



The Plasticity of R Wave Amplitude Due to Transmural Ischemia Triggered by Exercise Stress Test

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ABSTRACT

R wave enhancement is one of the important markers of ischemia in patients with coronary artery disease. It is usually difficult to detect all stages of transmural ischemia without myocardial infarction. Here, we described a patient exhibiting R wave enhancement in his electrocardiogram (ECG) during exercise stress test. R wave enhancement is an ECG pattern of exercise test associated with the hyperacute phase of transmural ischemia. A 42-year-old smoker male patient with a complaint of chest pain for 6 months was admitted to the outpatient clinic for further evaluation. An exercise stress test was performed for ten minutes with the Bruce protocol, and the heart rate reached 162 beats/min. We observed an R wave enhancement in leads II, III and aVF accompanied by ST segment elevation in the same leads. Moreover, reciprocal ST segment depression concurrently occurred. In the recovery period, the ischemic ECG findings reversed in the same order. In the absence of Q wave, it is very rare to observe the R wave enhancement followed by ST segment elevation. Our case report illustrates the nonspecific response of the myocardium to the triggered transmural ischemia.

Key Words: Ischemia; electrocardiography; R wave enhancement; inferior leads

Egzersiz Stres Testi ile Tetiklenen Transmural İskemiye Bağlı R Dalga Amplitüdünün Plastisitesi

ÖZET

Koroner arter hastalığı olan hastalarda R dalga artışı iskeminin önemli göstergelerinden biridir. Miyokard enfarktüsü olmaksızın transmural iskeminin tüm aşamalarını tespit etmek genellikle çok zordur. Biz burada, transmural iskeminin hiperakut fazında görülebilen bir elektrokardiyogram (EKG) paterni olan egzersiz stres testi sırasında EKG’de R dalgası amplitüdünde artış görülen bir hasta sunmaktayız. 42 yaşında sigara öyküsü olan erkek hasta 6 aydır devam eden göğüs ağrısı şikayeti ile ileri değerlendirme için polikliniğe başvurdu. Bruce protokolünde on dakika boyunca egzersiz stres testi yapıldı ve 162 atım/dk kalp atış hızına ulaştı. II, III ve aVF derivasyonlarında ST segment yükselmesi ile birlikte aynı derivasyonlarda R dalga artışı gözlemlendi. Ayrıca, aynı anda resiprok derivasyonlarda ST segment depresyonu meydana geldi. Dinlenme fazında iskemik EKG bulguları aynı sırayla düzeldi. Q dalgası yokluğunda ST segment yükselmesi ile birlikte R dalgasının belirginleşmesini gözlemlemek oldukça çok nadir bir durumdur. Bu olgumuz, miyokardın tetiklenen transmural iskemiye spesifik olmayan cevabını göstermektedir.

Anahtar Kelimeler: İskemi; elektrokardiyografi; R dalga artışı; inferior derivasyonlar

INTRODUCTION

Electrocardiography (ECG), which is the milestone modality of ischemic heart disease, has an important value both for the management and prognosis of the patients with coronary artery disease. Moreover, a newly appeared ST segment elevation is a well-accepted cardiac emergency necessitating instant reperfusion. Unfortunately, some patients with serious coronary artery stenosis still present to outpatient clinics without any sign of ischemia on ECG. Unconventional ECG signs of ischemia may improve the recognition of patients with coronary artery disease, especially in exercise stress test. Apart from ST segment elevation, R wave enhancement is one of the most important fundamental of ischemia can easily be stimulated during an exercise stress test. Here, we described a 42-year-old male patient whose exercise stress test had a very demonstrative and deductive pattern in regard to coronary ischemia.

