

Evaluation of Low-Molecular-Weight Heparin Dose Adjusted by Weight for Patients with Cardiovascular Disease

Thromboembolic diseases are a common cause of cardiovascular morbidity and mortality, and their prevention depends on adequate antithrombotic therapy. Drug dose recommendations are based on clinical trials evaluating the risk-benefit of the therapies employed. This is particularly important for heparins that require doses adjusted by body weight. Precise weight-adjusted dosing has been performed in randomized trials testing these drugs. Based on these results, recommendation guidelines for atrial fibrillation (AF), pulmonary thromboembolism (PTE), deep vein thrombosis (DVT), and acute coronary syndromes (ACS) suggest anticoagulation therapy with low-molecular-weight heparin (LMWH) at 1 mg/kg body weight every 12 hours to prevent thromboembolic events with low risk of bleeding.

This study has been conducted to determine the actual usefulness of LMWH depending on estimated weight-adjusted dosing.

To understand the usefulness of LMWH in patients requiring anticoagulation for cardiac reasons, an analysis of patients under LMWH was performed by comparing estimated weight-adjusted dosing versus actual weight-adjusted dosing.

Before discharge, inpatient data with conditions requiring LMWH were collected from both public and private tertiary cardiology services. The administered dose and its relationship with the body weight estimated by the attending physician were evaluated, and the actual patients' weight was measured with an analog scale. A range of ± 10 kg of the estimated weight compared with the actual weight was considered adequate. Patients with contraindications for LMWH, creatinine clearance (Cockcroft-Gault equation) < 30 ml/min, or weighing > 100 kg were excluded from the study. Continuous variables were expressed as mean \pm standard deviation, and events, in percentages.

A total of 102 patients were included with mean age of 68 years; 33% were women. The reason for anticoagulation was 1 patient with left ventricular thrombus, 11 patients with mechanical valve, 19 patients with acute coronary syndromes, 27 patients with DVT/PTE, and 43 patients with atrial flutter/AF (Table 1). Mean creatinine clearance was 89.2 ml/min, and enoxaparin was the heparin used in all patients. After adjusting LMWH dose by actual weight (1 mg/kg every 12 h), it was seen that 62.8% of patients were improperly anticoagulated. Among the anticoagulated patients under improper dosage, 95.2% was receiving only one dose.

This series revealed that estimated weight entails incorrect LMWH anticoagulation doses in pa-

Table 1. Baseline characteristics and results

Baseline characteristics and results	
Patients (n)	102
Male (%)	67.6
Mean age (years)	68 \pm 12
Reason for anticoagulation (n)	
*Atrial fibrillation/Atrial flutter	43
*DVT/PTE	27
*ACS	19
*Mechanical valve	11
*LV thrombus	1
Cr Clearance (mean ml/min)	89.2 \pm 56
Mean weight (kg)	77.7 \pm 13
Sub-therapeutic range (%)	95.2
Enoxaparin (%)	100

ACS: Acute coronary syndrome. Cr Clearance: Creatinine clearance. DVT/PTE: Deep vein thrombosis/Pulmonary thromboembolism. LV thrombus: Left ventricular thrombus

tients with cardiovascular diseases. Not measuring patients' weight systematically is the major determinant of proper dosage failure. In a study of patients with ACS, Macie et al. observed that only 1 out of 10 patients treated with LMWH had had their weight measured in the Coronary Care Unit. (1) In this regard, weight estimation implies an error of 9-10 kg, even reaching an interindividual variability of up to 20%. This estimation differs depending on who calculates it. Patients' own weight estimates are likely to be more accurate than those of physicians or nurses. (2, 3) There are other factors involved in correct dosing; weight changes during the course of hospitalization, generally decreasing as a result of diet, and in patients with cardiovascular disease, weight is related to heart failure treatment. This is a very important aspect to be considered in daily dose adjustment. In our registry, patients were weighed within two days of hospitalization, so as to prevent weight changes from influencing the results.

