

Coronary Bypass in Pediatric Patients with Congenital Heart Disease

In certain circumstances, coronary artery bypass grafting (CABG) is the only option to solve congenital diseases derived from cardiac anatomical defects in pediatric patients.

There are five groups of patients for whom CABG is the only option for restoring normal cardiac function:

1. Anomalous origin of the left main coronary artery from the pulmonary artery (ALCAPA);
2. Atresia of the left main coronary artery;
3. Acute and chronic coronary events in the transposition of great arteries operation (switch procedure);
4. Pediatric Ross procedure complicated with infective endocarditis;
5. Unexpected coronary artery injury in pediatric cardiac surgery.

We describe two resolved clinical cases from groups 1 and 3. Case 1: Anomalous origin of the left main coronary artery from the pulmonary artery. Case 2: Reoperation for pulmonary homograft placement in a patient with transposition of great arteries procedure.

Case 1

This is the case of a 7-month-old patient with heart failure, severely impaired left ventricular function, and moderate mitral regurgitation. Clinical and echocardiographic data evidenced an undernourished infant with low cardiac output. Cardiac catheterization revealed atresia of the left main coronary artery ostium (Figure 1 A & B).

On March 28, 2018, surgery was performed through median sternotomy, cannulation of the ascending aorta, bicaval cannulation, extracorporeal circulation and cardiac arrest with Del Nido antegrade cardioplegia. Transection of the pulmonary artery showed the anomalous origin of the left main coronary artery, unable to be reimplemented in the aorta since it was in the left lateral wall of the main pulmonary artery. Coronary artery bypass grafting of the coronary ostium excised from the pulmonary artery was performed using the internal thoracic artery (ITA) (Figure 1 C & D). Postoperative course was favorable, and the patient was discharged 14 days after the procedure. Color Doppler echocardiography performed on February 10, 2020 showed normal ventricular function, decreased chamber diameters and volumes compared to preoperative values, and normal mitral valve function, without regurgitation.

Case 2

This is the case of a female patient born in February 2018 with prenatal diagnosis of double outlet right

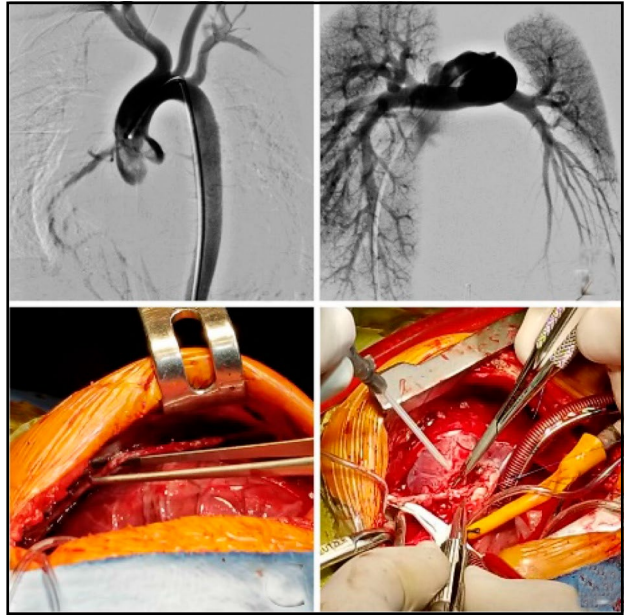


Fig. 1. Case 1. A: coronary mapping, absence of the origin of the left coronary artery; **B:** pulmonary artery mapping; **C:** preparation of left internal thoracic artery (LITA) with skeletonized technique; **D:** LITA bypass to anterior descending artery (ADA).

ventricle, transposed vessels, ventricular septal defect, and single origin of left sinus coronary arteries. Arterial switch operation with closure of ventricular septal defect was performed at 10 days of life. Because the right coronary artery was entangling the neo-pulmonary trunk, which was slightly hypoplastic, the patient progressed with residual severe pulmonary stenosis, requiring subsequent angioplasties of the pulmonary arteries and surgical repair of the pulmonary trunk and its main branches between May 2018 and July 2019. Given the progression of pulmonary stenosis, further surgery was decided on February 11, 2020, with the corresponding angiographic mapping (Figure 2 A & B). Right ventricular outflow tract enlargement, homograft of 15 mm diameter in pulmonary position, pulmonary branch enlargement with pericardial patch, and CABG using in situ right ITA to the proximal right coronary artery (RCA) were performed (Figure 2 C & D). Coronary artery bypass grafting to RCA was necessary in order to enlarge the right ventricular outflow tract and avoid complications with the RCA course. On February 15, 2020, the patient was discharged with good progress and normal RV pressure and ventricular function.

