Journal of the American College of Cardiology
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Published by Elsevier Inc.

Vol. 51, No. 24, 2008
ISSN 0735-1097/08/$34.00
doi:10.1016/j.jacc.2008.03.019

Clinical Utility of C-Reactive Protein Measured at Admission, Hospital Discharge, and 1 Month Later to Predict Outcome in Patients With Acute Coronary Disease

The RISCA (Recurrence and Inflammation in the Acute Coronary Syndromes) Study

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Objectives This study was designed to prospectively determine, in patients with an acute coronary syndrome, whether the inflammatory marker, C-reactive protein (CRP), measured at hospital admission, discharge, and 1 month later has incremental value to predict outcomes at 1 year.

Background The clinical utility of CRP measurements in patients with acute coronary syndromes remains uncertain. Limitations of previous studies have been retrospective design and incomplete adjustment for readily available clinical prognosticators.

Methods The CRP marker was measured at admission, hospital discharge, and 1 month later in consecutive patients hospitalized for acute coronary syndromes in 8 tertiary and secondary hospitals. The primary outcome was a composite of death, nonfatal myocardial infarction (MI), and unstable angina (UA) with electrocardiogram (ECG) changes at 1 year.

Results A total of 1,210 patients, age 62 ± 12 years, 64% with acute myocardial infarction (MI) and 36% with unstable angina (UA), were recruited. At 1 year, the primary outcome occurred in 142 patients (11.7%) and included 58 deaths (4.8%), 79 nonfatal MIs (6.5%), and 26 UA episodes with ECG changes (2.1%). The unadjusted odds ratios (ORs) (95% confidence intervals) of CRP values at admission, hospital discharge, and 1 month later for the occurrence of the primary outcome were 1.20 (1.06 to 1.36), 0.98 (0.85 to 1.14), and 1.23 (1.00 to 1.50), respectively. After multivariate adjustment, ORs were 1.04 (0.91 to 1.20), 0.90 (0.77 to 1.06), and 1.12 (0.93 to 1.34), respectively. The individual components of the primary outcome were also not independently associated with any of the 3 CRP measurements.

Conclusions The modest predictive ability of CRP following admission for an acute coronary syndrome disappeared after adjusting for common clinical variables. This large prospective study does not support the incremental value of measuring CRP in this clinical setting. (J Am Coll Cardiol 2008;51:2339–46) © 2008 by the American College of Cardiology Foundation

Atherosclerosis is considered an inflammatory disease, and its acute manifestations of unstable angina (UA), myocardial infarction (MI), and sudden coronary death are associated with more intense expression of blood-borne inflammatory markers and mediators (1–11). It is therefore plausible that the presence of higher titers of blood inflammatory markers such as

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Manuscript received November 6, 2007; revised manuscript received March 3, 2008, accepted March 4, 2008.

Abbreviations and Acronyms

CRP = C-reactive protein
ECG = electrocardiogram
MI = myocardial infarction
PCI = percutaneous coronary intervention
UA = unstable angina

C-reactive protein (CRP) at presentation of an acute coronary syndrome may be associated with a greater risk of poorer outcomes. It is also plausible that the persistence of more elevated blood inflammatory markers, presumed to reflect a more active or smoldering inflammatory atherosclerotic substrate, may indicate which patients are at greater risk of continuing and future coronary instability.

An important and elusive clinical challenge in patients with UA or MI is to better identify those patients who have a high risk of recurrence of acute coronary instability. Some but not all recent studies have suggested that the serum inflammatory marker, CRP, measured during the acute episode, may provide this predictive information (3,5,12–21). Limitations of these studies include retrospective designs (5,13,14,21), small samples with low power (3,15,17,18,20), highly selective patient cohorts (5,12,14,16,21), and inadequate control of other prognostically important clinical variables (3,5,12–15,17,18,20,21). As a result, despite the large number of clinical studies that have associated inflammatory markers, mostly CRP, with prognosis in the acute coronary syndromes, it is still unclear whether inflammatory markers are truly useful in the clinical management of patients admitted with acute coronary disease.

We therefore undertook this prospective multicenter study in a relatively large and unselected group of patients hospitalized with acute coronary syndromes to determine whether CRP measured at any of 3 points—on admission, at hospital discharge, and 1 month later—might reliably predict those patients at risk of recurrent coronary instability and death and whether this information would be of incremental value to known and readily available clinical prognosticators.

