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Case Report

COVID-19 associated perimyocarditis

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ABSTRACT

Perimyocarditis is a well-known acute inflammation of the pericardium and the underlying myocardium. Most commonly perimyocarditis is of viral aetiology, specifically the coxsackie B virus. However, nowadays SARS-CoV-2 associated with COVID-19 infections has emerged as a potential rare cause of perimyocarditis. This case report will demonstrate a case of a young female with perimyocarditis as diagnosed by magnetic resonance imaging (MRI) accompanied by antigens indicating a past COVID-19 infection. Clinical status as well as Findings at MRI, echocardiography and lab results will be reviewed.

1. Introduction

"Perimyocarditis" refers to cases presenting as acute pericarditis with elevated troponin and LV ejection fraction less than 55%. The diagnostic approach to perimyocarditis is the same as for myocarditis [1]. Ultimately, perimyocarditis represents an acute inflammation of the pericardium and the underlying myocardium and is mostly of viral aetiology [2]. COVID-19 has been declared a global pandemic by the World Health Organization and is caused by the SARS-CoV-2 virus. Myocardial injury is relatively common in patients with COVID-19, accounting for 7%–23% of cases, and is associated with a higher rate of morbidity and mortality. Nowadays, more and more evidence is emerging of COVID-19 infections causing perimyocarditis [3]. This case report will demonstrate a case of a young female with perimyocarditis as diagnosed by magnetic resonance imaging (MRI) accompanied by antigens indicating a past COVID-19 infection.

2. Case presentation

A 15-year-old previously healthy female presented to the emergency department on the 5th day of a febrile episode, accompanied by mild cough with hemoptysis on one occasion, epigastric pain with vomiting on day 3 and 4, and diarrhea. In addition, she reported chest pain worsening in supine position, head and neck pain and general discomfort. She did not recall any respiratory symptoms in the period preceding the current episode, nor has she been in close contact with people having respiratory complaints. On physical examination she was alert and not acutely ill. She was tachypnoeic (RR 24/min) and had an increased heart rate (130/min) with a blood pressure of 90/50 mmHg and cold extremities with a normal central capillary refill time. Her oxygen saturation was 95–97% on 2 L of low flow oxygen. There were no audible cardiac murmurs or abnormal chest sounds. Abdominal examination showed no abnormalities except for some epigastric tenderness. There were no signs of arthritis or lymphadenopathy and no rash was present. There was a high index of suspicion towards a COVID-19 infection and patient was admitted to our hospital. However, SARS-CoV-2 presence could not be demonstrated using PCR in two separate nasopharyngeal swabs, a throat swab and a stool sample (all taken on day of admission = day 5). Serological testing was performed on a serum sample collected on day 6 (Wantai SARS-CoV-2 Ab ELISA, Beijing Wantai Biological Pharmacy Enterprise, Beijing, China). IgM was negative, but IgG was strongly positive, consistent with a past infection with SARS-CoV-2 in our patient.

Chest CT-scan without intravenous (IV) contrast showed very subtle ground-glass opacities in the most dorsobasal regions of the lungs (Fig. 1. Panel A-D) without any crazy paving, vascular dilatation, traction bronchiectasis or subpleural bands. There were also no consolidations. This finding is non-specific and could possibly be related to COVID-19 pneumonia, but could also be gravity-dependent atelectasis. Echocardiography (supplementary imaging content I) showed a

