Evaluation of Coagulation Markers for Early Diagnosis of Acute Coronary Syndromes in the Emergency Room

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Background: Diagnosis of acute coronary syndromes (ACS) is a major challenge for emergency physicians. Because soluble fibrin (sF) has been suggested as a potential early marker of impending myocardial ischemia, we were interested whether a sF bedside test could help in early identification of patients with ACS in the emergency department.

Methods: We evaluated plasma coagulation markers, including a newly developed sF bedside test, prothrombin fragment (F1+2), sF, and D-dimer, in a cross-sectional trial with 184 patients suggestive of ACS.

Results: Whereas 76% (13 of 17) of patients with unstable angina pectoris (UAP) had a positive sF bedside test, only 10 of 33 patients (30%) with non-ST-segment-elevation myocardial infarction and 10 of 44 patients (23%) with ST-elevation myocardial infarction tested positive. Three percent of controls (1 of 33) and 11% of patients (6 of 57) with preexisting stable angina had a positive sF bedside test (\( P < 0.001 \) for noncardiac chest pain vs ACS), yielding an overall specificity of 92% and a sensitivity of 35%. The sensitivity of the established coagulation markers was significantly less to detect ACS (11% for F1+2, 20% for thrombus precursor protein, and 18% for D-dimer; \( P < 0.02 \) vs sF bedside test). The sF bedside test presented the earliest objective indicator of impending myocardial damage in the majority (10 of 13) of ACS patients with a normal or nondiagnostic electrocardiogram (ECG).

Conclusions: A sF bedside test offers a specific tool for early identification of patients with ACS in an emergency department setting, although its sensitivity seems sufficient only for the early identification of patients with UAP. A sF bedside test could be useful, particularly in UAP patients with a nondiagnostic ECG.

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The term acute coronary syndrome (ACS)\(^4\) has evolved as an operational term to refer to any constellation of clinical syndromes that are compatible with acute myocardial ischemia (1). It encompasses acute myocardial infarction (AMI) as well as unstable angina pectoris (UAP).

In addition to medical history and physical examination, the electrocardiogram (ECG) is critical for the initial evaluation of patients with suspected ACS. However, in 20–50% of cases, the ECG is nondiagnostic at hospital admission (2–4). Thus, biochemical cardiac markers have become important diagnostic tools for ACS. It is currently standard that patients presenting with chest pain to an emergency department (ED) are evaluated not only by ECG but also by determination of biochemical cardiac markers, including creatine kinase (CK), creatine kinase-MB isoenzyme (CK-MB), and cardiac troponins (1).

To identify patients with ACS, serial assessment of CK-MB and cardiac troponin I (cTnI) or cardiac troponin I at 4- to 6-h intervals is obligatory because of the time lag of CK-MB and cTnI increases after the onset of chest pain (5). Determination of myoglobin appears to have higher sensitivity for the early detection of myocardial damage

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4 Nonstandard abbreviations: ACS, acute coronary syndrome; AMI, acute myocardial infarction; UAP, unstable angina pectoris; ECG, electrocardiogram; ED, emergency department; CK, creatine kinase; CK-MB, creatine kinase-MB isoenzyme; cTnI, cardiac troponin I; sF, soluble fibrin; NSTEMI, non-ST-segment-elevation myocardial infarction; CAD, coronary artery disease; and TpP, thrombus precursor protein.
All these markers, however, merely detect myocardial necrosis after MI but totally fail to detect UAP. Additionally, there is a relatively long time lag before these markers increase. Hence, novel early markers of ACS are desirable.

Because tissue factor-induced thrombin formation is a cardinal feature of ACS (7, 8), a promising approach to find early markers for ACS involves the measurement of fibrin formation and fibrin degradation products. The main pathophysiologic mechanism for ACS is rupture or erosion of an atherosclerotic plaque followed by exposure of thrombogenic contents to the circulation. This induces coagulation and platelet activation and, finally, thrombus formation. The key event in the activation of plasmatic coagulation is the conversion of prothrombin into thrombin, which in turn converts fibrinogen into fibrin (9). The use of fibrinopeptide A as a marker of fibrin formation is limited because of preanalytical and technical difficulties (10). To overcome these limitations, measurement of soluble fibrin (sF) as a direct indicator of thrombin activity has been proposed (11). In addition, fibrin monomer antigen has predicted mortality in patients with AMI (12, 13).

