Original research

Prevalence and diagnostic significance of de-novo 12-lead ECG changes after COVID-19 infection in elite soccer players

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ABSTRACT

Background and aim The efficacy of pre-COVID-19 and post-COVID-19 infection 12-lead ECGs for identifying athletes with myocarditis has never been reported. We aimed to assess the prevalence and significance of de-novo ECG changes following COVID-19 infection.

Methods In this multicentre observational study, between March 2020 and May 2022, we evaluated consecutive athletes with COVID-19 infection. Athletes exhibiting de-novo ECG changes underwent cardiovascular magnetic resonance (CMR) scans. One club mandated CMR scans for all players (n=30) following COVID-19 infection, despite the absence of cardiac symptoms or de-novo ECG changes.

Results 511 soccer players (median age 21 years, IQR 18–26 years) were included. 17 (3%) athletes demonstrated de-novo ECG changes, which included reduction in T-wave amplitude in the inferior and lateral leads (n=5), inferior leads (n=4) and lateral leads (n=4); inferior T-wave inversion (n=7); and ST-segment depression (n=2). 15 (88%) athletes with de-novo ECG changes revealed evidence of inflammatory cardiac sequelae. All 30 athletes who underwent a mandatory CMR scan had normal findings. Athletes revealing de-novo ECG changes had a higher prevalence of cardiac symptoms (71% vs 12%, p<0.001) and longer median symptom duration (5 days, IQR 3–10) compared with athletes without de-novo ECG changes (2 days, IQR 1–3, p<0.001). Among athletes without cardiac symptoms, the additional yield of de-novo ECG changes to detect cardiac inflammation was 20%.

Conclusions 3% of athletes demonstrated de-novo ECG changes post COVID-19 infection, of which 88% were diagnosed with cardiac inflammation. Most affected athletes exhibited cardiac symptoms; however, de-novo ECG changes contributed to a diagnosis of cardiac inflammation in 20% of athletes without cardiac symptoms.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ The efficacy of pre-COVID-19 and post-COVID-19 infection 12-lead ECGs for identifying athletes with myocarditis has never been reported.

WHAT THIS STUDY ADDS

⇒ De-novo ECG patterns following COVID-19 infection (3%) characterised by anomalies in the inferior and lateral leads, including low-amplitude T waves, flat T waves or inverted T waves, identify athletes with cardiac inflammation (88%) on cardiovascular magnetic resonance (CMR) imaging.

⇒ Whereas most affected athletes express cardiac symptoms, such de-novo ECG changes identify an additional 20% athletes presenting with non-cardiac symptoms.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The presence of cardiac symptoms and/or de-novo ECG changes offer a greater diagnostic yield to detect cardiac inflammation in comparison to offering a mandatory CMR, irrespective of symptomatology or ECG findings.

⇒ Despite a different approach, our data on the prevalence of inflammatory cardiac sequelae (2.9%) following COVID infection are similar to data reported in North American professional and collegiate athletes.

⇒ Our approach is a cheap and pragmatic method of identifying athletes with sub-clinical myocarditis in a setting where serial cardiac assessments including ECGs are conducted on an annual basis and may also be applicable to athletes playing at the grass-roots level.
INTRODUCTION

Observational studies reveal that up to 30% of individuals hospitalised for severe COVID-19 infection show biochemical evidence of myocardial injury. Old age, obesity, the presence of multiple risk factors for atherosclerosis and concomitant cardiac comorbidity are recognised risk factors, suggesting that plaque rupture is an important cause of myocardial injury; however, it is also well established that COVID-19 infection predisposes to myocarditis.

While caution must be advised when extrapolating these data to the younger and generally healthier athletic population, a diagnosis of myocarditis has serious implications in athletes due to its association with fatal arrhythmias during vigorous exercise. Reports from large cohorts of collegiate and professional athletes in the USA indicate that the prevalence of myocarditis following mild to moderate COVID-19 infection is 0.6%–3.0%. These figures are a justified incentive for identifying affected athletes as evidenced by several consensus-based evaluation protocols for infected athletes in North America and Europe. Given its widespread availability, the inclusion of a 12-lead ECG is common to all protocols; however, a single cross-sectional ECG is problematic because several physiological repolarisation changes affecting the J point, ST segment and T wave are also common manifestations in individuals with myopericarditis. Most elite sporting organisations, however, recommend annual assessments in athletes which provide an opportunity to investigate the significance of de-novo electrical changes during or shortly after viral infection.

