It Is Time for a Routine Measurement of Aortic Stiffness in Inflammatory Bowel Disease

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Dear Editor,

In a recent article published in the *Turkish Journal of Gastroenterology*,[1] Lu et al[1] performed an aggregated data meta-analysis to assess whether carotid-femoral pulse wave velocity (CF-PWV), a well-accepted measure of aortic stiffness, is increased in patients with inflammatory bowel disease (IBD). Authors concluded that CF-PWV is higher in both Crohn's disease (CD) and ulcerative colitis (UC) patients compared with their respective control subjects and that it is comparable between CD and UC patients; these results are in accordance with 2 independent aggregated data meta-analyses and a recent individual participant data meta-analysis performed in IBD and similar meta-analyses performed in other models of inflammation.[2] The increase of aortic stiffness in IBD is clinical of interest and can be considered a vascular biomarker of inflammation and an extraintestinal manifestation of IBD.[3] Moreover, considering that the cardiovascular (CV) risk is increased in IBD patients despite the prevalence of CV risk factors is lower than in the general population (this phenomenon is known as "IBD paradox"), it has been suggested that the increased aortic stiffness in CD and UC patients could be linked with the inflammatory state of the patients. In this regard, the role of acute and chronic inflammation on the arterial stiffening process has been recently discussed in a position statement from the European Society of Hypertension Working Group on Vascular Structure and Function and the ARTERY Society.[2] Inflammation can potentially lead to functional (and more easily reversible) and structural arterial stiffening. An important limitation of the aggregated data meta-analysis performed by Lu et al[1] is that the potential mechanisms of aortic stiffening in IBD were not explored. Interestingly, arterial stiffness is positively associated with the disease duration and white blood cell count in IBD, whereas vascular dysfunction is positively associated with erythrocyte sedimentation rate, C-reactive protein, and scores of disease activity in other models of chronic severe inflammation.[2] These findings suggest that chronic and active inflammation can be both involved in the stiffening process of IBD patients. In addition, a recent multicenter longitudinal study reported that aortic stiffness is reduced by tumor necrosis factor-inhibitors (TNFi) and that their effect is more evident when used in patients with a recent diagnosis of IBD,[4] suggesting that inflammation leads, at least in part, to functional alterations of the arterial wall in IBD. Considering that the reduction of aortic stiffness leads to a parallel reduction of the CV risk in other diseases, it is reasonable that the same positive effect would be present also in IBD. However, considering the peculiar arterial phenotype of patients with IBD,[5] large prospective studies in IBD are needed to confirm this hypothesis.

Finally, the results of Lu et al[1] are also in line with similar findings reported in other diseases characterized by severe inflammation.[2] Therefore, it would be of interest to test whether aortic stiffness is increased in a novel model of inflammation, the coronavirus disease 2019 (COVID-19), characterized by a peak of severe inflammation during the acute phase of the disease and the persistence of several symptoms in the mid-term (the so-called "long COVID"). Ongoing studies have been designed to solve this question.

In conclusion, the results of the meta-analysis performed by Lu et al[1] are in line with the recent indication of aortic stiffening as an extraintestinal manifestation of IBD and support the routine measurement of aortic stiffness in patients with UC and CD. In this regard, we suggest that it is time to include the measurement of aortic stiffness

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in the protocols for the evaluation of the extraintestinal manifestation of IBD.

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