

In conclusion, although the authors concluded that only YKL-40 level was established as the determinant of CAE, but YKL-40 is not used for inflammation in clinical practice. So, we believe that not only YKL-40 but also routine, inexpensive, easy inflammatory tests like red cell distribution width, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio and mean platelet volume should be evaluated in future studies.

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isolated CAE compared with patients with normal coronary arteries (NCA) and coronary artery disease (CAD). Increased YKL-40 levels may be observed due to many causes and if other concomitant diseases are not ruled out, the application as cardiac marker can lead to misinterpretation. We accept that YKL-40 is not a specific vascular, inflammatory biomarker however, red cell distribution width, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio, mean platelet volume are neither specific nor routinely used in clinical practice (2). We have been criticized for not excluding potential factors that might affect YKL-40, however as far as we know, we excluded malignancy, infectious diseases and inflammatory conditions, hepatic and renal failure. It would have been better, although exhausting, if a selected patient population for isolated CAE had been composed. In addition to obstructive sleep apnea syndrome (OSAS) and non-alcoholic fatty liver disease (NAFLD), a possible related mechanism may be increased epicardial adipose tissue (3).

Based on previous arguments, although we cannot conclude the underlying pathologic process of CAE, we believe that further studies searching signaling on ectatic process in coronary vasculature are needed to clarify more accurately the mechanisms of CAE and the specific roles of YKL-40, and to confirm the importance of modulating real underlying process to improve clinical outcome.

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YKL-40 as new cardiac biomarker

The publications on YKL-40 as a new cardiac biomarker is very interesting (1, 2). According to the report by Erdoğan et al. (2) a "Increased YKL-40 levels in patients with isolated coronary artery ectasia: an observational study" in *Anadolu Kardiyol Derg* 2013; 13: 465-70. It was concluded that "YKL-40 levels in patients with isolated CAE compared to patients with normal coronary arteries (NCA) were found significantly high and only YKL-40 level was established

Author's Reply

To the Editor,

We thank the authors for their comments on our article in *Anadolu Kardiyol Derg* 2013; 13: 465-70. (1) entitled as 'YKL-40 levels in Patients with Coronary Artery Ectasia' in their letter. The goal of this study was to investigate YKL-40 and C-reactive protein (CRP) levels in patients with

as the determinant of CAE ." It is no doubt that YKL-40 might be applied as a good cardiac biomarker. However, there are many concerns of this biochemical test. First, as it is widely discussed, this biomarker is considered a non - specific marker (3). Its increase level can be due to many causes and if there is no good ruling out of other concomitant disease, the application as cardiac marker can lead to misinterpretation. Second, the standardization of the technique is very important. At least, the consensus to develop the international laboratory procedure guideline and reference range setting is needed. Bojesen et al. (4) found that "plasma YKL-40 increases with age within and across healthy individuals from the general population " and concluded for the necessity of "age-stratified or age-adjusted reference levels"

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Author's Reply

To the Editor,

We would like to thank the authors for their comments on our article (1) entitled as 'YKL-40 as new cardiac biomarker' in *Anadolu Kardiyol Derg* 2013; 13: 465-70. The aim of our study was to investigate YKL-40 and C-reactive protein (CRP) levels in patients with isolated CAE compared to patients with normal coronary arteries (NCA) and coronary artery disease (CAD). We demonstrated increased serum YKL-40 levels without increased systemic inflammatory response (The serum C-reactive protein [CRP] concentration was used as a surrogate marker of systemic inflammation) in patients with isolated CAE. YKL-40 as well as CRP might be non-specific markers of inflammation; however both are strong predictors of cardiovascular outcome (2). Therefore, in the event of carefully selected study population with a matching control group, our results carry important predictive and diagnostic meaning. As the authors stated that YKL-40 may be increased by ageing, we performed multivariate analyzes and did not identify YKL-40 as an inde-

pendent factor for CAE. We may hypothesize that YKL-40 may reflect silent atherosclerosis in a group of healthy people with varying ages (2), however; in a carefully constructed group by means of diagnostic coronary angiography, YKL-40 may be related to atherosclerosis but not to aging as documented in our study. We do share the opinion of the authors on standardization of the technique.

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The relationship between neutrophil-to-lymphocyte ratio and coronary artery disease

To the Editor,

We read the article "Relation of neutrophil-to-lymphocyte ratio with the presence and compleentitled of coronary artery disease" by Sönmez et al. (1) in *Anadolu Kardiyol Derg* 2013; 13: 662-7. The neutrophil-to-lymphocyte ratio (NLR), which represents an inflammatory state, was significantly higher in patients with coronary artery disease (CAD) compared to patients with normal coronary arteries. They concluded that NLR is a strong clinical laboratory value that is associated with presence and complexity of CAD. Thanks to the authors for their contribution.

The SYNTAX score is used for grading the complexity of CAD. It has been reported that elevated SYNTAX score is associated with higher rates of long term major adverse cardiovascular events and revascularization after percutaneous coronary intervention or coronary artery bypass graft. Stabil CAD is different from acute coronary syndrome. It is well known that this score has some limitations including the inability to estimate precisely coronary plaque burden or to identify vulnerable plaques and inter-observer variability inherent to visual estimation of vessel stenosis (2).