Incidence and outcomes of unstable angina compared to NSTEMI

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Background

Unstable angina and non-ST-elevation myocardial infarction (NSTEMI) are often thought to have similar incidence, characteristics, pathophysiology, and outcome, and are therefore treated similarly. The advent of high-sensitivity cardiac troponin (hs-cTn) assays has markedly improved the detection of myocardial necrosis. Little data exists concerning unstable angina in the era of hs-cTn.

Methods

Two independent prospective multicenter diagnostic studies (Advantageous Predictors of Acute Coronary Syndromes Evaluation (APACE) and High-Sensitivity Troponin in the Evaluation of Patients With Acute Coronary Syndrome (High-STEACS)) enrolling patients with acute chest discomfort presenting to the emergency department. Central adjudication of the final diagnosis was done by two independent cardiologists using all clinical information including serial measurements of high-sensitivity cardiac troponin (hs-cTn). All-cause death and future non-fatal MI were assessed at 30-days and 1-year. Unstable angina was diagnosed in patients with ischemic symptoms and no evidence of acute myocardial necrosis (rise and/or fall in hs-cTn, with at least one measurement above the 99th percentile).

The following criteria were interpreted as increasing the likelihood, although were not mandatory for the diagnosis of unstable angina: typical angina pectoris at rest; worsening/deterioration of a previously stable angina; cardiac stress test showing myocardial ischaemia; coronary angiography revealing a diameter stenosis of at least 70%, fractional flow reserve documenting functional significance of a coronary lesion, and sudden cardiac death or myocardial infarction occurred during 60-day follow-up. However, perhaps reflecting some imprecision in the current diagnostic guidelines, the significance of troponin in the adjudication of unstable angina was considered differently in the APACE and High-STEACS cohorts. In the appropriate clinical context, patients in APACE could be adjudged to have unstable angina in the presence of mild hs-cTn elevations above the 99th percentile without dynamic changes on serial testing. In contrast, within the High-STEACS population, any measurement of hs-cTn above the 99th percentile precluded this diagnosis being made. To investigate the clinical significance of this diagnostic discrepancy, a post-hoc sensitivity analysis was performed aligning the definitions.

Results

8992 patients were enrolled at 11 centres. UA was adjudicated in 8.9% [95%CI 8.0-9.7] and 2.8% [2.3-3.3] patients in APACE and High-STEACS, respectively, and NSTEMI in 15.1% [14.0-16.2] and 13.4% [12.4-14.3]. Coronary artery disease was pre-existing in 73% and 76% of patients with UA. At 30-days, all-cause mortality in UA was substantially lower than as compared to NSTEMI (0.5% versus 3.7%, p=0.002 in APACE, 0.7% versus 7.4%, p=0.004 in High-STEACS). Similarly, at 1-year in UA all-cause mortality was 3.3% [1.2-5.3] vs 10.4% [7.9-12.9] in APACE, and 5.1% [0.7-9.5] vs 22.9% [19.3-26.4] in High-STEACS, and similar to non-cardiac chest pain (NCCP). In contrast, future non-fatal MI in APACE was comparable in UA and NSTEMI (11.2%, [7.8-14.6] and 7.9%, [5.7-10.2]), and higher than in NCCP (0.6%, [0.2-1.0]). In multivariable analysis, patients with UA had a mortality at 1-year similar to patients with NCCP (Hazard ratio (HR) 1.3, 95% CI 0.7-2.6).

In the total cohort, 1-year mortality and rate of future MI was lower in UA patients without vs with any hs-cTn concentration above the 99th percentile (2.6% vs 9.5%, p=0.001, and 5.7% vs 14.6%, p=0.001, respectively.

Conclusion

The relative incidence and mortality of UA is substantially lower than that of NSTEMI, while the rate of future non-fatal MI is similar.

Conflicts of Interest

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