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COVID-19 Associated Myocarditis: A Monocentric Series of 33 Cases

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Abstract

Introduction: Myocarditis is defined by an inflammatory myocardial infiltrate with necrosis of non-ischemic origin in three forms: fulminant, acute, and chronic. Diagnosis is guided by clinical presentation, ECG, echocardiography, and biology, and confirmed by MRI and myocardial biopsy. The prognosis depends on clinical manifestations, echocardiographic features, and serum troponin levels. Management is based on the treatment of heart failure (HF). For two years, the world has been experiencing a pandemic related to SARS-CoV2 that can affect the heart with ischemic or non-ischemic lesions (myocarditis, most often fulminant) whose treatment is nonspecific. Trials with corticosteroids and immunosuppressant drugs have yielded discordant results.

Objective: To describe the evolutionary modalities of COVID-19-associated myocarditis and identify factors of poor ejection fraction recovery under HF treatment.

Method: This observational, retrospective, single-center study, in 2021, included patients with non-fulminant COVID-19-associated myocarditis suspected at echocardiography and biology and confirmed on MRI. Patients with previous HF and reduced left ventricular ejection fraction (LVEF) were excluded (n=06). Patients were divided into two groups according to LVEF three months later (LVEF>50% v. LVEF<50%) and compared to identify factors predicting a poor LVEF recovery.

Results: 33 patients (19♂/14♀) aged between 30–61 years with acute non-fulminant COVID-19-associated myocarditis were included. All had ECG repolarization abnormalities. The mean LVEF at baseline was 44.3% +/- 6.3 (30–52%) with an average troponin level 480 times normal (20–2,100). Beta-blocker and RASB treatment was initiated in all patients, spironolactone (37.5 mg) in 13 patients with LVEF <40%, and furosemide if congestive signs (17 patients/51.5%). Clinical, electrical, biological, and echocardiographic monitoring was performed at one and three months. Eight patients developed uncomplicated pericardial effusion. A significant improvement in LVEF>50% was observed in 29 patients. One patient with LVEF of 38% presented with incessant ventricular tachyarrhythmia that necessitated an ICD. Three patients kept LVEF<50%. Sex, congestive signs, ECG, and coronary angiogram abnormalities do not seem to influence the LVEF evolution (*p* at 0.62, 1.00, 1.00, 0.56, 0.50, and 0.23, respectively). Age >60 years, troponins >1,200 times normal, pericardial effusion, and a combined criterion of the three seem to be a good predictor of poor LVEF evolution (*p* at 0.07, 0.02, 0.035, and 0.01, respectively).

Discussion: The absence of fulminant forms in our series explains the absence of mortality at three months (>30% in the literature). Acute non-fulminant COVID-19-associated myocarditis has a good prognosis with LVEF recovery in 87.88%. The factors of poor LVEF recovery are the age >60 years, troponins >1,200 times normal, pericardial effusion, and the combined criterion of the three (*p* respectively at 0.07, 0.02, 0.035, 0.01). The routine prescription of corticosteroids in the COVID-19 protocol made it impossible to analyze its impact on COVID-19-associated myocarditis.

Interpretation: Cardiac manifestations are not uncommon during COVID-19; they can be ischemic or non-ischemic. There is no specific therapy for non-fulminant COVID-19-associated myocarditis and the evolution seems favorable. Patients with predictive factors of poor progress should have longer follow-ups.

Informed consent: All participants gave their informed consent to participate in this study and share the results.

Keywords: Patient series, fulminant myocarditis, ACE2 receptor, SARS-CoV-2, troponins, cardiac MRI

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INTRODUCTION

Myocarditis is defined by the presence of an inflammatory infiltrate with myocardial necrosis of non-ischemic origin, in three forms: fulminant, acute, and chronic [1, 2]. The diagnosis is guided by clinical signs, electrocardiogram (ECG), echocardiography, and biology (troponins); it is then confirmed by magnetic resonance imaging (MRI) and myocardial biopsy [3–8]. The prognosis depends on the initial clinical manifestations, echocardiographic findings, and troponin levels [9]. Management is based on the classic treatment of chronic heart failure: loop diuretics in case of overload, blockers of the renin-angiotensin system, beta-blockers, and spironolactone. Specific treatments such as immunosuppressant drugs, corticosteroids, and immunoglobulins have been tested in various situations, with results varying from one histological type to another [9].

In 2019, the world began experiencing an unprecedented pandemic linked to the respiratory spread of a single-stranded RNA virus (SARS-CoV2), which enters cells thanks to the ACE2 receptor, present in the lung, heart and kidney cells [10].

Cardiac involvement during COVID-19, estimated at 20% [11], is defined by an increase in troponin levels associated with ECG abnormalities and hypokinesia at echocardiography. These abnormalities are of ischemic origin (plaque fracture related to inflammatory stress, thrombosis in situ or coronary embolism related to hypercoagulability) or non-ischemic (more often a fulminant myocarditis) [10, 12].