The anomalous origin of the left main coronary artery from the pulmonary artery has two names: Bland-White-Garland syndrome, named after those who described it, and ALCAPA (anomalous origin of

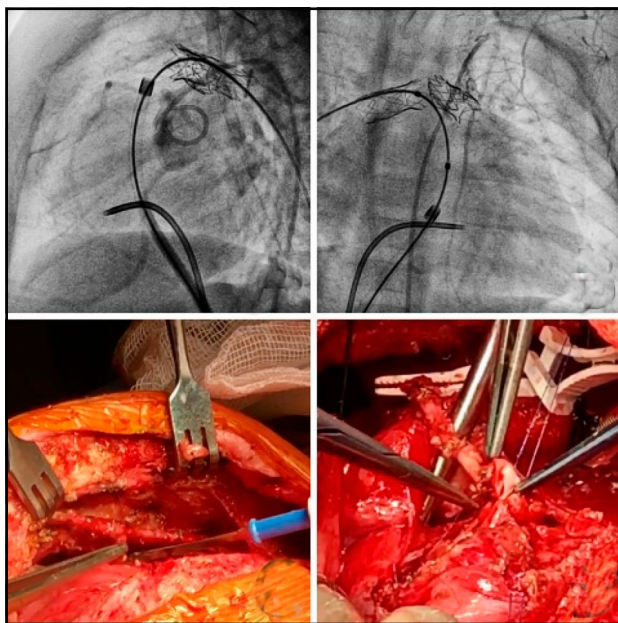


Fig. 1. Case 2. A: coronary mapping; **B:** in situ LITA mapping; **C:** preparation of LITA with skeletonized technique; **D:** LITA to ADA bypass grafting

the left coronary artery from the pulmonary artery). It comprises 0.24% to 0.5% of congenital heart anomalies. (1) It usually presents as an isolated anomaly and has a mortality rate of 90% in the first year of life. In children, it clinically presents dilated cardiomyopathy, with arrhythmias, heart failure, and sudden death. (1) Alexi-Meskishvili et al. published the initial experience in reimplantation of the left main coronary artery ostium into the aorta, (2) and this technique is widely used in the management of this condition.

In certain circumstances, ligation of the left coronary artery in the presence of severe myocardial dysfunction and hemodynamic instability was a life-saving procedure in some patients, as cited by Kreutzer et al. (3) However, these authors recognized the importance of the left coronary system revascularization to achieve long-term survival in these patients. Ginde et al. (4) described their experience with Takeuchi's technique consisting of a pulmonary artery wall tunnel extending from the left main coronary artery ostium to the aortic wall, and reported valvular complications and/or postoperative fistulas.

The technique used in both cases reported was direct anterior descending artery CABG using ITA and closure of the left ostium that originated anomalously in the pulmonary artery. Some reports support this technique, (5) understanding that ITA is a graft that grows along with the developing child with clear benefits compared to venous grafts.

There are many potential causes of early coronary artery obstruction following switch procedures such as kinking and thrombus formation secondary

to intramural or intimal injury, extreme compression or stretching. From the experience reported, it appears that CABG using in situ ITA is the best option. Priftia et al. discussed several reports of successful CABG with ITA in different scenarios, with short- and long-term benefits. (6) Postoperative angiography demonstrated good patency of the thoracic arteries at 8 and 30 months postoperatively. Internal thoracic artery grafts are live conduits with potential growth and adaptation; reports on catheterization demonstrated graft and coronary anastomosis growth as body surface area increased.

