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CMR Tissue Characterization and Course of Acute SARS-CoV-2 type B.1.1.529-Associated Myocarditis in a Professional Soccer Player

CMR-Gewebecharakterisierung und Verlauf einer akuten SARS-CoV-2 Typ B.1.1.529 assoziierten Myokarditis bei einem Profifußballer

CASE REPORT

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Summary

- **We report the case** of a young professional soccer player who underwent cardiac MRI (CMR) for work-up of discrete intermittent chest pain and subtle ST segment elevations in the ECG after having been tested positive for SARS-CoV-2 type B.1.1.529 despite full vaccination including recent mRNA booster.
- **Troponin levels** were significantly increased and myocarditis was suspected. Comprehensive CMR including CINE and late gadolinium enhancement as well as multi-parametric T1/T2 mapping techniques revealed local hypokinesia and swelling of the posterolateral wall with non-ischemic late gadolinium enhancement and increased T2 relaxation time compatible with acute viral myocarditis. The patient was admitted to a cardiology ward for rhythm and troponin monitoring and was discharged after two days of uneventful rhythm monitoring and with decreased troponin levels.
- **Adhering to current recommendations** the patient was advised to abstain from moderate- to high-intensity sports and exercise for 3-6 months. After 6 months of exercise avoidance, follow-up ECG showed regression of prior ST segment elevations, and Holter ECG as well as a treadmill exercise stress test did not reveal any abnormalities. Follow-up CMR was performed before return-to-sports which revealed persisting myocardial fibrosis but complete regression of myocardial edemam and excluded ongoing inflammation.
- **This example** underscores the value of multi-parametric CMR tissue characterization for the work-up of suspected SARS-CoV-2 associated myocarditis, as well as for follow-up before return-to-sports.

KEY WORDS:
COVID-19, Late Gadolinium Enhancement
Cardiovascular MR, Myocardial Mapping

Zusammenfassung

- **In diesem Fallbericht** berichten wir die Diagnostik und den Krankheitsverlauf eines jungen Profifußballers, welcher sich mit diskreten intermittierenden Thoraxschmerzen und ST-Hebungen im 12-Kanal-EKG nach kürzlichem Nachweis einer COVID-19 Infektion (SARS-CoV-2 Typ B.1.1.529) trotz vollständigem mRNA-Impfschutz vorstellte.
- **Die Labordiagnostik** ergab eine deutliche Troponinerhöhung (hoch-sensitives Troponin I: 7804 ng/l; Oberer Referenzwert: 17.5 ng/l), sodass bei atypischen Beschwerden und fehlendem kardiovaskulären Risikoprofil die Verdachtsdiagnose einer Myokarditis gestellt wurde. Eine umfassende kardiale MRT-Diagnostik (CINE, late gadolinium enhancement, parametrisches T1/T2 Mapping) zeigte eine lokale Schwellung und Hypokinesie der posterolateralen linksventrikulären Wand mit nicht-ischämischem late gadolinium enhancement sowie verlängerter T2-Relaxationszeit vereinbar mit einer viralen Myokarditis.
- **Basierend auf diesen Befunden** und der deutlichen Troponinerhöhung erfolgte eine stationäre Aufnahme zur Rhythmuskontrolle und Verlaufskontrolle der Herzenzyme. Bei unauffälliger Rhythmusüberwachung und fallenden Troponinwerten konnte der Patient nach 2 Tagen entlassen werden. Entsprechend den aktuellen Empfehlungen wurde eine Sportpause für 3-6 Monate empfohlen. Nach 6-monatiger Sportpause zeigte sich eine Normalisierung des EKGs, ein Langzeit-EKG und ein Belastungs-EKG fielen ebenfalls unauffällig aus. Vor dem Wiederbeginn des Trainings erfolgte eine MRT-Verlaufskontrolle, welche eine persistierende myokardiale Fibrose bei vollständiger Regression des myokardialen Ödems zeigte, sodass kein Hinweis auf eine anhaltende Inflammation vorlag.
- **Anhand dieses Falls** demonstrieren wir die entscheidende Rolle der kardialen MRT zur Diagnosestellung und Verlaufsbeurteilung von Sportlern mit (vermuteter) Myokarditis im Rahmen einer SARS-CoV-2-Infektion.

