Aortitis as Cause of Chest Pain: Role of FDG-PET in a Multimodal Diagnostic Approach

Diagnostic imaging plays a critical role in identifying aortitis. (1)

Computed tomography (CT) angiography of the aorta and thoracic magnetic resonance imaging (MRI) are essential resources to discriminate the different causes of chest pain. Nonetheless, once the aorta is identified as responsible for the symptoms, there are several conditions that can cause confusion in the diagnosis of aortitis, such as acute aortic syndrome (intramural hematoma, aortic dissection, and penetrating atherosclerotic ulcer) and different types of stent-graft leaks.

Inflammation of the aortic wall may be caused by inflammatory, infectious, paraneoplastic or idiopathic diseases. Use of radioisotope techniques such as 18-fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET) is an accurate tool for the diagnosis of this infectious or noninfectious inflammation. (2)

We report the case of a patient with chest pain, where PET/CT scan revealed an aortic inflammatory process that redefined the diagnosis and treatment.

This 74-year-old male patient was a heavy smoker and had hypertension and dyslipidemia. A control chest CT scan after radiation therapy for laryngeal cancer revealed an atherosclerotic, penetrating aortic ulcer, with maximum diameter of 22 mm in the lower wall of the juxtaductal region, which was treated with a thoracic stent-graft. The patient progressed with recurrent chest pain requiring new hospitalization. The condition was interpreted as of coronary etiology, and a coronary angiography (CA) with angioplasty and stent placement in the mid-circumflex artery was therefore performed. The patient was always afebrile, but was admitted in our center due to persistent chest pain. Lab tests on admission showed white blood cell count of 10 600/mm^3; erythrocyte sedimentation rate (ESR), 92 mm/h; C-reactive protein (CRP), 12.7 mg/dL; high-sensitivity troponin T, 117 mg/dL; and negative blood cultures. A new CA showed no lesions and a patent stent. Thoracic aortography ruled out aortic dissection and stent-graft endoleaks.

An ^{18}F-FDG PET/CT scan (Figure 1) exposed a thickened aortic arch wall, with intravenous contrast enhancement and radiotracer uptake with a maximum standardized uptake value (SUVmax) of 14.4, predominantly in the medial sector and, to a lesser extent, in the inferior sector. The ulcer was properly excluded with no evidence of endoleaks or expansion. The aortic stent showed no signs of pathological uptake.

Due to suspected diagnosis of inflammatory aortitis, systemic inflammatory markers were requested, including VDRL, FTA-Abs, FAN in Hep-2, ANCA c and p, HLA-B*51 (for Behçet's disease), and IgG4, with negative results.

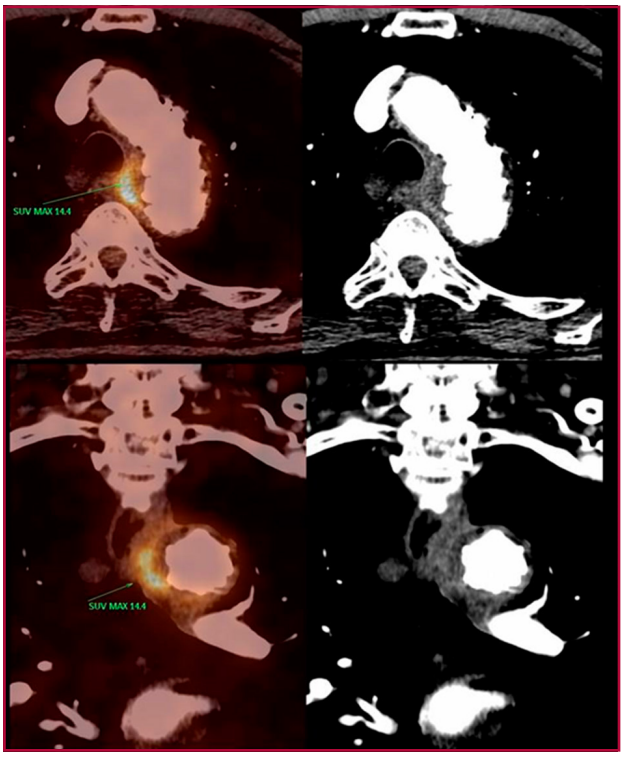

Fig. 1.

