

etiological treatment. The general approach includes routine measures, such as infusion of amines or implantation of VA-ECMO as a bridge to recovery.¹ Myocardial inflammation underlies the acute myocarditis, and SARS-CoV-2 particles have recently been demonstrated in the myocardium of these patients.² In addition to respiratory and circulatory support measures, treatment includes the use of corticosteroids and immunoglobulins. Stress cardiomyopathy in COVID-19 may be triggered by catecholamine discharge secondary to hypoxemia or sepsis, by myocardial injury related to the systemic inflammatory process, by direct myocardial infection with the virus, or by a mixture of factors.³ Treatment consists of administration of amines and mechanical support in addition to the other therapeutic measures used in COVID-19 (table 1).

Pulmonary thromboembolism is common in the hypercoagulability state provoked by COVID-19 and can lead to cardiogenic shock with high mortality. All available measures should be applied, such as VA-ECMO, thrombolysis, and percutaneous treatment, particularly if there is a contraindication for thrombolysis or if this measure fails. Another thrombotic complication that can cause cardiogenic shock is the development of acute coronary thrombosis in segments proximal to the main coronary arteries. This case was treated with percutaneous stent placement, as is recommended in the related clinical practice guidelines.

One consideration to mention is the absence of pathological studies in these patients, as they were not allowed in our setting at the beginning of the pandemic.

This limited series illustrates the variety and severity of cardiovascular manifestations in COVID-19 patients, including acute myocarditis, stress cardiomyopathy, acute coronary syndrome, and pulmonary thromboembolism.

CONFLICTS OF INTEREST

Á. Sánchez-Recalde is an associate editor of *Revista Española de Cardiología*. The editorial procedure established in the journal was followed to guarantee impartial management of the manuscript.

Pulmonary infarction secondary to pulmonary thromboembolism in COVID-19 diagnosed with dual-energy CT pulmonary angiography



Infarto pulmonar secundario a tromboembolia pulmonar en COVID-19 diagnosticada con angiotomografía computarizada pulmonar con energía dual

To the Editor,

We report the cases of 2 patients from Barcelona, Spain, admitted to the emergency department of our hospital secondary to COVID-19 (formerly known as SARS-CoV-2) pneumonia, confirmed with a real-time reverse-transcription polymerase chain-reaction test^{1,2}; both patients showed respiratory deterioration and elevated serum D-dimer levels. Figure 1 illustrates the case of a 32-year-old man, with no comorbidities or risk factors, admitted to our emergency department on day 14 after symptom onset with dry cough, asthenia, arthromyalgia, fever, and right pleuritic pain. A baseline electrocardiogram showed sinus rhythm, 97 bpm, normal PR interval (120 ms) and normal QRS complex (80 ms), aQRS 0°. QTc (Friderica) 415 msec. Echocardiography was not performed but initial physical examination showed systemic blood pressure values of 136/79 mmHg, regular rhythm with no murmurs, present and symmetrical distal pulses, and no signs of

Acknowledgements

We thank Dr. Luisa Salido Tahoces, Dr. José Luis Mestre-Barcia, Dr. Rosa Ana Hernández-Antolín, and Dr. Ana Pardo Sanz for their critical assessment and in-depth revision of the manuscript.

Ángel Sánchez-Recalde,^{a,*} Jorge Solano-López,^a Javier Miguelena-Hycka,^b Jesús Javier Martín-Pinacho,^c Marcelo Sanmartín,^a and José L. Zamorano^a

^aServicio de Cardiología, Hospital Universitario Ramón y Cajal, Madrid, Spain

^bServicio de Cirugía Cardiovascular, Hospital Universitario Ramón y Cajal, Madrid, Spain

^cServicio de Radiología, Hospital Universitario Ramón y Cajal, Madrid, Spain

* Corresponding author:

E-mail address: recalde@secardiologia.es (Á. Sánchez-Recalde).