SCHLÜSSELWÖRTER:
COVID-19, Late Gadolinium Enhancement
kardiale Magnetresonanztomographie, Mapping

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Introduction

A 19 year-old professional soccer player was referred for Cardiovascular magnetic resonance (CMR) work-up of suspected myocarditis seven days after testing positive for SARS-CoV-2 type B.1.1.529 by PCR in January 2022. Initial symptoms included headache, sore throat and nasal congestion despite a documented vaccination with two doses Vaxzevria (Astra Zeneca) in spring and summer 2021, as well as a mRNA booster dose Comirnaty (Pfizer-Biontech) in November 2021.

The patient reported new onset of discrete atypical chest pain resulting in additional 12-lead ECG and blood testing. ECG demonstrated normal ›

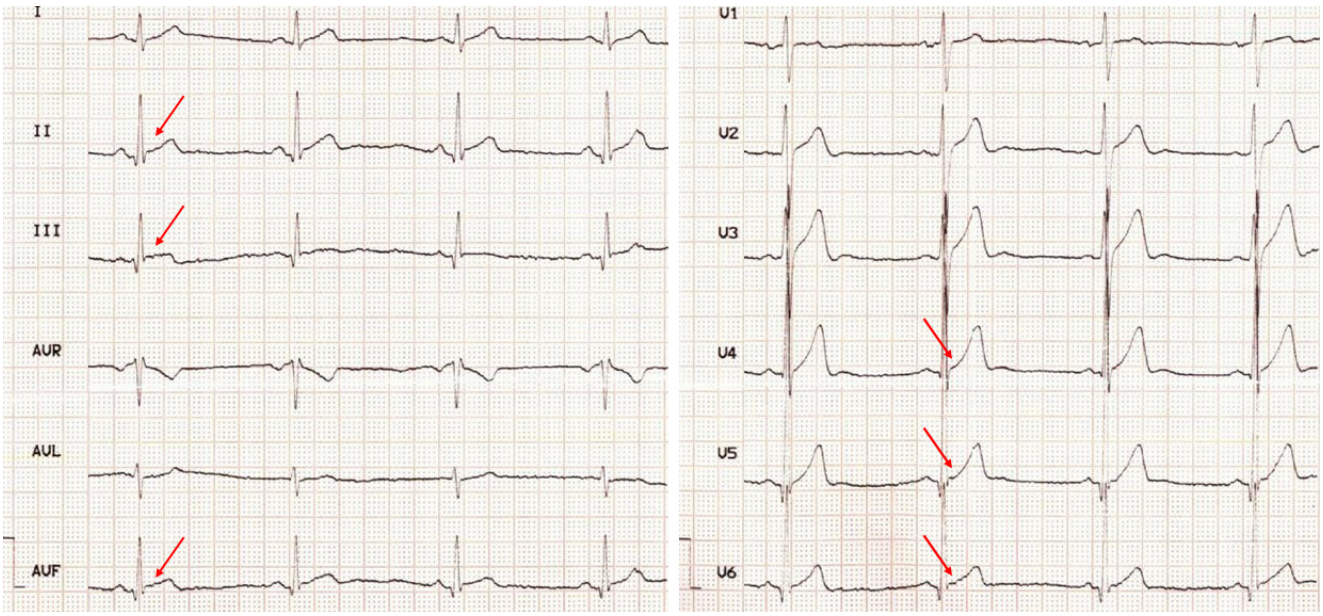

Figure 1

Resting 12-lead ECG during initial presentation demonstrating ST segment elevations in leads II, III, aVF and V3 to V6 (red arrows).

