



## LETTER TO THE EDITOR

### The relation between inflammation and coronary artery ectasia



### A relação entre a inflamação e a ectasia arterial coronária

Dear Editor,

We read the article “Gamma glutamyltransferase, inflammation and cardiovascular risk factors in isolated coronary artery ectasia” by Dogan et al.<sup>1</sup> They aimed to investigate major cardiovascular risk factors, serum gamma-glutamyltransferase (GGT) and high-sensitivity C-reactive protein (hs-CRP) levels in a large population of patients with coronary artery ectasia (CAE). They concluded 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.

Coronary artery ectasia is defined as a localized or diffuse non-obstructive lesion of the epicardial coronary arteries with a luminal dilation exceeding 1.5 times the normal adjacent segment or vessel diameter. Although the etiology of CAE is not well understood, atherosclerosis may be considered a major part of the process.<sup>2</sup> Inflammation is an important step in the atherosclerotic process.<sup>3</sup> We have reported elevated inflammatory markers in CAE compare with controls.<sup>4–6</sup> In the present study,<sup>1</sup> the authors showed a relation between CAE and inflammation. In this respect, we argue that some aspects should be considered when assessing the relation between CAE and inflammation. Firstly, classification of CAE is an important factor in study design. In the CAE classification proposed by Markis et al.,<sup>7</sup> in decreasing order of severity, diffuse ectasia of two or more vessels was classified as type I, diffuse disease in one vessel and localized disease in another vessel as type II, diffuse ectasia of one vessel only as type III and localized or segmental ectasia only as type IV. Inflammatory markers may be different depending on the severity of CAE according to Markis’ classification. Secondly, certain

conditions including metabolic syndrome,<sup>8</sup> thyroid dysfunction, and known malignancy<sup>9</sup> may alter inflammatory conditions in these patients. Besides, we have reported elevated inflammatory markers in inflammatory diseases like Behçet’s disease.<sup>10,11</sup> We have also shown that some medications decrease inflammatory markers.<sup>12</sup>

Furthermore, in their article the authors showed an association between serum GGT and CAE, but some important issues should be addressed. Serum GGT activity is a marker of oxidative stress and endothelial dysfunction. We previously showed that serum GGT levels may be an independent marker of the severity of cardiovascular disease.<sup>5</sup> GGT may change as a result of hepatic dysfunction even if there is no overt liver disease. Secondly, Gilbert syndrome is a common condition worldwide that can affect liver function tests. GGT is also a widely measured serum enzyme and is commonly elevated in cases of biliary epithelial damage and excessive alcohol intake.

In conclusion, although the authors concluded that CAE can be independently and positively associated with inflammation in their study, we strongly believe that inflammatory conditions in CAE patients should be thoroughly assessed in future studies.

### Conflicts of interest

The authors have no conflicts of interest to declare.

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