Turkish Journal of Medical Sciences

Volume 35 | Number 2 Article 11

1-1-2005

Transient Silent Ischemia after PTCA Manifested with a Bizarre ECG

HAKAN ÖZHAN

RAMAZAN AKDEMİR

SADIK DURAN

MEHMET YAZICI

HÜSEYİN ARINÇ

See next page for additional authors

Transient Silent Ischemia after PTCA Manifested with a Bizarre ECG

Authors
HAKAN ÖZHAN, RAMAZAN AKDEMİR, SADIK DURAN, MEHMET YAZICI, HÜSEYİN ARINÇ, HÜSEYİN GÜNDÜZ, and CİHANGİR UYAN

Turk J Med Sci
35 (2005) 127-130

SHORT REPORT

Transient Silent Ischemia after PTCA Manifested with a Bizarre ECG

Hakan ÖZHAN[1], Ramazan AKDEMİR[1], Sadık DURAN[1], Mehmet YAZICI[1], Hüseyin ARINÇ[2], Hüseyin GÜNDÜZ[2], Cihangir UYAN[2]

[1]Department of Cardiology, Düzce Faculty of Medicine, Abant İzzet Baysal University, Düzce - Turkey

[2]Department of Cardiology, İzzet Baysal Faculty of Medicine, Abant İzzet Baysal University, Bolu - Turkey

Received: July 16, 2004

Key Words: silent ischemia, coronary spasm

Silent myocardial ischemia (SMI) is defined as a transient alteration in myocardial perfusion in the absence of chest pain or the usual anginal equivalents. SMI is recognized as a common manifestation after percutaneous coronary interventions (PCIs) (1-2). PCIs can induce coronary artery spasm that causes ischemia (2). As catheterization laboratory procedures have become progressively more invasive with multivessel interventions, prolonged balloon inflation times, use of multiple guiding catheters and the advent of coronary stenting, the propensity for PCI-induced spasm has increased. We report a patient who had a brief period of intense epicardial ischemia after percutaneous transluminal coronary angioplasty (PTCA), possibly due to coronary artery spasm. Although this clinical picture is not unusual, the authors find the ECG changes during SMI very exceptional.

Case Report

A 54-year-old male patient was admitted to the emergency clinic with a complaint of intense chest pain for the previous 4 h. His arterial blood pressure was 110/70 mmHg and pulse was 74/min on admission. Physical examination revealed normal findings. Electrocardiogram demonstrated ST segment elevation in leads V1 to V4. He was diagnosed with acute anterior myocardial infarction (MI) and primary PTCA was decided on. He was administered 300 mg of aspirin, 300 mg of clopidogrel and 6.25 mg of captopril p.o. in the emergency ward. His coronary angiogram showed occlusion of the left anterior descending (LAD) artery in the proximal part (Figure 1a). He had no significant stenosis in the circumflex or right coronary arteries. Primary PTCA was performed without any complications and a 3.0 x 15 mm Ephesos (Nemed Corp., İstanbul, Turkey) stent was implanted. There was TIMI III flow without any residual stenosis in the infarction related artery (Figure 1b). Subsequently, 50 mg of metoprolol, 75 mg of clopidogrel, 300 mg of aspirin, 18.75 mg of captopril and 10 mg of simvastatin daily p.o. were started. Intravenous heparin was also administered continuously. The dose was adjusted according to the activated clotting time to be 2-3 times the normal values. Control ECG early after PTCA demonstrated (-) T waves and rS forms in leads V1-5 (Figure 2). On the second routine ECG, recorded 18 h after the procedure placing the precordial electrodes on exactly the same place on the chest of the patient, he had bizarre, notched large (-) T waves in the V1-6, D3 and aVF leads (Figure 3). He did not have any chest pain at that time and no medication was administered. Intensive care unit staff were unable to appreciate the importance of the changes in the patients ECG and the control ECG could only be recorded several hours later. The findings were much like the first one (Figure 4). The patient was discharged 7 days later without any other in-hospital adverse event. He had no complaints on his first-month visit.

SMI is a common manifestation after PCI (1-2). SMI can be classified into 3 groups: type A, totally asymptomatic patients with no history of angina or MI;

