Quality Improvement

High-Sensitivity Cardiac Troponin I vs a Clinical Chemistry Score for Predicting All-Cause Mortality in an Emergency Department Population

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ABSTRACT

Background: For patients investigated for suspected acute coronary syndrome, there is uncertainty if a single measurement of high-sensitivity cardiac troponin I (hs-cTnI) at emergency department (ED) presentation can identify patients at both low and high risk for mortality.

Methods: We included consecutive adult patients in the ED who had a Clinical Chemistry Score (CCS) taken at presentation (ie, combination of glucose, creatinine for estimated glomerular filtration rate determination, and hs-cTnI assay) in a Canadian city between 2012 and 2013. Outcomes were 3-month, 1-year, and 5-year all-cause mortality using the provincial death registry. Mortality rates and test performance (eg, sensitivity and specificity) with 95% confidence intervals (CIs) were obtained for the CCS or hs-cTnI assay alone using established cutoffs for these tests.

Results: Our cohort included 5974 patients with a 1-year mortality rate of 17.2% (95% CI, 16.2-18.3). A CCS ≥ 1 yielded a sensitivity of

RÉSUMÉ

Contexte : Dans le cas des patients chez qui l'on soupçonne un syndrome coronarien aigu, des doutes subsistent à savoir si la mesure de la troponine I cardiaque à haute sensibilité (TnIc-hs) à l'arrivée au service des urgences peut, à elle seule, permettre de repérer les patients présentant un risque de mortalité faible ou élevé.

Méthodologie : L'étude portait sur les patients adultes qui se sont présentés consécutivement au service des urgences dans une ville canadienne entre 2012 et 2013 et pour lesquels un score CCS (*Clinical Chemistry Score*, ou score des paramètres biochimiques cliniques, c'est-à-dire glycémie, créatininémie [aux fins du calcul du débit de filtration glomérulaire estimé] et dosage de la TnIc-hs) a été établi à leur arrivée. Les critères d'évaluation étaient la mortalité toutes causes confondues à 3 mois, à 1 an et à 5 ans, déterminée à partir des actes de décès inscrits au registre provincial. Les taux de mortalité et la fiabilité des tests (sensibilité et spécificité) avec des intervalles de confiance (IC) à 95 % ont été déterminés pour le score CCS et pour le

In patients with suspected acute coronary syndrome (ACS), multicenter studies worldwide have indicated that a single measurement of high-sensitivity cardiac troponin I (hs-cTnI)

≥ 5 ng/L at emergency department (ED) presentation may be suitable to rule out myocardial infarction (MI) and identify a low-risk group of patients who may avoid unnecessary hospital admissions.[1-3] These findings were generated with an Abbott Laboratories (Abbott Park, IL) hs-cTnI assay, and recent publications demonstrating a 5 ng/L cutoff might be suitable for one of Siemens Healthcare Diagnostics (Munich, Germany) hs-cTnI assays, suggesting an overall low concentration cutoff of 5 ng/L may be applicable to more than 1 hs-cTnI assay.[4,5] Accordingly, this leads to the concept of a single rule-out cutoff when using hs-cTnI assays, which has been published by the European Society of Cardiology guidelines[6] and most recently discussed in the Fourth Universal

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99.2% (95% CI, 98.4-99.6) compared with the hs-cTnI ≥ 5 ng/L cutoff sensitivity of 88.4% (95% CI, 86.3-90.3), with the mortality rate being significantly lower for patients with CCS < 1 (2.0%; 95% CI, 0.9-4.0) vs patients with hs-cTnI < 5 ng/L (5.0%; 95% CI, 4.2-6.0) at 1 year ($P = 0.01$). A CCS of 5 also yielded a higher specificity (88.5%; 95% CI, 87.5-89.3) compared with hs-cTnI > 26 ng/L (83.9%; 95% CI, 82.9-84.9), with no difference in mortality rates (37.4% vs 36.3%; $P = 0.66$). This trend was consistent at 3-month and 5-year mortality.

Conclusion: For patients in the ED with a potential cardiac issue, using the CCS cutoffs can better identify patients at low and high risk for mortality than using published cutoffs for hs-cTnI alone.

dosage de la TnIc-hs seulement au moyen des valeurs seuils établies pour ces tests.

