imab in patients with STEMI treated with primary PCI does not appear to be beneficial in those who receive pre-PCI thienopyridines (3). However, their comments are limited to five abciximab series and cannot be compared with the main results of our retrospective study in a total of 994 patients with STEMI in whom TRT was used prior to, during, or after primary PCI. Recent studies confirmed our positive results on upstream TRT (4, 5).

Intracoronary TRT was the choice in all patients of the peri-PCI TRT group, whereas only the intravenous route was used in the upstream or post-PCI TRT groups. Although the median difference in pain-to-balloon time was only 25 min between the upstream and peri-PCI TRT groups, more positive results with upstream TRT can be considered consistent with the potential benefit of earlier TRT over intracoronary injection of this drug at Cath Lab.

At the time of the enrollment, a manual aspiration catheter was not available in our center. In our opinion, "pain-to-balloon time" instead of "first medical contact-to-balloon time" seems to be a more appropriate measure for the estimation of total ischemic time, and the definition also includes the time delay from the occurrence of pain to the first medical contact. Data from angiographic and ST-segment resolution in the pre-PCI, peri-PCI, and post-PCI TRT subsets can answer your question concerning the effect of TRT on the no-reflow phenomenon. All patients with no-reflow or high thrombus burden without satisfactory ST-segment resolution underwent repeat angiography after TRT. In case of renal insufficiency, bolus TRT was not followed by infusion.

Finally, I would like to thank you for this letter, which led to a discussion concerning the use of upstream TRT as an adjunct treatment to DAPT in patients who underwent primary PCI.

Cihangir Kaymaz
Department of Cardiology, Kartal Koşuyolu Yüksek İhtisas Training and Research Hospital; İstanbul-*Turkey*

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Address for Correspondence: Dr. Cihangir Kaymaz
Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Kartal, İstanbul-*Türkiye*
Phone: +90 216 459 44 40
Fax: +90 216 500 15 00
E-mail: cihangirkaymaz2002@yahoo.com

Uniform criteria for diagnosing noncompaction by cMRI and echocardiography are warranted

To the Editor,

We read with great interest the article by Akhbour et al. (1) published in Anatol J Cardiol 2015; 15: 550-5 entitled "Electrocardiographic findings in correlation to magnetic resonance imaging patterns in African patients with isolated ventricular noncompaction" on cardiac magnetic resonance imaging (cMRI) and electrocardiographic (ECG) findings in 24 patients with left ventricular hypertrabeculation (LVHT)/noncompaction. Systolic function and arrhythmia were not correlated with the number of non-compacted segments or the number of segments showing late gadolinium enhancement (LGE) (1). We have the following comments and concerns.

Though LVHT is presumably congenital in majority of the cases, it can be also acquired, such as in neuromuscular disorders (NMDs), (2) pregnant females (3), and athletes (4). Acquired LVHT suggests that LVHT is not only due to the failure of the embryonic compaction process but also may result from the adaptation of the myocardium to hemodynamic dysfunction.

We do not agree with the definition of LVHT for not allowing the presence of any other cardiac abnormality except LVHT (isolated LVHT). Non-isolated LVHT is frequent and is also LVHT.

How do the authors explain the missing correlation between the number of LGE segments and ventricular tachycardia? Was the group size too small? Was the correlation different when subendocardial, transmural, and mid-myocardial LGE were separately evaluated? Was the LGE pattern patchy or diffuse? Possibly, cMRI fails to display all degrees of fibrosis, particularly fibrosis of the endocardium or early evolving fibrosis? Possibly, ventricular arrhythmias are not correlated with the number of LG -segments but with the volume or area of the LGE lesions? It is also conceivable that fibrosis in LVHT is ethnically different; for instance, Caucasians show a positive correlation between fibrosis and arrhythmias, whereas Africans do not, similar to the results in the present study. How did the authors quantify arrhythmias to correlate them with the number of LVHT fibrotic segments?

Arrhythmias may not only result from myocardial fibrosis but also result from ischemia. There are some indications that perfusion of the non-compacted layer is worse than that of the compacted layer (5). Possibly, the amount of arrhythmias correlates with myocardial scintigraphy. The frequent occurrence of LBBB may not only result from myocardial fibrosis but also from trabeculations, which predispose for prolonged propagation of the excitation.

