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CASE REPORT
Heart failure

Consensus recommendations for COVID-19-related myocarditis in athletes: proof of concept—case report

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Background Post-viral myocarditis has been associated with sudden cardiac death in athletes. Since the beginning of the COVID-19 pandemic, the concern of post-viral myocarditis impacting the professional athletic community has been present.

Case summary An elite-level basketball player presented after a positive COVID-19 test with findings consistent with ventricular tachycardia related to myocardial fibrosis/scar from a COVID-19-related myocarditis. Although rare, COVID-19 myocarditis can occur. This case illustrates how the consensus guidelines for return-to-play correctly identified the player as high risk with appropriate downstream evaluation by cardiac magnetic resonance (CMR) imaging. The stepwise approach is illustrated in this case and highlights the utility and success of the algorithm when approaching athletes with COVID-19-related myocarditis risk and determining a return to exercise.

Discussion Diligence is required to identify competitive athletes with features suggestive of myocarditis at the initial presentation and with the return to exercise. Cardiopulmonary symptoms in the setting of recent COVID-19 infection should prompt additional testing in a stepwise fashion and often benefit from CMR in addition to the triad testing with electrocardiography, echocardiography, and cardiac troponin measurement to further investigate clinical presentations of COVID-19-related myocarditis.

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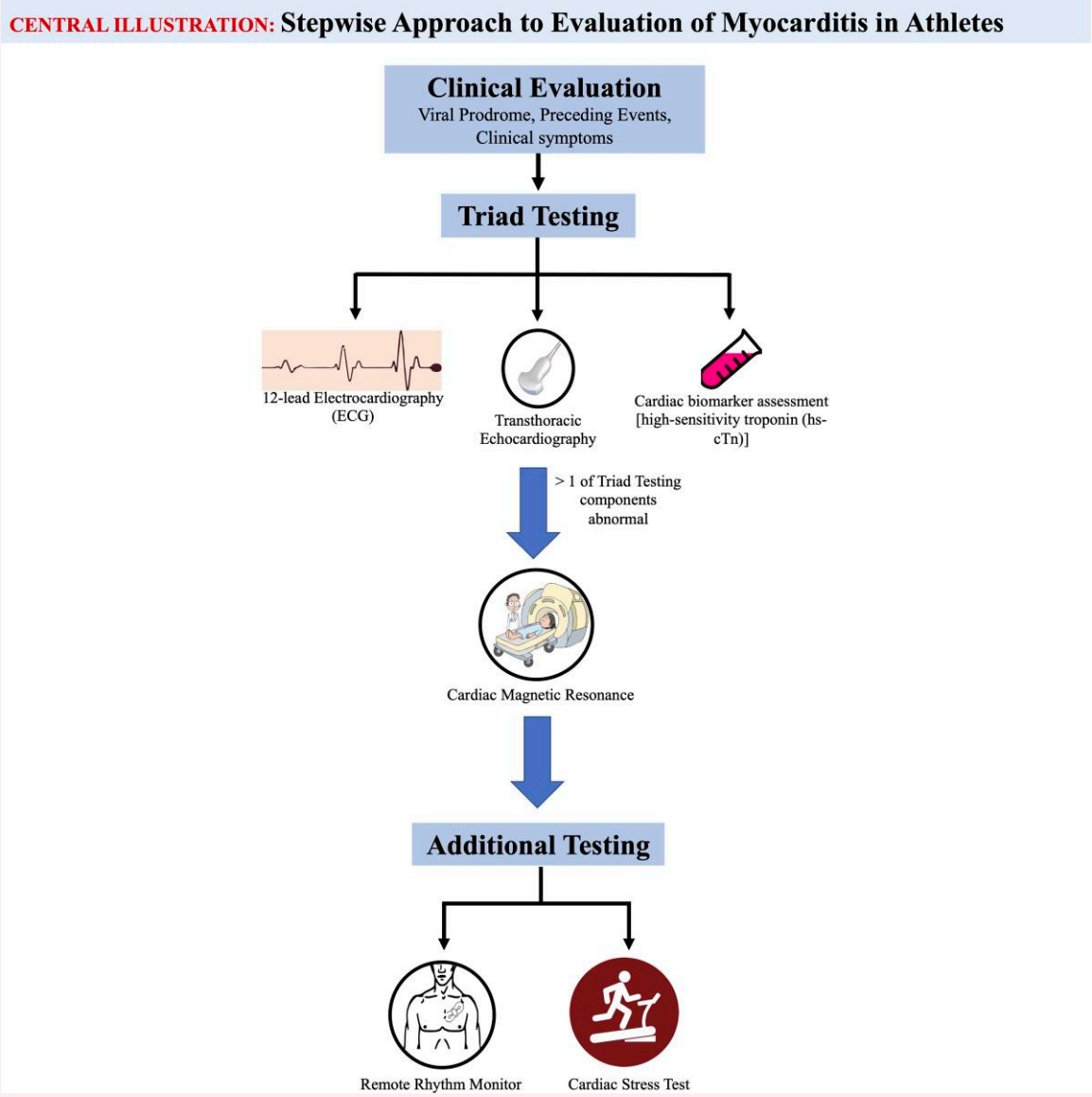
Graphical Abstract



