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Article:
Gorecka, MM, Thirunavukarasu, S, Levelt, E et al. (2021) Progressive myocardial dysfunction following COVID-19. BMJ Case Reports, 14 (11). e246291. ISSN: 1757-790X

https://doi.org/10.1136/bcr-2021-246291

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BMJ Case Reports

TITLE OF CASE

Progressive myocardial dysfunction following COVID-19

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Word count: 407

DESCRIPTION

A 66-year-old male with background of type 2 diabetes mellitus, hypertension and functionally non-significant coronary artery disease (CT coronary angiogram 2 years prior demonstrated moderate ostial atheroma in the first diagonal branch and in the dominant right coronary artery, however a subsequent stress echocardiogram did not reveal any inducible ischaemia) presented to the hospital with non-productive cough and pyrexia, and was subsequently confirmed to have COVID-19. He denied dyspnoea and chest pain. Laboratory testing revealed elevated Troponin T (peak 296ng/L; ULN<57ng/L), elevated D-dimer (peak 504 ng/ml; ULN<230ng/ml) and acute kidney injury stage III. He was treated conservatively with intravenous fluid replacement and made an uneventful recovery.

He underwent cardiovascular magnetic resonance (CMR) imaging as part of a UK, multi-centre, Urgent Public Health research study (ISRCTN 58667920).[1] CMR revealed mild impairment in left ventricular ejection fraction (LVEF 45%).Stress perfusion CMR revealed no inducible ischaemia, whereas late gadolinium enhancement (LGE) images showed patchy mid-wall hyper-enhancement consistent with COVID-19 myocarditis (Figure 1A). T1-mapping confirmed myocardial injury in a non-ischaemic pattern (Figure 1B) and global myocardial T2 was also elevated consistent with acute inflammation (Figure 1C).

At 6-months, the patient felt well. Repeat investigations however revealed persistent elevation of Troponin T (597ng/L) and BNP (494ng/L; ULN<331ng/L). Repeat CMR showed significant deterioration in LVEF to 28%, persisting mid-wall hyper-enhancement (Figure 2A) and elevated

myocardial T1 (Figure 2B). Myocardial T2 had normalised (Figure 2C) and stress perfusion CMR was again unremarkable.

Despite the absence of cardiovascular symptoms and an uneventful recovery from COVID-19, CMR demonstrated progressive myocardial dysfunction, which warranted introduction of heart failure therapies and immunosuppression.[2] The patient was treated with a beta-blocker and an angiotensin-II receptor blocker in the community. The ultimate goal was to introduce an angiotensin receptor neprilysin inhibitor, mineralocorticoid receptor antagonist and a sodium-glucose co-transporter-2 alongside immunosuppression, however the patient has since failed to attend his hospital appointments.

Although the differential diagnosis in such a case is quite broad, in view of the functionally non-significant (non-obstructive) coronary artery disease, lack of any specific cardiac symptoms and an elevated, but relatively static Troponin T rise, COVID-19 associated myocardial injury was felt to be the most likely clinical diagnosis (biopsy data were not available). As the patient failed to attend further hospital appointments, the final case disposition is uncertain.

We believe this case highlights the importance of follow up imaging in cases of COVID-19 myocarditis, as ongoing and progressive myocardial injury may occur in the absence of any obvious symptoms.

LEARNING POINTS/TAKE HOME MESSAGES (2-3 bullet points)

- COVID-19 can cause progressive myocardial dysfunction
- Follow up imaging is required to establish the diagnosis and to guide appropriate therapies

REFERENCES

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2. Nicol M, Cacoub L, Baudet M, et al. Delayed acute myocarditis and COVID-19-related multisystem inflammatory syndrome. *ESC Heart Fail.* 2020;7(6):4371-6.

FIGURE CAPTIONS

Figure 1. Baseline CMR scan. Panel A - Short-axis late gadolinium enhancement (LGE) image demonstrates patchy mid-wall hyper-enhancement consistent with COVID-19 myocarditis; Panel B - T1-mapping image shows myocardial injury in a non-ischaemic pattern; Panel C – image of a T2 map demonstrates global elevation of T2 consistent with acute inflammation.

Figure 2. Follow up scan at 6-months. Panel A – LGE image shows persisting mid-wall hyper-enhancement; Panel B – T1-mapping image demonstrates elevated myocardial T1; Panel C – T2

map shows normalisation of T2.

PATIENT'S PERSPECTIVE

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Date: 27.10.2021