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Cardiac abnormalities in Long COVID 1-year post-SARS-CoV-2 infection

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ABSTRACT

Background Long COVID is associated with multiple symptoms and impairment in multiple organs. Cross-sectional studies have reported cardiac impairment to varying degrees by varying methodologies. Using cardiac MR (CMR), we investigated a 12-month trajectory of abnormalities in Long COVID.

Objectives To investigate cardiac abnormalities 1-year post-SARS-CoV-2 infection.

Methods 534 individuals with Long COVID underwent CMR (T1/T2 mapping, cardiac mass, volumes, function and strain) and multiorgan MRI at 6 months (IQR 4.3–7.3) since first post-COVID-19 symptoms. 330 were rescanned at 12.6 (IQR 11.4–14.2) months if abnormal baseline findings were reported. Symptoms, questionnaires and blood samples were collected at both time points. CMR abnormalities were defined as ≥1 of low left or right ventricular ejection fraction (LVEF), high left or right ventricular end diastolic volume, low 3D left ventricular global longitudinal strain (GLS), or elevated native T1 in ≥3 cardiac segments. Significant change over time was reported by comparison with 92 healthy controls.

Results Technical success of multiorgan and CMR assessment in non-acute settings was 99.1% and 99.6% at baseline, and 98.3% and 98.8% at follow-up. Of individuals with Long COVID, 102/534 (19%) had CMR abnormalities at baseline; 71/102 had complete paired data at 12 months. Of those, 58% presented with ongoing CMR abnormalities at 12 months. High sensitivity cardiac troponin I and B-type natriuretic peptide were not predictive of CMR findings, symptoms or clinical outcomes. At baseline, low LVEF was associated with persistent CMR abnormality, abnormal GLS associated with low quality of life and abnormal T1 in at least three segments was associated with better clinical outcomes at 12 months.

Conclusion CMR abnormalities (left entricular or right ventricular dysfunction/dilatation and/or abnormal T1mapping), occurred in one in five individuals with Long COVID at 6 months, persisting in over half of those at 12 months. Cardiac-related blood biomarkers could not identify CMR abnormalities in Long COVID.

Trial registration number NCT04369807.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Acute COVID-19 can be associated with various cardiovascular complications, including myocarditis, ventricular disfunction or acute coronary syndrome, however, the evolution of cardiac impairment, especially in non-hospitalised patients has not been fully investigated.

WHAT THIS STUDY ADDS

⇒ We specify the nature of cardiac abnormalities in Long COVID, linked to clinical characteristics at 1 year. Within a multiorgan context, we provide a holistic view of Long COVID assessment, developed in a community cohort of mainly non-hospitalised individuals with varying severity of symptoms.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Comprehensive cardiac MRI assessment may guide clinical decision making and improve healthcare resource utilisation. Evidence of cardiac involvement could inform follow-up assessment and identification of Long COVID subtypes in research and practice, as well as interventional trials to evaluate cost-effective therapies.

INTRODUCTION

Cardiovascular disease is linked to COVID-19 severity and mortality since the first reports from Wuhan in late 2019.[1–3] However, associations between Long COVID symptoms and cardiac impairment are unclear, and the subtypes more likely to recover have not been identified.

In a large post-COVID-19 assessment service in the UK, almost half of individuals where cardiac MR (CMR) scans were performed had evidence of mild myocarditis[4] and in a smaller study, symptom improvement at 6 months was neither correlated with improvement on CMR imaging nor lung parenchymal recovery.[5] A systematic review of CMR findings post-COVID-19 identified myocarditis as the most prevalent diagnosis (14%),[6] though

not all classical features are evident on biopsy,[7 8] and T1 abnormalities and oedema on T2 as the most common findings, and occasional late gadolinium enhancement (LGE).[8] These findings may be present even in absence of elevated cardiac blood biomarkers (eg, troponin or NT-pro-BNP, natriuretic peptide pro B-type natriuretic peptide).[6 9 10] Pericardial effusion and reduced LV and RV function have been occasionally reported, but pericarditis is rare. Nevertheless, to date there is no clear definition of cardiac change post-COVID-19 and cardiac abnormalities in Long COVID at baseline and over time are ill defined in the community setting.

Although echocardiography is often the first choice for assessment of cardiac function, CMR is the gold-standard assessment, ensuring a more accurate assessment of cardiac structure and function. We; therefore, conducted a prospective, longitudinal 1-year study using CMR alongside multiorgan MRI assessment, in the largest Long COVID community cohort available to date, to investigate: (1) The evolution of cardiac abnormalities over 1 year after SARS-CoV-2 infection in a multiorgan context; (2) the prevalence and severity of cardiac abnormalities in the non-hospitalised versus the hospitalised population and (3) the associations to patient outcomes that could be used to guide clinical pathway design and identification of at risk individuals.

METHODS

Population and study design

The COVERSCAN study (NCT04369807) is a prospective study of organ function using quantitative MRI in individuals recovering from SARS-CoV-2 infection with persistent COVID-19 symptoms in a community setting. Individuals were recruited via advertisement, including in Long COVID support groups and hospital referral (online supplemental methods 1), and invited to undergo CoverScan (Perspectum, Oxford, UK), a multiparametric MRI assessment of lungs, heart, liver, pancreas, kidneys and spleen. All imaging assessments were performed at Perspectum (Oxford), Mayo Clinic (London) and Chenies Mews Imaging Centre (London), between April 2020 and October 2021 (figure 1). Healthy controls were recruited within the same period,

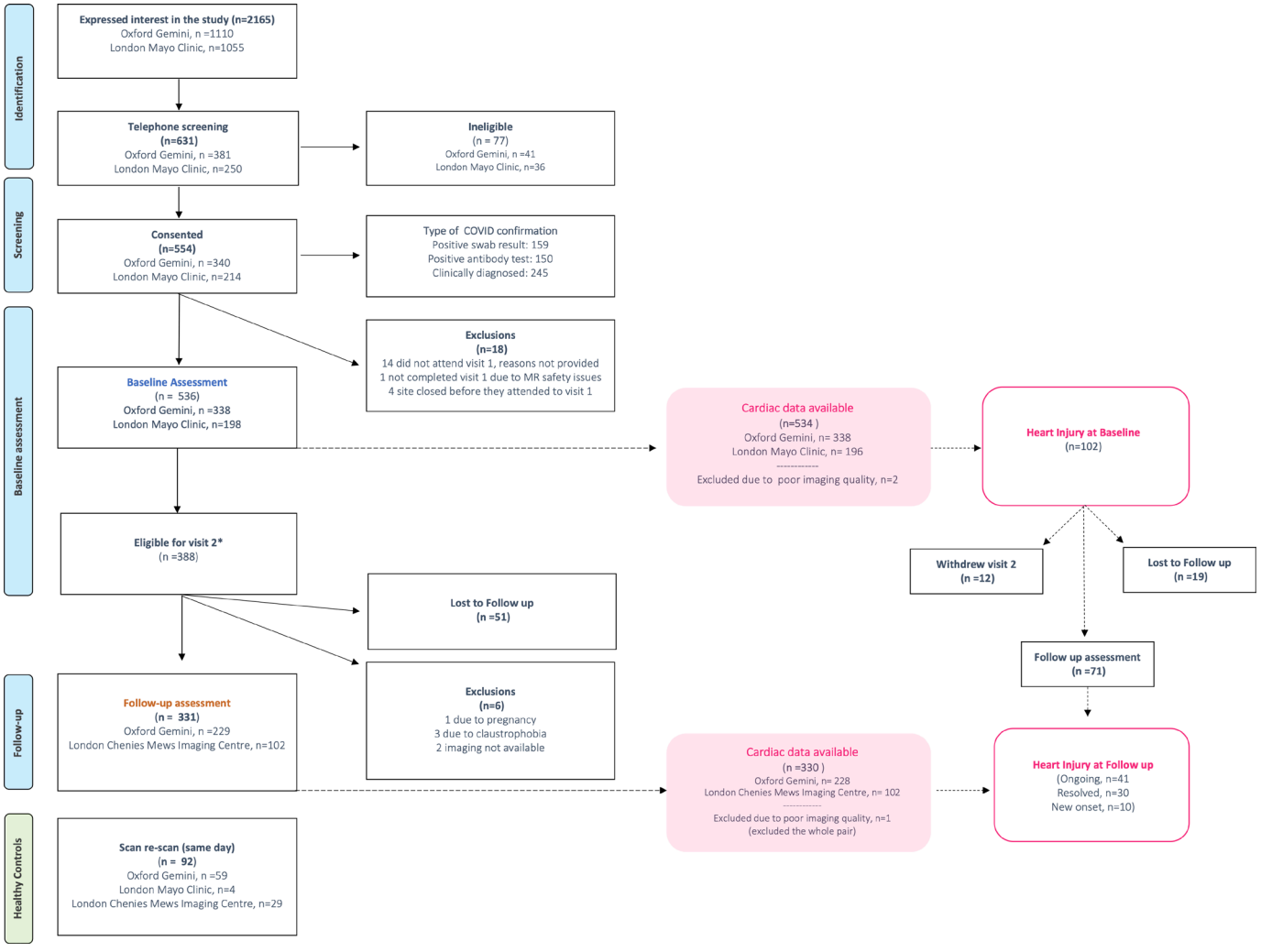

Figure 1 Study population for cardiac complications of long COVID. *Individuals were eligible for follow up when MRI abnormality or abnormal bloods in any organ were found at baseline.

based on self-reporting medical history, and scanned twice on the same date to derive reference ranges and assess repeatability. COVID-19 was classified by either laboratory-confirmed SARS-CoV-2 infection (159 tested SARS-CoV-2-positive by oropharyngeal/nasopharyngeal swab for reverse-transcriptase PCR; 150 individuals with positive antibodies) or strong clinical suspicion of SARS-CoV-2 infection with typical symptoms/signs confirmed by 2 clinicians (245 individuals). Exclusion criteria were symptoms of active respiratory viral infection (temperature >37.8°C or ≥3 episodes of coughing in 24 hours), hospital discharge in the last 7 days and contraindications to MRI, including implanted pacemakers, defibrillators, other metallic implanted devices and claustrophobia. Participants gave written informed consent. Those with organ abnormality at baseline MRI scan (in ≥1 of the following organs: lungs, heart, liver, pancreas, spleen, kidneys) or blood tests were invited back for 6-month follow-up, corresponding to 1-year postinfection. Incidental findings classified as benign and/or not requiring follow-up by an experienced radiologist were not invited for follow-up.

Symptoms, quality of life and function

Presence and severity of symptoms were assessed by self-report and validated questionnaires: EQ-5D-5L (EuroQoL-5 dimension-5 level; utility score and quality of life related to usual activities), and Dyspnoea-12 at baseline and follow-up, when Left Ventricular Dysfunction Questionnaire (LVD-36) was also conducted (online supplemental methods 2). For self-reported symptoms at baseline, participants were asked to report only new symptoms arising since the COVID infection; at follow-up, they were asked to report symptoms since baseline. Time off work due to Long COVID was recorded as total number of days at follow-up.

Blood investigations

Two blood samples were taken at both timepoints, on the same day as the MRI scan: one immediately sent for analysis, the other fractionated and frozen for later analysis (online supplemental methods 3).

Multiorgan imaging

Participants were scanned at Perspectum Gemini (Oxford: n=338; MAGNETOM Aera 1.5T scanner) and Mayo Clinic (London: n=198; MAGNETOM Vida 3T) (both scanners: Siemens Healthcare, Erlangen, Germany), at baseline and follow-up with multiorgan, multiparametric MRI assessment (total ~40 min duration). All imaging methods were deployed in standard clinical MRI scanners using slightly modified versions of previously published methods[11 12] and using short (<14 s) breath-holds except for lung imaging (online supplemental methods 4 and 5).

After each visit, participants and if requested their primary care physicians also, received a clinical summary and a report informing on the MRI data, where quantitative metrics were referenced against the healthy control population, and one on the blood biomarker data.

Reference ranges and repeatability coefficients

In parallel, 92 sex-matched and age-matched healthy individuals (online supplemental methods 6 tables S1,S2) were recruited and scanned twice on the same day, to derive a control group. Reference ranges using the healthy control population were calculated for each metric by computing 2.5% and 97.5% percentiles using bootstrapping (100 000 permutations), except pancreas proton-density fat fraction (PDFF), where the 95% percentile was for the upper limit, and liver cT1 and PDFF, where we used established thresholds.[13] Reference ranges for organ length and volume required larger sample size for sex and height stratification, so we used a sample of 1836 individuals from UK Biobank without self-reported diabetes or hypertension. To evaluate measurement repeatability, two separate scans were performed in healthy controls (1.5T, n=59; 3T, n=33) on the same day. After first scan, the participant had a 10 min break out of the scanner before a second identical scan. Technical success was assessed by quality-assured measures for each variable, and overall, in report delivery for each patient (online supplemental table S1).

Definition of cardiac and multiorgan abnormality

CMR abnormalities were defined by consensus among expert cardiologists with experience of Long COVID patients and following literature review of common cardiac findings post-COVID-19 as: ≥1 of the following outside reference range left or right ventricular ejection fraction (LVEF or RVEF) or left or right ventricular end diastolic volume, global longitudinal strain (GLS) (abnormal will be referred as low, in absolute values) or ≥3 quantitative T1 mapping segments. Two cardiologists independently reviewed all CMR findings ahead of statistical analysis in this work. Multiorgan impairment was defined as ≥2 measurements outside reference ranges in a further organ (excluding elevated liver or kidney volume)[11] (further details in online supplemental methods 5 table S1).

Statistical analysis

We used R software V.4.0.4 and p values <0.05 defined statistical significance. Normality was assessed using Shapiro test. To describe parametric and non-parametric variables, we used mean (SD) and median (IQR), respectively. For categorical variables, we reported frequencies (percentage). For groupwise comparisons of continuous parametric and non-parametric, and categorical variables, t-test, Wilcoxon rank sum and Fisher's exact tests, respectively, were used, without correction for multiple testing as analyses were exploratory. Baseline and follow-up metrics were assessed using reference ranges calculated in healthy controls. Repeatability coefficients (RC) for each CMR metric in healthy controls

determined the smallest detectable difference between repeated measures.[14] For cases with CMR abnormalities at baseline, findings were considered: (A) ongoing when CMR metrics were outside reference ranges at follow-up, independently from RC, (B) resolved when change was >RC and CMR metrics were within reference ranges at follow-up. In cases without baseline CMR abnormalities, participants were considered: (A) never affected when CMR was within reference ranges at follow-up, independently from RC, (B) with new onset findings when change was >RC and CMR metrics were outside reference ranges at follow-up. Associations with all exposures were by logistic and linear regression for categoric and continuous dependent variables, respectively. Variables with a significance >0.05 in the univariable models were included in the multivariable analyses. Goodness of fit was performed comparing the actual versus predicted values for an outside validation cohort and doing a visual inspection of residuals of the model. Multivariable stepwise regressions were performed to assess which cardiac metrics at baseline, as continuous variables, were most predictive of poor quality of life, reduced symptom severity and ongoing CMR findings between baseline and follow-up to inform future clinical care.