Coronary artery bypass grafting using ITA is a feasible and interesting option for coronary revascularization in the context of congenital heart defects, such as ALCAPA with difficult reimplantation and cardiac reoperations in infants.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable.

**Guillermo N. Vaccarino¹ ,
Guillermo S. Gutierrez¹ ,
Gustavo Bastianelli¹, Daniel Klinger¹,
Benjamin Chiostrì¹, Christian Kreutzer¹**

¹ Hospital Universitario Austral, Pilar,
Pcia. de Buenos Aires, Argentina.
E-mail: guillermovaccarino@gmail.com

REFERENCES

1. Agarwal PP, Dennie C, Pena E, Nguyen E, LaBounty T, Yang B, et al. Anomalous Coronary Arteries That Need Intervention: Review of Pre- and Postoperative Imaging Appearances. *Radiographics* 2017;37:740-57. <https://doi.org/10.1148/rg.2017160124>.
2. Alexi-Meskishvili V, Nasser B, Nordmeyer S, Schmitt B, Weng YG, Böttcher W, et al. Repair of anomalous origin of the left coronary artery from the pulmonary artery in infants and children. *J Thorac Cardiovasc Surg* 2011;142:868-74. <https://doi.org/10.1016/j.jtcvs.2011.04.006>
3. Kreutzer C, Schlichter A, Roman M, Kreutzer G. Emergency ligation of anomalous left coronary artery arising from the pulmonary artery. *Ann Thorac Surg* 2000;69:1591-2. [https://doi.org/10.1016/S0003-4975\(00\)01179-6](https://doi.org/10.1016/S0003-4975(00)01179-6)
4. Backer CL, Stout MJ, Zales VR, Muster AJ, Weigel TJ, Idriss FS, et al. Anomalous origin of the left coronary artery. A twenty-year review of surgical management. *J Thorac Cardiovasc Surg* 1992;103:1049-58. [https://doi.org/10.1016/S00225223\(19\)34868-8](https://doi.org/10.1016/S00225223(19)34868-8)
5. Ginde S, Earing MG, Bartz PJ, Cava JR, Tweddell J S. Late complications after Takeuchi repair of anomalous left coronary artery from the pulmonary artery: case series and review of literatura. *Pediatr Cardiol* 2012;33:1115-23. <https://doi.org/10.1007/s00246-012-0260-5>.
6. Priftia E, Bonacchib M, Vincenzo S, Vaninia V. Coronary revascularization after arterial switch operation. *Eur J Cardiothorac Surg* 2002;21:111-3. [https://doi.org/10.1016/S1010-7940\(01\)01074-0](https://doi.org/10.1016/S1010-7940(01)01074-0)

Comprehensive Phenotypic Assessment of Hypertrophic Cardiomyopathy with Spectral Computed Tomography

In parallel to its established role for the diagnosis of ischemic heart disease, computed tomography (CT) has gained ground in the assessment of myocardial perfusion and delayed contrast enhancement. (1) Therefore, it is an alternative to other imaging methods such as cardiac magnetic resonance imaging (CMR) to assess cardiomyopathies. (2)

In addition, cardiac CT quantifies extracellular volume fraction (ECV), with comparable results to CMR T1 mapping techniques. (3) Spectral CT is a tool that can mitigate or eliminate artifacts that can simulate perfusion defects in conventional CT, and significantly improves tissue characterization. Effectively, although conventional CT assesses delayed contrast enhancement accurately in the context of acute myocardial infarction (in which increased ECV is associated to cellular rupture), its ability to assess delayed enhancement in chronic pathologies—in which increased ECV is associated to interstitial fibrosis—is very limited.

In this regard, dual-layer spectral detector CT provides spectral multiparametric imaging following the standard acquisition protocol and with the possibility of assessing conventional CT imaging. (4) Thus, images are generated at different monoenergetic levels to evaluate parameters related to different tissue composition.

