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.

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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

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personal relationships that could have appeared to influence the work reported in this paper.

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Benefit of Single Antiplatelet Therapy Over Dual Antiplatelet Therapy After Transcatheter Aortic Valve Implantation

Current practice guidelines recommend dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for 3 to 6 months followed by lifelong aspirin after transcatheter aortic valve implantation (TAVI). However, recently published POPular TAVI trial[1] and other trials[2–4] that have compared DAPT with single antiplatelet therapy (SAPT) have shown decreased bleeding events and noninferiority with respect to ischemic stroke, all-cause mortality, and myocardial infarction (MI) among patients receiving SAPT. Using the existing randomized control trials (RCTs) evaluating the efficacy of DAPT versus SAPT in patients undergoing TAVI who did not have an indication for oral anticoagulation, we computed the net clinical benefit (NCB) of SAPT.

The 2 groups (DAPT and SAPT) were compared for the following outcomes: Major or life-threatening bleeding, stroke, all-cause mortality, and MI at the longest available follow-up. We used Mantel-Haenszel method with Paule-Mandel estimator for tau2, Hartung-Knapp adjustment for random effects model and Q-profile method for confidence interval of tau2 and tau to calculate risk ratio (RR) with 95% confidence interval (CI). Heterogeneity was assessed using Higgins I2 statistics. All statistical analysis was carried out using R version 3.6.4. NCB for SAPT was defined as stroke event prevented by SAPT minus major or life-threatening bleeding caused by SAPT multiplied by a weighting factor.[5] The weighting factor reflects the relative impact, with regard to death and disability, of major or life-threatening bleeding compared with stroke. In addition to a weighting factor of 1, sensitivity analyses by using weighting factors 0.5, 1.5, and 2 were also performed.

Net Clinical Benefit $_{SAPT}$ = *(Stroke rate* $_{DAPT}$ *- Stroke rate* $_{SAPT}$*) − Weight factor (Major or life-threatening bleeding* $_{SAPT}$ *- Major or life-threatening bleeding* $_{DAPT}$*)*

Four RCTs[1–4] were included in the final analysis. Together, these studies included 1086 patients: 541 with SAPT (aspirin only) and 545 patients with DAPT (aspirin and clopidogrel). The average duration of follow up was 5.5 months (range: 1 to 12 months). DAPT was associated with a significantly higher risk of major bleeding or life-threatening bleeding in comparison

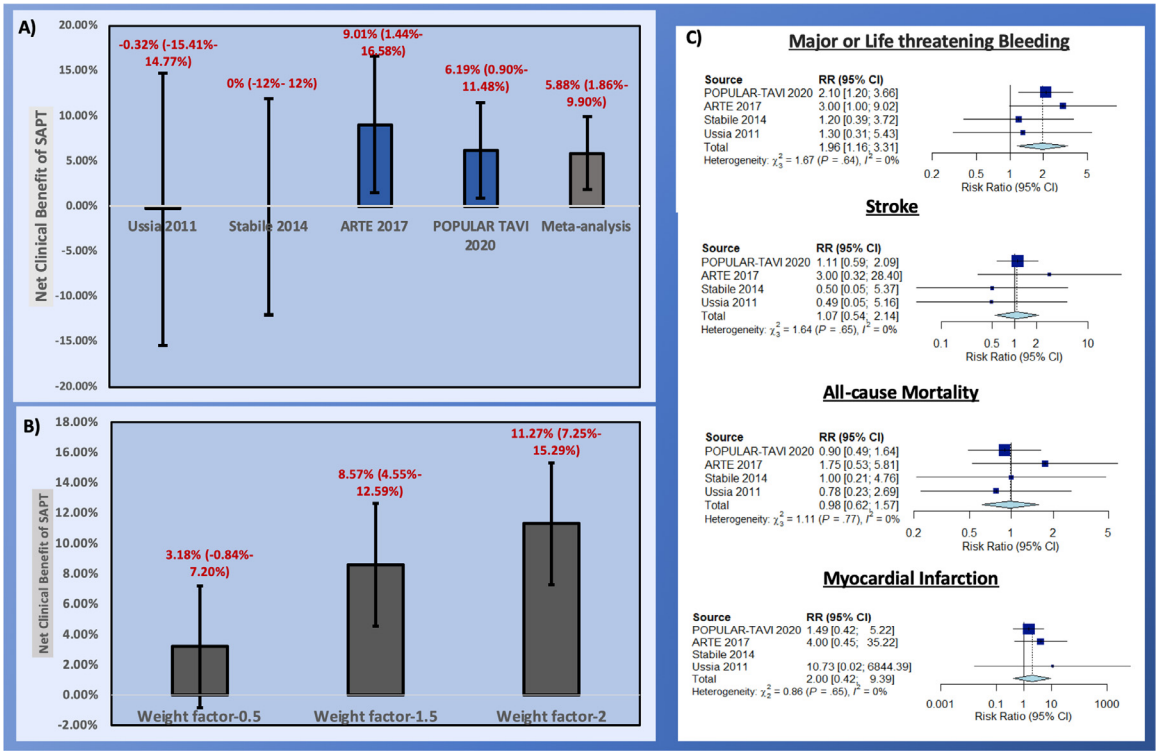

Figure 1. *(A)* Net clinical benefit of single antiplatelet therapy (SAPT) over dual antiplatelet therapy (DAPT) for the four RCTs and the pooled meta-analysis. With a weight factor of 1, pooled effect of RCTs showed a NCB of SAPT over DAPT (5.88% [95% CI: 1.86% to 9.90%])
(B) Sensitivity analysis of net clinical benefit of SAPT compared with DAPT (pooled meta-analysis) with weighting factors 0.5, 1.5, and 2.
(C) Pooled risk ratios and 95% confidence intervals of major or life-threatening bleeding, stroke, all-cause mortality and myocardial infarction; RR = risk ratio; CI = confidence interval. Single antiplatelet therapy was associated with significantly reduced risk of major bleeding or life-threatening bleeding compared with dual antiplatelet therapy.