CASE REPORT

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COVID-19-induced silent myocarditis and newly developed hypertension in a 3-year-old boy

Elaheh Malakan Rad[1*] and Sara Momtazmanesh[2]

Abstract

Background: COVID-19 myocarditis occurs in 7–28% of patients admitted in the hospital with or without multisystem inflammatory syndrome. It may present as fulminant myocarditis. Dilated cardiomyopathy as a sequela of COVID-19 myocarditis has been reported in the pediatric population. However, to date, no case of silent COVID-19 myocarditis progressing to dilated cardiomyopathy has been reported in children. Furthermore, although newly developed hypertension as a sequela of COVID-19 infection has been reported in adults, there is no report of newly developed COVID-induced hypertension in children. We report a 3-year-old boy with silent COVID-19 myocarditis progressing to dilated cardiomyopathy and newly developed systemic hypertension.

Case presentation: A 3-year-old boy was referred to the emergency department because of respiratory distress. The parents gave a history of SARS-CoV-2 infection in the child 5 months ago that was manifested as fever and cough, for which he was treated as an outpatient. Echocardiographic examination revealed a severe decrease in left ventricular systolic function in favor of dilated cardiomyopathy. Cardiac magnetic resonance imaging established the diagnosis of myocarditis. The patient left ventricular systolic function did not improve after 2 weeks of intravenous inotropic support. Therefore, the child was transferred to another tertiary center with extracorporeal membrane oxygenation and pediatric cardiac transplantation facilities.

Conclusions: COVID-19 can induce silent myocarditis with progression to dilated cardiomyopathy and newly developed systemic hypertension. Thus, a thorough examination of the heart and measurement of blood pressure are mandatory in every child with COVID-19 infection. Cardiac MR is an indispensable tool in the diagnosis, follow-up, and prognostication of COVID-19 myocarditis. Moreover, four-chamber speckle tracking strain imaging showed apical rocking in all the four heart chambers in this child with opposite direction in the failed left ventricle compared with other cardiac chambers. Lastly, the presence of septal flash on M-mode echocardiography, apical rocking and prestretch–rebound stretch patterns on longitudinal strain imaging of the failed left ventricle in this child may be of predictive value for response to cardiac resynchronization therapy.

Keywords: COVID-19, Myocarditis, Dilated cardiomyopathy, Strain imaging, Speckle-tracking, Echocardiography, Magnetic resonance imaging, Hypertension, Case report

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Background

COVID-19 myocarditis occurs in 7–28% of patients admitted in the hospital with or without multisystem inflammatory syndrome (MIS) [1]. It may present as fulminant myocarditis [2]. Dilated cardiomyopathy as a sequela of COVID-19 myocarditis has been reported in the pediatric population [3]. However, to date, no case

of silent COVID-19 myocarditis progressing to dilated cardiomyopathy has been reported in children. Furthermore, although newly developed hypertension as a sequela of COVID-19 infection has been reported in adults, there is no report of newly developed COVID-induced hypertension in children [4]. We report a 3-year-old boy with silent COVID-19 myocarditis progressing to dilated cardiomyopathy and newly developed systemic hypertension. We also present the findings on speckle-tracking strain imaging echocardiography of the four chambers of the heart in this patient.

Case presentation

A three-year-old was referred to the emergency department because of respiratory distress. He was a well-nourished boy with normal temperature and a respective heart rate and respiratory rate of 140 beats and 63 per minute on physical examination. His blood pressure was 135/110 mmHg. Cardiac auscultation revealed muffled heart sounds with a grade 2/6 regurgitant systolic murmur at the cardiac apex. The rest of the physical examination was unremarkable. The parents gave a history of SARS-CoV-2 infection in the child five months ago that was manifested as fever and cough, for which he was treated as an outpatient. Furthermore, he had a history of complete cardiac evaluation for a cardiac murmur, including a comprehensive echocardiographic examination by a pediatric cardiologist one year ago, which confirmed a normal structure and function and the innocent nature of the murmur. Family history was negative except a COVID-19 infection in five months ago in other family members.

Cardiomegaly, pulmonary edema, and blunted costophrenic angles were evident on the chest X-ray (Fig. 1). The electrocardiogram showed T wave inversion in left precordial leads (Fig. 2).

Comprehensive two-dimensional color Doppler echocardiography revealed a severe decrease in left ventricular systolic function in favor of dilated cardiomyopathy, significant left ventricular enlargement, mild to moderate holosystolic mitral regurgitation with eccentric posterolateral jet, trivial aortic regurgitation, and septal flash (Fig. 3). Left ventricular ejection fraction by Simpson's method was 26%, and tricuspid annular plane systolic excursion (TAPSE) was 21.8 mm. There was no coarctation of the aorta.

Speckle-tracking longitudinal, circumferential, radial and transverse atrial and ventricular strain imaging was performed using velocity vector imaging (VVI) of Siemens Accuson SC 2000 (Siemens Medical Solutions USA, Inc.) (Figs. 4, 5, 6, 7, Additional files 1, 2, 3, 4, 5, 6, 7, 8, and 9: Movie clips 1–9). Compared with normal pediatric values, left ventricular longitudinal,

Fig. 1 Chest X-ray of the patient showing cardiomegaly and pulmonary congestion
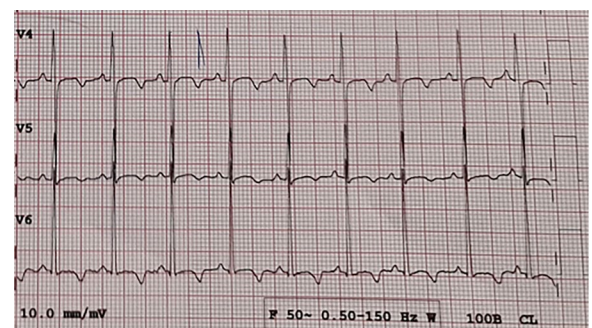