As initiation of clotting plays a pivotal role in the pathogenesis of ACS, measurement of activation of coagulation may predict myocardial ischemia before myocardial necrosis occurs (13). The usefulness of measuring activation markers of coagulation may, however, be compromised by two factors: (a) a relatively small thrombus in a small sited coronary artery may not yield detectable elution of activation markers into systemic circulation, particularly when the vessel is already occluded; and (b) experience with fibrinolytic therapy and autopsy studies suggests that only ~50% of occlusive thrombi may be considered fibrin rich. This could significantly reduce the sensitivity of coagulation tests to detect ACS.

Nonetheless, we hypothesized that sF could serve both to predict the outcome in patients with AMI (12, 13) and to detect ACS at a very early stage, even before myocardial damage (CK-MB and cTnT release). We therefore set out to investigate whether a newly developed bedside test for sF may help in early identification of patients with ACS presenting to the ED with clinical symptoms suggestive of myocardial ischemia.

**Materials and Methods**

**STUDY PROTOCOL**

The study protocol was approved by the Ethics Committee of the University of Vienna. All patients with chest pain were seen at the Emergency Department, University Hospital, a tertiary care facility equipped with ECG monitoring where patients can remain under observation for a maximum of 24 h. Consecutive patients with chest pain suggestive of myocardial ischemia were eligible for the study, and all gave informed consent before inclusion. We enrolled 200 consecutive patients over a period of 3 months. Exclusion criteria were signs of systemic inflammation, i.e., fever, increased C-reactive protein or fibrinogen >4.5 g/L; suspected thromboembolic disease; severe skeletal muscle damage or trauma; cardiac resuscitation; and inability or refusal to give informed consent.

Patients with ECG abnormalities on admission that were strongly suggestive of AMI were immediately transferred to the critical care unit of the ED and received standard therapy, including thrombolysis or percutaneous transluminal angioplasty, at the discretion of the physician. Patients with new episodes of chest pain or accelerated angina accompanied by typical ECG changes were also referred to the critical care unit for standard treatment. Blood sampling from these patients continued for at least 24 h. All other patients remained in an ambulatory setting for observation and blood sampling for at least 6 h.

The attending physicians noted the time of onset of symptoms. A history was taken and physical examinations and ECGs were performed before any laboratory results were reported. Blood samples were drawn immediately after insertion of an indwelling intravenous line. All samples for analysis of activation markers of coagulation were obtained, after discarding the initial 20 mL (used for clinical chemistry), into evacuated tubes containing 129 mmol/L sodium citrate (Vacutainer; Greiner Bio-One) before any therapeutic intervention. This does not produce measurable concentrations of thrombin activation or fibrin generation (14).

Clinical data from the primary evaluation, including the history and the results of the examinations, were recorded by the ED physicians according to a standardized protocol. A third investigator was involved to determine diagnosis if there was a disagreement regarding the diagnosis between the ED physician and/or the independent investigator. A final diagnosis was established by chart reviews from two independent investigators as follows.

**Acute ST-segment-elevation MI.** According to the WHO criteria and the Consensus Document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction (15), we defined AMI as a typical increase and gradual decrease (cardiac troponin) or more rapid increase and decrease (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following: typical chest pain, ST-segment elevations of at least 2 mm in two or more precordial leads or of at least 1 mm in two or more corresponding extremity leads, and development of pathologic Q-waves on the ECG.

UAP. UAP was classified according to the Braunwald classification. Basically, UAP is characterized by episodes of chest pain with reversible ST- and T-wave changes but no biochemical markers in the circulation (1).
Non-ST-segment-elevation MI (NSTEMI). Patients with clinical UAP and positive cTnT were described as NSTEMI (1).

Stable coronary artery disease (CAD). This category included patients with known ischemic heart diseases but without reversible ST- and T-wave changes in the ECG and/or increases in biochemical markers (cTnT or CK-MB) as specified above.

Others. Others included patients with chest pain of non-cardiac origin and patients with nonischemic cardiac disease that might cause myocardial damage and subsequent increases in cTnT and CK-MB (e.g., myocarditis and pericarditis).