Another factor is that LMWH comes in fixed doses, which prevents an accurate weight adjustment without drug discarding. All these factors generally determine subtherapeutic anticoagulation doses, and to a lesser extent, doses higher than suggested. The Thrombolysis in Myocardial Infarction (TIMI) 11A trial revealed that a 25% increase in the dose of heparin increased the rate of bleeding without reducing thrombotic events; however, the thrombotic effect of lower dosing is controversial. (4) Xu et al. have demonstrated that lower-than-required dose in anticoagulated patients with LMWH was not associated with increased risk of thrombotic events, whereas an overdose caused major bleeding according to multivariate analysis. Neverthe-

less, the sub-therapeutic LMWH dose is associated with the individual effect on anti-Xa activity. Although low anti-Xa activity increases 30-day mortality, implications in our series are unknown because it was not measured. (5)

This report evidences the value of using weight-adjusted LMWH dosing and the difficulty of such adjustment in daily practice, which may require anti-Xa activation measurement. Another possibility is to consider the use of new oral anticoagulation agents that do not involve weight adjustment or factor measurement to prevent thromboembolic events with low bleeding risk.

Anticoagulation with estimated weight-based LMWH dosing entails an inadequate dose of heparin, generally due to weight underestimation. Measuring actual body weight, not usually performed, is essential for a correct anticoagulation therapy.

Conflicts of interest

None declared.

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

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REFERENCES

1. Macie C, Forbes L, Foster GA, Douketis JD. Dosing practices and risk factors for bleeding in patients receiving enoxaparin for the treatment of an acute coronary syndrome. *Chest* 2004;125:1616-21. <http://doi.org/c7kv2d>
2. Anglemyer BL, Hernandez C, Brice JH, Zou B. The accuracy of visual estimation of body weight in the ED. *Am J Emerg Med* 2004;22:526-9. <http://doi.org/cg9bmb>
3. Fernández CM, Clark S, Price A, Innes G. How accurately do we estimate patients' weight in emergency departments? *Can Fam Physician* 1999;45:2373-6.
4. Thrombolysis in Myocardial Infarction (TIMI) 11A Trial Investigators. Dose-ranging trial of enoxaparin for unstable angina: results of TIMI 11A. *J Am Coll Cardiol* 1997;29:1474-82. <http://doi.org/dfbk9f>
5. Xu H, Cai H, Qian Z, Xu G, Yan X, Dai H. (2012). Dosing practice of low molecular weight heparins and its efficacy and safety in cardiovascular inpatients: a retrospective study in a Chinese teaching hospital. *BMC cardiovascular disorders*, 12. <http://doi.org/gbcb2x>

REV ARGENT CARDIOL 2017;85:445-446. <http://dx.doi.org/10.7775/rac.v85.i5.11655>

Prosthetic Dehiscence as Cause of Acute Coronary Syndrome

Prosthetic dehiscence is a complication consisting in a solution of continuity of the sutures that connect the valve prosthesis with its ring, resulting in eccentric valve regurgitation.

The incidence of significant paravalvular regurgitation in a prosthetic valve is 1-5%. (1, 2) It can be

mild and have a benign course or present symptoms of heart failure or hemolytic anemia, which is the complication reflected in our uncommon clinical case. Some of the predisposing factors include a severely calcified valve annulus, infective endocarditis (IE), chronic use of steroids, and poor surgical technique. (3) Its early postoperative onset is common in patients with severe mitral annulus calcification, making prosthesis fixation difficult. If there is no hemodynamic involvement, and if failure is not severe, the treatment approach can be conservative, since it is usually mild and decreases, or even disappears with time. Late dehiscence occurs commonly as the result of IE and its diagnosis is clinical and echocardiographic.

