Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

TABLE 1 Echocardiographic Structural and Systolic/Diastolic Functional Indices in COVID-19 Patients With and Without Cardiac Injury

	Without Cardiac Injury (n = 89)	With Cardiac Injury (n = 26)	p Value
Septal wall thickness, mm	10.7 ± 2.3	11.5 ± 2.3	NS
LV posterior wall thickness, mm	9.8 ± 1.2	10.8 ± 1.1	NS
LV end-diastolic diameter, mm	48.7 ± 4.8	49.9 ± 6.7	NS
LV end-systolic diameter, mm	33.4 ± 3.8	35.4 ± 5.2	NS
Mitral peak A velocity, m/s	0.72 ± 0.32	0.73 ± 0.3	NS
Mitral peak E/A ratio	0.91 ± 0.3	0.88 ± 0.3	NS
LV ejection fraction %	56.2 ± 4.4	54.4 ± 4.6	NS
RV basal tract diameter, mm	31.2 (2.6)	35.8 (4.2)	<0.01
RV mid-tract diameter, mm	28.6 (3.7)	32.4 (3.9)	<0.01
Tricuspid peak E velocity, m/s	0.15 ± 0.08	0.12 ± 0.06	<0.01
Tricuspid peak A velocity, m/s	0.13 ± 0.08	0.16 ± 0.06	<0.01
Tricuspid peak E/A ratio	1.1 ± 0.6	0.77 ± 0.5	<0.01
TRV, m/s	2.9 ± 0.5	3.4 ± 0.6	<0.001
PASP, mm Hg	36.1 ± 5.3	46.8 ± 4.8	<0.001
MPAP, mm Hg	23.6 ± 2.7	29.6 ± 2.9	<0.001
TAPSE, mm	20.3 ± 4.7	15.4 ± 3.2	<0.001

Values are mean ± SD or n (%).

COVID-19 = coronavirus disease-2019; E/A = ratio of peak velocity blood flow from LV relaxation in early diastole [E wave] to peak velocity flow in late diastole caused by atrial contraction [A wave]); LV = left ventricle; MPAP = mean pulmonary artery pressure; NS = not significant; PASP = pulmonary artery systolic pressure; RV = right ventricle; TAPSE = tricuspid annular systolic plane excursion; TRV = tricuspid regurgitation velocity.

portably to facilitate sanitation, and the examination was performed by a single operator, reducing patient contact with the device. RV end-diastolic diameters and mPAP values were significantly increased in patients with cardiac injury, whereas the tricuspid inflow E/A ratio (the ratio of peak velocity blood flow from LV relaxation in early diastole [E wave] to peak velocity flow in late diastole caused by atrial contraction [A wave]) and TAPSE were reduced (Table 1). All patients were treated with oxygen, and the percentages of use of noninvasive ventilation and invasive mechanical ventilation were 16.5 % (19 patients) and 9.5 % (11 patients), respectively. A greater proportions of patients with cardiac injury required invasive or noninvasive mechanical ventilation (17 of 26 [65.4%] vs. 20 of 89 [22.4%], respectively; $p < 0.001$). The common complication in patients with cardiac injury was acute respiratory distress syndrome (14 of 26 [63.5%] vs. 15 of 89 [16.8%], respectively; $p < 0.001$). Patients with cardiac injury had higher mortality than those without cardiac injury (13 of 26 [50%] vs. 8 of 89 [7.8%], respectively; $p < 0.0001$). In a Cox regression model in COVID-19 patients, troponin levels (hazard ratio [HR]: 4.33 [95% confidence interval [CI]: 1.87 to 8.49]; $p < 0.001$), partial pressure of oxygen (Pao_2) at admission (HR: 0.26 [95% CI: 0.15 to 0.74; $p < 0.01$]; mPAP (HR: 3.8 [95% CI: 1.72 to 6.39] $p < 0.001$), and RV TAPSE (HR: 0.5 [95% CI: 0.22 to 0.74] $p < 0.001$) emerged as the only independent predictors of in-hospital death.