Myocardial biopsies (most often post-mortem) confirm the existence of an inflammatory lymphocytic infiltrate without detection of the SARS-CoV-2 virus. [13, 14].

The most likely hypothesis to explain virus-induced myocarditis is probably the ACE2 receptors hypothesis; the latter are internalized during the virus penetration, resulting in a decrease in their expression on the surface of cardiomyocytes. This phenomenon limits the reduction of angiotensin II and results in a direct toxicity on cardiomyocyte apoptosis but also an indirect toxicity through vasoconstriction, edema, and ischemia [10].

Finally, cases of acute myocarditis have been reported after vaccination against COVID-19 with messenger RNA vaccines, without a clear cause being identified (effects on the ACE2 receptor or an immunoallergic phenomenon) [15].

The treatment of myocarditis occurring during a COVID-19 infection is nonspecific; it involves strict rest in the acute phase and the prevention of intense physical activity for three to six months. Treatment for heart failure is associated with inotropic and vasoactive treatment or even circulatory assistance in the case of the fulminant form. Trials with corticosteroids and immunosuppressants have been attempted with discordant results [16].

The subsequent follow-up and evolution of these patients remains unknown and has been based on the repetition of anatomical examinations (especially MRI) [17].

Objective

The objective of this study is to describe the treatment and evolutionary modalities of COVID-19-associated myocarditis and to identify factors of poor LVEF recovery in this situation under heart failure treatment at three-month follow-up.

MATERIALS AND METHODS

Study design: This is an observational, retrospective, single-center study.

Setting: The study was conducted from August 2021 to March 2022, during the third COVID-19 pandemic wave, in a referral cardiology department of a university hospital, from a prospective registry collecting clinical, biological, and imaging data on myocarditis patients (from all forms). Patients who were registered in our myocarditis registry from August to December 2021, and who met the inclusion criterion for this work, were enrolled. A follow-up period of three months was observed in all these patients. Data collection lasted until March 2022.

Participants: We included all patients presenting a non-fulminant COVID-19-associated myocarditis suspected on echocardiography and biology and confirmed

on cardiac MRI (total of 39 patients). Patients with previous HF and reduced LVEF were excluded (n=06).

Patients were then divided into two groups according to LVEF recovery three months later (LVEF>50% v. LVEF<50%) and compared to identify factors predicting a poor LVEF evolution.

We did not explore for a follow-up period of three months any patient lost to follow-up or who had died. All participants gave their informed consent to participate in this study and share the results.

Variables: For all patients, we collected data on symptoms and clinical examination, troponin level, ECG, and echocardiography. All patients had an MRI and a coronary angiogram.

Measurement: Symptoms and clinical examination were assessed and mentioned in the patient's medical record and the department's myocarditis registry at each consultation (0, 1, and 3 months).

Troponins were assessed on Biomerieux vidas automaton and mentioned in the patient's medical record and department's myocarditis registry at the first visit.

ECGs were performed on 12-lead devices, and echocardiographic parameters were measured on a GE ultrasound machine at each consultation (0, 1, and 3 months). The LVEF was measured by the Simpson Biplane method. A summary of the report was archived in the patient's medical record and the department's myocarditis registry.

Coronary angiograms were performed at the first visit, on GE Optima Cath Lab with radial 6F access and Judkins left and right sheaths. A summary of the report was archived in the patient's medical record and the department's myocarditis registry.

MRIs were performed in two other cardiac MRI reference centers on GE 1.5 Tesla machines at the first visit. A summary of the report was archived in the patient's medical record and the department's myocarditis registry.

Biases:

Selection bias: In order to reduce these biases and make the study population as representative as possible of daily practice, we did not limit the origin of patients whose recruitment was successive.

Verification bias: All patients benefited from the reference test (MRI to confirm myocarditis).

Interpretation bias: A double-blind determination was made by two echocardiographists; the results were averaged if the difference in LVFE was <10% at baseline, one, and three months for all patients.

Disease evolution bias: To avoid this situation, the maximum delay between suspected diagnosis (TTE and troponins) and confirmation by cardiac MRI was one month (MRI studies suggested myocardial recovery after a delay of 3–6 months [7]).

Study size: We consecutively included all patients with the inclusion criterion (non-fulminant COVID-19-associated myocarditis) from August to December 2021, bringing the total number of patients to 39. After applying the exclusion criterion (patient with HF and reduced LVEF), 33 patients were retained in this work.

Quantitative variables: Based on the work described in the literature, we divided our patients according to the evolution of their LVEF into two groups LVEF<50% and LVEF>50%.

In order to assess the impact of age on LVEF recovery, patients were classified as patients aged > 60 years and those aged < 60.

Statistical methods: All data were collected on the EPI-INFO 7 software. Results were expressed as a percentage for qualitative variables and average ± standard deviation (SD) for quantitative variables. Bivariate analyses of all parameters according to LVEF evolution subgroups were carried out according to the Fisher test. A *p*-value of <0.10 was considered statistically significant.