Fig. 2 T wave inversion in left precordial leads of the electrocardiogram of the patient
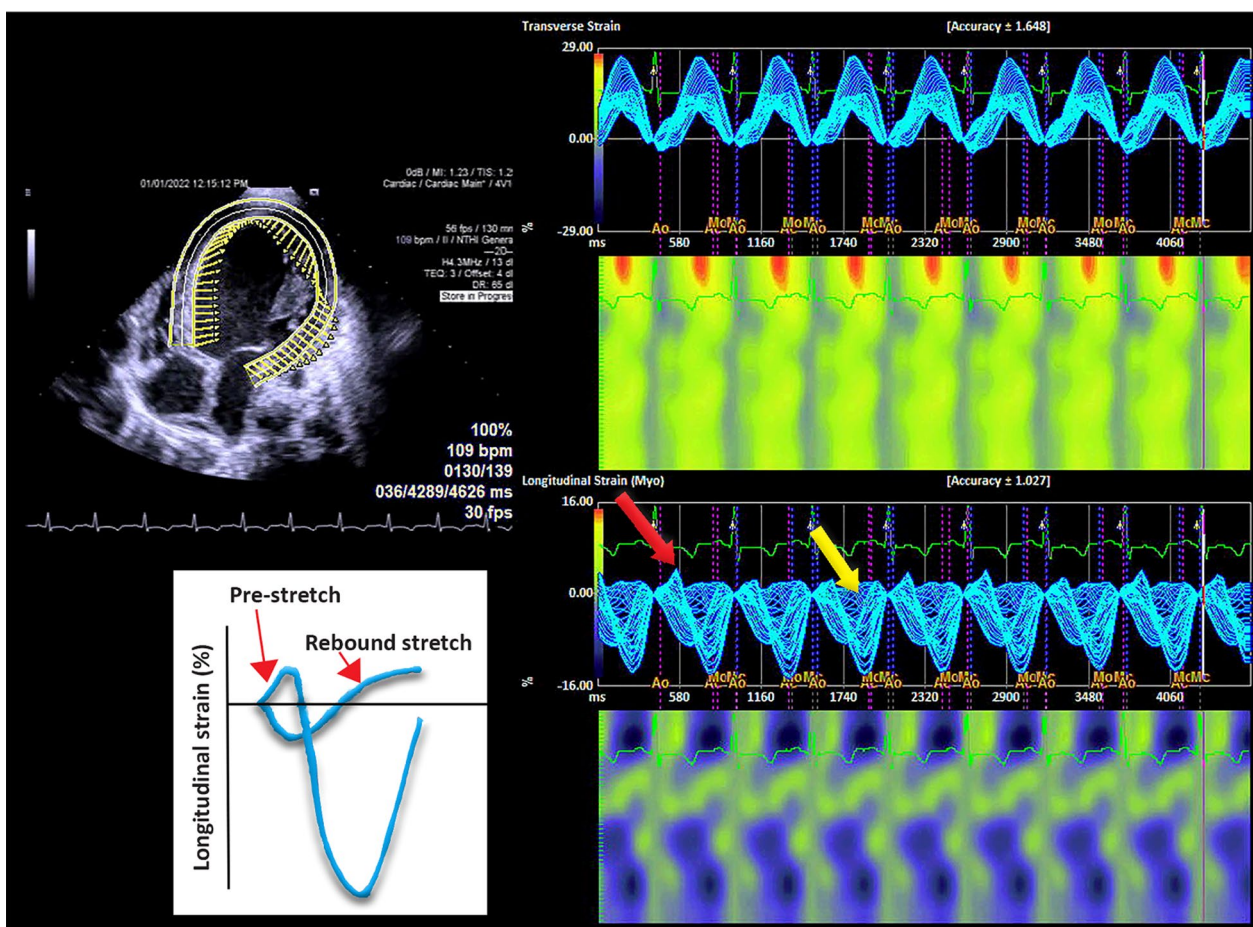
transverse, circumferential, and radial strain were decreased [5–8]. Furthermore, there was a prestretch and rebound stretch pattern in the longitudinal strain of the left ventricle. Notably, apical rocking was detected in all four heart chambers with the direction of the apical movement in LV opposite to the rocking in the right ventricle, right atrium, and left atrium (Additional files 1, 2, 3, 4: Movie Clips 1 to 4). The results of the speckle-tracking strain imaging are depicted in Table 1.