The condition was thus interpreted as isolated aortitis after aortic stent graft placement in the context of preexisting inflammatory aortic disease. Although giant cell arteritis —an entity accounting for 70% of aortitis in patients >50 years of age, and involving the aortic wall in 15-22% of cases in a diffuse or segmental form, with high probability of causing aortic aneurysms— cannot be definitively ruled out as a differential diagnosis, (3) this diagnosis is unlikely in our case as the patient did not meet the classification criteria or the typical clinical manifestations.

The patient was started on meprednisone and made good progress, with resolution of symptoms and normalization of inflammatory parameters (ESR and CRP).

A follow-up ^{18}F-FDG PET/CT scan at 2 months (Figure 2) showed persistent metabolic activity in the medial and inferior wall of the aortic arch, extending to the lateral sector, and the same uptake values as in the previous study.

Aortitis is a nonspecific term that refers to inflammatory changes in the aortic wall, often caused by a systemic inflammatory, infectious or non-infectious disease, with different clinical variables and presentation. Therefore, the diagnosis often requires a multimodal imaging approach. (4) In our patient, after an initial CT angiography and two invasive procedures, a PET/CT scan made it possible to redefine the diagno-

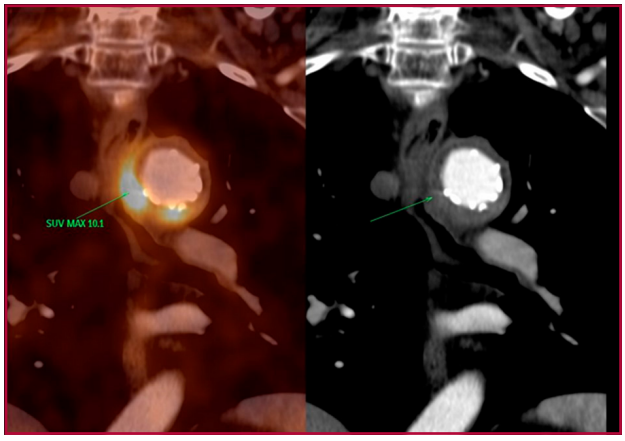

Fig. 2.

sis, initially oriented to a coronary disease, as aortitis. FDG PET for the diagnosis of inflammatory aortic disease has 98% specificity, a positive predictive value of 93%, and a negative predictive value of 80%. (5) These data are also relevant for the follow-up and treatment of these patients, based on the inflammatory activity provided by the images and the evolving clinical and serological data. (6)

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/ Supplementary material).

Ethical considerations

Not applicable.

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REFERENCES

1. Restrepo C, Ocazionez D, Suri R, Vargas D. Aortitis: Imaging spectrum of the infectious and inflammatory conditions of the Aorta. RadioGraphics 2011;31:435-51. https://doi.org/10.1148/rg.312105069
2. Gornik H, Creager M. Aortitis. Circulation 2008;117:3039-51. https://doi.org/10.1161/CIRCULATIONAHA.107.760686
3. Pacini D, Leone O, Turcis S, Giunchi F, Martinelli G, Di Bartolomeo, R. Incidence, etiology, histologic findings, and course of thoracic inflammatory aortopathies. Ann Thorac Surg 2008;86:1518-23. https://doi.org/10.1016/j.athoracsur.2008.07.039
4. Wurmann P, Sabugo F, Cruz J, Díaz G, Sánchez F, Pino S. Aortitis, causas infrecuentes en Reumatología. Rev Med Chile 2014;142:924-9. https://doi.org/10.4067/S0034-98872014000700015
5. Blockmans D, Stroobants S, Maes A, Mortelmans L. Positron emission tomography in Giant Cell Arteritis: evidence for inflammation of the aortic arch. Am J Med 2000;108:246-9. https://doi.org/10.1016/S0002-9343(99)00424-6
6. Flórez-Muñoz P, Martín-Fernández M, Capín-Sampedro E, Barreiro-Pérez M, Álvarez-Pichel, I. Aortitis: una causa de dolor torácico agudo poco común. Rev Esp Cardiol 2013;66:673-4. https://doi.org/10.1016/j.recesp.2013.03.007

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http://dx.doi.org/10.7775/rac.v89.i3.20364

5-Fluorouracil-Induced Reversible Cardiogenic Shock

5-Fluorouracil (5-FU) is a fluoropyrimidine (FP) antimetabolite agent used broadly in the treatment of a variety of solid tumors, and is only second to anthracyclines in terms of incidence of cardiotoxicity.

