

A clinical risk score identified high-risk patients with chest pain but no ST-segment deviation and normal troponin levels

Sanchis J, Bodí V, Núñez J, et al. A practical approach with outcome for the prognostic assessment of non-ST-segment elevation chest pain and normal troponin. *Am J Cardiol.* 2007;99:797-801.

Clinical impact ratings: Emergency Med ★★★★★☆ GIM/FP/GP ★★★★★☆ Hospitalists ★★★★★☆ Cardiology ★★★★★☆

QUESTION

In patients with suspected acute coronary syndromes (ACSs) but no ST-segment deviation or important increases in troponin levels, does a clinical risk score (CRS) identify patients at increased risk for death or myocardial infarction (MI) at 1 month and in the longer term?

METHODS

Design: Prospective cohort study.
Setting: Emergency department (ED) of a university hospital in Spain.
Patients: Patients presenting to the ED with chest pain of possible coronary origin were evaluated by a cardiologist using a protocol consisting of clinical history, a previously developed chest pain questionnaire, electrocardiogram, serial troponin I determination, and early exercise testing in eligible patients. Patients with ST-segment elevation, new left branch bundle block, history of nonischemic heart disease, or heart failure at admission were excluded. Based on the results of the tests, the patients were divided into 3 cohorts: those with troponin increases ($n = 552$, mean age 68 y, 69% men), those with ST-segment depression but normal troponin levels ($n = 106$, mean age 68 y, 68% men), and those with no ST-segment deviation and normal troponin levels. The last cohort was further divided, based on a CRS that was previously derived and validated in the same group of patients, into those at high risk for a poor prognosis ($n = 158$, mean age 70 y, 71% men) and those at low risk ($n = 633$, mean age 62 y, 66% men). Patients received appropriate treatment at their physician's discretion.

Description of prediction guide: The CRS (range 0 to 6) categorized patients into low-risk (score < 3) or high-risk (score \geq 3) groups. The CRS was a summation of 5 clinical variables: score \geq 10 points on the chest pain questionnaire, \geq 2 episodes of chest pain in < 24 hours, age \geq 67 years, insulin-dependent diabetes (2 points), and previous percutaneous coronary angioplasty.
Outcomes: A composite endpoint of death or nonfatal MI at 30 days and longer term (median duration of follow-up 26 mo).

MAIN RESULTS

Risk for death or MI, at both 30 days and longer term, was lowest in the group with no ST-segment deviation and normal tro-

ponin levels that was categorized by the CRS as low risk; the other 3 groups did not differ (Table).

CONCLUSION

A simple clinical risk score identified a subgroup of high-risk patients with chest pain, non-ST-segment deviation, and normal troponin levels for whom the outcome was similar to that of patients with increased troponin levels or ST-segment depression.

Sources of funding: Instituto de Salud Carlos III and Fundación Española del Corazón.

For correspondence: Dr. J. Sanchis, Hospital Clinic Universitari, València, Spain. E-mail sanchis_juafor@gva.es. ■

Composite endpoint of death or myocardial infarction in patients with non-ST-segment elevation chest pain, categorized by presence or absence of ST-segment depression and increased troponin levels and, in the absence of both, by risk score*

Risk group	Incidence at 30 d (95% CI)	Adjusted odds ratio at 30 d (CI)†	Incidence at median 26 mo (CI)	Adjusted hazard ratio at median 26 mo (CI)†
Low risk (score < 3) non-ST-segment depression, normal troponin	1.7% (0.9 to 3.1)	0.2 (0.1 to 0.4)	9.3% (7.2 to 12)	0.4 (0.3 to 0.6)
High risk (score \geq 3) non-ST-segment depression, normal troponin	10.8% (6.4 to 17)	0.9 (0.5 to 1.7)	26% (19 to 34)	0.7 (0.5 to 1.1)
ST-segment depression, normal troponin	6.6% (2.7 to 13)	0.6 (0.3 to 1.4)	30% (22 to 40)	1.2 (0.8 to 1.7)
Troponin increase	9.4% (7.1 to 12)	1 (reference)	25% (21 to 29)	1 (reference)

*CI defined in Glossary.

†Odds ratios and hazard ratios adjusted for other risk factors, such as age, hypertension, diabetes, and previous myocardial infarction.

COMMENTARY

Accurate risk stratification is the cornerstone of the often-challenging task of evaluating a patient with suspected ACS. The difficulty is heightened by imperfect biomarkers, the limited utility of a normal electrocardiogram, and the fact that most patient presentations ultimately "rule out" ACS. Discriminating between patients at high and low risk for short-term adverse events is crucial for deciding which patients require aggressive treatment strategies (early percutaneous coronary intervention) and which patients can be safely discharged.

The work by Sanchis and colleagues points to the potential value of a 5-item CRS in identifying high-risk (and low-risk) patients among those who are troponin-negative and do not demonstrate ST-segment deviations. However, these findings are constrained by a relatively narrow, single-center validation, reliance on composite endpoints as outcomes for both follow-up periods, the apparent lack of blinding in outcome assessment and rule interpretation, and the relatively wide confidence intervals around the adverse event rates in the low-risk group (upper 95% confidence limit 3% at 30 d).

Fortunately, there is a more accurate, discriminating, and widely validated CRS that can provide useful guidance. The easy-to-use and intuitive 7-item Thrombolysis In Myocardial Infarction (TIMI) risk score, derived and initially validated in 2 large populations of patients with ACS (in contrast to the CRS of Sanchis and colleagues, which was derived in patients with acute chest pain of uncertain coronary origin), is the most well-studied CRS in ACS, showing utility in patients with a clear diagnosis, as well as those with undifferentiated chest pain (1-3). Although not sufficiently sensitive to "rule-out" ACS and discharge patients on its own, the TIMI score provides meaningful and graded measures of risk that can serve as powerful supplements to clinical judgment.

Eddy Lang, MD
 SMBD Jewish General Hospital, McGill University
 Montreal, Quebec, Canada

References

1. Antman EM, Cohen M, Bernink PJ, et al. *JAMA.* 2000;284:835-42.
2. Chase M, Robey JL, Zogby KE, et al. *Ann Emerg Med.* 2006;48:252-9.
3. Pollack CV Jr., Sites FD, Shofer FS, Sease KL, Hollander JE. *Acad Emerg Med.* 2006;13:13-8.