However, very few reports on the usefulness of spectral CT in patients with hypertrophic cardiomyopathy (HCM) are available in the literature. (3) In those patients, it is important to determine maximum wall thickness and the presence of fibrosis. In addition, many of these patients—who may have ischemia associated with supply and demand imbalance or arteriolar dysplasia—develop symptoms associated with exertion that first require ruling out obstructive coronary artery disease. Therefore, we report, to our knowledge, the first case of HCM comprehensively evaluated with dual-layer spectral detector CT. (5)

This is the case of a 75-year-old female patient with a history of non-insulin diabetes, hypertension, dyslipidemia, and smoking, with a pre-existing diagnosis of non-obstructive HCM and progressive dyspnea during the past months (functional class II-III), without angina.

Physical examination was normal, with no changes in the electrocardiogram compared with previous ones (Figure 1A). CT-angiography was performed with a spectral CT scanner (IQon Spectral CT®, Philips Healthcare, Best, The Netherlands) using volumetric acquisition with retrospective electrocardiographic gating for the arterial phase (Figure 1), and with prospective gating for the delayed enhancement images (Figure 2) obtained at 5 minutes without additional contrast agent, using tube modulation techniques to

reduce the radiation dose. A total of 65 ml contrast agent was used.

The main coronary arteries showed mild calcification, especially in the anterior descending artery, while the first diagonal branch, with a very small diameter, was occluded at the proximal level (Figure 1B). Asymmetric left ventricular (LV) hypertrophy with anterior-basal and septal predominance (Figure 1D), discrete apical right ventricular (RV) hypertrophy (Figure 2C), and increased myocardial trabeculation of the left ventricle were evidenced using end-diastolic reconstructions. Global systolic function was preserved. Spectral evaluation revealed intramyocardial focal areas of hypoperfusion (Figure 1E).

Images obtained 5 minutes after contrast injection showed multiple focal areas of predominantly intramyocardial delayed enhancement (Figure 2). In addition, a hypodense nucleus within the basal anteroseptal transmural late enhancement area (Figure 2D), consistent with microvascular obstruction, was identified. Minimal subendocardial enhancement was also observed at the anterior and mid-anterolateral levels and at the level of the anterolateral papillary muscle (Figure 2A), and the right ventricular apex (Figure 2C). A ECV of 36% was calculated using iodine maps.

The filling defect in the left atrial appendage evidenced during arterial acquisition (Figure 1C) com-

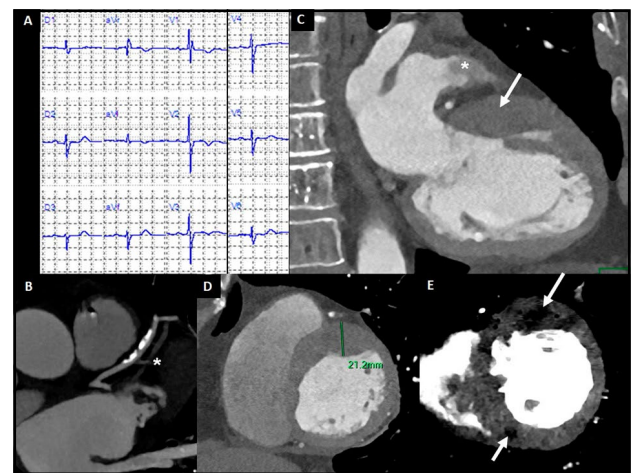


Fig. 1. (A) ECG with upper limit PR interval; indeterminate axis with QRS complexes (of normal duration) and low voltage in the limb leads; Q-waves in DII-DIII-aVF suggestive of inferior inactivation; atypical incomplete right bundle branch block due to the presence of R < initial R in V1; abnormal decrease of R-waves from right to left precordial leads; ST-T alteration in DI, to VL and V2-V4; normal QTc duration. (B-E) Arterial acquisition (retrospective ECG gating). (B) Non-significant atherosclerosis of the anterior descending artery, an occluded very small first diagonal branch. (*) (C) Anterior-basal hypertrophy (arrow) and gradual filling defect in left atrial appendage. (*) (D) End-diastolic reconstruction with asymmetric hypertrophy, with anterior and basal anteroseptal predominance. (E) Assessment of 40-keV monoenergetic spectral images showing focal perfusion defects with anterior and inferoseptal intramyocardial predominance (arrows).