Stepwise approach to the evaluation of myocarditis in athletes.

Keywords Athletes heart • COVID-19 • Myocarditis • Case report

ESC Curriculum 2.3 Cardiac magnetic resonance • 8.1 Sports cardiology • 6.1 Symptoms and signs of heart failure • 2.1 Imaging modalities

Learning points

- To be able to make a differential diagnosis of myocarditis with multimodality imaging.
- To understand the role of symptoms interpretation during the initial post-COVID-19 infection phase among athletes and the return to exercise portion of recovery.
- To incorporate the return-to-play guidelines and multimodality assessment of suspected myocarditis in professional athletes.

Introduction

Since the beginning of the SARS-coronavirus-2 (COVID-19) pandemic, there has been a rising concern in the field of sports cardiology, with the increased risk of sudden cardiac death (SCD) among athletes during training and competition if infected with COVID-19. Prior epidemiological studies highlighted the associated SCD risk with acute viral myocarditis or inflammatory heart disease.[1,2] News headlines of high-profile athletes not participating in their professional tournaments due to presumptive COVID-19-related myocarditis have gained popularity over the past year. Subsequently, the growing concerns about the safety of permitting athletes to return-to-play (RTP) after COVID-19 infection has brought to light the American College of Cardiology's (ACC) Sports & Exercise Cardiology Council revised consensus recommendations regarding RTP for athletes after COVID-19 infection.[3,4] During the early stages of the COVID-19 pandemic, little was known regarding the COVID-19-related myocarditis prevalence and its clinical outcomes in athletes. Therefore, the RTP recommendations remained purely consensus-based.[3] We describe a case of COVID-19-related myocarditis in an avid athlete with a reflection on RTP recommendations and a validated proof-of-concept stepwise approach in diagnosis and management among athletes.

Timeline

Timeline	Sequence of events
Days 1–9	Onset of COVID-19 symptoms
Day 20	Return-to-play (RTP) following initial investigations and cardiac imaging
Day 27	Recurrence of shortness of breath after RTP prompting further investigation, including cardiac magnetic resonance imaging (MRI) that revealed mild apical lateral hypokinesis and subepicardial late gadolinium enhancement involving the apical lateral segment
Days 30–90	Held from participation in sports
Month 3	Resolution of symptoms, stress test performed showing significant burden of premature ventricular complexes and non-sustained ventricular tachycardia (VT), leading to holding from activity for an additional 3 months
Month 6	Repeat MRI showed the resolution of scar/fibrosis, and a repeat stress test showed normal exercise tolerance with the absence of non-sustained VT at peak heart rate. Decision was made to RTP
Month 7	After 1 month of exercise, he wore an ambulatory monitor for 1 week, which did not demonstrate any significant arrhythmias with exercise