According to the classification of Singh et al. for categorization of LV diastolic function based on the left atrial strain imaging, the patient had grade 3 LV diastolic dysfunction [9].
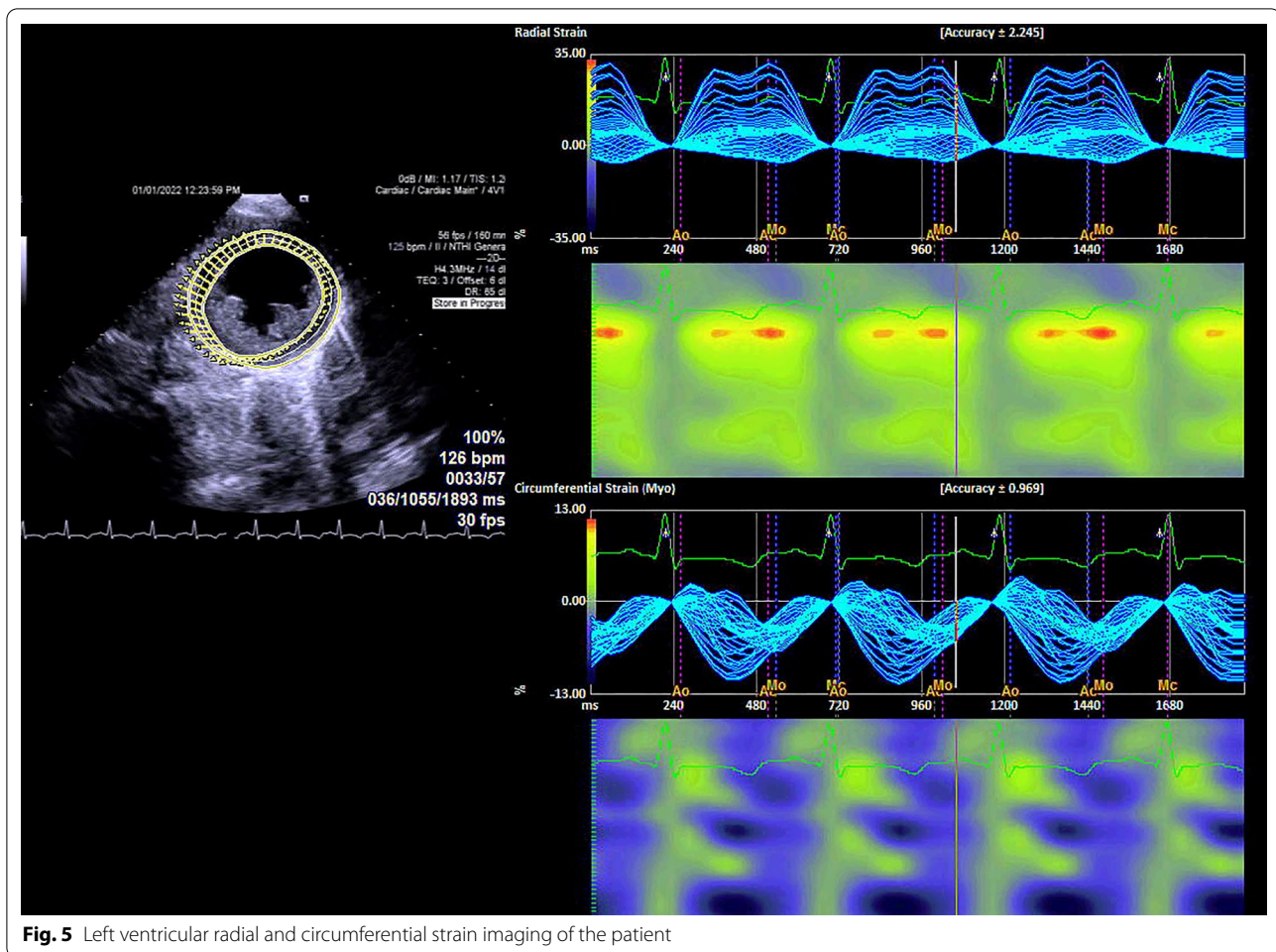
Given the past medical history of confirmed COVID-19 infection in the past months and normal echocardiogram in the last year, the clinical diagnosis of silent
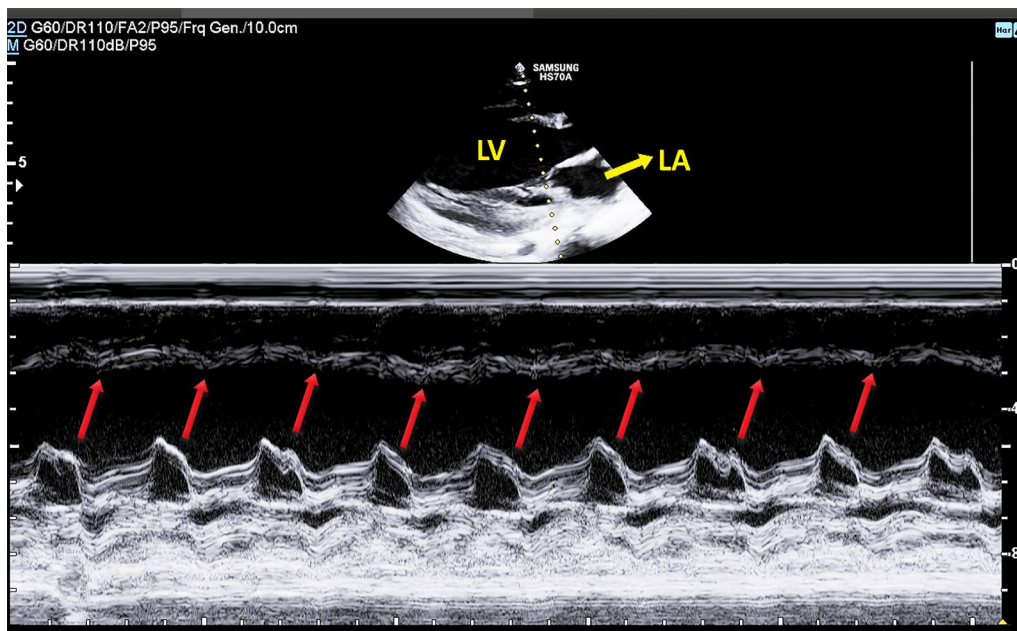
Fig. 3 Septal flash on M-mode echocardiography of the patient

Fig. 4 Left ventricular transverse and longitudinal strain imaging of the patient

Fig. 5 Left ventricular radial and circumferential strain imaging of the patient

and undiagnosed COVID-19 myocarditis was suspected. COVID-19 reverse transcription-polymerase chain reaction (RT-PCR) and SARS-CoV-2 IgG and IgM antibodies were negative. Similarly, the polymerase chain reaction for other viruses, including adenovirus, enterovirus, Coxsackievirus, Epstein-Barr virus, Influenza virus, Cytomegalovirus, Human Immunodeficiency Virus, Hepatitis B and C virus, Herpes viruses were negative. The level of troponin I was normal, but NT-proBNP was elevated.

To look for Lake Louise criteria of myocarditis, cardiac magnetic resonance (CMR) imaging was performed with gadolinium study and Short Tau Inversion Recovery (STIR)/T2 weighted-sequences [10]. This evaluation revealed mild pericardial effusion, severely enlarged left ventricle without left ventricular hypertrophy, evidence of diffuse myocardial edema, and severely reduced systolic function. Left ventricular and right ventricular ejection fractions were 13% and 45%, respectively, on cardiac MRI. Late-gadolinium enhancement was observed in the left ventricle's basal and mid-posterolateral segments in favor of post-myocarditis scarring (Fig. 8).