The aim of our study was to report on the prevalence and diagnostic significance of de-novo ECG patterns following COVID-19 infection in a well-defined cohort of elite soccer players in the English, Dutch and Brazilian leagues.

METHODS

Setting

The English, Dutch and Brazilian Football Associations mandate at least an annual cardiac assessment in soccer players aged 16 years old and above. Our sports cardiology units, led by senior authors AK, AM, AV, DR, HJ, MP, RC, SS serve several clubs and maintain records of serial assessments for all athletes. During the ongoing COVID-19 pandemic, several clubs requested a minimum of a 12-lead ECG in athletes with confirmed COVID-19 infection. We compared ECGs performed during the last assessment, prior to testing positive for COVID-19 infection with ECGs performed following COVID-19 infection.

Subjects

Fully anonymised data from 511 athletes were collected from nine sports cardiologists serving 36 elite soccer clubs between 1 March 2020 and 15 May 2022 (figure 2). All athletes who were referred to a sports cardiologist following testing positive for COVID-19 infection on PCR and/or antibody test and in whom a previous ECG was available were included. All previous ECGs were reported in accordance with the international recommendations for ECG interpretation in athletes.

Athletes with a previously abnormal baseline ECG were excluded (n=6). These included athletes who were under regular surveillance due to marked repolarisation changes indicative of an overt cardiomyopathic phenotype. All pre-COVID-19 ECGs were performed during the routine cardiac evaluation at the onset of the soccer season.

All athletes underwent an initial clinical evaluation including a clinical history and 12-lead ECG. The clinical history pertained to the presence and duration of cardiovascular symptoms, as well as a comprehensive systems review. A standard 12-lead ECG was conducted in a supine position at a paper speed of 25 mm/s and voltage of 10 mm/mV.

De-novo ECG patterns: criteria for an abnormal post COVID-19 ECG

The international recommendations for ECG interpretation recommend further investigations in athletes with several anomalies that may reflect myopericarditis such as atrioventricular block, left bundle branch block, ST-segment depression, T-wave inversion (TWI) and ventricular extrasystoles. Given that some repolarisation changes overlap between myopericarditis and athletes’ heart, we also considered the following ECG patterns as abnormal if they were not detected on the pre-COVID-19 infection ECG: PR-segment depression, new J-point and ST-segment...
elevation (≥0.2 mV in the precordial leads and ≥0.1 mV in the limb leads), low QRS voltages, complete right bundle branch block, QRS fragmentation, new ST-segment depression, new TWI, biphasic T waves and a reduction in the T-wave amplitude by 50% or T-wave flattening.

Further investigation
Athletes with de-novo ECG changes underwent transthoracic echocardiography (TTE) and cardiovascular magnetic resonance (CMR) imaging. One institution included CMR imaging in all athletes, with COVID-19 infection, irrespective of non-cardiac symptoms, mild disease severity or the absence of de-novo ECG changes.

Transthoracic echocardiography
TTE was performed by sonographers accredited by the British Society of Echocardiography (BSE) and/or the European Association of Cardiovascular Imaging. Cardiac measurements and interpretation were conducted in accordance with a standardised minimum dataset and a joint policy statement of the BSE and Cardiac Risk in the Young, which accounts for physiological cardiac adaptations to vigorous exercise.18

CMR imaging
CMR imaging was considered as the gold-standard non-invasive modality to assess for myocarditis. Scans were performed either using a 1.5 or 3.0 T scanner with ECG-synchronised cine acquisitions. Sequences included multiple white blood steady-state free precession images and cines, multiple turbo spin echo black blood images, T2 short-tau inversion recovery images, tissue characterisation and delayed enhancement images following gadolinium. In athletes with suspected acute or active myocardial inflammation, we applied the Updated Lake Louise Imaging criteria.19 All scans were performed in large-volume tertiary centres and reported by experts formally accredited in CMR with particular expertise in inherited cardiac conditions and sports cardiology. Furthermore, most scans were dual reported by at least two experts in the field.

