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Case Report

A case of transient constrictive pericarditis after COVID-19☆

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ABSTRACT

Cardiovascular disorders have been associated with coronavirus disease 2019 (COVID-19). Here, we describe a case of transient constrictive pericarditis after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. A few days following SARS-CoV-2 pneumonia, a 55-year-old man developed fever and chest pain exacerbated by movement and breathing, and acute pericarditis was diagnosed. After two weeks, he progressively developed fatigue, dyspnea, peripheral edema, ascites, and bilateral pleural effusion. The patient's clinical condition, as well as imaging findings, were consistent with a diagnosis of constrictive pericarditis. Therefore, medical therapy was optimized with a progressive clinical improvement. Follow-up echocardiography showed full recovery of pericardial constriction. Transient constrictive pericarditis, defined as a reversible pericardial constriction followed by resolution, can be spontaneous or treatment-related, and represents an uncommon complication of acute pericarditis. Although a broad spectrum of COVID-19-related cardiac diseases (including pericarditis) have already been reported, transient pericardial constriction after SARS-CoV-2 infection has not previously been described.

Learning objective: Transient constrictive pericarditis is an uncommon complication of acute pericarditis that can occur sporadically after viral acute pericarditis. We hereby describe a case of coronavirus disease 2019-related transient pericardial constriction. This case confirms that pericardial constriction after viral acute pericarditis often resolves with medical therapy.

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is a heterogeneous clinical condition, which may present an asymptomatic clinical course as well as lead to more severe presentations such as multiorgan failure. Although the main clinical manifestation is interstitial pneumonia (with or without respiratory failure), SARS-CoV-2 has also been implicated in several extrapulmonary manifestations, including cardiovascular disorders such as myocardial injury, arrhythmias, acute coronary syndrome, and thromboembolism [1]. Furthermore, inflammatory cardiac diseases, including pericarditis and myocarditis, have also been reported [2].

In general, very few cases of acute pericarditis progress into a transient constrictive pericarditis, an uncommon complication characterized by a temporary form of constriction due to inflammation, which often resolves once the underlying inflammatory process is resolved [3].

We hereby describe a case of transient constrictive pericarditis which occurred after SARS-CoV-2 infection.

Case report

A 55-year-old Caucasian man presented to the emergency department (ED) with peripheral edema, shortness of breath, and abdominal swelling.

Medical history included arterial hypertension and an unspecified respiratory disease; previous spirometry was normal, as well as carbon monoxide diffusion capacity.

After a month of in-hospital stay, the patient developed SARS-CoV-2 pneumonia with acute hypoxemic respiratory failure. The patient was treated and eventually discharged. Three days after discharge, he

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presented to another ED with fever and chest pain exacerbated by movement and breathing. A baseline electrocardiogram (ECG) showed sinus tachycardia and widespread ST elevation, while a bedside echocardiography showed normal left and right ventricular dimensions and function, with also normal inferior vena cava (IVC); during the examination, a moderate anechoic pericardial effusion (15 mm width) was found, without any sign of cardiac tamponade.

Laboratory tests were consistent with a systemic inflammatory response, with high C-reactive protein (CRP; 237 mg/L, reference range < 5 mg/L) and white blood cells 25.4 × 10^3/ml. Serum creatinine was 0.84 mg/dL, and high-sensitivity cardiac troponin I also remained within the normal laboratory range. A diagnosis of acute pericarditis was made, and the patient was discharged with ibuprofen 600 mg every 8 h. The dosage of non-steroidal ibuprofen was eventually reduced after a few days, due to gastrointestinal intolerance, and treatment with colchicine 1 mg/day was started.

After one week, the patient presented to our ED with progressive signs and symptoms of heart failure.

Laboratory tests showed: white blood cells 16 × 10^3/L, hyponatremia (130 mEq/L), serum creatinine 1.6 mg/dL, CRP 39.8 mg/L. ECG showed sinus tachycardia with a heart rate of 105 beats/min, and widespread nonspecific T-wave abnormalities.

