Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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13 November 2020
1 December 2020

1. Green P, Woglom AE, Genereux P, Daneault B, Paradis J-M, Schnell S, Hawkey M, Maurer MS, Kirtane AJ, Kodali S, Moses JW, Leon MB, Smith CR, Williams M. The impact of frailty status on survival after transcatheter aortic valve replacement in older adults with severe aortic stenosis: a single-center experience. *JACC Cardiovasc Interv* 2012;5:974–981.
2. Goldfarb M, Lauck S, Webb JG, Asgar AW, Perrault LP, Piazza N, Martucci G, Lachapelle K, Nicolas N, Kim DH, Popma JJ, Lefèvre T, Labinaz M, Lamy A, Peterson MD, Arora RC, Morais JA, Morin JF, Rudski LG, Afilalo J. Malnutrition and mortality in frail and non-frail older adults undergoing aortic valve replacement. *Circulation* 2018;138:2202–2211.
3. González Ferreiro R, Muñoz-García AJ, López Otero D, Avanzas P, Pascual I, Alonso-Briales JH, González-Juanatey JR, Pun F, Jiménez-Navarro MF, Hernández-García JM, Morís C, Nouche RT. Nutritional risk index predicts survival in patients undergoing transcatheter aortic valve replacement. *Int J Cardiol* 2019;276:66–71.
4. Shimura T, Yamamoto M, Kano S, Sago M, Tsunaki T, Kagase A, Koyama Y, Tsujimoto S, Otsuka T, Yashima F, Tada N, Naganuma T, Araki M, Yamanaka F, Shiirai S, Mizutani K, Tabata M, Ueno H, Takagi K, Higashimori A, Watanabe Y, Hayashida K, OCEAN-TAVI investigators. Predictors and prognostic impact of nutritional changes after transcatheter aortic valve replacement. *Cardiovasc Revasc Med* 2020. https://doi.org/10.1016/j.carrev.2020.08.031.
5. Habaybeh D, de Moraes MB, Slee A, Avgerinou C. Nutritional interventions for heart failure patients who are malnourished or at risk of malnutrition or cachexia: a systematic review and meta-analysis. *Heart Fail Rev* 2020. https://doi.org/10.1007/s10741-020-09937-9.
6. von Haehling S, Doehner W, Anker SD. Nutrition, metabolism, and the complex pathophysiology of cachexia in chronic heart failure. *Cardiovasc Res* 2007;73:298–309.

https://doi.org/10.1016/j.amjcard.2020.12.009

The Utility of CHA(2)DS(2)-VASc Scores as a Risk Assessment Tool in Low-Risk In-Hospital Patients With Coronavirus Disease 2019 Infection

Coronavirus disease 2019 (COVID-19) infections can have serious consequences such as cardiac manifestations, severe coagulopathy, and thromboembolism.[1–4] We read with interest the study by Ruocco et al published in *the American Journal of Cardiology*.[5] This study, performed on an Italian cohort, showed that the CHA(2)DS(2)-VASc score could aid prognostication of mortality and a composite end-point of inpatient death or invasive ventilation across CHA(2)DS(2)-VASc tertiles in COVID-19 patients.

In Singapore, there has been a demographic shift in COVID-19 cases initially involving at-risk elderly population in the community, and transitioning to cluster outbreaks in densely-populated foreign worker dormitories.[6] Active case finding of dormitory residents resulted in a surge of swab-positive COVID-19 patients who were either asymptomatic or mildly symptomatic. They were admitted to hospital for risk assessment before transfer to a nonhospital isolation facility. This policy provided an opportunistic insight into the low-risk COVID-19 cohort.

Our hospital registry of 554 in-hospital COVID-19 patients, recruited between 23rd January to 30th April 2020, were admitted to a tertiary

Table 1
Clinical characteristics and outcomes of patients hospitalised for COVID-19 infection categorised according to CHA(2)DS(2)-VASc score

