Report of Two Cases with Giant Inverted T Waves and Review of the Literature

Dev T Dalgası Negatifliği Olan İki Olgu Sunumu ve Literatürün Gözden Geçirilmesi

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ABSTRACT Cardiac repolarization is detected on the surface 12-lead ECG as the T wave. In general the direction of the T wave is in the same direction with the QRS complex. In case of myocardial injury, recovery may be delayed regionally causing subendocardial muscle fibers recover first. Repolarization from endocardium to epicardium results inversion of the T waves. Although inverted T waves are constantly seen in electrocardiograms and that can be associated with myriad of pathologies or nonspecific changes, it is often a manifestation of myocardial ischemia. Giant deep inverted T waves are a rare evidence for the clinical conditions. Giant T waves can be seen with apical (Yamaguchi) variant of hypertrophic cardiomyopathy, high intracranial pressure, subarachnoid hemorrhage, severe myocardial ischemia, pulmonary embolism and in posttachycardia syndrome. We report here two cases with giant inverted T waves after myocardial infarction and discuss the potential mechanisms in the light of the literature.

Key Words: Electrocardiography; pathology

ÖZET Kardiyak repolarizasyon 12-derivasyonlu EKG'de T dalgası şeklinde ortaya çıkar. Genellikle T dalgası yönü o derivasyondaki QRS dalgası ile aynı yöndedir. Miyokard hasarı sonrası repolarizasyon, bölgesel olarak gecikerek subendokardiyal kas demetlerinin erken toparlamasına yol açar. Repolarizasyonun endokarddan epikarda doğru olması T dalgasının negatifleşmesine neden olur. T negatifliği elektrokardiyografilerde çok sık görülen,çeşitli ciddi durumlara işaret eden ya da non-spesifik olabilen sıklıkla da miyokard iskemisi ile ilişkili bir durumdur. Dev derin negatif T dalgaları ise çok daha nadir görülen bir klinik bozukluktur. Dev T dalgası hipertrofik kardiyomiyopatinin bir varyantı olan apikal hipertrofi (Yamaguchi) sendromu, yüksek intrakranial basınç, subaraknoid kanama, ciddi miyokardiyal iskemi, pulmoner emboli ve posttaşikardi sendromlarında rapor edilmiştir. Burada miyokard infarktüsü sonrası dev T negatif dalgaları olan iki olgu sunulmakta ve potansiyel mekanizmalar literatür ışığında tartışılmaktadır.

Anahtar Kelimeler: Elektrokardiyografi; patoloji

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The differential diagnoses of T-wave inversions are diverse and include ischemia, inflammation, electrolyte abnormalities, cocaine use and trauma.[1] T-wave inversion can be normal in leads aVR, III, and V1. It may be seen in young adults (juvenile T-wave pattern). T-wave inversion is also seen in anxiety states, hyperventilation syndrome and with digoxin use.[2] However, deep symmetrically inverted T waves (>5 mm), also called giant inverted T waves, are classically described in rare conditions.

CASE REPORTS

CASE 1

An 81-year-old man was admitted with a severe retrosternal chest pain which had started one hour before admission. The pain had an acute onset, progressive course and a compressing nature. History revealed tobacco use. His initial heart rate was 63 beats/min; blood pressure was 110/70 mmHg. Cardiac examination revealed normal S1, S2 and the rest of the physical examination was normal. Twelve-lead electrocardiography (ECG) showed elevated ST-waves in leads V2-V5. The diagnosis of acute anterior myocardial infarction (AMI) was concluded and the patient was admitted to catheterization laboratory for primary percutaneous coronary intervention (PCI). Coronary angiogram showed total occlusion of left anterior descending (LAD) artery. Reperfusion was stored with LAD artery stenting. After PCI, ECG showed slight ST-wave elevation in leads V2-3 and T wave inversion in leads V2-6, D1, D2 and aVL. His hospital stay was uneventful and he was discharged with ACE inhibitor, beta-blocker, aspirin and clopidogrel therapy. Two weeks later, at his follow-up in outpatient clinic, his ECG revealed bradycardia and giant T wave inversion in the same leads at discharge (Figure 1). He had complaint of dizziness. Cardiac enzymes and electrolytes were normal. Echocardiography revealed anterior hipokinesia, an ejection fraction of 45% and mild mitral regurgitation. Cranial tomography was normal except cerebral atrophy with enlargement of subarachnoid space. Beta-blocker treatment was cancelled and he had no complaints at his first month follow-up visit.

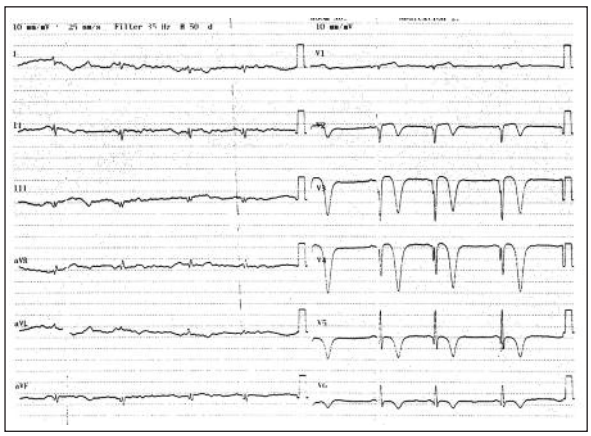

FIGURE 1: First case's ECG at second admission showing bradycardia and giant T wave inversion in leads V2-V6.