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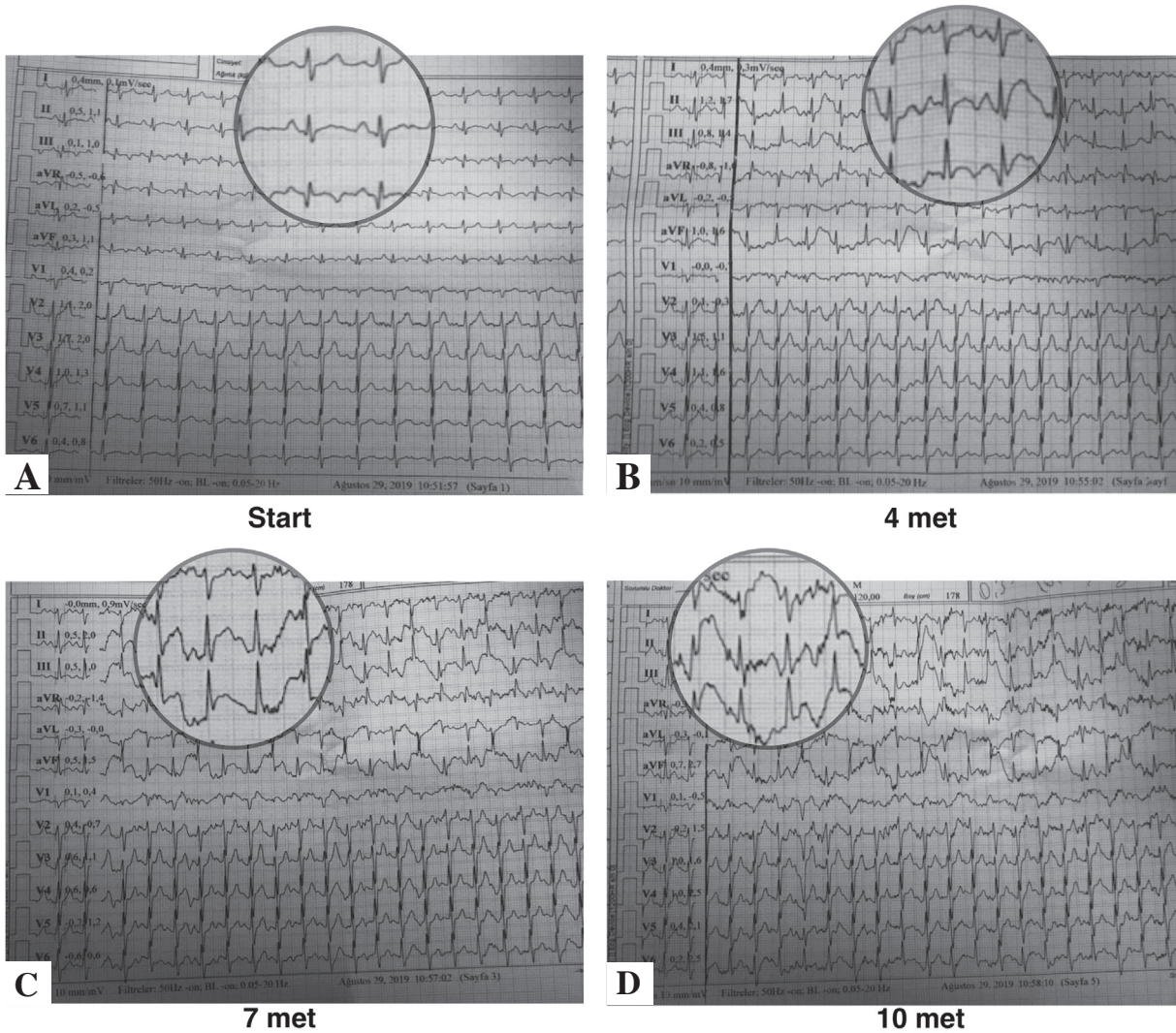


Figure 1. A. B. C. D. Showing R wave enhancement and concomitant ST segment elevation in leads II, III and aVF during the course of the exercise stress test.