Variable	All patients (N = 554)	CHA(2)DS(2)-VASc scores			p Value
		≤1 (N = 532)	2-3 (N = 18)	≥4 (N = 4)	
Sex (male)	482 (87.0)	473 (88.9)	9 (50.0)	0	<0.001
Age (years)	36 (11)	36 (10)	54 (13)	67 (19)	<0.001
Smoking status					0.330
Current smoker	30 (5.6)	27 (5.3)	3 (16.7)	0	
Ex-smoker	3 (0.6)	3 (0.6)	0	0	
Hypertension	53 (12.3)	34 (8.3)	16 (88.9)	3 (75.0)	<0.001
Diabetes mellitus	21 (5.1)	8 (2.0)	10 (62.5)	3 (75.0)	<0.001
Hyperlipidemia	34 (8.1)	17 (4.3)	13 (72.2)	4 (100.0)	<0.001
Atrial fibrillation	0	0	0	0	
Ischemic heart disease	5 (1.2)	1 (0.3)	3 (16.7)	1 (25.0)	<0.001
Congestive heart disease	3 (0.7)	0	1 (6.3)	2 (50.0)	<0.001
Stroke	2 (0.5)	0	0	2 (50.0)	<0.001
Chronic kidney disease	3 (0.7)	1 (0.3)	0	2 (50.0)	<0.001
Medications					
Statin	26 (6.2)	11 (2.8)	11 (61.1)	4 (100)	<0.001
Beta-blocker	8 (1.9)	4 (1.0)	3 (16.7)	1 (25.0)	<0.001
Calcium channel blocker	29 (6.9)	17 (4.3)	10 (55.6)	2 (50.0)	<0.001
ACE-I	6 (1.5)	2 (0.5)	3 (16.7)	1 (25.0)	<0.001
ARB	12 (2.9)	8 (2.0)	3 (16.7)	1 (25.0)	<0.001
Diuretics	5 (1.2)	3 (0.8)	0	2 (50.0)	<0.001
Metformin	18 (4.3)	8 (2.0)	10 (62.5)	0	<0.001
Insulin	4 (1.0)	2 (0.5)	1 (6.3)	1 (25.0)	<0.001
Study outcomes					
Mortality	2 (0.5)	2 (0.5)	0	0	0.952
Intensive care unit admission	19 (3.4)	15 (2.8)	2 (11.1)	2 (50.0)	<0.001
Mechanical ventilation	16 (2.9)	12 (2.3)	2 (11.1)	2 (50.0)	<0.001
Composite end-point	59 (10.6)	52 (9.8)	3 (16.7)	4 (100.0)	<0.001

ACE-I = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; COVID-19 = coronavirus disease 2019.

healthcare institution for risk assessment before down-triaging to a nonhospital isolation facility. They were allocated into 3 groups based on the CHA(2)DS(2)-VASc scores (CV ≤1; CV2-3; CV ≥4). Primary outcomes were intensive care unit (ICU) admissions, mechanical ventilation, all-cause mortality, and a composite end-point. The composite end-point was defined as the presence of one of the following: ICU admissions, mortality, mechanical ventilation, or any COVID-related organ involvement (pneumonia, acute respiratory distress syndrome, acute kidney injury, pulmonary embolism, coagulopathy, acute myocardial infarction, heart failure, and stroke).

The baseline characteristics of our study population differed from the Ruocco et al study.[5] Our cohort had lower median age of 34 (28 to 43) years compared with the previous study (65 [53 to 76] years). Our median CHA(2)