SAMPLE SIZE ESTIMATION
A classic sample size estimation was difficult to perform because we did not know the prevalence of a positive test result in our population of interest. For estimates of sensitivity and specificity, ~50 patients in each of the three groups (UAP, AMI, and stable angina) were considered to be sufficient. For clinical (therapeutic) reasons, it seemed appropriate to define the comparison of patients with and without ACS as the comparison of primary interest, i.e., AMI and UAP vs stable angina and patients with chest pain of noncardiac origin as controls. Hence we estimated that a sample size of 100 individuals with and without ACS and a test specificity of 90% would provide significant results at a two-sided P value of 0.05 when the test has a sensitivity of 20%. We took into consideration that only 50% of patients with AMI respond adequately to thrombolytic therapy (16), which corresponds with the prevalence of fibrin-rich coagulation thrombi at autopsy (17). Hence, it must be assumed that activation of plasmatic coagulation is the predominant feature in only 50% of patients with acute occlusive thrombi and that the remainder of patients may not have measurable activation of coagulation.

BLOOD SAMPLING AND ANALYSES
Blood was sampled before any intervention, and medical histories were taken and ECG, laboratory analyses, and diagnostic/therapeutic interventions were performed as appropriate.

Blood samples were centrifuged at 3000g for 15 min immediately after collection, and citrated (129 mmol/L) plasma was stored as 0.5-mL aliquots at −80 °C until analysis. The following enzyme immunoassays were used: F₁₋₂ (Behring); D-dimer (Boehringer Mannheim); and sF [thrombus precursor protein (TpP); American Biogenetic Sciences]. These assays, except the PredictaClot, have been used and described in detail in several recent publications (18–20). The TpP is an enzyme-linked immunoassay for the quantitative determination of sF polymers. The reference interval of the TpP ELISA was based on measurements of 20 healthy patients and was <1.75 mg/L (mean ± 2 SD); the inter- and intraassay CVs were <10% and <6%, respectively, for a concentration range of 0.13–4.2 mg/L.

The semiquantitative sF bedside test, called PredictaClot, was also obtained from American Biogenic Sciences. It is a lateral flow immunoassay that uses the same capture antibody (MH1) as in the TpP ELISA. The antibody is a murine IgG₁ that is quite specific for a conformational epitope on fibrin that includes D-dimer/fragment E complex. The reporting antibody has been coupled to colloidal gold by standard conjugation methods. The detector antibody in the sF bedside test is the Fab’2 fragment of MH1 and is applied at a predefined area on the nitrocellulose strip. There is no cross-reaction with fibrinogen or the major degradation products of either fibrinogen or fibrin.

We performed a preliminary evaluation comparing the bedside test with the TpP enzyme immunoassay in 78 patients with diagnosed thrombotic disorders (deep venous thrombosis and pulmonary embolism) and found a high level of agreement (0.87) (21).

Before starting the trial, we validated the sF bedside assay with 10 plasma samples from patients with D-dimer concentrations 3 mg/L and 20 samples from healthy volunteers (D-dimer concentration <0.5 mg/L). Whereas test results from healthy persons were all negative, patients with high D-dimer concentrations all had positive test results. Additionally, we tested 20% of our study samples as duplicates with two different assessors, blinded with regard to clinical and test results. The agreement between the duplicate analyses and/or the observers was >95%.

The sF bedside test was mostly performed immediately after blood sampling by two trained persons (a laboratory technician and a medical doctor). In some cases citrated plasma, obtained after centrifugation of blood samples for 15 min at 3000g, was stored at −80 °C until assayed. The assay procedure was carried out according to the manufacturer’s instructions. Briefly, with a delivery pipette, four drops of plasma were placed into a microtube containing 400 μL of plasma dilution buffer. Four drops of the diluted plasma sample were added into the sample well of the lateral flow device with a new delivery pipette. When the sample was absorbed in the sample well, two drops of a chase buffer were placed into the sample well. Two different analysts, blinded with regard to clinical outcome of the patients, evaluated the assays 15 min after application of the sample to the sF bedside test. The appearance of a red line in both control and test areas indicated a positive test, whereas a negative test had a red line only in the control area. The absence of a red line in the control area indicated an inconclusive test, which was repeated with a new device. The test reagent set also included a positive control, which was used for quality-control purposes every other day. In addition, aliquots of patient samples with high D-dimer content, known to react positively, were used weekly as a positive control.
cTnT was measured in the routine laboratory with the Elecsys 1010 analyzer (Roche Diagnostics) as described elsewhere (22). The reference values are \( \leq 0.01 \, \mu \text{g}/\text{L} \). CK-MB was determined with the Hitachi 917, a fully automated analyzer (Roche). The reference values are <10 U/L. C-reactive protein was measured in the hospital’s central laboratory by a fully automated chemiluminescence immunoassay (Roche).