Despite the high prevalence of normal CUS, it was found that increases in mPAP and RV dysfunction were common conditions among patients with COVID-19 pneumonia and cardiac injury, associated with higher risk of in-hospital mortality. Use of empiric therapeutic anticoagulation in certain COVID-19 patients who did not have deep vein thrombosis has been advocated, but this remains controversial because of the risk of major bleeding and lack of data about the true incidence of pulmonary embolism (4). However, assessment of RV function and of pulmonary pressures during the recovery of these patients may represent a key point in the prognostic stratification.

Antonello D'Andrea, MD, PhD*
Raffaella Scarafile, MD
Lucia Riegler, MD
Biagio Liccardo, MD
Fabio Crescibene, MD
Rosangela Cocchia, MD
Eduardo Bossone, MD

*Department of Cardiology
University of Campania Luigi Vanvitelli
Umberto I° Hospital - Nocera Inferiore (Salern)
Corso Vittorio Emanuele 121A
80121, Naples
Italy
E-mail: antonellodandrea@libero.it

https://doi.org/10.1016/j.jcmg.2020.06.004

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

REFERENCES

1. Guo T, Fan Y, Chen M, et al. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol 2020;5:811–8.

2. Bonow RO, Fonarow GC, O'Gara PT, Yancy CW. Association of coronavirus disease 2019 (COVID-19) with myocardial injury and mortality. JAMA Cardiol 2020;5:751–3.

3. Bossone E, D'Andrea A, D'Alto M, et al. Echocardiography in pulmonary arterial hypertension: from diagnosis to prognosis. J Am Soc Echocardiogr 2013;26:1–14.

4. Obi AT, Tignanelli CJ, Jacobs BN, et al. Empirical systemic anticoagulation is associated with decreased venous thromboembolism in critically ill influenza A H1N1 acute respiratory distress syndrome patients. J Vasc Surg Venous Lymphat Disord 2019;7:317–24.

Coronary Artery Calcification and Complications in Patients With COVID-19

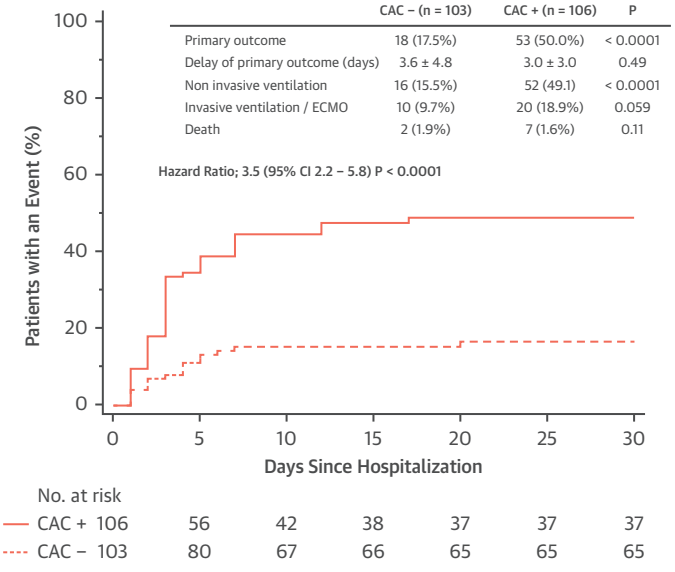
In the coronavirus disease-2019 (COVID-19) pandemic, early risk stratification of severe forms is essential. Patients with COVID-19 and cardiovascular

(CV) risk factors such as hypertension and diabetes have higher morbidity and mortality (1). In patients at high CV risk, coronary artery calcification (CAC) is associated with a higher incidence of nonfatal and fatal outcomes (2). Chest CT has become a key tool for the diagnosis of COVID-19, but CAC detection is not carried out routinely (3). Yet CAC could represent an easy integrative marker of worse prognosis in patients with COVID-19.