Methods

Study population. Consecutive patients were recruited from 4 tertiary and 4 community hospitals, 7 in Quebec and 1 in New Brunswick, Canada. These hospitals were selected because their clinical investigators were already collaborating in a clinical research network. Each hospital committee on human research approved the study, and all patients gave written informed consent.

To be eligible, patients had to have an urgent admission with a diagnosis of either acute MI or UA and had to be recruited into the study with a first study blood sample obtained within 24 h of symptom onset. Patients transferred from other hospitals were excluded.

The primary composite outcome was the first occurrence of death, MI, or UA (see following discussion for complete details) at 1 year. The original protocol required a history of characteristic chest discomfort or pain with an elevation of creatine kinase-myocardial band to ≥1.5 times the upper normal limit for the diagnosis of MI. The protocol was subsequently amended so that the diagnosis of MI required only characteristic chest pain and an abnormal troponin elevation diagnostic of MI. Thus, although the definition of MI changed during the study to include positive troponin despite negative creatine kinase-myocardial band, this had a neutral effect on primary composite outcome criteria because the latter change only reclassified some patients from UA to MI. The amended MI outcome definition was retained for all analyses reported herein. A diagnosis of UA required either 1 episode lasting ≥10 min or ≥2 episodes lasting ≥5 min of characteristic discomfort or pain at rest or with minimal exertion. This could be either new-onset angina or an abrupt and significant change in the pattern of established angina and creatine kinase-myocardial band had to be negative (<1.5 times the upper normal limit). To increase specificity, UA patients had to have at least 1 of the following features showing objective evidence of myocardial ischemia or high probability of coronary atherosclerosis: electrocardiogram (ECG) changes (≥0.5-mm ST-segment depression or transient ST-segment elevation or ≥2-mm T-wave inversion in ≥2 contiguous leads); a cardiac troponin I or T value in the positive range (subsequently reclassified in the amended protocol as MI); history of MI or coronary revascularization; previous coronary angiogram with at least 1 vessel showing ≥50% stenosis; previous noninvasive test showing myocardial ischemia or evidence of MI; known peripheral arterial disease; known ischemic cerebrovascular disease; or presence of diabetes. A UA, for the purposes of the primary outcome, was defined as a characteristic clinical syndrome requiring either significant ECG changes from previous ECG (1-mm ST-segment depression in ≥2 contiguous leads or T-wave inversion ≥3 mm in ≥3 contiguous leads) or a positive troponin value (the latter becoming a MI outcome, as previously discussed, during the course of the study). Secondary outcomes included singly and in various combinations death, cardiovascular death, MI, and UA, the latter defined as previously as well as in 2 other less stringent ways: 1) a broad definition of a characteristic chest pain syndrome requiring hospitalization (referred to as "soft UA") and 2) the same as the soft UA plus requiring a supporting diagnostic test (referred to as "test positive UA").

Although there were no formal age limits or comorbidity exclusion criteria, patients who could not comprehend the consent form or who were too ill to sign it or in whom a return visit 1 month after hospital discharge was felt to be problematic were not recruited. A registry was kept of all potentially eligible patients detailing reasons for exclusion.

Blood samples were taken: 1) acutely; 2) at hospital discharge; and 3) 1 month after hospital discharge. After centrifugation, serum and plasma were distributed in aliquots, stored locally at −70°C and then transferred on dry ice to the core laboratory. There, samples were stored at −80°C until analysis for CRP and cardiac troponin T (acute

sample only). All measurements were performed in a single batch. The CRP was measured with the N High-Sensitivity CRP mono assay using the BN ProsPec Nephelometer (Dade Behring, Deerfield, Illinois). Cardiac troponin T was measured using a commercial assay (Roche Inc, Mannheim, Germany) with a detection limit for myocardial injury of 0.1 μg/l.