RESULTS

Participants: A total of 39 patients were included in our study. After the analysis of the exclusion criterion, six were excluded, bringing the final number to 33. These all participated in the inclusion visit and the one- and three-month control visit (Figure 1).

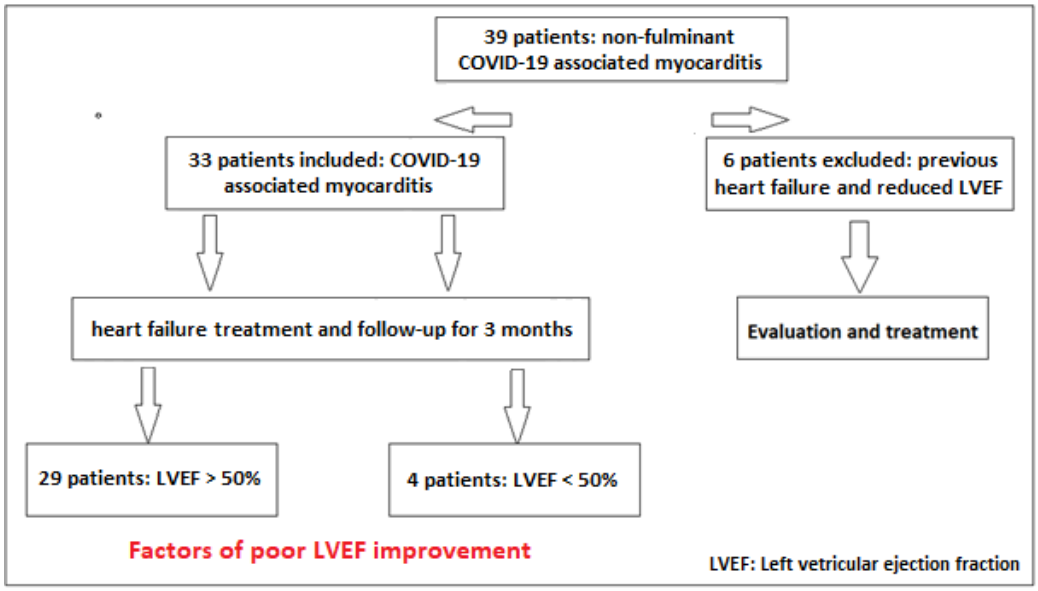

Figure 1. Study the flow diagram

Descriptive data: This observational single-center study included 33 patients (19 men and 14 women) aged 30–61 years with COVID-19-associated myocarditis strongly suspected on echocardiography and biology (significant elevation of troponin) and confirmed on cardiac MRI made less than one month after.

All patients had a non-severe form of COVID-19 (SpO2 ≥ 94%, respiratory rate <30 breaths/min, lung involvement <50% on chest CT scan, and admitted in non-ICU) and a non-fulminant form of myocarditis (hemodynamically stable).

Following chest pain and the observation of repolarization abnormalities, these patients were referred to our service. All patients received echocardiography, coronary angiography, and cardiac MRI (with a maximum delay of one month).

All patients (100%) had ST-T wave changes (21 patients with negative T waves, 7 with ST deviation, and 5 with T and ST abnormality).

The mean LVEF in our series at baseline was 44.3% +/- 6.3 (30–52%) with an average troponin level of 480 times normal (20 to 2,100 times normal).

The coronary angiography revealed two cases of in situ thrombosis, without significant coronary stenosis, which required urgent revascularization (MRI was in favor of myocarditis in these two cases); the other patients did not have a critical or unstable coronary lesion that could explain the elevation of troponins.

MRI confirmed subepicardial involvement in 100% of patients.

Treatment with beta-blocker (bisoprolol 5–10 mg) and ACE inhibitor (ramipril 2.5–10 mg) was initiated in all patients; spironolactone (37.5 mg) was introduced in only 13 patients with LVEF <40%. Furosemide was introduced only in cases of congestive signs (17 patients or 51.5%) and was discontinued in 11 patients after clinical improvement. None of our patients received ARNI (sacubitril/valsartan) or SGLT2 inhibitor (dapagliflozin) at baseline.

A systematic corticosteroid therapy was introduced in the COVID-19 treatment protocol for all of our patients. They all were followed for three months without loss of follow-up or death.

Outcome data: A clinical, electrical, biological, and echocardiographic control (with double-blind determination performed by two echocardiographers and results averaged if the difference was <10% concerning the LVEF) was performed at one and three months for all patients.

Eight patients presented a pericardial

effusion of variable importance during this follow-up, without tamponade, which regressed in five with the introduction of colchicine.

Main results: The evolution under treatment resulted in a significant improvement in LVEF (LVEF>50%) in 29 of our patients. One patient with LVEF at 38% presented with incessant ventricular tachyarrhythmia that necessitated an implantable cardioverter defibrillator. The final three patients kept an LVEF decreased to 42, 45, and 46% each and are still under treatment and supervision without the introduction of ARNI or SGLT2 inhibitors.

The improvement in psychological status was appreciated by our psychologist, who estimated that 25 patients felt better in themselves, although she detected depressive disorders in eight patients (24.2%).