Community-delivered diagnostic assessment

Technical success of CMR was determined by reporting quality-assured measures for each variable reported here, and of multiorgan MRI overall, in delivering a report for each patient. For cardiac T1 and T2, technical success was based on value availability for least three AHA segments. Clinical utility of MRI metrics was not directly assessed during the study, as they were used for research only.

RESULTS

Characteristics of cardiac abnormalities at 6 months

Of 536 individuals enrolled at baseline, 534 had available CMR data at a median 6 (IQR (4.33–7.26)) months after first COVID-19 symptoms (table 1, figure 1). Of those, 6 (1%) presented with raised cardiac blood biomarkers (high hs-cTnI, n=4 and high NT-proBNP, n=2), but only 1/6 had abnormal CMR with both low LVEF and RVEF at 6 months and acute COVID-19 hospitalisation. However, an additional group of 101 individuals (19%) presented with abnormalities on CMR and normal cardiac blood biomarkers (figure 2, online supplemental tables S2–S4).

Demographic differences between groups are presented in table 1, the 102 individuals with CMR findings at 6 months were mostly characterised by reduced LVEF (21/102, 21%) or RVEF (21/102, 21%), low GLS (21/102, 21%) or T1 findings (46/102, 45%) (T1 topographical abnormalities are shown in S5) (table 2). Multiorgan involvement (≥3 organs) was more common in those with CMR abnormalities compared with those without (14% vs 5.7%, p=0.005) (table 1).

In exploratory analyses, no blood investigations were predictive of CMR abnormalities at 6 months and a full table with prevalence of blood abnormalities and group can be found in online supplemental table S4. At 6 months, 62/102 (62%) individuals with CMR abnormalities presented with severe Long COVID, based on questionnaires (Supplementary methods). Forty-three (43%) and 44 (44%) individuals had severe and moderate symptoms, respectively; most commonly fatigue (100%), shortness of breath (88%), headache (83%), chest pain (81%) and cough (80%). Symptom prevalence was similar regardless of the CMR abnormalities category (table 1).

Follow-up CMR data were available in 330/331 individuals at a median 12.7 (IQR: 11.6–14.3) months since first symptoms; these individuals were all symptomatic at baseline. At 12 months, 51/330 (15%) presented with CMR abnormalities. Of the 102 individuals with CMR abnormalities at 6 months, 71 had follow-up data available (figure 1).

Resolved CMR abnormalities

At 12 months CMR abnormalities had resolved in 30/71 (42%). At 6 months, CMR in this group showed elevation in T1 (57%), low GLS (21%) and reduced LVEF (20%), with full resolution by 1 year (table 2). By 12 months, 53% had fully resolved multiorgan impairment, and only 1 individual had impairment in ≥3 organs (table 3). Alongside resolution of CMR findings, elevation of NT-proBNP observed at baseline in a single patient of 41 years had resolved by 12 months. No blood investigations were predictive of cardiac recovery (online supplemental table S4).

Of these individuals, 13/30 (43%) presented with severe Long COVID at baseline, with less symptom burden at follow-up in all but 1 (median 10 and 4 symptoms at 6 and 12 months, respectively) and 5/30 (17%) fully resolving their symptoms (table 1). CMR abnormalities affected quality of life 1 year after infection (mean LVD-36 score 36%) and 13/30 (43%) still presented moderate to severe problems with usual activities. Of 30, 9 (30%) had required acute COVID-19 hospitalisation, and 3 (10%) were hospitalised between 6 and 12 months postinfection.

Ongoing CMR abnormalities

At 12 months, abnormalities by CMR persisted in 58% (41/71) of individuals. At 6 months, reduced LVEF (p=0.04) and low GLS (p=0.02) were more common, and at 12 months, LVEF, GLS and RVEF were consistently lower (p=0.05, p=0.04 and p=0.04, respectively) (table 4). One individual presented with abnormal T2 imaging at 12 months. Multiorgan impairment was more common in those individuals not resolving their CMR abnormalities (≥2 organs impaired in 49% with ongoing CMR abnormalities, p=0.002) (table 1).

Symptoms and impact on usual activities as well as quality of life were similar between the ongoing and resolved CMR abnormalities groups. Of 41, 16 (39%) individuals with ongoing CMR abnormalities still presented with severe Long COVID; however, most of

Table 1 Demographics and characteristics

	6 months							12 months		
	Overall cohort n=534	CMR abnormalities n=102	No CMR abnormalities n=424	P value	CMR abnormalities and hospitalised n=19	CMR abnormalities and non-hospitalised n=83	P value	Ongoing CMR abnormalities n=41	Resolved cardiac function n=30	P value
Demographics										
Age (median (IQR) or mean (SD))	44 (38–52)	43 (37–51)	44 (38–52)	0.41	45 (41–53)	41 (35–51)	0.2	45 (13)	48 (12)	0.22
Sex (% male)	147 (28%)	42 (41%)	103 (24%)	**0.001**	11 (58%)	31 (37%)	0.1	19 (46%)	11 (37%)	0.41
BMI kg/m^2 (median (IQR))	25.5 (22.6– 29.3)	26.3 (23.1–29.0)	25.3 (22.6–29.4)	0.28	28.0 (23.4–32.0)	26.0 (23.0–28.4)	0.31	25.6 (23.4–28.4)	27.4 (24.5–33.8)	0.09
BMI ≥25 to <30 kg/m^2 (%)	172 (32%)	38 (37%)	131 (31%)	0.22	7 (37%)	31 (37%)	0.97	15 (37%)	11 (37%)	0.99
BMI ≥30 kg/m^2 (%)	119 (22%)	23 (23%)	96 (23%)	0.98	6 (32%)	17 (20%)	0.36	8 (20%)	10 (33%)	0.19
Hypertension (%)	44 (8.2%)	12 (12%)	32 (7.5%)	0.17	2 (11%)	10 (12%)	1	5 (12%)	6 (20%)	0.51
Diabetes (%)	10 (1.9%)	3 (2.9%)	7 (1.7%)	0.42	0 (0%)	3 (3.6%)	1	2 (4.9%)	0 (0%)	0.51
Asthma (%)	101 (19%)	22 (22%)	78 (18%)	0.46	4 (21%)	18 (22%)	1	9 (100%)	5 (100%)	1
Previous heart disease	9 (1.7%)	2 (2%)	7 (1.7%)	0.82	0 (0%)	2 (2.4%)	0.49	1 (2.4%)	0 (0%)	0.38
Ethnicity (%): white	475 (89%)	88 (86%)	382 (90%)	0.57	14 (74%)	74 (89%)	0.16	38 (93%)	23 (77%)	**0.02**
Asian	24 (4.5%)	7 (6.9%)	16 (3.8%)		3 (16%)	4 (4.8%)		1 (2.4%)	6 (20%)	
Black	13 (2.4%)	3 (2.9%)	9 (2.1%)		1 (5.3%)	2 (2.4%)		0 (0%)	1 (3.3%)	
Mix	21 (3.9%)	4 (3.9%)	16 (3.8%)	0.15	1 (5.3%)	3 (3.6%)	0.68	2 (4.9%)	0 (0%)	0.9
Other	1 (0.2%)	0 (0%)	1 (0.2%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
Smoking status (%): current	348 (65%)	5 (4.9%)	7 (1.7%)		0 (0%)	5 (6.0%)		2 (4.9%)	1 (3.3%)	
Never	13 (2.4%)	66 (65%)	275 (65%)		14 (74%)	52 (63%)		31 (76%)	22 (73%)	
Past	172 (32%)	31 (30%)	141 (33%)		5 (26%)	26 (31%)		8 (20%)	7 (23%)	
Time from first symptom to scan (median (IQR))	182 (132–221)	162 (118–213)	183 (140–223)	**0.05**	141 (77)	173 (72)	0.12	359 (339–394)	380 (323–422)	0.27
Severity										
Hospitalisation at the acute stage (%)	72 (14%)	19 (19%)	51 (12%)	0.08	100 (100%)	0 (0%)	–	7 (17%)	9 (30%)	0.2
Long COVID severity from questionnaires (%):										
Mild	175 (34%)	38 (38%)	135 (33%)	0.38	11 (58%)	27 (33%)	**0.047**	20 (54%)	13 (45%)	0.46
Severe	338 (66%)	62 (62%)	270 (67%)		8 (42%)	54 (67%)		17 (46%)	16 (55%)	
Self-reported symptom severity (%): critical	11 (2.1%)	1 (1.0%)	9 (2.1%)	0.23	1 (5.3%)	0 (0%)	**0.01**	16 (39%)	9 (30%)	0.6
Mild	42 (7.9%)	13 (13%)	29 (6.9%)		0 (0%)	13 (16%)		11 (27%)	7 (23%)	
Moderate	232 (44%)	44 (44%)	186 (44%)		5 (26%)	39 (48%)		14 (34%)	13 (43%)	
Severe	246 (46%)	43 (43%)	198 (47%)		13 (68%)	30 (37%)		0 (0%)	1 (3.3%)	
EQ-5D-5L quality of life (Utility score) (median (IQR))	0.67 (0.49–0.77)	0.66 (0.43–0.77)	0.68 (0.50–0.77)	0.66	0.74 (0.57–0.81)	0.65 (0.42–0.77)	0.08	0.72 (0.55–0.81)	0.71 (0.33–0.84)	0.89

Continued

Table 1 Continued

	6 months							12 months		
	Overall cohort n=534	CMR abnormalities n=102	No CMR abnormalities n=424	P value	CMR abnormalities and hospitalised n=19	CMR abnormalities and non-hospitalised n=83	P value	Ongoing CMR abnormalities n=41	Resolved cardiac function n=30	P value
Dyspnoea 12 score (median (IQR))	6 (2–14)	6 (2–12)	7 (2–14)	0.43	4 (2–8)	6 (2–13)	0.52	4 (2–11)	4 (1–11)	0.91
LVD-36 (average, SD)	–	–	–	–	–	–	–	39% (31.3)	36% (28.3)	0.67
Time off work (median (IQR))	56 (14–180)	60 (21–180)	56 (14–180)	0.55	NA	124.8 (129.6)	–	96 (35–270)	135 (40–302)	0.45
Vaccination status (vaccinated at least one dose-%)	10 (1.9%)	2 (2%)	8 (1.9%)	0.96	0 (0%)	2 (2.4%)	0.5	19 (46.3%)	18 (60%)	0.25
Multiorgan impairment										
No organ impairment (%)	227 (43%)	0 (0%)	222 (52%)	**<0.001**	0 (0%)	0 (0%)	1	0 (0%)	16 (53%)	**<0.001**
≥2 organs impaired (%)	118 (22%)	47 (46%)	69 (16%)	**<0.001**	11 (58%)	36 (43%)	0.3	20 (49%)	4 (13%)	**0.002**
≥3 organs impaired (%)	38 (7.1%)	14 (14%)	24 (5.7%)	**0.005**	6 (32%)	8 (9.6%)	**0.02**	5 (12%)	1 (3.3%)	0.39
Symptoms										
No of symptoms (median, IQR)	9[7 11]	10 (8,11)	10 (8,11)	1	10 (8,11.5)	10 (8.5, 11)	0.44	2 (0,5)	4 (0,6)	0.26
Fever (%)	374 (70%)	69 (68%)	299 (71%)	0.62	15 (79%)	54 (66%)	0.27	1 (2.4%)	2 (7%)	0.57
Cough (%)	397 (75%)	81 (80%)	312 (74%)	0.19	16 (84%)	65 (79%)	0.76	2 (4.9%)	9 (30%)	**0.01**
Sore throat (%)	379 (71%)	70 (69%)	302 (72%)	0.65	11 (58%)	59 (72%)	0.23	6 (15%)	5 (17%)	1
Runny nose (%)	175 (33%)	35 (35%)	137 (32%)	0.67	9 (47%)	26 (32%)	0.2	2 (4.9%)	4 (13%)	0.23
Wheezing (%)	268 (50%)	51 (50%)	214 (51%)	0.97	15 (79%)	36 (44%)	**0.01**	4 (9.8%)	1 (3.3%)	0.39
Chest pain (%)	435 (82%)	82 (81%)	347 (82%)	0.81	15 (79%)	67 (82%)	0.75	14 (34%)	12 (40%)	0.61
Muscle aches (%)	472 (89%)	81 (80%)	384 (91%)	**0.002**	14 (74%)	67 (82%)	0.52	13 (32%)	11 (37%)	0.66
Joint pain (%)	394 (74%)	69 (68%)	319 (76%)	0.13	11 (58%)	58 (71%)	0.28	12 (29%)	11 (37%)	0.51
Fatigue or Malaise (%)	522 (98%)	101 (100%)	413 (98%)	0.22	19 (100%)	82 (100%)	1	20 (49%)	19 (63%)	0.22
Shortness of breath (%)	473 (89%)	89 (88%)	377 (89%)	0.72	19 (100%)	70 (85%)	0.12	13 (32%)	16 (53%)	0.07
Inability to walk (%)	177 (33%)	41 (41%)	132 (31%)	0.07	10 (53%)	31 (38%)	0.24	2 (4.9%)	0 (0%)	0.51
Headache (%)	446 (84%)	84 (83%)	356 (84%)	0.77	13 (68%)	71 (87%)	0.08	13 (32%)	10 (33%)	0.89
Seizures (%)	5 (0.9%)	1 (1.0%)	4 (0.9%)	1	0 (0%)	1 (1.2%)	1	0 (0%)	0 (0%)	1
Abdominal pain (%)	285 (54%)	59 (58%)	221 (52%)	0.27	10 (53%)	49 (60%)	0.57	6 (15%)	5 (17%)	1
Diarrhoea (%)	304 (57%)	59 (58%)	242 (57%)	0.85	12 (63%)	47 (57%)	0.64	5 (12%)	5 (17%)	0.73

Characteristics for overall population, CMR abnormalities versus no CMR abnormalities at 6 and 12 months in individuals with Long Covid. Values are presented as mean (SD) and p values calculated with t-test when the data were normally distributed. For variables where data were not normally distributed data are presented with median (IQR) and p values are calculated with Wilcoxon rank sum test. p ≤0.05 are in bold.
BMI, body mass index; CMR, cardiac MR; LVD-36, Left Ventricular Dysfunction Questionnaire.