All basic demographic and medical data, conventional risk factors, clinical and paraclinical diagnostic and therapeutic information including recurrent ischemia, cardiac function, and use of invasive cardiac procedures were recorded in a comprehensive case report form. This information was independently verified for consistency and, then in all cases, systematically reverified by on-site visits. Items in the patient history such as previous heart failure or MI and clinical features such as heart failure during the index hospitalization required firm documentation that fulfilled predefined criteria. The ECG motivating the admission and the final ECG before hospital discharge were obtained. The ECG analysis was performed at the core center. Follow-up was ascertained 1 month after hospital discharge (at the time of blood sampling) and by telephone contact at 1 year, obtaining, where necessary, confirmatory hospital files. All study outcomes were systematically recorded, as well as all other hospitalizations and their causes, cardiac catheterizations, and percutaneous and surgical revascularizations. All events were verified by on-site visits and examination of all necessary supporting documents. Finally, all prospective and potential outcomes were centrally adjudicated independently by 2 cardiologist investigators. In the case of a disagreement, the final decision was reached by a third cardiologist investigator.

Statistical analysis. Our sample size was calculated on an anticipated 10% 1-year composite event rate. Therefore, recruitment of 1,200 patients would result in about 120 events, providing sufficient precision for the numerous requisite prediction parameters that would be included in a multivariate model (10 to 20 events per regression term). Continuous and dichotomous variables were analyzed using Student *t* test and the chi-square test, respectively. The cohort was divided into quartiles of CRP values obtained at each of the 3 points (admission, discharge, 1 month later) to obtain event curves from the Nelson-Aalen estimator of the cumulative hazard rate. The log-rank test and a test for trends were used to compare quartiles. For logistic regression analyses, the selection process was performed using univariate analysis of each variable. Variables whose univariate test had a p value <0.20 were entered into a multivariate model. The selected variables were analyzed using both stepwise forward and backward approaches in the logistic regression model. Both approaches gave similar results. Analyses of CRP were based on log-transformed values. The results were considered significant with p values <0.05. The data were analyzed using the statistical package program SAS version 9.1.3 (SAS Institute Inc., Cary, North Carolina).

Results

During the period of recruitment (2001 to 2002), 2,672 patients were admitted with chest pain or discomfort in the 8 participating centers. This number excludes any patients who died in the emergency room before they could be approached for this study. A flow diagram depicts the reasons for exclusions in the constitution of the study cohort (Fig. 1). The clinical characteristics of the 1,210 patients of the study (mean age 62 ± 12 years, 75% males) are shown in Table 1. About one-half of the patients had a previous history of coronary artery disease. The admission diagnosis was MI in 772 (64%) patients with UA in the remainder. During initial hospitalization, there were 364 (30%) patients with ST-segment elevation MI of whom 77% received reperfusion therapy (235 patients had fibrinolytic therapy and 45 patients had primary percutaneous coronary intervention [PCI]). Coronary angiography was performed at some time during initial hospitalization in 678 (56%) patients; PCI was performed in 437 (36%) patients and coronary bypass surgery in 112 (9%) patients. Heart failure occurred in 109 (9%) patients.

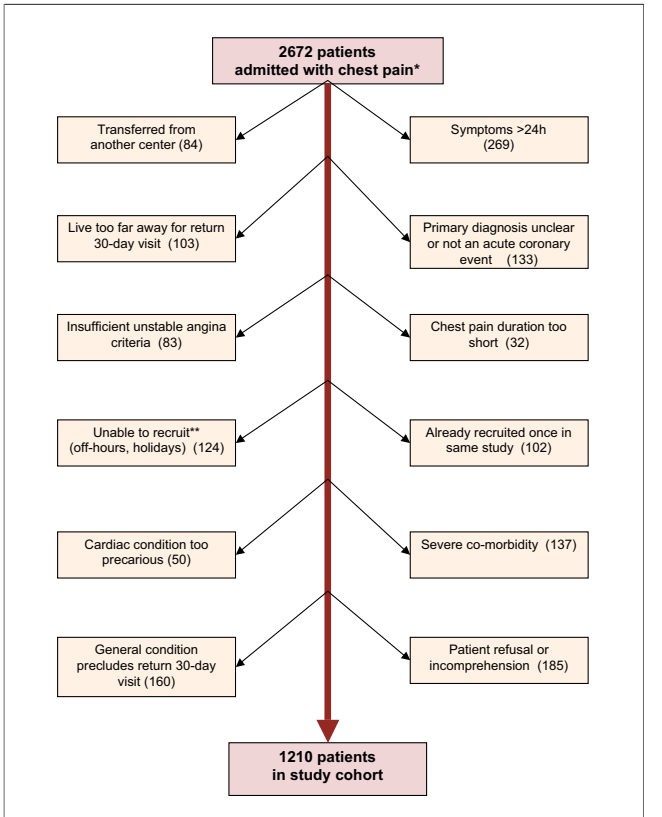

Figure 1 Selection of Study Cohort

Flow diagram of screened patients admitted with chest pain, depicting reasons for exclusions in selection of study cohort. *Excludes any patients who died before they could be approached for this study. **In some centers.

