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Anti-inflammatory treatment improves endothelial glycocalyx, peripheral and coronary microcirculatory function and myocardial deformation in inflammatory bowel disease patients

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Introduction: Inflammatory bowel diseases (IBD) alter gastrointestinal physiology and mucosal immunity through a complex inflammatory process. The extensive inflammation leads to significant arterial endothelial dysfunction as well as modification of cardiac structure and function. This study is performed to test the hypothesis that treatment with TNF-a inhibitor or surgical intervention in the IBD population improves cardiovascular function through anti-inflammatory mechanisms.

Methods: Thirty-seven IBD patients (28 CD and 9 UC, 39 ± 12 years, 62% male) were examined at baseline and 4 months after pharmaceutical (TNF-a inhibitor) (16 patients) or surgical intervention (21 patients). Subjects with a history of established cardiovascular risk factors were excluded.

We measured a) carotid-femoral pulse wave velocity (PWV - Complior SP ALAM), central systolic blood pressure (cSBP) and augmentation index (AI), b) flow mediated dilatation (FMD) of the brachial artery), c) perfused boundary region(PBR) of the sublingual arterial microvessels using Sideview Darkfield imaging, d) LV longitudinal strain (GLS), strain rate (GLSR) and (PWV/GLS) as a marker of ventricular-arterial coupling, e) peak LV twisting, peak twisting velocity (pTwVel) and peak untwisting velocity (pUtwVel) using speckle tracking echocardiography, f) mitral annulus velocities by tissue doppler imaging (S' and E') and mitral inflow velocity (E), g) coronary flow reserve (CFR) by Doppler echocardiography, h) C-reactive protein (CRP), white blood cells (WBC).

IBD severity was quantified using Mayo score and Harvey-Bradshaw Index (HBI) for UC and CD respectively, and correlated with the cardiovascular disease markers.

Results: At baseline, the disease severity score was significantly correlated with markers of diastolic dysfunction (lateral mitral E' velocity r=-0.352, p < 0.05, UntwVelE r = 0.389, p < 0.05), while the WBC values were negatively associated with lateral mitral E' velocity: r=-0.5, p < 0.05 and CFRvti (r=-0.332, p = 0.05). Four months after anti-inflammatory treatment, there was a reduction of CRP (15.5 ± 4.7 mg/L vs 5.1 ± 2.1 mg/L, p < 0.05) and WBC values (8.6 ± 0.6 vs 6.6 ± 0.7 x 103, p = 0.06). Moreover, post-treatment, there was a significant reduction of central arterial AI (3.58 ± 4.13 vs 0 ± 4.96, p < 0.05), PBR10-19 (2.47 ± 0.09 vs 2.24 ± 0.08 μm, p < 0.05) and PBR5-25 (2.31 ± 0.08 vs 2.14 ± 0.06 μm, p = 0.05) and increase of FMD (7.6%±0.7 vs 12.2%±1.7, p < 0.05), CFR (2.6 ± 0.1 vs 3.2 ± 0.14, p < 0.05), GLS (-18.7 ± 0.46 vs -20 ± 0.49, p < 0.05) and PWV/GLS (-0,48 ± 0.027 vs -0,42 ± 0.028, p < 0.05). No difference in the examined markers was observed between patients treated with anti-TNFa or surgery (p = NS).

Conclusion: IBD severity is associated with vascular and diastolic dysfunction. Anti-TNFa inhibition treatment or surgical intervention in IBD lead to improved myocardial deformation, endothelial and coronary microcirculatory function possibly through the reduction of excess inflammatory burden.