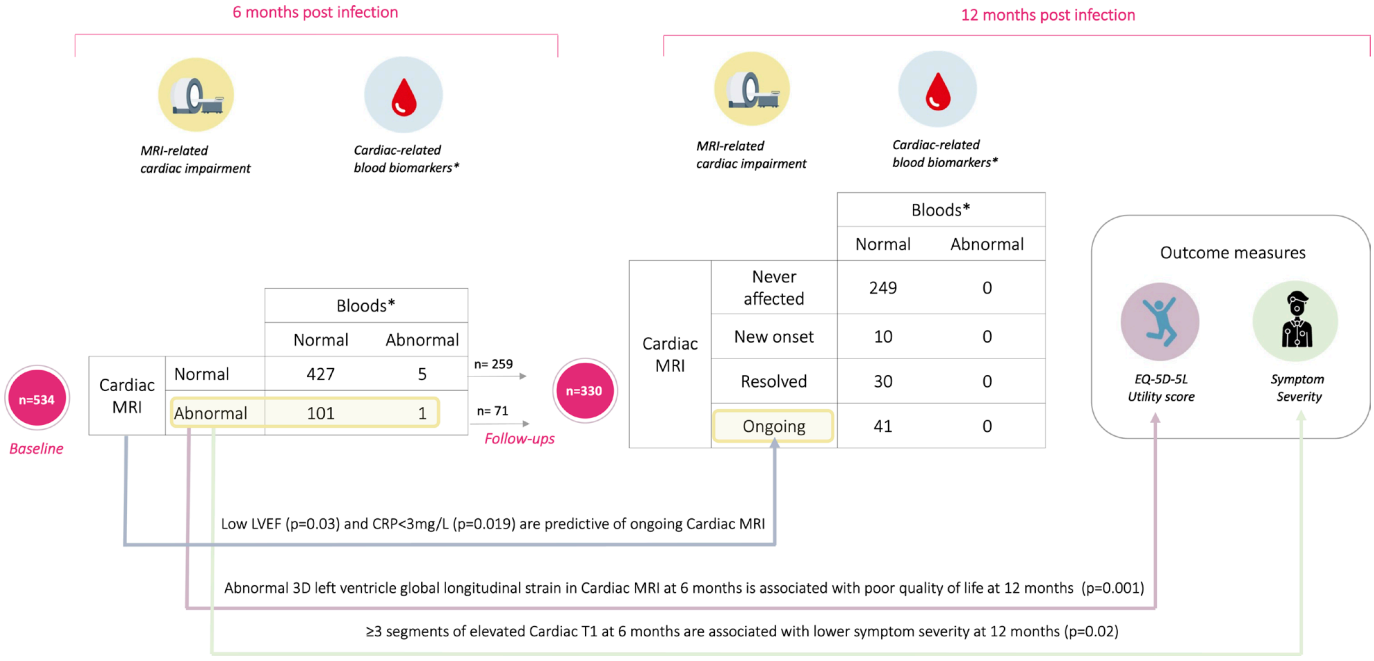

Figure 2 Central illustration. Evolution and characteristics of cardiac abnormalities in Long Covid 1-year post-SARS-CoV-2 infection. Numbers in the table are referring to number of patients. *Referring to high sensitivity cardiac troponin I and B-type natriuretic peptide. CRP, C reactive protein; EQ-5D-5L, EuroQoL-5 dimension-5 level; LVEF, left ventricular ejection fraction.

them reduced the number of symptoms (median 10 and 2 symptoms at 6 and 12 months, respectively) and 6/41 (15%) patients become asymptomatic (table 1). Of 41, 7 (17%) individuals had acute COVID-19 hospitalisation. Only 1/41 (2%) required hospitalisation between visits. Average time off work was not significantly different between resolved and ongoing impairment groups. Ten individuals with normal cardiac function at 6 months developed CMR abnormalities by 12 months (elevated cardiac T1: n=6, low RVEF: n=4, low LVEF: n=1) (online supplemental table S6).

Impact of hospitalisation versus non-hospitalisation in the acute stage and CMR abnormalities

Most individuals (83/102 (81.4%)) with CMR abnormalities did not require hospitalisation at the acute stage. Nevertheless, acute COVID-19 hospitalisation in those with CMR abnormalities (19%) was associated with severe symptoms (68% vs 37%, p=0.01), T1 elevation by CMR (68% vs 40%, p=0.02) and multiorgan involvement (≥3 organs; 32% vs 9.6%, p=0.02), compared with non-hospitalised individuals (tables 1–2).

Associations of cardiac markers and outcomes in long COVID populations at risk of CMR abnormalities

CMR abnormality at 12 months was mainly predicted by having low LVEF (p=0.03) and CRP levels ≤3 mg/L (p=0.019) at 6 months, based on stepwise multivariable logistic regression. CMR abnormalities as a composite group at 6 months were not predictive of any clinical outcome measures at 12 months; however, low GLS and elevated cardiac T1 at 6 months were predictive of poor quality of life (OR: 0.78 (95% CI 0.67 to 0.91), p=0.001) and lower symptom severity (OR: 0.71 (95% CI 0.52 to 0.96), p=0.02) at 12 months (figure 2).

Multiorgan MRI (including CMR) and integrated clinical assessment

Technical success of multiorgan MRI was 99.1% and 98.3% at baseline and follow-up assessments, respectively. Technical success of CMR and integrated in-person assessment was 99.6% at first visit and 98.8% at follow-up.

DISCUSSION

In the largest community-based study to-date with cardiac MR follow-up over 1 year in a mainly non-hospitalised, post-COVID-19 cohort with little prior cardiac disease, we report three new findings. First, CMR abnormalities were common (one in five individuals at 6 months) and commonly persisted (three out of five individuals at 12 months). Second, CMR abnormalities were found even without acute COVID hospitalisation (83/462, 18%). Third, cardiac blood biomarkers and symptoms were not predictive of composite CMR abnormalities but abnormal individual CMR parameters (eg, LVEF, 3D global longitudinal strain and cardiac T1) were associated with ongoing CMR findings, lower quality of life or reduced symptom severity at 12 months.

Characteristics and trajectory of cardiac abnormalities

Our results indicate that, despite women being more affected by Long COVID, men have higher risk of cardiac abnormalities.[15] Potential contributory factors include: influence of biological sex on expression and regulation of ACE 2, sex differences in genetic and hormonal

Table 2 CMR abnormalities in long Covid at 6 and 12 months postinfection

	6 months						12 months		
	CMR abnormalities n=102	No CMR abnormalities n=424	P value	CMR abnormalities and hospitalised, n=19	CMR abnormalities not hospitalised, n=83	P value	Ongoing CMR abnormalities n=41	Resolved CMR abnormalities n=30	P value
Elevated T1	46 (45%)	0 (0%)	**<0.001**	13 (68%)	33 (40%)	**0.02**	13 (32%)	0 (0%)	**<0.001**
Left ventricle									
High end diastolic volume	4 (3.9%)	0 (0%)	**0.001**	1 (5.3%)	3 (3.6%)	0.6	2 (4.9%)	0 (0%)	0.32
High end systolic volume	6 (5.9%)	4 (0.9%)	**0.005**	1 (5.3%)	5 (6.0%)	1	3 (7.3%)	0 (0%)	0.15
Low ejection fraction	21 (21%)	0 (0%)	**<0.001**	5 (26%)	16 (19%)	0.5	9 (22%)	0 (0%)	**0.008**
High stroke volume	1 (1.0%)	3 (0.7%)	0.58	0 (0%)	1 (1.2%)	1	0 (0%)	0 (0%)	1
High ventricular muscle mass	6 (5.9%)	18 (4.2%)	0.44	2 (11%)	4 (4.8%)	0.3	2 (4.9%)	2 (6.7%)	0.60
High ventricular maximum wall thickness	11 (11%)	26 (6.1%)	0.09	3 (17%)	8 (9.6%)	0.4	4 (9.8%)	4 (13%)	0.23
Low global circumferential strain	11 (11%)	13 (3.1%)	**0.002**	2 (11%)	9 (11%)	1	6 (15%)	1 (3.3%)	0.17
Low global longitudinal strain	21 (21%)	0 (0%)	**<0.001**	1 (5.6%)	20 (25%)	0.1	7 (17%)	0 (0%)	**0.02**
Right ventricle									
High end diastolic volume	6 (5.9%)	0 (0%)	**<0.001**	2 (11%)	4 (4.8%)	0.3	3 (7.3%)	0 (0%)	0.15
High end systolic volume	7 (6.9%)	2 (0.5%)	**<0.001**	3 (16%)	4 (4.8%)	0.1	3 (7.3%)	2 (6.7%)	0.79
Low ejection fraction	21 (21%)	0 (0%)	**<0.001**	4 (21%)	17 (20%)	1	12 (29%)	0 (0%)	**<0.001**
High stroke volume	4 (3.9%)	0 (0%)	**0.001**	1 (5.3%)	3 (3.6%)	0.6	2 (4.9%)	0 (0%)	0.32

Prevalence of abnormal CMR findings at 6 and 12 months in individuals with Long Covid.
p ≤0.05 are in bold.
CMR, cardiac MR.

Table 3 Multiorgan impairment (non-cardiac) in individuals at baseline and follow-up

	6 months							12 months		
	Overall cohort n=534	CMR abnormalities n=102	No CMR abnormalities n=424	P value	CMR abnormalities and hospitalised, n=19	CMR abnormalities not hospitalised, n=83	P value	Ongoing CMR abnormalities n=41	Resolved CMR abnormalities n=30	P value
Liver										
cT1 (high)	58 (11%)	13 (13%)	43 (10%)	0.48	3 (16%)	10 (12%)	0.71	6 (15%)	1 (3.4%)	0.23
PDFF (high)	119 (24%)	25 (26%)	92 (24%)	0.62	6 (33%)	19 (25%)	0.55	9 (23%)	5 (17%)	0.56
Volume (high)	35 (6.6%)	6 (5.9%)	29 (6.9%)	0.72	2 (11%)	4 (4.8%)	0.31	3 (7.3%)	3 (10%)	0.69
Kidneys										
Cortex T1 both kidneys (high)	28 (5.3%)	8 (7.8%)	20 (4.8%)	0.21	3 (16%)	5 (6.0%)	0.17	2 (4.9%)	0 (0%)	0.51
Volume, both kidneys (high)	18 (3.4%)	6 (5.9%)	12 (2.9%)	0.14	3 (16%)	3 (3.6%)	0.08	2 (4.9%)	2 (6.9%)	1
Left cortex T1 (high)	61 (12%)	12 (12%)	48 (11%)	0.92	5 (26%)	7 (8.4%)	**0.04**	4 (9.8%)	3 (10%)	1
Right cortex T1 (high)	46 (8.7%)	8 (7.8%)	38 (9.1%)	0.70	3 (16%)	5 (6.0%)	0.17	5 (12%)	1 (3.4%)	0.40
Left volume (high)	28 (5.3%)	7 (6.9%)	21 (5.0%)	0.45	3 (16%)	4 (4.8%)	0.12	2 (4.9%)	2 (6.9%)	1
Right volume (high)	38 (7.2%)	8 (7.8%)	30 (7.1%)	0.81	4 (21%)	4 (4.8%)	**0.04**	4 (9.8%)	2 (6.9%)	1
Pancreas										
T1 (high)	46 (9.1%)	8 (8.4%)	37 (9.2%)	0.81	2 (11%)	6 (7.8%)	0.64	2 (5.9%)	4 (17%)	0.22
PDFF (high)	77 (15%)	14 (14%)	62 (15%)	0.86	4 (22%)	10 (12%)	0.28	4 (11%)	6 (23%)	0.30
Spleen										
Volume (high)	42 (7.9%)	6 (5.9%)	36 (8.6%)	0.37	1 (5.3%)	5 (6.0%)	1	4 (9.8%)	1 (3.4%)	0.39
Length (high)	43 (8.1%)	8 (7.8%)	35 (8.3%)	0.88	1 (5.3%)	7 (8.4%)	1	3 (7.3%)	0 (0%)	0.26
Lungs										
Total deep fractional area change (low)	12 (2.4%)	10 (2.5%)	2 (2.2%)	1	0 (0%)	2 (2.7%)	1	0 (0%)	0 (0%)	1

Prevalence of abnormal CMR findings for multiorgan scans at 6 and 12 months in individuals with Long Covid.
p ≤0.05 are in bold.
CMR, cardiac MR; PDFF, proton-density fat fraction; PDFF, proton-density fat fraction.

