

The frequency of silent myocardial ischemia associated with femoral sheath removal and hemostasis after percutaneous coronary intervention: evaluation with 12-lead ST-segment monitoring

Perkütan koroner girişim sonrası femoral kılıf çekilmesi ve hemostaz ile ilişkili sessiz iskemi sıklığı: 12 derivasyonlu ST-segment izlemiyle değerlendirme

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Objectives: Silent myocardial ischemia (SMI) is the objective documentation of ischemia in the absence of angina or anginal symptoms. We aimed to determine the frequency of SMI before and after sheath removal (SR) following elective percutaneous coronary interventions (PCI).

Study design: Sixty-six patients (51 men, 15 women; mean age 59.5±10.3 years) were prospectively monitored with 12-lead ST monitoring after elective PCI for six hours. Transient ischemic episode was defined as the detection of transient ST-segment shift of at least 1 mm and lasting for at least 1 min in any lead. The monitoring period was divided into three intervals: two hours before and after SR, and the last two hours. The number of SMI episodes and maximal ST-segment changes were calculated for each interval.

Results: Throughout monitoring, SMI was detected in 32 patients (48.5%), during which 44, 121, and 65 SMI episodes were recorded and 11 (16.7%), 20 (30.3%), and 1 (1.5%) patients exhibited *de novo* SMI episodes in two hours before and after SR, and the last two hours, respectively. The number of patients with SMI was significantly greater in the first two hours after SR compared to two hours before SR ($p<0.001$) and the last two hours ($p=0.022$). Moreover, the number of SMI episodes per patient was significantly greater in this period (1.8±3.8) compared to the period before SR (0.7±2.4, $p<0.001$) and the last period (1.0±3.0, $p<0.001$). Maximum ST-segment shifts were significantly greater in both the first and second two hours after SR compared to the period before SR (0.82±0.30 mm and 0.77±0.36 mm vs. 0.68±0.32 mm; $p<0.001$ and $p=0.008$, respectively).

Conclusion: Our data indicate that SMI occurs more frequently during the early hours after SR in patients undergoing elective PCI.

Key words: Angioplasty, balloon, coronary; electrocardiography/methods; myocardial ischemia/diagnosis; prevalence.

Amaç: Sessiz miyokart iskemisi (SMİ), angina veya angina eşdeğeri semptomlar yokken iskeminin nesnel olarak gösterilmesidir. Bu çalışmada, elektif perkütan koroner girişim (PKG) uygulanan hastalarda kılıf çekilmesi (KÇ) öncesi ve sonrasında SMİ sıklığı araştırıldı.

Çalışma planı: Elektif PKG uygulanan 66 hasta (51 erkek, 15 kadın; ort. yaş 59.5±10.3) işlem sonrasında ileriye dönük olarak 12 derivasyonlu ST-segment izlem aygıtıyla altı saat takip edildi. Geçici iskemik atak, herhangi bir derivasyonda en az 1 mm ve en az 1 dk süreli ST-segment değişikliği olması olarak tanımlandı. İzlem dönemi, KÇ öncesi ve sonrası iki saat ve son iki saat olarak üç bölüme ayrıldı. Her bir dönem için SMİ atağı sayısı ve en büyük ST-segment değişiklikleri hesaplandı.

Bulgular: İzlem süresince 32 hastada (%48.5) SMİ atağı saptandı. Kılıf çekilmesi öncesi ve sonrası iki saatlik dönemlerde ve son iki saatte sırasıyla 44, 121 ve 65 SMİ atağı kaydedilirken, aynı dönemlerde yeni SMİ atağı görülen hasta sayısı sırasıyla 11 (%16.7), 20 (%30.3), and 1 (%1.5) idi. Kılıf çekilmesi sonrası ilk iki saatte SMİ atağı saptanan hasta sayısı, KÇ öncesi ($p<0.001$) ve son iki saatlik ($p=0.022$) dönemlerden anlamlı olarak daha fazlaydı. Aynı dönemde hasta başına düşen SMİ atağı sayısı (1.8±3.8) da KÇ öncesi (0.7±2.4, $p<0.001$) ve son iki saatlik (1.0±3.0, $p<0.001$) dönemlerden fazla bulundu. Kılıf çekilmesi sonrası ilk iki ve son iki saatlerdeki en büyük ST-segment değişiklikleri de KÇ öncesi döneme göre anlamlı olarak daha fazlaydı (sırasıyla 0.82±0.30 mm, 0.77±0.36 mm ve 0.68±0.32 mm; $p<0.001$ ve $p=0.008$).

