Cardiology in the Young

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The diagnosis of SARS-CoV-2-related myocarditis requires confirmation by endomyocardial biopsy or autopsy

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Letter to the Editor

Cite this article: Finsterer J and Stöllberger C (2025) The diagnosis of SARS-CoV-2-related myocarditis requires confirmation by endomyocardial biopsy or autopsy. *Cardiology in the Young* **35**: 439–440. doi: 10.1017/S1047951125000265

Received: 26 November 2024
Accepted: 23 December 2024

Keywords:
Myocarditis; SARS-CoV-2 infection; dilated cardiomyopathy; late gadolinium enhancement; heart failure

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We read with interest the article by Suarez et al. about a 10-year-old girl with idiopathic dilated cardiomyopathy (dCMP) and a history of heart failure (HF) that recurred after SARS-CoV-2 infection (SC2I).[1] Although the patient benefited from adequate HF therapy and remdesivir for SC2I, she eventually had to undergo heart transplantation (HTX) 7 months after SC2I.[1] The study is encouraging, but several points should be discussed.

The first point is that myocarditis was suspected on the basis of late gadolinium enhancement (LGE) on contrast-enhanced cardiac MRI. However, the patient did not undergo an endomyocardial biopsy (EMB) to determine whether the deterioration in dCMP was indeed due to SARS-CoV-2-related myocarditis. To determine whether the LGE is indeed inflammation and not edema or connective tissue, an EMB would have been mandatory. Complications of EMB are generally rare.

The second point is that the cause of the dilated CMP is not clear. Was it primary or secondary? Was the family history positive for dCMP? Was a genetic panel performed for mutations in genes associated with dCMP? Was the family history positive for a hereditary disease in general?

The third point is that the explanted heart was not tested for myocarditis, SARS-CoV-2 or the cause of dCMP. To determine whether dCMP is a primary or secondary disease, it would have been useful to perform an autopsy of the explanted heart. Was SARS-CoV-2 isolated from the explanted heart?

The fourth point is that LGE can also be due to myocardial fibrosis and does not necessarily represent edema or myocarditis.[2] Did the LGE persist after the myocarditis subsided? In the case of fibrosis, LGE usually persists.

The fifth point is that dCMP is often associated with neuromuscular disorders.[3] Was there evidence of muscle weakness or wasting in the index patient? Was creatine kinase elevated? dCMP may even be the first manifestation of, for example, dystrophinopathies.[4] Has the patient ever been examined by a neurologist? Did the patient develop progressive neurological abnormalities over time?

The sixth point is that LGE can also be associated with left ventricular noncompaction (LVNC).[5] Was there any evidence of LVNC on echocardiography or cardiac MRI? Was the LGE due to myocarditis, fibrosis or LVNC?

The seventh point is that the time course is unclear.[1] In the case description, the first echocardiogram was performed in March 2021 and another one 9 months later. However, the discussion mentions that the HTX was performed seven months after the SC2I. This discrepancy should be clarified.

Since the patient had received an implantable cardioverter defibrillator (ICD) prior to HTX, it would be interesting to know if the ICD was ever discharged.

To summarize, this interesting study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and support the message of the study. All unresolved issues need to be addressed before readers can uncritically accept the study's conclusions. The diagnosis of SARS-CoV-2-related myocarditis requires confirmation by EMB or autopsy.

Availability of data and material. All data are available from the corresponding author.

Acknowledgements. None.

Author contributions. JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. CS, FS, AF: contributed to literature search, discussion, correction, and final approval.

Financial support. None received.

Completing interests. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Ethical approval. Not applicable.

Consent to participation. Not applicable.

Consent for publication. Not applicable.

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https://doi.org/10.1017/S1047951125000265 Published online by Cambridge University Press