Case presentation

A 21-year-old African-American male basketball player presented after a positive COVID-19 PCR test 1 month earlier for an RTP assessment. His COVID-19 symptoms included diarrhoea, cough, shortness of breath, and nasal congestion for 9 days. No intervention or hospitalization was needed, and he felt he was fully recovered and was asymptomatic. His vital signs and physical examination were normal. His cardiac exam was within normal limits, including no abnormal heart sounds, regular pulse and rhythm, and equal strong peripheral pulses. His respiratory exam showed no abnormal breath sounds with equal inspiratory effort. His resting electrocardiography (ECG) is shown in *Figure 1*. He had no significant past family history of cardiomyopathies or sudden cardiac arrest before the age of 50 years. He denied any persistent dyspnoea, chest pain, palpitations, lightheadedness, dizziness, near syncope, or syncope. At that time, as per published guidance, an echocardiogram revealed normal left ventricular (LV) size and function (normal wall motion, diastolic function, and LV ejection fraction = 58%) (see Supplementary material online, *File S1*).[5,6] Troponin was undetectable. As the player was currently without symptoms, he returned to activity with a ramp-up of exercise.

He returned to exercise as per the recommendations and noted symptoms of shortness of breath and chest tightness, which began within a week of return to basketball practice.[5,6] This prompted a halt in exercise and additional testing. Cardiac magnetic resonance (CMR) imaging revealed normal size and function with an ejection fraction of 55% with mild apical lateral hypokinesis and subepicardial late gadolinium enhancement involving the apical lateral segment. No active oedema was present. This finding was consistent with myocardial fibrosis/scar related to myocarditis (*Figure 2A*). He was held from exercise as per the ACC/AHA recommendations for return to sport.[5] After 2 months, he was symptom-free; we decided to proceed with a stress test that was positive for inferolateral repolarization abnormalities at rest and positive for a significant burden of premature ventricular complexes (PVCs) (right bundle superior axis) likely originating from the LV inferolateral at the base and non-sustained ventricular tachycardia (VT) with exercise during the recovery phase (*Figure 3*). He was again held from the activity for an additional 3 months. A repeat CMR and echocardiography showed the resolution of scar/fibrosis and the prior mild apical lateral hypokinesis (*Figure 2B*). A repeat stress test found rare, isolated PVCs (right bundle superior axis) initially at rest that did not escalate with exercise and the absence of non-sustained VT at peak heart rate. Moreover, the patient had a normal exercise tolerance, normal hazard ratio, and blood pressure response without sustained arrhythmias.

Moreover, on follow-up ECG at 3 and 6 months, the PVC morphology remained unchanged. At this point, he began a slow RTP by ramping up his activities. Of note, the patient did not receive any medical therapy for myocarditis throughout the observation period, as his cardiac function was preserved. After 1 month of exercise, he wore an ambulatory monitor for 1 week, inclusive of a graded return to an exercise regimen, which did not demonstrate any significant arrhythmias with exercise.

Discussion

Post-viral myocarditis has been associated with SCD in athletes. Since the beginning of the COVID-19 pandemic, the concern of post-viral myocarditis impacting the professional athletic community has been present. The hallmark of acute post-viral myocarditis is based on a clinical syndrome with a preceding viral illness, including heart failure symptoms such as dyspnoea on excretion, angina-type chest pain, or myopericarditis of <3 months duration, and an otherwise unexplained elevation in serum troponin, electrocardiogram characteristics of high-degree atrioventricular (AV) block or arrhythmias, cardiac ischemia, and echocardiographic and CMR features of wall motion abnormality. Several consensus screening recommendations have been published,[3,6] all endorsing a conservative RTP screening approach early on. This includes the so-called triad testing with 12-lead ECG, cardiac biomarker assessment (high-sensitivity troponin), and transthoracic echocardiography to exclude cardiac injury.[7] Multiple large registries were created to track US professional and collegiate athletes who had recovered from COVID-19. Both the US professional cohort[8] and multicentre