Coronary vasospasm is its main cardiotoxic mechanism manifested as precordial pain associated with transient electrocardiographic (ECG) changes, rarely causing myocardial infarction.

However, there are other rare manifestations of cardiotoxicity, such as dilated cardiomyopathy, ventricular arrhythmia, and sudden death.

We describe a case of cardiogenic shock following 5-FU infusion.

This is a 35-year-old patient with a history of stage IV gastric adenocarcinoma. Throughout his specific treatment, the patient completed several chemotherapy regimens: EOX (epirubicin -cumulative dose 324 mg/m2-, oxaplatine, and capecitabine) as first-line therapy; paclitaxel / ramucirumab as second-line therapy, and irinotecan as third-line therapy (Figure 1). During those regimens, the patient had no signs of cardiotoxicity; transthoracic echocardiography (TTE) revealed preserved left ventricular ejection fraction (LVEF) (>55%), with normal global longitudinal strain (<18%, normal range: - 19% ± 2%). Finally, a fourth-line therapy with FOLFOX (5-fluorouracil, oxaplatine and folinic acid) in continuous infusion was started.

During the first 5-FU infusion, the patient presented typical precordial pain associated with ECG transient ST-segment elevation, which disappeared once the drug was discontinued. No high-sensitivity troponin T elevation was detected. This event was interpreted as coronary vasospasm. The patient was discharged under diltiazem therapy.

Forty-eight hours later, the patient was admitted in the emergency room with a second episode of precordial pain associated with diaphoresis and dizziness. Physical examination showed an awake, hypotensive patient with signs of poor peripheral perfusion. Lab tests on admission revealed acute kidney failure, with increased cardiac biomarkers: high-sensitivity troponin: 20 pg/mL (normal <15 pg/mL), NT-proBNP: 3000 pg/mL (normal 125 pg/mL), and increased blood lactic level: 3 mmol/L. The ECG showed no acute ischemic changes. TTE revealed slightly increased LV diameters, with global hypokinesis and severe LVEF impairment: 25%.

In the Cardiac Intensive Care Unit (CICU), the

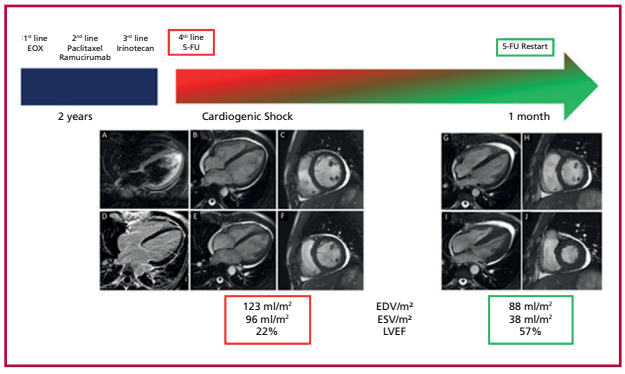
patient required vasopressors, with clinical improvement in 48 hours. Cardiac magnetic resonance imaging (MRI) confirmed diffuse hypokinesis and severe ventricular dysfunction (LVEF 22%), with no myocardial edema in T2-weighted sequences. Late gadolinium enhancement (LGE) sequences ruled out myocardial fibrosis (Figure 1).

The patient made good progress and was discharged 5 days later under beta-blockers and angiotensin-converting enzyme inhibitors. LVEF gradually improved during follow-up in the Cardio-Oncology Unit, reaching full recovery (LVEF: 55%) at 15 days. This recovery was confirmed by MRI, showing 57% LVEF at 1 month (Figure 1). After LVEF recovery, a multidisciplinary team decided to continue treatment with 5-FU. The 5 pending chemotherapy cycles were completed in the CICU under continuous monitoring, with no further cardiac complications.