Inflammatory cardiac sequelae
To facilitate onward management and risk stratification, in a cohort of ostensibly healthy athletes, the authors considered the following clinical definitions to indicate inflammatory cardiac sequelae:

► Acute pericarditis. the presence of at least two of the following criteria: (1) chest pain—typically sharp and pleuritic, alleviated by sitting up and leaning forward; (2) ECG changes—with new PR depression or ST elevation; and (3) the presence of a pericardial effusion, either using TTE or CMR imaging.

► Definitive myocarditis: the presence of at least three of the following criteria (1) cardiac symptoms; (2) where
performed, an elevated serum cardiac troponin; (3) the presence of de-novo ECG changes; (4) regional wall motion abnormalities or impaired left ventricular ejection function (LVEF <50%) on TTE; and (5) CMR findings consistent with an episode of active inflammation.

- **Possible myocarditis**: the presence of at least three of the following criteria (1) cardiac symptoms; (2) where performed, an elevated serum cardiac troponin; (3) the presence of de-novo ECG changes; (4) regional wall motion abnormalities or impaired left ventricular function (LVEF <50%) on TTE but where an athlete did not undergo CMR, or in absence of acute inflammation on CMR.

- **Probable myocarditis**: the presence of at least three of the following criteria (1) cardiac symptoms; (2) where performed, an elevated serum cardiac troponin; (3) the presence of de-novo ECG changes; (4) regional wall motion abnormalities or impaired left ventricular function (LVEF <50%) on TTE; and (5) CMR findings consistent with sub-papillary or mid-wall scar.

**Statistical analysis**

Data are expressed as medians (IQRs) or percentages as appropriate and analysed with Microsoft Excel V.16.61.1 and Statistics V.13.1. Comparison between groups was performed using the Student t-test or U-Mann Whitney test for continuous variables, the decision on the test used was based on the outcome of the Shapiro-Wilk test for data distribution. The chi-square test was used for categorical variables, with the Fisher exact test considered in the cases with more than 20% of cells having expected frequencies of <5. Statistical significance was defined as a p value of <0.05.

**RESULTS**

The median age of athletes was 21 years (IQR 18–26 years), of which 88% were male. The cohort was ethnically diverse, consisting of 58% white athletes, 18% black athletes and 17% athletes of mixed ethnicity. In accordance with public health guidance at the time of the study, following a mandatory period of isolation of 7–10 days, the median time for a sports cardiology specialist review following a positive COVID-19 PCR/antibody test was 11 days (IQR 10–14 days). From February 2022, the legal requirements around self-isolation for individuals who tested positive for COVID-19 were abolished in the UK, which shortened the gap between an athlete testing positive for COVID-19 infection and being reviewed by a sports cardiologist was a median of 7 days (IQR 6–9 days). The median duration of illness was 2 days (IQR 1–3 days). The median time interval between the pre-COVID-19 and post-COVID-19 ECGs was 239 days (IQR 164–462 days).

A total of 494 (97%) athletes had a normal post COVID-19 ECG. Of these, 4 (0.8%) reported persistent cardiac symptoms, with a median duration of illness of 12 days (IQR 9–17 days), and downstream investigations, including CMR imaging, resulted in a diagnosis of pericarditis in all four athletes. None of the athletes in this group revealed overt features of definite, probable or possible myocarditis.

Seventeen (3%) athletes demonstrated de-novo ECG changes, which included a reduction in T-wave amplitude by 50% in ≥2 contiguous leads with the preceding R-wave taller than 0.3 mV in the inferior and lateral leads (n=5), isolated inferior leads (n=4) and isolated lateral leads (n=4); new TWI in the inferior leads (n=7), inferior and lateral leads (n=1), anterior and lateral leads (n=1) and isolated lateral leads (n=1); ST-segment depression (n=2); biphasic T-waves (n=2); new J-point and ST-segment elevation of ≥0.2 mV in the precordial leads (n=1) and new J-point and ST-segment elevation of ≥0.1 mV in the limb leads (n=2) (figure 2 and online supplemental file table 1). The median time from a positive PCR to a 12-lead ECG in individuals who revealed de-novo ECG changes was 13 days (IQR 10–15 days).