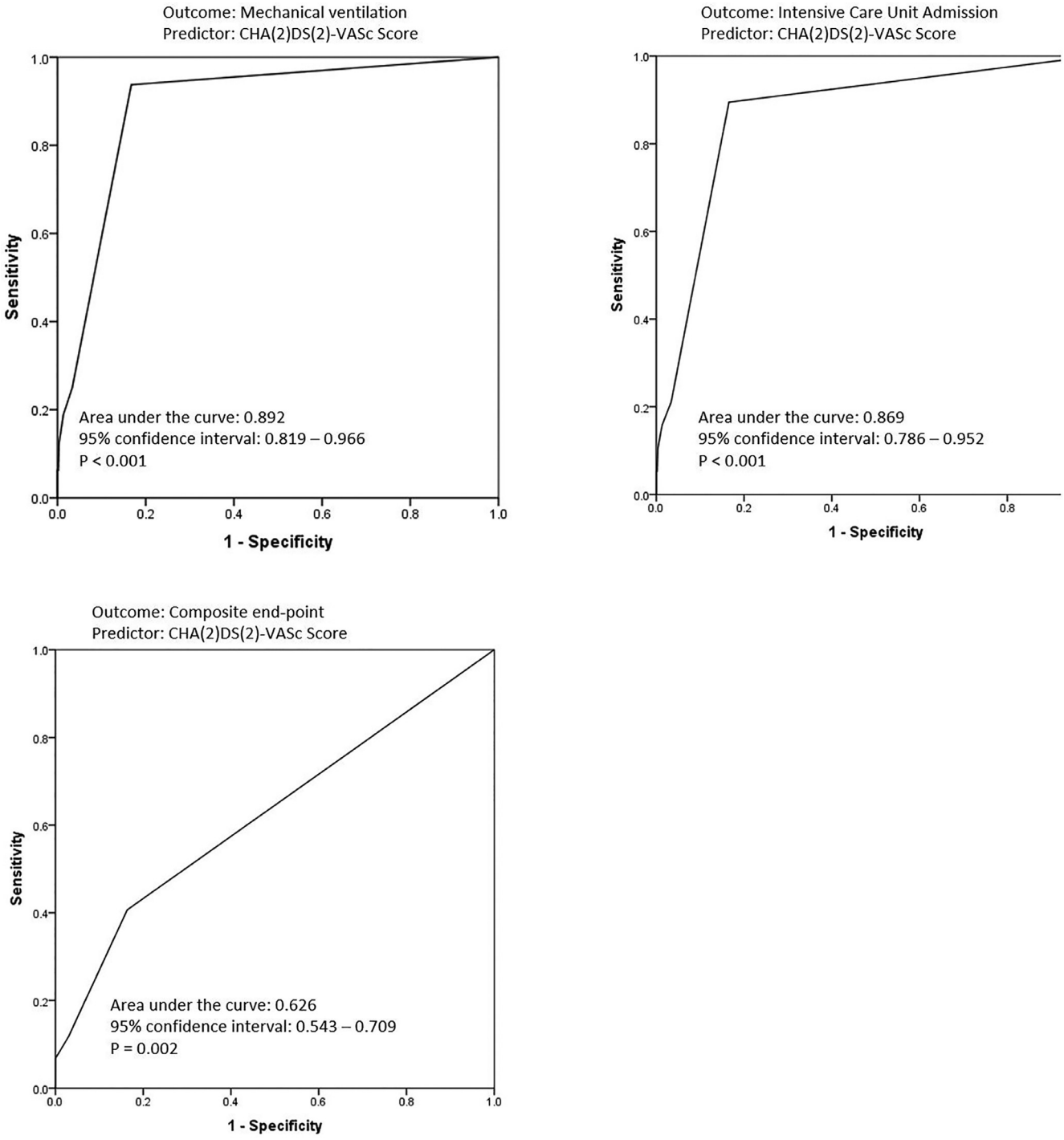

Figure 1. Receiver operating characteristic curves for mechanical ventilation, intensive care unit admission, and study composite end-point for the predictor of CHA(2)DS(2)-VASc score. Composite end-point was defined as the presence of one of the following: ICU admissions, mortality, requiring mechanical ventilation, or one of the COVID-related organ involvements (pneumonia, acute respiratory distress syndrome, acute kidney injury, pulmonary embolism, coagulopathy, acute myocardial infarction, ventricular tachycardia, myocardial injury or myocarditis, heart failure, and stroke).

DS(2)-VASc score of 0 was also lower (2 [1 to 3] in the cited study).[5] Seventy-six study participants (13.7%) remained asymptomatic. There was lower prevalence of co-morbidities in our cohort compared with the cited study (hypertension 12.3% vs 48.6%, diabetes 5.1% vs 15.7%, ischemic heart disease 1.2% vs 11.2%, stroke 0.5% vs 7.6%, and heart failure 0.7% vs 6.1%, respectively).[5] Chronic medication use was less in our study cohort. In terms of study outcomes, there were 2 deaths in our low-risk group, both of whom were in the CV ≤1 group. There were 19 (3.4%) patients requiring ICU admission, 16 (2.9%) requiring mechanical ventilation, and 59 (10.6%) with the composite end-point. We observed significant increases in ICU admissions (CV ≤1: 2.8%, CV2-3: 11.1%, CV ≥4: 50.0%, p <0.001), mechanical ventilation (CV ≤1: 2.3%, CV2-3: 11.1%, CV ≥4: 50.0%, p <0.001), and composite end-point (CV ≤1: 9.8%, CV2-3: 16.7%, CV ≥4: 100.0%, p <0.001) across the groups (Table 1). Univariable logistic regression analysis demonstrated significantly increased risk of mechanical ventilation in the CV2-3 (odds ratio [OR] 5.778, 95% confidence interval [CI] 1.187 to 28.121, p = 0.030) and CV ≥4 (OR 28.889, 95% CI 4.415 to 189.038, p <0.001) groups compared with CV ≤1 group (reference group). There was significantly increased risk of ICU admission in the CV ≥4 (OR 22.844, 95% CI 3.551 to 146.951, p = 0.001) group compared with CV ≤1 group (reference), with a trend toward increased risk of ICU admission in the CV2-3 group (OR 4.569, 95% CI 0.958 to 21.790, p = 0.057). Significant increased risk of the adverse composite end-point was observed in the CV ≥4 group (OR 36.923, 95% CI 4.051 to 338.550, p = 0.001) compared with the CV ≤1 group (reference). There was no statistical difference in composite end-point between CV2-3 (OR 1.978, 95% CI 0.550 to 7.110, p = 0.296) and CV ≤1 groups.