We have described the case of a young male patient with no cardiovascular history, who presented cardiogenic shock following 5-FU infusion.

The most common 5-FU-related adverse events include mucositis, diarrhea, and myelosuppression. Cardiotoxicity is a severe adverse effect causing from electrocardiographic changes in asymptomatic patients to transient angina, myocardial infarction, or even life-threatening cardiogenic shock. The exact mechanism of cardiotoxicity remains unclear, although several mechanisms from animal models, case reports, and small clinical studies have been proposed.

Coronary spasm and acute myocardial ischemia are the best-described adverse effects of fluoropyrimidines, manifested as precordial pain, ischemic ECG changes, and increased biomarkers as high-sensitivity troponin. Two mechanisms have been proposed to explain this phenomenon. Mosseri et al. found that protein kinase C could mediate vasoconstriction, and demonstrated endothelium-independent vasoconstriction with increasing doses of 5-FU in an animal model. (1) In addition, increased concentration of endothelin-1, a potent vasoconstrictor, was detected in patients with 5-FU-induced cardiotoxicity. (2) Our patient reported 5-FU-induced precordial pain 48 hours before the episode of cardiogenic shock. However, no high-sensitivity troponin elevation was detected, and MRI evidenced no signs of myocardial ischemia, since it showed global hypokinesis with no signs of edema in T2-weighted sequences, and no fibrosis in LGE sequences. Vasospasm was therefore ruled out as the underlying mechanism causing ventricular dysfunction.



Chronology of clinically significant events: timing of treatment, onset and recovery from cardiotoxicity (A). Cardiac magnetic resonance imaging (MRI), T2-weighted sequences with no signs of myocardial edema. End-diastolic cine sequences show increased left ventricular volumes: four chamber (B) and short axis (C) views. (D) Late gadolinium enhancement in MRI shows no signs of fibrosis. End-systolic cine images show severe left ventricular dysfunction: four chamber (E) and short axis (F) views. MRI at 1 month: cine images showing significant decrease in left ventricular volumes and significant improvement in left ventricular ejection fraction (LVEF). End-diastole: four chamber (G) and short axis (H) views. End-systole: four chamber (I) and short axis (J) views. EDV/m²: LV end-diastolic volume indexed to body surface area. ESV/m²: LV end-systolic volume indexed to body surface area.

Fig. 1. Timeline

Direct toxic effect of 5-FU on cardiomyocytes is another proposed mechanism. Alpha-fluoro-beta-alanine (FBAL), a degradation product of 5-FU, plays a key role in this regard. In a case report, Muneoka et al. demonstrated increased concentrations of FBAL in patients with 5-FU-induced cardiotoxicity. No cardiac symptoms occurred after the initiation of the prodrug S-1 administration (an oral fluoropyrimidine that lacks FBAL as a metabolite). (3)

Endothelial dysfunction and inadequate oxygen delivery constitute another proposed mechanism. Vascular dysfunction associated with microthrombi formation is a mechanism that could potentially cause cardiotoxicity. Microthrombi occlusion is usually undetected by coronary arteriography. (4) However, in our patient, we were unable to demonstrate evidence of microvascular obstruction by MRI on LGE sequences. Use of anticoagulation therapy has been suggested to mitigate this adverse effect, but further research is required to standardize their indication. Some authors believe free oxygen radicals could also play a role in cytotoxic endothelial dysfunction. (5)

The last mechanism was described by Spasojevic et al. who demonstrated that 5-FU causes changes to the erythrocyte membrane leading to increased blood fluidity and conversion of the erythrocyte from its usual biconcave shape to an echinocyte shape. The resulting membrane changes diminish the erythrocyte ability for oxygen transportation, resulting in myocardial ischemia and injury. (6)

In our patient, it was not possible to recognize a single pathophysiological mechanism to explain LV dysfunction, but we believe that it is due to a combination of several factors.