We tried to identify factors of poor LVEF evolution (LVEF <50% at three months of follow-up) (Table 1).

Table 1. Influence of different factors on LVEF improvement

Exposure		LVEF < 50%: 4 patients		LVEF > 50%: 29 patients		*p*
		Number	%	Number	%	
Gender: male		3	15.8%	16	84.2%	0.62
Age	**> 60 years**	3	30%	7	70%	**0.07**
Congestive signs		2	11.8%	15	88.2%	1.00
ECG abnormality	**T**	3	14.3%	18	85.7%	1.00
	ST	0	0%	7	100%	0.56
	T and ST	1	25%	4	75%	0.42
Coronary lesion		1	50%	1	50%	0.23
Serum troponin level >1,200		3	42.9%	4	57.1%	**0.02**
Pericardial effusion		3	37.5%	5	62.5%	**0.035**
Combined: age, troponin, effusion		2	100%	0	0%	**0.01**

- Gender did not seem to influence the LVEF evolution (♂: 3/19 VS ♀: 1/14 kept an LVEF<50% at 3 months, *p*=0.62).

- Age >60 years was associated with poor LVEF recovery (age: 3/10 v. young: 1/23 kept an LVEF<50% at 3 months, *p*=0.07) (Figure 2).

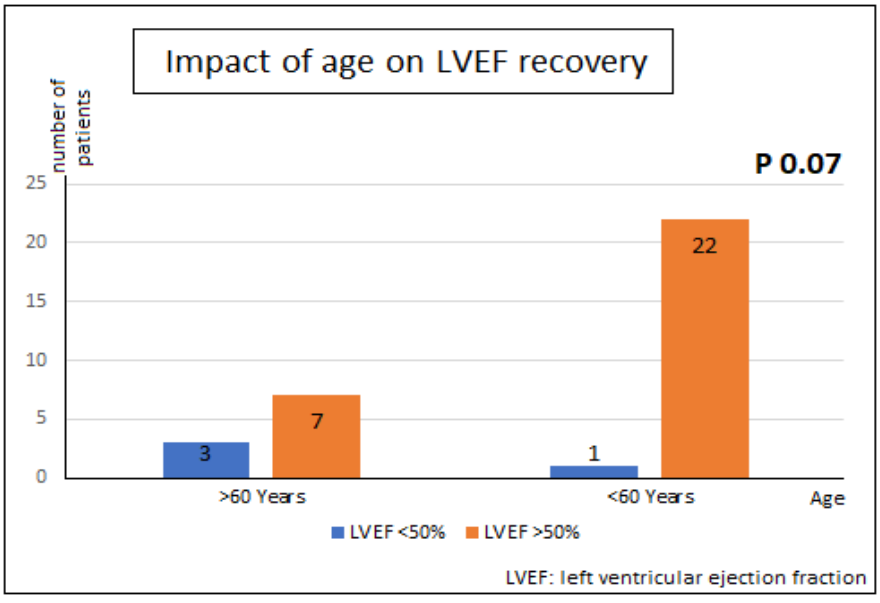

Figure 2. Impact of age on LVEF recovery

- The existence of congestive signs did not seem to influence the course of LVEF (2/17 patients with congestive signs against 2/16 without congestive signs kept an LVEF<50% at 3 months, *p*=1.00).

- The existence of an anomaly of the T wave or the ST or association of the two did not seem to influence the course of LVEF (3/21 patients with T abnormalities, none/7 with ST deviation and 1/5 patients with T and ST abnormality kept LVEF<50% at 3 months, *p*=1.00, 0.56 and 0.50, respectively).

- The existence at coronary angiography of a significant coronary lesion also did not seem to influence the LVEF evolution (1/2 patients with coronary lesion kept an LVEF<50% at 3 months, *p*=0.23).

- A serum troponin level >1,200 times the normal seemed to influence the poor LVEF evolution (3/7 patients' troponin >1,200 v. 1/26 patients' troponin <1,200 kept LVEF<50% at 3 months, *p*=0.02) (Figure 3).

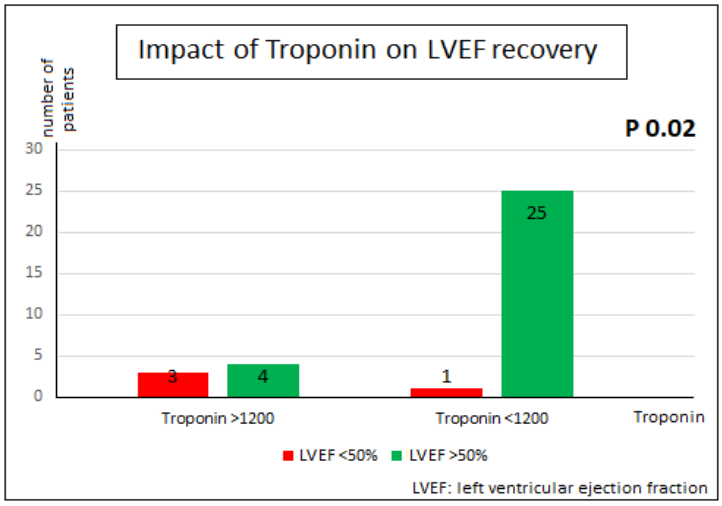

Figure 3. Impact of Troponin on LVEF recovery

- The appearance of pericardial effusion also seemed to influence the poor LVEF evolution (3/8 patients with effusion v. 1/25 patients without effusion kept LVEF<50% at 3 months, *p*=0.035) (Figure 4).

