**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- and post-COVID-19 infection 12-lead electrocardiograms (ECGs) for identifying athletes with myopericarditis has never been reported. We aimed to assess the prevalence and significance of de-novo ECG patterns following COVID-19 infection.

**Methods:** In this multicentre observational study, between March 2020–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 sequalae. All 30 athletes who underwent a mandatory CMR in the absence of cardiac symptoms or de-novo ECG changes had normal findings. Athletes revealing de-novo ECG changes, had a higher prevalence of cardiac symptoms (71% v 12%; p<0.0001) 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.

**Clinical Perspective**

**What is new?**

1. The efficacy of pre- and post-COVID-19 infection 12-lead electrocardiograms (ECGs) for identifying athletes with myopericarditis has never been reported.
2. 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.
3. Whereas most affected athletes express cardiac symptoms, such de-novo ECG changes identify an additional 20% athletes presenting with non-cardiac symptoms.

**What are the clinical implications?**

1. 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.
2. Despite a different approach, our prevalence of inflammatory cardiac sequalae (2.9%) following COVID infection is similar to data reported in North American professional and collegiate athletes.
3. 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.1,2 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.2,3 Whilst 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 (figure 1).4–6

Reports from large cohorts of collegiate and professional athletes in the United States, indicate that the prevalence of myocarditis following mild to moderate COVID-19 infection is 0.6-3%.7,8 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.9–15 Given its widespread availability, the inclusion of a 12-lead electrocardiogram (ECG) is common to all protocols,9–14 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.16 Most elite sporting organisations, however, recommend annual assessments in athletes which provides 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 league.

**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, RS and SS serve several clubs and maintain records of serial assessments for all athletes. During the on-going 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. The study was given ethical approval by the St. George’s Research Ethics Committee (SGREC) 2021.0139.

**Subjects**

Deidentified data from 511 athletes was collected from 9 sports cardiologists serving 36 elite soccer clubs between 1st March 2020 and 15th May 2022 (central illustration). All athletes who were referred to a sports cardiologist following testing positive for COVID-19 infection on polymerase chain reaction (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.16 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 a covert 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 10mm/mV. In women, the ECG electrodes were placed under the breast tissue according to American Heart Association recommendations.17

**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 (AV) block, left bundle branch block, ST-segment depression, T-wave inversion (TWI) and ventricular extrasystoles.16 Given that some repolarisation changes overlap between myopericarditis and athlete’s 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 (≥0.2mV in the precordial leads and ≥0.1mV in the limb leads), low QRS voltages, complete right bundle branch block, QRS fragmentation, new ST-segment depression, new T-wave inversion, 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)

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

Cardiovascular magnetic resonance (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 tesla or 3 tesla scanner with ECG-synchronised cine acquisitions. Sequences included: multiple white blood steady-state free precession (SSFP) images and cines, multiple turbo spin echo (TSE) black blood images, T2 short tau inversion recovery (STIR) 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 sequalae**

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 sequalae:

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

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

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

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

**Statistical analysis**

Data are expressed as medians (IQRs) or percentages as appropriate and analysed with Microsoft excel version 16.61.1 and Statistica 13.1. Comparison between groups was performed using Student t-test or U-Mann Whitney test for continuous variables and chi-square test for categorial variables. Statistical significance was defined as p<0.05.

**Results**

The median age of athletes was 21 years; interquartile range [IQR]: 18 to 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 7 to 10-day period of isolation, the median time for a sports cardiology specialist review following a positive COVID-19 PCR/antibody test was of 11 days (IQR: 10-14 days). From February 2022, the legal requirements around self-isolation for individuals who tested positive for coronavirus was abolished in the United Kingdom, 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). 72 (14%) players reported cardiovascular symptoms, which included chest pain (n=41; 8%); dyspnoea (n=37; 7%); and palpitation (n=6; 1%). The median time interval between the pre and post COVID-19 ECG was 239 days (IQR: 164-462 days).

494 (97%) athletes had a normal post COVID-19 ECG. Of these, 4 (0.8%) had 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 4 athletes. None of the athletes in this group revealed overt features of definite, probable, or possible myocarditis.

17 (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 T-wave inversion 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 ≥ 0.2mV in the precordial leads (n=1) and new J-point and ST-segment elevation ≥ 0.1mV in the limb leads (n=2) (central illustration, 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).