Sonuç: Bulgularımız, elektif PKG uygulanan hastalarda KÇ sonrası erken dönemde daha sık SMİ geliştiğini göstermektedir.

Anahtar sözcükler: Anjiyoplasti, balon, koroner; elektrokardiografi/yöntem; miyokart iskemisi/tanı; prevalans.

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Silent myocardial ischemia (SMI) is the objective demonstration of ischemia in the absence of angina or anginal symptoms.^[1] It can be detected by means of several methods. ST-segment monitoring (STSM) is one of the most common methods used to determine SMI. Although different lead configurations have been used, 12-lead STSM is currently recommended as the most accurate way to detect SMI.^[2,3] Albeit not mandatory for stable patients, cardiac monitors can be equipped with ST monitoring in the postprocedure unit and STSM can be activated in the immediate postintervention period and continued for 4 to 8 hours after uncomplicated elective percutaneous coronary interventions (PCI).^[4] ST-segment monitoring may be useful to detect abrupt reocclusion and distinguish ischemic from nonischemic chest pain.^[5] In addition, ST-segment changes after PCI may identify high risk patients.^[6]

The purpose of this study was to determine the frequency of SMI using 12-lead STSM in patients undergoing successful elective PCI.

PATIENTS AND METHODS

Patients. The study was conducted between March 2005 and January 2006 in 66 patients (51 males, 15 females; mean age 59.5 ± 10.3 years) who underwent elective PCI by different operators and followed-up in the coronary care unit (CCU) for the development of SMI. Patients who met all of the following conditions were considered eligible for inclusion: (i) elective PCI of one or more vessels and follow-up in the CCU; (ii) removal of femoral artery sheaths by physicians two hours after PCI; (iii) uncomplicated, successful PCI; (iv) absence of glycoprotein IIb/IIIa antagonist administration before or after intervention. Patients were excluded if they had any of the following: (i) left ventricular hypertrophy, conduction defects, or any other condition that may interfere with interpretation of ST-segment deviation; (ii) atrial arrhythmias, sinus bradycardia, or bundle branch block; (iii) atrioventricular nodal block, sick sinus syndrome, or an implanted pacemaker; (iv) uncontrolled hypertension or hypotension; (v) left ventricular aneurysm; (vi) primary PCI due to acute myocardial infarction; (vii) chronic renal failure; (viii) mean heart rate >100 beat/min during follow-up; (ix) a psychological problem.

Demographic and clinical characteristics of the patients and technical features of PCI were recorded. Coronary artery stenosis was evaluated with quantitative angiographic measurements and $\geq 70\%$ stenosis ($\geq 70\%$ luminal diameter narrowing) was accepted as

a significant lesion. An intravenous bolus of 100 IU/kg unfractionated heparin was administered immediately before PCI. The study protocol was approved by the local ethics committee and all the patients gave written informed consent.

Definition of silent ischemia. Transient ischemic episode was defined as transient ST-segment depression or elevation, in any lead, of at least 1 mm (0.1 mV) compared to the baseline, lasting for at least 1 min. An interval between the episodes by at least 1 min was regarded to show two separate episodes.^[5]