**Statistics**

We defined the sensitivity of the sF bedside test to detect ACS as our primary focus of interest. Sensitivity and specificity were calculated within the cohorts of patients with and without ACS, respectively, by the following formulas: sensitivity = true positive/(true positive + false negative); and specificity = true negative/(true negative + false positive).

Dichotomous variables were compared with the \( \chi^2 \) test. Data are presented as means and the 95% confidence intervals. All statistical comparisons of continuous variables were made with nonparametric tests. The Kruskal–Wallis ANOVA, Mann–Whitney \( U \)-test, and Spearman rank correlation test were used. A two-sided \( P \) value <0.05 was considered as significant for the primary outcome variable. \( P \) value corrections were performed for the additional outcome variables.

All statistical calculations were performed with commercially available statistical software (Statistica, Ver. 5.0).

**Results**

Two hundred consecutive patients with chest pain presenting to the ED were enrolled in this study. None of the patients had impaired renal function as defined by plasma creatinine concentrations >120 \( \mu \text{mol}/\text{L} \). Chart reviews revealed that 6 patients did not meet the inclusion criteria because of infection (herpes zoster, \( n = 1 \); pneumonia and/or fever, \( n = 4 \)) or thrombophlebitis (\( n = 1 \)), and 10 patients were excluded because of incomplete data collection, mainly because the patients decided to leave the ED before a repeat ECG or blood sampling. The demographic data for the remaining 184 patients with chest pain of suspected cardiac origin are shown in Table 1. Ninety-four patients were finally diagnosed to have ACS: 17 with UAP, 33 with NSTEMI, and 44 with AMI. Patients with and without ACS did not differ with regard to age, duration of chest pain, or sex distribution (although more men than women were included). The median duration of chest pain was \( \sim 6 \) h for UAP and AMI and 3 h for NSTEMI.

Thirty-five percent (33 of 94) of patients with ACS had a positive sF bedside test compared with 3% (1 of 33) of controls or 11% (6 of 57) of patients with preexisting stable angina (Table 2; \( P <0.001 \) between patients with and without ACS). The specificity of the sF bedside to detect all ACS was 92%, and the sensitivity was 35% in the setting of an ED.

**Table 1. Demographics of patients with chest pain.**

<table>
<thead>
<tr>
<th>Age, years</th>
<th>Other chest pain</th>
<th>ACS</th>
</tr>
</thead>
<tbody>
<tr>
<td>61 ± 13</td>
<td>64 ± 14</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>63 M/27 F</td>
<td>55 M/39 F</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preexistent known CAD, %</td>
<td>56</td>
<td>38</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>47</td>
<td>42</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>42</td>
<td>21</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>32</td>
<td>42</td>
</tr>
<tr>
<td>Hyperlipidemia, %</td>
<td>34</td>
<td>49</td>
</tr>
<tr>
<td>Family history, %</td>
<td>33</td>
<td>32</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27 ± 3</td>
<td>27 ± 4</td>
</tr>
<tr>
<td>Duration of pain, h</td>
<td>4.5 (0.5–133)</td>
<td>4 (0.3–133)</td>
</tr>
</tbody>
</table>

**Table 2. Diagnostic performance of the sF bedside test in patients with suspected myocardial ischemia.**

<table>
<thead>
<tr>
<th>Other chest pain (n = 90)</th>
<th>Positive/Negative</th>
<th>% positive tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>7/83</td>
<td>8</td>
</tr>
<tr>
<td>Controls</td>
<td>1/32</td>
<td>3</td>
</tr>
<tr>
<td>Stable angina</td>
<td>6/51</td>
<td>11</td>
</tr>
</tbody>
</table>

**ACS (n = 94)**

| Total | 33/61 | 35* |
| UAP | 13/4 | 76 |
| NSTEMI | 10/23 | 30 |
| MI | 10/34 | 23 |

\( ^* P <0.001 \) vs other chest pain.
of the onset of symptoms had a positive sF bedside test. In contrast, only 4 of 26 patients (13%) presenting after 6 h from the onset of symptoms had a positive test result \( (P = 0.16, \text{early vs late presenters}) \).