From March 15, 2020 to May 3, 2020, we performed a cross-sectional study including consecutive patients hospitalized with COVID-19, 40 to 80 years of age, who underwent noncontrast chest computed tomography on the day of admission. Patients with histories of CV disease were excluded. We analyzed the presence or absence of CAC (CAC+ and CAC−, respectively), defined as any area ≥1 mm² with density >130 Hounsfield units (HU) on the known coronary tract. Agatston score was also measured, despite technical limitations (no electrocardiographic gating and slice thickness 2.0 mm) (4). Analysis was performed by 2 certified operators blinded to clinical history (1 radiologist and 1 cardiologist). The primary outcome was the first occurrence of mechanical noninvasive or invasive ventilation, extracorporeal membrane oxygenation, or death (scores of 5, 6, and 7 on the World Health Organization Blueprint scale) (5) within 30 days following hospital admission. Because CAC is highly correlated with age, the primary outcome was segmented by median age group. The study was approved by the Paris university hospital ethics committee.

Among 356 patients screened, 147 were excluded (54 had previous CV disease, and 93 did not undergo chest computed tomography within 24 h of admission). Ultimately, 209 consecutive patients were included. The median age was 62 years (interquartile range: 51 to 70 years), 72% were men, and all patients were at grade 3 or 4 on the World Health Organization Blueprint scale at entry (hospitalization without or with nasal oxygen). CAC was detected in 106 patients (50.7%). The primary outcome occurred in 50.0% of CAC+ patients compared with 17.5% of CAC− patients ($p < 0.0001$) (**Figure 1**). Using Kaplan-Meier analysis, CAC was significantly associated with the primary outcome (hazard ratio [HR]: 3.5; 95% confidence interval [CI]: 2.2 to 5.8; $p < 0.0001$) (**Figure 1**). Among patients <62 years of age ($n = 104$), CAC was detected in 32%, and the primary outcome occurred in 55% of CAC+ patients compared with 20% of CAC− patients (HR: 5.4; 95% CI: 2.4 to 12.2; $p < 0.0001$). Among patients ≥62 years of age ($n = 105$), CAC was detected in 69%, and the primary outcome occurred in 48% of CAC+ patients compared with 13% of CAC− patients (HR: 3.2; 95% CI: 1.6 to 6.3; $p = 0.02$). In a multivariate analysis with a Cox proportional hazards model including age, sex, hypertension, smoking, and diabetes, CAC was independently associated with the primary outcome (HR: 4.4; 95% CI: 2.4 to 8.0; $p < 0.0001$).

FIGURE 1 Event Rate and Primary Outcome According to CAC



Event rate and primary outcome (first occurrence of noninvasive or invasive mechanical ventilation, extracorporeal membrane oxygenation [ECMO], or death during hospitalization) according to absence or presence of coronary artery calcification (CAC) on chest computed tomography performed for the diagnosis of coronavirus disease-2019. The cumulative incidences were estimated using Kaplan-Meier analysis. CI = confidence interval.

The median Agatston score was 8 (interquartile range: 0 to 116); 28.7% of patients had Agatston scores >100, and 2.9% had scores >400. The crude HR for the primary outcome for Agatston score (per quartile) was 1.6 (95% CI: 1.3 to 2.0; $p < 0.0001$), and using the same Cox proportional hazards model, the HR for Agatston score was 1.8 (95% CI: 1.5 to 2.3; $p < 0.0001$). In CAC+ patients, mean calcification density was 457 ± 176 HU in patients without events, compared with 387 ± 153 HU in patients with events ($p = 0.03$).

There was 1 acute ST-segment elevation myocardial infarction and no stroke during follow-up. A significant increase in peak high-sensitivity cardiac troponin I was detected in 9.1% of CAC+ patients compared with 3.4% of CAC− patients ($p = 0.16$).

Our study shows that the presence and extent of CAC are associated with a worse prognosis in

hospitalized patients with COVID-19. The severity of immune response, endothelial dysfunction, and myocardial stress due to COVID-19 could be exacerbated in patients with subclinical coronary atherosclerosis.