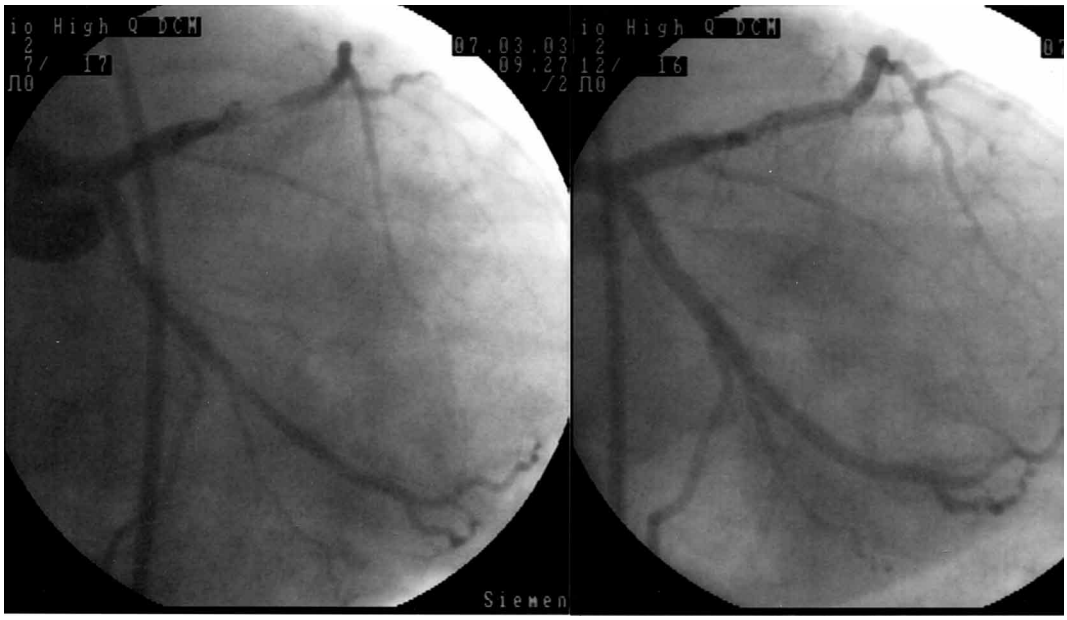

Figure 1a. Coronary angiogram shows occlusion of the left anterior descending artery in the proximal part (left panel).

Figure 1b. Restored TIMI III flow without any residual stenosis in the infarction related artery after PTCA/ stenting.

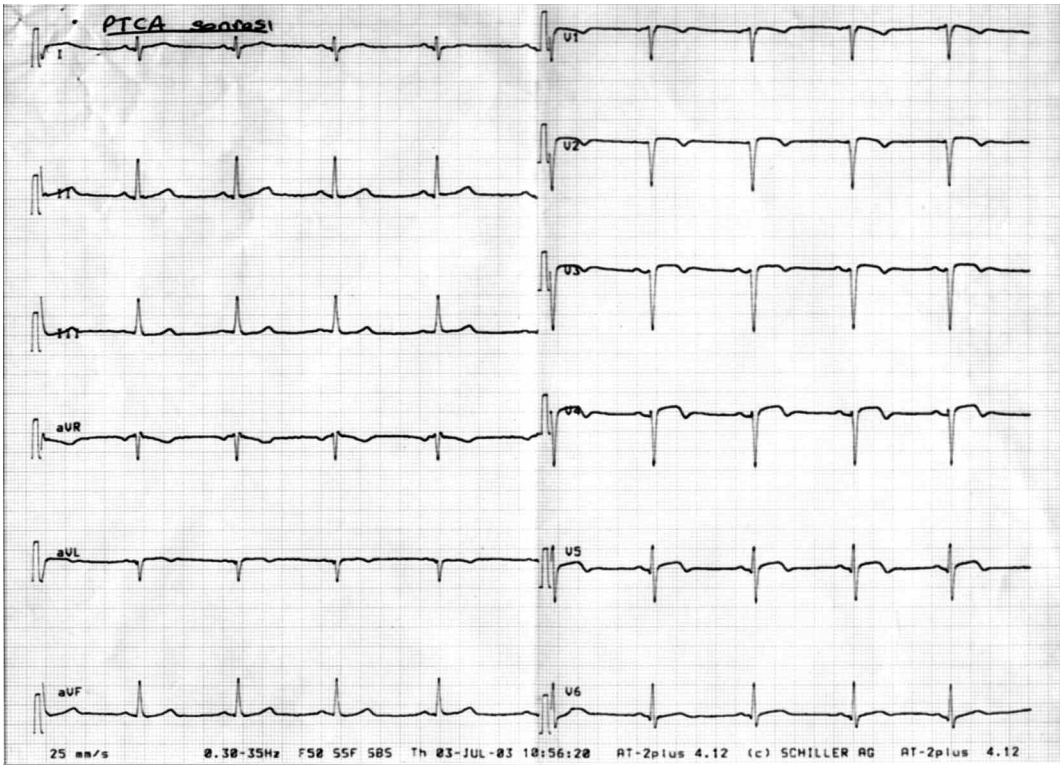

Figure 2. The ECG recording early after the PTCA procedure.