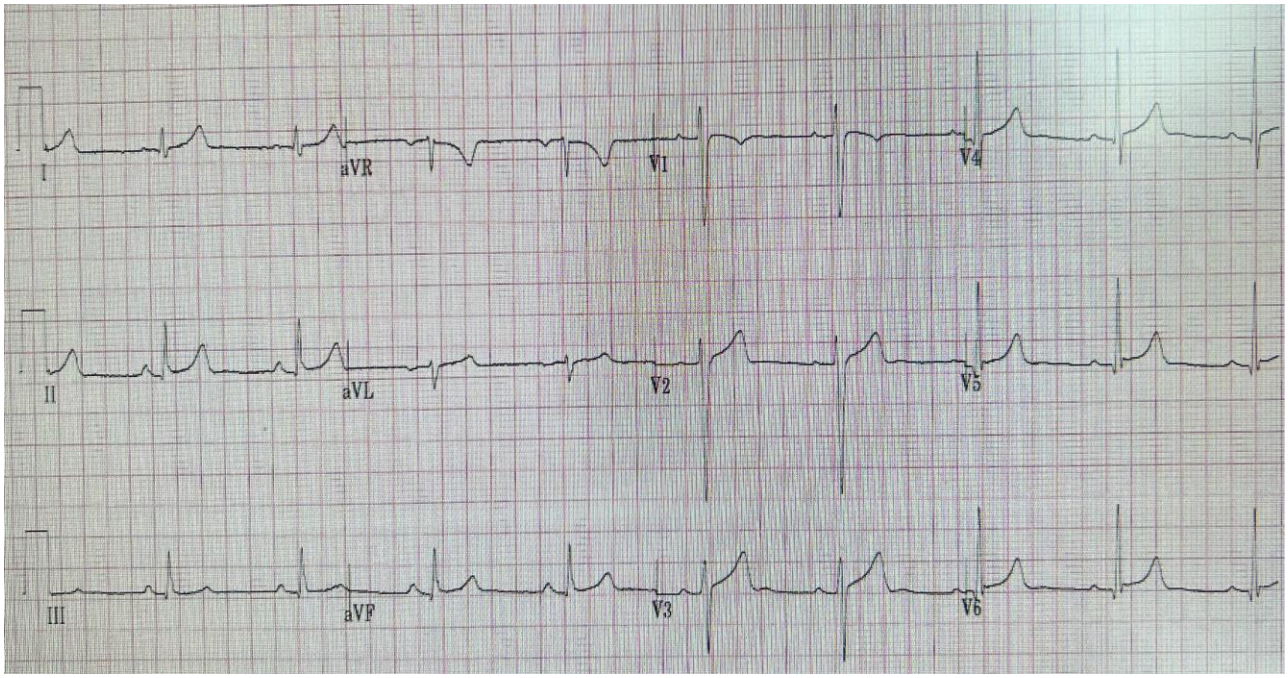


Figure 1 Resting electrocardiogram prior to participation in sports.