Complete workup including endocrinologic, nephrologic, neurologic, and rheumatologic causes of hypertension was performed. The only abnormal finding was significantly elevated renin (> 550 micro IU/mL, normal levels at supine position: 4.2–59.7 micro IU/mL). Measurement of aldosterone and angiotensin II was not available. Kidney function and Doppler ultrasound of renal arteries and veins were normal. Cortisol and ACTH were normal. The absence of left ventricular hypertrophy on CMR and echocardiography, absence of hypertension in the past medical history of the patient, and negative workup for other causes of hypertension were in favor of COVID-19-induced newly developed systemic hypertension.

Intravenous infusion of Dopamine (7.5 μg/kg/min), dobutamine (7.5 μg/kg/min), and milrinone (0.6 μg/kg/min), in conjunction with aspirin at a dose of 5 mg/kg/day, were administered for the patient. The patient's hypertension was controlled by the administration
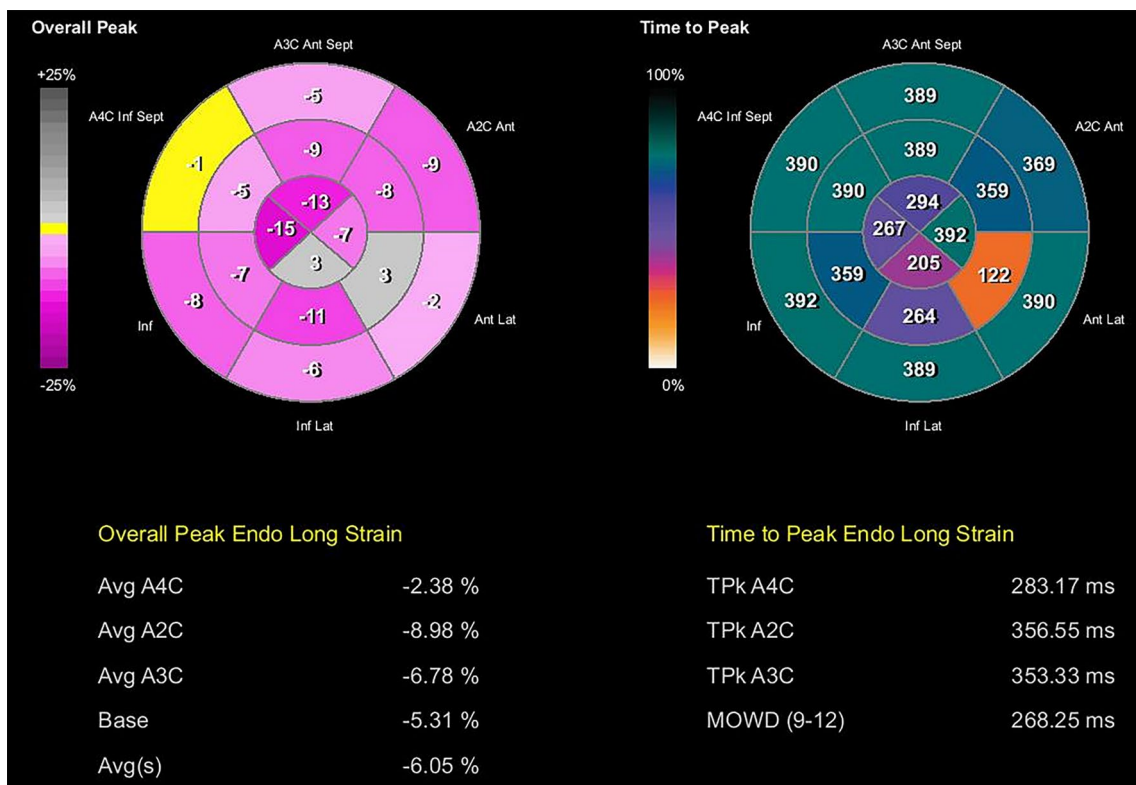
Fig. 6 Bull's eye plot of left ventricular longitudinal strain and time to peak longitudinal strain
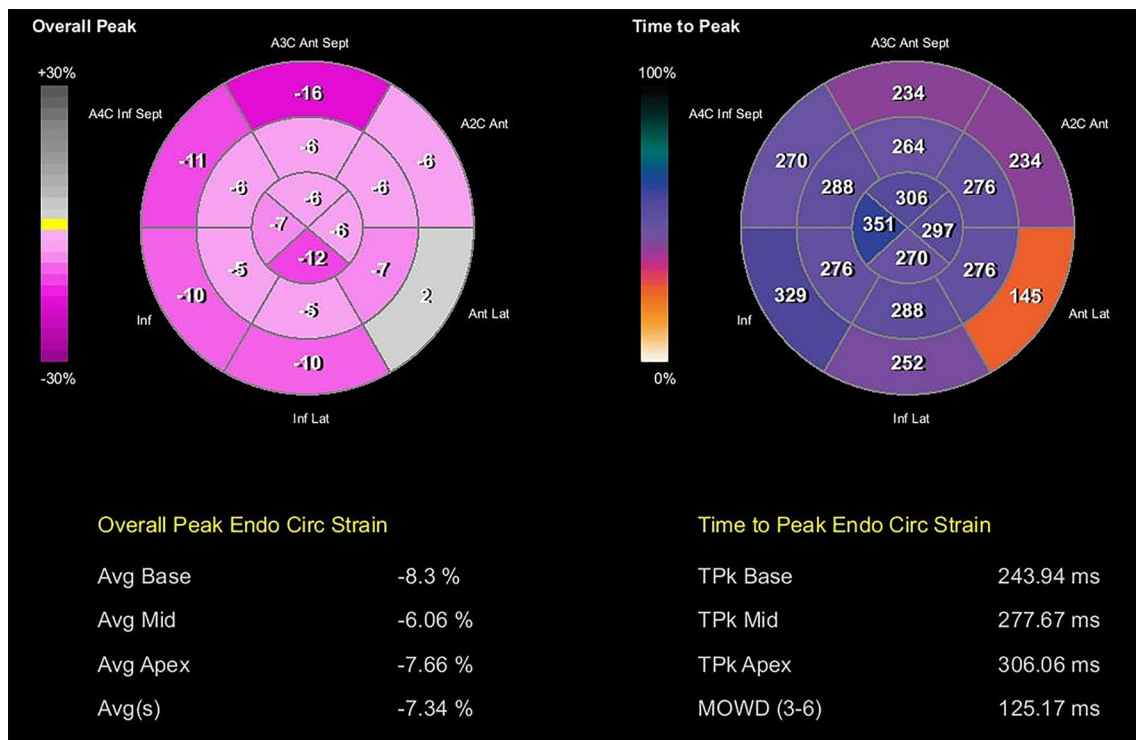
Fig. 7 Bull's eye plot of left ventricular circumferential strain and time to peak circumferential strain

