Annals of Vascular Surgery

CORRESPONDENCE | ARTICLES IN PRESS

Deepening hemodynamics of iliac artery tortuosity

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Published: April 24, 2023 • DOI: https://doi.org/10.1016/j.avsg.2023.04.005

The interesting study b 35 y Song et al. on iliac arteries (IAs) tortuosity deserves to be deepened in some hemodynamic features.1 At histology, IAs, as elastic vessels, are prone to an ageing-related remodeling, expressing in the tunica media an increase in type I collagen fibers at the expense of those of type III and elastic lamellae, which undergo a process of fragmentation and dispersion. In addition, the smooth muscle cells lose their interconnections and become spaced each other (Fig. 1, A-D).2 The often superimposed atherosclerosis promotes infiltration of the tunica adventitia by leukocytes and other immune competent cells, in particular B lymphocytes, producing matrix metalloproteases, and activating macrophages and mast cells. This process releases Fibroblast and Vascular Endothelial Growth Factors, which, by their angiogenetic properties, induce a vascular remodeling.3 In IAs common and external segments, typical “conducting" vessels, this pathology causes a prevailing lengthening and a proportionally lowered blood flow kinetic energy, firstly for the Hagen-Poiseuille law. Secondly, parietal stiffness, often atherosclerosis-correlated, nullifies the "windkessel" effect, normally transferring part of the kinetic energy from the systolic to the diastolic phase of the arterial sphygmic wave, through an elastic mechanism of expansion and rebound. Thirdly, the amplified angle of the abdominal aorta bifurcation with IAs, their curved layout, and multiple three-dimensional inflections and bends increase energy dispersion, generating centrifugal forces. Fourthly, repeated changes in IAs size make the blood flow turbulent with a high Reynolds number, and subdivide it into multiple streams, some of which, for the forces parallelogram, collide against the original vector. Fifth, by the Bernoulli equation, dysmorphic and sequential changes in IAs diameter further reduce the blood flow kinetic energy (Fig.1, E). This chain of factors, often each other interconnected, increases the IAs thrombotic risk and reduce the blood supply to collaterals and femoral arteries, muscular conduits, where a higher hemodynamic resistance is encountered.5,7-9 Moreover, in trans-catheter procedures or endovascular surgery, it predisposes to possible unexpected complications, such as embolisms, arterial injuries, endoprosthesis malpositions, and endoleaks. The same hemodynamics negatively expresses also in IAs aneurysms, increasing their risk of thrombosis and rupture.4 Surgeons have to be alerted about this pathology and its complications, mainly today when their precise assessment can be achieved by 3D CT or quantitative 4D flow MRI angiography.6-10

Fig. 1 **Iliac arteries remodeling: [A] tunica media: smooth muscle cells dispersed between collagen fibers (hematoxylin-eosin staining, 40X); [B] tunica intima: endothelium with alternating zones of remodeling, destruction and missed elastic membrane (hematoxylin104 eosin staining, 100X); [C] tunica media: increased population of collagen fibers, blue stained, wrapping scattered smooth muscle cells, red stained (Masson-Goldner staining, 200X); [D] tunica media: elastic fibers disappeared, smooth muscle cells, rose stained, fragmented and rarefied inside an abundant extracellular matrix, pale yellow stained (Weigert-Van Gieson staining, 200X). [E] Diagram of decreased kinetic energy in iliac arteries.**

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Article info

Publication history

Accepted: April 3, 2023
Received in revised form: April 3, 2023
Received: March 30, 2023

Publication stage

In Press Journal Pre-Proof

Identification

DOI: https://doi.org/10.1016/j.avsg.2023.04.005

ScienceDirect

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