15 (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) (central illustration, table 1, figure 2 and figure 3). 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. 2 (0.4%) athletes 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 3%. Among athletes without cardiac symptoms, the additional yield of de-novo ECG changes to detect cardiac inflammation was 20% (n=3; table 1: athlete number 4,5, and 7 demonstrated the presence of late gadolinium enhancement, LGE).

12 athletes identified with inflammatory cardiac sequalae, demonstrated resolution of de-novo ECG changes (Figure 3 and 4); the ECG changes persisted in 1 athlete with healed myocarditis; and follow up ECG were not available in 2 players who moved clubs (table 1, figure 3 and figure 4).

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 (table 2). Athletes revealing de-novo ECG changes had a longer symptom duration (median 5 days [IQR 3-10 days]) compared to 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 2).

**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 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%.20,21 Amongst athletes, the overlap between physiological repolarization 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 patterns following COVID-19 infection in athletes, has not been investigated. Our study revealed that 3% of professional soccer players revealed de-novo ECG changes, of which, 88% demonstrated features compatible with inflammatory cardiac sequalae.

ECG changes have been reported with other viral infections and may have prognostic relevance. Almost 30% of hospitalized patients with H1NI influenza virus revealed anomalous patterns including T-wave inversion and ST-segment depression.22 Specific ECG changes such as diminished QRS amplitude in COVID-19 infection or influenza infected hospitalized patients with pre-existing comorbidities was independently observed to precede clinical decompensation and were associated with an increased mortality.23,24 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 are consistent with the typical cardiac MRI findings in individuals with myocarditis, who demonstrate a predilection for myocardial scar localised to the basal inferolateral wall.25

Only 3 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.26,27 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 athletes7, which included troponin measurements, our prevalence (2.9%) of inflammatory cardiac sequalae post COVID-19 infection, based on identical disease definitions7,8,28 is similar to the upper limit of the prevalence reported by from American studies (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.10,28 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.

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-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 0.4% of athletes 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 athletes16, reported that as many as 4% of 378 collegiate athletes in whom pre and post COVID-19 ECGs were available revealed abnormalities following COVID-19 despite normal cardiac imaging29. The precise significance of the ECG abnormalities in this cohort is 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 patterns 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, by which time we were mindful that measures of inflammation might have normalised. Furthermore, many of our asymptomatic athletes continued to partake in regular physical activity whilst isolating which is a recognised cause of transient elevation in their serum cardiac troponin.

**Conclusions**

3% of athletes developed de-novo ECG changes following COVID-19 infection, of which 88% revealed inflammatory cardiac sequalae. Whereas most athletes in question reported cardiac symptoms and would have been assessed as standard of care, a small minority 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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**Table 1:** Clinical characteristics of COVID-19 positive (PCR) athletes with de-novo ECG patterns diagnosed with inflammatory cardiac sequalae

**Table 2:** Comparison of CMR data between athletes demonstrating de-novo ECG patterns and athletes undergoing a mandatory CMR.

**Figure 1:** Challenges in establishing a diagnosis of myocarditis in athletes following COVID-19 infection.

**Figure 2:** Prevalence and diagnostic significance of de-novo 12-lead ECG patterns following COVID-19 infection: study overview. ECG: electrocardiogram; TWI: T wave inversion.

**Figure 3:** Athlete case number 2. 25-year-old-white male. 20-day history of cardiovascular symptoms post COVID-19 infection. In comparison to his pre COVID-19 ECG (panel A), new T-wave inversion was observed in leads II, III, aVF, V3-V6 (panel B); a cardiac MRI (panel C) demonstrated mid-wall late gadolinium enhancement (LGE) in the basal inferior & inferolateral wall; subepicardial LGE in the mid lateral, apical lateral & inferior walls; with resolution of ECG changes 93 days post positive PCR (panel D).

**Figure 4:** Athlete case number 3. 19-year-old-white male. 28-day history of symptoms post COVID-19 infection. In comparison to his pre COVID-19 ECG (panel A), a reduction in T-wave amplitude II, aVF; biphasic T III was observed post COVID-19 infection (panel B). A cardiac MRI (panel 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 observed 170 days post positive PCR (Panel D).