

Effusive-constrictive pericarditis following mild COVID-19: a case of combined surgical and medical therapy

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SOUHRN

Dnes je již prokázáno, že infekční onemocnění SARS-CoV-2, zprvu považované za respirační onemocnění, způsobuje kardiovaskulární komplikace včetně vzácných případů efuzivně-konstriktivní perikarditidy. Popisujeme případ 45letého muže, u něhož došlo po překonání infekčního onemocnění covid-19 s mírným průběhem k rozvoji efuzivně-konstriktivní perikarditidy. Pacient byl původně přijat pro akutní perikarditidu s malým perikardiálním výpotkem léčeným ibuprofenem a colchicinem. O dva týdny později se muž dostavil k vyšetření znovu pro zhoršující se symptomy srdečního selhání včetně ortopnoe, otoků a únavy. Vyšetření srdce echokardiograficky a magnetickou rezonancí (cardiac magnetic resonance, CMR) ukázala na přítomnost efuzivně-konstriktivní perikarditidy a subakutní myokarditidy. Přes protizánětlivou léčbu a podávání diuretik symptomy přetrvávaly, a vyžádaly si tak perikardiektomii. Perikard byl silně ztluštělý s rozsáhlými srůsty. Symptomy srdečního selhání přetrvávající po operaci a nález fibrózního perikardu zobrazovacími metodami vedly k zahájení léčby kortikosteroidy s následným vymizením symptomů. Tento případ názorně ukazuje náročnost diagnostiky a léčby efuzivně-konstriktivní perikarditidy v kontextu infekčního onemocnění covid-19. Identifikaci aktivního zánětu perikardu a vedení léčby napomohlo použití neinvazivních zobrazovacích metod, zvláště CMR. Zatímco perikardiektomie představuje i nadále rezolutní intervenci v jinak neřešitelných případech, popsáný případ ukazuje na potenciální přínos imunosupresivní léčby pro vymizení reziduálních symptomů. Vzhledem k omezenému množství literatury na téma perikarditidy v souvislosti s infekčním onemocněním covid-19 je pro optimalizaci výsledku léčby a směřování dalšího výzkumu nutný multidisciplinární přístup.

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ABSTRACT

SARS-CoV-2 infection, initially perceived as a respiratory illness, is now recognized to cause cardiovascular complications, including rare cases of effusive-constrictive pericarditis. We present a case of a 45-year-old male who developed effusive-constrictive pericarditis following mild COVID-19. The patient was initially admitted with acute pericarditis and moderate pericardial effusion, treated with ibuprofen and colchicine. Two weeks later, he returned with worsening symptoms of heart failure, including orthopnea, edema, and fatigue. Echocardiography and cardiac magnetic resonance (CMR) revealed findings consistent with effusive-constrictive pericarditis and subacute myocarditis. Despite anti-inflammatory and diuretic therapy, symptoms persisted, necessitating pericardiectomy. The pericardium was severely thickened with extensive adhesions. Postoperatively, persistent heart failure symptoms and imaging findings of fibrotic pericardium led to initiation of corticosteroid therapy, which resolved symptoms. This case underscores the diagnostic and therapeutic challenges of effusive-constrictive pericarditis in the context of COVID-19. Non-invasive imaging, particularly CMR, was instrumental in identifying active pericardial inflammation and guiding treatment. While pericardiectomy remains a critical intervention in refractory cases, this case highlights the potential benefit of immunosuppressive therapy in resolving residual symptoms. Given the limited literature on COVID-19-associated pericarditis, multidisciplinary management is essential for optimizing outcomes and guiding future research.

Keywords:

Constrictive pericarditis

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Introduction

Initially regarded as a respiratory illness, SARS-CoV-2 has been associated with extrapulmonary manifestations, including cardiovascular disorders like myocardial injury, arrhythmias, acute coronary syndrome, thromboembolism, pericarditis and myocarditis. The risk of constrictive pericarditis is usually low for viral or idiopathic acute pericarditis, while it is more frequent for specific etiologies, including bacterial and tuberculous pericarditis.¹

There are only a few cases reported of SARS-CoV-2 related effusive-constrictive pericarditis.

Case description

A 45-year-old male, diagnosed with mild COVID-19 in the previous week, was presented with acute pericarditis with moderate pericardial and bilateral pleural effusions. Admission tests revealed elevated NT-proBNP (221pg/ml, N 0–125 pg/mL) and C-reactive protein (15 mg/dL, N 0–0.5 mg/dL), normal troponin I levels and sinus tachycardia and diffuse negative T waves on ECG. Echocardiogram showed circumferential pericardial effusion (11 mm), without hemodynamic compromise; normal biventricular systolic function; no valve alterations; dilated inferior vena cava (IVC) without respiratory variation. The patient started colchicine and ibuprofen and was discharged under this treatment after reduction of the effusions. One week

later, colchicine was discontinued due to abdominal discomfort.