Transthoracic echocardiography (TTE) is useful to identify paravalvular prosthetic valve regurgitation. Acoustic shadowing, so problematic in mitral prosthesis, is a minor inconvenience for prosthetic aortic regurgitation. However, transesophageal echocardiography (TEE) provides uniform, better images than those from TTE for prosthetic valve assessment, given the proximity of the esophagus to cardiac structures. Two-dimensional (2D)-TEE can miss significant findings as it only presents images from one plane of the heart. Three-dimensional (3D) TEE, on the other hand, provides improved spatial resolution compared to conventional 2D TEE, allowing a complete visualization of the prosthesis. (4, 5)

Echocardiographic findings will include a high velocity and intense eccentric regurgitant jet, which usually courses adhered to the receptive chamber wall and its level can be determined using the usual criteria for valve diseases. If dehiscence is significant, pros-



Fig. 1

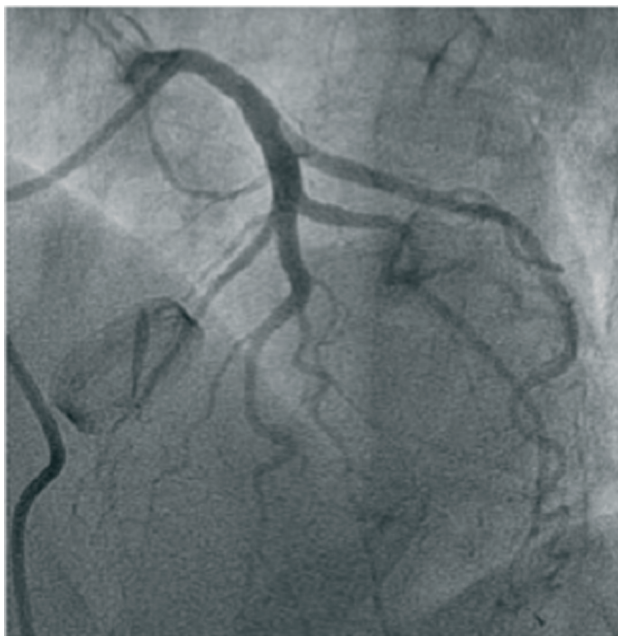


Fig. 2

thetic excursion can be detected by 2D, 3D, or M-mode echocardiography. In these cases, an excessive movement of the prosthetic ring is observed, somewhat independent of the movement of the other cardiac structures. (6) Significant paravalvular regurgitation requires intervention, which may be surgical or percutaneous. Traditionally, surgical treatment of paravalvular regurgitation has been the standard practice. However, over the past 20 years, transcatheter closure methods have been developed for paravalvular regurgitation. (4)

We describe the case of a 70-year old woman with history of psoriatic arthritis, recurrent uveitis, and with a mechanical 23 mm CarboMedics aortic prosthesis since 2001 due to severe aortic regurgitation. On admission, the patient presented with clinical signs of chest pain, dyspnea, and slightly increased myocardial necrosis markers, without ECG abnormalities, and was admitted to the Cardiac Intermediate Care Unit with diagnosis of non-ST segment elevation acute coronary syndrome (NSTEMI-ACS) and heart failure (HF). Cardiac catheterization evidenced aortic prosthetic excursion, suggestive of prosthetic dehiscence (Video 1). Coronary angiography showed acute thrombotic occlusion of the mid segment of the anterior descending coronary artery (Figure 1), which was effectively treated with aspiration thrombectomy, without need for a stent (Figures 2). 2D and 3D TEE confirmed prosthesis dysfunction due to dehiscence along the anterior portion of the ring, causing severe aortic regurgitation (Video 2). The patient underwent emergency aortic valve replacement with a 26 mm St Jude Trifecta Valve prosthesis. The aspect of the inflammatory periprosthetic tissue was remarked

during the procedure; however, all the cultures were negative.

This case shows the importance of always bringing into consideration a possible prosthetic dysfunction in prosthetic valve patients, even in cases of uncommon ACS, with echocardiography being the essential diagnostic tool.

Conflicts of interest

None declared.

