

Case Report

Heart and Lung Fibrosis in a Patient with COVID-19-Related Myocarditis

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Abstract

A COVID-19 patient, in whom pneumonia lesions were first detected by chest computed tomography, was further evaluated by cardiac magnetic resonance (CMR) due to a suspected myocarditis. Beyond heart alterations, CMR revealed peculiar features of affected pulmonary areas in T1 mapping sequences and showed a particular distribution of late gadolinium enhancement in the same regions. The noninvasive assessment of the cellular, fluid, or fibrotic content of lung lesions may provide key information about the underlying pathophysiological pathways in the search of a tailored medical therapy and ventilatory support for COVID-19 patients.

Keywords: Cardiac magnetic resonance, COVID-19, interstitial pneumonia, late enhancement, myocarditis, T1 mapping

INTRODUCTION

A patient affected by angina and COVID-19 interstitial pneumonia underwent cardiac magnetic resonance (CMR), which suggested peculiar heart and lung abnormalities. Few studies are actually available about tissue characterization of lung parenchyma.

CASE REPORT

A 55-year-old male patient was referred to our COVID-19-dedicated coronary care unit following typical chest pain, nonspecific electrocardiographic ST-T wave changes, and increased high-sensitive cardiac troponin T (hs-cTnT) levels. Nasopharyngeal swab tested positive for SARS-CoV-2; chest computed tomography revealed interstitial pneumonia. Blood tests showed lymphopenia (490/ μ l; normal range: 1000–4800/ μ l) and high hs-cTnT (peak: 4649 ng/L; normal value <14 ng/L). At arterial blood gas analysis (21% fraction of inspired oxygen), pO₂ was 75 mmHg; pCO₂ was 35 mmHg; SatO₂ was 97%; and PaO₂/FIO₂ was 357. Echocardiography showed left ventricular (LV) global hypokinesia, normal right ventricle function, and mild pericardial effusion. During the following days, there was a slight improvement in LV wall motion and a progressive decrease in hs-cTnT.

On day 3, CMR was performed using a 1.5 T scanner (Siemens Aera, Germany). Beyond heart disease, CMR highlighted lung parenchyma abnormalities. Specifically, T2 short tau inversion recovery images showed diffuse myocardial and lung edema [Figure 1a and b]. Native T1 mapping assessment within affected lung areas revealed higher values as compared with remote uninvolved ones [Figure 1c and d]; late gadolinium enhancement (LGE) was detected in the same pulmonary regions [Figure 1e and f]. Moreover, CMR showed intramyocardial high signal intensity in the left ventricle [Figure 1a and b], systolic function was reduced (ejection fraction: 40%) with global hypokinesia. On native T1 mapping sequences, modified Look–Locker inversion values were increased^[1] [Figure 1c and d], LGE was detected in the damaged areas with subendocardial sparing [Figure 1e and f].

A diagnosis of myocarditis in the course of SARS-CoV-2 infection was hypothesized. Interestingly, CMR provided additional information about the cellularity of lung involvement:

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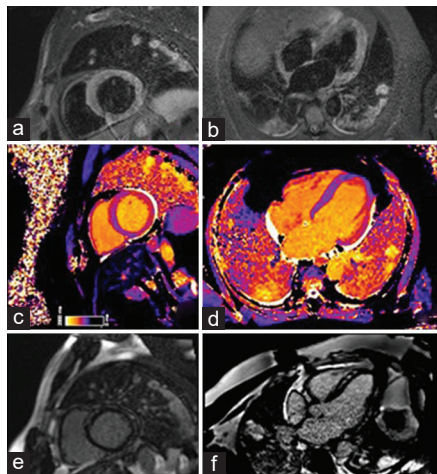


Figure 1: Myocardial and lung edema in T2 short tau inversion recovery images in short axis (a) and four chamber view (b); higher values of T1 mapping in the affected pulmonary areas in short axis (c) and four chamber view (d); late gadolinium enhancement in the same pulmonary regions (e and f). See text for details

in affected areas, interstitial expansion was well depicted, native T1 values were raised, hypothetically signifying ongoing inflammation and increased water and cellular content within the airway wall and/or lumen.

The patient received guideline-directed medical therapy for heart failure, corticosteroids, and enoxaparin for 10 consecutive days. He was discharged asymptomatic, without the need of oxygen support. A follow-up visit was scheduled after 1 month.

DISCUSSION

The onset of myocardial injury in the context of COVID-19 is well described in the literature.^[2,3] Its underlying causes include hypoxia, inflammation due to the cytokine storm, or, less frequently, a direct damage from the virus. Increased cTnT levels over the 99th percentile and electrocardiographic abnormalities are the most common signs of myocardial involvement. Transthoracic echocardiography generally contributes to make a differential diagnosis. Beyond ischemic heart disease, other cardiac conditions can frequently arise in the context of a viral infection and a systemic inflammatory response, such as myocarditis. CMR is a noninvasive and safe technique, frequently used as a first-line examination to diagnose myocarditis,^[4] especially in COVID-19 patients where an endomyocardial biopsy cannot be simply obtained. Endomyocardial biopsy is certainly recommended but usually performed only in specific critical situations. Coronary angiography is often not performed in the setting of clinically suspected myocarditis in COVID-19 patients. When evaluating CMR, findings incidental to the cardiac examination may be encountered, some of which may be clinically relevant.^[5] Notwithstanding,

while several data are available on the accuracy of CMR in the diagnosis of myocarditis, there are few studies in the literature concerning tissue characterization of lung lesions incidentally detected.^[6,7] These suggested a possible role for LGE evaluation of lungs to assess the degree of fibrosis and contributing to the differential diagnosis of respiratory diseases. Some limitations have been reported related to the need to take a longer breath-hold and to the inversion time of the lung parenchyma, which is similar to that of the lung blood pool. Even less is known about the role of native T1 mapping in the assessment of pulmonary lesions.

Following a histopathologically driven approach, since mapping imaging reflects the presence of diffuse fibrosis as well as edema at cardiac level, it might be reasonable a larger application of this technique in the assessment of pulmonary pathologies, to help clinicians chose a tailored medical therapy and the correct type of ventilatory support for each COVID-19 patient.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published, and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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