An echocardiography described a partially organized circumferential pericardial effusion, without signs of cardiac tamponade; IVC was dilated and not collapsing, with also a prominent hepatic vein expiratory diastolic flow reversal. Septal bounce, respiratory variation of the mitral peak *E*-wave velocity about 29 %, reduction of the right ventricular systolic function, and the phenomenon of "annulus reversus" (mitral annular lateral e' to medial e' ratio < 1) were also observed during the examination. Moreover, global longitudinal strain (GLS) showed an asymmetric involvement of myocardium, with a relative sparing of the septal segments compared to the posterior and lateral segments (so called "strain reversus" bull's-eye plot) (Fig. 1). Bilateral pleural effusion and ascites were also found.

The patient therefore underwent cardiac magnetic resonance, that showed slightly reduced ventricular volumes with substantially preserved function, thickening of the pericardial sheets (4 mm) with late gadolinium enhancement (LGE), and absence of LGE of the ventricular walls. Finally, a circumferential pericardial effusion, partially organized, with a maximum thickness of 10 mm was also observed (Online materials). These findings confirmed the diagnosis of pericarditis with pericardial constriction.

Medical therapy was therefore optimized, but a transient reduction of the glomerular filtration rate required the replacement of ibuprofen with prednisone and colchicine. Diuretics were administered to reduce systemic congestion, and once adequate hemodynamic compensation was obtained, the patient was discharged. At discharge, laboratory test results improved compared with baseline: white blood cells 9.34 × 10^3/L, sodium 139 mEq/L, serum creatinine 1 mg/dL, CRP 5.7 mg/L.

During follow-up, a progressive improvement in the patient's clinical condition was observed after a few weeks, with partial resolution of ECG abnormalities and persistence of non-specific inversion of T