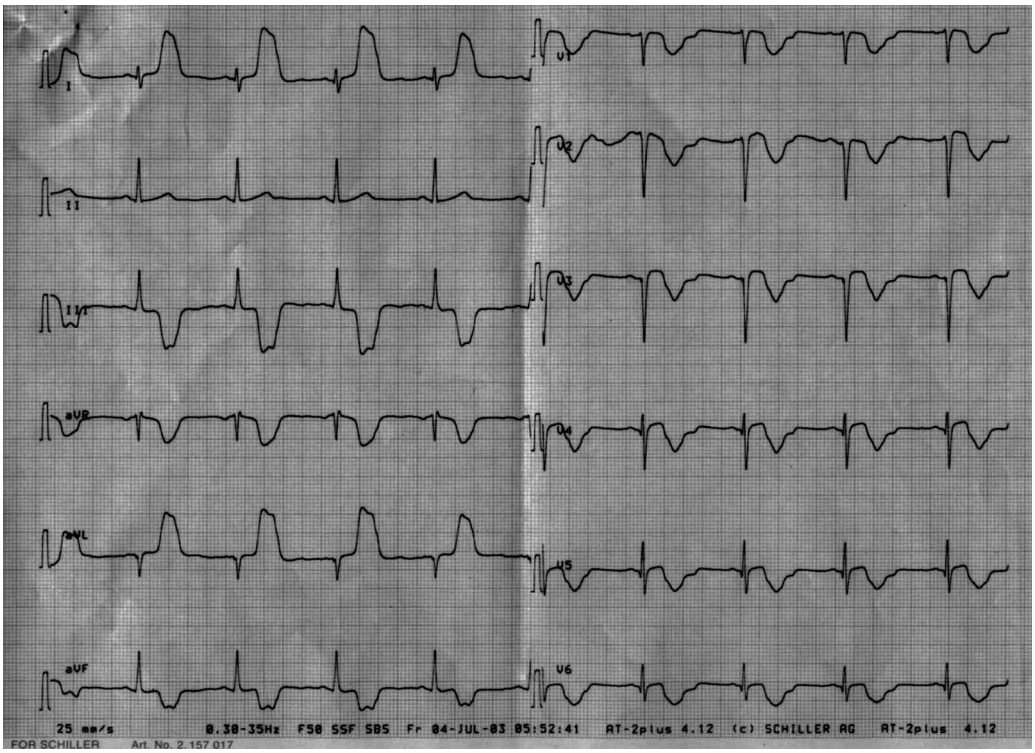

Figure 3. Bizarre, large-notched inverted T waves in V1-6, D3 and aVF leads recorded routinely 18 h after the procedure while the patient was asymptomatic.

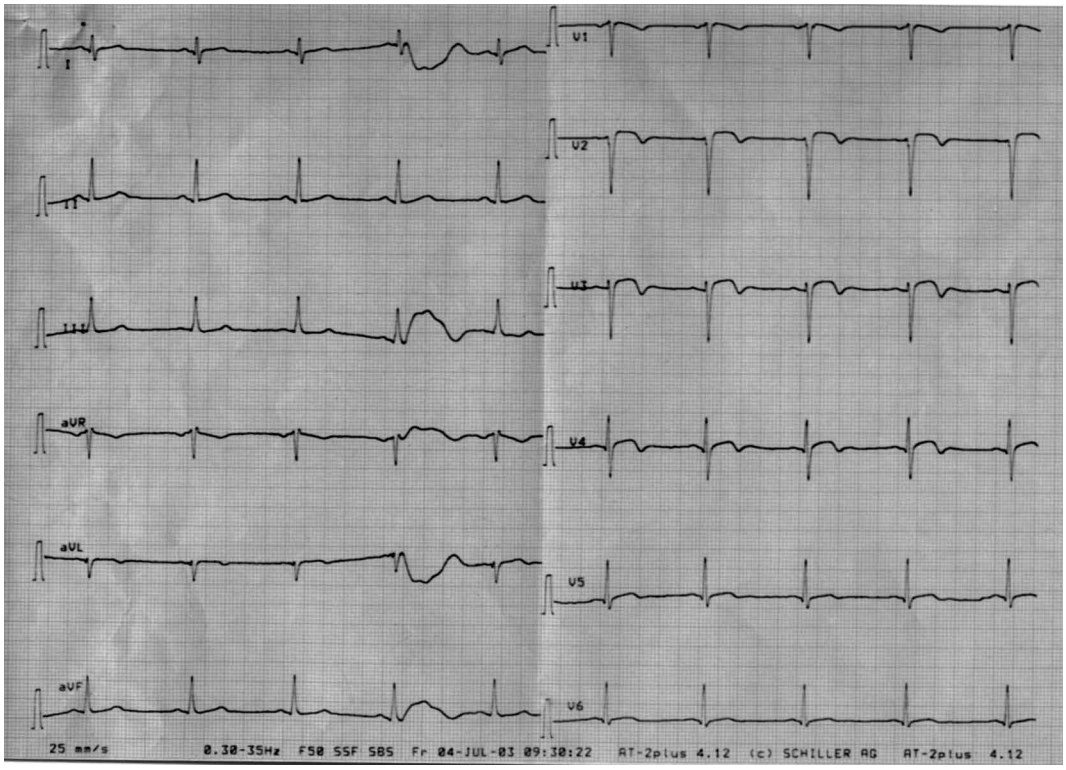

Figure 4. Control ECG demonstrating inverted T waves and rS forms in V1-5 leads.