Available online 27 April 2020

REFERENCES

- García-Carreño J, Sousa-Casasnovas I, Devesa-Cordero C, Gutiérrez-Ibañes E, Fernández-Aviles F, Martínez-Sellés M. Reanimación cardiopulmonar con ECMO percutáneo en parada cardiaca refractaria hospitalaria: experiencia de un centro. *Rev Esp Cardiol*. 2019;72:880–882.
- Tavazzi G, Pellegrini C, Maurelli M, et al. Myocardial localization of coronavirus in COVID-19 cardiogenic shock. *Eur J Heart Fail*. 2020. <http://dx.doi.org/10.1002/ehf.1828>.
- Meyer P, Degrauwe S, Van Delden C, Ghadri J-R, Templin C. Typical takotsubo syndrome triggered by SARS-CoV-2 infection. *Eur Heart J*. 2020. <http://dx.doi.org/10.1093/eurheartj/ehaa306>.

<https://doi.org/10.1016/j.rec.2020.04.012>
1885-5857/

© 2020 Sociedad Española de Cardiología. Published by Elsevier España, S.L.U. All rights reserved.

deep vein thrombosis. Laboratory data showed elevated ferritin levels (615 ng/mL), C-reactive protein (CRP) = 3.6 mg/dL, and increased interleukin-6 (IL-6) (144.7 pg/mL). Coagulation studies: prothrombin time (PT) 12 seconds, international normalized ratio 1.1, partial thromboplastin time (aPTT) 28.2 seconds. Lupus anticoagulant testing was positive. Immunoglobulin G and immunoglobulin M anticardiolipin antibodies were also tested with a negative result. D-dimer levels were elevated up to 2460 ug/L and therefore, due to high suspicion of pulmonary thromboembolism, dual-energy pulmonary computed tomography (CT) angiography (CTPA) was performed. CTPA confirmed bilateral thromboembolism associated with multiple opacities compatible with viral pneumonia (figure 1A,B). Iodine map images showed a triangular peripheral pulmonary infarction (figure 1C).

The patient received therapy with hydroxychloroquine at a loading dose of 400 mg on the first day followed by a maintenance dose of 200 mg/d for the next 4 days. Azithromycin 500 mg/d for 3 days and enoxaparin 80 mg/12 h for 10 days were also prescribed. Throughout the admission, the patient showed clinical improvement with no respiratory support requirements, maintaining oxygen saturation levels around 97% to 99% on room air. On the 10th day after admission, 24 days after symptom onset, the patient was discharged with good health status and was asymptomatic. Given the positivity to lupus anticoagulant auto-antibodies, thrombophilia testing will be performed in 3 months. A



Figure 1. A: computed tomography angiography maximum intensity projection oblique coronal reconstruction image showing filling defects (white arrows) in bilateral segmental and subsegmental branches of pulmonary arteries. B: transverse computed tomography image obtained with lung window settings showing wedge-shaped bilateral opacities with surrounding ground-glass opacities compatible with viral pneumonia. C: iodine map images showing a triangular peripheral area of decreased perfusion (yellow arrow) in the right lower, distal to PE (red arrow) lobe compatible with pulmonary infarction.

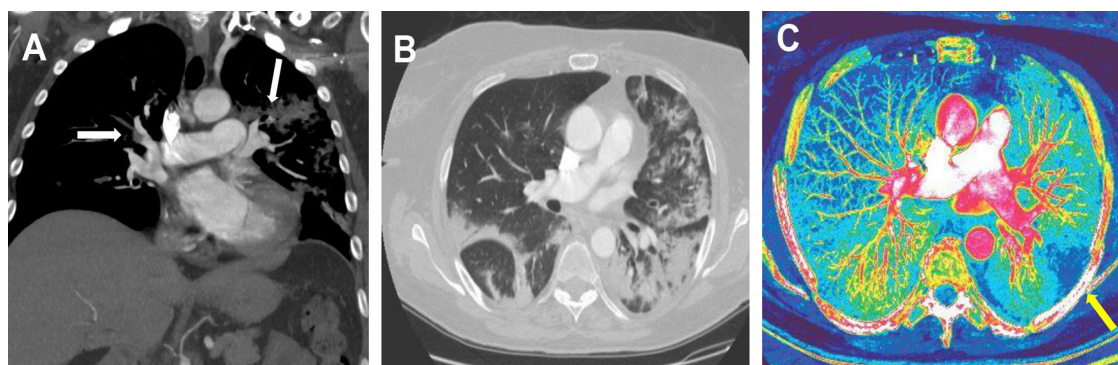


Figure 2. A: computed tomography angiography maximum intensity projection oblique coronal reconstruction image showing filling defects in bilateral segmental and subsegmental branches of pulmonary arteries. B: transverse computed tomography image obtained with lung window settings showing wedge-shaped bilateral opacities with surrounding ground-glass opacities compatible with viral pneumonia. C: iodine map images showing a peripheral, triangular and hypoperfused area in the left lower lobe (yellow arrow), inside the peripheral mnemonic opacities, suggestive of pulmonary infarction.

full-dose anticoagulation regimen (80 mg/12 h) was prescribed for 6 months.