Jean Guillaume Dillinger, MD, PhD
Fatima Azzahra Benmessaoud, MD
Théo Pezel, MD
Sebastian Voicu, MD, PhD
Georgios Sideris, MD, PhD
Naima Chergui, MD
Lounis Hamzi, MD
Anthony Chauvin, MD
Pierre Leroy, MD
Jean François Gautier, MD, PhD
Damien Sène, MD, PhD
Patrick Henry, MD, PhD*
on behalf of the COVID Research Group of Lariboisiere Hospital

*Department of Cardiology
Lariboisiere Hospital
Assistance Publique-Hôpitaux de Paris
2 rue A Pare
75010 Paris
France
E-mail: patrick.henry@aphp.fr

https://doi.org/10.1016/j.jcmg.2020.07.004

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

REFERENCES

1. Guan W-J, Ni Z-Y, Hu Y, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med 2020;382:1708–20.

2. Erbel R, Möhlenkamp S, Moebus S, et al. Coronary risk stratification, discrimination, and reclassification improvement based on quantification of subclinical coronary atherosclerosis: the Heinz Nixdorf Recall study. J Am Coll Cardiol 2010;56:1397–406.

3. Ye Z, Zhang Y, Wang Y, Huang Z, Song B. Chest CT manifestations of new coronavirus disease 2019 (COVID-19): a pictorial review. Eur Radiol 2020;30:4381–9.

4. Blaha MJ, Mortensen MB, Kianoush S, Tota-Maharaj R, Cainzos-Achirica M. Coronary artery calcium scoring. J Am Coll Cardiol Img 2017;10:923–37.

5. Cao B, Wang Y, Wen D, et al. A trial of lopinavir-ritonavir in adults hospitalized with severe COVID-19. N Engl J Med 2020;382:1787–99.

A Plea Not to Forget Infective Endocarditis in COVID-19 Era

Although recent publications have stressed that the diagnosis and management of acute coronary syndrome should not be neglected during coronavirus disease-2019 (COVID-19) pandemic (1), the issue of infective endocarditis (IE) has not been addressed. IE is associated with a mortality rate of more than 20%, and even 50% when surgery is indicated and not performed (2,3).

We compared the number of cases of IE diagnosed in 2 regional tertiary reference centers from Belgium and France between January 24, 2020 (first COVID-19 case diagnosed in France), and April 30, 2020 (2 weeks after the pandemic peak in France and Belgium), with the number of cases of IE during the same time frame last year. Additionally, we compared the rate of in-hospital complications and mortality between the 2 periods. Data were extracted from a dedicated database compiling all IE cases referred from these regions. This survey was approved by the local ethical committees of both centers and was carried out in accordance with the ethical principles for medical research involving human subjects established by the Declaration of Helsinki, protecting the privacy of all participants.

The percentage of diagnosed IE decreased by 33% during the COVID-19 pandemic (47 patients in 2020 vs. 70 patients in 2019) (**Figure 1**). Furthermore, we observed a worse prognosis in patients diagnosed with IE during the pandemic (i.e., cerebral embolism rate was 18.5% [n = 13] in 2019 vs. 56% [n = 26] in 2020). In-hospital IE mortality reached 61% (n = 29) during the pandemic versus 31% (n = 22) in 2019, which was similar to EURO-ENDO (European Infective Endocarditis Registry) registry results (2). This probably also underlines that patients were referred late, but this hypothesis requires further analysis.

These findings might have several explanations. In the current COVID-19 pandemic, symptoms related to endocarditis might be incorrectly attributed to a diagnosis of SARS-CoV-2 (severe acute respiratory syndrome-coronavirus-2) infection. Moreover, patients might avoid medical care, and hospital resources might be limited due to reorganization during the crisis.

Despite transesophageal echocardiography (TEE) being a very sensitive examination for the diagnosis of IE, current recommendations suggest that its use should be restricted due to high risk of contamination (4). Consequently, we observed a decrease of 49% in the number of TEEs during the pandemic compared with the same time frame in 2019 (498 TEEs in 2019 vs. 244 TEEs in 2020). The substitution of TEE with transthoracic echocardiography might be an explanation for the worse prognosis of patients diagnosed with IE during the pandemic, who potentially have larger vegetations, which might be an indirect gauge of severity.