CASE REPORT

A 42-year-old smoker, male patient with a complaint of chest pain for 6 months was admitted to the outpatient clinic for further evaluation. He described episodes of awakening with chest pain in the morning subsiding in less than 5 minutes. His father, with a history of diabetes and smoking, had died of a sudden cardiac arrest at 46 years of age. Physical examination, routine laboratory tests, and electrocardiography were completely normal. Transthoracic echocardiography revealed normal left ventricular systolic function with an ejection fraction of 64%. An exercise stress test was performed for ten minutes with the Bruce protocol, and the heart rate reached 162 beats/min. The patient had a desire to stop because of a burning in the throat and retro-sternum. At the recovery period, the patient developed ST segment elevation up to 3 mm concomitant with R wave amplitude enhancement, and ST segment depression

reciprocal to inferior and posterior epicardial ischemia. R wave amplitude was enhanced and already prominent while the patient was at the level of 4 MET (Figure 1). The amplitude of R waves in leads II, III and aVF were tripled at the level of 10 MET (Figure 1). ST segment elevation in leads II, III and aVF and reciprocal ST segment depression appeared in the recovery period (Figure 2). The considerable enhancement of R wave amplitude in leads II, III and aVF during exercise and decrease of the enhanced R wave amplitude in the recovery period were frankly presented in Figure 3. The patient had no symptoms of ischemia after the test, and his ECG was completely normal. He was hospitalized for further evaluation, and follow-up cardiac enzymes were negative. The patient underwent cardiac catheterization showing subtotal lesion in the proximal right coronary artery, and there was a milder disease in left anterior descending and circumflex artery (Figure 4A). The patient un-