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Table 1 Clinical Characteristics of Study Group

	Patients (n = 1,210)
Age, yrs	62 ± 12
Male	903 (75%)
Current smoker	368 (30%)
Arterial hypertension	616 (51%)
Diabetes	242 (20%)
Previous MI	341 (28%)
History of angina	451 (37%)
History of arterial (noncoronary) disease	247 (20%)
History of cerebrovascular disease	106 (9%)
History of heart failure	69 (6%)
Previous PCI	220 (18%)
Previous CABG	176 (15%)
First ever cardiac hospitalization	618 (51%)

CABG = coronary artery bypass graft; MI = myocardial infarction; PCI = percutaneous coronary intervention.

The composite primary outcome occurred at 1 year in 142 (11.7%) patients. Follow-up was obtained in 1,207 of the 1,210 patients. Central adjudication of outcomes was performed by agreement of 2 investigators in all cases but 1 that required the intervention of a third investigator. Clinical events during initial hospitalization, at 1 month, and at 1 year are detailed in Table 2. Univariate analyses of clinical characteristics in patients with and without occurrence of the primary end point at 1 year are shown in Table 3. Significant univariate clinical predictors included age, female gender, diabetes with and without insulin, history of heart failure, history of MI, heart failure developing during initial hospitalization, admission heart rate >100/min, ST-segment depression on admission, glycemia on admission, left ventricular ejection fraction <35%, and renal insufficiency (creatinine clearance <50 ml/min).

All 1,210 patients had an admission CRP measurement; a hospital discharge CRP was obtained in 95.5% and a 1-month CRP was obtained in 91.4% of patients. The CRP values at admission, discharge, and at 1 month in patients with and without the primary outcome at 1 year are shown in Table 4 with corresponding unadjusted odds ratios and 95% confidence intervals (CIs). The CRP at admission and at 1 month after hospital discharge had modest ability (1.20 [95% CI 1.06 to 1.36] and 1.23 [95% CI 1.00 to 1.50], respectively) to predict occurrence of the primary outcome at 1 year. The CRP value at hospital discharge was not predictive of the primary outcome at 1 year.

The CRP values at admission, hospital discharge, and 1 month later of patients in whom the primary outcome as well as its individual components occurred compared with patients without events are shown in Figure 2. Although patients who were dying had higher CRP values than survivors, there was otherwise no difference in CRP concentrations between those with and without events. Results were similar when analyzed from the perspective of CRP quartiles (data not shown). Admission CRP quartiles stratified risk of death at 1 year ($p = 0.001$, log-rank test for trend) and drove the prediction of the primary outcome ($p = 0.01$, log-rank test for trend). Admission CRP quartiles did not predict the occurrence of MI at 1 year ($p = 0.3$, log-rank test for trend). Nor did the analysis of quartiles of CRP at hospital discharge or 1 month later provide additional data in terms of the prediction of events at 1 year (data not shown).

The independent predictors for the occurrence of the primary outcome at 1 year were age, diabetes with insulin therapy, a history of heart failure, and heart failure occurring during initial hospitalization (Table 5). The association of CRP with the occurrence of the primary outcome at 1 year was no longer statistically significant after multivariate adjustment for these independent predictors (Table 5).

Independent predictors of death alone at 1 year were age, a history of heart failure, ST-segment depression at admission, glycemia at admission, and heart failure occurring during initial hospitalization. The ability of CRP at admission and at hospital discharge to predict death was again no longer present after multivariate adjustment (Table 6). Use of a dichotomous CRP value (>3 mg/l signifying increased risk) (18) at the 3 points did not change these findings.