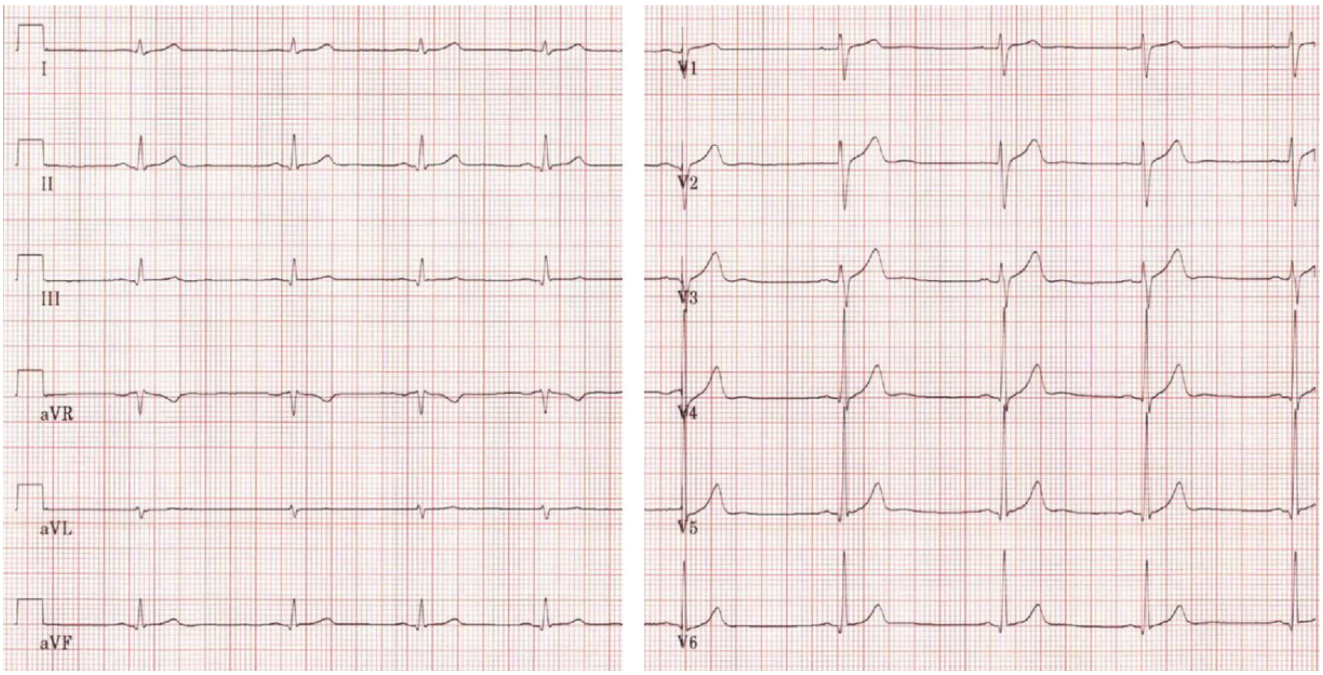

Figure 4

Normalization of the 12-lead resting ECG after 6 months.

sinus rhythm with subtle ST segment elevations in the posterolateral leads (figure 1). Blood work revealed a significant increase of troponin levels (high-sensitive troponin I of 7804 ng/l; ULN 17.5 ng/l).

Methods and Results

For further work-up of suspected COVID-19 myocarditis, as recommended by current expert consensus papers (1, 6), CMR was performed using a 1.5T Magnetom Aera. Cine-SSFPs revealed overall normal left ventricular ejection fraction (LV-EF) (62%) with local hypokinesia and swelling in the posterolateral wall (figure 2A, 2C, white arrows). Breath-hold T2 mapping was performed before gadolinium contrast and demonstrated myocardial T2 signal increase in the posterolateral wall matching the area of swelling and hypokinesia described above, as well as the location of late gadolinium enhancement (LGE) to become visible after contrast administration (figure 2B, 2D, white arrows). T1-mapping was obtained using a modified look-locker inversion-recovery sequence (MOLLI) during breath-hold before and after gadolinium. LGE images were acquired on average 5-10 minutes after contrast administration using segmented IR-FLASH constantly adjusting inversion time. LGE revealed typical non-ischemic enhancement of the posterolateral wall (figure 2B, 2D), which is frequently seen in viral myocarditis (4).