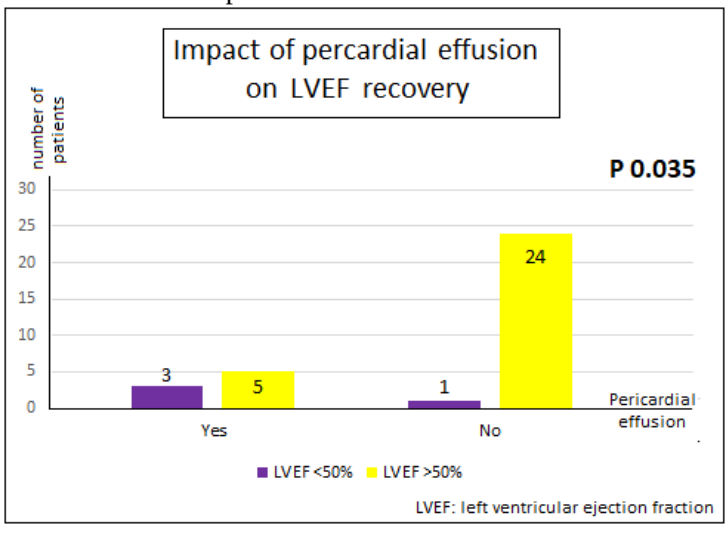

Figure 4. Impact of pericardial effusion on LVEF recovery

- A combined criterion associating age>60 years, troponin level>1,200 times normal, and pericardial effusion seems to be a good predictor of a poor LVEF progression (2/2 patients with the combined endpoint v. 2/31 without the combined endpoint kept an LVEF<50% at months, p=0.01) (Figure 5).

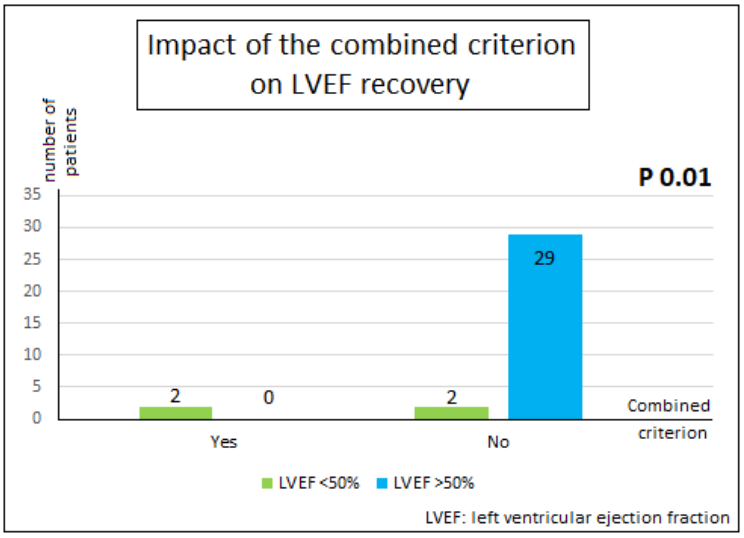

Figure 5. Impact of the combined criterion on LVEF recovery

DISCUSSION

Key results: Acute non-fulminant forms of COVID-19-associated myocarditis appear to have a good prognosis with LVEF recovery in 87.88% of cases.

Severe rhythmic complications of COVID-19-associated myocarditis in our series were rare compared to hemodynamic complications and pericardial effusions.

Factors that may influence poor LVEF recovery (LVEF<50% at 3 months of treatment) are age >60 years (p=0.07), troponin levels >1,200 times normal (p=0.02), the onset of pericardial effusion (p=0.035) and the combined criterion of the three: age, troponins, and effusion (p=0.01).

The psychological impact of COVID-19 cardiac involvement is poorly understood and seems to concern a quarter of the patients.

There are very limited data on the prognosis of COVID-19-associated myocarditis in the literature. Patients with elevated serum troponin or low LVEF during their disease have worse outcomes and higher mortality [16, 17], but there is no data about the cut-off value. The minimum duration of medical therapy remains to be determined.

Limitations: The small number of patients in our series does not allow the identification of predictive factors of the installation of myocarditis during COVID-19 infection.

The absence of fulminant forms in our series is a selection bias that probably explains the absence of mortality at three months, while the mortality from COVID-19-associated myocarditis is given at >30% in the literature [10, 18].