Table 4 CMR metrics in those with between ongoing and resolved cardiac abnormalities

			6 months			12 months		
		Healthy controls n=92	CMR abnormalities ongoing at 12 months, n=41	CMR abnormalities resolving at 12 months, n=41	P value	CMR abnormalities ongoing at 12 months, N=41	CMR abnormalities resolving at 12 months, N=30	P value
Global T1 (ms)	1.5T	968 (962, 988)	974 (35)	987 (31)	0.2	982 (26)	970 (36)	0.2
	3T	1172 (1150, 1192)	1196 (37)	1200 (27)	0.8	1200 (1172,1209)	1194 (1188, 1204)	0.9
Left ventricle								
End diastolic volume (mL)		86 (79, 97)	88 (73, 97)	80 (70, 88)	0.18	86 (16)	82 (17)	0.30
End systolic volume (mL)		35 (31, 41)	39 (30, 46)	34 (28, 41)	0.07	37 (10)	33 (8)	0.07
Ejection fraction (%)		59.5 (56.6, 62.7)	55.0 (5.8)	58.1 (6.0)	**0.04**	57.7 (6.0)	60.0 (3.9)	**0.05**
Stroke volume (mL)		52 (46, 58)	45 (40, 53)	46 (43, 50)	0.99	48 (43, 54)	48 (42, 53)	0.96
Ventricular muscle mass (g)		78 (64, 96)	87 (19)	85 (24)	0.72	86 (76, 100)	84 (68, 108)	0.79
Ventricular max wall thickness (mm)		8.91 (8.16, 10.20)	9.45 (8.46, 10.50)	9.24 (8.30, 10.33)	0.50	9.75 (8.77, 10.74)	9.61 (8.64, 10.58)	0.56
Global circumferential strain (%)		−21.28 (2.31)	−19.64 (2.67)	−21.16 (2.44)	**0.02**	−20.43 (2.68)	−21.34 (2.16)	0.13
Global longitudinal strain (%)		−14.68 (−15.95, −13.69)	−12.85 (−14.56, −11.49)	−13.93 (−15.03, −11.89)	0.28	−13.29 (2.59)	−14.49 (2.13)	**0.04**
Right ventricle								
End diastolic volume (mL)		87 (78, 101)	83 (69, 95)	80 (67, 88)	0.52	81 (72, 98)	82 (68, 91)	0.43
End systolic volume (mL)		38 (31, 45)	37 (30, 46)	34 (27, 39)	0.16	36 (29, 43)	34 (24, 40)	0.18
Ejection fraction (%)		57.6 (4.5)	54.9 (5.7)	57.3 (5.5)	0.09	56.1 (6.1)	58.9 (5.1)	**0.04**
High stroke volume (mL)		50 (45, 58)	44 (39, 52)	46 (39, 49)	0.66	46 (40, 53)	46 (41, 50)	0.96

Detailed findings of CMR at 6 and 12 months in individuals with ongoing and resolved cardiac abnormalities. Values are presented as mean (SD) and p values calculated with t-test when the data were normally distributed. For variables where data were not normally distributed data is presented with median (IQR) and p values are calculated with Wilcoxon rank sum test.
p ≤0.05 are in bold.
CMR, cardiac MR.

regulation of immune responses,[16] sex-dependent patterns of coagulation, smoking or drinking.[4 5 17 18]

Published CMR studies in Long COVID vary by study design, cohort, follow-up duration, definition of cardiac abnormalities and estimated prevalence of cardiac abnormalities (26%–60%).[6 11 19–23] A recent review[9] highlighted under-representation of affected individuals from community-based settings, especially monitoring non-hospitalised individuals over time, which we address in this study. When COVID-19-related and classical myocardial injury are compared,[8] only 9% of individuals fulfil acute myocarditis criteria and those with more severe disease are more likely to exhibit chronic inflammation and impaired cardiac function. We report prevalence of CMR abnormalities (19% and 15% at 6 and 12 months) consistent with previous studies, providing standardisation of metrics and definition, which can be used at scale in research and practice to document and monitor cardiac abnormalities.[6 11 16 19 20] We confirm that abnormalities in T1 (in line with previous research,[6 9–11 19 22] T2 and LGE, as well as functional abnormalities,[5 11 23 24] are most common in Long COVID patients. Acute COVID can present with myocardial inflammation; ongoing COVID-19 patients can also have myocarditis, but it is harder to diagnose, and often missed with echocardiography. More pertinently, the observed functional changes may be due to inflammation and other aetiologies (eg, pulmonary disease, microinfarctions, metabolic dysregulation), and further mechanistic work is required to explore associations with CMR markers seen here.

In 58 hospitalised individuals, 3 months post-COVID-19, there were persistent abnormalities in cardiac T1 (26%) and multiple organs (eg, 29% with increased cortical T1, a marker of kidney inflammation). At 6 months, 52% had persistent symptoms and CMR abnormalities.[19] In the first 201 individuals in our study, we observed multiorgan impairment (29%; cardiac: 26%; renal: 4%).[11] In 443 individuals, 10 months after mild-to-moderate COVID-19, subclinical multiorgan impairment was associated with CMR abnormalities (reduced left and right ventricular systolic function).[10] At 12 months, the longest follow-up duration to-date, we confirm 54% of individuals with CMR abnormalities do not fully recover.

Impact of acute hospitalisation for COVID-19 on cardiac abnormalities

Most individuals presenting with CMR abnormalities at baseline did not require acute COVID-19 hospitalisation (81%). One individual with elevated cardiac-related blood biomarkers had CMR abnormalities at 6 months and acute COVID-19 hospitalisation. Blood biomarkers and symptoms did not differentiate hospitalised and non-hospitalised groups. On MRI, cardiac T1 abnormalities[4 25] and multiorgan involvement (particularly renal)[5 11 19] were more prevalent in those with CMR abnormalities and acute COVID-19 hospitalisation, as in other published studies.[8 13 26 27]

Clinical management pathways in Long COVID populations at risk of cardiac abnormalities

Cardiac-related blood biomarkers may be raised in early convalescence from COVID-19,[28] but did not aid detection of CMR abnormalities in Long COVID in our study, despite 19% having CMR abnormalities, supported by other research.[5 23 24] Burden and improvement in symptoms 6 months after COVID-19 were neither correlated with resolution on CMR nor lung parenchymal recovery.[5] Early MRI assessment may identify organ-specific impairment (including cardiac), leading to early referral for appropriate specialist assessment and treatment, in contrast to the experience of many patients who are currently having multiple appointments with multiple specialists for multiple assessments. In a cluster-randomised design, the STIMULATE-ICP trial is currently evaluating whether multiorgan MRI (Coverscan) can aid diagnosis and follow-up of cardiac and multiorgan impairment in Long COVID, and reduce burden to healthcare systems, already struggling due to COVID-19-related lack of resources and backlogs, while achieving integrated care.[29]

Cardiac findings could inform design of Long COVID treatment algorithms. Abnormal GLS is associated with cardiac remodelling (indicative of more severe cardiac disease),[26] and predictive of low quality of life at 12 months. Elevated T1 was predictive of lower symptom severity at 12 months. There may be multiple cardiac subgroups in Long COVID, potentially detected by CMR early postinfection. These subtypes may be related to pulmonary hypertension,[13] pre-existing comorbidities[27] and post-COVID-19 myocardial inflammation,[8] but require further study and validation.

Comprehensive multiorgan MRI assessment may help clinical decision making and improve healthcare access and provision. Evidence of cardiac involvement could guide follow-up assessment and identification of Long COVID subtypes in research and practice. Interventional trials with prespecified subgroup analysis and improved definitions of cardiac abnormality (not only myocarditis centred), are required to inform cost-effective therapies.

Strengths and limitations

This is the largest longitudinal study to-date of cardiac abnormality in Long COVID with detailed biochemical and imaging characterisation of multiorgan function starting in April 2020. We included healthy, age-matched controls. All MRI was non-contrast. We recruited a real-world cohort at lower risk of COVID-19 severity and mortality. Unlike other studies,[30] our approach offers quick, scalable assessment using standard MRI scanners. There are limitations. First, our CMR protocol excluded gadolinium contrast, the main reason for this was to reduce the scanning times, contact-time between the patient and the healthcare worker, and to avoid potential renal complications related to COVID-19. This was backed by previous research, supporting the use of native non-invasive T1 mapping to characterise myocardial

inflammation,[26] and did not have sufficient statistical power in cardiac T2 collection, relying on native non-invasive T1 mapping to characterise myocardial inflammation, validated for acute myocarditis.[31] Second, we are not able to define whether these individuals presented with multiorgan abnormalities before their COVID-19 infection, although clinical diagnoses were recorded. Third, we did not have follow-up scans on individuals without impairment at baseline and a third of patients with CMR abnormalities at baseline withdrew or were lost to follow-up. Fourth, we did not have pre-COVID-19 cardiac or multiorgan imaging available in participants. Fifth, our study population was not ethnically diverse, and COVID-19 has disproportionately affected non-white individuals. In addition, our study recruited patients during the first wave of the pandemic, when testing was not broadly available, mainly via patient support groups rather than a systematic screen of post-COVID-19 patients, as Long COVID clinics were only set up at the end of our recruitment and this may represent a bias.

CONCLUSION

CMR shows that cardiac abnormality persists in Long COVID in some individuals up to 12 months after first symptoms. CMR abnormalities (left ventricular or right ventricular dysfunction/dilatation and/or abnormal T1mapping), are associated with acute COVID-19 hospitalisation and male gender, but subtypes of disease (based on symptoms, examination and investigations) are yet to be established. Therapeutic options and effective clinical pathways require urgent clinical trials.

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Competing interests AD, AT, VC, ABo, SF, MP, ARF, HTB, MK, MR, MB and RB are employees of Perspectum.

Patient consent for publication Not applicable.

Ethics approval The protocol received full ethical approval from South Central - Berkshire B Research Ethics Committee (20/SC/0185). Participants gave informed consent to participate in the study before taking part.

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Data availability statement All data relevant to the study are included in the article or uploaded as online supplemental information.

Author note Short Tweet: Among 534 people with Long COVID, CMR abnormalities were present in 1 in 5 individuals with Long COVID at 6 months, persisting in over half of those at 12 months. Cardiac-related blood biomarkers are unable to identify CMR abnormalities in Long COVID. #LongCOVID #ACC #CVD.

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Supplementary materials

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Supplementary methods

1. **Study recruitment:** The study website (coverscan.com) was advertised by Perspectum or by iWGC (an independent platform and service provider for patient experience data collection and analysis) via online posting (social media, website banners) and by global invitation emails to applicable charities and consented patient networks. Participants interested in the study registered their interest by responding to a link located in the study's webpage. Participant response overwhelmed capacity so we randomly selected participants to be contacted for phone screening interview, without a formal algorithm. Participation was voluntary (no remuneration).

2. **Classification of Severity:** Long COVID disease severity categories are described below (a, b).

   a. **Definition Long COVID, based on validated questionnaires** (1–3)**:**

	Dyspnoea 12 score (1)		EQ-5D-5L usual activity score*(4)
Severe Long COVID	≥10	or	≥3
Mild Long COVID	Not fulfilling neither of the conditions above		

*** EQ-5D-5L score is based on the scoring in the following question:**

"Please tick the ONE box that best describes your health TODAY"

USUAL ACTIVITIES (e.g., work, study, housework, family, or leisure activities)

- *I have no problems doing my usual activities (score 1)*
- *I have slight problems doing my usual activities (score 2)*
- *I have moderate problems doing my usual activities (score 3)*
- *I have severe problems doing my usual activities (score 4)*
- *I am unable to do my usual activities (score 5)*

   b. **Definition of symptom severity, based on self-reported perceived severity:**

Patient asked to select one of the following options:
Critical acute respiratory distress syndrome (ARDS)
Severe disease
Moderate disease
Mild disease
Asymptomatic disease

   c. **Severe breathlessness**
   Score of ≥10 in the Dyspnoea-12 validated questionnaire

3. **List of blood biomarkers assessed in this study:**

Haemoglobin, HCT, red cell count, MCV, MCH , MCHC, RDW , platelet count, MPV, white cell count , neutrophils, lymphocytes monocytes, eosinophils, basophils, ESR, sodium, potassium, chloride, bicarbonate, urea, creatinine, bilirubin, alkaline phosphatase, aspartate transferase , alanine transferase , LDH , CK , gamma , total protein , albumin , globulin, calcium, magnesium, phosphate, uric acid, triglycerides, fasting triglycerides, cholesterol, fasting cholesterol, HDL cholesterol, LDL cholesterol, iron, TIBC, transferrin saturation, CRP high

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sensitivity, troponin I high sensitivity, amylase, ferritin, lipase, thyroid stimulating hormone, testosterone, insulin , C-peptide, NTpro-BNP.

4. Imaging acquisitions:

- Cardiac imaging involved a combination of several gated cine series, two long axis cines (horizontal long axis – HLA and vertical long axis – VLA), and a complete short axis stack covering the left ventricle (LV) and right ventricle (RV). This acquisition mirrors the one used at the UK Biobank and is a standardized approach (5). Three short-axis were acquired at the basal, mid, and apical levels of the left ventricle, for T1 mapping using MOLLI, and for T2 mapping using a T2 preparation pulse applied with different T2 preparation times, to impart T2 signal contrast, and a subsequent readout is performed by using a steady-state free precession (SSFP) or a fast low angle shot (FLASH). Cardiac T2 was only collected at the tail end of recruitment and therefore there was insufficient sample size (27/534 at baseline) for inclusion in manuscript results. In line with recent research in amyloidosis and published consensus on myocardial inflammation (6)(7), late gadolinium enhancement was not undertaken in this study where a combination of speed, cost, convenience, and hepatotoxicity avoidance were required to deliver CMR at scale.

Cardiac mapping	
T1	
1.5T Siemens	Modified Look-Locker inversion recovery sequence (MOLLI): For HR >700 ms/beat, TR= ~312.64 ms, TE= ~1.33 or Min ms, Flip angle= 35°, FOV= 384 mm,SL= 8mm, Ny*Nx= 256x256 matrix, Inversion Scheme= 5(3)3, parallel imaging technique/factor= GRAPPA2, BW= 1085 Hz/pixel. For HR <700 ms/beat, TR= ~312.64 ms, TE= ~1.33 or Min ms, Flip angle= 35°, FOV= 384 mm,SL= 8mm, Ny*Nx= 192 x 192 matrix, Inversion Scheme= 5(3)3, parallel imaging technique/factor= GRAPPA2, BW= 1085 Hz/pixel.
3T Siemens	MOLLI: For HR >700 ms/beat,TR= ~ 280.56 ms, TE= ~ 1.12 or Min ms, Flip angle= 35°, FOV= 360 mm,SL= 8mm, Ny*Nx= 256x256 matrix, Inversion Scheme= 5(3)3, parallel imaging technique/factor= GRAPPA2, BW= 1085 Hz/pixel. For HR <700 ms/beat, TR= ~ 272.13ms, TE= ~ 1.2 or Min ms, Flip angle= 35°, FOV= 360 mm,SL= 8mm, Ny*Nx= 192 x192 matrix, Inversion Scheme= 5(3)3, parallel imaging technique/factor= GRAPPA2, BW= 1085 Hz/pixel.
T2	
1.5T Siemens	2D SSFP sequence (TrueFISP): TR= ~ 214.07 ms or Min, TE= ~ 1.23 ms, FOV= 384x308 mm^2, SL= 8mm, Ny*Nx= 192x116 matrix, segment =58, parallel imaging technique/factor= GRAPPA2, BW= 1185 Hz/pixel
3T Siemens	2D TurboFLASH sequence: TR= ~ 214.07 ms or Min, TE= Min, FOV= 360x288 mm^2, SL= 8mm, Ny*Nx= 192x116 matrix, segment = 58, parallel imaging technique/factor= GRAPPA2, BW= 1185 Hz/pixel.