Similar to the previous study,[5] our receiver operating characteristics (ROC) analysis (Figure 1) confirmed the prognostic ability of CHA(2)DS(2)-VASc score in the low-risk COVID-19 cohort for ICU admissions, mechanical ventilation requirement, and study composite end-point.

To date, a clinically simple risk stratification score for COVID-19 patients is lacking. Ruocco et al have called for an urgent need to characterize these patients to identify the at-risk patients of acute respiratory distress syndrome.[5] Our findings reinforce CHA(2)DS(2)-VASc score as a potential tool to identify at-risk COVID-19 individuals in a generally young, low-risk, asymptomatic, or mildly symptomatic cohort. The study demonstrated that CV2-3 and CV ≥4 groups displayed higher rates of mechanical ventilation, ICU admissions, and composite end-point, compared with the CV ≤1 group. However, we did not see a trend for all-cause mortality due to the low-risk nature of the cohort with its overall mortality rate of 0.5%.

The CHA(2)DS(2)-VASc score is indeed a simple and widely-available stratification tool that can be used in the outpatient setting or upon admission, as it is not restricted by laboratory measurements that is required in other proposed risk scores.[7] This is important as it allows clinicians to identify those who are at higher risk in the community, and may benefit from closer in-hospital monitoring.

Disclosure

The authors have no conflicts of interest to disclose.

Financial disclosure: None.

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28 November 2020

1. Pan A, Liu L, Wang C, Guo H, Hao X, Wang Q, Huang J, He N, Yu H, Lin X, Wei S, Wu T. Association of public health interventions with the epidemiology of the COVID-19 outbreak in Wuhan, China. *JAMA* 2020;323:1–9.
2. Fox SE, Akmatbekov A, Harbert JL, Li G, Quincy Brown J, Vander Heide RS. Pulmonary and cardiac pathology in African American patients with COVID-19: an autopsy series from New Orleans. *Lancet Respir Med* 2020;2600:30243–30245.
3. Ho JS, Sia CH, Chan MY, Lin W, Wong RC. Coronavirus-induced myocarditis: a meta-summary of cases. *Heart Lung* 2020;49:681–685.
4. Ho JS, Tambyah PA, Ho AF, Chan MY, Sia CH. Effect of coronavirus infection on the human heart: a scoping review. *Eur J Prev Cardiol* 2020;27:1136–1148.
5. Ruocco G, McCullough PA, Tecson KM, Mancone M, De Ferrari GM, D'Ascenzo F, De Rosa FG, Paggi A, Forleo G, Secco GG, Pistis G, Monticone S, Vicenzi M, Rota I, Blasi F, Pugliese F, Fedele F, Palazzuoli A. Mortality risk assessment using CHA(2)DS(2)-VASc scores in patients hospitalized with coronavirus disease 2019 infection. *Am J Cardiol* 2020;137:111–117.
6. Ngiam JN, Chew N, Tham SM, Beh DL, Lim ZY, Li TYW, Cen S, Tambyah PA, Santosa A, Sia CH, Cross GB. Demographic shift in COVID-19 patients in Singapore from an aged, at-risk population to young, migrant workers with reduced risk of severe disease. *Int J Infect Dis* 2020. https://doi.org/10.1016/j.ijid.2020.11.157. S1201-9712(20)32467-X.
7. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *J Thromb Haemost* 2020;18:844–847.

https://doi.org/10.1016/j.amjcard.2020.12.005

Comment on "A Practical Approach for the Use of High-Sensitivity Cardiac Troponin Assays in the Evaluation of Patients With Chest Pain"

In a recent article in the *American Journal of Cardiology*, Azar et al[1] refer to "creatinine kinase" (sic) in the article abstract. Rather than "creatinine kinase," presumably the authors were instead referring to the enzyme creatine kinase. Creatine kinase (also known as creatine phosphokinase) catalyzes the reversible phosphorylation of creatine to phosphocreatine, is frequently measured as a marker of muscle damage, and is commonly abbreviated as "CK."[2] Creatinine is neither a product nor substrate for creatine kinase and is instead formed from creatine and phosphocreatine via nonenzymatic reactions. The mistake of referring to creatine kinase as creatinine kinase is common. It is likely that most readers understood the authors as they intended. However, this misspelling has the potential to cause confusion, can complicate literature searches, and may result in a loss of reader confidence in what might otherwise be a high-quality publication.

Disclosures

The author declares that he has no known competing financial interests or