There is no standard treatment available for fluoropyrimidine-induced cardiotoxicity. Current consensus indicates 5-FU treatment discontinuation in case of suspected cardiotoxicity. In case of vasospasm, the patient should be treated symptomatically with antianginal drugs (nitrates or calcium blockers, such as diltiazem), to abort the symptoms and prevent recurrences. LV dysfunction should be initially treated

according to international guidelines. In some cases, cardiotoxicity is reversible after drug discontinuation, as occurred in our patient. A multidisciplinary approach by cardiologists and oncologists is crucial when managing this type of patients and making decisions about whether or not to continue treatment. Repeating treatment with 5-FU after cardiotoxicity implies a high risk of recurrence, between 82% and 100%. Our patient completed the remaining 5-FU cycles under careful monitoring in the CICU.

We believe our case is important to emphasize the diverse clinical manifestations, other than vasospasm, of 5-FU-induced cardiotoxicity, to be taken into account when planning strategies for patient monitoring.

Myocardial dysfunction and cardiogenic shock may be manifestations of reversible 5-FU-induced cardiotoxicity, and seem to be independent of coronary vasospasm. It is essential to be aware of this adverse effect in these patients' follow-up.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable

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REFERENCES

1. Mosseri M, Fingert HJ, Varticovski L, Chokshi S, Isner JM. In vitro evidence that myocardial ischemia resulting from 5-fluorouracil chemotherapy is due to protein kinase C-mediated vasoconstriction of vascular smooth muscle. Cancer Res 1993;53:3028-33.

2. Thyss A, Gaspard MH, Marsault R, Milano G, Frelin C, Schneider M. Very high endothelin plasma levels in patients with 5-FU cardiotoxicity. Ann Oncol 1992;3:88. https://doi.org/10.1093/oxfordjournals.annonc.a058084

3. Muneoka K, Shirai Y, Yokoyama N, Wakai T, Hatakeyama K. 5-Fluorouracil cardiotoxicity induced by alpha-fluoro-beta-alanine. Int J Clin Oncol 2005;10:441-3. https://doi.org/10.1007/s10147-005-0516-7

4. Layoun ME, Wickramasinghe CD, Peralta MV, Yang EH. Fluoropyrimidine-Induced Cardiotoxicity: Manifestations, Mechanisms, and Management. Curr Oncol Rep 2016;18:35. https://doi.org/10.1007/s11912-016-0521-1.

5. Focaccetti C, Bruno A, Magnani E, Bartolini D, Principi E, Dallaglio K, et al. Effects of 5-fluorouracil on morphology, cell cycle, proliferation, apoptosis, autophagy and ROS production in endothelial cells and cardiomyocytes. PLoS One 2015;10: e0115686. https://doi.org/10.1371/journal.pone.0115686

6. Spasojević I, Jelić S, Zakrzewska J, Bacić G. Decreased oxygen transfer capacity of erythrocytes as a cause of 5-fluorouracil related ischemia. Molecules 2008;14:53-67. https://doi.org/10.3390/molecules14010053

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Rev Argent Cardiol 2021;89:254-256.
http://dx.doi.org/10.7775/rac.v89.i3.20024

Impact of the COVID-19 Pandemic on a Department of Cardiology

In December 2019, the first cases of SARS-CoV-2 were identified in China; shortly after, the World Health Organization (WHO) declared COVID-19 an international emergency, and by February 2020, more than 80 000 cases had been confirmed. (1) The disease is characterized by acute respiratory distress syndrome and subsequent cardiac damage by different mechanisms. (2) To briefly describe the impact of the pandemic on cardiac patients, we consecutively analyzed consultations, practices, and admissions in the Coronary Care Unit (CCU) of our center, Hospital Santojanni. This is a public hospital receiving nearly one million consultations per year, 59% of which correspond to residents of the City of Buenos Aires (CABA). Cardiac patient care was considered essential; therefore, the areas of cardiac admission were kept mostly free from COVID-19 patients. During the critical months, part of the healthcare team was assigned to work in COVID areas. Telecare for arrhythmias and heart failure began in May, but the medical office for prevention always remained with in-person consultations. The Interventional Cardiology and Electrophysiology Units continued working and adapted to protection standards established by the Ministry of Health. Cancellation of scheduled procedures increased with high COVID bed occupancy, in order to free up CCU beds and save healthcare resources.