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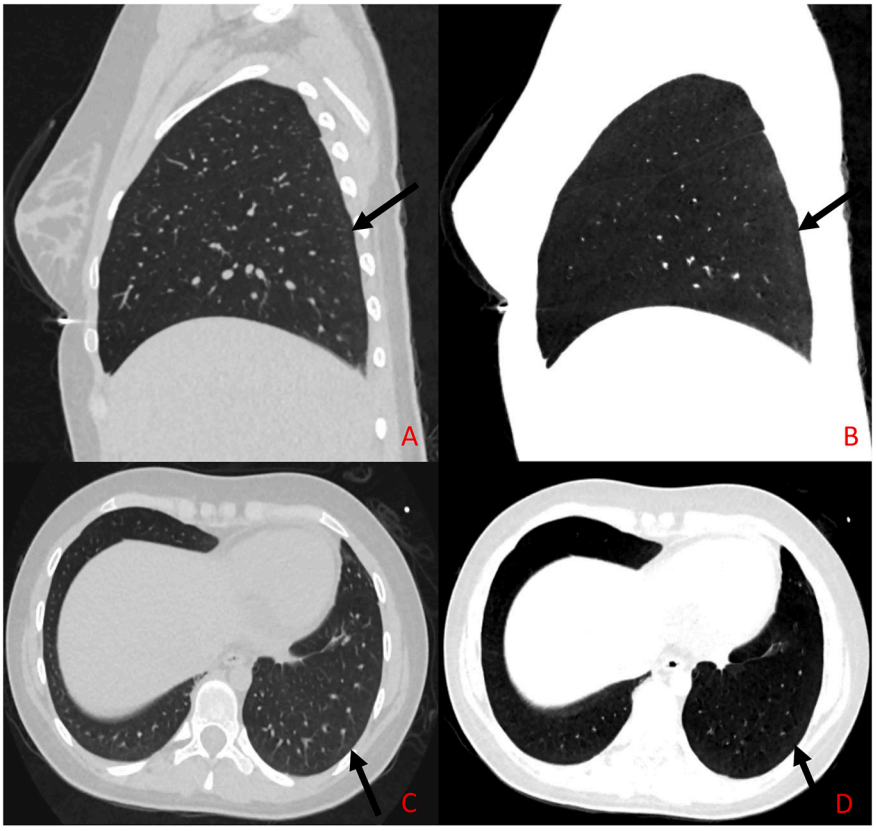

Fig. 1. Chest CT-scan without IV contrast. **(Panel A-D)** Very subtle ground ground-glass opacities in the most dorsobasal regions of the lungs (arrows).