Résultats : La cohorte réunissait 5 974 patients, et le taux de mortalité à 1 an s'établissait à 17,2 % (IC à 95 % : 16,2-18,3). Un score CCS ≥ 1 a été associé à une sensibilité de 99,2 % (IC à 95 % : 98,4-99,6) comparativement à 88,4 % (IC à 95 % : 86,3-90,3) pour une valeur seuil de TnIc-hs ≥ 5 ng/l, le taux de mortalité à 1 an étant significativement plus bas chez les patients ayant un score CCS < 1 (2,0 %; IC à 95 % : 0,9-4,0) que chez ceux ayant un taux de TnIc-hs < 5 ng/l (5,0 %; IC à 95 % : 4,2-6,0) ($p = 0,01$). Un score CCS de 5 a en outre été associé à une plus grande spécificité (88,5 %; IC à 95 % : 87,5-89,3) qu'un taux de TnIc-hs > 26 ng/l (83,9 %; IC à 95 % : 82,9-84,9); il n'y avait pas de différence entre les taux de mortalité (37,4 % vs 36,3 %; $p = 0,66$). Les résultats relatifs à la mortalité à 3 mois et à 5 ans concordaient avec cette tendance.

Conclusion : Dans le cas des patients admis au service des urgences en raison d'un problème cardiaque potentiel, les valeurs seuils du score CCS peuvent permettre de mieux repérer les patients qui présentent un risque de mortalité faible ou élevé, comparativement aux seules valeurs seuils publiées des taux de TnIc-hs.

Definition of MI, which provided the following balanced statement: "Some studies indicate that the single sample approach provides optimal sensitivity and negative predictive accuracy in patients otherwise at low risk and those with a normal electrocardiogram. However, one concern about short rule-out periods is that the precision of the assays may not permit small differences to be distinguished."[7]

In this regard, we have provided evidence of analytical issues that affect interpretation around and below 5 ng/L.[8-12] Laboratory recommendations on appropriate monitoring of the hs-cTnI assays will help mitigate some of these issues;[13] however, what has not been evaluated thoroughly is the impact of a low hs-cTnI result for risk stratification in a general ED population in North America. To address this gap, our goal was to compare Abbott hs-cTnI published cutoffs (eg, 5 ng/L and the overall 99th percentile of 26 ng/L) alone vs a simple laboratory algorithm (ie, Clinical Chemistry Score [CCS])[14] at presentation in a general ED population to determine low- and high-risk patients for subsequent all-cause death.

Methods

Study design and population

After research ethics board approval was obtained, from November 28, 2012, to February 28, 2013, all ethyl-enediaminetetraacetic acid blood samples from the ED at 3 adult hospitals in Hamilton, Ontario, Canada, that had an order for cardiac troponin had both the standard cardiac troponin I (cTnI) and hs-cTnI tests performed on the Abbott ARCHITECT instruments, with only the cTnI results being clinically reported. Patients included in this study were adult patients who were investigated for possible ACS as demonstrated by emergency staff ordering the "ED cardiac presentation panel," which included troponin I, glucose, creatinine, and other laboratory tests at the Hamilton General Hospital, St. Joseph's Healthcare Hamilton, and Juravinski Hospital

(note, troponin I used for clinical decision making during this timeframe was the Abbott Laboratories contemporary sensitive cTnI assay).[15] The Abbott hs-cTnI concentrations (limit of detection for the assay being 1 ng/L)[15] were obtained in real-time and on the same instruments in the hospitals with the hs-cTnI results not reported to the treating physician.[16]

The flow diagram in Supplemental Figure 1 shows the cohort selection. Briefly, only the presentation hs-cTnI result on the first ED encounter on 6641 patients who presented to the ED was used with further selection dependent on also having a glucose and creatinine result (ie, the "ED cardiac presentation panel," with creatinine for an estimated glomerular filtration rate [eGFR] as calculated by the CKD-Epidemiology Collaboration equation being performed in 2019).[17] Patients were also excluded if they were not Ontario residents, if age and sex were missing in the registered persons database (n = 625 individuals excluded), or if they were not Ontario Health Insurance Plan eligible at baseline (n = 42). The final cohort consisted of 5974 individuals with the treating emergency physicians blinded to the hs-cTnI in addition to eGFR results calculated by the CKD-Epidemiology Collaboration equation.

