeed, animal studies have shown that overproduction of GAGs in the aorta resulted in thinning of the elastic lamellae and therefore aortic stiffening. Also, inflammatory cytokines can cause vascular smooth muscle cell proliferation, and phenotypic transformation resulting in an increased bioapatite formation, which can lead to calcification and stiffening. Furthermore, the release of matrix metalloproteinases from leukocytes can degrade elastin fibres within the arterial media.