The systematic prescription of corticosteroids to all our patients made it impossible to analyze objectively the impact of corticosteroid therapy on the course of COVID-19-associated myocarditis. The RECOVERY trial showed a benefit of dexamethasone in COVID-19 patients with severe respiratory involvement but there are no data to apply this treatment to COVID-19-associated myocarditis [19, 20].

The impact of heart failure treatment could also not be assessed, as all patients had the same treatment regimen. Many controversies have emerged regarding the potential imputability of ACEs in the aggravation of patients with

COVID-19 disease. However, it has recently been shown that these treatments do not influence the prognosis [21].

Finally, administration of convalescent patients' plasma is safe, but controlled trials are needed to determine its efficacy [22].

Interpretation

Cardiac manifestations are not uncommon during COVID-19 infection. Many theories explain cardiac involvement during COVID-19 infection; these abnormalities are of ischemic origin (plaque fracture related to inflammatory stress, thrombosis in situ or coronary embolism related to hypercoagulability) or non-ischemic (more often fulminant myocarditis) [10, 12]. Its pathogenesis is not fully elucidated, but the most likely hypothesis to explain virus-induced myocarditis is probably the ACE2 receptors hypothesis. The latter are internalized during the virus penetration, resulting in a decrease in their expression on the surface of cardiomyocytes; this phenomenon limits the reduction of angiotensin II, leading to direct toxicity on cardiomyocytes apoptosis but also indirect toxicity through vasoconstriction, edema, and ischemia [10].

The elimination of an ischemic cause seems necessary, but the boundary between these two diagnoses remains blurred, as is the case for our two patients with coronary thrombosis and MRI in favor of myocarditis.

There is no specific therapy for non-fulminant COVID-19-associated myocarditis and the evolution seems favorable except in cases where ventricular tachyarrhythmias inhibit the patient's convalescence.

Patients with predictors of poor progress should have more intensive initial treatment and longer follow-up

Generalizability: The results of this work are very promising but should be confirmed by a greater prospective multicentric study.

What we know:

- COVID-19-associated myocarditis is a real entity with most often fulminant forms.
- Mortality from COVID-19 associated myocarditis is >30%.
- The treatment is based on classic heart failure treatment, with ongoing trials regarding corticosteroids and immunosuppressant agents.

What this study adds:

- Acute non-fulminant forms of myocarditis to COVID-19 exist and are of good prognosis (0% mortality and LVEF recovery at 3 months in 87.88% in our series).
- Predictors of poor LVEF recovery at three months are age>60 years, troponin levels >1,200 times normal, and the onset of pericardial effusion.

Informed consent:

All participants gave their informed consent to participate retrospectively in this study and share the results.

Ethics committee:

The hospital's ethics committee consented to this study and to share the results.

List of abbreviations

ACE 2: Angiotensin-converting Enzyme 2
CMV: Cytomegalovirus
CRP: C-reactive protein
ECG: Electrocardiogram
ECMO: Extracorporeal membrane oxygenation
EF: Ejection fraction
ESR: Erythrocyte sedimentation rate
HF: Heart failure
HIV: Human immunodeficiency virus
ICD: Implantable cardiac defibrillator
ICU: Intensive care unit
LVEF: Left ventricular ejection fraction
MRI: Magnetic resonance imaging
RASB: Renin angiotensin system blocker
RNA: Ribonucleic acid
TTE: Transthoracic echocardiography