Table 1 Results of speckle-tracking strain imaging of the atria and ventricles

Parameters	Right atrium	Left atrium	Right ventricle	Left ventricle
Ejection fraction %	49%	65%	–	20%
End-diastolic volume (mL)	13	13.2	–	103
End-systolic volume (mL)	6.4	4.5	–	82
Fractional area change (%)	–	–	54.2	17.9
Maximal area (cm2)	–	–	3.2	23.2
Minimal area (cm2)	–	–	1.4	19
Peak global longitudinal strain (%)	− 60%	− 10%	–	− 6.60
Average of peak segmental transverse myocardial strain ± standard deviation (SD)	− 22.50 ± 6.84	− 23.50 ± 6.27	30.5 ± 50.36	15.6 ± 5.57
Average of peak segmental longitudinal myocardial strain ± SD	12.60 ± 5.31	11.40 ± 2.48	− 5.50 ± 15.19	− 7.10 ± 3.57
Average of peak segmental radial strain ± SD	–	–	–	6.8 ± 6.75
Average of peak segmental myocardial circumferential strain ± SD	–	–	–	− 5.80 ± 1.87
Average of peak segmental transverse myocardial strain rate ± SD	2.4 ± 1.39	2.50 ± 1.07	1.70 ± 1.56	0.90 ± 0.22
Average of peak segmental longitudinal myocardial strain rate ± SD	− 0.80 ± 0.26	− 1.10 ± 0.36	− 1.20 ± 0.36	− 0.50 ± 0.20
Average of peak segmental radial myocardial strain rate ± SD	–	–	–	0.60 ± 0.22
Average of peak segmental circumferential myocardial strain rate ± SD	–	–	–	− 0.40 ± 0.16
Average of time to peak segmental myocardial transverse strain ± SD	–	–	227.80 ± 92.27	329 ± 76.28
Average of time to peak segmental myocardial longitudinal strain ± SD	–	–	266.70 ± 88.65	349.30 ± 88.38
Average of time to peak segmental radial strain ± SD	–	–	–	284.70 ± 87.41
Average of time to peak segmental myocardial circumferential strain ± SD	–	–	–	254.80 ± 41.86
Maximal opposing wall delay for transverse strain	0	18	161	125
Maximal opposing wall delay for longitudinal strain	0	35	232	71
Maximal opposing wall delay for transverse strain rate	36	36	196	36
Maximal opposing wall delay for longitudinal strain rate	107	0	304	107
Apical rocking -epicardial layer	+ 0.39	+ 0.28	–	− 0.05
Apical rocking -myocardial layer	+ 0.61	+ 0.12	+ 0.84	− 0.23
Apical rocking -endocardial layer	+ 0.72	+ 0.22	+ 0.29	− 0.17

of captopril (6 mg/kg/day every 6 h) and hydralazine (0.2 mg/kg/dose every 4 h). The patient left ventricular systolic function did not improve after 2 weeks of intravenous inotropic support. Therefore, the child was transferred to another tertiary center with extracorporeal membrane oxygenation and pediatric cardiac transplantation facilities.

Discussion

This is the first report of silent myocarditis and newly developed systemic hypertension after SARS-CoV-2 infection in a child.

Silent myocarditis has been reported in several diseases such as Takayasu arteritis, myasthenia gravis, and systemic sclerosis [11–13]. However, there is no report of silent myocarditis following viral infections, let alone COVID-19 disease, to the best of our knowledge.

COVID-19 infection in children is often milder than in adults [14]. Fever and cough are the most common manifestations in children aged 0–19 years [15]. In the meta-analysis of 9335 COVID-19 patients ≤ 19 years, 13% (as mean proportion) of children were asymptomatic [15]. The risk for the development of myocarditis during SARS-CoV-2 infection has been reported to be 0.146% [16].

Furthermore, the similarity of pulmonary and cardiac involvement manifestations makes distinguishing the cardiac from the pulmonary causes challenging. The associated fever and cough in this child and the child's young age that prevents communication of symptoms further distract the primary care physician of attention to the cardiac problem.