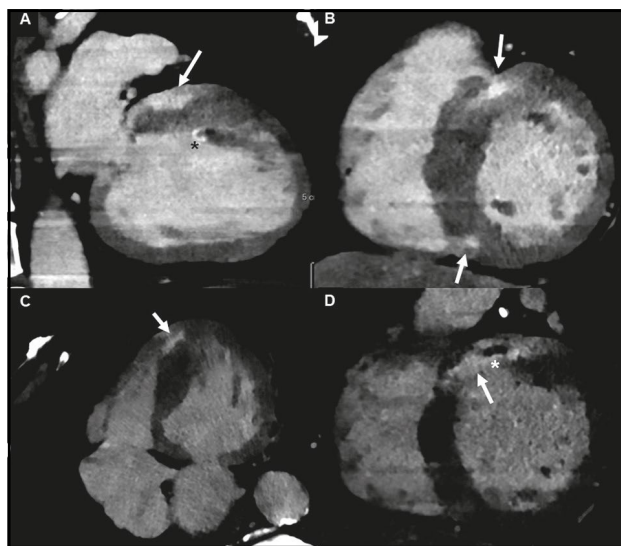


Fig. 1. Late phase, assessment of 40-keV monoenergetic spectral images. (A) Anterior-basal subepicardial fibrosis (arrow) and at the level of the papillary muscle, (*) and absence of thrombosis in the left atrial appendage. (B) Intracardiac patches of fibrosis at the anteroseptal and inferoseptal levels (arrows). (C) Four-chamber view showing marked septal hypertrophy and apical involvement of the right ventricle (arrow). (D) Basal anteroseptal transmural enhancement, with signs of microvascular obstruction. (*)

pletely resolved in the delayed acquisition (Figure 2A), confirming the presence of blood stasis without appendage thrombus.

In this case, we ruled out relevant obstructive coronary artery disease in the first instance. Also, these findings might identify the mechanisms underlying delayed enhancement (myocardial fibrosis), in this case possibly associated with microinfarcts, given the presence of significant hypertrophy and coronary atheromatosis that would generate insufficient perfusion to make up for the imbalance between oxygen supply and demand.

Spectral cardiac CT-angiography is an alternative to other methods for the characterization of cardiomyopathies, as well as for the simultaneous assessment of coronary artery disease and myocardial perfusion, and to rule out fibrosis.

In addition, low-energy monoenergetic images allow for lower contrast volumes, without need for additional contrast injection to evaluate delayed contrast enhancement, as would have been the case in a conventional CT. The pathophysiological substrate of CT delayed enhancement (iodine) is compared to delayed enhancement detected by CMR (gadolinium), with very similar pharmacokinetics, assessing the ECV expansion associated with interstitial fibrosis and myocardial damage.

Even though CMR provides excellent quality imaging to evaluate HCM, it has some limitations regarding patient selection, including claustrophobia and intolerance, renal failure, or implanted cardiac devices

incompatible with MRI.

In our patient, typical ECG signs of hypertrophy-ischemia, as detected in septal and apical forms, were absent, although the atypical findings described usually coincide with this type of cardiomyopathy.

In conclusion, spectral cardiac CT angiography is presented as a valid and feasible option in clinical practice for the evaluation of HCM, in patients with contraindications for CMR, particularly if new symptoms occur, or in obstructive forms in patients eligible for septal ablation whose anatomy of the septal branches is to be evaluated. Furthermore, this method could emerge with the potential to evaluate both coronary anatomy and the presence of perfusion disorders and myocardial fibrosis at the same time, comprehensively characterizing the HCM phenotype.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

**Lucía Fontana, Gastón Rodríguez Granillo ,
Carlos Ingino , Marcos Cerón, Pedro Lylyk**

Instituto Médico ENERI, Clínica La Sagrada Familia
Av. Libertador 6647 (C1428ARJ) Buenos Aires, Argentina
e-mail: grodriguezgranillo@gmail.com - Phone: (011) 40147012