REFERENCES

1. Richardson P, McKenna W, Bristow M, Maisch B, Mautner B, O'Connell J, *et al.* Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies. Circulation. 1996; 93(5): 841–842.
2. Bergelson JM, Cunningham JA, Droguett G, Kurt-Jones EA, Krithivas A, Hong JS, *et al.* Isolation of a common receptor for Coxsackie B viruses and adenoviruses 2 and 5. Science. 1997; 275(5304): 1320-1323.
3. Sarda L, Colin P, Boccara F, Daou D, Lebtahi R, Faraggi M *et al.* Myocarditis in patients with clinical presentation of myocardial infarction and normal coronary angiogram. J Am Coll Cardiol. 2001; 37(3): 786-792.
4. Narula J, Khaw BA, Dec GW, Palacios IF, Southern JF, Fallon JT *et al.* Brief report: recognition of acute myocarditis masquerading as acute myocardial infarction. N Engl J Med. 1993; 328(2): 100-104.
5. Felker GM, Boehmer JP, Hruban RH, Hutchins GM, Kasper EK, Baughman KL *et al.* Echocardiographic findings in fulminant and acute myocarditis. J Am Coll Cardiol. 2000; 36(1): 227-232.
6. Smith SC, Ladenson JH, Mason JW, Jaffe AS. Elevations of cardiac troponin I associated with myocarditis. Experimental and clinical correlates. Circulation. 1997; 95(1): 163-168.
7. Hinojar R, Foote L, Arroyo Ucar E, Jackson T, Jabbour A, Yu CY *et al.* Native T1 in discrimination of acute and convalescent stages in patients with clinical diagnosis of myocarditis: a proposed diagnostic algorithm using CMR. JACC Cardiovasc Imaging. 2015; 8(1): 37-46.
8. Holzmann M, Nicko A, Kühl U, Noutsias M, Poller W, Hoffmann W *et al.* Complication rate of right ventricular endomyocardial biopsy via the femoral approach: a retrospective and prospective study analyzing 3,048 diagnostic procedures over an 11-year period. Circulation. 2008; 118(17): 1722-1728.
9. Mason JW, O'Connell JB, Herskowitz A, Rose NR, McManus BM, Billingham Me *et al.* A clinical trial of immunosuppressive therapy for myocarditis. N Engl J Med. 1995; 333(5): 269-275.
10. Filippetti L, Selton-Suty C, Huttin O, Pace N, Marie P Y, Juillière Y. Myocardite et COVID-19, mythe ou réalité ? [Myocarditis and COVID-19, myth or fact?]. Archives des maladies du coeur et des vaisseaux Pratique. 2020; 2020(291): 5-10.
11. Peretto G, Sala S, Caforio A L P. Acute myocardial injury, MINOCA, or myocarditis? Improving characterization of coronavirus-associated myocardial involvement. Eur Heart J. 2020; 41(22): 2124-2125.
12. Doyen D, Moceri P, Ducreux D., Dellamonica J. Myocarditis in a patient with COVID-19: a cause of raised troponin and ECG changes. Lancet Lond Engl. 2020; 395(10235): 1516.
13. Lippi G, Lavie C J, Sanchis-Gomar F. Cardiac troponin I in patients with coronavirus disease 2019 (COVID-19): evidence from a meta-analysis. Prog Cardiovasc Dis. 2020; 63(3): 390-391.
14. Ali M, Shiwani H A, Elfaki M Y, Hamid M, Pharithi R, Kamgang R *et al.* COVID-19 and myocarditis: a review of literature. Egypt Heart J. 2022; 74(1): 23.
15. Abu Mouch S, Roguin A, Hellou E, Ishai A, Shoshan U, Mahamid L *et al.* Myocarditis following COVID-19 mRNA vaccination. Vaccine. 2021; 39(29): 3790-3793.

16. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z *et al.* Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan: A retrospective cohort study. Lancet. 2020; 395(10229): 1054-1062.
17. Agdamag ACC, Edmiston JB, Charpentier V, Chowdhury M, Fraser M, Maharaj VR *et al.* Update on COVID-19 Myocarditis. Medicina (Kaunas). 2020; 56(12): 678.
18. Rathore SS, Rojas GA, Sondhi M, Pothuru S, Pydi R, Kancherla N *et al.* Myocarditis associated with Covid-19 disease: A systematic review of published case reports and case series. Int J Clin Pract. 2021; 75(11): e14470.
19. Kamarullah W, Nurcahyani, Mary Josephine C, Bill Multazam R, Ghaezany Nawing A, Dharma S. Corticosteroid Therapy in Management of Myocarditis Associated with COVID-19; a Systematic Review of Current Evidence. Arch Acad Emerg Med. 2021; 9(1): e32.
20. Horby P, Lim WS, Emberson JR, Mafham M, Bell JL, Linsell L *et al.* The RECOVERY collaborative group. Dexamethasone in hospitalized patients with COVID-19 – preliminary report. N Engl J Med. 2021; 384(8): 693-704.
21. Fosbøl E.L., Butt J.H., Østergaard L., Andersson C., Selmer C., Kragholm K. Association of angiotensin-converting enzyme inhibitor or angiotensin receptor blocker use with COVID-19 diagnosis and mortality. JAMA. 2020; 324(2): 168-177.
22. Focosi D, Franchini M, Joyner MJ, Casadevall A. Are convalescent plasma stocks collected during former COVID-19 waves still effective against current SARS-CoV-2 variants? *Vox Sang.* 2022; 117(5): 641-646.

التهاب عضلة القلب المرتبط بـ " :COVID-19 حول سلسلة أحادية المركز من 33 حالة"

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الملخص

الخلفية والأهداف : يعرف التهاب عضلة القلب بارتشاح التهابي لعضلة القلب مع نخر من أصل غير إقفاري ولديه ثلاثة أشكال: خاطف، حاد ومزمن. يتم توجيه التشخيص من خلال العرض السريري، تخطيط القلب، تخطيط صدى القلب والبيولوجيا ويتم تأكيده بواسطة التصوير بالرنين المغناطيسي وخزعة عضلة القلب. يعتمد النذير على المظاهر السريرية، ميزات تخطيط صدى القلب ومستويات تروبونين المصل. أما الرعاية فتعتمد على علاج قصور القلب .(HF) يعاني العالم منذ عامين من جائحة مرتبطة بتفشي فايروس SARS-CoV2 الذي يمكن أن يؤثر على القلب بآفات إقفاريه أو غير إقفاريه (التهاب عضلة القلب الذي غالبًا ما يكون خاطفًا) والذي يبقى علاجه غير محدد إذ أسفرت التجارب التي أجريت على الكورتيكوستيرويدات ومثبطات المناعة عن نتائج متضاربة.
وصف الطرائق التطورية لالتهاب عضلة القلب المرتبط بـ COVID-19 وتحديد عوامل التنبؤ بالتعافي الضئيل للكسر القذفي المنخفض للبطين الأيسر (LVEF <50٪ (تحت علاج قصور القلب .