ST-segment monitoring. A baseline electrocardiogram was obtained before the procedure. Then, the electrodes were placed according to the standards of the American Heart Association for electrocardiographic monitoring.^[4] All the patients were continuously monitored with the 10-electrode connection cable (MultiMed 12 pod, Siemens Medical Solutions, Danvers, USA) and the modified standard 12-lead ECG system (Siemens SC 8000, Siemens Medical Solutions). ST-segment analysis was performed using the ARIES (Advanced Review of Ischemia Event System) bedside monitoring system and software. The patients were planned to be monitored for approximately six hours. The electrodes were not removed during the whole monitoring period. To control for the possible effects of body position on the ECG, patients were placed in the supine position. ST-segment changes were measured 60 msec after the J point to reduce the effect of heart rate.^[5] At the beginning of monitoring, the baseline ST-segment levels for each of the 12 leads were determined as the reference points. ST-segment alarm limits were set as ± 1 mm (0.1 mV) changes from the reference ST levels. The ARIES software provides quantitative ST measurements for each lead and graphical demonstration of ST-segment, blood pressure, and heart rate. This software continuously analyzes and stores ST-segment changes for each lead per minute (Fig. 1). All the reports stored on a computer were printed after monitoring. False alarms triggered by poor signal quality or transient arrhythmias were reviewed on these reports and eliminated. Total monitoring period was divided into three intervals; the first two hours until sheath removal (SR), the second two hours for hemostasis, and the remaining two hours. Time to hemostasis was accepted as the time interval from the removal of the femoral sheath until applying a bandage. The number of ischemic episodes and the degree of maximal ST-segment changes were manually calculated from the printouts for each interval. The number of SMI episodes per patient throughout these

| | | 16:31 | 16:32 | 16:33 | 16:34 | 16:35 | 16:36 | 16:37 | 16:38 | 16:39 | 16:40 | 16:41 | 16:42 |
|---------|------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
| HR | bpm | 115 | 116 | 116 | 118 | 116 | 112 | 114 | 113 | 108 | 109 | 107 | 107 |
| STIII | mm | 1.0 | 1.0 | 1.0 | 1.1 | 1.0 | 1.0 | 0.9 | 0.8 | 0.6 | 0.5 | 0.5 | 0.5 |
| STaVL | mm | -1.0 | -1.0 | -1.0 | -1.0 | -0.9 | -1.0 | -0.9 | -0.7 | -0.7 | -0.6 | -0.6 | -0.6 |
| STV1 | mm | 0.3 | 0.3 | 0.3 | 0.3 | 0.2 | 0.3 | 0.3 | 0.3 | 0.4 | 0.4 | 0.5 | 0.5 |
| NBP S | mmHg | | | | | | | | | | | | |
| NBP M | mmHg | | | | | | | | | | | | |
| NBP D | mmHg | | | | | | | | | | | | |
| %PACED | % | | | | | | | | | | | | |
| PVC/min | bpm | | | | | | | | | | | | |
| STI | mm | -1.0 | -1.0 | -1.0 | -0.9 | -0.9 | -1.0 | -0.9 | -0.8 | -0.7 | -0.7 | -0.7 | -0.7 |
| STII | mm | 0.0 | 0.0 | 0.0 | 0.1 | 0.1 | 0.0 | 0.0 | 0.0 | -0.1 | -0.1 | -0.2 | -0.2 |
| STaVR | mm | 0.4 | 0.5 | 0.4 | 0.4 | 0.4 | 0.4 | 0.4 | 0.4 | 0.4 | 0.4 | 0.5 | 0.4 |
| STaVF | mm | 0.5 | 0.5 | 0.5 | 0.6 | 0.5 | 0.5 | 0.5 | 0.3 | 0.2 | 0.2 | 0.1 | 0.1 |
| STV2 | mm | -0.5 | -0.5 | -0.5 | -0.5 | -0.4 | -0.3 | -0.3 | -0.1 | 0.0 | 0.0 | 0.1 | 0.2 |
| STV3 | mm | -0.9 | -1.0 | -1.0 | -0.8 | -0.7 | -0.6 | -0.7 | -0.3 | -0.2 | -0.1 | 0.0 | 0.1 |
| STV4 | mm | -1.7 | -1.8 | -1.8 | -1.6 | -1.9 | -1.8 | -1.9 | -1.7 | -2.0 | -1.6 | -1.6 | -1.4 |
| STV5 | mm | -1.0 | -1.1 | -1.0 | -0.9 | -1.1 | -1.1 | -1.2 | -1.4 | -2.0 | -1.7 | -1.7 | -1.7 |
| STV6 | mm | -0.8 | -0.9 | -0.9 | -0.7 | -0.9 | -1.0 | -1.0 | -1.5 | -2.2 | -1.9 | -2.1 | -2.1 |
| STVM | mm | 1.2 | 1.2 | 1.2 | 1.0 | 1.1 | 1.1 | 1.2 | 1.2 | 1.6 | 1.3 | 1.4 | 1.4 |
| STCVM | mm | 0.5 | 0.5 | 0.5 | 0.3 | 0.4 | 0.3 | 0.4 | 0.4 | 0.8 | 0.6 | 0.7 | 0.8 |

Figure 1. A sample showing real time ST-segment changes recorded from 12 leads.

three periods were compared. The lead showing the maximum ST-segment deviation was also recorded.

