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Electrocardiographic findings in young competitive athletes during acute SARS-CoV-2 infection

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Abstract

Initial guidelines recommended a 12-lead electrocardiogram (ECG) in young competitive athletes following SARS-CoV-2 infection to screen for myocarditis. However, no data are available that detail ECG findings before and after SARS-CoV-2 infection in young athletes without clinical or imaging evidence of overt myocarditis. This study applied the International Criteria for ECG interpretation in a cohort of 378 collegiate athletes to compare ECG findings at baseline and during the acute phase of SARS-CoV-2 infection. Our results suggest that ECG changes can occur in the absence of definitive SARS-CoV-2 cardiac involvement in young competitive athletes.

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Introduction

Pre-participation cardiovascular screening (PPCS) with a 12-lead electrocardiogram (ECG) is used to enhance detection of pathologic cardiac conditions in young competitive athletes. Given ECG changes can occur in the setting of viral myocarditis, ECG was recommended in the evaluation of competitive athletes following SARS-CoV-2 infection [1]. Studies have shown the prevalence of SARS-CoV-2 cardiac involvement to be low among competitive athletes (0.5–3%) [2–4]. However, the stability of the ECG after uncomplicated SARS-CoV-2 infection has not been assessed. The aim of this study was to assess whether SARS-CoV-2 infection results in ECG changes in athletes without a clinical or cardiac imaging diagnosis of myocarditis to better inform the role and accuracy of ECG testing after SARS-CoV-2 infection.

Material and methods

This prospective observational cohort study included institutions from the National Collegiate Athletic Association (NCAA) who submitted data to the Outcomes Registry for Cardiac Conditions in Athletes (ORCCA) from September 1, 2020 to May 21, 2021. A detailed description of the ORCCA study has been reported previously [2]. Inclusion criteria for this study were: 1) athletes with confirmed SARS-CoV-2 infection, 2) an available baseline (pre-infection) ECG with no pre-existing cardiac condition, 3) an available ECG during or immediately after acute infection, and 4) no abnormalities on cardiac imaging (transthoracic echocardiography [TTE] and/or cardiac magnetic resonance [CMR]) related to SARS-CoV-2 infection [2]. ECG findings were defined as normal or abnormal per the International Criteria [5]. Each ECG was interpreted by 2 experienced readers (NM, BJP, TWC) who were blinded to athlete characteristics (i.e. sport, institution) other than race and age. Discrepancies in interpretation were settled by consensus between the 3 readers, and all abnormal ECGs were reviewed by the entire author team for final consensus. Continuous variables are presented as mean (SD) or median (IQR). Categorical variables are presented as n (%). All aspects of this study were approved by the Mass General Brigham Institutional Review Board (Protocol #2020P002667).

Results

A total of 378 (10%) athletes met inclusion criteria (age = 20 ± 1 years, 32% female, 65% white, 26% black race, 74% symptomatic). The median time from baseline ECG to SARS-CoV-2 infection was 456 [IQR...
109,792] days and from SARS-CoV-2 diagnosis to SARS-CoV-2 infection ECG was 13 [IQR 11,18] days. There were 275 (73%) athletes who had a TTE, 85 (22%) with CMR, and 18 (5%) with both TTE and CMR. A total of 8/378 (2%) athletes had an abnormal baseline ECG. Of these, 4/8 (50%) athletes had a persistently abnormal SARS-CoV-2 infection ECG, while 4/8 (50%) athletes had normalization of their initial abnormal ECG findings (Fig. 1). There were 370/378 (98%) athletes with normal baseline ECG, of whom 15/370 (4%) had an abnormal SARS-CoV-2 infection ECG (Fig. 1). The most common new ECG abnormalities were T-wave inversions (6/15), ST-depressions (4/15) and bi-atrial enlargement (2/15).

Discussion

This study compared ECGs during SARS-CoV-2 infection with pre-infection baseline tracings among young competitive athletes with normal cardiac imaging. A large increase in ECG changes during SARS-CoV-2 infection was noted, suggesting that SARS-CoV-2 myocardial involvement was not seen. Normalization of 50% of abnormal baseline ECGs and new abnormalities in 4% of ECGs may represent electrical variation over time in competitive athletes. Without controlling for training intensity, type, and duration, it is not possible to determine if these changes are related to physiological adaptations or new pathology. The long-term stability of ECG patterns in competitive athletes requires a larger and longitudinal investigation and is an important area of future work. We also cannot exclude the possibility of ECG changes related to differences in lead placement or undetected myocardial tissue changes in athletes without CMR. Overall, our findings suggest that ECG changes can occur in the absence of definitive SARS-CoV-2 cardiac involvement in young competitive athletes.

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Dr. Harmon discloses stock options for 98point6 for which she is also on the medical advisory board.

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Declaration of Competing Interest
None.

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