High-sensitivity cardiac troponin I cutoffs and CCS

The cutoffs used for the Abbott Laboratories hs-cTnI assay were < 5 ng/L, 5 to 26 ng/L, and > 26 ng/L, as previously used in patients with suspected ACS.[1-3,14] The simple laboratory algorithm or CCS has been published in both a multicenter Canadian study and an international study.[14,18] Results from glucose, eGFR, and hs-cTnI are converted into an ordinal scale, with the sum ranging from 0 (low risk) to 5 (high risk).[14,18] The scores are generated as follows: glucose < 5.6 mmol/L = 0 points or ≥ 5.6 mmol/L = 1 point; eGFR ≥ 90 mL/min/1.73 m² = 0 points or < 90 mL/min/1.73 m² = 1 point; hs-cTnI < 4 ng/L = 0 points or 4-14 ng/L = 1 point or 15-30 ng/L = 2 points or > 30 ng/L = 3 points.[14,18] The CCS was calculated by the sum of the points from glucose,

Table 1. Characteristics of all-comer ED population classified by the CCS or hs-cTnI concentration categories

Characteristic		Clinical chemistry score							hs-cTnI concentration			
		0	1	2	3	4	5	P value	< 5 ng/L	5-26 ng/L	> 26 ng/L	P value
		N = 399	N = 923	N = 1405	N = 1486	N = 853	N = 908		N = 2374	N = 2324	N = 1276	
Age (y)	Median (IQR)	46 (36-53)	53 (44-63)	66 (56-78)	77 (67-84)	79 (67-86)	80 (69-87)	< 0.001	58 (47-70)	76 (65-84)	78 (66-86)	< 0.001
Sex, female	N (%)	210 (52.6%)	434 (47.0%)	802 (57.1%)	792 (53.3%)	373 (43.7%)	414 (45.6%)	< 0.001	1324 (55.8%)	1154 (49.7%)	547 (42.9%)	< 0.001
Risk factors												
Arrhythmia	N (%)	12 (3.0%)	49 (5.3%)	164 (11.7%)	295 (19.9%)	236 (27.7%)	231 (25.4%)	< 0.001	157 (6.6%)	509 (21.9%)	321 (25.2%)	< 0.001
Congestive heart failure	N (%)	11 (2.8%)	49 (5.3%)	177 (12.6%)	381 (25.6%)	329 (38.6%)	343 (37.8%)	< 0.001	172 (7.2%)	634 (27.3%)	484 (37.9%)	< 0.001
Diabetes	N (%)	26 (6.5%)	158 (17.1%)	390 (27.8%)	612 (41.2%)	342 (40.1%)	433 (47.7%)	< 0.001	526 (22.2%)	906 (39.0%)	529 (41.5%)	< 0.001
Hypertension	N (%)	110 (27.6%)	380 (41.2%)	931 (66.3%)	1181 (79.5%)	691 (81.0%)	752 (82.8%)	< 0.001	1181 (49.7%)	1840 (79.2%)	1024 (80.3%)	< 0.001
MI	N (%)	15 (3.8%)	44 (4.8%)	105 (7.5%)	169 (11.4%)	157 (18.4%)	184 (20.3%)	< 0.001	121 (5.1%)	318 (13.7%)	235 (18.4%)	< 0.001