We do not agree with the statement that cMRI is the method of choice to diagnose LVHT (1). The method of choice is echocardiography, but in case the echocardiographic diagnosis is uncertain, cMRI should be performed. Both techniques supplement each other, but they produce false positive and false negative results. As long as there are no common generally accepted LVHT diagnostic criteria either for cMRI or for echocardiography and as long as there is no gold standard for diagnosing LVHT, the reliability of both methods remains limited.

Atrial fibrillation was found in 17% of patients (1). Did these patients also present with thrombi within the intertrabecular spaces?

Was intraventricular thrombus formation associated with cardiac function?

Overall, this interesting study could profit from including patients other than Africans, from increasing the group size, and from evaluating the LGE extension. The negative correlation found could be explained by the absence of a uniform definition of LVHT, thus including patients who do not have LVHT or excluding patients who definitely have LVHT.

Josef Finsterer, Sinda Zarrouk-Mahjoub[1]
Krankenanstalt Rudolfstiftung; Vienna-*Austria*
[1]Genomics Platform, Pasteur Institute of Tunis; Tunis-*Tunisia*

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Address for Correspondence: Univ. Prof. Dr. Josef Finsterer
Postfach 20, 1180 Vienna-*Austria*
Phone: +43-1-71165-92085
Fax :+43-1-4781711
E-mail: fifigs1@yahoo.de

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Author`s Reply

The authors of this mentioned article did not send any reply to this Letter to the Editor in spite of our insistent requests.

Predictors of poor coronary collateral development in patients with stable coronary artery disease: Neutrophil-to-lymphocyte ratio and platelets

To the Editor,

We read with great interest the paper titled "Predictors of poor coronary collateral development in patients with stable coronary artery disease: Neutrophil-to-lymphocyte ratio and platelets" by Akın et al. (1) that was published in the April issue of the Anatol J Cardiol 2015; 15: 218-23. In this original article, the authors revealed that the neutrophil/lymphocyte (N/L) ratio is independently associated with the presence of coronary collateral circulation (CCS) in patients with stable angina pectoris. Also, they found that an N/L ratio higher than 2.55 could predict a good collateral circulation with 76% sensitivity and 63% specificity using ROC analysis.

Data in current scientific literature reveal that CCS is associated with metabolic syndrome and serum cholesterol levels (HDL and especially triglyceride) (2, 3). There are similar findings in the present study (1). In particular, the study found that a lower triglyceride level is significantly related with good CCS and is an independent predictor in multivariate regression analysis.

Exercise has a positive effect on the lipid profile, and it does improve metabolic syndrome parameters. Furthermore, Wang et al. (4) showed in their study that diet and exercise improve the N/L ratio. The study also demonstrated that a decrease in the N/L ratio is positively correlated with interleukin-6 levels. As a result, they concluded that diet and exercise have a positive impact on pro-inflammatory mediators.

The positive impact of exercise on CCS development has been well known for some time (5). As the positive effect of exercise on lipid parameters and the N/L ratio is already known, the questions that come to our mind are whether patients who have good CCS exercise more, and could a lower triglyceride level and a lower N/L ratio be related to exercise? However, for us to answer these questions, more information about the functional status and exercise capacity of the study patients is required, and this was not mentioned in the article. Furthermore, body mass index data for these patients are unavailable. In our opinion, the findings from this study could be related to the amount of exercise, and further studies in this area could answer this question.

Kamil Gülşen, Levent Cerit, Barçın Özcem*, Onur Akpınar
Departments of Cardiology and *Cardiovascular Surgery, Faculty of Medicine, Near East University; Nicosia-*Turkish Republic of Northern Cyprus*

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Address for Correspondence: Dr. Kamil Gülşen
Yakın Doğu Bulvarı, PK: 99138
Lefkoşa- KKTC, Mersin-*Türkiye*
Phone: +90 392 223 64 64
E-mail: kamilgulsen2000@yahoo.com

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