## Editöre Mektup — Letter to the Editor

### *Akut miyokard infarktüsü nedeniyle primer perkütan girişim uygulanan hastalarda başvuru anındaki hs-CRP düzeyinin önemi*

Sayın Editör,

Derginizin Ocak 2009 tarihli 1. sayısında, Çağlı ve ark. tarafından sunulan "Akut miyokard infarktüsü nedeniyle primer perkütan girişim uygulanan hastalarda başvuru anındaki hs-CRP düzeyinin önemi" adlı makaleyi ilgiyle okudum.

Öncelikle yazarları titizlikle hazırlanmış makalelerinden dolayı kutlarım.

Yazarların kullandıkları diğer yöntemlere değinmeyeceğim. Sadece 0 ve 1 TIMI kan akımı olan hastalarda TIMI kare sayımını değerlendirme yöntemlerini, tüm hastalarda perkütan koroner anjiyoplasti sonrası elde edilen TIMI akım değerlerini, ayrıca yaptıkları kıyaslamalarda perkütan koroner anjiyoplasti öncesi TIMI kare sayımı değerlerini neden kullanmadıklarını merak ediyorum.

Saygılarımla,

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#### *Yazarın yanıtı*

Sayın Editör,

Türk Kardiyoloji Derneği Arşivi'nin 2009 yılı, 1. sayısında yayımlanan "Akut miyokard infarktüsü nedeniyle primer girişim uygulanan hastalarda başvuru anındaki hs-CRP düzeyinin önemi" başlıklı makale ile ilgili bir okuyucunun sorusu ve buna yanıtımız aşağıda yer almaktadır.

Soru:

- Sadece 0 ve 1 TIMI kan akımı olan hastalarda TIMI kare sayımı değerlendirme yöntemi,

- Tüm hastalarda perkütan koroner anjiyoplasti sonrası elde edilen TIMI akım değerleri,

- Karşılaştırmalarda perkütan koroner anjiyoplasti öncesi TIMI kare sayımının kullanılmamasının nedeni.

Yanıt:

- Makalenin yöntem kısmında da belirtildiği üzere TIMI 0'da tıkanıklık distalinde akım ve perfüzyon yoktur, TIMI 1'de tıkanıklık distalinde penetrasyon var ancak perfüzyon yoktur. Bu nedenle, TIMI 0 ve 1 akım derecesine sahip damarlarda distal akım ve perfüzyon olmadığından distalde TIMI kare sayısını değerlendirmek mümkün olmamaktadır.

- Hastalardaki perkütan girişim sonrası TIMI akım değerleri Tablo 2'de hs-CRP'si düşük ve yüksek olmak üzere iki gruba ayrılarak verilmiştir. Buna göre, işlem sonrası TIMI akım derecesi hs-CRP düzeyi <0.98 mg/dl olan grupta 2.77±0.43 ve hs-CRP düzeyi >0.98 mg/dl olan grupta ise 2.57 ±0.67 olarak bulunmuş ve istatistiksel bir anlamlılık saptanmamıştır (p=0.248)

- İlk sorunun yanıtında olduğu gibi, işlem öncesi TIMI 0 ve TIMI 1 akım derecesine sahip olan hastalar çoğunluğu (34/43 hasta) oluşturduğundan işlem öncesi TIMI kare sayısını hesaplamak ve kullanmak doğru olmayacaktır. Bu nedenle, infarkt ile ilişkili damarlardaki TIMI kare sayısı sadece işlem sonrasında değerlendirilmiştir.

Saygılarımla,

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### *A case of simultaneous anterior, inferior, and right ventricular ST-segment elevation myocardial infarction due to occlusion of the wrapped left anterior descending coronary artery*

Dear Editor,

This report pertains to the article presented by Akpınar et al. in the July 2008 issue of *Archives of the Turkish Society of Cardiology*.[1] We would like to add some comments on the physiopathology and

clinical significance of ST-segment elevation (STE) in the right precordial leads in the setting of anterior myocardial infarction (MI). It is hoped that these will be clinically useful for both the clinical and interventional cardiologist and expand the information presented in the corresponding article.