Renal disease	N (%)	0 (0.0%)	6 (0.7%)	26 (1.9%)	61 (4.1%)	88 (10.3%)	104 (11.5%)	< 0.001	15 (0.6%)	124 (5.3%)	146 (11.4%)	< 0.001
Stroke	N (%)	≤ 5*	15 (1.6%)	23 (1.6%)	51 (3.4%)	36 (4.2%)	56 (6.2%)	< 0.001	35 (1.5%)	87 (3.7%)	64 (5.0%)	< 0.001
Angina	N (%)	12 (3.0%)	27 (2.9%)	79 (5.6%)	118 (7.9%)	59 (6.9%)	79 (8.7%)	< 0.001	97 (4.1%)	172 (7.4%)	105 (8.2%)	< 0.001
Percutaneous coronary intervention	N (%)	7 (1.8%)	31 (3.4%)	68 (4.8%)	78 (5.2%)	56 (6.6%)	66 (7.3%)	< 0.001	72 (3.0%)	148 (6.4%)	86 (6.7%)	< 0.001
Coronary artery bypass grafting	N (%)	≤ 5*	7 (0.8%)	29 (2.1%)	41 (2.8%)	40 (4.7%)	29 (3.2%)	< 0.001	25 (1.1%)	78 (3.4%)	44 (3.4%)	< 0.001
Serum creatinine, μmol/L	Median (IQR)	67 (61-73)	72 (65-79)	78 (69-94)	89 (74-116)	101 (77-144)	114 (89-169)	< 0.001	74 (67-85)	88 (73-119)	107 (80-163)	< 0.001
Glucose, mmol/L	Mean ± SD	4.98 ± 0.42	6.19 ± 2.17	7.03 ± 3.35	8.42 ± 5.60	7.92 ± 4.48	9.45 ± 4.82	< 0.001	6.84 ± 3.34	7.95 ± 5.03	8.41 ± 4.55	< 0.001
hs-cTnI, ng/L	Median (IQR)	2 (1-2)	2 (1-3)	4 (2-6)	8 (6-12)	24 (18-42)	67 (39-179)	< 0.001	2 (1-4)	10 (7-15)	62 (38-170)	< 0.001
eGFR, mL/min/1.73 m²	Median (IQR)	102 (96-110)	94 (85-103)	77 (63-87)	63 (46-78)	54 (34-76)	46 (27-66)	< 0.001	86 (73-98)	63 (44-81)	49 (29-74)	< 0.001
Outpatient cardiologist follow-up: 30 d after ED visit	N (%)	25 (6.3%)	71 (7.7%)	133 (9.5%)	192 (12.9%)	132 (15.5%)	155 (17.1%)	< 0.001	188 (7.9%)	287 (12.3%)	233 (18.3%)	< 0.001
Outpatient GP/FP follow-up: 30 d after ED visit	N (%)	190 (47.6%)	405 (43.9%)	673 (47.9%)	699 (47.0%)	380 (44.5%)	403 (44.4%)		1120 (47.2%)	1094 (47.1%)	536 (42.0%)	
No outpatient follow-up: 30 d after ED visit	N (%)	184 (46.1%)	447 (48.4%)	599 (42.6%)	595 (40.0%)	341 (40.0%)	350 (38.5%)		1066 (44.9%)	943 (40.6%)	507 (39.7%)	
All-cause mortality, 90 d	N (%)	≤ 5*	31 (3.4%)	68 (4.8%)	137 (9.2%)	145 (17.0%)	217 (23.9%)	< 0.001	65 (2.7%)	254 (10.9%)	282 (22.1%)	< 0.001
All-cause mortality, 1 y	N (%)	8 (2.0%)	47 (5.1%)	134 (9.5%)	253 (17.0%)	248 (29.1%)	340 (37.4%)	< 0.001	119 (5.0%)	448 (19.3%)	463 (36.3%)	< 0.001
All-cause mortality, 5 y	N (%)	32 (8.0%)	137 (14.8%)	335 (23.8%)	657 (44.2%)	526 (61.7%)	612 (67.4%)	< 0.001	350 (14.7%)	1113 (47.9%)	836 (65.5%)	< 0.001