Initially, 13 patients with ACS exhibited a normal ECG \( (n = 5) \) or nondiagnostic ECG \( (n = 8; \text{pacemaker ECG, bundle branch block, and atrial fibrillation}) \). Interestingly, the majority \( (10 \text{ of } 13) \) had a positive sF bedside test, but 5 of these 10 patients had a negative cTnT on admission, and 1 patient developed positive cTnT after 4 h of observation. The remaining four patients developed changes in the ECG obtained 4 h after admission.

Plasma concentrations of \( \text{F}_{1+2} \) fragment, sF, and D-dimer for ED patients at the time of admission are depicted in Fig. 1. The correlation between coagulation markers was weak \( (r = 0.40 \text{ for TpP and D-dimer}; r = 0.60 \text{ for TpP and } \text{F}_{1+2}; \text{ and } r = 0.37 \text{ for } \text{F}_{1+2} \text{ and D-dimer}; P < 0.001 \text{ for all determinations}) \).

Almost all patients within the highest quartile of sF values \( (\text{TpP} > 0.99 \text{ mg/L}) \) had CAD \( (97\%) \), 70\% had ACS, and 3\% (one patient) had chest pain of noncardiac origin. Identically, 97\% of patients within the highest quartile of D-dimer \( (\text{D-dimer} > 0.6 \text{ mg/L}) \) had CAD, 70\% had ACS, and 3\% had chest pain of noncardiac origin. Patients within the highest quartile for \( \text{F}_{1+2} (> 1.73 \text{ nmol/L}) \) also had a high prevalence of CAD \( (97\%) \), whereas 60\% had ACS and 3\% had noncardiac chest pain.

After univariate regression analysis, age, cTnT, and ECG changes at admission as well as changes in ECG wave patterns were entered into multiple regression analysis, which was performed with the whole cohort of patients. sF, D-dimer, and \( \text{F}_{1+2} \) values were used as dependent variables. The only significant finding was that changes in ECG wave pattern were associated with sF concentrations \( (P < 0.01) \).

Thirteen patients with ACS had a positive sF bedside assay and negative cTnT during their stay at the ED. Nine of them \( (69\%) \) had no cardiac event during 1 year of follow-up, one patient exhibited restenosis and underwent percutaneous transluminal angioplasty, one patient died, and two were lost to follow-up. Thirteen patients were within the highest quartile for sF measured by the TpP ELISA \( (> 0.99 \text{ mg/L}) \) and had negative cTnT values. Ten of these 13 \( (77\%) \) had an event-free 1-year follow-up, 2 \( (15\%) \) were hospitalized because of UAP, and 1 was lost to follow-up.

Patients with ACS had, on average, 26\% higher plasma \( \text{F}_{1+2} \) fragment, 46\% higher sF, and 111\% higher D-dimer \( (P < 0.001 \text{ for all markers}; \text{ Fig. 2}) \). Patients with a positive sF bedside test had, on average, 26\% higher plasma concentrations of \( \text{F}_{1+2} \) fragment, 59\% higher sF, and 180\%
higher D-dimer than those with a negative sF bedside test (P \leq 0.01 for all markers; Fig. 2).

Finally, we compared the sensitivity of the sF bedside test (35%) with the sensitivity of established coagulation markers at the same specificity (92%). The sensitivity was 11%, 20%, and 18% at cutoff concentrations of 2.52 nmol/L F_{1+2}, 1.19 mg/L TpP, and 1.0 mg/L D-dimer, respectively. Thus, the sensitivity of the sF bedside test was higher than that of the other three activation markers of coagulation (P \leq 0.02).

Discussion

Five million patients present to an ED with chest pain in the US each year. Triage of these patients is one of the most difficult challenges for emergency physicians (23). An ECG is not always helpful in the early decision-making process because the initial ECG is nondiagnostic or indeterminate in many cases, particularly in patients with coexisting cardiac pathology such as left ventricular hypertrophy or previous MI (2–4). In several studies, \sim 5% of patients with a normal ECG who were discharged from the ED were ultimately found to have ACS (24, 25).