Two weeks after discharge, the patient returned with orthopnea, edema of the lower limbs, fatigue, and decrease in urinary output in the previous three days. On examination, he exhibited heart rate of 104/min, normal blood pressure, auscultation with normal heart sounds with no rub or murmurs and decreased breath sounds on the right, jugular venous distension, ascites and significant lower limb swelling.

Laboratory results revealed elevated NT-proBNP (587 pg/mL), C-reactive protein (4,17 mg/dL), alkaline phosphatase (135 U/L), gamma GT (84 U/L), and hyperbilirubinemia (1.6 mg/dL), with normal albumin and INR. ECG revealed sinus tachycardia, chest X-ray showed right pleural effusion, and echocardiogram revealed mild pericardial effusion (7 mm, circumferential), no signs of hemodynamic compromise but with apparent septal bounce, normal global biventricular systolic function; no valve alterations and dilated IVC.

He was admitted to investigate polyserositis, while maintaining ibuprofen and starting furosemide.

Thoracentesis revealed a transudate according to light criteria and diagnostic tests for autoimmune, bacterial, and viral causes were negative (including IGRA). Echocardiography confirmed mild pericardial effusion, pericardial thickening, septal bounce, and respiration-related variation of atrioventricular transvalvular flow, without cardiac chambers collapse. The patient was discharged, with anti-inflammatory ther-

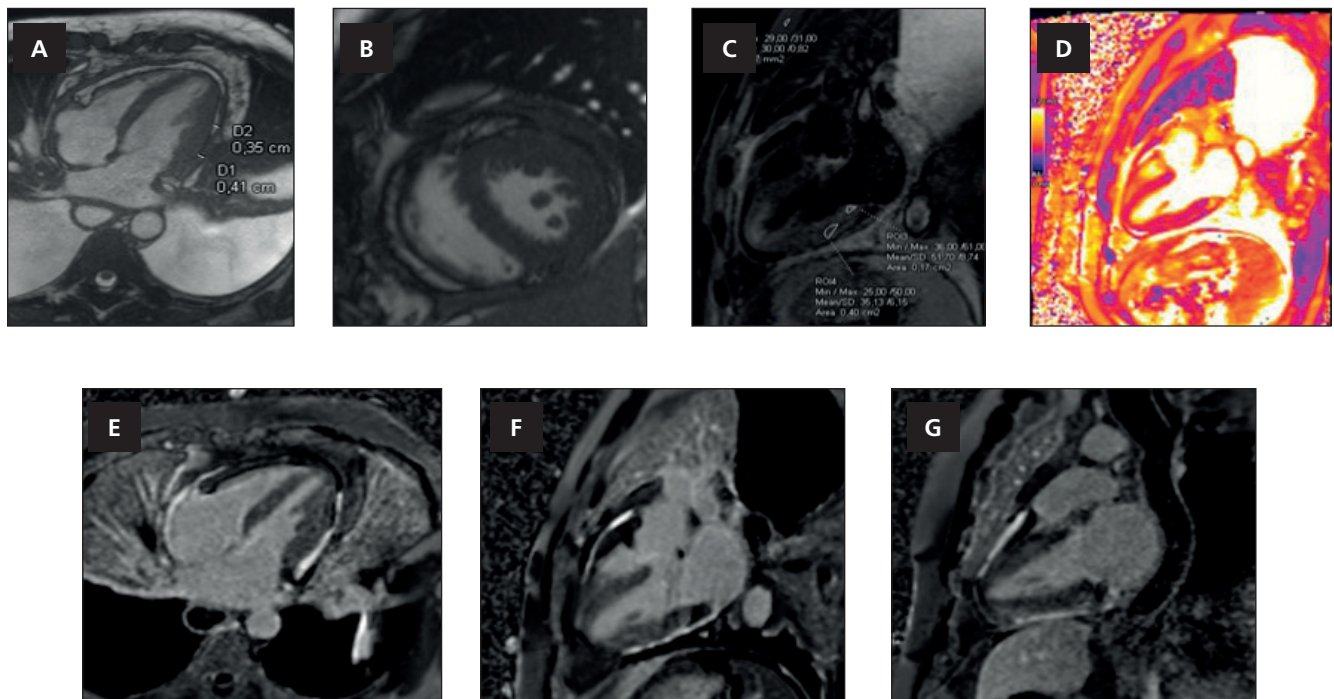


Fig. 1 – First cardiac magnetic resonance, performed on the day 49 after diagnosis of SARS-CoV-2 infection and after 39 days of anti-inflammatory therapy. Source: Clinical records of the patient. (A and B) Ventricular interdependence signs diffuse pericardial thickening and mild pericardial effusion. (C) T2 STIR without hypersignal. (D) T2 mapping with focally increased values in the basal inferolateral segment. (E, F) Late gadolinium enhancement with diffuse hypersignal of the pericardium and focal midventricular enhancement.