Moreover, while Chen et al. [4] reported newly developed hypertension as a consequence of SARS-CoV-2 infection in about 8% of the 190 patients, aged 40–86 years, this sequelae has not been reported in children before. The absence of left ventricular hypertrophy, the patient's past medical history, and the completely negative workup for cardiologic, endocrinologic, nephrologic, and neurologic causes of systemic hypertension and the significantly elevated renin level were in favor of COVID-19-induced systemic hypertension (CISH). Similar to this study, renin was elevated in our patient. This newly developed hypertension may be due to increased
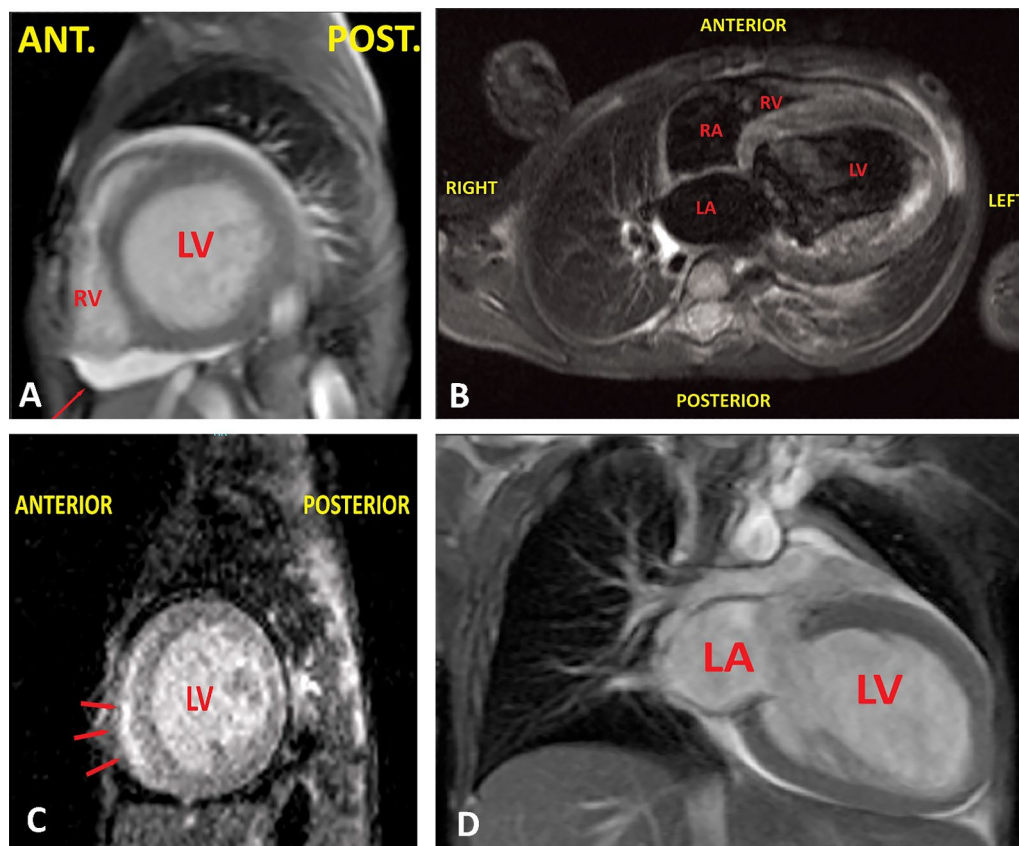
Fig. 8 Cardiac magnetic resonance imaging of the patient. **A** Pericardial effusion, **B** indicates diffuse myocardial edema, **C** depicts late gadolinium enhancement, and **D** shows a severe enlargement of the left ventricle

signaling of angiotensin II by SARS-CoV-2 [4]. Genetic characteristics may play a role in various clinical features of COVID-19 infection [17, 18].

Although we performed a comprehensive strain analysis including longitudinal, circumferential, radial, and transverse strain imaging in this patient, transverse and radial strains are reported to be less reliable [5, 7].

The presence of four-chamber-apical rocking with opposite direction of rocking in the failed left ventricle compared to the three other cardiac chambers has not been reported before. Apical rocking or transverse motion of the apex is recognized as a marker of left ventricular dyssynchrony. It has been reported as a parameter that predicts response to cardiac resynchronization therapy [19–22]. We may speculate that all three other chambers are working together to compensate for abnormal left ventricular apical rocking.

Moreover, his child had prestretch and rebound stretch in the left ventricular longitudinal strain imaging. Gorcsan et al., in a study on 422 patients with heart failure, reported the presence of systolic stretch as an indicator of favorable response to cardiac resynchronization therapy

in patients with a QRS duration of 120 149 ms or absent left bundle branch block [23]. Thus, the septal flash, apical rocking, and the peculiar strain pattern in this patient may predict a potential favorable response to CRT [22].

Evaluation of left atrial strain is a necessity in patients with heart failure. It provides information regarding the left atrium's reservoir, conduit, and booster function and is considered a parameter for assessing left ventricular diastolic function. Left atrial reservoir strain of less than 23% is abnormal [9, 24–28].

Cardiac magnetic resonance imaging is a robust gold standard tool for diagnosis, follow-up, and prognostication of myocarditis in acute, chronic, and healed states [29, 30]. The diagnosis of definite myocarditis was established for this patient by fulfilling the two major (myocardial edema and myocardial injury) and the two minor (pericarditis and left ventricular systolic dysfunction) criteria of the updated Lake Louise criteria [10]. Georgiopoulos and colleagues reported that late gadolinium enhancement (LGE) and anteroseptal location portend a worse prognosis in patients with acute myocarditis [31]. Myocardial edema may be seen in acute and chronic

myocarditis. However, the scar is a feature of healed myocarditis [10]. Furthermore, persistent myocardial edema has been reported in 25% of patients with COVID-19 myocarditis [1].

Conclusions

This is the first case report that indicates COVID-19 can induce silent myocarditis with progression to dilated cardiomyopathy and newly developed systemic hypertension. Accordingly, this case encompasses several critical lessons for primary care physicians and pediatric cardiologists. First and foremost, a thorough examination of the heart and measurement of blood pressure are mandatory in every child with COVID-19 infection. Second, the role of cardiac MR with appropriate sequences in the diagnosis, follow-up, and prognostication of myocarditis should not be underestimated. Third, four-chamber speckle tracking strain imaging showed apical rocking in all the four chambers of the heart in this child with secondary dilated cardiomyopathy with opposite direction in the failed left ventricle compared with other cardiac chambers. Fourth, the presence of septal flash on M-mode echocardiography, apical rocking, and prestretch–rebound stretch patterns on longitudinal strain imaging of the left ventricle may be of predictive value for response to CRT in these patients.

Abbreviations
CMR: Cardiac magnetic resonance imaging; CRT: Cardiac resynchronization therapy; LA: Left atrium; LV: Left ventricle; RA: Right atrium; RV: Right ventricle.

Supplementary Information
The online version contains supplementary material available at https://doi.org/10.1186/s43044-022-00282-w.