Figure 2 illustrates the case of a 59-year-old woman, with a history of idiopathic hypertension (treated with ramipril 5 mg/d) and hypothyroidism (treated with levothyroxine 112 µg/d), without other risk factors or comorbidities, admitted to our hospital for 10 days with dry cough, myalgia, and fever. The baseline electrocardiogram showed sinus rhythm, 86 bpm, normal PR interval (131 ms) and normal QRS complex (93 ms), aQRS 0°. QTc (Friderica) 412 ms. Eocardiography was not performed, but initial physical examination showed systemic blood pressure of 116/78 mmHg, regular rhythm, and present and symmetrical distal pulses, without signs of deep vein thrombosis. Laboratory data showed elevated ferritin levels (1127 ng/mL), CRP = 9.5 mg/dL, and increased serum IL-6 (75,60 pg/mL). Coagulation studies: prothrombin time (PT) 10.7 seconds, international normalized ratio 1.09, partial thromboplastin time (aPTT) 33.6 seconds. D-dimer at admission was 1320 µg/L. The patient received initial treatment with hydroxychloroquine at a loading dose of 400 mg/12 h on day 1 followed by a maintenance dose of 200 mg/12 h for 4 days. She was also prescribed azithromycin 500 mg/d for 5 days, anticoagulant prophylaxis with enoxaparin (40 mg/d), methylprednisolone 70 mg/d for 5 days, and a single intravenous dose of tocilizumab (400 mg).

On day 9 after admission, the patient showed oxygen desaturation and reported chest pain. D-dimer elevation up to 6120 µg/L was observed (previous 1870 µg/L) and therefore, due to high suspicion of pulmonary thromboembolism, CTPA with dual-energy mode was obtained and confirmed a bilateral acute pulmonary thromboembolism associated with bilateral pulmo-

nary opacities compatible with viral pneumonia (figure 2A,B). Iodine map images depicted a peripheral pulmonary infarction (figure 2C). A full anticoagulant regimen with enoxaparin 60 mg/12 h was added to the treatment from that day until discharge.

Considering the long hospitalization of patients, pulmonary thromboembolic complications are increasing and must be considered in the context of COVID-19 pneumonia.³⁻⁵ It is also important to evaluate the possible onset of pulmonary infarcts secondary to pulmonary thromboembolism, which change the patient's management and prognosis. In this clinical scenario, the use of advanced imaging methods, such as dual energy pulmonary angiography, allows differentiation between lung parenchyma affected by COVID-19 pneumonia and ischemic or infarcted areas.

Acknowledgments

We thank fellow health and nonhealth workers who helped us in this study and who are struggling in this global emergency.

Salvatore Marsico,^{a,*} Irene Espallargas Giménez,^a Santiago Javier Carbullanca Toledo,^a Luis Alexandre Del Carpio Bellido,^a José María Maiques Llácer,^a and Flavio Zuccarino^{a,b}

^aDepartamento de Radiología, Hospital del Mar, Barcelona, Spain

^bDepartamento de Radiología, Hospital Sant Joan de Déu, Esplugues de Llobregat, Barcelona, Spain

* Corresponding author:

E-mail address: 40161@parcdesalutmar.cat (S. Marsico).

Available online 27 April 2020

REFERENCES

1. Chung M, Bernheim A, Mei X, et al. CT imaging features of 2019 novel coronavirus (2019-nCoV). *Radiology*. 2020. <http://dx.doi.org/10.1148/radiol.2020020230>.
2. Lei J, Li J, Li X. CT imaging of the 2019 novel coronavirus (2019-nCoV) pneumonia. *Radiology*. 2020. <http://dx.doi.org/10.1148/radiol.2020020236>.
3. Yuanliang Xie, Xiang Wang, Pei Yang, Zhang S. COVID-19 Complicated by Acute Pulmonary Embolism. *Radiology: Cardiothoracic Imaging*. 2020. <http://dx.doi.org/10.1148/ryct.2020020067>.

4. Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *Lancet*. 2020;395:507–513.
5. Abdellatif W, Ebada MA, Alkanj S, et al. Diagnostic Accuracy of Dual-Energy CT in Detection of Acute Pulmonary Embolism: A Systematic Review and Meta-Analysis. *Can Assoc Radiol J*. 2020. <http://dx.doi.org/10.1177/0846537120902062>.

<https://doi.org/10.1016/j.rec.2020.04.013>
1885-5857/

© 2020 Sociedad Española de Cardiología. Published by Elsevier España, S.L.U. All rights reserved.

Telecardiology in times of the COVID-19 pandemic



Telecardiología en tiempos de la pandemia de COVID-19

To the Editor:

The health emergency caused by the COVID-19 pandemic led to a state of alarm being declared in Spain on 14 March 2020. Consequently, substantial changes were needed to reorganize Spanish health services. However, there has also been growing concern about diseases unrelated to COVID-19. To ensure continuity of quality health care for patients with heart disease, it is important to differentiate between deferrable and nondeferrable activity with specific objectives. At our hospital, this has meant that most scheduled activities were postponed. This scientific letter describes the gradual development of health care protocols for our Outpatient Cardiology Department. This organizational approach was developed to stratify patients, thus allowing patients in poor clinical condition to be seen personally and patients with stable disease to avoid in-person visits.

In the first phase (11-16 March 2020), a telephone follow-up protocol was implemented and in-person care was maintained for

new patients with the relevant additional tests, based on the usual model aimed at rapid resolution. [table 1](#) summarizes the care provided in the first 48 hours to a total of 278 patients during phase 1: 202 (72.7%) telemedicine visits and 76 (27.3%) new in-person visits. Among these, 14 (18.4%) new patients decided not to attend and follow-up visits were conducted with all patients except for 12 (5.9%), for whom telephone contact was not achieved and new appointments were made. In the second phase, all new and follow-up visits were performed remotely. This organizational structure requires the same number of cardiologists as in-person visits, but allows physicians at higher risk of contagion to be assigned to these positions.

An e-mail address was created for patients to send previous reports, electrocardiograms, and additional tests already carried out. Following telephone contact, the clinicians decided which patients required urgent assessment and which patients could be deferred, allowing discharges and appointments to be arranged for preferential or scheduled evaluations ([figure 1](#)). Last, a second telephone contact was possible for patients with any changes in treatment, to check on tolerance and ensure clinical stability.

As shown in [table 1](#), telemedicine visits proved to be suitable for medical discharges, medication changes, and additional medical

Table 1

Reasons for visit, history of heart disease, and actions taken in 278 patients

Type	Age, y	Women	Reason for visit/heart disease	Tests performed	Treatment	Follow-up, mo				
New, in-person	76 (27)	59 ± 19	38 (61.3)	Chest pain	12 (19.3)	Electrocardiogram	53 (85.5)	Discharge	35 (56.2)	8 ± 10
				Symptoms of HF	6 (9.7)	TTE	40 (64.5)	Treatment changes	8 (12.9)	
				Palpitations	11 (17.7)	Stress test	5 (6.5)	Additional tests	21 (33.8)	
				Dizziness/syncope	3 (4.8)					
				Murmur	3 (4.8)					
				ECG abnormalities	9 (14.5)					
				Family screening	9 (14.5)					
				Other	9 (14.5)					
				Follow-up visits, telemedicine visit	202 (73)	66 ± 17	78 (41.1)	Ischemic heart disease	50 (26.3)	
Valvular heart disease	35 (18.4)		Treatment changes					42 (22.1)		
HF	23 (12.1)		Additional tests					106 (55.8)		
Cardiomyopathies	17 (8.9)									
Arrhythmias	38 (20)									
Congenital heart disease	9 (4.7)									
Pulmonary hypertension	5 (2.6%)									
Other	13 (6.8)									

ECC, electrocardiography; HF, heart failure; TTE, transthoracic echocardiography. The values are expressed as No. (%) or mean ± standard deviation.