on BAV aortas with mean diameter as small as 3.9 cm, gene expression analysis is revealing significant alterations in resident cell phenotype and transforming growth factor-factor signalling (unpublished data).

Most likely, the authors’ results were only in part affected by the above flaws, and therefore they deserve serious consideration, as the alarming evidence is presented that we are currently unable to non-invasively detect aortopathy without dilatation, but also we currently replace, according to guidelines, a number of aortas with absent/mild histological alterations! Are those aortas really at risk of dissection or fast growth? Words of caution were recently issued about the current aggressiveness in prophylactic aorta replacement for BAV disease [3, 4].

The paper by Leone et al. [1], as well as our most recent research [5], underscores the need for further investigation on the pre-surgical stages of BAV aortopathy, to identify early predictors of aortic dilatation/dissection, including functional (rather than merely dimensional) imaging measurements [5] and humoral biomarkers [6].

References


LETTER TO THE EDITOR RESPONSE

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We thank Dr Della Corte et al. for their interest in our paper ‘The elusive link between aortic wall histology and echocardiographic anatomy in bicuspid aortic valve: implications for prophylactic surgery’ [1, 2].

(1) Clearly, the absence of correlation between echocardiographic and histological findings (which is the main result of our paper) has two consequences: part of the patients without severe ascending aortic dilatation has advanced histological abnormalities, whereas part of the patients with aortic dilatation leading to surgical intervention lacks histological abnormalities. In our cohort of patients, 18 (14%) had root dilatation (dilatation isolated or prevailing at the sinusual portion), a percentage similar to that reported in Della Corte et al.’s paper [3].

(2) Regarding the site of sample retrieval, the retrospective nature of the paper did not allow us to perform targeted sampling and aortic samples were randomly obtained from the ascending aortic tract, distally to the sino-tubular junction. Additionally, the samples were not oriented or targeted at the level of maximal diameter.

(3) Finally, we confirm that all patients with advanced atherosclerotic lesions were excluded from this study. Only patients with minor atherosclerotic lesions (including adaptive intimal thickening, intimal xantoma, pathological intimal thickening or early and small fibroatheroma) were included, according to the morphological classification of the atherosclerotic lesions by Virmani et al. [4] and more recently by Van Dijk et al. [5]. It is highly improbable that these lesions can have significant consequences on the medial layer structure.

References