REFERENCES

1. Nakamura S, Kitagawa K, Goto Y et al. Prognostic Value of Stress Dynamic Computed Tomography Perfusion With Computed Tomography Delayed Enhancement. *JACC Cardiovasc Imaging* 2020;13:1721-34. <https://doi.org/10.1016/j.jcmg.2019.12.017>
2. Kalisz K, Rajiah P. Computed tomography of cardiomyopathies. *Cardiovasc Diagn Therapy* 2017;7:539-56. <https://doi.org/10.21037/cdt.2017.09.07>
3. Lee HJ, Im DJ, Youn JC et al. Myocardial Extracellular Volume Fraction with Dual-Energy Equilibrium Contrast-enhanced Cardiac CT in Nonischemic Cardiomyopathy: A Prospective Comparison with Cardiac MR Imaging. *Radiology* 2016;280:49-57. <https://doi.org/10.1148/radiol.2016151289>
4. Danad I, Fayad ZA, Willeminck MJ, Min JK. New Applications of Cardiac Computed Tomography: Dual-Energy, Spectral, and Molecular CT Imaging. *JACC Cardiovasc Imaging* 2015;8:710-23. <https://doi.org/10.1016/j.jcmg.2015.03.005>
5. Rassouli N, Etesami M, Dhanantwari A, Rajiah P. Detector-based spectral CT with a novel dual-layer technology: principles and applications. *Insights Imaging* 2017;8:589-98. <https://doi.org/10.1007/s13244-017-0571-4>

Rev Argent Cardiol 2021;89:54-55.
<http://dx.doi.org/10.7775/rac.v89.i1.19665>

Giant Coronary Aneurysms and Acute Coronary Syndrome

Coronary artery aneurysms are rare anomalies. The first case was reported on post mortem examination by Morgagni in 1761, and later by Bourgon in 1812.

These aneurysms are defined as a fusiform or sac-

cular dilation of the coronary artery, exceeding 1.5 to 2 times the normal adjacent segment vessel diameter. They can be fusiform, with the longitudinal diameter larger than the transverse diameter, or saccular, with the transverse diameter larger than the longitudinal diameter. Fusiform aneurysms are more common than saccular aneurysms.

The American Heart Association Committee has defined giant aneurysms as those >8 mm diameter; other authors consider an aneurysm to be giant when it is >20 mm. (1) Giant aneurysms have an incidence of 0.02%, and most of them involve the right coronary artery. (2)

Atherosclerosis is the most prevalent cause, accounting for more than 50% of coronary aneurysms in adults. Reported complications include thrombo-

sis and distal embolization, rupture and vasospasm. Coronary aneurysms cause ischemia, heart failure, arrhythmias and, less frequently, compression of surrounding structures and fistulization into one of the cardiac chambers. (3)

It is a rare condition, with an estimated incidence between 0.15 and 4.9%. The most common location is the right coronary artery, followed by the anterior descending artery. Involvement of three vessels or of the left main coronary artery is rare. Its incidence is growing due to increased coronary catheterizations and stent implantations. (4, 5)

Patients may present with a condition consistent with acute coronary syndrome or with other symptoms caused by extrinsic compression of adjacent structures (in the case of giant aneurysms). The most common complications include myocardial ischemia or infarction, and aneurysm rupture. Giant aneurysms can be associated with cardiac chamber fistulas causing heart failure, ECG changes or infective endocarditis.

Aneurysms can be diagnosed by noninvasive techniques including transthoracic echocardiography, transesophageal echocardiography, computed tomography (CT), and magnetic resonance angiography (MRA). Coronary angiography remains the gold standard for the evaluation of aneurysmal coronary artery disease. (6)

We report the case of multiple coronary aneurysms in an elderly female patient with several cardiovascular risk factors and angina pectoris.

This is the case of a 70-year-old female patient, with hypertension, dyslipidemia, and a history of hemorrhagic stroke due to brain aneurysm, and infrarenal abdominal aortic aneurysm. In the previous month, she had been admitted for acute myocardial infarction (AMI), which was resolved with medical treatment, without performing complex studies.

Once again, the patient presented with an episode of typical angina, arterial hypotension, and changes in baseline ECG with ST-segment depression in the inferolateral wall, so she was urgently evaluated in the emergency room.