- Liver and pancreas imaging used the LiverMultiScan acquisition protocol (Perspectum, Oxford, UK), which involves 3 single 2D axial slice breath-held acquisitions that

separately are sensitive to the fat content (proton density fat fraction, or PDFF), to T2* (which is representative of liver iron content) and a MOLLI-T1 measurement (providing a measurement of tissue water), additionally a volumetric scan was used that covers the entire liver (8).

- Lungs: Two dynamic cine MR acquisitions were acquired in the coronal plane with a 306.91ms temporal resolution: one 40 s acquisition with the patient instructed to breathe normally and a second 30 s acquisition with the patient instructed to breathe deeply.
- Kidney: single coronal view that was able to image both kidneys. Imaging contrasts were MOLLI-T1, and a spoiled gradient recalled acquisition (SPGR).
- Spleen: Volumetric SPGR MRI images

5. Image Analysis:

- Cardiac: Experienced cardiac MRI analysts used CVI42v5.11 (Cardiovascular Imaging Inc, Canada) to trace manually the myocardium in the end-diastolic and end-systolic phases in each of the short-axis views, following the standard UK Biobank evaluation approach as previously described (9). We reported ventricular function; end systolic and diastolic volume; stroke volume and ejection fraction in both ventricles; left ventricular muscle mass and ventricular max wall thickness and global longitudinal and circumferential 3D strain metrics. Mean Cardiac T1 and T2 were determined for each of the 16 cardiac segments (of the AHA 17 segment model excluding the apex)(10).
- Liver Images were analysed by data analysts experienced at using the LiverMultiScan (Perspectum, Oxford, UK) software. This yielded global metrics in each liver of PDFF (proton density fat fraction), T2*, and cT1 (cT1 is a measurement of T1 that has been corrected for the confounding effects of iron and standardised to 3 Tesla; it is elevated with disease).
- Pancreas images were analysed in an equivalent manner to the above except the software used was not FDA-cleared and iron correction was not performed. The output T1 was standardized to 3 Tesla.
- Lung cine imaging allowed the measurement of the area of the left and right lungs through the breathing cycle in the coronal plane, which used automated methods that were reviewed by image analysts. The periodicity of the area fluctuations was used to determine the respiratory rate. All analysis was performed in-house using MATLAB based tools. The method was validated by measuring the correlation between the change in area and the forced vital capacity, the latter being measured using spirometry. Patient respiration was assessed by imaging a single 2D coronal slice of the lungs over 30 seconds using a dynamic cine MRI acquisition, during which the patient instructed to breathe deeply.
- Kidney: assessed using in-house tools to fit parametric maps and to allow trained analysts to make measurements. The kidney cortex was manually segmented using the MOLLI-T1 map to guide the boundary. Multiple regions-of-interests were manually placed within the cortex to extract a median value of cortical T1 in each kidney. Volumetric delineations of the kidneys were derived from SPGR MRI images. Automated delineations were produced using a 3D convolutional neural network, trained on expert annotations. Delineations were manually checked, and corrected, if necessary, for each subject. In addition to kidney cortex T1 and kidney volume, we also derived kidney length measurements, in the inferior-superior axis, from the same organ

segmentations and assessed the correlation of kidney length and kidney volume measurements

- Spleen: Volumetric delineations were derived from SPGR MRI images. Automated delineations were produced using a 3D convolutional neural network, trained on expert annotations. Delineations were manually checked, and corrected, if necessary, for each subject.
- Organ abnormality: Calculated for each organ based on evidence of any of the measurements appearing out of reference range (*Liver:* elevated cT1 or Fat; *Kidney:* elevated T1 or volume; *Pancreas:* elevated sT1 or Fat; *Heart:* elevated T1 in 3 or more segments, decreased RV or LV EF or increased LV or RV EDV or increased LV global longitudinal strain; *Spleen*: elevated volume; *Lung:* reduced fractional area volume). Single organ impairment was based on ≥1 organ impairment and multi-organ impairment was based on ≥2 organ impairments.

6. Reference Ranges for imaging markers:

All values but organ volumes were calculated using sex and age matched HC (n=92) (Healthy Controls) scanned at 1.5T and 3T for this study calculating 2.5% (lower threshold) and 97.5% percentiles (upper threshold). Organ volumes were calculated from a combined cohort of the 92 healthy controls and 1744 BMI matched participants (N=1836 from N=36) from the UK Biobank, (11), representing all sex and height subgroups, as these are known confounders of organ size.(12) (*) Reference ranges for the liver cT1 and liver PDFF have been taken from the available literature (13), as the LMS technology have been widely used and tested in multiple clinical trials and research settings. For pancreas PDFF, which has a positive skew in the distribution, reference ranges were extracted with the 95% percentile. ($) T2 repeatability coefficients are not provided as this metric was only really available for follow up patients. (§) Right and left cortical T1 limits were averaged for analysis (Table S1).

147 Table S1: Reference Ranges for imaging metrics across organs

	Gender	Field Strength	Height (cm)	Lower threshold	Upper threshold (*)	Repeatability coefficient
CARDIAC METRICS						
Field strength independent variables (BSA corrected)						
Left end diastolic volume (mL)	F	-	-	-	108	17
Left end diastolic volume (mL)	M	-	-	-	132	17
Right end diastolic volume (mL)	F	-	-	-	110	19
Right end diastolic volume (mL)	M	-	-	-	139	19
Left end Systolic volume (mL)	F	-	-	-	47	12.5
Left end Systolic volume (mL)	M	-	-	-	57	12.5
Right end Systolic volume (mL)	F	-	-	-	49	12
Right end Systolic volume (mL)	M	-	-	-	60	12
Left Stroke volume (mL)	F	-	-	-	66	16
Left Stroke volume (mL)	M	-	-	-	84	16
Right Stroke volume (mL)	F	-	-	-	65	16
Right Stroke volume (mL)	M	-	-	-	84	16
Field strength independent variables (non-BSA corrected)						
Global circumferential strain 3D (%)	F	-	-	-	-18.1	2.5
Global circumferential strain 3D (%)	M	-	-	-	-16.8	2.5
Global longitudinal strain 3D (%)	F	-	-	-	-11.5	5.1
Global longitudinal strain 3D (%)	M	-	-	-	-7.8	5.1
Left ventricle ejection fraction (%)	F	-	-	52	-	6.6
Left ventricle ejection fraction (%)	M	-	-	51	-	6.6
Right ventricle ejection fraction (%)	F	-	-	50	-	7.0
Right ventricle ejection fraction (%)	M	-	-	50	-	7.0
Left ventricular max wall thickness (mm)	F	-	-	-	10.6	2.1
Left ventricular max wall thickness (mm)	M	-	-	-	14	2.1
Left ventricular muscle mass (g)	F	-	-	-	95	13
Left ventricular muscle mass (g)	M	-	-	-	151	13
Field strength Dependent variables: 1.5T						
Global T1 ref range (ms)	F	1.5T	-	-	1042	-
Global T1 ref range (ms)	M	1.5T	-	-	997	-
Segment 1: T1 basal anterior (ms)	F	1.5T	-	-	1043	42
Segment 1: T1 basal anterior (ms)	M	1.5T	-	-	1000	42
Segment 2: T1 basal anteroseptal (ms)	F	1.5T	-	-	1031	49
Segment 2: T1 basal anteroseptal (ms)	M	1.5T	-	-	1022	49
Segment 3: T1 basal inferoseptal (ms)	F	1.5T	-	-	1031	54
Segment 3: T1 basal inferoseptal (ms)	M	1.5T	-	-	1001	54
Segment 4: T1 basal inferior (ms)	F	1.5T	-	-	1091	57
Segment 4: T1 basal inferior (ms)	M	1.5T	-	-	995	57
Segment 5: T1 basal inferolateral (ms)	F	1.5T	-	-	1042	55
Segment 5: T1 basal inferolateral (ms)	M	1.5T	-	-	998	55
Segment 6: T1 basal anterolateral (ms)	F	1.5T	-	-	1041	54
Segment 6: T1 basal anterolateral (ms)	M	1.5T	-	-	979	54
Segment 7: T1 mid anterior (ms)	F	1.5T	-	-	1014	52
Segment 7: T1 mid anterior (ms)	M	1.5T	-	-	969	52
Segment 8: T1 mid anteroseptal (ms)	F	1.5T	-	-	1030	39
Segment 8: T1 mid anteroseptal (ms)	M	1.5T	-	-	1006	39
Segment 9: T1 mid inferoseptal (ms)	F	1.5T	-	-	1036	37
Segment 9: T1 mid inferoseptal (ms)	M	1.5T	-	-	994	37
Segment 10: T1 mid inferior (ms)	F	1.5T	-	-	1035	44
Segment 10: T1 mid inferior (ms)	M	1.5T	-	-	1023	44
Segment 11: T1 mid inferolateral (ms)	F	1.5T	-	-	1016	44
Segment 11: T1 mid inferolateral (ms)	M	1.5T	-	-	982	44
Segment 12: T1 mid anterolateral (ms)	F	1.5T	-	-	1029	62
Segment 12: T1 mid anterolateral (ms)	M	1.5T	-	-	979	62
Segment 13: T1 apical anterior (ms)	F	1.5T	-	-	1059	86
Segment 13: T1 apical anterior (ms)	M	1.5T	-	-	1004	86
Segment 14: T1 apical septal (ms)	F	1.5T	-	-	1065	48
Segment 14: T1 apical septal (ms)	M	1.5T	-	-	992	48
Segment 15: T1 apical inferior (ms)	F	1.5T	-	-	1070	43
Segment 15: T1 apical inferior (ms)	M	1.5T	-	-	1003	43
Segment 16: T1 apical lateral (ms)	F	1.5T	-	-	1040	70
Segment 16: T1 apical lateral (ms)	M	1.5T	-	-	1011	70
Global T2 ref range (ms) ($)	-	1.5T	-	-	51	-
Field strength Dependent variables: 3T						
Global T1 ref range (ms)	F	3T	-	-	1255	-
Global T1 ref range (ms)	M	3T	-	-	1214	-

	Gender	Field Strength	Height (cm)	Lower threshold	Upper threshold (*)	Repeatability coefficient
Segment 1: T1 basal anterior (ms)	F	3T	-	-	1226	72
Segment 1: T1 basal anterior (ms)	M	3T	-	-	1201	72
Segment 2: T1 basal anteroseptal (ms)	F	3T	-	-	1248	70
Segment 2: T1 basal anteroseptal (ms)	M	3T	-	-	1218	70
Segment 3: T1 basal inferoseptal (ms)	F	3T	-	-	1251	74
Segment 3: T1 basal inferoseptal (ms)	M	3T	-	-	1218	74
Segment 4: T1 basal inferior (ms)	F	3T	-	-	1271	112
Segment 4: T1 basal inferior (ms)	M	3T	-	-	1231	112
Segment 5: T1 basal inferolateral (ms)	F	3T	-	-	1240	109
Segment 5: T1 basal inferolateral (ms)	M	3T	-	-	1209	109
Segment 6: T1 basal anterolateral (ms)	F	3T	-	-	1200	61
Segment 6: T1 basal anterolateral (ms)	M	3T	-	-	1193	61
Segment 7: T1 mid anterior (ms)	F	3T	-	-	1266	90
Segment 7: T1 mid anterior (ms)	M	3T	-	-	1161	90
Segment 8: T1 mid anteroseptal (ms)	F	3T	-	-	1264	89
Segment 8: T1 mid anteroseptal (ms)	M	3T	-	-	1219	89
Segment 9: T1 mid inferoseptal (ms)	F	3T	-	-	1272	74
Segment 9: T1 mid inferoseptal (ms)	M	3T	-	-	1226	74
Segment 10: T1 mid inferior (ms)	F	3T	-	-	1279	84
Segment 10: T1 mid inferior (ms)	M	3T	-	-	1228	84
Segment 11: T1 mid inferolateral (ms)	F	3T	-	-	1226	60
Segment 11: T1 mid inferolateral (ms)	M	3T	-	-	1210	60
Segment 12: T1 mid anterolateral (ms)	F	3T	-	-	1278	75
Segment 12: T1 mid anterolateral (ms)	M	3T	-	-	1228	75
Segment 13: T1 apical anterior (ms)	F	3T	-	-	1271	63
Segment 13: T1 apical anterior (ms)	M	3T	-	-	1227	63
Segment 14: T1 apical septal (ms)	F	3T	-	-	1280	62
Segment 14: T1 apical septal (ms)	M	3T	-	-	1230	62
Segment 15: T1 apical inferior (ms)	F	3T	-	-	1257	57
Segment 15: T1 apical inferior (ms)	M	3T	-	-	1202	57
Segment 16: T1 apical lateral (ms)	F	3T	-	-	1254	77
Segment 16: T1 apical lateral (ms)	M	3T	-	-	1214	77
Global T2 ref range (ms) ($)	-	3T	-	-	46	-
LIVER METRICS						
Field strength independent variables						
cT1 ROI (ms)	-	-	-	-	800 (*)	48
PDFF %	-	-	-	-	5 (*)	1.5
Volume (mL)	F	-	<164	-	1778	64
Volume (mL)	M	-	<164	-	2003	64
Volume (mL)	F	-	≥ 164, < 250	-	2049	64
Volume (mL)	M	-	≥ 164, < 250	-	2284	64
KIDNEY METRICS						
Field strength independent variables						
Left Volume (mL)	F	-	<164	-	177	10
Left Volume (mL)	M	-	<164	-	221	10
Left Volume (mL)	F	-	≥ 164, < 250	-	192.	10
Left Volume (mL)	M	-	≥ 164, < 250	-	255	10
Right Volume (mL)	F	-	<164	-	176	8
Right Volume (mL)	M	-	<164	-	207	8
Right Volume (mL)	F	-	≥ 164, < 250	-	186	8
Right Volume (mL)	M	-	≥ 164, < 250	-	229	8
Field strength Dependent variables: 1.5T						
Cortex T1 (ms) (§)	-	1.5T	-	-	1154	76
Field strength Dependent variables: 3T						
Cortex T1 (ms) (§)	-	3T	-	-	1512	68
PANCREAS						
Field strength independent variables						
sT1 ROI (ms)	-	-	-	-	821	74
PDFF %	-	-	-	-	6.6 (*)	2.8
SPLEEN						
Field strength independent variables						
Volume (mL)	F	-	<164	-	254	17
Volume (mL)	M	-	<164	-	392	17
Volume (mL)	F	-	≥ 164, < 250	-	293	17
Volume (mL)	M	-	≥ 164, < 250	-	411	17.
LUNG						
Field strength independent variables						