Sheath removal and hemostasis. Prior to SR, 10 ml of subcutaneous 2% procaine was applied around the sheath insertion site. Care was taken to keep the sheath as fixed as possible until removal. The sheaths were removed approximately two hours after PCI by a physician. Activated clotting time or activated partial thromboplastin time tests were not performed before SR. Hemostasis was achieved by manual compression and maintained with a tight bandage. The bandage was not removed during the whole monitoring period.

Blood pressure, heart rate, and complications. Blood pressure was measured noninvasively hourly using an automated cuff during the follow-up, and heart rate was automatically recorded every minute. The patients were specifically questioned for the presence of chest pain, urinary problems, and back pain. Anxiety was assessed subjectively by the physician based on observable patient behaviors. Puncture site complications were defined as the presence of major hematoma (>5 cm in diameter), minor hematoma (>2.5 cm), bleeding requiring transfusion, retroperitoneal bleeding, vasovagal attack, femoral artery occlusion, pseudoaneurysm, or arteriovenous fistula. Symptomatic ischemia was defined as detection of an ischemic episode in the course of chest pain. Creatine kinase-MB (CK-MB) and troponin I were measured at 0, 6, and 24 hours after admission to the CCU. Non-ST-segment elevation myocardial infarction was defined by CK-

MB exceeding three times the upper normal limit or troponin I exceeding the upper normal limit.

Statistical analysis. The variables are presented as mean \pm standard deviation for continuous data and as proportions for categorical data. The Kolmogorov-Smirnov test was used to identify whether continuous variables were normally distributed. Descriptive statistics were used to calculate demographic and clinical characteristics. Associations between two categorical variables were tested using the chi-square or Fisher's exact test as appropriate. Data were also analyzed with the Friedman ANOVA test, Wilcoxon signed-rank test, Mann-Whitney U-test, or Kruskal-Wallis test, whenever appropriate. Two-sided *p* values of less than 0.05 were considered significant. Statistical analysis was performed using a commercially available software package (SPSS 9.0 for Windows).

RESULTS

Demographic and clinical characteristics of the patients are given in Table 1. A total of 88 lesions were treated in 66 subjects by PCI. A total of 71 stents (56 uncovered) were implanted in 56 patients. Ten patients were treated with only balloon angioplasty. Procedural data and distribution of lesions are given in Table 2.

Frequency of SMI. Patients were monitored for approximately six hours after PCI, during which SMI was detected in 32 patients (48.5%). The numbers of SMI episodes were 44 (11 patients, 16.7%), 121 (29 patients, 43.9%), and 65 (20 patients, 30.0%) before SR, within the first two hours after SR, and in the remain-

Table 1. Demographic and clinical characteristics of the patients

| | n | % | Mean±SD |
|-----------------------------------------------|----|-------|-----------|
| Age (years) | | | 59.5±10.3 |
| Sex | | | |
| Male | 51 | 77.3 | |
| Women | 15 | 22.7 | |
| Body mass index (kg/m ²) | | | 26.5±3.0 |
| Hypertension | 35 | 53.0 | |
| Current smoker | 26 | 39.4 | |
| Diabetes mellitus | 22 | 33.3 | |
| Dyslipidemia | 34 | 51.5 | |
| Obesity | 11 | 16.7 | |
| Family history of coronary artery disease | 22 | 33.3 | |
| Previous myocardial infarction | 36 | 54.6 | |
| History of percutaneous coronary intervention | 20 | 30.3 | |
| Previous coronary artery bypass graft surgery | 15 | 22.7 | |
| Sinus rhythm | 66 | 100.0 | |
| Ischemia on admission ECG | 10 | 15.2 | |
| ECG evidence for old myocardial infarction | 35 | 53.0 | |
| Ejection fraction (%) | | | 48.9±10.8 |
| Coronary artery disease | | | |
| Single vessel | 27 | 40.9 | |
| Double vessel | 20 | 30.3 | |
| Triple vessel | 19 | 28.8 | |

