



## LETTER TO THE EDITOR

### Reply to the letter “The relation between inflammation and coronary artery ectasia”



#### Resposta à carta «A relação entre a inflamação e a ectasia arterial coronária»

Dear Editor,

We would like to thank Balta et al.<sup>1</sup> for their interest in our article.<sup>2</sup> In our study, we evaluated the frequency of major cardiovascular risk factors and serum levels of gamma-glutamyltransferase (GGT) and high-sensitivity C-reactive protein (hs-CRP) in a relatively large population of patients with isolated coronary artery ectasia (CAE). We found that CAE can be independently and positively associated with obesity, GGT and hs-CRP levels, but inversely with diabetes. Moreover, its severity may be related to GGT and hs-CRP levels.<sup>2</sup>

Coronary artery ectasia is usually defined as dilation exceeding 1.5 times the diameter of adjacent normal segments in epicardial coronary arteries.<sup>3,4</sup> Its underlying causes are poorly understood, but it has frequently been considered as a variant of atherosclerotic vascular disease.<sup>3</sup> Atherosclerosis is regarded as a low-grade inflammatory process<sup>5</sup> and thus it is not surprising that there may be a close association between inflammation and CAE. As reported by Balta et al.,<sup>1</sup> various studies have shown such an association.<sup>6–10</sup> Moreover, inflammation may be related to severity of CAE. However, the extent of CAE can be differently defined. Markis et al. categorized CAE in four types, with decreasing severity of CAE from type I to IV according to its topographical extent in the major coronary arteries.<sup>3</sup> Based on this definition, types I, II and III CAE can be considered severe ectasia. By contrast, we defined severe CAE as diffuse involvement ( $\geq 2$  segments) in at least two vessels. This definition corresponds to type I CAE according to Markis’s classification. Our definition has also been used in previous studies.<sup>8,9</sup>

We agree with Balta et al. that some disorders such as obesity, thyroid dysfunction and malignancy can trigger an

inflammatory state in which inflammatory markers like hs-CRP may be elevated. We excluded patients with thyroid dysfunction and known malignancies and inflammatory diseases from the study. With obesity, we consecutively included CAE patients in the study and selected age- and gender-matched controls with normal coronary arteries. Although mean body mass index was comparable between CAE patients and controls, the obesity rate was slightly higher in CAE patients (16% vs. 9%,  $p=0.06$ ) and obesity was positively associated with CAE. We think that inflammatory activity may contribute to the development of CAE in obese patients.

Serum GGT, a major antioxidant, can oxidize low-density lipoprotein cholesterol, and has a role in the pathogenesis of atherosclerosis.<sup>11</sup> GGT can also act as a proinflammatory protein in atherogenesis and is associated with atherosclerotic risk factors including obesity, dyslipidemia, metabolic syndrome, hypertension and diabetes.<sup>12,13</sup> Primary hepatic diseases such as alcoholic or viral hepatitis and Gilbert syndrome can lead to elevated transaminases, GGT, and bilirubin levels in hepatic function tests. We did not enroll patients who had previously known hepatic disease and who regularly drank ethyl alcohol. Accordingly, we think that the relatively elevated levels of GGT may be associated with CAE itself rather than other disorders or situations.

In conclusion, there is growing evidence that an inflammatory state may be activated in CAE patients and that its markers may be elevated in CAE patients as in obstructive coronary artery disease. As an oxidative stress and proinflammatory marker, GGT may also have a role in the pathogenesis of CAE. This consideration needs future studies.

### Conflicts of interest

The authors have no conflicts of interest to declare.

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