Medical history calculated using 5-y look-back.

CCS, Clinical Chemistry Score; ED, emergency department; eGFR, estimated glomerular filtration rate; FP, family practice; GP, general practice; hs-cTnI, high-sensitivity cardiac troponin I; IQR, interquartile range; MI, myocardial infarction.

* Suppressed for privacy reasons.

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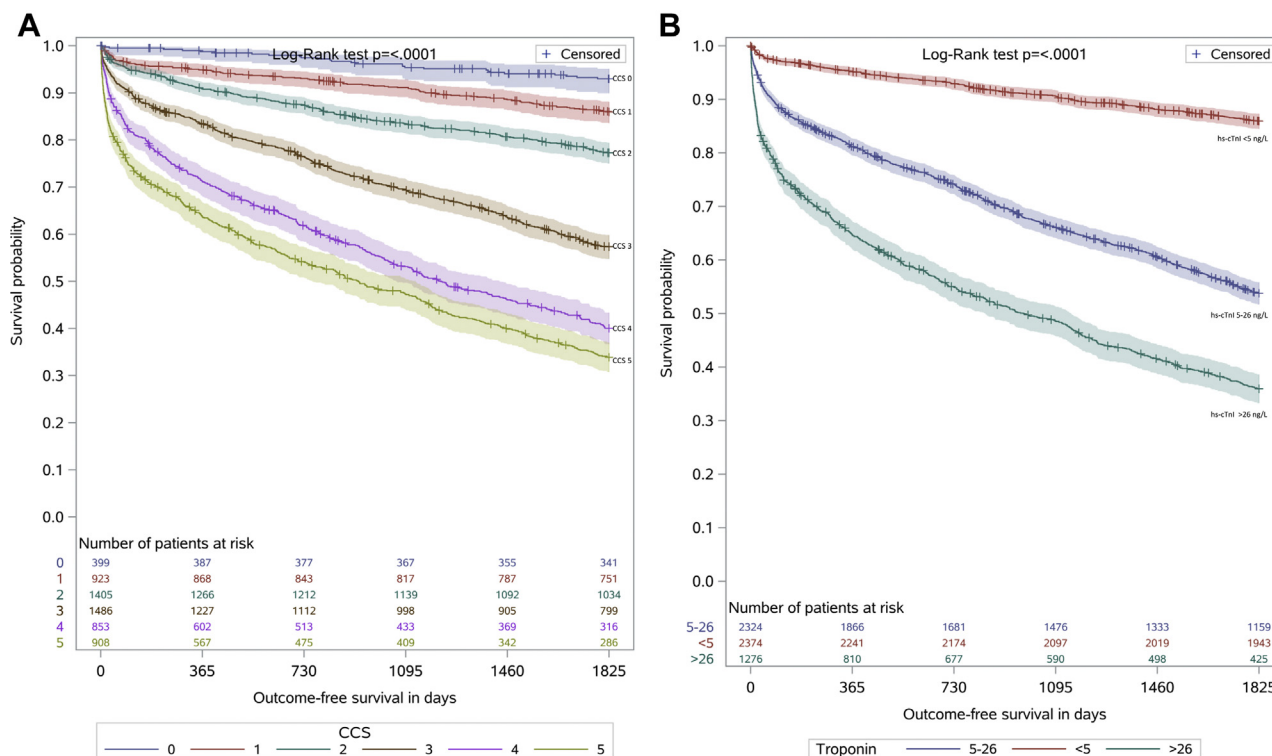
Figure 1. Kaplan—Meier survival curves for all-cause death based on **(A)** Clinical Chemistry Score (CCS) (0 to 5) and **(B)** high-sensitivity cardiac troponin I (hs-cTnI) categories (<5 ng/L, 5-26 ng/L, >26 ng/L).

eGFR, and hs-cTnI. For example, a CCS < 1 (or CCS = 0) would be obtained if glucose < 5.6 mmol/L, eGFR ≥ 90 mL/min/1.73 m^2, and hs-cTnI < 4 ng/L, whereas a CCS = 5 would be obtained if glucose ≥ 5.6 mmol/L, eGFR < 90 mL/min/1.73 m^2, and hs-cTnI > 30 ng/L from the ED presentation blood work.

Outcomes and statistical analysis

We evaluated all-cause death at 3 months, 1 year, and 5 years after the presentation blood work.[19-21] Administrative and clinical databases linked at ICES (Toronto, Ontario, Canada) via unique encrypted patient identifier were used to obtain medical history and outcomes. The Ontario registered persons database contained all information on patient demographics and death date. All inpatient hospital discharges were captured in the Canadian Institute for Health Information (CIHI) Discharge Abstract Database. We used the Ontario Health Insurance Plan database to capture all physician billings and outpatient visits.

We compared baseline characteristics (demographic and clinical) with a 5-year look-back across the following categorizations: (1) hs-cTnI (<5 ng/L vs 5-26 ng/L vs >26 ng/L) and (2) CCS (all individual values on 0-5 ordinal scale). To assess test performance, we calculated sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) (with 95% confidence intervals [CIs]). These metrics were calculated to assess the prognostic performance of hs-cTnI alone vs in combination with other laboratory tests to rule out those at low risk of experiencing the outcome and rule in those at high risk. We compared hs-cTnI ≥ 5 ng/L vs CCS ≥ 1 and hs-cTnI ≥ 5 ng/L vs CCS ≥ 2 to assess if

sensitivity ≥ 99% or NPV ≥ 99.5%, 2 metrics that have been selected as necessities for a test to rule out.[22] In contrast, for high risk, we compared hs-cTnI > 26 ng/L (the overall 99th percentile) vs CCS = 5 and hs-cTnI > 26 ng/L vs CCS ≥ 4 to assess if specificity ≥ 90% or PPV ≥ 75% could be attained.[22] We also compared the rate of death between the CCS of < 1 or < 2 with that of hs-cTnI < 5 ng/L as surveys of Canadian, American, and Australasian ED physicians suggest a miss rate not exceed 2%.[23] Kaplan—Meier survival curves for all-cause mortality over 5 years were also constructed for both the CCS categories and hs-cTnI ranges with censoring at the end of the 5-year observation window (*P* value by log-rank). Analyses were performed using SAS 9.1.3 software (SAS Institute Inc., Cary, NC) and MedCalc Statistical Software version 19.1.6 (MedCalc Software Ltd., Ostend, Belgium).