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

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REFERENCES

1. Tominaga R, Kurisu K, Ochiai Y, Tomita Y, Masuda M, Morita S, et al. A 10-year experience with the Carbomedics cardiac prosthesis. *Ann Thorac Surg.* 2005;79:784-9. <http://doi.org/fkrgtg>
2. Koo HJ, Yang DH, Kang JW, Han K, Chung CH, Song JK, et al. Demonstration of prosthetic aortic valve dehiscence in a patient with noninfectious aortitis by multimodality imaging findings of echocardiography and computed tomography. *Circulation* 2013;128:759-61. <http://doi.org/chtr>
3. Miller DL, Morris JJ, Schaff HV, Mullany CJ, Nishimura RA, Orszulak TA. Reoperation for aortic valve periprosthetic leakage: identification of patients at risk and results of operation. *J Heart Valve Dis.* 1995;4:160-5.
4. Tsang W, Weinert L, Kronzon I, Lang RM. Ecocardiografía tridimensional en la evaluación de las válvulas protésicas. *Rev Esp Cardiol.* 2011;64:1-7. <http://doi.org/fmz2kq>
5. Ionescu A, Fraser AG, Butchart EG. Prevalence and clinical significance of incidental paraprosthetic valvar regurgitation: a prospective study using transoesophageal echocardiography. *Heart.* 2003;89:1316-21. <http://doi.org/ff77gz>
6. Rodríguez Padiá L. *Ecocardiografía.* Barcelona: Edicomplet; 2006.

Videos are available at:

<https://youtu.be/cSxXnNrRE40>

<https://youtu.be/UOsgUzSblCY>

REV ARGENT CARDIOL 2017;85:446-447. <http://dx.doi.org/10.7775/rac.v85.i5.10174>

Infective Endocarditis Caused by *Streptococcus suis*

Streptococcus suis infection (*S. suis*) is an uncommon emerging zoonosis, secondary to being exposed to swine food or swine-related occupations. (1) This pathogen can cause severe systemic infection in humans (2), including meningitis in the first place, followed by sepsis, arthritis, endocarditis, and endophthalmitis. (3) The first human case was reported in Denmark in 1968, and since then, an increasing number of human cases have been reported in Southeast Asia and Northern Europe. Recently, this pathogen has been identified as the first and third agent caus-

ing bacterial meningitis in Vietnam and Hong Kong, respectively. (4)

In Latin America, the first two cases were reported in Argentina in 2005-2008. Currently, Argentina is among the Western countries with the highest number of human cases reported, after the Netherlands, France, and the UK, with a number of cases similar to Poland (Figure 1). However, the European countries began the identification of *S. suis* in humans more than 15 years before Argentina, so the total number of human cases in our country may have been underestimated. (6)

S. suis natural habitat is the pig's upper respiratory tract, particularly the tonsils and nasal cavities, as well as the genital, and alimentary tracts. (7) It has also been isolated in other animals, like rodents, cats, dogs, deer, and horses, and it is thought to be a commensal in the intestinal flora. Infection in humans is caused through lesions in the skin, oral mucosa, and nasal cavity, or by ingesting contaminated food. Among the 35 known serotypes, serotype 2 is the most pathogenic both for pigs and humans, and the most commonly isolated in humans, with serotype 1, 4, 14, 16 reported in a fewer number of cases. (4)

Patients do not usually have previous conditions at the time of *S. suis* infection, although some predisposing factors such as splenectomy, diabetes mellitus, alcoholism, malignant tumors, and structural heart diseases have been reported.

Misdiagnosing *S. suis* infection is not uncommon, both by conventional biochemical testing and commercial identification systems. Bacteria are often reported as *Streptococcus viridans* in 70% of cases. (5)

According to the information available, penicillin,

ceftriaxone, or vancomycin are the drugs used to correctly treat meningitis caused by this pathogen. No clinical data are available regarding the treatment of the infection in places other than the central nervous system. (7)

We report the case of a 42-year old male rural worker dedicated to swine husbandry. He had a history of herniated intervertebral thoracic disc, for which he was being treated with depot corticosteroid 6 months prior to consultation, associated to daily fever episodes of 40 °C during the previous month. During hospitalization, sinus tachycardia and systo-diastolic murmur of grade 3/6 intensity and maximum auscultation in the aortic area were detected, with no signs of heart failure. Echocardiography targeted mobile vegetation in the aortic valve and sessile vegetation in the anterior mitral leaflet with severe aortic regurgitation and mild mitral regurgitation (Figure 2).