type B, asymptomatic patients with previous MI; and type C, patients with angina and asymptomatic ischemic episodes (3). SMI has been found in 2.5% of all healthy males aged 40-59 and in 20% of all postinfarction patients. A study by Mizutani et al. revealed that the frequency of SMI during PCI is 16% and this proportion did not differ in clinical or angiographic groups except for diabetes mellitus patients, 60% of whom had silent ischemia (2). Arnim et al. demonstrated that 93% of the patients who were followed up in the coronary care unit with acute coronary syndrome and post-PTCA had pathologic changes on a portable ECG computer, which monitors the 12-lead ECG every 20 s, and 82% of the recorded episodes of transient ischemia were silent (1). SMI patients have generally reduced sensitivity to pain, felt during ischemic episodes. Seemingly healthy asymptomatic patients and patients with coronary artery disease are all at higher risk of subsequent cardiovascular morbidity if there is evidence of silent ischemia (4).

Coronary spasm plays an important role in the pathogenesis of SMI (5). Gentle rubbing of large vessels with a blunt instrument or balloon can result in nearly complete endothelial denudation (6). Catheter-induced coronary spasm does not require extensive intra-coronary manipulation (7). Endothelial dysfunction may contribute to but is not necessary for the development of coronary vasospasm. Instead pathologic changes in the underlying vascular smooth muscle are most likely to be etiologically involved in the vasospastic process (8).

Ischemia predominantly causes ST-T wave changes. In our patient, transient SMI was possibly caused by a spasm in the very proximal LAD (the area in which the stent was implanted). The bizarre ECG changes (large- notched inverted T waves) were thought to be associated with a specific pattern of abnormal left ventricular motion (9). Echocardiography is a valuable tool showing contraction abnormalities and would have strengthened this assumption if it could have been performed in this patient during the ECG changes. Another possibility is the transient occlusion of one of the diagonal branches of the LAD artery, since there was electrocardiographic evidence of ST wave elevation in the DI and aVL leads during SMI. Angiographic control could not be performed during ECG changes.

The authors think that although the clinical picture of the present case is frequent in clinical practice, the ECG findings during SMI are unique. Finally, it should be noted that ischemic episodes, whether silent or asymptomatic, are quite common after coronary interventions. Screening patients with computerized systems at least in the first 24 h after PCI can help to detect and prevent coronary adverse events.

Acknowledgment

The authors thank J. Willis Hurst, MD, MACP, Chairman of Cardiology, Emory University School of Medicine, for his contribution to the article.

Corresponding author:
Hakan ÖZHAN
Department of Cardiology,
Düzce Faculty of Medicine,
Abant İzzet Baysal University,
81620 Konuralp, Düzce - Turkey
E-mail: ozhanhakan@yahoo.com

References

1. Von Arnim T, Reuschel-Janetschek E. Continuous bedside monitoring of the ECG for detection of silent myocardial ischaemia. Coron Artery Dis 7: 535-40, 1996.
2. Mizutani M, Ben Freedman S, Barns E et al. ST monitoring for myocardial ischemia during and after coronary angioplasty. Am J Cardiol 66: 389-93, 1990.
3. Loskot F, Novotny P. Asymptomatic myocardial ischemia. Z Gesamte Inn Med 45: 370-73, 1990.
4. Mahmarian JJ, Pratt CM. Silent myocardial ischemia in patients with coronary artery disease. Possible links with diastolic left ventricular dysfunction. Circulation 81: Suppl: III, 33-40, 1990.
5. Kawano H, Ogawa H. Endothelial dysfunction and coronary artery spasm. Eur Heart J 9 Suppl. 9: 89-92, 1988.
6. Brum JM, Sufan Q, Lane G et al. Increased vasoconstrictor activity of proximal coronary arteries with endothelial damage in intact dogs. Circulation 70: 1066-1073, 1984.
7. Moukarbel GV, Dakik HA. Diffuse coronary artery spasm induced by guidewire insertion. J Invasive Cardiol 15: 353-354, 2003.
8. Konidala S, Gutterman DD. Coronary vasospasm and the regulation of coronary blood flow. Prog Card Dis 46: 349-373, 2004.
9. Malfatto G, Beria G, Sala S et al. Quantitative analysis of T wave abnormalities and their prognostic implications in the idiopathic long QT syndrome. J Am Coll Cardiol 23: 296-301, 1994.