منهجية الدراسة : شملت هذه الدراسة الأحادية المركز والقائمة على الملاحظة، بأثر رجعي، في عام 2021، المرضى الذين يعانون من التهاب عضلة القلب غير الخاطف المرتبط بـ COVID-19 المشتبه بهم في تخطيط صدى القلب والبيولوجيا والذين تم تأكيد تشخيصهم بالتصوير بالرنين المغناطيسي. كما استبعد المرضى الذين يعانون سابقا من قصور القلب مع كسر قذفي منخفض للبطين الأيسر (ن = 06). ثم تم تقسيم المرضى إلى مجموعتين وفقًا لـ LVEF بعد 3 أشهر (LVEF> 50 ٪ VS LVEF <50٪ (ومقارنتهما لتحديد العوامل التي تتنبأ بالتعافي الضعيف لـ . LVEF

النتائج: تم تضمين 33 مريضًا (19♂ / 14♀) تتراوح أعمارهم بين 30−61 عامًا مصابين بالتهاب عضلة القلب المرتبط بـ COVID-19 الحاد، 100 ٪ منهم يعانون من اضطرابات عودة استقطاب تخطيط القلب؛ كان متوسط LVEF عند خط الأساس 44.3٪ +/− 6.3 (30−52٪) بمتوسط مستوى تروبونين 480 ضعف العتبة (20−2100). أعطي علاج Betablockers و RASBلجميع المرضى، سبيرونولاكتون (37.5 مجم) لـ 13 مريضاً يعانون من LVEF أقل من 40٪ وفوروسيميد في حالة وجود علامات الاحتقان (17 مريضاً / 51.5٪). ثم أجريت المراقبة السريرية، الكهربائية، البيولوجية وتخطيط صدى القلب بعد 1 و3 أشهر لجميع المرضى. طور ثمانية مرضى انصباب غير معقد للتامور. لوحظ تحسن كبير في الكسر القذفي للبطين الأيسر LVEF> 50 ٪ لدى 29 مريضًا، وتعرض مريض واحد بـ38 LVEF ٪ لاضطراب النظم البطيني المتسارع الذي استلزم زرع مقوم نظم القلب ومزيل الرجفان القابل للغرس، بينما احتفظ 3 مرضى على LVEF أقل من 50٪. لا يبدو أن الجنس، علامات الاحتقان، اضطرابات تخطيط القلب وتشوهات تصوير الأوعية التاجية تؤثر على تطور P) LVEF عند 0.62 ،1.00 ،1.00 ،0.56، 0.50 و0.23 على التوالي .(يبدو أن العمر> 60 عامًا، التروبونين> 1200 ضعف العتبة، الانصباب التأموري والمعيار المشترك للثلاثة يعتبرون مؤشرات جيدة للتعافي الضئيل للكسر القذفي المنخفض للبطين الأيسر P) عند 0.07 ،0.02 ،0.035 و0.01 على التوالي.(

المناقشة: يفسر غياب الأشكال الخاطفة في سلسلتنا غياب الوفيات بعد 3 أشهر (والتي تفوق 30٪ في بعض الدراسات). يتمتع التهاب عضلة القلب المرتبط بفيروس كورونا المستجد غير الخاطف بنذير جيد مع انتعاش LVEF في 87.88٪. عوامل التعافي الضئيل للكسر القذفي المنخفض للبطين الأيسر هي العمر> 60 عامًا، التروبونين> 1200 ضعف العتبة، الانصباب التامور والمعيار المشترك للثلاثة P) على التوالي عند 0.07، 0.02، 0.035 و0.01 .(جعلت الوصفة الروتينية للكورتيكوستيرويدات في بروتوكول COVID-19 من المستحيل تحليل تأثير هذا الأخير على التهاب عضلة القلب المرتبط بـ.COVID-19

الاستنتاجات : يعد تلف القلب شائعًا أثناء الإصابة بـ COVID-19 حيث يمكن أن تكون هذه النوبات القلبية إقفاريه أو غير إقفاريه. لا يوجد علاج محدد لالتهاب عضلة القلب المرتبط بـ COVID-19 غير الخاطف ويبدو أن التطور موات على المدى القصير والمتوسط. يجب أن يستفيد المرضى الذين يعانون من عوامل التنبؤ بالتعافي الضئيل للكسر القذفي المنخفض للبطين الأيسر من متابعة أطول من العادة .وأعطى جميع المشاركين موافقتهم المستنيرة على المشاركة في هذه الدراسة وبث النتائج.

الكلمات الدالة: سلسلة مرضى، التهاب عضلة القلب الخاطف، مستقبلات الإنزيم المحول للأنجيوتنسين 2، SARS-CoV-2، تروبونين، تصوير القلب بالرنين المغناطيسي.