Admission to the coronary care unit was decided, with a diagnosis of post-AMI angina. The urgent coronary arteriography showed a 12-15 mm fusiform aneurysm of the left main coronary artery, proximal occlusion of the anterior descending artery, proximal fusiform aneurysm of the anterior descending artery, and large tandem saccular aneurysms proximal to the circumflex artery. (Figures A-D)

Pre-surgical Doppler echocardiography showed slightly enlarged left ventricle with moderate systolic function impairment, Simpson's biplane ejection fraction of 38%, akinesis of the anterior septum from base to apex and of the entire apical cap; mid-anterior and mid-lateral hypokinesis, moderate to severe aortic stenosis, restrictive mitral filling pattern, and moderate tricuspid regurgitation with severe pulmonary hyper-

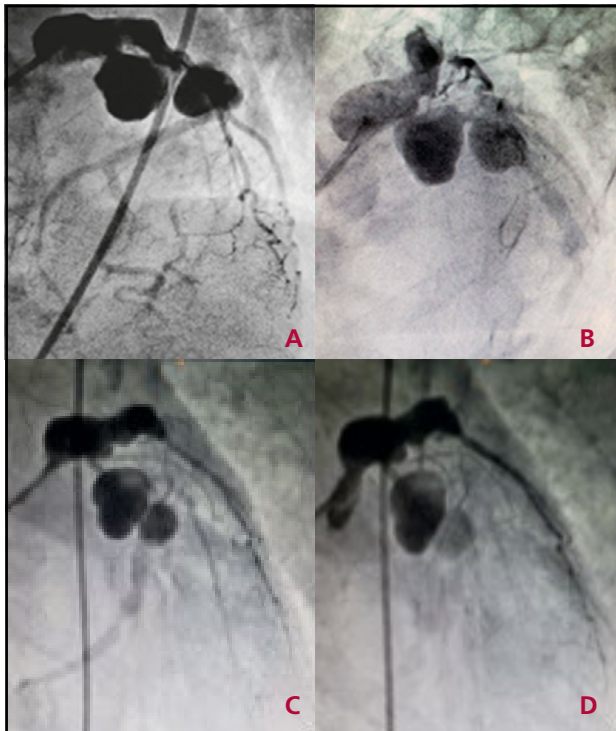


Fig. A: Right caudal oblique projection showing fusiform aneurysm of the left main coronary artery extending into the proximal third of the anterior descending artery, plus two 22-20 mm saccular aneurysms in the proximal third of the circumflex artery. **Fig. B:** Left caudal oblique projection showing fusiform aneurysm of the left main coronary artery extending into the proximal third of the anterior descending artery. Tandem saccular aneurysms in the proximal third of the circumflex artery. The marginal branch arises from the second aneurysm with severe obstruction at the origin. **Fig. C:** Fusiform aneurysm of the left main coronary artery extending into the proximal third of the anterior descending artery —which is completely hidden, showing only the mid and distal third of the vessel with homo-coronary circulation. **Fig. D:** Right caudal oblique projection showing left main coronary artery without involvement of the origin, immediately following a 12 mm diameter fusiform aneurysm extending into the proximal third of the anterior descending artery. Giant saccular aneurysms in the proximal third of the circumflex artery.

tension (PASP of 50 mmHg).

Incomplete revascularization during coronary artery bypass grafting (internal thoracic artery to anterior descending artery, and venous bypass to circumflex artery) associated with aortic bioprosthetic valve replacement #23 with longer cardiopulmonary bypass perfusion time and prolonged total aortic clamping were performed.

Evolution was torpid, with acute renal failure due to acute tubular necrosis, resulting in 16 hemodialysis sessions that improved renal function and diuretic rhythm. During a hemodialysis session, the patient suffered a cardiopulmonary arrest for a few minutes, but recovered vital parameters and sinus rhythm.

The patient progressed with cardiogenic shock, requiring a Swan Ganz catheter for monitoring, inotropic and vasoactive drugs and volume and pressure management. As a result, clinical and hemodynamic improvement was achieved.

Once hemodynamically stable, the patient was discharged after prolonged hospitalization.