We repeated all analyses substituting the broader UA definitions of soft UA and test positive UA. We then repeated all analyses replacing death due to any cause with cardiovascular death. None of these analyses yielded positive results for CRP after multivariate adjustment. Again, as for the primary outcome, CRP at admission had modest unadjusted predictive value for cardiovascular death at 1 year and CRP at 1 month had similar predictive value at 1 year. These results were not sustained after multivariate adjustment. In all these analyses, CRP at hospital discharge showed no predictive value. Finally, because recent angiography and/or PCI and coronary bypass surgery could raise CRP measured at hospital discharge and confound interpretation, we examined the predictive ability of discharge CRP in those patients who did not have coronary bypass surgery at any time before hospital discharge blood was taken nor coronary angiography and/or PCI in the previous

Table 2 Clinical Events on Follow-Up

	Initial Hospitalization	1 Month	1 Year
Death	21 (1.7%)	33 (2.7%)	58 (4.8%)
Cardiovascular death	21 (1.7%)	32 (2.6%)	50 (4.1%)
MI (by troponin definition)	21 (1.7%)	39 (3.2%)	79 (6.5%)
UA (clinical diagnosis or soft UA)*	—	45 (3.7%)	148 (12.2%)
UA (with supporting diagnostic test)*	—	23 (1.9%)	75 (6.2%)
UA (with ECG changes)*	—	6 (0.005%)	26 (2.2%)
Primary outcome (death, MI, or UA with ECG changes)*	—	69 (5.7%)	142 (11.7%)
Any nonelective rehospitalization	—	136 (11.4%)	413 (34.1%)

*Not counted during initial hospitalization.
ECG = electrocardiogram; UA = unstable angina; other abbreviation as in Table 1.

Table 3 Univariate Analysis of Clinical Characteristics in Patients Without and With Occurrence of the Primary End Point at 1 Year

	No Primary End Point (n = 1,068)	Primary End Point (n = 142)	p Value
Age, yrs	61.6 ± 11.4	68.2 ± 11.5	<0.001
% males	811 (75.9%)	92 (64.8%)	0.004
History of myocardial infarction	284 (26.6%)	57 (40.1%)	0.0007
History of heart failure	42 (3.9%)	27 (19%)	<0.0001
History of diabetes without insulin	140 (13.1%)	31 (21.8%)	<0.0001
History of diabetes with insulin	50 (4.7%)	21 (14.8%)	<0.0001
HR >100/min on first ECG	68 (6.4%)	21 (14.8%)	0.0003
↓ ST-segment on admission	149 (14%)	38 (28.8%)	<0.0001
↑ ST-segment on admission	325 (30.4%)	40 (28.2%)	0.6
Admission glycemia, mmol/l	7.7 ± 3.0	9.2 ± 4.0	<0.0001
CK >1,000 U/l	249 (23.5%)	25 (17.6%)	0.12
Troponin diagnostic of myocardial injury	621 (58.2%)	85 (60.7%)	0.6
Ejection fraction <35%	42 (4.0%)	20 (14.5%)	<0.0001
Heart failure during index hospitalization	68 (6.4%)	42 (29.6%)	<0.0001
Creatinine clearance <50 ml/min	509 (47.8%)	93 (65.5%)	<0.0001

CK = creatine kinase; ECG = electrocardiogram; HR = heart rate.

48 h. The univariate odds ratio of CRP at hospital discharge to predict the primary outcome at 1 year in these 742 patients was 1.09 (95% CI 0.92 to 1.29; p = 0.32). Results were unchanged if this analysis was enlarged to include patients who had coronary angiography but not PCI in the 48 h preceding the obtaining of the CRP hospital discharge sample.

Discussion

This multicenter prospective study was specifically designed to determine whether there is clinical prognostic utility to measuring CRP, which is the most studied marker of inflammatory status, following an acute coronary episode. We tested this "inflammatory hypothesis" at 3 points that are potentially relevant on mechanistic grounds in addition to being practical: acutely, at hospital discharge because this is the time when patients are considered stabilized whatever the length of the hospital stay, and 1 month later because this is the time when persistent inflammation might augur recurrent instability as well as being generally within the time frame for an outpatient visit after the acute episode. Additionally, CRP blood levels 1 month after hospital discharge might be less likely to be confounded by the inflammatory contribution of myocardial necrosis if the acute event was MI.

We found that CRP acutely and 1 month after hospital discharge had modest predictive value for the occurrence of a combined outcome of death, MI, and UA, and particularly for death alone. This predictive value was no longer present after adjustment for readily available clinical prognosticators. The CRP measured at hospital discharge showed no predictive value. This latter "negative" result remained unchanged when the analysis was restricted to patients who did not have cardiac surgery or recent coronary angiography and/or PCI.