	Gender	Field Strength	Height (cm)	Lower threshold	Upper threshold (*)	Repeatability coefficient
Total deep fractional area change (%)	-	-	-	22	-	15.9

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149 Table S2: Demographics of HC compared to the Long COVID cohort, with and without CMR abnormalities
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Demographic	HC, N = 91	COVID, N = 534	Post-COVID CMR abnormalities n=102	Post-COVID No CMR abnormalities n=424	P (HC vs COVID cohort)	P (HC vs Post-COVID CMR abnormalities)	P (HC vs Post-COVID No CMR abnormalities)
Age	44 (33, 53)	44 (38, 52)	43 (37, 51)	44 (38, 52)	0.6	>0.9	0.5
Sex (% male)	30 (33%)	147 (28%)	42 (41%)	103 (24%)	0.3	0.2	0.086
BMI kg/m^2 (Median (IQR))	22.8 (20.9, 25.1)	25.5 (22.6, 29.3)	26.3 (23.1, 29.0)	25.3 (22.6, 29.4)	**<0.001**	<0.001	<0.001
BMI ≥25 to <30 kg/m^2 (%)	20 (22%)	172 (32%)	38 (37%)	131 (31%)	**0.051**	**0.021**	0.090
BMI ≥30 kg/m^2 (%)	3 (3.3%)	119 (22%)	23 (23%)	96 (23%)	**<0.001**	**<0.001**	**<0.001**
Ethnicity (%)					0.3	0.3	0.3
White	84 (92%)	475 (89%)	88 (86%)	382 (90%)			
Asian	6 (6.6%)	24 (4.5%)	7 (6.9%)	16 (3.8%)			
Black	0 (0%)	13 (2.4%)	3 (2.9%)	9 (2.1%)			
Mix	1 (1.1%)	21 (3.9%)	4 (3.9%)	16 (3.8%)			
Other	0 (0%)	1 (0.2%)	0 (0%)	1 (0.2%)			
Smoking					**0.02**	**0.013**	**0.01**
Never	71 (78%)	348 (65%)	66 (65%)	275 (65%)			
Current	3 (3.3%)	13 (2.4%)	5 (4.9%)	7 (1.7%)			
Past	17 (19%)	172 (32%)	31 (30%)	141 (33%)			
Hypertension	0 (0%)	44 (8.2%)	12 (12%)	32 (7.5%)	**0.005**	**<0.001**	**0.007**

Diabetes	0 (0%)	10 (1.9%)	3 (2.9%)	7 (1.7%)	0.4	0.20	0.60
Asthma	2 (2.2%)	101 (19%)	22 (22%)	78 (18%)	**<0.001**	**<0.001**	**<0.001**
Previous heart disease	0 (0%)	9 (1.7%)	2 (2%)	7 (1.7%)	**0.21**	**0.5**	**0.6**

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154 Table S3: Differences in CMR abnormalities and symptoms by COVID-19 diagnosis method
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	Clinically diagnosed COVID-19	COVID-19 positive PCR test result	Proportion test P value
Prevalence of CMR abnormalities at baseline			
CMR abnormalities	43 (18%)	59 (20%)	0.557
No CMR abnormalities	196 (82%)	236 (80%)	0.557
Total	239	295	
Severity of symptoms at baseline			
Mild/moderate	113 (47%)	161 (55%)	0.138
Severe/Extreme	125 (52%)	132(45%)	0.138
Total	238	293	
Trajectory of CMR abnormalities at 12 months			
Never	117(80%)	132 (72%)	0.08
New	3 (2%)	7 (4%)	0.35
Ongoing	13 (9%)	28 (15%)	0.09
Resolved	13 (9%)	17 (9%)	0.92
Total	146	184	-

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Table S4: Blood investigations by abnormalities on CMR

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
Haemoglobin													
H	6 (1.2%)	4 (1.0%)	2 (2.2%)		0 (0%)	2 (2.6%)		1 (2.6%)	0 (0%)		1 (2.4%)	0 (0%)	
L	13 (2.6%)	9 (2.2%)	3 (3.3%)	0.36	1 (6.7%)	2 (2.6%)	0.60	2 (5.3%)	0 (0%)	0.51	1 (2.4%)	0 (0%)	1.00
N	487 (96%)	393 (97%)	87 (95%)		14 (93%)	73 (95%)		35 (92%)	27 (100%)		39 (95%)	27 (100%)	
Missing	28	18	10		4	6		3	3		0	3	
HCT													
H	10 (2.0%)	9 (2.2%)	1 (1.1%)		0 (0%)	1 (1.3%)		1 (2.6%)	0 (0%)		1 (2.4%)	2 (7.4%)	
L	8 (1.6%)	5 (1.2%)	2 (2.2%)	0.69	1 (6.7%)	1 (1.3%)	0.42	1 (2.6%)	0 (0%)	1.00	0 (0%)	0 (0%)	0.56
N	488 (96%)	392 (97%)	89 (97%)		14 (93%)	75 (97%)		36 (95%)	27 (100%)		40 (98%)	25 (93%)	
Missing	28	18	10		4	6		3	3		0	3	
Red cell count													
H	14 (2.8%)	13 (3.2%)	1 (1.1%)		0 (0%)	1 (1.3%)		0 (0%)	0 (0%)		1 (2.4%)	1 (3.7%)	
L	17 (3.4%)	12 (3.0%)	3 (3.3%)	0.69	1 (6.7%)	2 (2.6%)	0.52	2 (5.3%)	0 (0%)	0.51	3 (7.3%)	0 (0%)	0.38
N	475 (94%)	381 (94%)	88 (96%)		14 (93%)	74 (96%)		36 (95%)	27 (100%)		37 (90%)	26 (96%)	
Missing	28	18	10		4	6		3	3		0	3	
MCV													
H	1 (0.2%)	0 (0%)	1 (1.1%)		0 (0%)	1 (1.3%)		1 (2.6%)	0 (0%)		2 (4.9%)	0 (0%)	
L	10 (2.0%)	8 (2.0%)	2 (2.2%)	0.20	0 (0%)	2 (2.6%)	1.00	2 (5.3%)	0 (0%)	0.51	0 (0%)	1 (3.7%)	0.30
N	495 (98%)	398 (98%)	89 (97%)		15 (100%)	74 (96%)		35 (92%)	27 (100%)		39 (95%)	26 (96%)	
Missing	28	18	10		4	6		3	3		0	3	
MCH													
H	4 (0.8%)	3 (0.7%)	1 (1.1%)		0 (0%)	1 (1.3%)		1 (2.6%)	0 (0%)		1 (2.4%)	0 (0%)	
L	8 (1.6%)	7 (1.7%)	1 (1.1%)	0.84	0 (0%)	1 (1.3%)	1.00	1 (2.6%)	0 (0%)	1.00	1 (2.4%)	1 (3.7%)	1.00
N	494 (98%)	396 (98%)	90 (98%)		15 (100%)	75 (97%)		36 (95%)	27 (100%)		39 (95%)	26 (96%)	
Missing	28	18	10		4	6		3	3		0	3	
MCHC													
H	105 (21%)	76 (19%)	26 (28%)		2 (13%)	24 (31%)		13 (34%)	4 (15%)		10 (24%)	2 (7.4%)	
L	0 (0%)	0 (0%)	0(0%)	**0.04**	0 (0%)	0 (0%)	0.22	0 (0%)	0 (0%)	0.08	0 (0%)	0 (0%)	0.07
N	401 (79%)	330 (81%)	66 (72%)		13 (87%)	53 (69%)		25 (66%)	23 (85%)		31 (76%)	25 (93%)	
Missing	28	18	10		4	6		3	3		0	3	

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
RDW													
H	12 (2.4%)	10 (2.5%)	2 (2.2%)	0.94	0 (0%)	2 (2.6%)	1.00	2 (5.3%)	0 (0%)	0.53	0 (0%)	0 (0%)	1.00
L	26 (5.1%)	22 (5.4%)	4 (4.3%)		0 (0%)	4 (5.2%)		3 (7.9%)	1 (3.7%)		1 (2.4%)	0 (0%)	
N	467 (92%)	373 (92%)	86 (93%)		15 (100%)	71 (92%)		33 (87%)	26 (96%)		40 (98%)	27 (100%)	
Missing	29	19	10		4	6		3	3		0	3	
Platelet count													
H	22 (4.4%)	15 (3.7%)	6 (6.6%)	0.13	1 (6.7%)	5 (6.6%)	1.00	4 (11%)	1 (3.7%)	0.49	1 (2.4%)	3 (11%)	0.29
L	2 (0.4%)	1 (0.2%)	1 (1.1%)		0(0%)	1 (1.3%)		1 (2.7%)	0(0%)		0(0%)	0(0%)	
N	479 (95%)	388 (96%)	84 (92%)		14 (93%)	70 (92%)		32 (86%)	26 (96%)		40 (98%)	24 (89%)	
Missing	31	20	11		4	7		4	3		0	3	
MPV													
H	8 (1.6%)	6 (1.5%)	1 (1.1%)	1.00	0 (0%)	1 (1.3%)	1.00	1 (2.6%)	0 (0%)	1.00	2 (4.9%)	0 (0%)	0.51
L	0 (0%)	0 (0%)	0 (0%)		0 (0%)	0 (0%)		0(0%)	0 (0%)		0 (0%)	0 (0%)	
N	496 (98%)	398 (99%)	91 (99%)		15 (100%)	76 (99%)		37 (97%)	27 (100%)		39 (95%)	27 (100%)	
Missing	30	20	10		4	6		3	3		0	3	
White cell count													
H	19 (3.8%)	13 (3.2%)	3 (3.3%)	1.00	0 (0%)	3 (3.9%)	1.00	2 (5.3%)	0 (0%)	0.51	0 (0%)	0 (0%)	1.00
L	1 (0.2%)	1 (0.2%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
N	486 (96%)	392 (97%)	89 (97%)		15 (100%)	74 (96%)		36 (95%)	27 (100%)		41 (100%)	27 (100%)	
Missing	28	18	10		4	6		3	3		0	3	
Neutrophils													
H	8 (1.6%)	7 (1.7%)	1 (1.1%)	0.60	0 (0%)	1 (1.3%)	0.66	1 (2.6%)	0 (0%)	0.78	0 (0%)	0 (0%)	1.00
L	30 (5.9%)	20 (4.9%)	7 (7.6%)		0 (0%)	7 (9.1%)		3 (7.9%)	1 (3.7%)		3 (7.3%)	2 (7.4%)	
N	468 (92%)	379 (93%)	84 (91%)		15 (100%)	69 (90%)		34 (89%)	26 (96%)		38 (93%)	25 (93%)	
Missing	28	18	10		4	6		3	3		0	3	
Lymphocytes													
H	2 (0.4%)	1 (0.2%)	1 (1.1%)	0.27	0 (0%)	1 (1.3%)	0.65	0 (0%)	1 (3.7%)	0.59	1 (2.4%)	1 (3.7%)	0.22
L	38 (7.5%)	32 (7.9%)	5 (5.4%)		0 (0%)	5 (6.5%)		2 (5.3%)	2 (7.4%)		7 (17%)	1 (3.7%)	
N	466 (92%)	373 (92%)	86 (93%)		15 (100%)	71 (92%)		36 (95%)	24 (89%)		33 (80%)	25 (93%)	
Missing	28	18	10		4	6		3	3		0	3	
Monocytes													
H	4 (0.8%)	2 (0.5%)	1 (1.1%)	0.64	0 (0%)	1 (1.3%)	1.00	1 (2.6%)	0 (0%)	1.00	0 (0%)	0 (0%)	1.00
L	2 (0.4%)	2 (0.5%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
N	500 (99%)	402 (99%)	91 (99%)		15 (100%)	76 (99%)		37 (97%)	27 (100%)		41 (100%)	27 (100%)	
Missing	28	18	10		4	6		3	3		0	3	

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
Eosinophils													
H L N Missing	14 (2.8%) 0 (0%) 492 (97%) 28	10 (2.5%) 0 (0%) 396 (98%) 18	4 (4.3%) 0 (0%) 88 (96%) 10	0.30	0 (0%) 0 (0%) 15 (100%) 4	4 (5.2%) 0 (0%) 73 (95%) 6	1.00	0 (0%) 0 (0%) 38 (100%) 3	1 (3.7%) 0 (0%) 26 (96%) 3	0.42	2 (4.9%) 0 (0%) 39 (95%) 0	1 (3.7%) 0 (0%) 26 (96%) 3	1.00
Basophils													
H L N Missing	2 (0.4%) 0 (0%) 504(100%) 28	2 (0.5%) 0(0%) 404 (100%) 18	0 (0%) 0 (0%) 92 (100%) 10	1.00	0 (0%) 0 (0%) 15 (100%) 4	0 (0%) 0 (0%) 77 (100%) 6	1.0	0 (0%) 0 (0%) 38 (100%) 3	0 (0%) 0 (0%) 27 (100%) 3	1.00	0 (0%) 0 (0%) 41 (100%) 0	2 (7.4%) 0 (0%) 25 (93%) 3	0.15
ESR													
H L N Missing	40 (7.9%) 0 (0%) 468 (92%) 26	32 (7.9%) 0 (0%) 375 (92%) 17	8 (8.6%) 0 (0%) 85 (91%) 9	0.81	1 (6.7%) 0 (0%) 14 (93%) 4	7 (9.0%) 0 (0%) 71 (91%) 5	1.00	4 (11%) 0 (0%) 34 (89%) 3	3 (11%) 0 (0%) 24 (89%) 3	1.00	3 (7.3%) 0 (0%) 38 (93%) 0	4 (15%) 0 (0%) 23 (85%) 3	1.0
Sodium													
H L N Missing	1 (0.2%) 17 (3.4%) 488 (96%) 28	1 (0.2%) 13 (3.2%) 392 (97%) 18	0 (0%) 4 (4.3%) 88 (96%) 10	0.62	0 (0%) 0 (0%) 15 (100%) 4	0 (0%) 4 (5.2%) 73 (95%) 6	1.00	0 (0%) 4 (11%) 34 (89%) 3	0 (0%) 0 (0%) 27 (100%) 3	0.13	0 (0%) 4 (9.8%) 37 (90%) 0	0 (0%) 0 (0%) 28 (100%) 2	0.14
Potassium													
H L N Missing	231 (49%) 0 (0%) 243 (51%) 60	188 (49%) 0 (0%) 194 (51%) 42	42 (49%) 0 (0%) 43 (51%) 17	0.97	6 (46%) 0 (0%) 7 (54%) 6	36 (50%) 0 (0%) 36 (50%) 11	0.80	17 (47%) 0 (0%) 19 (53%) 5	12 (52%) 0 (0%) 11 (48%) 7	0.71	8 (32%) 0 (0%) 17 (68%) 16	9 (47%) 0 (0%) 10 (53%) 11	0.30
Chloride													
H L N Missing	10 (2.0%) 11 (2.2%) 485 (96%) 28	7 (1.7%) 6 (1.5%) 393 (97%) 18	2 (2.2%) 5 (5.4%) 85 (92%) 10	**0.05**	0 (0%) 0 (0%) 15 (100%) 4	2 (2.6%) 5 (6.5%) 70 (91%) 6	0.71	1 (2.6%) 3 (7.9%) 34 (89%) 3	0 (0%) 0 (0%) 27 (100%) 3	0.26	0 (0%) 2 (4.9%) 39 (95%) 0	0 (0%) 0 (0%) 28 (100%) 2	0.51
Bicarbonate													
H L N Missing	25 (4.9%) 49 (9.7%) 432 (85%) 28	18 (4.4%) 37 (9.1%) 351 (86%) 18	7 (7.6%) 10 (11%) 75 (82%) 10	0.30	2 (13%) 0 (0%) 13 (87%) 4	5 (6.5%) 10 (13%) 62 (81%) 6	0.24	4 (11%) 4 (11%) 30 (79%) 3	2 (7.4%) 4 (15%) 21 (78%) 3	0.82	2 (4.9%) 1 (2.4%) 38 (93%) 0	1 (3.6%) 1 (3.6%) 26 (93%) 2	1.00
Urea													