Table 2. Procedural data on percutaneous coronary intervention (PCI)

| | n | % |
|------------------------------------|----|-------|
| Femoral arterial puncture | 66 | 100.0 |
| Sheath size | | |
| 6 French | 48 | 72.7 |
| 7 French | 18 | 27.3 |
| Percutaneous coronary intervention | | |
| Only stent | 28 | 42.4 |
| Only balloon | 10 | 15.2 |
| Both stent and balloon | 28 | 42.4 |
| The number of vessels treated | | |
| One vessel | 53 | 80.3 |
| Two vessels | 12 | 18.2 |
| Three vessels | 1 | 1.5 |
| The number of lesions treated | | |
| One lesion | 46 | 69.7 |
| Two lesions | 18 | 27.3 |
| Three lesions | 2 | 3.0 |
| The number of stents | | |
| None | 10 | 15.2 |
| One stent | 41 | 62.1 |
| Two stents | 15 | 22.7 |
| Revascularization | | |
| Complete | 35 | 53.0 |
| Incomplete | 31 | 47.0 |
| Clopidogrel treatment | | |
| Regular use before PCI | 38 | 57.6 |
| Loading dose (300 mg) | 9 | 13.6 |
| Loading dose (450 mg) | 19 | 28.8 |

ing two hours, respectively. The number of patients who suffered *de novo* SMI in these three consecutive periods were 11 (16.7%), 20 (30.3%) and 1 (1.5%), respectively (Table 3).

There were more patients experiencing SMI in the first two hours after SR than in the period before SR ($p<0.001$) and in the last two hours of ST monitoring ($p=0.022$). The number of SMI episodes per patient during the first two hours after SR was also significantly higher compared to the period before SR (1.8 ± 3.8 vs. 0.7 ± 2.4 , $p<0.001$).

The incidence of incomplete revascularization was 47%. The number of SMI episodes per patient was significantly higher in patients with incomplete revascularization compared to patients with complete revascularization (5.4 ± 10.2 vs. 1.8 ± 4.3 , $p=0.039$).

There was no difference between the types of PCI procedures (only stent, only balloon, both stent and balloon) in terms of the number of SMI episodes ($p>0.05$). The number of SMI episodes per patient was significantly higher in subjects with anxiety than in patients without anxiety (6.5 ± 10.1 vs. 2.2 ± 7.0 , $p=0.005$). Heart rate and blood pressures of the pa-

tients are shown in Table 4. There was no difference between patients with or without hypotensive attacks in terms of the number of SMI episodes per patient (3.8 ± 8.7 vs. 1.2 ± 2.9 , $p=0.25$).

ECG derivations showing maximum ST shift and false alarms. The first three derivations containing a maximum ST-segment shift were V3 (26.5%), V2 (16.6%), and D III (12.8%). The recorded maximum ST-segment shifts were significantly greater in both the first and second two hours after SR compared to the period before SR (0.82 ± 0.30 mm and 0.77 ± 0.36 mm vs. 0.68 ± 0.32 mm, $p<0.001$ and $p=0.008$, respectively). At least one false ST alarm was recorded in 21 patients (31.8%).

In-hospital complications. The most frequent complications were groin pain ($n=25$, 37.9%), anxiety ($n=19$, 28.8%), and minor hematoma ($n=13$, 19.7%). There were no significant differences among the levels of CK-MB and troponin I at 0, 6 and 24 hours in all pairwise comparisons ($p>0.05$).

DISCUSSION

The main finding of this study suggests that SMI is more frequently observed during time of hemostasis

Table 3. Distribution of patients having silent myocardial ischemia (SMI) in the three periods of ST-segment monitoring

| Occurrence of SMI during intervals | | | |
|----------------------------------------|------------------------------------------------------|------------------------------------------|----------------|
| Before sheath removal (11 patients) | Within 2 hours after sheath removal (29 patients) | Within the last 2 hours (20 patients) | No of patients |
| + | + | + | 9 |
| + | - | - | 2 |
| + | + | - | 0 |
| + | - | + | 0 |
| - | + | - | 10 |
| - | + | + | 10 |
| - | - | + | 1 |
| - | - | - | 34 |