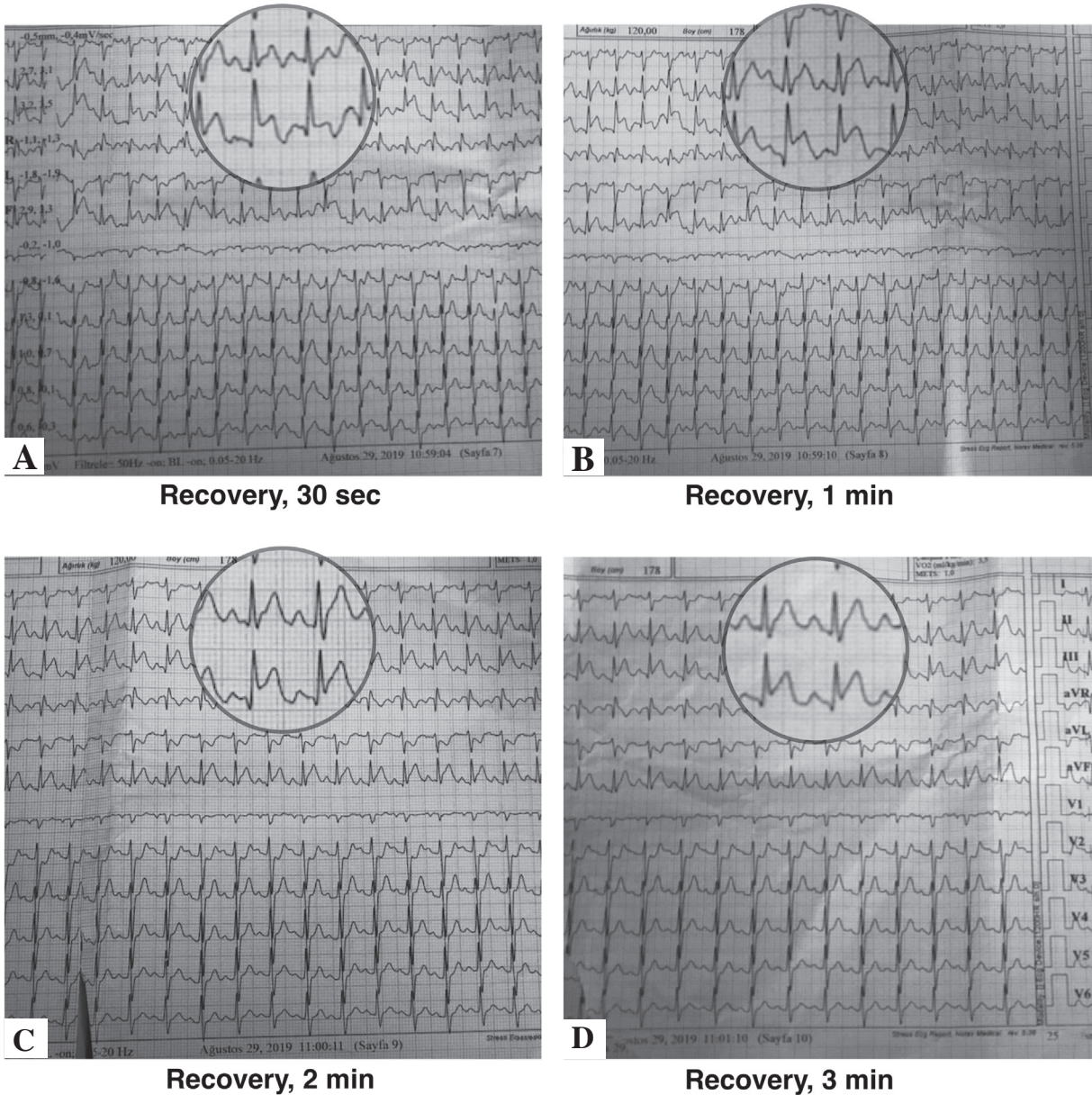


Figure 2. A. B. C. D. Showing R wave enhancement and concomitant ST segment elevation in leads II, III and aVF during the course of the exercise stress test.

derwent successful percutaneous transluminal coronary angioplasty and a drug eluting stent was successfully implanted to the culprit lesion (Figure 4B). He was discharged well on his 4th day and is currently asymptomatic in the follow-up period.

DISCUSSION

The basic indications for terminating an exercise stress test are progressively worsening of typical chest pain, ST-segment elevation in the absence of Q waves and marked ST-segment depression (> 3 mm of additional ST-segment depression compared to beginning)⁽¹⁾. All of the aforementioned indications are strong predictors of ischemic heart disease in exercise

stress test. Furthermore, similar to our case report, an increase in R wave amplitude with stress test has been proven to be an identifier of present myocardial ischemia⁽²⁾.

The augmented R waves are identified in several clinical circumstances, such as myocardial infarction, Prinzmetal angina or ischemia during exercise stress test. Thus, it is not easy to distinguish the exact etiology of the increase in R wave. Animal laboratory studies have repeatedly evaluated the effect of coronary ligations on R wave amplitude and demonstrated the enhanced R wave response to transmural ischemia⁽³⁾. Another different theoretical etiologic mechanism for R wave enhancement secondary to ischemia is the triggered left ventricular dilatation in

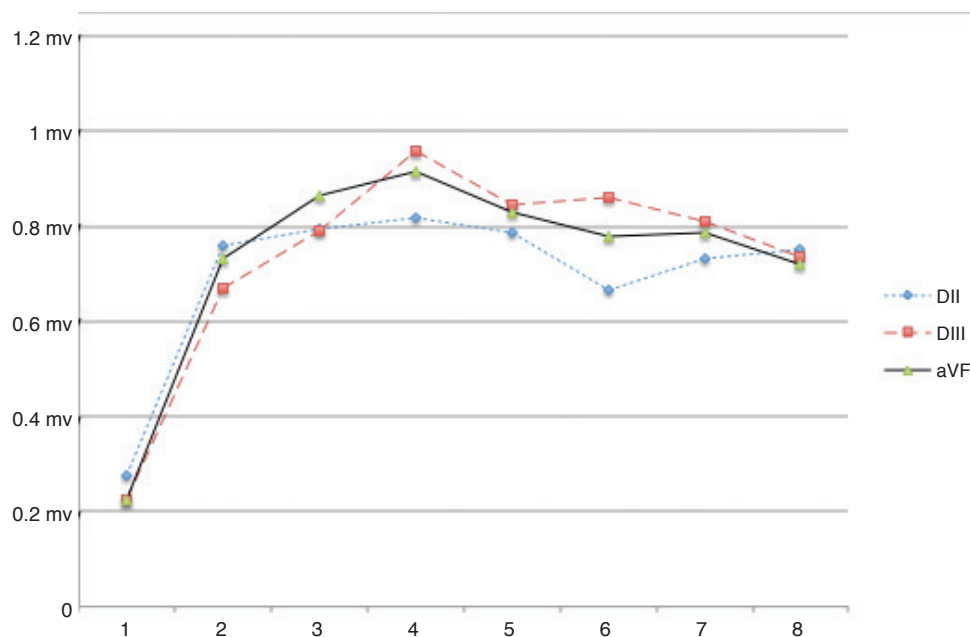


Figure 3. Showing R wave changes during the exercise stress test in leads II, III and aVF.