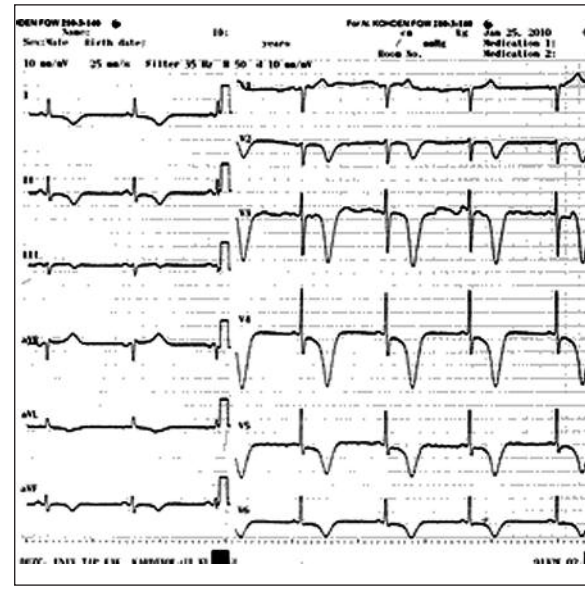

FIGURE 2: Second case's ECG on the day after admission showing giant T wave inversion especially in leads V2-V5.

CASE 2

A 77-year-old man was admitted with a severe retrosternal chest pain, which had resolved ten minutes after the onset. The pain had an acute onset and a compressing nature. He had hypertension and was using antihypertensive medication during the last twenty years. His initial heart rate was 74 beats/min; blood pressure was 120/80 mmHg. Cardiac examination revealed normal S1, S2 and the rest of the physical examination was normal. ECG in the emergency room was inconclusive. The patient was hospitalized for acute coronary syndrome. Cardiac enzymes and electrolytes were normal. Echocardiography revealed normal left ventricular function and an ejection fraction of 60%. Aspirin, beta-blocker, clopidogrel and enoxaparin treatment was started. ECG on the day after admission revealed T wave inversion in all leads except aVR and V1 (Figure 2). The patient was admitted to catheterization laboratory for invasive treatment. Coronary angiogram showed 70% stenosis in the proximal

part and a myocardial bridge causing 50% stenosis in the middle part of the left anterior descending artery and 80% stenosis in the proksimal part of the intermediary artery. Coronary artery bypass grafting was recommended, however the patient refused the operation. He had no pain during the hospital stay and serial cardiac enzymes were within normal limits. On the second day of admission he had complaint of acute onset headache. Due to co-incidental giant T wave inversion again cranial tomography was performed which was normal. He was discharged on aspirin, clopidogrel, ace inhibitor and beta-blocker treatment.

DISCUSSION

Conditions usually associated with deep symmetrically inverted T waves (>5 mm) include left ventricular apical hypertrophy, raised intracranial pressure, anesthesia induction and myocardial ischemia.[3]

ECG findings of apical hypertrophic cardiomyopathy (HCM) are most clearly reflected by large deep inverted T-waves in the precordial leads, particularly in V4 and V5.[4] Prominent T waves are also seen in cerebrovascular accidents especially in subarachnoid hemorrhage. T waves are diffuse with a widely splayed appearance with a prolonged QT interval. The proposed pathogenesis is cardiomyocytolysis from excessive sympathetic stimulation. Similar changes are reported after truncal vagotomy, bilateral carotid endarterectomy.[2] In a study conducted in Turkey, the prevalence of prominent T wave inversion was reported to be 23.6%.[5]

Some patients may develop strongly negative T waves following anesthesia or maxillofacial surgery.[6] The T wave returns to normal approximately 4 months later.

The most common cause of very deeply inverted T wave is possibly myocardial ischemia. Deep T-wave inversions are described in subendocardial infarction and occur due to regional delay in ventricular repolarization.[7] T-wave inversion is frequently seen after acute myocardial infarction and persist for days to weeks. Delayed normalization is observed mainly in patients with ischemic myocardium who have undergone revascularization. Persistent negative T waves correspond to either extensive necrosis or non-revascularized, jeopardized myocardium.[8] Nakajima et al reported T wave inversion in 98% of their study group after reperfusion.[9] They also showed that the deeper the negativity of the T waves recorded, the greater is the effectiveness of reperfusion of the myocardium.

In our first case, the cause of giant T wave inversion was possibly the effectively reperfused myocardium. A silent cerebrovascular accident was excluded with cranial tomography. Apical hypertrophic cardiomyopathy was also excluded with echocardiography. In the light of this case, we conclude that giant inverted T waves (>20 mm) can be seen normally several weeks after successful reperfusion of a large amount of jeopardized myocardium. Clinicians should exclude the most common causes, taking into account that it is a benign finding after successful treatment of acute myocardial infarction.

In our second case, the cause of giant T wave inversion was possibly the large area of ischemic myocardium. The change of ECG was dramatic, from almost normal to giant T wave inversion especially on chest leads. He might possibly have a transient spasm at the site of the left anterior descending artery stenosis, which caused transient ischemia later causing T wave inversion. Alegría et al have studied 73 patients with anterior T wave inversion and showed that all of them had an LAD involvement and in 5% of the cases the reason was due to non-atherosclerotic alterations (milking or spasm).[10]

In conclusion, giant T waves may be seen infrequently, some of the differential diagnosis being potentially fatal. Clinicians should be vigilant about taking a concise history and directing the evaluation of the patient based on his/her symptoms.

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