In the three medical offices analyzed, the 2020 overall drop rate in health care was 17% compared with 2019. A deficit of 1007 consultations was observed, with 4923 consultations in 2020 versus 5930 in 2019. When the exclusive in-person medical office for prevention was analyzed, an average drop rate of 62% was observed in the critical months, with a total of 1019 fewer consultations (– 31%) than in 2019. However, no difference was found in the medical offices that performed telecare. The top panel of Table 1 shows the total number consultations per quarter and the quarterly average, comparing 2019 and in 2020 in the in-person medical offices in 2019 that added telemedicine in 2020, and in those that maintained only in-person consultation. The analysis is repeated at the bottom panel of Table 1, taking into account only the last 3 quarters of each year, to assess specifically the impact of the pandemic.

Regarding invasive procedures, there were fewer cardiac catheterizations (coronary angiography and coronary angioplasty). A total of 942 invasive procedures were performed in 2019 and 605 in 2020, that is, 36% fewer procedures, with an average reduction of 66% from April to July. A drop in the total number of coronary angioplasties, including

Number of outpatient consultations						
	In-person	In-person & telemedicine		In-person		
Quarter	2019	2020	%	2019	2020	%
First	586	577	-1.5%	468	636	35.9%
Second	687	655	-4.7%	878	381	-56.6%
Third	692	681	-1.6%	893	662	-25.9%
Fourth	669	733	9.6%	1057	598	-43.4%
Total	2634	2646	0.5%	3296	2277	-30.9%
	659.5 (95% CI 610 - 707)	661.5 (95% CI 598 - 725)	p = 0.9	824.0 (95% CI 578 - 1070)	569.3 (95% CI 444 - 595)	p = 0.09

Number of outpatient consultations						
	In-person	In-person & telemedicine		In-person		
Quarter	2019	2020	%	2019	2020	%
Second	687	655	-4.7%	878	381	-56.6%
Third	692	681	-1.6%	893	662	-25.9%
Fourth	669	733	9.6%	1057	598	-43.4%
Total	2048	2069	1.0%	2828	1641	-42.0%
	682.7 (95% CI 669 - 696)	689.7 (95% CI 654 - 735)	p NS	942.7 (95% CI 830 - 1055)	547.0 (95% CI 380 -713)	p = 0.002

Number of outpatient consultations									
	Cardiac catheterization			Electrophysiology implant			Coronary angioplasty		
Quarter	2019	2020	%	2019	2020	%	2019	2020	%
First	229	213	-7%	20	15	-25%	72	68	-6%
Second	226	67	-70%	22	19	-14%	67	27	-60%
Third	260	155	-40%	15	6	-60%	84	49	-42%
Fourth	227	170	-25%	16	18	13%	74	60	-19%
Total	942	605	-36%	73	58	-21%	297	204	-31%
	235.5 (95% CI 219 - 252)	151 (95% CI 91 - 211)	p = 0.04	18.3 (95% CI 15 - 22)	14.5 (95% CI 9 - 20)	p = 0.1	74.3 (95% CI 67 - 81)	51 (95% CI 33 - 68)	p = 0.04

Number of outpatient consultations									
	Cardiac catheterization			Electrophysiology implant			Coronary angioplasty		
Quarter	2019	2020	%	2019	2020	%	2019	2020	%
Second	226	67	-70.4%	22	19	-13.6%	67	27	-59.7%
Third	260	155	-40.4%	15	6	-60.0%	84	49	-41.7%
Fourth	227	170	-25.1%	16	18	12.5%	74	60	-18.9%
Total	713	392	-45%	53	43	-18.9%	225	136	-39.6%
	237.7 (95% CI 216 - 260)	130.7 (95% CI 67 - 193)	p = 0.04	17.7 (95% CI 13 - 22)	14.3 (95% CI 6 - 21)	p = 0.2	73.0 (95% CI 65 - 85)	16.8 (95% CI 26 - 64)	p = 0.03

primary percutaneous coronary intervention (PCI) in acute myocardial infarction (AMI), was also observed with respect to 2019. An annual drop of 21% was found in electrophysiology implants, with an average reduction of 46% from May to September 2020 (Table 2).

