

Frequent constriction-like echocardiographic findings in elite athletes following mild COVID-19: in the grasp of SARS-CoV-2?

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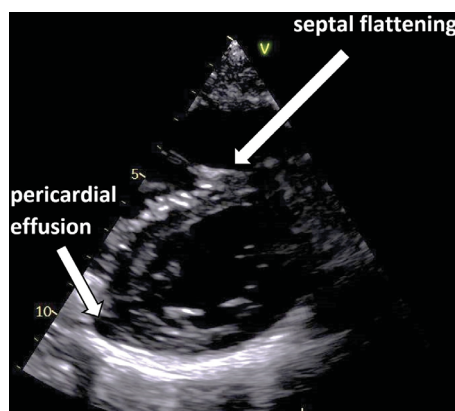
The COVID-19 pandemic had a major impact on the sports community as well. Despite the vast majority of athletes experiencing mild symptoms, potential cardiac involvement and complications have to be explored to support a safe return to play. Accordingly, we were aimed at a comprehensive echocardiographic characterization of post-COVID athletes (P-CA) by comparing them to a propensity-matched healthy, non-COVID athlete (N-CA) cohort.

One hundred and seven elite athletes with COVID-19 were prospectively enrolled after an appropriate quarantine period and formed the P-CA group (23±6 years, 23% female). From our retrospective database comprising 425 elite athletes, 107 age-, gender-, body surface area-, and weekly training hours-matched subjects were selected as a reference group using propensity score matching (N-CA group). All athletes underwent a comprehensive clinical investigation protocol comprising 2D and 3D echocardiography. Left (LV) and right ventricular (RV) end-diastolic volumes (EDVi) and ejection fractions (EF) were quantified using dedicated softwares. To characterize LV longitudinal deformation, 2D global longitudinal strain (GLS) and the ratio of free wall versus septal longitudinal strain (FWLS/SLS) were also calculated. In order to describe septal flattening (SF – frequently seen in P-CA), LV eccentricity index (EI) was measured. P-CA and N-CA athletes had comparable LV and RV EDVi (P-CA vs N-CA; 77±12 vs 78±13mL/m²; 79±16 vs 80±14mL/m², respectively). P-CA

group had significantly higher LV EF (58±4 vs 56±4%, $p<0.001$) and GLS (-18.2 ± 1.8 vs $-17.6\pm2.2\%$, $p<0.05$). Eccentricity index was significantly lower in P-CA (0.89 ± 0.10 vs 0.99 ± 0.04 , $p<0.001$), which was attributable to a distinct subgroup of P-CA athletes with a prominent SF ($n=34$, 32%), further provoked by inspiration. In this subgroup, the eccentricity index was markedly lower compared to the rest of the P-CA group (0.79 ± 0.07 vs 0.95 ± 0.07 , $p<0.001$).

In the SF subgroup, LV EDVi was significantly higher (80 ± 14 vs 75 ± 11 mL/m², $p<0.001$), while RV EDVi did not differ (82 ± 16 vs 78 ± 15 mL/m²). Moreover, the FWLS/SLS ratio was significantly lower in the SF subgroup (0.92 ± 0.09 vs 0.97 ± 0.08 , $p<0.01$). Interestingly, P-CA athletes with SF experienced fatigue (17 vs 34%, $p<0.05$) or chest pain (0 vs 15%, $p=N/A$) less frequently during the course of the infection; however, the presence of a mild pericardial effusion was more common (41 vs 12%, $p<0.01$).

Elite athletes following COVID-19 showed distinct morphological and functional cardiac changes compared to a propensity score-matched control athlete group. These results are mainly driven by a subgroup, which presented with some echocardiographic features characteristic of constrictive pericarditis (septal flattening, lower FWLS/SLS ratio, pericardial effusion). Follow-up of athletes and further, higher case number studies are warranted to determine the clinical significance and potential effects on exercise capacity of these findings.



Post-Covid athlete with SF