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Helical flow as a new determinant of coronary plaque vulnerability: a glimpse into the future

El flujo helicoidal como determinante de placa vulnerable: una mirada al futuro

To the Editor,

Over the last 2 decades, a growing body of evidence has emerged from both in vitro and in vivo studies regarding the relationship between the hemodynamic properties of blood flow and atherosclerotic plaque formation on coronary arteries. It is well known that atherosclerotic lesions typically develop at areas of low wall shear stress (WSS), which are associated with the development of unstable, rupture-prone atherosclerotic plaques.[1] In this context, low-WSS segments develop larger plaques and promote necrotic core progression as well as constrictive remodeling, while high-WSS segments favor larger necrotic core formation, calcium progression and excessive expansive changes, leading to a more vulnerable plaque phenotype.[2]

An intriguing novel determinant of WSS is represented by helical flow (HF). Indeed, a counterrotating-bi-helical-flow pattern, which is physiologically present in coronary arteries, has been described to be inversely associated with HF intensity. HF, per se, is characterized by high velocity and high WSS, which can confer atheroprotective effects facilitating the diffusion of oxygen, reducing the uptake of low-density lipoprotein, and the adhesion of inflammatory cells on the endothelium.[2] In this regard, Morbiducci et al.[3] observed that this flow pattern was also present on coronary artery bypass grafts, showing that its value leads to a lower WSS temporal gradient at the proximal graft. In more recent years, De Nisco et al.[4] reported that HF intensity was significantly correlated with WSS magnitude while coronary artery regions exposed to high baseline levels of HF intensity exhibited a significantly lower wall thickness growth than segments with either mid or low HF intensity.

The prompt and noninvasive identification of unstable coronary artery plaques still represents an important unmet medical need. HF could be a novel surrogate determinant of plaque vulnerability. In the near future, it will be possible to use software similar to that of computed tomography angiography-derived fractional flow reserve,[5] which is currently entering clinical practice as an alternative to invasive fractional flow reserve, and online computational fluid dynamic analysis to identify coronary segments and plaques at low HF, and thus exposed to the risk of rupture.

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M. Zuin: conceptualization, drafting the article; G. Rigatelli: drafting the article, references; G. Zuliani: reviewing and editing; L. Roncon: reviewing, editing, and supervision.

CONFLICTS OF INTEREST

None of the authors have any conflicts of interest to declare.

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