The main argument about the etiology of coronary artery ectasia: is it inflammation or not?

To the Editor,

We read with interest the article entitled "Association of neutrophil to lymphocyte (N/L) ratio with presence of isolated coronary artery ectasia (CAE)" by Isık et al.[1] They investigated a possible association between the N/L ratio and the presence of isolated CAE. They concluded that the N/L ratio is a readily available clinical laboratory value that is associated with the presence of isolated CAE.

The etiopathogenesis of CAE has been poorly understood. We think that CAE is the histopathological pattern of atherosclerosis. First, CAE and coronary artery disease (CAD) have similar risk factors. Second, the two conditions exhibit similar histopathological features.[2] The association between the N/L ratio and CAD in the general population is not very well defined. The relationship between the N/L ratio and CAE is precisely defined and inflammation and oxidative stress are likely to play a role.[3] Elevated differential cell counts, including eosinophil, neutrophil, and monocyte counts, are associated with the presence of CAD. The N/L ratio is an easy, cheap, noninvasive, and widely available laboratory marker used to assess systemic inflammatory conditions in patients. Furthermore, the N/L ratio may predict independent prognostic factors for many conditions.

However, abnormal thyroid function tests, local or systemic infection, and inflammatory diseases can potentially influence the N/L ratio.[3] Moreover, some medications used, such as antihypertensive therapy, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and statins, may also influence the N/L ratio.[4] Thus, it would have been better if the authors had mentioned these factors.

CAE classification is also important. The CAE classification previously described by Markis et al.,[5] in decreasing order of severity, is as follows: Type I: diffuse ectasia of 2 or 3 vessels, Type II: diffuse disease in 1 vessel and localized disease in another, Type III: diffuse ectasia of only 1 vessel, and Type IV: localized or segmental ectasia. The N/L ratio was significantly associated with severity of CAE in a previous study.[6] Thus, if the authors had classified CAE according to Markis's classification, the results of the study would have been more useful.

In addition, the authors did not evaluate markers of inflammation such as C-reactive protein (CRP), although the role of inflammation was previously demonstrated in these patients. If CRP levels of these patients had been assessed and correlated with the N/L ratio, the results of the present study would be helpful. Recently, we have come to believe that the N/L ratio without other inflammatory markers may not provide information to clinicians about chronic endothelial inflammation.[7]

Obstructive sleep apnea syndrome (OSAS) and nonalcoholic fatty liver disease (NAFLD) are commonly seen in clinical practice. Cardiovascular diseases and related comorbidities frequently associated with OSAS have been linked to morbidity and mortality in these patients based on endothelial dysfunction.[8] Furthermore, the presence and degree of NAFLD are related to higher inflammatory parameters in nonhypertensive, nondiabetic individuals. Additionally, common pathways involved in the pathogenesis of NAFLD include hepatic insulin resistance, subclinical inflammation, and atherosclerosis.[9] From this point of view, because NAFLD and OSAS are associated with higher inflammatory status, it would have been useful if the authors had mentioned these factors.

In conclusion, the N/L ratio is a readily available clinical laboratory value that is associated with the presence of isolated CAE, as presented in the current study. However, these findings will enlighten further studies about the N/L ratio as a surrogate marker of severity in CAE. Not only the N/L ratio, but also mean platelet volume, red cell distribution width, platelet distribution width, platecrit, total bilirubin, uric acid, and gamma-glutamyl transferase are simple, cheap and routine markers to evaluate the relation between inflammation and CAE.[10] Thus, we think that the N/L ratio should be evaluated together with other inflammatory indicators.

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Düzeltme
Erratum

Türk Kardiyoloji Derneği Arşivi, Cilt 41, Sayı 8, s. 699-704

Türk Kardiyoloji Derneği Arşivi, Cilt 41, 8. sayısında, sayfa 699-704'de ***"Perkütan koroner girişim sonrası -mobiliteye izin veren-kontrollü baskı kemerinin kum torbasıyla karşılaştırılması: Pilot çalışma / Comparison of controlled pressure belt -allowing mobility-to sandbags after percutaneous coronary intervention: pilot study"*** adıyla yayımlanan makale yazarlarından Murat Yüce'nin adı PubMed MEDLINE dizininde hatalı çıkmıştır. Doğrusu "Yüce M" olacaktır. Düzeltiriz.