Local vascular inflammation involving the adventitial layer (the 'forgotten layer') is associated with the coronary hyper-reactivity in vasospastic angina,” comments John Beltrame (University of Adelaide, Australia) on the basis of new research published in JACC. According to the study, patients with coronary artery spasm have increased perivascular adipose tissue (PVAT) volume and perivascular inflammation.

Histologically, arterial walls can be divided into the intima (with an endothelial lining), media (containing vascular smooth muscle cells), and adventitia (featuring connective tissue and adipose cells). Vasospastic angina is the clinical manifestation of epicardial coronary artery spasm, which investigators have now shown can be caused by PVAT-associated inflammation.

Hiroaki Shimokawa and colleagues studied 27 consecutive patients with angina and acetylcholine-induced diffuse spasm in the left anterior descending coronary artery and 13 individuals with suspected angina but without coronary lesions or spasm. CT angiography showed that PVAT volume was larger in patients with vasospasm than in individuals without. Furthermore, use of 18F-FDG PET–CT showed that levels of perivascular inflammation were higher with vasospasm (image) than without. The presence of vasospasm was also associated with increased adventitial vasa vasorum formation, as assessed by optical coherence tomography, and a higher level of Rho-kinase activity (a molecular switch for vascular smooth muscle contraction) in circulating leukocytes.

Of note, medical treatment with calcium-channel blockers significantly reduced 18F-FDG uptake in the spastic artery and Rho-kinase activity. “These results demonstrate for the first time that coronary spasm is a dynamic disorder influenced by adventitial inflammatory changes,” concludes Shimokawa.

“Coronary inflammation has long been proposed in the pathogenesis of vasospastic angina,” states Beltrame, who was not involved in the research. “This study takes these observations to a new level because it supports the presence of inflammation within the coronary vessel wall, rather than as an associated systemic phenomenon.”

“Current therapies (calcium-channel blockers, nitrates, and nicorandil) are primarily focused on reducing vascular reactivity,” continues Beltrame. “This study now paves the way forward for an alternative therapeutic target in the management of vasospastic angina — modulating perivascular inflammation.”

Gregory B. Lim

Perivascular inflammation in coronary spasm

INFLAMMATION