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
H L N Missing	1 (0.2%) 1 (0.2%) 504(100%) 28	0 (0%) 1 (0.2%) 405 (100%) 18	0 (0%) 0 (0%) 92 (100%) 10	1.00	0 (0%) 0 (0%) 15 (100%) 4	0 (0%) 0 (0%) 77 (100%) 6	1.00	0 (0%) 0 (0%) 38 (100%) 3	0 (0%) 0 (0%) 27 (100%) 3	1.00	1 (2.4%) 0 (0%) 40 (98%) 0	0 (0%) 0 (0%) 28 (100%) 2	1.00
Creatinine													
H L N Missing	6 (1.2%) 25 (4.9%) 475 (94%) 28	6 (1.5%) 20 (4.9%) 380 (94%) 18	0 (0%) 5 (5.4%) 87 (95%) 10	0.65	0 (0%) 2 (13%) 13 (87%) 4	0 (0%) 3 (3.9%) 74 (96%) 6	0.19	0 (0%) 2 (5.3%) 36 (95%) 3	0 (0%) 2 (7.4%) 25 (93%) 3	1.00	1 (2.4%) 2 (4.9%) 38 (93%) 0	0 (0%) 2 (7.1%) 26 (93%) 2	1.00
Bilirubin													
H L N Missing	17 (3.4%) 0 (0%) 489 (97%) 28	11 (2.7%) 0 (0%) 395 (97%) 18	4 (4.3%) 0 (0%) 88 (96%) 10	0.49	1 (6.7%) 0 (0%) 14 (93%) 4	3 (3.9%) 0 (0%) 74 (96%) 6	0.52	3 (7.9%) 0 (0%) 35 (92%) 3	0 (0%) 0 (0%) 27 (100%) 3	0.26	2 (4.9%) 0 (0%) 39 (95%) 0	2 (7.1%) 0 (0%) 26 (93%) 2	1.00
Alkaline phosphatase													
H L N Missing	12 (2.4%) 13 (2.6%) 481 (95%) 28	9 (2.2%) 9 (2.2%) 388 (96%) 18	2 (2.2%) 4 (4.3%) 86 (93%) 10	0.44	0 (0%) 0 (0%) 15 (100%) 4	2 (2.6%) 4 (5.2%) 71 (92%) 6	1.00	1 (2.6%) 2 (5.3%) 35 (92%) 3	0 (0%) 1 (3.7%) 26 (96%) 3	1.00	0 (0%) 0 (0%) 41 (100%) 0	0 (0%) 1 (3.6%) 27 (96%) 2	0.41
Aspartate transferase													
H L N Missing	43 (8.8%) 0 (0%) 443 (91%) 48	33 (8.4%) 0 (0%) 359 (92%) 32	10 (12%) 0 (0%) 76 (88%) 16	0.35	1 (7.1%) 0 (0%) 13 (93%) 5	9 (12%) 0 (0%) 63 (88%) 11	1.00	4 (11%) 0 (0%) 31 (89%) 6	2 (8.3%) 0 (0%) 22 (92%) 6	1.00	6 (15%) 0 (0%) 34 (85%) 1	2 (8.0%) 0 (0%) 23 (92%) 5	0.47
Alanine transferase													
H L N Missing	73 (14%) 7 (1.4%) 426 (84%) 28	62 (15%) 6 (1.5%) 338 (83%) 18	11 (12%) 1 (1.1%) 80 (87%) 10	0.81	1 (6.7%) 0 (0%) 14 (93%) 4	10 (13%) 1 (1.3%) 66 (86%) 6	0.74	5 (13%) 0 (0%) 33 (87%) 3	4 (15%) 1 (3.7%) 22 (81%) 3	0.69	7 (17%) 1 (2.4%) 33 (80%) 0	5 (18%) 0 (0%) 23 (82%) 2	1.00
LDH													
H L N Missing	80 (16%) 18 (3.6%) 400 (80%) 36	63 (16%) 12 (3.0%) 326 (81%) 23	16 (18%) 3 (3.4%) 70 (79%) 13	0.79	1 (6.7%) 0 (0%) 14 (93%) 4	15 (20%) 3 (4.1%) 56 (76%) 9	0.40	9 (25%) 0 (0%) 27 (75%) 5	4 (15%) 2 (7.7%) 20 (77%) 4	0.19	10 (24%) 0 (0%) 31 (76%) 0	7 (26%) 0 (0%) 20 (74%) 3	0.89
CK													
H L	40 (7.9%) 2 (0.4%)	31 (7.6%) 1 (0.2%)	9 (9.8%) 1 (1.1%)	0.28	1 (6.7%) 0 (0%)	8 (10%) 1 (1.3%)	1.00	3 (7.9%) 1 (2.6%)	3 (11%) 0 (0%)	0.82	4 (9.8%) 1 (2.4%)	4 (15%) 0 (0%)	0.82

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
N	464 (92%)	374 (92%)	82 (89%)		14 (93%)	68 (88%)		34 (89%)	24 (89%)		36 (88%)	23 (85%)	
Missing	28	18	10		4	6		3	3		0	3	
Gamma GT													
H	32 (6.3%)	26 (6.4%)	5 (5.4%)		0 (0%)	5 (6.5%)		3 (7.9%)	1 (3.7%)		3 (7.3%)	0 (0%)	
L	12 (2.4%)	8 (2.0%)	3 (3.3%)	0.66	0 (0%)	3 (3.9%)	0.76	2 (5.3%)	0 (0%)	0.53	3 (7.3%)	0 (0%)	0.16
N	462 (91%)	372 (92%)	84 (91%)		15 (100%)	69 (90%)		33 (87%)	26 (96%)		35 (85%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Total protein													
H	2 (0.4%)	1 (0.2%)	1 (1.1%)		0 (0%)	1 (1.3%)		1 (2.6%)	0 (0%)		0 (0%)	0 (0%)	
L	7 (1.4%)	5 (1.2%)	2 (2.2%)	0.30	0 (0%)	2 (2.6%)	1.00	1 (2.6%)	1 (3.7%)	1.00	1 (2.4%)	0 (0%)	1.00
N	497 (98%)	400 (99%)	89 (97%)		15 (100%)	74 (96%)		36 (95%)	26 (96%)		40 (98%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Albumin													
H	27 (5.3%)	21 (5.2%)	6 (6.5%)		0 (0%)	6 (7.8%)		4 (11%)	2 (7.4%)		1 (2.4%)	0 (0%)	
L	0 (0%)	0 (0%)	0 (0%)	0.61	0 (0%)	0 (0%)	0.58	0 (0%)	0 (0%)	1.00	0 (0%)	0 (0%)	1.00
N	479 (95%)	385 (95%)	86 (93%)		15 (100%)	71 (92%)		34 (89%)	25 (93%)		40 (98%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Globulin													
H	2 (0.4%)	2 (0.5%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
L	14 (2.8%)	11 (2.7%)	3 (3.3%)	0.82	0 (0%)	3 (3.9%)	1.00	2 (5.3%)	0 (0%)	0.51	1 (2.4%)	0 (0%)	1.00
N	490 (97%)	393 (97%)	89 (97%)		15 (100%)	74 (96%)		36 (95%)	27 (100%)		40 (98%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Calcium													
H	7 (1.4%)	4 (1.0%)	3 (3.3%)		0 (0%)	3 (3.9%)		2 (5.3%)	1 (3.7%)		0 (0%)	0 (0%)	
L	8 (1.6%)	6 (1.5%)	2 (2.2%)	0.12	0 (0%)	2 (2.6%)	1.00	0 (0%)	1 (3.7%)	0.75	1 (2.4%)	0 (0%)	1.00
N	491 (97%)	396 (98%)	87 (95%)		15 (100%)	72 (94%)		36 (95%)	25 (93%)		40 (98%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Magnesium													
H	2 (0.4%)	2 (0.5%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
L	1 (0.2%)	0 (0%)	1 (1.1%)	0.21	0 (0%)	1 (1.3%)	1.00	1 (2.6%)	0 (0%)	1.00	1 (2.4%)	0 (0%)	1.00
N	503 (99%)	404 (100%)	91 (99%)		15 (100%)	76 (99%)		37 (97%)	27 (100%)		40 (98%)	28 (100%)	
Missing	28	18	10		4	6		3	3		0	2	
Phosphate													
H	13 (2.6%)	8 (2.0%)	5 (5.4%)		1 (6.7%)	4 (5.2%)		2 (5.3%)	2 (7.4%)		2 (4.9%)	2 (7.1%)	
L	53 (10%)	46 (11%)	6 (6.5%)	0.08	0 (0%)	6 (7.8%)	0.66	3 (7.9%)	3 (11%)	0.88	4 (9.8%)	5 (18%)	0.56
N	440 (87%)	352 (87%)	81 (88%)		14 (93%)	67 (87%)		33 (87%)	22 (81%)		35 (85%)	21 (75%)	
Missing	28	18	10		4	6		3	3		0	2	

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
Uric acid													
H L N Missing	29 (5.7%) 59 (12%) 418 (83%) 28	22 (5.4%) 48 (12%) 336 (83%) 18	7 (7.6%) 10 (11%) 75 (82%) 10	0.71	3 (20%) 3 (20%) 9 (60%) 4	4 (5.2%) 7 (9.1%) 66 (86%) 6	**0.03**	1 (2.6%) 4 (11%) 33 (87%) 3	4 (15%) 2 (7.4%) 21 (78%) 3	0.21	1 (2.4%) 7 (17%) 33 (80%) 0	4 (14%) 2 (7.1%) 22 (79%) 2	0.11
Triglycerides													
H L N Missing	26 (20%) 0 (0%) 102 (80%) 406	20 (20%) 0 (0%) 81 (80%) 323	4 (17%) 0 (0%) 20 (83%) 78	1.00	0 (0%) 0(0%) 3 (100%) 16	4 (19%) 0 (0%) 17 (81%) 62	1.00	3 (27%) 0 (0%) 8 (73%) 30	1 (20%) 0 (0%) 4 (80%) 25	1.00	3 (10%) 0 (0%) 26 (90%) 12	5 (26%) 0 (0%) 14 (74%) 11	0.24
Fasting triglycerides													
H L N Missing	44 (12%) 0 (0%) 334 (88%) 156	39 (13%) 0 (0%) 266 (87%) 119	5 (7.4%) 0 (0%) 63 (93%) 34	0.21	1 (8.3%) 0 (0%) 11 (92%) 7	4 (7.1%) 0 (0%) 52 (93%) 27	1.00	2 (7.4%) 0 (0%) 25 (93%) 14	1 (4.5%) 0 (0%) 21 (95%) 8	1.00	1 (8.3%) 0 (0%) 11 (92%) 29	0 (0%) 0 (0%) 9 (100%) 21	1.00
Cholesterol													
H L N Missing	68 (53%) 0 (0%) 60 (47%) 406	54 (53%) 0 (0%) 47 (47%) 323	13 (54%) 0 (0%) 11 (46%) 78	0.95	1 (33%) 0 (0%) 2 (67%) 16	12 (57%) 0 (0%) 9 (43%) 62	0.58	5 (45%) 0 (0%) 6 (55%) 30	3 (60%) 0 (0%) 2 (40%) 25	1.00	10 (34%) 1 (3.4%) 18 (62%) 12	8 (42%) 0 (0%) 11 (58%) 11	0.86
Fasting cholesterol													
H L N Missing	167 (44%) 0 (0%) 211 (56%) 156	143 (47%) 0 (0%) 162 (53%) 119	24 (35%) 0 (0%) 44 (65%) 34	0.08	4 (33%) 0 (0%) 8 (67%) 7	20 (36%) 0 (0%) 36 (64%) 27	1.00	8 (30%) 0 (0%) 19 (70%) 14	8 (36%) 0 (0%) 14 (64%) 8	0.62	9 (75%) 0 (0%) 3 (25%) 29	5 (56%) 0 (0%) 4 (44%) 21	0.40
HDL cholesterol													
H L N Missing	176 (35%) 40 (7.9%) 290 (57%) 28	141 (35%) 31 (7.6%) 234 (58%) 18	31 (34%) 8 (8.7%) 53 (58%) 10	0.94	6 (40%) 1 (6.7%) 8 (53%) 4	25 (32%) 7 (9.1%) 45 (58%) 6	0.91	13 (34%) 5 (13%) 20 (53%) 3	7 (26%) 3 (11%) 17 (63%) 3	0.72	12 (29%) 2 (4.9%) 27 (66%) 0	6 (21%) 5 (18%) 17 (61%) 2	0.22
LDL cholesterol													
H L N Missing	166 (33%) 0 (0%) 332 (67%) 36	137 (34%) 0 (0%) 264 (66%) 23	28 (31%) 0 (0%) 61 (69%) 13	0.63	5 (36%) 0 (0%) 9 (64%) 5	23 (31%) 0 (0%) 52 (69%) 8	0.71	8 (22%) 0 (0%) 28 (78%) 5	9 (35%) 0 (0%) 17 (65%) 4	0.28	15 (37%) 0 (0%) 26 (63%) 0	9 (32%) 0 (0%) 19 (68%) 2	0.70
Iron													

