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.

pitalization cost and LoS was noted. The limitations of this study are primarily due to the inherent characteristics of the NIS database.[7] The NIS is an administrative database and thus the accuracy of the analysis depends on the precision of the records collected; moreover, the use of ICD codes in identifying records may have led to inherent inaccuracies in estimating the studied diagnoses. Due to the nature of the database, the cohort is also limited by the lack of objective echocardiographic assessment of heart failure and long-term follow-up.

Disclosures

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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7 October 2020

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More Good News on Statins and COVID-19

We read with interest the meta-analysis (4 studies, 8,990 coronavirus disease 2019 (COVID-19) patients) by Kow et al[1] reporting that statin use was associated with a significantly lower risk of fatal or severe COVID-19 (pooled hazard ratio: 0.70, 95% confidence intervals 0.53 to 0.94) compared with nonuse. This finding strongly supports the clinical importance of continuing or initiating (according to current guidelines[2]) statin treatment in the COVID-19 era. The authors mention some pathophysiological mechanisms that could explain this beneficial impact of statins, including anti-inflammatory actions and upregulation of angiotensin-converting enzyme 2 expression. There are also other mechanisms that have been described, including the degradation of lipid rafts, directly affecting severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) S-protein, and reducing the cholesterol important for viral entry into host cells.[3,4]

Another mechanism could involve acute kidney injury and acute cardiac injury, both of which are predictors of COVID-19 mortality.[5,6] Statins have been shown to prevent contrast-induced acute kidney injury[7,8] and stabilize atherosclerotic plaques,[9] thus protecting from acute adverse events from the cardiac and renal system. Furthermore, statins may favorably affect immunomodulation, oxidative stress, and thrombosis.[10]

It should be noted that a combination of statins/angiotensin II receptor blockers improved outcomes and increased survival of patients infected during the 2014 Ebola virus disease epidemic in Sierra Leone.[11]

Drug-drug interactions should also be considered when treating COVID-19 patients on statins with antibiotics (e.g., macrolides) and antiviral drugs, due to an increased risk of statin-associated muscle symptoms.[12,13] Furthermore, the use of lovastatin and simvastatin is contraindicated in patients on ritonavir/lopinavir therapy since they might increase the risk of rhabdomyolysis.[14] Specific drugs used to combat SARS-CoV2 infections could have pharmacokinetic interactions with statins that affect plasma concentrations and toxicity of both statins and/or antiviral medication.[12] Knowledge in this field might help prescribers to use suitable doses and preparations and thus avoid treatment discontinuation for COVID-19 infection and prevent its complications.

Overall, statins can exert beneficial effects on the heart, vascular, and lung function, as well as inflammation,[15] thus strongly supporting their continuation or initiation, based on recommendations,[2] during the COVID-19 pandemic.

Conflict of interest

NK has given talks, attended conferences and participated in trials sponsored by Astra Zeneca, Bausch Health, Boehringer Ingelheim, Elpen, Mylan, Novo Nordisk, Sanofi and Servier. **MB** speakers bureau: Abbott/Mylan, Abbott Vascular, Actavis, Akcea, Amgen, Biofarm, KRKA, MSD, Polpharma, Sanofi-Aventis, Servier and Valeant; consultant to Abbott Vascular, Akcea, Amgen, Daichii Sankyo, Esperion, Freia Pharmaceuticals, Lilly, MSD, Polfarmex, Resverlogix, Sanofi-Aventis; Grants from Sanofi and Valeant. **DPM** has given talks and attended conferences sponsored by Amgen, Novo Nordisk, and Libytec.

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9 October 2020

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https://doi.org/10.1016/j.amjcard.2020.10.026

Correlation Between Exercise Metabolic Equivalents and Risk Factors in Nonathletes With Atrial Fibrillation

Despite the extensive benefits of exercise on cardiovascular health, recent data demonstrate that long durations of intense exercise may increase risk for developing atrial fibrillation (AF), in particular for individuals with lone AF (ie, AF in the absence of traditional risk factors [RF]).[1,2] Despite knowledge of elevated risk among dedicated athletes, it remains unclear if there is a correlation between risk of developing AF in nonathletes and level of functional exercise capacity. This study examined a cohort of patients aged <50 years and matched counterparts >50 years with diagnosis of AF and recent exercise stress testing. The hypothesis was that patients with minimal RFs (0-1) and AF would have achieved greater METs (metabolic equivalents—a standard measurement of energy expenditure ability) than patients with multiple RFs.

Patients were identified retrospectively from the Rush University Medical Center Patient Database between 06/01/2007 and 05/01/2020. Patient eligibility consisted of diagnosis of AF before the age of 50 and an exercise stress test within 5 years of AF diagnosis. RF for AF—including hypertension, CAD, moderate to severe valvular disease, obesity (BMI >40), OSA/COPD, CKD stage III or greater, history of alcohol abuse, and hyperthyroidism—were evaluated in correlation with exercise performance. The primary outcome was the difference in METs achieved by patients with low number of AF RF (0-1) versus patients with multiple RF (≥2). Propensity score matching in 1:1 ratio on AF RF and modifiers of exercise capacity (orthopedic injuries, asthma, degenerative joint disease) was used to compare patient groups <50 years of age and >50 years of age with AF (optimal matching; standardized mean difference<20% for all matching variables). Statistical analyses were performed using R statistical package (R Core Team, Vienna, Austria).

Among 100 patients aged <50 years (median age 41.0 years [34.0, 45.0]; male 68%), 49 patients (49%) had 0-1 risk factors for AF. Of those patients, 92% had paroxysmal AF and 6% were on chronic anticoagulation. Of the 3 patients on anticoagulation, 2 had prior strokes with known PFOs, and 1 had Factor V Leiden mutation. In the group with 0-1 risk factors, 11 patients (23%) underwent ablation and 6 cardioversion (12%), 7 (15%) received antiarrhythmic and 17 (35%) rate control therapy, and 13 (27%) were untreated. Two patients (4%) had stroke/TIAs subsequent to AF diagnosis. A correlation was not observed between total number of METs achieved and left atrial size by volume index or diameter.

The median number of METs in the low RF group (0-1 risk factors) was significantly higher than in the high RF group (11.7 METS vs 8.9 METS; $p < 0.001$; Fig. 1). Additionally, there was an inverse correlation between the number of RF for AF and the total number of METs performed ($p = 0.002$; Fig. 1). In comparison, this trend between number of risk factors and METs performed was not observed in the propensity-matched group of older patients (>50 years of age) with AF ($p = 0.08$). For those who achieved >10 METSs, patients in the younger AF group were more likely to have only 0-1 risk factors than the older group (21.8% vs 12.8%; $p = 0.01$).

Discussion Among patients with AF diagnosed before the age of 50, we found that higher exercise capacity on treadmill exercise stress testing was associated with a lower number of traditional RF for AF. This same correlation was not observed in a comparable group of patients above the age of 50 with AF who were 1:1 propensity-matched for baseline covariates, suggesting levels of exercise activity may have greater influence on proclivity for AF in younger patients with minimal traditional risk factors. Several mechanisms have been postulated for the underlying causes of AF in athletes including left atrial dilation and increased vagal tone.[3] Given that vagal