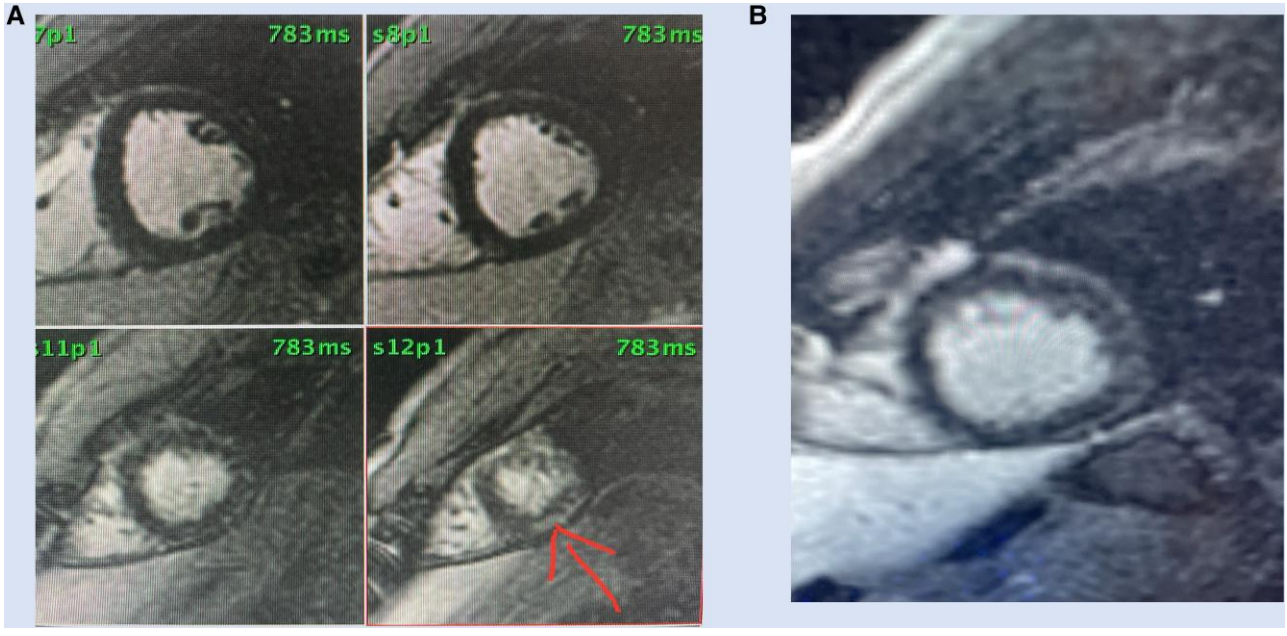


Figure 2 (*A*) Cardiac magnetic resonance imaging at the onset of symptoms; mild apical lateral hypokinesis with subepicardial late gadolinium enhancement involving the apical lateral segment. (*B*) Cardiac magnetic resonance imaging at follow-up; resolution of scar/fibrosis.

collegiate athlete cohort [Outcomes Registry for Cardiac Conditions in Athletes (ORCCA)][7] documented a low prevalence of cardiac injury (<0.7%) among previously infected athletes. Within these registries, CMR was generally obtained only if any initial triad testing was abnormal or if symptoms were suggestive of myocarditis.[7,8] In competitive athletes with concerning persistent findings such as syncope, palpitations, chest tightness/discomfort, or exertional cardiopulmonary symptoms, following the initial testing and RTP, more sensitive imaging with CMR is then warranted to further investigate the underlying subclinical pathology.[1,9] Hence, a stepwise approach to evaluating myocarditis