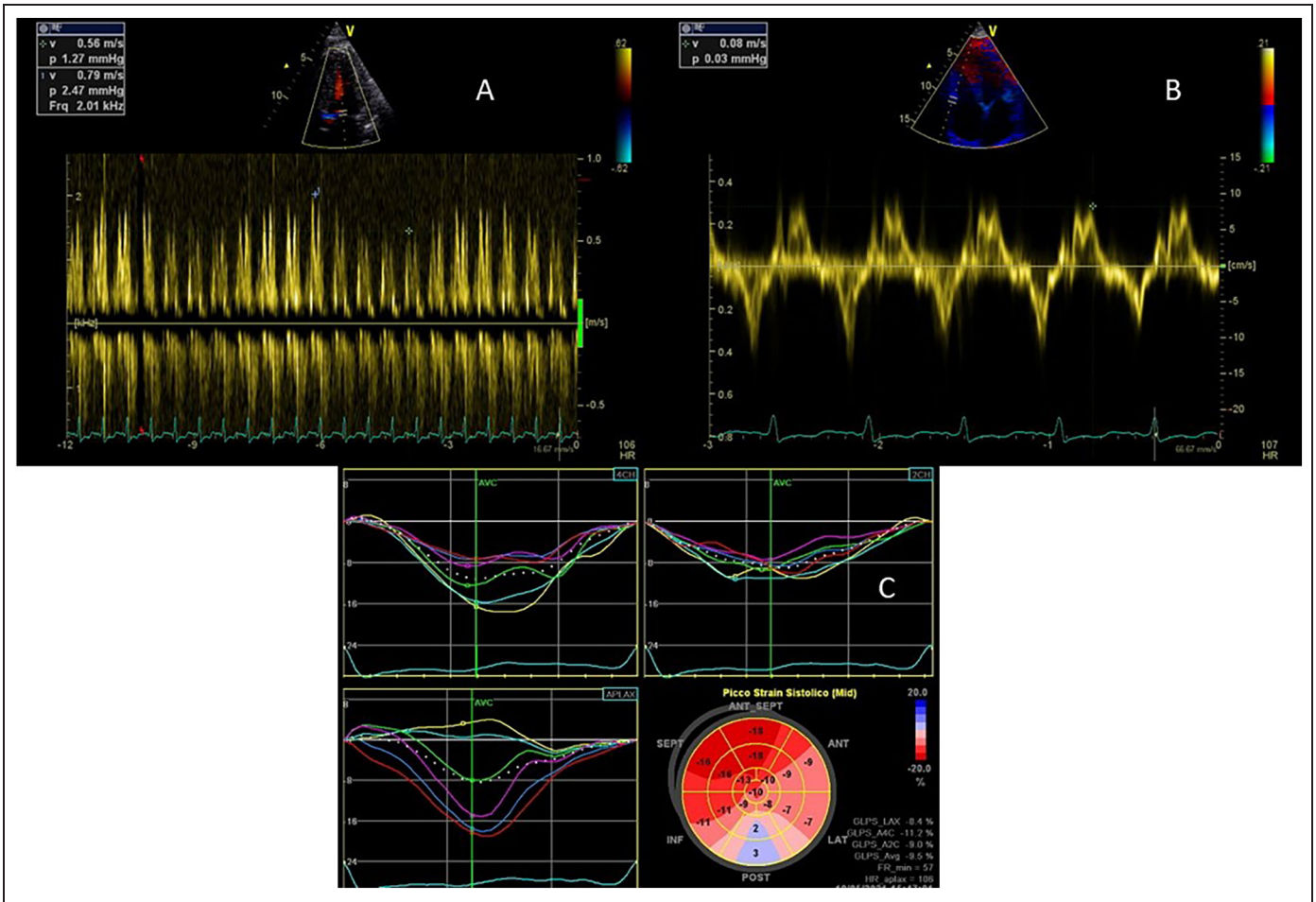

Fig. 1. Echocardiography at the time of diagnosis.
(A) Respiratory variation of the mitral peak E-wave about 29 % (pulsed wave Doppler sample volume at the level of the mitral leaflets). (B) Reduction of the right ventricular systolic longitudinal function (tricuspid annular velocity S′ wave with tissue Doppler imaging, 8 cm/s); (C) Left ventricle global longitudinal strain at the time of diagnosis (−9.5 %). A clear reduction of longitudinal strain is observed in the lateral and posterior regions of the bull's eye (so called "strain reversus" phenomena).

Table 1
Echocardiographic measures at the time of diagnosis and at follow up.

	Diagnosis	Follow up	Normal value [10]
Respiratory variation of the mitral peak E-wave (%)	29	21	<25
Left lateral annular velocity (cm/s)	9	17	>10
Interventricular septum annular velocity (cm/s)	10	11	>7
Left ventricular GLS (%)	−9.5	−18	>−20 %
Stroke volume	42 ml	88 ml	60–80 ml
Left ventricular cardiac output	3.96 l/m	7.42 l/m	4–8 l/min
Tricuspid annular velocity (TDI, cm/s)	8	12	>9.5
TAPSE (mm)	13	19	>17
RVOT acceleration time (msec)	97	148	>130
FAC (%)	27	52	>35
TDI MPI	0.65	0.31	<0.54
LVEDD (mm)	40	48	≤58.4
LVESD (mm)	25	22	≤39.8
LVEDV normalized by BSA (ml/m^2)	40	57.5	34–74
LVESV normalized by BSA (ml/m^2)	19	13.5	11–31
LVEF (%)	52	74	>52
LA diameter (mm)	37	38	<40
LAVi (ml/m^2)	24	16	≤34
RAVi (ml/m^2)	30	24	25 ± 7
BSA (m2)	2		

TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area change; MPI, myocardial performance index; TDI, tissue Doppler imaging; RVOT, right ventricular outflow tract; GLS, global longitudinal strain; LA, left atrial; LAVi, left atrial volume index; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; RAVi, right atrial volume index; BSA, body surface area.

waves only in the inferior leads. Diuretics were tapered and ultimately suspended after two months. The patient continued treatment with colchicine and prednisone for three months after discharge, and then steroids were tapered and finally discontinued after 4 months from admission.

