Release of cardiac troponin-T following successful percutaneous transluminal coronary angioplasty

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Cardiac troponin T (cTn-T) measurements were studied in 34 patients who underwiew successful percutaneous transluminal coronary angioplasty (PTCA) for defining myocardial damage during procedure. Blood samples were drawn before and between 7 and 14 h (mean of 10 h) following PTCA. Before PTCA, high cTn-T levels were found in 8 of 14 patients with stable and 15 of 20 patients with unstable angina pectoris. None of the patients had significant increases in cTn-T after uncomplicated PTCA (1.0±1.9 ng/ml; 2.2±5.2 ng/ml, p<0.05). In conclusion, successful PTCA does not cause myocardial damage irrespective of clinical stability. [Turk J Med Res 1994; 12(5): 222-223]

Key Words: Coronary angioplasty, Troponin T, Angina pectoris

Percutaneous transluminal coronary angioplasty (PTCA) is established as a common technique for myocardial revascularization. There is a controversy whether PTCA causes myocardial damage or not (1-3). Up to 20% of patients who undergo this procedure have mild elevations in creatine phosphokinase-MB (1,2,4). It is traditionally used to identify myocardial injury in clinical practice. Myoglobin is also an early and sensitive indicator of myocardial damage but not specific (5). Recently some studies suggested cardiac troponin T (c-Tn T) which had different amino acid sequence of protein in skeletal muscle, was sensitive and specific for myocardial injury (6-11).

In this study, using cTn-T, myocardial injury was evaluated during successful PTCA.

MATERIALS AND METHODS

The study population was consisted of 34 patients (27 men and 7 women, median age years; range 34 to 70) who were undergoing elective PTCA for stable (in 14 patients) or unstable (in 20 patients) angina pectoris. Of all patients, 12 had myocardial infarction at least 2 months ago.

Coronary angioplasty was made by Judkins technique from right femoral artery. Before angioplasty procedure, a standard drug treatment (diltiazem 90-180 mg and aspirin 300 mg, daily) was instituted within 48 hours. Patients were fully heparinized during procedure. A total of 41 stenosis were subjected to PTCA. Fifteen patients had stenosis in the left anterior descending coronary artery, 5 in the left circumflex artery, 7 in the right coronary artery, 3 in the left anterior descending and right coronary arteries, 2 in the left anterior descending and circumflex and 2 in the left circumflex and right coronary arteries.

The degree of stenotic lesions was between 80% and 100%. The angioplasty balloon was inflated at 6 to 8 atmospher. An average inflation duration was 60-90 ms and the average two inflations were used in each lesions. There were not any complications during procedure. After balloon deflation no patient complained of chest pain or had ST segment-T wave changes on electrocardiography or signs of a new myocardial infarction. Residuel stenosis were below 30%. All patients had successful PTCA.

Blood samples were withdrawn before and between 7 and 14 h (mean of 10 h) following the angioplasty procedure. cTn-T concentrations were measured by the enzyme immunoassay (Boehringer Mannheim, Germany). The upper limit of cTn-T was accepted 0.5 ng/ml in normal healthy group.

Statistics

All data was reported as mean±SD. Student's t test and Chi-Square test were used to test differences between variables.

RESULTS

A total of 41 stenosis in 34 patients was succesfully dilated by PTCA. In all patients, cTn-T concentrations showed in significant changes after PTCA. The results were given in Table 1.

cTn-T measurements were above 0.5 ng/ml in 8 (57%) patients with stable angina pectoris, but 15 (75%) with unstable angina pectoris (p>0.05). In comparison of stable and unstable angina groups, the mean values of cTn-T were not statistically different before and after procedure (p>0.05; p>0.05).

DISCUSSION

In the last several decades, serum levels of cardiac enzymes and isoenzymes have become the final arbiters

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Table 1. cTn-T measurements before and after PTCA

PTCA	cTn-T ng/ml before	after	P
Stable angina n=14	0.6±0.3	2.5±6.7	>0.05
Unstable angina n=20	1.2±2.5	2.0±3.9	>0.05
Total n-34	1.0+1.9	2.2±5.2	>0.05

by which myocardial damage is diagnosed or excluded. Because conventionally used enzymes are neither perfectly sensitive nor specific, there is need for a new sensitive and cardiospecific marker of myocardial damage (7). cTn-T measurements are highly sensitive and specific in diagnosis of myocardial injury (6-15).

PTCA is widely used to dilate stenosis of coronary arteries. After PTCA, cTn-T Is a sensitive marker for determining wheather myocardial injury is present or not. In uncomplicated PTCA patients, Tn-T does not increase in serum (16). But angiographically visible occlusion of smaller side branches, although not accompained by ST segment changes or chest pain, leads to an increase in cTn-T above normal range. ReocclıHon of a successfully dilated stenosis causes a marked rise in cTn-T. The extent of myocardial damage after PTCA can be estimated by cTn-T measurement (16). In another study, troponin measurement was applied to detect myocardial injury during PTCA and it was found that uncomplicated PTCA did not cause significant rise in Tn-T levels (17).

Both of these studies consisted of patients with stable and unstable angina pectoris, and no significant myocardial damage was seen as a result of successful PTCA irrespective of the stability of the coronary artery disease (16,17). There is also some studies that clinically and angiographically successful PTCA can cause the elevation of cTn-T, indicating myocardial damage (2,4).

Our result suggested uncomplicated PTCA was not associated with myocardial injury as they were confirmed by others (16,17). Before PTCA, the high cTn-T concentrations in stable and unstable angina pectoris were interesting finding. It is known up to 50% of patients with unstable angina have increased cTn-T concentrations (9,10,13,15) and it may be a useful prognostic myocardial necrosis (7,10). Because of its release kinetics the early and late diagnosis of myocardial damage can be made by this measurement (6,7,18). In our knowledge, the high cTn-T serum levels have not been showed in stable ang'na pectoris The cumulative effects of recurrent brief ischemic- attacts may be responsible from micro and patchy myocardial damage (19). Additionally, stable angina pectoris is a cl'nic diagnosis and asymptomatic, silent ischemia ftyav be effective an increased cTn-T measurements M is suggested that biochemical marker release may or may not reflect irreversibl ischemia (4,9). A gradual release of Hytosdlic enzymes from reversibly injured myocardial cells was showed after short periods of coronary occlusion without myocardial necrosis (20).

In concluvior. increased cTn-T in serum can identify a subgroup of ischemic heart disease patients who have serious p'onlosis p.nd may consider revascularization as soon as po^ibie. If PTCA is applied successfully, no myocardial damage is occurred irrespective of clinical stability.

Başarılı perkutan transluminal koroner anjioplasti sonrası kardiyaktroponin-T salınımı

Başarılı perkutan transluminal koroner anjioplasti (PTKA) sırasında, miyokard hasarı olup olmadığının değerlendirilmesi amacı ile 34 hastada kardiyak troponin T (kTN-T) ölçümleri yapıldı. Kan örnekleri anjioplastiden önce ve 7-14 saat (ortalama 10 saat) sonra alındı. PTKA öncesi, 14 stabil anginalı hastanın 8'inde, unstabil anginası olan 20 hastanın 15'inde kTn-T seviyeleri yüksek bulundu. Komplikasyonsuz PTKA sonrası, kTn-T hiçbir hastada anlamlı artış göstermedi (1.0±1.9 ng/ml, 2.2±5.2 ng/ml; p>0.05). Başarılı PTKA'nin, klinik stabiliteden bağımsız olarak, miyokart hasarına yol açmadığı sonucuna varıldı. [Turk J Med Res 1994; 12(5): 222-223]

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