Our findings suggest that there is little if any incremental clinical utility to measuring CRP in acute coronary disease at any of these 3 points. An examination of the predictive ability of the individual components of the primary outcome showed that it was the future occurrence of death, and not MI or UA, that was associated with higher CRP values at admission and at 1 month after hospital discharge. This could be explained by the stimulus of inflammatory cytokine release that occurs in confounding conditions such as heart failure as well as other comorbidities and organ dysfunctions that are associated with decreased survival (22–24). Other studies have also shown that CRP predicts death after an acute coronary event but have not always adjusted for other important and readily available predictors of increased mortality (25–27). We found that age, diabetes, a history of heart failure, and heart failure developing during the index hospitalization were strong independent predictors of mortality after an acute coronary syndrome. After adjustment

Table 4 CRP Values (mg/l; Mean ± SD) in Relation to Primary End Point and Unadjusted Odds Ratios

	Event at 1 Year (n = 142)	No Event at 1 Year (n = 1,068)	Unadjusted Odds Ratio (95% CI)
CRP at hospital admission	19.5 ± 44.2	13.6 ± 26.1	1.20 (1.06–1.36)
CRP at hospital discharge	18.8 ± 28.7	22.1 ± 33.4	0.98 (0.85–1.14)
CRP at 1 month after discharge	5.5 ± 7.5	4.9 ± 10.1	1.23 (1.00–1.50)

CI = confidence interval; CRP = C-reactive protein.

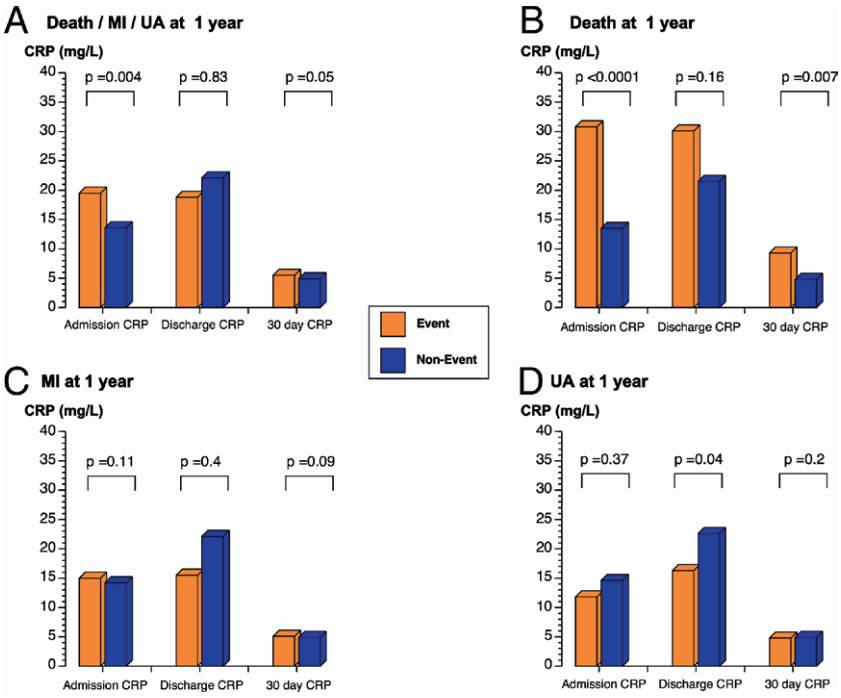

Figure 2 **CRP Values in Patients With and Without Events**

The C-reactive protein (CRP) values at admission, at hospital discharge, and at 1 month after discharge in patients with and without occurrence of the primary outcome (death, myocardial infarction [MI] by troponin definition, or unstable angina [UA] with electrocardiographic changes) and its individual components.

for these factors, an independent contribution of CRP was not demonstrated.

Indeed, previous studies that have suggested that the measurement of CRP has clinical utility in patients with acute coronary syndromes have several limitations. They have involved small and/or selected (nonconsecutive) cohorts (3,5,12–18,20,21,27). They have been retrospective and perhaps subject to publication bias (5,13,14,21,27). They have especially tended not to adequately adjust for readily available clinical parameters such as a history of heart failure, the presence of ECG changes, the presence or degree of myocardial necrosis, the status of left ventricular function and renal function, the presence of diabetes, and importantly the occurrence of heart failure during the index hospitalization (3,5,12–15,17,18,20,21,25–27). The strengths of this prospective study in addressing the question of whether there is predictive clinical utility in monitoring inflammatory status in patients with an acute coronary syndrome are its design, carefully validated clinical characterization, relatively large sample size, the inclusion of a broad spectrum of patients afflicted with an acute coronary syndrome, the examination of CRP at 3 pertinent points, the consideration of a detailed combination of clinical outcomes, and adjustment for relevant and readily available clinical prognosticators. The rates of invasive cardiac interventions are also fairly comparable to those found in contemporary registry studies of patients with acute coronary syndromes (28–30).