Fifteen (88%) athletes with de-novo ECG changes were diagnosed with inflammatory cardiac sequelae including pericarditis (n=5), probable healed myocarditis (n=3), definitive myocarditis (n=3), possible myocarditis (n=2) and healed myocarditis (n=2) (online supplemental file table 1 and figures 2–4). These changes occurred in individuals who had a median illness duration of 7 days (IQR 4–14 days), and 12 (80%) of the athletes reported cardiac symptoms. Two (0.4%) athletes who demonstrated new inferior TWI (n=1), and inferolateral T-wave flattening (n=1) showed no evidence of inflammatory cardiac changes on downstream testing. The overall prevalence of inflammatory cardiac sequelae in the cohort based on de-novo ECG changes was 2.9%. Among athletes without cardiac symptoms, the additional yield of de-novo ECG changes to detect cardiac inflammation was 20% (n=3; online supplemental file table 1: athlete numbers 4, 5 and 7 demonstrated the presence of late gadolinium enhancement (LGE)).

Twelve athletes identified with inflammatory cardiac sequelae demonstrated resolution of de-novo ECG changes (online supplemental file table 1 and figures 3 and 4); the ECG changes persisted in one athlete with healed myocarditis; and follow-up ECG was not available in two players who moved clubs.

None of the 30 asymptomatic or paucisymptomatic athletes who were investigated with a CMR despite a normal ECG revealed cardiac inflammation compared with 88% of the athletes with de-novo ECG changes (tables 1 and 2). Athletes revealing de-novo ECG changes had a longer symptom duration (median 5 days, IQR 3–10 days) compared with athletes undergoing mandatory CMR assessment (median 0 days (asymptomatic), IQR 0–3 days; p<0.0001). There were no significant differences in quantitative CMR parameters between athletes with de-novo ECG changes and athletes who underwent mandatory CMR despite the absence of cardiac symptoms or abnormal ECG (table 1).

**Follow-up**

All athletes with confirmed cardiac inflammation were appraised by experts in the field. Conventional risk stratification included a maximal exercise stress test, ambulatory Holter monitor, prolonged arrhythmia monitoring for up to 2 weeks in certain cases, and close surveillance by the club doctors and respective sports cardiologist. None of the athletes revealed significant impairment in functional and haemodynamic status on exercise testing, and arrhythmia monitoring did not reveal any evidence of complex ventricular arrhythmia or myocardial ischaemia.

During a median follow-up of 270 days (IQR 133–487 days), there were no adverse cardiac events among infected athletes in the entire cohort. All athletes identified with cardiac inflammation returned to play following conventional risk stratification tests and tailored exercise prescriptions without adverse events over a median follow-up period of 479 days (IQR 268–520 days).

**DISCUSSION**

The resting 12-lead ECG has a low sensitivity for identifying individuals with myocarditis, which is estimated at <50%. Among athletes, the overlap between physiological
Special populations

repolarisation changes and myocarditis indicates that the specificity of the ECG may be even lower than the sensitivity. However, the precise significance of de-novo ECG

Figure 3  Athlete case 2. Twenty-day history of cardiovascular symptoms post COVID-19 infection. In comparison to his pre-COVID-19 ECG (A), new T-wave inversion was observed in leads II, III, aVF and V3–V6 (B); a cardiac MRI (C) demonstrated mid-wall LGE in the basal inferior and inferolateral wall; subepicardial LGE in the mid-lateral, apical lateral and inferior walls; with resolution of ECG changes 93 days post positive PCR (D). LGE, late gadolinium enhancement.

Figure 4  Athlete case 3. Twenty-eight-day history of symptoms post COVID-19 infection. In comparison to his pre-COVID-19 ECG (A), a reduction in T-wave amplitude II, aVF, biphasic T III was observed post COVID-19 infection (B). A cardiac MRI (C), a pericardial effusion around the basal inferior wall with ↑ signal on T2-STIR images, an abnormal T1 signal in the mid-inferolateral wall and subepicardial LGE in the basal and mid-inferolateral wall. Resolution of ECG changes was observed 170 days post positive PCR (D). LGE, late gadolinium enhancement; STIR, short-tau inversion recovery.
worthy that these criteria were derived from a cohort of acutely affected patients with H1N1 influenza virus revealed anomalous changes in ostensibly healthy young athletes with CO.