«—— p<0.001 ——» «—— p=0.022 ——»
«———— p=0.012 ————»

and in the first hours after SR. These findings might be attributable to SR-induced physical and mental stress, hemostasis procedure, or tight bandage. To reduce the risk for SMI during and after hemostasis, analgesics and sedatives may be given more effectively. The use of femoral closure devices might also be an option. Even though there are substantial data on silent ischemia, its prognostic importance is still controversial. Histopathological studies propose the idea that recurrent ischemia may be associated with irreversible myocardial changes^[7] resulting in scarred or fibrotic myocardium, predisposing to life-threatening arrhythmias or congestive cardiac failure.

The frequency of SMI is expected to be less in patients who have undergone nonurgent uncomplicated PCI, particularly after stent implantation.^[8] Although SMI was found in 11% of patients undergoing percutaneous transluminal balloon angioplasty,^[9] Kathiresan et al.^[10] reported the incidence as 33% even after stenting. Similarly, we detected SMI in nearly half of our patients (48.5%). This high rate may partly be due to the use of different ST-segment monitoring systems in diverse studies and the high rate of incomplete revascularization in our study.

False alarms make the evaluation of SMI difficult. Drew et al.^[11] showed that 40% of patients who had been monitored bedside for an average of 41 hours had at least a false ST alarm, the most common cause being a change in the body position. Similarly, at least one false ST alarm was determined throughout the monitoring period in 31.8% of our patients.

The best three-lead combination for ST-segment monitoring includes the leads III, V3, and V5. The most sensitive leads for the right coronary artery and left anterior descending artery are leads III and V2-V3, respectively. For the left circumflex artery, a variety of leads may be involved, depending on the myocardial zone affected.^[5] In the present study, the most frequent derivation in which maximum ST shifts were seen was V3, probably because of the fact that most PCI procedures involved the left coronary system.

Compared to the other two periods, the mean heart rate was significantly higher and diastolic and systolic blood pressures were significantly lower in the first two hours after SR. Significant increases in the mean heart rate and significant decreases in systolic and diastolic blood pressures may be due to excessive adrenaline

Table 4. Clinical data of patients in the three periods of ST-segment monitoring

| | Before sheath removal | Within 2 hours after sheath removal | Within the last 2 hours |
|---------------------------------|-----------------------|-------------------------------------|-------------------------|
| Monitoring time (min) | 124.0±8.4 | 120.0±0.0 | 120.0±0.0 |
| SMI episodes per patient | 0.7±2.4 | 1.8±3.8* | 1.0±3.0 |
| Maximum ST shift (mm) | 0.68±0.32 | 0.82±0.30* | 0.77±0.36 |
| Heart rate (beats/min) | 72.7±10.1 | 75.9±10.5* | 73.1±11.6 |
| Systolic blood pressure (mmHg) | 124.1±15.9 | 120.7±15.5* | 123.8±18.2 |
| Diastolic blood pressure (mmHg) | 74.6±10.4 | 71.6±8.9* | 74.2±9.2 |

*p<0.05 compared to two other periods. SMI: Silent myocardial ischemia.

secretion associated with anxiety, which may have an inhibitory effect on the vasovagal response.

Study limitations. The number of patients in this study may be too small for the strength of our data. Silent myocardial ischemia associated with SR was studied as a separate factor following uncomplicated PCI, i.e. not related to the pathology or to the type of the procedure. The time between the procedure and SR may be evaluated for other types of PCI for comparison and more solid results. Hemostasis procedures were carried out by different physicians, which might affect the results of the study. Monitoring periods were of short duration. Both the number of patients and the frequency of major adverse cardiac events (MACE) during hospitalization were very low for statistical analysis. The patients were not followed-up after discharge from hospital. These limitations did not allow to investigate associations between in-hospital or long-term MACE and SMI. Because vasovagal attack and troponin release were determined in a limited number of patients, the associations of these factors with SMI could not be analyzed. Another limitation was that anxiety was not measured objectively.

In conclusion, SMI is more frequently observed during the early hours after SR. Since there was no rise in cardiac markers or symptoms, SR was not a cause of significant SMI. Therefore, new SMI during SR and for the next two hours needs careful interpretation to avoid unnecessary measures or intervention.

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