Serial echocardiograms were performed during follow-up, and specifically early after discharge, and then at 2 and 7 months. The mean differences of the echocardiographic parameters at baseline and then at the last follow-up control are reported in Table 1. Overall, progressive improvements were observed, with the last echocardiogram showing normal dimensions of the IVC, reduction of the respiratory variation of the mitral peak E-wave velocity (21 %), and normalization of the right ventricular systolic function (Fig. 2).

Discussion

Acute pericarditis is a well-known cause of chest pain and accounts for around 0.1 % of all hospital admissions. A simplified classification of pericardial diseases is based on whether the primary underlying cause of the disease is infectious or not. In developed countries, approximately 80–90 % of cases are diagnosed as idiopathic, likely after viral infections [4].

Constrictive pericarditis is considered a rare complication of viral or idiopathic acute pericarditis, while it is more frequent for specific etiologies, including bacterial and tuberculous pericarditis [5].

Transient constrictive pericarditis represents a peculiar subtype of constrictive pericarditis, defined by a reversible pattern of pericardial constriction followed by resolution, either spontaneous or with medical therapy. The reversibility of this condition is thought to be related by the pathophysiological mechanisms underlying this condition, which includes inflammation, fibrin deposits, and edema rather than fibrosis or calcification (less likely associated with resolution). This hypothesis has been also corroborated by novel imaging techniques [3,6]. Indeed, alongside the traditional echocardiographic signs of pericardial constriction (i.e. respiratory-related ventricular septal bounce, pericardial thickening and calcifications, respiratory variation of the mitral peak E-wave velocity of >25 %, IVC plethora), new signs have recently been proposed. In particular, the well-known phenomenon of "annulus reversus" (mitral annular lateral e' to medial e' ratio < 1), calculated with tissue Doppler, seems to reflect the specular phenomenon documented with 2D speckle tracking. Specifically, we observed reduced systolic peak strain values at left ventricle free wall compared to the septal (medial) peak systolic strain (the so-called "strain reversus") [6]. Similarly, the finding of typical strain reversus (so called "hot septum") observed at tissue Doppler could be explained by the tight adherence of the lateral mitral annulus to the thickened adjacent pericardium. Besides echocardiography, other invasive imaging techniques, such as hemodynamic assessment performed through right heart catheterization, can be particularly helpful in uncertain cases; however, given the overall good clinical course of our patient, we decided not to perform invasive procedures.

Although coronavirus disease 2019 (COVID-19) has been extensively studied, the pathogenesis of the interplay between COVID-19 and cardiovascular disease remains largely unclear. During the pandemic, several cases of acute pericarditis, pericardial effusion, and cardiac tamponade allegedly related to COVID-19 have been described [2], but only three cases of constrictive or effusive-constrictive pericarditis in SARS-CoV-2 infection have already been reported [7–9]. Specifically, in two cases the patients developed constrictive-effusive pericarditis during the acute phase of COVID-19, and subsequently died of severe respiratory failure [8,9]. In the third case, pericardial effusion with cardiac tamponade was observed in a patient with several in-hospital complications, four months after COVID-19; the patient ultimately needed a pericardiocentesis [7].

To our knowledge, this is the first described case of COVID-19-related transient pericardial constriction. Indeed, our patient presented clinical, echocardiographic, and radiological findings compatible with constrictive pericarditis which disappeared in the following months (Figs. 1 and 2). Although there is no definitive guidance on how to manage these patients, it is conceivable that a prompt diagnosis, as well as appropriate anti-inflammatory therapy, may play a crucial role to reduce inflammation and prevent progression to a more severe and irreversible pericardial constriction, as we observed in our patient.

Conclusion

The main clinical manifestation of COVID-19 is interstitial pneumonia, but SARS-CoV-2 has also been implicated in several extrapulmonary manifestations, including various cardiovascular disorders. Transient constrictive pericarditis represents a rare cardiac complication of COVID-19, which requires awareness and specific echocardiographic evaluation.

Declaration of competing interest

None of the authors has any conflicts of interest to declare.