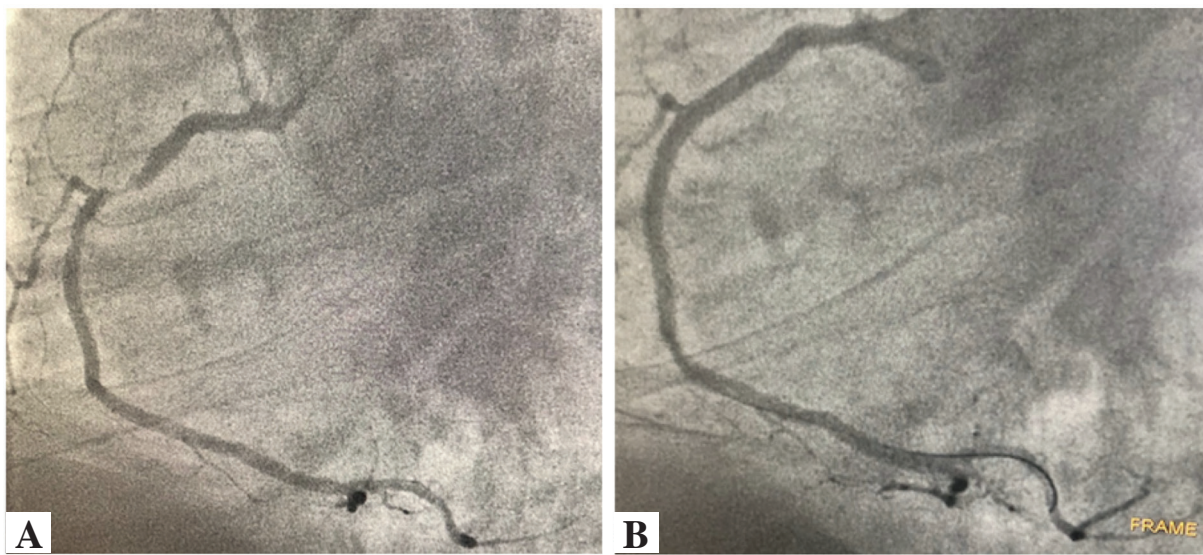


Figure 4. A. Showing a subtotal lesion in the proximal right coronary artery. B. Showing the final view of the right coronary artery after stent implantation.

the exercise stress test⁽⁴⁾. R wave enhancement in exercise stress test is also associated with ST segment depression in different cohorts⁽⁵⁾. In our patient, R wave amplitude was augmented in leads II, III and aVF throughout the exercise stress test, and it was highest in amplitude at the level of 10 MET. ST segment elevation in leads II, III and aVF and reciprocal ST depression followed up the R wave enhancement in inferior derivations. There was no Q wave due to short period of ischemia in the exercise stress test, and the patient had ischemic symptoms that would be wrong to force the patient to carry on the exercise stress test.

The plasticity of the QRS complex was presented owing to the return of its beginning state in the reverse way. From a different perspective, a short period of transmural ischemia resulted in R wave augmentation and ST segment elevation followed R wave augmentation. The ischemic duration was not sufficient to cause pathologic Q wave in our patient.

CONCLUSION

In conclusion, in an exercise stress test, it is very rare to observe R wave enhancement tracked by ST segment eleva-

tion in the absence of pathologic Q wave. This temporary ECG evolution remarks to severe transmural ischemia caused by a critically stenosed coronary artery. Our case report illustrates the nonspecific response of the myocardium to the triggered transmural ischemia.

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