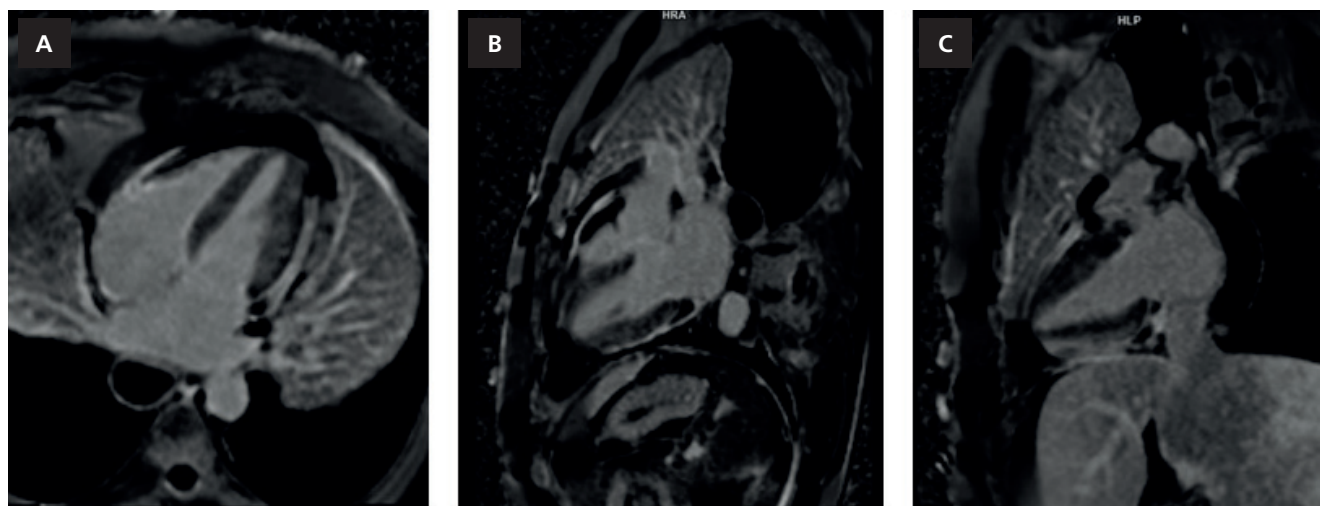


Fig. 2 – Second cardiac magnetic resonance, performed on the day 70 after diagnosis of SARS-CoV-2 infection and after 60 days of anti-inflammatory therapy. Source: Clinical records of the patient. (A–C) Reduced extent of the pericardial late gadolinium enhancement.

apy and furosemide, awaiting cardiac magnetic resonance (CMR).

The CMR, performed on the 39th day of anti-inflammatory therapy showed signs of ventricular interdependence with septal bounce and respiration-related shift of the interventricular septum; myocardium with increased extracellular volume, focal increase of T2 mapping values in the basal inferolateral segment of the left ventricle, diffuse areas of midventricular late gadolinium enhancement (basal and middle segments of anterolateral, inferolateral and inferior walls, basal anterior, middle anteroseptal and infero-septal and apical inferior, lateral and septal); pericardium with diffuse thickening, mild pericardial effusion and diffuse late gadolinium enhancement; moderate bilateral pleural effusion; preserved systolic function of both ventricles; compatible with subacute myocarditis and effusive-constrictive pericarditis (Fig. 1). Symptoms worsened despite therapy, and he was readmitted.

He maintained anti-inflammatory therapy with ibuprofen, restarted colchicine and needed high doses of IV diuretics and sequential nephron block for right heart failure compensation. Cardiac catheterization performed on the 52nd day of anti-inflammatory therapy showed atypical right ventricle pressure curve without typical features of constriction.

CMR was repeated on the 60th day of therapy, with reduced extent of pericardial late gadolinium enhancement and less marked signs of ventricular interdependence (Fig. 2). After intensive intravenous diuretic therapy and hydro saline restriction, he showed progressive improvement, allowing discharge to the outpatient clinic.

The case was discussed with cardiac surgery, and the patient was subsequently submitted to pericardiectomy, revealing severely thickened pericardium (8 mm) with extensive adhesions.

Post-surgery CMR showed absence of pericardium in anterior topography, maintaining extensive areas of

thickened pericardium with diffuse late enhancement with clear hyposignal that suggests a concomitant fibrotic process and thin lamina of pericardial effusion.

Persistent heart failure symptoms necessitated corticosteroid therapy (prednisolone 1 mg/kg/day), leading to symptom resolution.

Relevance and final considerations

Effusive-constrictive pericarditis diagnosis typically relies on intrapericardial pressure measurements,² but non-invasive imaging may be equally useful for its diagnosis.

CMR is critical for identifying active pericardial inflammation. The reversibility of the condition is thought to be related by the underlying pathophysiological mechanisms, which includes inflammation, fibrin deposits, and edema, whereas fibrosis and calcification indicate poorer outcomes.³

Limited data exist on COVID-19-associated pericarditis, with only two reported cases of effusive-constrictive pericarditis, one requiring pericardiectomy.^{4,5}

Further research on immunosuppressive therapy and optimal timing for surgical intervention is needed. A multidisciplinary approach involving infectious disease specialists, cardiologists, and surgeons is essential for management.

Conflict of interest

Nothing to disclose.

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Informed consent

The patient provided informed written consent for the publication of the study data.

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