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
H	24 (4.7%)	19 (4.7%)	4 (4.3%)	1.00	0 (0%)	4 (5.2%)	1.00	3 (7.9%)	1 (3.7%)	0.78	1 (2.4%)	0 (0%)	1.00
L	10 (2.0%)	8 (2.0%)	2 (2.2%)		0 (0%)	2 (2.6%)		1 (2.6%)	0 (0%)		2 (4.9%)	2 (7.1%)	
N	472 (93%)	379 (93%)	86 (93%)		15 (100%)	71 (92%)		34 (89%)	26 (96%)		38 (93%)	26 (93%)	
Missing	28	18	10		4	6		3	3		0	2	
TIBC													
H	19 (3.8%)	15 (3.7%)	3 (3.3%)	1.00	0 (0%)	3 (4.0%)	1.00	2 (5.4%)	0 (0%)	0.51	1 (2.4%)	0 (0%)	0.64
L	1 (0.2%)	1 (0.2%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	1 (3.7%)	
N	479 (96%)	385 (96%)	87 (97%)		15 (100%)	72 (96%)		35 (95%)	26 (100%)		40 (98%)	26 (96%)	
Missing	35	23	12		4	8		4	4		0	3	
Transferrin saturation													
H	9 (1.8%)	8 (2.0%)	1 (1.1%)	0.64	0 (0%)	1 (1.3%)	1.00	1 (2.7%)	0 (0%)	0.86	0 (0%)	0 (0%)	1.00
L	78 (16%)	60 (15%)	17 (19%)		3 (20%)	14 (19%)		7 (19%)	6 (23%)		6 (15%)	3 (11%)	
N	412 (83%)	333 (83%)	72 (80%)		12 (80%)	60 (80%)		29 (78%)	20 (77%)		35 (85%)	24 (89%)	
Missing	35	23	12		4	8		4	4		0	3	
CRP highly sensitive													
H	37 (7.3%)	33 (8.1%)	4 (4.3%)	0.21	1 (6.7%)	3 (3.9%)	0.52	0 (0%)	2 (7.4%)	0.17	2 (4.9%)	2 (7.1%)	1.00
L	0 (0%)	0 (0%)	0 (0%)		0 (0%)	0 (0%)		0(0%)	0 (0%)		0 (0%)	0 (0%)	
N	468 (93%)	372 (92%)	88 (96%)		14 (93%)	74 (96%)		38 (100%)	25 (93%)		39 (95%)	26 (93%)	
Missing	29	19	10		4	6		3	3		0	2	
Troponin I highly sensitive													
H	4 (0.9%)	4 (1.1%)	0 (0%)	1.00	0 (0%)	0 (0%)	1.00	0 (0%)	0 (0%)	1.00	0 (0%)	0 (0%)	1.00
L	0 (0%)	0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)		0 (0%)	0 (0%)	
N	458(99%)	368 (99%)	83 (100%)		18 (100%)	65 (100%)		33 (100%)	24 (100%)		32 (100%)	24 (100%)	
Missing	72	52	19		1	18		8	6		9	6	
Amylase													
H	34 (7.3%)	23 (6.2%)	8 (9.6%)	**0.03**	2 (11%)	6 (9.2%)	0.73	3 (9.1%)	3 (12%)	0.84	3 (9.4%)	4 (17%)	0.82
L	10 (2.2%)	5 (1.3%)	5 (6.0%)		0 (0%)	5 (7.7%)		1 (3.0%)	1 (4.2%)		1 (3.1%)	0 (0%)	
N	418 (91%)	344 (92%)	70 (84%)		16 (89%)	54 (83%)		29 (88%)	20 (83%)		28 (88%)	20 (83%)	
Missing	72	52	19		1	18		8	6		9	6	
Ferritin													
H	61 (13.2%)	48 (13%)	13 (16%)	0.76	2 (11%)	11 (17%)	0.78	6 (18%)	3 (12%)	0.72	6 (19%)	4 (17%)	1.00
L	11 (2.4%)	10 (2.7%)	1 (1.2%)		0 (0%)	1 (1.5%)		0 (0%)	0 (0%)		1 (3.1%)	0 (0%)	
N	390 (84%)	314 (84%)	69 (83%)		16 (89%)	53 (82%)		27 (82%)	21 (88%)		25 (78%)	20 (83%)	
Missing	72	52	19		1	18		8	6		9	6	
Lipase													
H	36 (7.7%)	29 (7.7%)	5 (5.9%)	0.88	1 (5.6%)	4 (6.0%)	0.26	2 (5.9%)	2 (8.0%)	1.00	3 (9.4%)	1 (4.2%)	0.63

	6 months										12 months		
	COVID, N=534	No CMR abnormalities, N = 424	CMR abnormalities, N = 102	P	CMR abnormalities hospitalised, N = 19	CMR abnormalities non hospitalized, N = 83	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P	Ongoing CMR abnormalities, N = 41	Resolved CMR abnormalities, N = 30	P
L N Missing	5 (1.1%) 425 (91%) 68	4 (1.1%) 341 (91%) 50	1 (1.2%) 79 (93%) 17		1 (5.6%) 16 (89%) 1	0 (0%) 63 (94%) 16		0 (0%) 32 (94%) 7	0 (0%) 23 (92%) 5		0 (0%) 29 (91%) 9	0 (0%) 23 (96%) 6	
Thyroid stimulating hormone													
H L N Missing	3 (0.6%) 0 (0%) 464 (99%) 67	3 (0.8%) 0 (0%) 372 (99%) 49	0 (0%) 0 (0%) 85 (100%) 17	1.00	0 (0%) 0 (0%) 18 (100%) 1	0 (0%) 0 (0%) 67 (100%) 16	1.00	0 (0%) 0 (0%) 34 (100%) 7	0 (0%) 0 (0%) 25 (100%) 5	1.00	1 (3.1%) 0 (0%) 31 (97%) 9	1 (4.2%) 0 (0%) 23 (96%) 6	1.00
Testosterone													
H L N Missing	19 (4.1%) 9 (1.9%) 434 (94%) 72	17 (4.6%) 6 (1.6%) 349 (94%) 52	1 (1.2%) 3 (3.6%) 79 (95%) 19	0.30	0 (0%) 3 (17%) 15 (83%) 1	1 (1.5%) 0 (0%) 64 (98%) 18	**0.01**	0 (0%) 1 (3.0%) 32 (97%) 8	0 (0%) 1 (4.2%) 23 (96%) 6	1.00	0 (0%) 2 (6.2%) 30 (94%) 9	0 (0%) 2 (8.3%) 22 (92%) 6	1.00
Insulin													
H L N Missing	41 (8.9%) 10 (2.2%) 408 (89%) 75	32 (8.6%) 7 (1.9%) 332 (89%) 53	8 (9.9%) 3 (3.7%) 70 (86%) 21	0.59	2 (12%) 0 (0%) 15 (88%) 2	6 (9.4%) 3 (4.7%) 55 (86%) 19	1.00	3 (9.4%) 1 (3.1%) 28 (88%) 9	4 (17%) 0 (0%) 19 (83%) 7	0.68	3 (9.4%) 0 (0%) 29 (91%) 9	3 (12%) 0 (0%) 21 (88%) 6	1.00
C peptide													
H L N Missing	19 (4.1%) 0 (0%) 443 (96%) 72	16 (4.3%) 0 (0%) 357 (96%) 51	2 (2.4%) 0 (0%) 80 (98%) 20	0.75	1 (5.9%) 0 (0%) 16 (94%) 2	1 (1.5%) 0 (0%) 64 (98%) 18	0.37	2 (6.1%) 0 (0%) 31 (94%) 8	0 (0%) 0 (0%) 23 (100%) 7	0.51	3 (9.4%) 0 (0%) 29 (91%) 9	3 (12%) 0 (0%) 21 (88%) 6	1.00
NT-proBNP													
H L N Missing	2 (0.4%) 0 (0%) 460 (99%) 72	1 (0.3%) 0 (0%) 371 (99%) 52	1 (1.2%) 0 (0%) 82 (99%) 19	0.45	1 (5.6%) 0 (0%) 17 (94%) 1	0 (0%) 0 (0%) 65 (100%) 18	0.22	0 (0%) 0 (0%) 33 (100%) 8	1 (4.2%) 0 (0%) 23 (96%) 6	0.42	0 (0%) 0 (0%) 32 (100%) 9	0 (0%) 0 (0%) 24 (100%) 6	1.00

Values presented as count and %. Cells with red shading indicating significant differences

Abbreviations: H, high; L, Low; N, Normal range; HCT, haematocrit test; MCV, Mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RDW, red cell distribution width; MPV, mean platelet volume; ESR, erythrocyte sedimentation rate; LDH, Lactate dehydrogenase; CK, Creatine Kinase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TIBC, total iron-binding capacity; CRP, C-reactive protein; NT-proBNP, N-terminal pro B-type natriuretic peptide.

Figure S5: T1 topographical abnormalities in participants classified as CMR abnormal at 6 months (n=102)

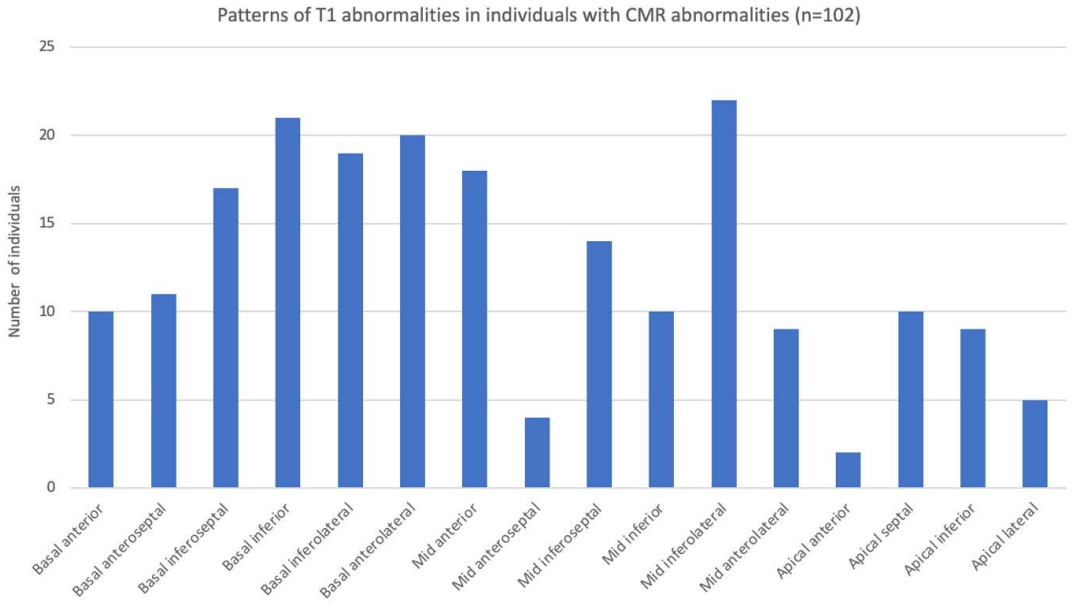

Table S6. Detailed CMR findings in new onset cardiac abnormalities at 12 months. Red cells indicate abnormal values

		A	B	C	D	E	F	G	H	I	J
Field Strength		1.5T	1.5T	1.5T	1.5T	1.5T	3T	1.5T	1.5T	3T	3T
sex		F	F	F	M	M	F	F	M	M	F
Age range		56-60	41-45	46-50	51-55	66-70	46-50	46-50	56-60	30-35	30-35
Global T1	baseline	1018	968	976	985	952	1219	1001	942	1141	1182
	follow up	1025	1018	1024	998	988	1278	998	934	1170	1238
≥ 3 elevated T1 segments	baseline	No	No	No	No	No	No	No	No	No	No
	follow up	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No	Yes
Global T2	follow up	48	NA	47	47	47	45	NA	46	NA	NA
≥ 3 elevated T2 segments	follow up	No	No	No	No	No	Yes	No	No	No	No
Left end diastolic volume (mL)	baseline	65	81	80	89	81	90	75	117	80	73
	follow up	64	91	83	78	86	91	68	103	79	54
Left end systolic volume (mL)	baseline	27	29	34	35	28	34	29	49	34	28
	follow up	23	31	31	36	33	38	30	49	35	27
Left ejection fraction (%)	baseline	58	64	58	61	65	63	62	58	57	62
	follow up	64	66	63	55	61	58	56	53	55	51
Left stroke volume (mL)	baseline	38	52	46	54	53	57	47	68	46	45
	follow up	41	60	52	42	53	53	38	54	44	27
Left ventricular max wall thickness (mm)	baseline	10	9	7	9	9	9	8	10	10	9
	follow up	11	8	8	12	9	7	9	12	8	11
Left ventricular muscle mass (mm)	baseline	79	79	61	99	84	56	63	134	74	83
	follow up	89	75	65	118	82	58	67	149	70	92
Left global circumferential strain (%)	baseline	-20	-25	-24	-23	-21	-20	-24	-19	-20	-23
	follow up	-21	-25	-24	-19	-20	-22	NA	-17	-19	-19
Left global longitudinal strain (%)	baseline	-13	-18	-14	-15	-15	-15	-16	-9	-14	-16
	follow up	-13	-17	-18	-16	-12	-17	NA	-10	-13	-14
Right end diastolic volume (mL)	baseline	68	79	80	86	94	90	77	132	75	77
	follow up	62	81	78	56	98	92	71	130	86	56
Right end systolic volume (mL)	baseline	31	28	34	32	39	40	31	60	33	28
	follow up	31	28	31	23	41	41	37	71	44	28
Right ejection fraction (%)	baseline	54	64	58	63	58	56	59	55	56	63
	follow up	50	65	60	58	58	55	48	45	49	49
Right stroke volume (mL)	baseline	36	51	46	54	55	50	46	72	42	48
	follow up	31	52	47	33	57	51	34	59	42	27

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