Initially, blood cultures revealed growth of alpha hemolytic, gram-positive cocci in chains. Treatment with penicillin G 3,000,000 units every 4 hours and gentamicin 80 mg/8 hours was started. The Department of Bacteriology decided to send the sample for identification to a specialized laboratory.

Patient progressed with heart failure and no response to medical treatment so an emergency bivalvular mechanical mitral-aortic valve replacement was performed on the 4th day of hospitalization. Immediate postoperative course presented with left facio-brachio-crural hemiparesis. Brain magnetic resonance imaging with SWAN technique showed hypointense focal images of cortical-subcortical location in both cerebral hemispheres, and others in the cerebellar hemispheres associated with microhemorrhages.

The report from a specialized laboratory (*Hospital Malbrán*) on the specimen collected revealed isolation of *S. suis*, and antibiotic treatment was continued.

During the late postoperative course, the patient had evening fever episodes. Different causes were studied, with no positive results. The condition resolved and the patient was discharged after a 6-week course of intravenous antibiotics.

A case of infective endocarditis was reported with very rare pathogen detection in our setting, *S. suis*. The patient worked in swine husbandry and marketing without any protection against animal handling (gloves, protective goggles, etc.), which increases susceptibility to infection associated to immunosuppression caused by chronic corticosteroid therapy.

The isolation of this pathogen raised a campaign from the Department of Epidemiology of *Hospital Castro Rendón* to detect it at the place where the patient comes from, collecting samples from the upper airway tract of the animals they take care of. In turn, the contact with *Hospital Malbrán* and Dr. Marcelo Gottschalk's laboratory (Canada) to confirm the serotype and determine the sequence type (ST) by multilocus sequence typing (MLST) is still pending.

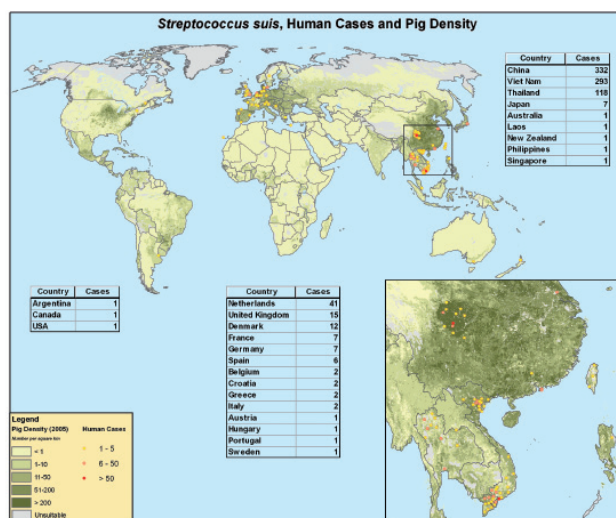


Fig. 1. World map of human *Streptococcus suis* cases with background pig density data. Published with permission from the Infectious Diseases Research Foundation (World Atlas of Infectious Diseases Project). (7)