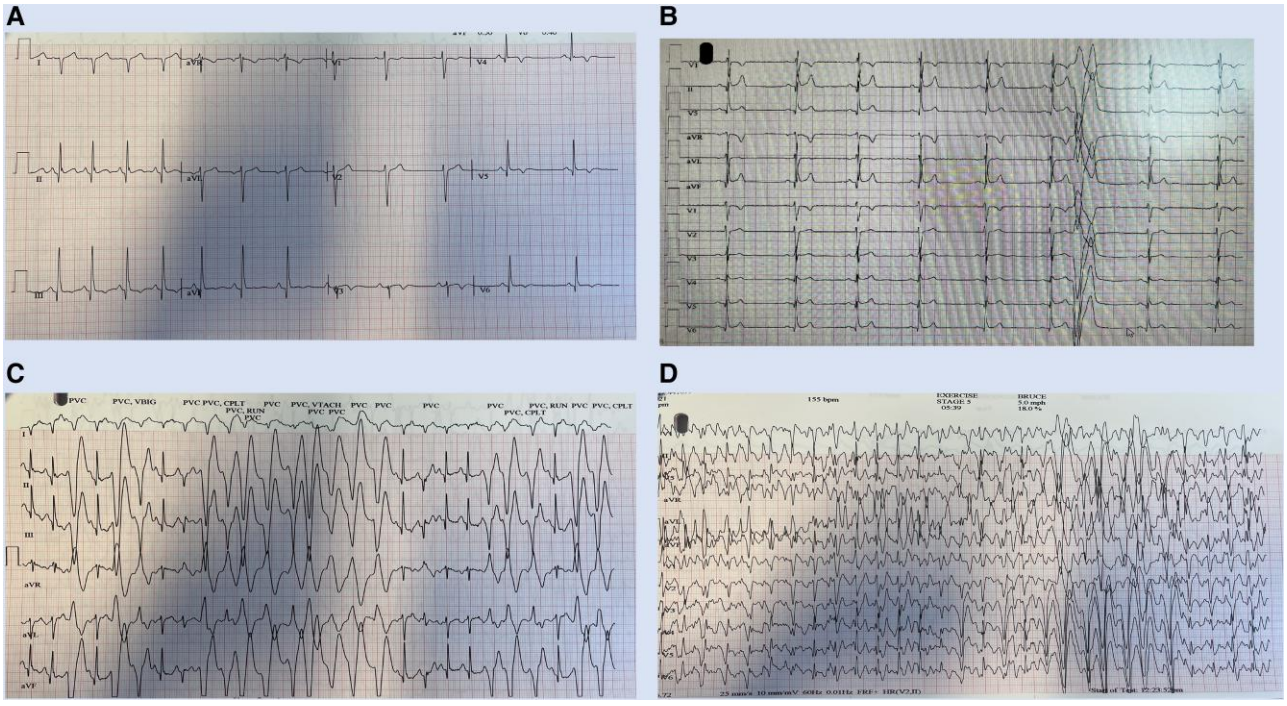


Figure 3 Stress electrocardiogram at the onset of symptoms. (*A*) Pretest supine electrocardiogram, (*B*) pretest standing electrocardiogram, (*C*) exercise electrocardiogram (Stage 5; 5.0 miles/h), and (*D*) recovery electrocardiogram.

among athletes is proposed (*Graphical Abstract*). In addition, cardiopulmonary symptoms suggestive of myocarditis were predictive of SARS-CoV-2 cardiac involvement.[7]

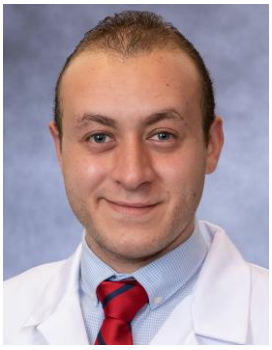
The concept of CMR for universal screening for all athletes, in addition to triad testing, was tested in a cohort of 1597 athletes from the Big Ten COVID-19 cardiac Registry, irrespective of COVID-19 symptomatology.[1] The addition of CMR to the triad testing did detect abnormalities suggestive of myocarditis in 2.3% of the athletes, although there was marked heterogeneity amongst the sites regarding CMR findings.[1] Overall, however, the results were consistent with the ORCCA and the professional athlete registries, with only nine athletes (0.6%) with clinical symptoms and findings consistent with acute myocarditis.[1,7] Importantly, no confirmed cases of cardiac death in the COVID-19 registries of athletes have been identified.[1,7,8] Similar findings have been noted in an unselected, non-athletic cohort of healthcare workers, in whom mild COVID-19 symptoms were not associated with cardiovascular involvement.[10] As illustrated in our case, cardiopulmonary symptoms are likely to be of greater relevance than age and athlete competition level when assessing the probability of underlying cardiac involvement.[7,11] Given that there is a large collective experience agreeable to significant cardiac complications detection with the utilization of symptom guidance and triad testing with CMR as downstream testing, any improvement in outcomes is unlikely with the addition of universal CMR utilization for all cases.

This case illustrates the importance of diligence regarding symptoms during the initial phase and the return to exercise portion of recovery and the wisdom of current published RTP guidance. Our athlete had symptoms with a return to exercise, which is an important finding regarding possible cardiac involvement after viral infection. This should prompt additional testing with a pause on activity while this is investigated further. In this case, CMR confirmed the suspicion of myocarditis, and high-risk features of ventricular arrhythmias with exercise were confirmed. Moreover, initial detection of oedema and late gadolinium enhancement on CMR, especially during initial presentation, does not necessarily reflect definite fibrosis synonymous with irreversible damage, emphasizing the importance of CMR re-evaluation during the recovery phase.[12] Therefore, a graded RTP approach with supervised stepwise-exercise programmes is implemented on all athletes after COVID-19 infection to ensure safe practice of their sports and close monitoring for new cardiopulmonary symptoms.

Conclusion

Identification of athletes with COVID-19 myocarditis is rare. Diligence is required to identify competitive athletes with features suggestive of myocarditis at the initial presentation and with the return to exercise. Universal testing for all does not yield benefits when compared with a stepwise approach. Cardiopulmonary symptoms in the setting of recent COVID-19 infection should prompt additional testing and often benefit from CMR in addition to the triad testing with ECG, echocardiography, and cardiac troponin measurement to further investigate clinical presentations of COVID-19-related myocarditis.

Lead author biography



Dr Omar M. Abdelfattah completed his research fellowship training at Cleveland Clinic Foundation with the Cardiovascular Medicine Department and is currently completing his Internal Medicine residency training at Morristown Medical Center and his subsequent Cardiovascular Medicine fellowship training at the University of Texas Medical Branch, Galveston, TX, USA. Dr Abdelfattah is an active fellow of the European Society of Cardiology

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Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including the images and associated text have been obtained from the patient in line with COPE guidance.

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