There was a reduction in the number of cardiovascular patients hospitalized in 2020 in the CCU, and an increase in the number of patients referred from intensive care units. A decrease in hospitalizations due to acute myocardial infarction (AMI) with ST-segment elevation (STEMI) or non-ST-segment elevation (NSTEMI) was evidenced during 2020, and this difference was maximal in the critical months of COVID-19 bed occupancy. A total of 248 AMI patients were hospitalized in 2019, and 191 in 2020, a drop of 23%. This reduction was observed in both STEMI (122 in 2019 and 88 in 2020) and NSTEMI (126 in 2019 and 103 in 2020). Given that the center also receives referrals from the AMI network, the number of STEMI patients is higher than the number of patients hospitalized with this diagnosis, as some of them return to their referral centers after PCI. So, a total of 176 STEMI patients were hospitalized in 2019, and 116 in 2020 (a drop of 34%).

The maelstrom of the pandemic should not pre-

vent us from analyzing how we work. The CCU continues to receive patients with severe conditions, such as AMI or heart failure, with longer delays or treatment withdrawal. Considering the marked reduction of consultations in the critical periods of 2020, it is likely that the cardiovascular mortality rate has then increased, as described in international reports showing an increase in out-of-hospital events. (3) Especially, we observed a significant reduction in consultations and hospital admissions in the second quarter, when few COVID-19 cases were still being admitted. This was a "missed opportunity" for care, perhaps due to general health policies and center-specific reasons, among others. The same situation was described in other countries. (4, 5) Prolonged lockdown delayed transmission, but this situation was only used for the general hospital reorganization, when the care of patients with heart diseases could have been encouraged, as was timely expressed by different sectors of Argentine cardiology. (6) Experience shows, as we observed months later with a greater number of COVID-19 inpatients, that cardiac patient care can be safely sustained.

2020 was a unique experience due to the pandemic; we observed a poorer care of patients with heart diseases and a possible increase in cardiovascular morbidity and mortality in the untreated population. In the City of Buenos Aires, the number of cases increased gradually in 2020, but a new wave of infections in 2021 might not be mitigated with social behavior. Our challenge will be to sustain quality of care without postponing patient medical attention to avoid the complications mentioned above.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/ Supplementary material).

Ethical considerations

Not applicable

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REFERENCES

1. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, et al. Clinical Characteristics of Coronavirus Disease 2019 in China. The China Medical Treatment Expert Group for Covid-19. N Engl J Med 2020;382:1708-20. https://doi.org/10.1056/NEJM 2002032.

2. Fernández A, Barisani JL, Guetta J, Bosio M, Chertcoff J, Marino J, et al. COVID – 19. Su repercusión cardiovascular. Una revisión. Rev Argent Cardiol 2020;88:253-74. http://dx.doi.org/10.7775/rac.es.v88.i3.18230

3. Baldi E, Sechi G, Mare C, Canevari F, Brancaglione A, Primi R, et al, Lombardia CARe Researchers Collaborators.Out-of-Hospital Cardiac Arrest during the Covid-19 Outbreak in Italy. . N Engl J Med. 2020;383:496-8. https://doi.org/10.1056/NEJMc2010418

4. Rodrıguez-Leor O, Alvarez B, Perez de Prado A, Rossello X, Ojeda S,Serrador A, et al, for the Working Group on the Infarct Code of the Interventional Cardiology Association of the Spanish Society of Cardiology Investigators. Impact of COVID-19 on ST-segment elevation myocardial infarction care.The Spanish experience. Rev Esp Cardiol 2020;73:994–1002.

5. De Rosa S, Spaccarotella C, Basso C, Calabro M, Curcio A, Perrone Filardi P, et al; on behalf of Societa`Italiana di Cardiologia and the CCU Academy investigators group. Reduction of hospitalizations for myocardial infarction in Italy in the COVID-19 era. Eur Heart J 2020;41:2083-8. https://doi.org/10.1093/eurheartj/ehaa409.

6. https://www.sac.org.ar/evento/infarto-con-suprast-que-estamos-aprendiendo-durante-la-pandemia-de-covid-19/

Rev Argent Cardiol 2021;89:256-258.
http://dx.doi.org/10.7775/rac.v89.i3.20363