Additional file 1. Right atrial segmental myocardial transverse and longitudinal strain imaging. As shown in the clip, apical rocking is + 0.61.

Additional file 2. Left atrial segmental myocardial transverse and longitudinal strain imaging. As shown in the clip, apical rocking is + 0.12.

Additional file 3. The right ventricular segmental myocardial transverse and longitudinal strain imaging. As shown in the clip, apical rocking is + 0.84.

Additional file 4. The left ventricular segmental myocardial transverse and longitudinal strain imaging. As shown in the clip, apical rocking is - 0.23.

Additional file 5. The right atrial global and segmental ejection fraction and segmental volume changes.

Additional file 6. The left atrial global and segmental ejection fraction and segmental volume changes.

Additional file 7. The left ventricular global and segmental ejection fraction and segmental volume changes.

Additional file 8. The right ventricular fractional area change.

Additional file 9. The left ventricular fractional area change.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent was obtained from the parents.

Competing interests
The authors have no conflict of interest to declare.

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References
1. Li DL, Davogustto G, Soslow JH, Wassenaar JW, Parikh AP, Chew JD, Dendy JM, George-Durrett KM, Parra DA, Clark DE, Hughes SG (2022) Characteristics of COVID-19 myocarditis with and without multisystem inflammatory syndrome. Am J Cardiol 168:135–141
2. Kohli U, Meinert E, Chong G, Tesher M, Jani P (2020) Fulminant myocarditis and atrial fibrillation in child with acute COVID-19. J Electrocardiol S0022–0736(20):30571–30579
3. Azeka E, Arshad A, Martins C, Dominguez AC, Siqueira A, Loss AS, Jatene M, Miura N (2021) Case report: dilated cardiomyopathy in a newborn, a potential association with SARS-COV-2. Front Pediatr 9:674300
4. Chen G, Li X, Gong Z, Xia H, Wang Y, Wang X, Huang Y, Barajas-Martinez H, Hu D (2021) Hypertension as a sequela in patients of SARS-CoV-2 infection. PLoS ONE 16(4):e0250815
5. Dallaire F, Slorach C, Bradley T, Hui W, Sarkola T, Friedberg MK, Jaeggi E, Dragulescu A, Mahmud FH, Daneman D, Mertens L (2016) Pediatric reference values and Z score equations for left ventricular systolic strain measured by two-dimensional speckle-tracking echocardiography. J Am Soc Echocardiogr 29(8):786-793.e8
6. Marcus KA, Mavinkurve-Groothuis AM, Barends M, van Dijk A, Feuth T, de Korte C, Kapusta L (2011) Reference values for myocardial two-dimensional strain echocardiography in a healthy pediatric and young adult cohort. J Am Soc Echocardiogr 24(6):625–636
7. Marwick TH, Kosmala W (2022) Strain imaging applications and techniques. In: Marwick HH (ed) ASE's comprehensive strain imaging. Elsevier, Philadelphia, pp 1–19
8. Tsugu T, Postolache A, Dulgheru R, Sugimoto T, Tridetti J, Nguyen Trung ML, Piette C, Moonen M, Manganaro R, Ilardi F, Chitroceanu AM, Sperlongano S, Go YY, Kacharava G, Athanassopoulos GD, Barone D, Baroni M, Cardim N, Hagendorff A, Hristova K, Lopez T, de la Morena G, Popescu BA, Penicka M, Ozyigit T, Rodrigo Carbonero JD, van de Veire N,

Von Bardeleben RS, Vinereanu D, Zamorano JL, Rosca M, Calin A, Magne J, Cosyns B, Galli E, Donal E, Santoro C, Galderisi M, Badano LP, Lang RM, Lancellotti P (2020) Echocardiographic reference ranges for normal left ventricular layer-specific strain: results from the EACVI NORRE study. Eur Heart J Cardiovasc Imaging 21(8):896–905

9. Singh A, Addetia K, Maffessanti F, Mor-Avi V, Lang RM (2017) LA strain for categorization of LV diastolic dysfunction. JACC Cardiovasc Imaging 10(7):735–743

10. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, Kindermann I, Gutberlet M, Cooper LT, Liu P, Friedrich MG (2018) Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. J Am Coll Cardiol 72:3158–3176

11. Chattopadhyay A, Singhal M, Debi U, Sharma A, Jain S (2020) Silent myocarditis in Takayasu arteritis. J Clin Rheumatol 26(5):e99

12. Mavrogeni S, Koutsogeorgopoulou L, Karabela G, Stavropoulos E, Katsifis G, Raftakis J, Plastiras S, Noutsias M, Markousis-Mavrogenis G, Kolovou G (2017) Silent myocarditis in systemic sclerosis detected by cardiovascular magnetic resonance using Lake Louise criteria. BMC Cardiovasc Disord 17(1):187

13. Mavrogeni S, Ntoskas T, Gialafos E, Karabela G, Krommida M, Gatzonis S, Siatouni A, Kolovou G, Zouvelou V, Stamboulis E (2016) Silent myocarditis in myasthenia gravis. Role of cardiovascular magnetic resonance imaging. Int J Cardiol 202:629–630

14. Sanna G, Serrau G, Bassareo PP, Neroni P, Fanos V, Marcialis MA (2020) Children's heart and COVID-19: up-to-date evidence in the form of a systematic review. Eur J Pediatr 179(7):1079–1087

15. Irfan O, Muttalib F, Tang K, Jiang L, Lassi ZS, Bhutta Z (2021) Clinical characteristics, treatment and outcomes of paediatric COVID-19: a systematic review and meta-analysis. Arch Dis Child 106(5):440–448

16. Boehmer TK, Kompaniyets L, Lavery AM, Hsu J, Ko JY, Yusuf H, Romano SD, Gundlapalli AV, Oster ME, Harris AM (2021) Association between COVID-19 and myocarditis using hospital-based administrative data—United States, March 2020–January 2021. MMWR Morb Mortal Wkly Rep 70(35):1228–1232