Table 5 **Independent Predictors and Adjusted Odds Ratios of CRP for the Occurrence of the Primary Outcome at 1 Year**

	Odds Ratio (95% CI)	p Value
Age, yrs	1.04 (1.02–1.06)	<0.001
Diabetes treated with insulin	2.58 (1.39–4.79)	0.02
History of heart failure	2.28 (1.25–4.18)	0.008
Heart failure during index hospitalization	1.75 (1.37–2.27)	<0.001
CRP at hospital admission	1.04 (0.91–1.20)	0.56
CRP at hospital discharge	0.90 (0.77–1.06)	0.20
CRP at 1 month after discharge	1.12 (0.93–1.34)	0.24

Abbreviations as in Table 4.

Table 6 **Independent Predictors and Adjusted Odds Ratios of CRP for the Occurrence of Death at 1 Year**

	Odds Ratio (95% CI)	p Value
Age, yrs	1.1 (1.06–1.14)	<0.001
History of heart failure	2.5 (1.2–5.4)	0.017
ST-segment depression on admission	2.6 (1.4–5.1)	0.004
Glycemia on admission	1.1 (1.0–1.2)	0.038
Heart failure during index hospitalization	5.4 (2.7–10.7)	<0.001
CRP at hospital admission	1.1 (0.9–1.4)	0.4
CRP at hospital discharge	1.1 (0.8–1.4)	0.7
CRP at 1 month after discharge	1.4 (0.9–2.1)	0.14

Abbreviations as in Table 4.

The notion that an inflammatory marker might predict acute coronary recurrence has rested on the assumption that persistent inflammation, presumably both systematically and more specifically within the coronary arterial circulation, would be associated with or might even stimulate coronary atherosclerotic plaque reactivation and recurrent coronary instability. The purest expression of the latter is acute MI because it is clearly related to the presence of occlusive thrombus on an active ruptured or eroded plaque. This pathophysiological link is less clear for death or even for cardiovascular death where the underlying substrate is likely more heterogeneous because other immediate causes besides pro-inflammatory plaque rupture may be implicated such as arrhythmia, cardiac failure, and contributory comorbidity. The consistent link with inflammatory plaque rupture is also less clear for UA, because the latter can be a more benign and uncertain diagnosis as well as a more heterogeneous entity that may implicate other destabilizing mechanisms such as increased myocardial demand on a substrate of critically reduced coronary arterial reserve. Therefore, the ideal mechanistic "terrain" on which to test the inflammatory hypothesis—that a raised CRP value at some point in the clinical evolution of patients hospitalized with an acute coronary syndrome might be a harbinger for recurrent or persistent acute coronary instability—is the subset of patients in whom MI occurs on follow-up. The finding of this study is especially pertinent in this regard because no statistically significant relation was found between CRP measured acutely, at hospital discharge, or 1 month later and the occurrence of this clinical event. This suggests that the mechanism underlying the occurrence of acute MI in patients previously hospitalized with an acute coronary syndrome is more complex than the presence of a persistent and heightened—and measurable—inflammatory blood profile.

Conclusions

In summary, this prospective study in a contemporary and broad spectrum of patients hospitalized with an acute coronary syndrome has found that CRP measured acutely and 1 month after hospital discharge has modest, but not independent, ability to predict death but does not predict MI or UA. The CRP measured at hospital discharge appears to have no predictive ability. This study does not support clinical use of CRP to aid clinical management in patients hospitalized with an acute coronary syndrome. The need for better predictive tools in these patients remains an elusive clinical challenge.

Acknowledgments

The authors thank Rémy Theriault, PhD, Department of Research, Laval Hospital, Quebec City, Quebec, Canada, for the creation and maintenance of the database. They are grateful for the hard work and dedication of the research personnel listed in the Online Appendix.

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APPENDIX

For a list of the participating centers of the study, please see the online version of this article.