In the present study, we have demonstrated that a bedside test for sF detected 46% of patients with UAP or NSTEMI. sF was the earliest objective indicator of risk for myocardial damage in one-fifth of patients with ACS. In 5 of these 10 ACS patients, the initial cTnT was negative, and only 1 patient developed a positive cTnT 4 h after presentation. This is in line with the well-known time lag before established biochemical markers such as cTnT and CK-MB increase after myocardial injury (5). It has even been estimated that 7- and 12-h observation periods will be necessary for CK-MB and cTnT to rule out AMI with a certainty of \sim 92–95% (6). In contrast, our findings show that sF was already increased in a subset of patients with ACS before markers of myocardial damage were detectable. This is in good agreement with two previously published studies reporting that sF concentrations were markedly increased even in those patients who presented early after onset of chest pain (26, 27).

Given the fact that intravenous thrombolytic therapies fail to achieve coronary flow in as many as 50% of patients with AMI (16) and that 50% of occlusive thrombi are fibrin rich at autopsy (17), one may not expect perfect sensitivity of a test that measures coagulation activation. Although the specificity of the sF bedside test to detect all ACS was rather good (92%), the sensitivity (35%) was lower: the sF bedside test detected 76% of cases with UAP. In contrast, the sensitivity of the sF bedside test was 30% to detect NSTEMI and 23% to detect ST-elevation AMI. This compares with a 66% sensitivity of cTnT to detect AMI on admission in our study and, by definition, a 0% sensitivity of cTnT to detect UAP. The low sensitivity of the sF bedside test in ST-segment-elevation AMI is not a major concern because diagnosis is made by ECG. However, the difference in sensitivities to detect UAP and AMI still warrants additional discussion.

Does a positive sF bedside test reflect local coagulation activation by the thrombus in the coronary artery or enhanced systemic activation of coagulation? It is difficult to understand why a tiny thrombus that occludes a stenosed artery should cause systemic coagulation activation, particularly when there is no flow in the artery that could elute sF into the circulation of patients with AMI. Along similar lines, we postulate that markers of coagulation can increase in the systemic circulation only as long as blood flows over the thrombus. This concept is strongly supported by two previous studies showing that sF concentrations are particularly increased early after onset of MI (26, 27). Only two of eight patients presenting with AMI after 6 h had abnormal concentrations of sF in one of these trials, whereas none of the late presenters had abnormal concentrations in the other trial. A similar trend was also observed in our trial. At the time patients with AMI present to the ED, increased sF concentrations may still reflect residual circulating concentrations of sF that were generated when blood still superfused the thrombus. Interestingly, the highest concentrations of sF have previously been observed in individuals with stable angina pectoris or UAP rather than AMI (26). This may well explain why the sF bedside test detected patients with UAP with much higher sensitivity than patients with AMI.

The second issue we have to address is the relative performance of the sF bedside test compared with more established markers of coagulation activation. At a similar specificity (92%), measurement of F_{1+2} and D-dimer provided sensitivities of 11% and 18%, respectively. The low sensitivity of D-dimer is in good agreement with a previous observation of increased concentrations of crosslinked fibrin degradation products in a minority of patients with UAP (28). The considerable overlap in D-dimer concentrations between patients with stable CAD and controls (Fig. 1) is also in line with previous observations (29).

Finally, the question arises whether it is worthwhile to test patients suspected to have ACS with a sF bedside test. Point-of-care testing technologies have gained increasing importance because point-of-care testing allows more rapid clinical decision-making by reducing the turnaround time spent in test ordering, specimen collection and transport, and data reporting (30). Although introduction of a sF test may increase costs, there may be considerable benefit as well: the test may have monitory function, as it may be positive even before increased cTnT concentrations are seen, as discussed above. Whereas the sF bedside test does not detect myocardial damage, it may help to expeditiously initiate anticoagulation in patients suspected to have ACS. This may salvage myocardial tissue even before an increase in cTnT concentrations is detected and improve outcome (31). This concept, however, needs to be confirmed by prospective clinical trials.

One limitation of our study is the cross-sectional trial design, with limited follow-up of patients. Furthermore,
the study population was small; the study, therefore, should be deemed preliminary. Finally, we tested the usefulness of a bedside test in the daily routine of an ED and were interested in fast results to rapidly establish appropriate diagnosis and treatment for ACS patients in the future. Therefore, the bedside test was performed only at admission, and we cannot exclude increased sensitivity of bedside testing when serial measurements are performed.

References