17. Aschenbrenner AC, Mouktaroudi M, Krämer B, Oestreich M, Antonakos N, Nuesch-Germano M, Gkizeli K, Bonaguro L, Reusch N, Baßler K, Saridaki M, Knoll R, Pecht T, Kapellos TS, Doulou S, Kröger C, Herbert M, Holsten L, Horne A, Gemünd ID, Rovina N, Agrawal S, Dahm K, van Uelft M, Drews A, Lenkeit L, Bruse N, Gerretsen J, Gierlich J, Becker M, Händler K, Kraut M, Theis H, Mengiste S, De Domenico E, Schulte-Schrepping J, Seep L, Raabe J, Hoffmeister C, ToVinh M, Keitel V, Rieke G, Talevi V, Skowasch D, Aziz NA, Pickkers P, van de Veerdonk FL, Netea MG, Schultze JL, Kox M, Breteler MMB, Nattermann J, Koutsoukou A, Giamarellos-Bourboulis EJ, Ulas T, Altmüller J, Angelov A, Bals R, Bartholomäus A, Becker A, Bitzer M, Bonifacio E, Bork P, Casadei N, Clavel T, Colome-Tatche M, Diefenbach A, Dilthey A, Fischer N, Förstner K, Franzenburg S, Frick J-S, Gabernet G, Gagneur J, Ganzenmüller T, Göpel S, Goesmann A, Hain T, Heimbach A, Hummel M, Iftner A, Iftner T, Janssen S, Kalinowski J, Kallies R, Kehr B, Keller A, Kim-Hellmuth S, Klein C, Kohlbacher O, Köhrer K, Korbel J, Kühnert D, Kurth I, Landthaler M, Li Y, Ludwig K, Makarewicz O, Marz M, McHardy A, Mertes C, Nöthen M, Nürnberg P, Ohler U, Ossowski S, Overmann J, Pfeffer K, Poetsch AR, Pühler A, Rajewsky N, Ralser M, Rieß O, Ripke S, Nunes da Rocha U, Rosenstiel P, Saliba A-E, Sander LE, Sawitzki B, Schiffer P, Schneider W, Schulte E-C, Schultze JL, Sczyrba A, Singh Y, Sonnabend M, Stegle O, Stoye J, Theis F, Vehreschild J, Vogel J, von Kleist M, Walker A, Walter J, Wieczorek D, Winkler S, Ziebuhr J, German C-OI (2021) Disease severity-specific neutrophil signatures in blood transcriptomes stratify COVID-19 patients. Genome Med 13(1):7

18. Fricke-Galindo I, Falfan-Valencia R (2021) Genetics insight for COVID-19 susceptibility and severity: a review. Front Immunol 12:622176

19. Ghani A, Delnoy PP, Smit JJ, Ottervanger JP, Ramdat Misier AR, Adiyaman A, Elvan A (2016) Association of apical rocking with super-response to cardiac resynchronisation therapy. Neth Heart J 24(1):39–46

20. Ghani A, Delnoy PP, Ottervanger JP, Misier AR, Smit JJ, Adiyaman A, Elvan A (2015) Apical rocking is predictive of response to cardiac resynchronization therapy. Int J Cardiovasc Imaging 31(4):717–725

21. Szulik M, Tillekaerts M, Vangeel V, Ganame J, Willems R, Lenarczyk R, Rademakers F, Kalarus Z, Kukulski T, Voigt JU (2010) Assessment of apical rocking: a new, integrative approach for selection of candidates for cardiac resynchronization therapy. Eur J Echocardiogr 11(10):863–869

22. Bax Jeroen J, van der Bijl P (2022) Apical rocking and septal flash. JACC Cardiovasc Imaging 15(2):221–223

23. Gorcsan J 3rd, Anderson CP, Tayal B, Sugahara M, Walmsley J, Starling RC, Lumens J (2019) Systolic stretch characterizes the electromechanical substrate responsive to cardiac resynchronization therapy. JACC Cardiovasc Imaging 12(9):1741–1752

24. Cameli M, Mandoli GE, Loiacono F, Sparla S, Iardino E, Mondillo S (2016) Left atrial strain: a useful index in atrial fibrillation. Int J Cardiol 220:208–213

25. Donal E, Galli E, Schnell F (2017) Left atrial strain: a must or a plus for routine clinical practice? Circ Cardiovasc Imaging 10(10):e007023

26. Gorcsan J 3rd (2021) Can left atrial strain forecast future fibrillation? JACC Cardiovasc Imaging 14(1):145–147

27. Mandoli GE, Sisti N, Mondillo S, Cameli M (2020) Left atrial strain in left ventricular diastolic dysfunction: have we finally found the missing piece of the puzzle? Heart Fail Rev 25(3):409–417

28. Morris DA, Belyavskiy E, Aravind-Kumar R, Kropf M, Frydas A, Braunauer K, Marquez E, Krisper M, Lindhorst R, Osmanoglou E, Boldt LH, Blaschke F, Haverkamp W, Tschope C, Edelmann F, Pieske B, Pieske-Kraigher E (2018) Potential usefulness and clinical relevance of adding left atrial strain to left atrial volume index in the detection of left ventricular diastolic dysfunction. JACC Cardiovasc Imaging 11(10):1405–1415

29. Chetrit M, Friedrich MG (2018) The unique role of cardiovascular magnetic resonance imaging in acute myocarditis. F1000Res 7: F1000 Faculty Rev-1153.

30. Dasgupta S, Iannucci G, Mao C, Clabby M, Oster ME (2019) Myocarditis in the pediatric population: a review. Congenit Heart Dis 14(5):868–877

31. Georgiopoulos G, Figliozzi S, Sanguineti F, Aquaro GD, di Bella G, Stamatelopoulos K, Chiribiri A, Garot J, Masci PG, Ismail TF (2021) Prognostic impact of late gadolinium enhancement by cardiovascular magnetic resonance in myocarditis: a systematic review and meta-analysis. Circ Cardiovasc Imaging 14(1):e011492

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