Almost 30% of hospitalised patients with COVID-19 and post-COVID-19 infection, based on similar disease definitions. As far as we are aware, this is the first study reporting the prevalence and significance of de-novo ECG changes in ostensibly healthy young athletes with COVID-19 infection of mild to moderate severity.

The most common ECG alterations following infection affected the inferior and lateral leads and were consistent with the typical cardiac MRI findings in individuals with myocarditis who demonstrated a predilection for myocardial scar localised to the basal inferolateral wall. Only three of our athletes fulfilled the Updated Lake Louise criteria for acute myocardial inflammation; however, it is noteworthy that these criteria were derived from a cohort of acutely unwell patients, where the CMR scan was performed in a timely fashion to detect myocardial oedema. In contrast, our athletes had mild or subclinical inflammation where the temporal relationship between confirmation of infection and CMR findings was probably too long to reveal acute inflammation but identified residual scar in most cases.

In the absence of a baseline CMR, it may be argued that the scar tissue may have been a compensatory response to decades of vigorous exercise. However, we did not find any evidence of inflammation or scar in 30 asymptomatic or paucisymptomatic athletes who underwent a mandatory CMR despite an unchanged ECG.

Despite a different approach to the triad testing in North American studies in athletes, which included troponin measurements, our prevalence (2.9%) of inflammatory cardiac sequelae post COVID-19 infection, based on similar disease definitions is similar to the upper limit of the prevalence reported in North America (3%).

Most athletes (80%) exhibiting de-novo ECG changes revealed symptoms compatible with myopericarditis and were symptomatic for a median of 7 days (IQR 4–14 days). Our observations are in keeping with North American and European recommendations which indicate that cardiac symptoms should be the main driver for selecting athletes for cardiac investigations post infection.

Although based on a small number of athletes, we noted that a minority of athletes who did not report cardiac symptoms but revealed de-novo ECG changes showed evidence of myocardial inflammation. It is well recognised from autopsy series that coryzal symptoms have predominated in a large proportion of decedents with viral myocarditis.

The uncertainty surrounding the current pandemic dictates that some sporting organisations may continue to investigate asymptomatic athletes with COVID-19 infection. Our study shows that comparison of pre-COVID-19 and post-COVID-19 ECGs is a cheap and effective method for detecting asymptomatic athletes with cardiac inflammation and may also be applicable to athletes playing at the grass-roots level.

Our approach raises concern about the possibility of false-positive ECG results; however, our observations revealed that only two athletes (12%) with de-novo ECG changes failed to demonstrate any evidence of cardiac inflammation during subsequent investigation. A recent study which applied the international criteria for ECG interpretation in athletes reported that as many as 4% of 378 collegiate athletes in whom pre-COVID-19 and post-COVID-19 ECGs were available revealed abnormalities following COVID-19 despite normal cardiac imaging. The precise significance of the ECG abnormalities in this cohort is

demonstrated features compatible with inflammatory cardiac sequelae.

ECG changes have been reported with other viral infections and may have prognostic relevance. Almost 30% of hospitalised patients with H1N1 influenza virus revealed anomalous patterns including TWI and ST-segment depression. Specific ECG changes such as diminished QRS amplitude in hospitalised patients infected with COVID-19 or influenza and pre-existing comorbidities were independently observed to precede clinical decompensation and were associated with an increased mortality. As far as we are aware, this is the first study reporting the prevalence and significance of de-novo ECG changes in ostensibly healthy young athletes with COVID-19 infection of mild to moderate severity.

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**Table 1** Comparison of CMR data between athletes demonstrating de-novo ECG changes and athletes undergoing a mandatory CMR