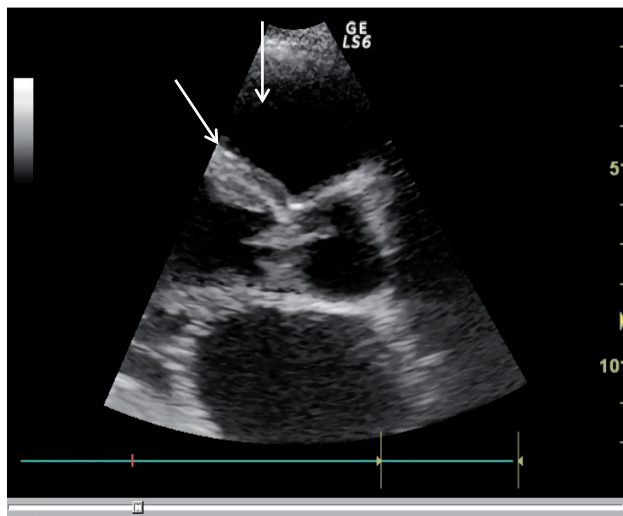


Fig. 2. Transthoracic echocardiography image, zoom of left parasternal axis. The arrows show vegetation in the aortic and mitral valves.

Conflicts of interest

None declared.

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

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REFERENCES

- Huong VT, Ha N, Huy NT, Horby P, Nghia HD, Thiem VD, et al. Epidemiology, clinical manifestations, and outcomes of *Streptococcus suis* infection in humans. *Emerg Infect Dis*. 2014;20:1105-14. <http://doi.org/f58nmm>
- Lun ZR, Wang QP, Chen XG, Li AX, and Zhu XQ. *Streptococcus suis*: an emerging zoonotic pathogen. *Lancet Infect Dis* 2007;7:201-9. <http://doi.org/bjt8qj>
- Burjel J, Aliandre V, Lamarca D, Pacello F, Stagno M, Majó C, et al. Meningitis a *Streptococcus Suis*. Reporte de un caso a propósito de una zoonosis emergente. Congreso Uruguayo de Neurología, poster N.º 2, 5-7 Noviembre 2013 (Arch. Inst. Neurol. (Montev). ed. Especial, 2014).
- Zalas-Wiecek P, Michalska A, Grabczewska E, Olczak A, Pawłowska M, Gospodarek E. Human meningitis caused by *Streptococcus suis*. *J Med Microbiol* 2013;62:483-5. <http://doi.org/chts>
- Lopreto C, Lopardo HA, Bardia MC, Gottschalk M. Meningitis primaria por *Streptococcus suis*: primer caso en humanos descrito en América Latina. *Enferm Infecc Microbiol Clin* 2005;23:110-2. <http://doi.org/ckdkjq>
- Wertheim HF, Nghia H, Taylor W, Schultsz C. *Streptococcus suis*: an emerging human pathogen. *Clin Infect Dis* 2009;48:617-25. <http://doi.org/fkzj6j>
- Callejo R, Zheng H, Du P, Prieto M, Xu J, Zielinski G, et al. *Streptococcus suis* serotype 2 strains isolated in Argentina (South America) are different from those recovered in North America and present a higher risk for humans. *JMM Case Reports* 2016;3:e005066. <http://doi.org/gbngd9>

Congenital Fistula Between the Internal Mammary Artery and the Pulmonary Trunk

Congenital internal mammary artery to pulmonary artery fistula is a rare condition, and its diagnosis is exceptional in pediatric patients. (1) The first case of mammary artery to pulmonary artery fistula was published in 1947, (2) and very few cases have been described since. This rare condition can be either congenital (it occurs in 1 out of 50,000 patients with congenital heart disease) or acquired (usually secondary to coronary artery bypass surgery, traumas, inflammation, or neoplasia), (3) and its diagnosis is exceptional in patients with no evidence of disease or triggering factors. (4)

Congenital forms are associated to pulmonary atresia or tetralogy of Fallot among other heart diseases, and to pulmonary sequestration and arteriovenous malformations. The embryonic origin of these connections is not well known, although it is believed that in these cases, systemic-pulmonary fistulas are formed when the main pulmonary arterial system does not develop continuity with the embryonic lung and cannot form a normal pulmonary arterial tree; (4) this is supported by the common embryonic origin of the chest wall and the pulmonary tree. Several authors have suggested that congenital fistulas arise because pulmonary capillary vessels and the aorta, which connect systemic and pulmonary circulation in the fetus, fail to regress. (5)

In general, patients with congenital fistulas but no other associated anomalies are asymptomatic. It is diagnosed in a study to detect heart murmur, although its clinical presentation depends partly on the functional repercussion of the fistula, which will be proportional to the size of the implicated vessels, and where it is located in relation to the heart or drainage site.