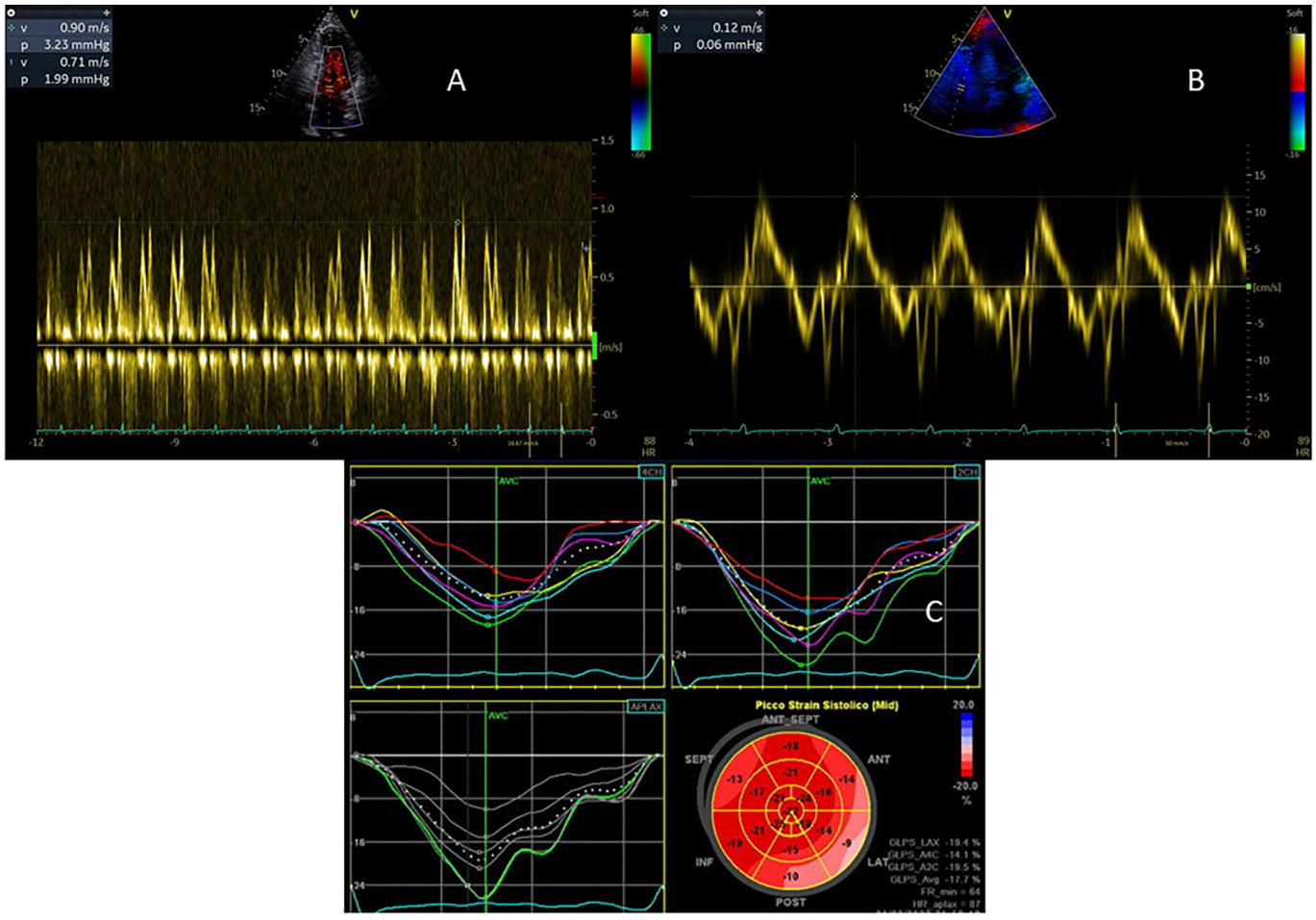

Fig. 2. Echocardiography at follow up.
(A) Normalization of the respiratory variation of the mitral peak E-wave (21 %); (B) Normalization of the tricuspid annular velocity wave with tissue Doppler imaging, 12 cm/s; (C) Almost normal left ventricle global longitudinal strain (GLS -18 %) with a net improvement in GLS in the lateral and inferior regions.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jccase.2022.07.006.

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