Akpınar et al. presented a very interesting case of "anterior-inferior" MI with simultaneous STE in leads V3R and V4R.[1] While STE in lead V4R ≥1 mm is diagnostic of right ventricular infarction (RVI) during inferior MI, this case reminds us that STE in the right precordial leads and especially lead V3R may also be recorded in 30% to 40% of cases of anterior MI.[2,3] Right ventricular infarction does occur during anterior MI as well, albeit less frequently than during inferior MI; however, STE in the right precordial leads fails in its diagnosis.[4-6] In fact, STE in lead V3R and its anatomically closest lead, namely lead V1, during anterior MI indicates infarction of the right side of the anterior interventricular septum (IVS).[2,3] This report presents some comments on the physiopathology and clinical significance of STE in the right precordial leads during anterior MI.

**Anatomic considerations**

While the right coronary artery supplies the most of the right ventricular (RV) free wall, left anterior descending artery (LADA)-derived RV branches may supply a substantial area of the anterior RV free wall, namely >30% of the entire RV free wall, in 24% of human hearts.[4] The LADA may also supply the inferoposterior RV free wall adjacent to the apex in 22% of human hearts where it exhibits a wrap-around the apex trajectory.[4] Right ventricular infarction during inferior MI is common with an incidence of up to 50%, being limited to the posterior RV free wall in the majority of cases and is larger than anterior MI-related RVI (28% *vs*. 7% of total infarct size, respectively).[4,7] Similarly, Andersen et al.[8] documented significantly larger posterior than anterior MI-related RVIs at autopsy, accounting for 53% and 5% of the infarcted RV myocardium, respectively. Tahirkheli et al.[4] reported a 10% incidence of anterior and/or anteroapical RVI during anteroseptal MI at autopsy, involving 22% of the RV wall mass, while in three cases, the inferoapical RV wall was also involved due to occlusion of a wrap-around the apex LADA.

**Electrocardiographic considerations**

ST-segment elevation in the right precordial leads has been shown to be diagnostic of autopsy-proven posterolateral but not anterior RVI.[5,6] This has been correlated to the topography and size of infarction, in that a posterolateral RVI is larger, located near the atrioventricular groove and oriented towards the right lateral hemithorax, producing STE in the right precordial leads and sometimes in lead V1.[5,8] In contrast, anterior RVIs are small and primarily located adjacent to the apex; hence, remote from the right precordial leads.[8] Moreover, patients with anterior MI without pathologic evidence for RVI have been shown to exhibit STE in the right precordial leads.[5] The assumption that this may signify septal MI has been demonstrated by Ben-Gal et al.[2] who showed that lead V1 STE (≥1.5 mm) was strongly correlated with lead V3R STE and a small-sized conus branch during anterior MI, implying the right side of the anterior IVS (right paraseptal area) being supplied solely by septal branches of the LADA or along with the conus branch. They also assumed that detection of absence of infarction of the anterior IVS owing to the presence of a large conus branch, as shown by the absence of STE in leads V1 and V3R, may identify patients without right ventricular "steal"; hence, less left ventricular ischemic burden. Furthermore, Zhong-qun et al.[3] demonstrated that lead V3R STE of at least 1.0 mm during anterior MI was significantly more prevalent among patients with proximal than distal to the first septal branch LADA occlusion while lead V3R STE of at least 1.5 mm was predictive, with 84.0% sensitivity, of the presence of a small conus branch not reaching the IVS.

**Conclusion**

In the case presented by Akpınar et al., infarction of the apical and adjacent inferior RV wall very likely occurred owing to occlusion of a wrapped LADA. Scrutiny of the ECG presented shows at least 2 mm STE in lead V3R and at least 1.5 mm STE in lead V1. These signs are much more supportive of right paraseptal involvement due to LADA occlusion proximal to the first or major septal branch and the presence of a small conus branch rather than RVI. At least the data so far support this concept and, when taken into account, may facilitate risk stratification although further validation is needed.

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