<table>
<thead>
<tr>
<th>Athletes undergoing mandatory CMR assessment (n=30)</th>
<th>Athletes demonstrating de-novo ECG changes undergoing CMR assessment (n=17)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>26 (19–30)</td>
<td>22 (19–25)</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>2 (1.9–2.1)</td>
<td>2 (1.9–2.1)</td>
</tr>
<tr>
<td>Symptom duration (days, 0=asymptomatic)</td>
<td>0 (0–3)</td>
<td>5 (3–10)</td>
</tr>
<tr>
<td>Positive PCR to ECG</td>
<td>11 (11–11)</td>
<td>12 (10–14)</td>
</tr>
<tr>
<td>Positive PCR to CM</td>
<td>11 (11–11)</td>
<td>15 (11.5–19.5)</td>
</tr>
<tr>
<td>LV Mass (g/m²)</td>
<td>10 (9–10)</td>
<td>9 (9–11)</td>
</tr>
<tr>
<td>LV Mass indexed (g/m²)</td>
<td>59 (57–62)</td>
<td>60 (57–64.3)</td>
</tr>
<tr>
<td>LV mass indexed (g/m²)</td>
<td>72 (54–83)</td>
<td>82 (65–93)</td>
</tr>
<tr>
<td>RVEF (%)</td>
<td>53 (51–60)</td>
<td>58 (51–62.5)</td>
</tr>
</tbody>
</table>

*P value <0.05 deemed statistically significant. Values are expressed as median (IQR).

**Table 2** Summary of cardiovascular symptom burden, de-novo ECG changes and MRI findings in overall cohort

<table>
<thead>
<tr>
<th>CV symptoms</th>
<th>De-novo ECG changes</th>
<th>CMR imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cohort, n=511</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No de-novo ECG changes, n=494</td>
<td>Persistent symptoms: n=4 (0.8%)</td>
<td>None: n=494 (97%)</td>
</tr>
<tr>
<td>Includes mandatory CMR group, n=30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>De-novo ECG changes group, n=17</td>
<td>Yes: n=12 (71%)</td>
<td>All underwent CMR: Normal: n=2 (12%)</td>
</tr>
</tbody>
</table>

*The clinical assessment as a minimum included a comprehensive history, systems review, examination and ECG. Further onward investigations were performed based on the respective sports cardiologists, club or team doctor policy. CMRI, cardiovascular magnetic resonance; CV, cardiovascular.
still unclear since only 22% of athletes with new ECG changes underwent CMR as opposed to all our athletes.

In our study, during a median follow-up period of 270 days (IQR 133–487 days), there were no adverse outcomes, which is reassuring, though long-term studies are required to ascertain the outcomes in athletes with myocardial inflammation post COVID-19 infection.

Limitations
Our study has several limitations which warrant mention. The cohort of interest, notably, athletes with de-novo ECG changes, was relatively small and reflects the low prevalence of myocarditis in athletes infected with COVID-19. It is possible that some of the de-novo ECG changes reflected change in variation in training intensity. However, most of our pre-COVID-19 ECGs were performed as part of routine cardiac evaluations just prior to the onset of the season when players are extremely conditioned. The absence of cardiac inflammation in equal numbers of athletes with normal ECGs who underwent CMR suggests that the de-novo ECG changes we observed were a genuine representation of cardiac pathology. Importantly, we did not rely on inflammatory markers and/or biomarkers of cardiac damage and systemic inflammation. In view of public health measures to curtail viral spread, which largely included a mandatory period of self-isolation for athletes, we were mindful that measures of inflammation might have normalised. Furthermore, many of our asymptomatic athletes continued to partake in regular physical activity while isolating, which is a recognised cause of transient elevation in serum cardiac troponin.

CONCLUSIONS
Three per cent of athletes developed de-novo ECG changes following COVID-19 infection, of which 88% revealed inflammatory cardiac sequelae. Whereas most of the athletes in question reported cardiac symptoms and would have been assessed as standard of care, a small minority either had mild non-cardiac symptoms or were asymptomatic. In the absence of cardiac symptoms, de-novo ECG changes post COVID infection contributed to 20% of all cases of cardiac inflammation.

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Contributors
All authors were involved in conception, design, analysis and interpretation of data, and drafting the article. All authors critically revised the manuscript and approved the final version to be published. MP and SS contributed jointly as senior authors and are both guarantors for the work.

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Competing interests
SS is on the editorial board of Heart BMJ.

Patient and public involvement
Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans of this research.

Patient consent for publication
Not applicable.

Ethics approval
The study was given ethical approval by the St. George’s Research Ethics Committee. Written informed consent was obtained from each athlete, in accordance with the Football Association governance department, respective sporting bodies and team management in the Netherlands and Brazil, which acknowledge that anonymised clinical information may be used for medical research purposes.

Provenance and peer review
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Data availability statement
Data are available upon reasonable request. Requests should be made to the senior author SS.

Supplemental material
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