The myocardial extracellular volume (ECV) was calculated using the formula ECV = (1 – hematocrit) × (ΔR1myocardium/ΔR1blood). A visual color map of the calculated ECV is depicted

in figure 1F. Note that the area of enlarged ECV (black arrows) exactly matches the area of LGE as expected based on the known mechanism of LGE (figure 2D, 2F). Furthermore, the area of elevated T2 also matches the area of LGE (figure 2D, 2E), suggesting that relevant edema in the setting of myocarditis is only present in an area of irreversible cell damage (LGE and increased ECV), as known from ischemic heart disease (3).

Based on the results described above, the patient was admitted to a cardiology ward for rhythm and troponin monitoring. He stayed as an in-patient for two days and was discharged after arrhythmias were ruled out and troponin levels decreased. Medical treatment with ACE inhibitor was initiated and the patient was advised to abstain from moderate to high-intensity sports and exercise for 3-6 months according to European Society of Cardiology (ESC) guidelines and current consensus documents (1, 2, 5). During the first 3 months, the patient was advised to strictly abstain from any exercise training, afterwards he slowly resumed low-intensity exercise training. During this time, the patient was closely followed by his team doctor and outpatient cardiologist.

Follow-up CMR was performed at our center after 6 months demonstrating shrinking of the area of myocardial fibrosis (figure 3B, 3D, 3F) as previously described (4). Most importantly, however, despite the persistence of focal fibrosis, T2 was normalized at follow-up CMR suggesting complete regression of myocardial edema/inflammation (figure 3E). Follow-up ECG showed regression of prior ST segment elevations (figure 4). A 24h holter ECG and a treadmill exercise stress test (up to 325 watts with a maximum heart rate of 174 bpm) did not reveal any abnormalities. Based on these results the asymptomatic athlete was allowed to return-to-sports using graded exercise under regular medical surveillance (1, 5). ›

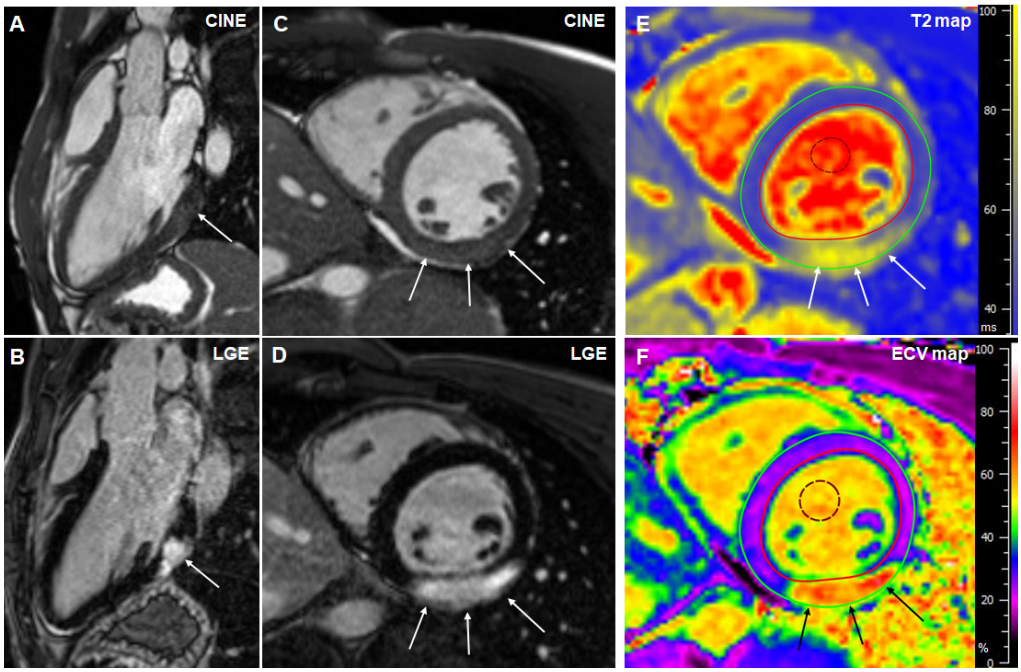

Figure 2

Detection of SARS-CoV2 associated myocarditis using multi-parametric cardiovascular MR. Panels A and C depict SSFP cine images before contrast administration. Note local hypokinesia and swelling in the posterolateral wall. Matching LGE images using segmented IR-GRE can be viewed in panels B and D. Typical non-ischemic contrast enhancement is marked by white arrows. Panels on the right side demonstrate results of T2 mapping (panel E) and calculated ECV (panel F, see text for details). Note that the area of increased T2 values (white arrows), as well as the area of enlarged extracellular volume (black arrows) match the area of hypokinesia, swelling and LGE.