She is currently asymptomatic, with good clinical outcome and adequate response to medical and surgical treatment.

Coronary artery aneurysms are uncommon, and usually an incidental finding. The main reason for coronary angiography is precordial pain, and the most common cardiovascular risk factors include hypertension and dyslipidemia. (4)

Aneurysmal segments produce slow and turbulent coronary blood flow, and microcirculatory dysfunction resulting in decreased coronary flow reserve leading to myocardial ischemia, especially during exertion. Infarction has been attributed to intra-aneurysmal thrombosis or embolization of thrombus distally to the aneurysmal sac.

We are uncertain about the mechanism that produces coronary aneurysms, but it is believed that they are triggered by coronary atherosclerosis, which affects the middle layer of the vessel and causes endothelial release of vasodilator substances.

It is known that coronary angiography is the diagnostic method of choice, but they can also be detected by Doppler echocardiography, and magnetic resonance imaging, which accurately assesses aneurysms, detects mural thrombi, and is very useful for noninvasive follow-up of these patients.

In the *Coronary Artery Surgery Study (CASS) Registry*, the angiographic incidence of coronary aneurysm was 4.9% in a group of 20,087 patients [12], which exceeded the incidence reported in many other angiographic studies (0.37–2.53%).

Furthermore, due to the lack of consensus to define

this entity as well as the variability of its symptoms and its morbidity and mortality, management of coronary aneurysms is not well defined. Sometimes, treatment with anticoagulants and antiplatelet drugs or covered stent implantation in the area of the lesion are considered, and aggressive modification of coronary artery risk factors should also be implemented.

Coronary artery bypass surgery is recommended in patients with recurrent infarction or angina associated with high risk of giant aneurysms, that increases in cases of saccular and large aneurysms, or in the presence of symptomatic compression, with good mid-term survival.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

**Agustín Bartoli , Camila Chort , Ramiro Ayala ,
Kenneth Schmidt , Jorge Montecinos ,
Luis Mantilla**

Department of Cardiology. Sanatorio Adventista del Plata,
Entre Ríos, Argentina
e-mail: agustinbartoli90@hotmail.com
Phone: +54 (9) 341 15 5443665
Fax: (0343) 4200-290

REFERENCES

1. Babes E, Babes V, Zdrinca M, Brihan I, Vicas R, Motorca, M et al. Multiple and giant coronary artery aneurysm – case report and a review of the literatura. *Rom J Morphol Embryol* 2020;61:551-4. <https://doi.org/10.47162/RJME.61.2.26>
2. Halapas A, Lausberg H, Gehrig T, Friedrich I, Hauptmann K. Giant Right Coronary Artery Aneurysm in an Adult Male Patient with Non-ST Myocardial Infarction. *Hellenic J Cardiol* 2013;54:69-76. https://www.hellenicjcardiol.org/archive/full_text/2013/1/2013_1_69.pdf
3. Núñez G, Alberca P, Nieves G, Nombela L, Salinas P, Fernández A. Giant coronary aneurysm culprit of an acute coronary síndrome. *Rev Portug Cardiol* 2018;37:203.e1-203.e5. <https://doi.org/10.1016/j.repc.2016.11.017>
4. Flamarique S, Cembreroa H, Artaizc M, Rábagob G, Hernández-Estefanía R. Características morfológicas de los aneurismas de arterias coronarias. Incidencia e implicancia clínica. *Cir Cardio* 2014;21:252–8 <http://dx.doi.org/10.1016/j.circv.2014.01.009>
5. Liévano M, Sánchez J, Acosta G, Acosta J, Olaya H. Enfermedad aneurismática coronaria. *Rev Colomb Cardiol* 2020; 27:485-90. <https://doi.org/10.1016/j.rccar.2019.09.006>
6. ElGuindy M, ElGuindy A. Aneurysmal coronary artery disease: An overview. *Glob Cardiol Sci Pract* 2017;26. <http://dx.doi.org/10.21542/gcsp.2017.26>

Rev Argent Cardiol 2021;89:55-57.

<http://dx.doi.org/10.7775/rac.v89.i1.19722>