Results

The median age (interquartile range) for the study population was 70 years (56-82), 50.6% were female, and approximately one-third of the population (32.8%) had diabetes. The prevalence of cardiovascular disease and risk factors increased with the higher hs-cTnI concentrations and the higher CCS (Table 1), with survival curves also displaying separation of low- and high-risk groups per hs-cTnI ranges or the CCS over 5 years (Fig. 1).

Overall, 40.8% of the population was discharged home from the ED with 79.4% (95% CI, 70.9-88.7) of patients with a CCS < 1 discharged compared with 64.4% (95% CI, 61.2-67.7) of patients with an hs-cTnI < 5 ng/L (*P* < 0.01). The mortality rate was ≤ 2.0% in patients (n = 399 or 6.7%

of the total population) with a CCS < 1 (ie, CCS = 0) at both 3 months and 1 year compared with 2.7% and 5.0% in patients (n = 2374 or 39.7% of the total population) with hs-cTnI < 5 ng/L at these timeframes, respectively (Table 1). At 1 year, the difference in mortality rates was significant (CCS < 1 mortality was 2.0%; 95% CI, 0.9-4.0 vs mortality in patients with hs-cTnI < 5 ng/L of 5.0%; 95% CI, 4.2-6.0; P = 0.01). By applying the CCS cutoff of ≥ 1, the sensitivity for mortality was 99.5% (95% CI, 98.5-99.9) at 3 months, 99.2% (95% CI, 98.4-99.6) at 1 year, and 98.6% (95% CI, 98.0-99.0) at 5 years (Table 2). Patients with a CCS < 2 had outcome rates similar to those patients with only hs-cTnI < 5 ng/L; however, the sensitivities for a CCS ≥ 2 were higher (3-month sensitivity = 94.3% to 5-year sensitivity = 92.6%) than hs-cTnI ≥ 5 ng/L (3-month sensitivity = 89.1% to 5-year sensitivity = 84.7%) (Table 2). The highest observed NPV (99.2%; 95% CI, 97.8-99.8) was at 3 months when using the CCS cutoff ≥ 1. At 5 years, only the CCS of 5 yielded a specificity ≥ 90% (91.9%; 95% CI, 91.0-92.8) for all-cause mortality, with no PPVs > 75% for the CCS or hs-cTnI above the 99th percentile cutoffs.

Discussion

In an ED population who were investigated for ACS, were clinically managed with a contemporary cTnI assay, and had hs-cTnI results that were blinded to the treating physicians, our findings indicate that an hs-cTnI cutoff of ≥ 5 ng/L at ED presentation would not be sufficiently safe to rule out mortality because the sensitivity estimates were below 90%. Only a CCS ≥ 1 would yield diagnostic test estimates that could be considered safe for identifying low-risk patients because the sensitivity was > 99% and the event rate was ≤ 2% (for those with a CCS < 1) for up to 1 year after patient presentation. For identifying high-risk patients, only the CCS of 5 yielded a specificity > 90% for mortality, but this was evident at only 5 years. These data extend previous findings on the CCS[14] to a general ED population, who are more representative of patients who have cardiac troponin ordered in the emergency setting. Moreover, these findings further support data that patients with hs-cTnI concentrations below the 99th percentile (or "normal levels") early after ED presentation may still be at a higher risk for an adverse outcome over both the short and long term.[19,20,24] Additional laboratory and clinical variables may further aid in risk stratification as demonstrated by the simple CCS in this setting.