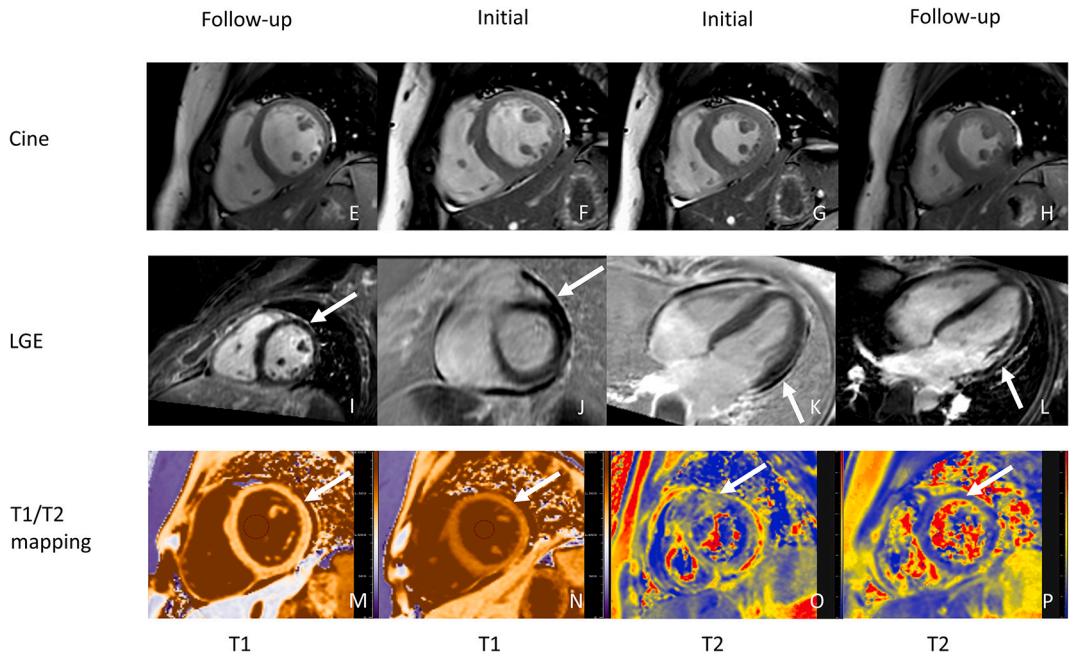

Fig. 2. Initial and follow-up diagnostic assessment with cardiac MRI which included cine, LGE and T1/T2 mapping. **(Panel F and G)** Initial cine showed global LV hypokinesia and decreased LV function with an EF of 38%. **(Panel E and H)** Follow-up cine showed improved LV function (EF 52%) with mild persisting global hypokinesia. **(Panel J and K)** Initial LGE enhancement of the visceral and parietal pericardium with slight pericardial effusion (arrows). **(Panel I and L)** At follow-up there is decreased LGE enhancement of the visceral and parietal pericardium with only minor persisting enhancement and pericardial effusion (arrows). **(Panel N and O)** Initial native T1 and T2 mapping showed strongly increased values (1500–1600 ms and 67 ms respectively) of the LV myocardium (arrows). **(Panel M and P)** Follow-up native T1 and T2 mapping values of the LV myocardium normalized (1200–1250 ms and 45 ms respectively) (arrows).

structurally normal heart with normal origins of the coronary arteries but with significant biventricular systolic dysfunction (LV Fractional shortening 15%, spontaneous contrast). Magnetic resonance imaging (MRI) of the heart was performed the following day, which included cine, native T1 and T2 mapping and delayed contrast enhancement, showing a non-dilated right and LV and normal right ventricle function. As expected from the echocardiography, cine showed global LV hypokinesia and decreased LV function with an ejection fraction (EF) of 38% (Fig. 2. Panel F and G). Additionally, there was LGE enhancement of the visceral and parietal pericardium with slight pericardial effusion in between (Fig. 2. Panel J and K). Native T1 and T2 mapping showed strongly increased values (1500–1600 ms and 67 ms respectively) of the LV myocardium (Fig. 2. Panel N and O), which has a broad differential diagnosis, including myocardial edema, diffuse fibrosis, inflammation and infiltrative diseases. However, in this clinical context accompanied by a rise in troponin (87 ng/L) this was compatible with non-ischemic CMP and suggestive of perimyocarditis. Repeated echocardiography (supplementary imaging content II) on day 23 showed improvement in cardiac function (LV fractional shortening 28–30%) This was confirmed by cardiac MRI after 3 months by an improved LV function with an EF of 52% and mild persisting global hypokinesia (Fig. 2. Panel E and H). Furthermore, MRI also showed decreased LGE enhancement of the visceral and parietal pericardium with only minor persisting enhancement and pericardial effusion (Fig. 2. Panel I and L). Native T1 and T2 mapping values of LV myocardium also normalized (1200–1250 ms and 45 ms respectively) (Fig. 2. Panel M and P), which in this clinical context was compatible with normalization of an earlier episode of diffuse myocardial edema due to perimyocarditis.

3. Discussion

The possibility of perimyocarditis being caused directly by COVID-19 infection itself, would require demonstrating SARS-CoV-2 presence in cardiac tissue by cardiac biopsy. This was however not conducted because of strongly improved clinical status and because of the always present risks of biopsy. Based on current knowledge [4], the serology and clinical status which was consistent with recent COVID-19 infection and the results of MRI indicating perimyocarditis, might indeed indicate a rare clinical entity and could possibly best be described as “COVID-19 associated perimyocarditis”. Quite striking in our case is the lack of severe respiratory symptoms accompanied by an almost normal chest computerized tomography.

4. Conclusion

This case highlights the importance of recognising COVID-19 infections with atypical clinical presentations such as perimyocarditis and may prove helpful in treating patients with this unique manifestation of clinical symptoms.

Declaration of Competing Interest

All Authors have nothing to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.mri.2021.08.012.

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