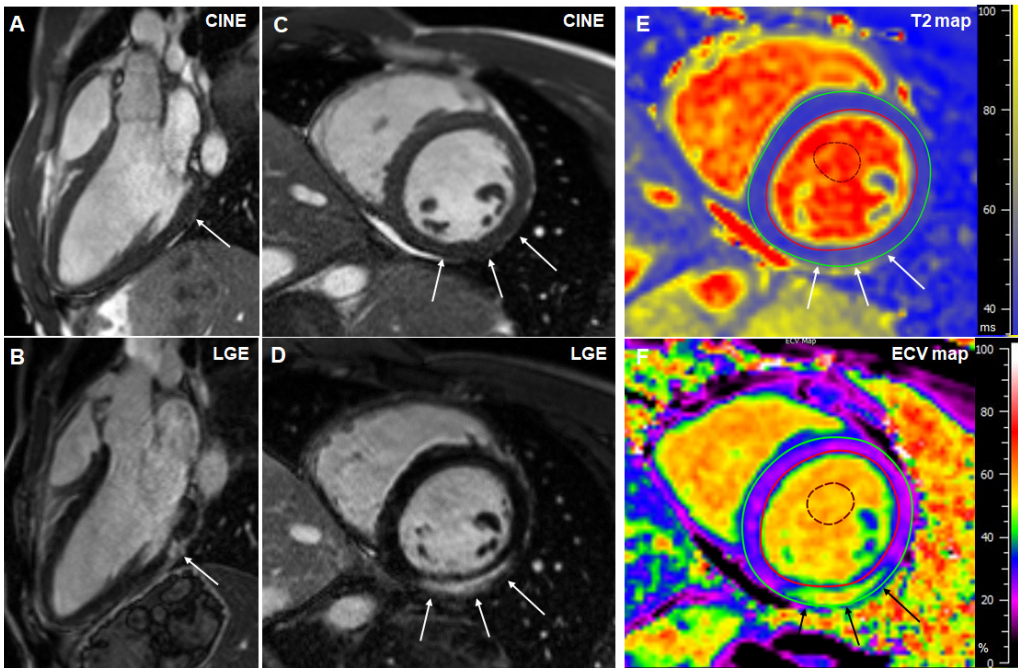

Figure 3

Follow-up CMR demonstrating regression of myocardial edema/inflammation before return-to-sport. Panels A and C before contrast administration demonstrate regression of hypokinesia and swelling (white arrows). Persistent but shrinked area of LGE is depicted in panels B and D (white arrows). Panel E demonstrate normalization of T2 mapping in the posterolateral wall suggesting complete regression of myocardial edema/inflammation. Note that the area of persistently enlarged extracellular volume (black arrows) at 6-months follow-up CMR still matches the area of LGE.

Limitation

The presented patient fulfilled the criteria for definite COVID-19 myocarditis according to the recent American College of Cardiology (ACC) expert consensus paper (1), however, in the absence of histopathological work-up concomitant SARS-CoV-2 infection and other causes of myocarditis (e.g. viral non-COVID-19 myocarditis or autoimmune myocarditis) cannot be excluded.

Discussion

This case nicely illustrates that SARS-CoV-2 type B.1.1.529 is capable of causing viral myocarditis with irreversible myocyte damage in a young competitive athlete despite complete vaccination and recent mRNA booster. Thus, a careful work-up of potential cardiac affection may be important in the setting of young SARS-CoV-2 patients even when presenting with mild clinical symptoms. Although CMR is only one component besides laboratory, resting and exercise ECG, as well as holter ECG in the work-up of athletes with myocarditis (5), this example underscores the value of CMR tissue characterization for evaluation of suspected viral myocarditis, as well as the importance of follow-up CMR assessment of myocardial edema/inflammation before return-to-sports.

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Conflict of Interest

The authors have no conflict of interest.

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