Limitations

Our study includes some important limitations. First, we were unable to capture the time of pain onset and thus cannot differentiate early pain onset from late pain onset because hs-cTn testing for rule-out is not recommended in the population with early chest pain onset.[3,6] Second, we only included all-cause death, a hard end point, whereas others have also included MI and other cardiovascular outcomes. Third, the study population was derived from 3 hospitals from the same city. Further research could validate the results from other geographic locations. Fourth, the laboratory ordering practice of including glucose and creatinine with cardiac troponin (ie, the "ED cardiac presentation panel") may differ at different hospitals. To our understanding, there are no

Table 2. Prognostic performance of CCS ≥ 1 or ≥ 2 for rule-out and CCS = 5 or ≥ 4 for rule-in vs hs-cTnI alone (≥ 5 ng/L for rule-out or > 26 ng/L for rule-in) for all-cause mortality

Parameter	Time	Rule-out			Rule-in		
		CCS ≥ 1	CCS ≥ 2	hs-cTnI ≥ 5 ng/L	CCS = 5	CCS ≥ 4	hs-cTnI > 26 ng/L
Sensitivity	3 mo	**0.995 (0.985-0.999)**	0.943 (0.921-0.960)	0.891 (0.864-0.915)	0.361 (0.322-0.400)	0.602 (0.561-0.641)	0.462 (0.422-0.503)
	1 y	**0.992 (0.984-0.996)**	0.946 (0.931-0.959)	0.884 (0.863-0.903)	0.330 (0.301-0.359)	0.570 (0.540-0.601)	0.442 (0.412-0.473)
	5 y	0.986 (0.980-0.990)	0.926 (0.915-0.936)	0.847 (0.832-0.862)	0.266 (0.248-0.284)	0.495 (0.474-0.515)	0.358 (0.339-0.378)
NPV	3 mo	0.992 (0.978-0.998)	0.974 (0.964-0.982)	0.972 (0.965-0.978)	0.924 (0.916-0.931)	0.943 (0.935-0.950)	0.931 (0.924-0.938)
	1 y	0.979 (0.960-0.991)	0.958 (0.946-0.968)	0.949 (0.940-0.958)	0.863 (0.854-0.873)	0.895 (0.885-0.904)	0.878 (0.868-0.887)
	5 y	0.919 (0.888-0.944)	0.872 (0.853-0.889)	0.852 (0.837-0.866)	0.667 (0.653-0.680)	0.724 (0.710-0.737)	0.688 (0.674-0.701)
Specificity	3 mo	0.073 (0.066-0.081)	0.239 (0.228-0.251)	0.429 (0.416-0.443)	0.871 (0.862-0.880)	0.739 (0.727-0.751)	0.819 (0.808-0.829)
	1 y	0.079 (0.071-0.087)	0.256 (0.244-0.268)	0.456 (0.442-0.470)	0.885 (0.875-0.893)	0.762 (0.750-0.774)	0.839 (0.829-0.849)
	5 y	0.099 (0.090-0.110)	0.313 (0.298-0.329)	0.550 (0.534-0.566)	**0.919 (0.910-0.928)**	0.830 (0.817-0.842)	0.884 (0.873-0.894)
PPV	3 mo	0.107 (0.099-0.115)	0.121 (0.112-0.131)	0.148 (0.137-0.160)	0.239 (0.211-0.268)	0.205 (0.186-0.225)	0.222 (0.199-0.246)
	1 y	0.183 (0.173-0.193)	0.209 (0.198-0.221)	0.253 (0.238-0.267)	0.374 (0.342-0.406)	0.333 (0.311-0.356)	0.365 (0.338-0.392)
	5 y	0.406 (0.393-0.419)	0.457 (0.443-0.472)	0.541 (0.524-0.557)	0.674 (0.642-0.704)	0.646 (0.623-0.668)	0.660 (0.633-0.686)

CCS, Clinical Chemistry Score; hs-cTnI, high-sensitivity cardiac troponin I; NPV, negative predictive value; PPV, positive predictive value.
Bold text represent estimates that are ≥ 99% sensitivity or ≥ 90% specificity.

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recommendations on what laboratory tests besides cardiac troponin should be ordered when considering myocardial injury.[7,13] Fifth, no adjustments were made for subsequent MI post-ED assessment, other cardiovascular disease risk factors, or medication use during the 5-year period for long-term mortality because there may be other important variables that have contributed to long-term mortality. Sixth, the prevalence of patients with a CCS < 1 (~7%) was approximately 6-fold lower compared with patients with an hs-cTnI < 5 ng/L (~40%). Clinicians may want to use additional screening tools, besides laboratory tests, to identify patients at low risk for an adverse outcome.

Conclusions

In patients in the ED being investigated for suspected ACS, using a simple CCS at presentation improves the ability to identify both low-risk and high-risk patients for short- and long-term mortality.

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Disclosures

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Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at https://www.cjcopen.ca/ and at https://doi.org/10.1016/j.cjco.2020.03.004.