With time, fistulas may cause vessel dilation and symptoms such as congestive heart failure, bacterial endocarditis and/or rupture. Treating this condition is controversial and the options are an expectant attitude, percutaneous closure, or surgery. (2)

We describe the case of a 10-year old boy with no relevant medical history, who was referred for heart murmur evaluation. Physical examination showed a continuous heart murmur at the left superior sternal border, with no signs of heart failure. The electrocardiogram showed no repolarization changes or other anomalies. Doppler-echocardiography detected diastolic flow suggestive of fistula at the pulmonary trunk, without coronary artery dilation or anomalies or evidence of the origin of such drainage. No other structural abnormalities or change in the size of heart chambers were targeted. Although the initial suspicion was of coronary artery fistula, the evaluation was completed with a contrast computed tomographic angiography, which revealed an anomalous vessel with a tortuous path at the level of the internal

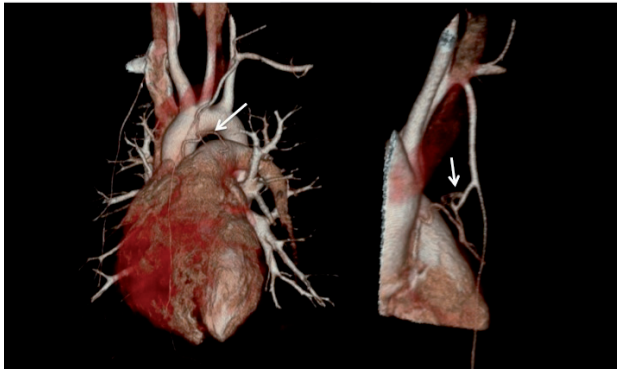


Fig. 1

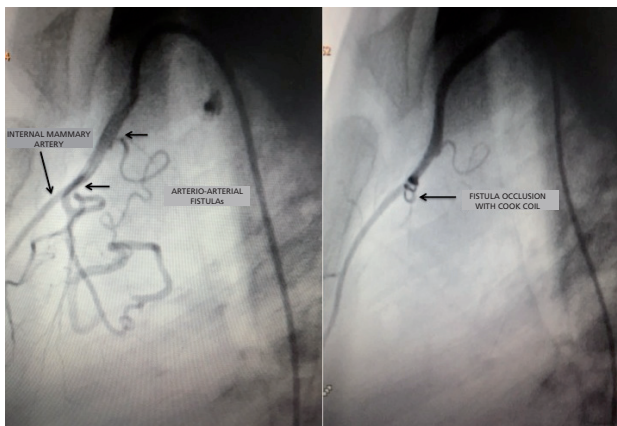


Fig. 2

mammary artery draining in the main pulmonary artery (Figure 1). Further angiographic evaluation was decided, which confirmed the fistula and another one of smaller caliber in the immediately upper portion of the internal mammary artery. Despite the patient was asymptomatic, given that the fistula size was significant and could cause complications in the future, uneventful embolization of the larger vessel was successfully performed with a Cook coil (Figure 2). The patient has remained asymptomatic and without further complications.

Although coronary artery fistulas are quite common anomalies, they are difficult to differentiate from those originated at another level, as is the case of this arterio-arterial fistula with greater potential risk for long-term complications. Therefore, when in doubt, diagnosis should be completed with other imaging techniques or with angiography, which is useful to determine the size, location, and shunt hemodynamics.

When the patient is asymptomatic and presents no other disorders, treating this condition is contro-

versial because its progression is unknown due to the limited caseload. Some authors choose the expectant attitude approach, although in most cases surgery is the option due to its positive outcome and low morbidity and mortality. (2) Percutaneous closure of these defects is relatively recent, the case described by Fernández et al. in 2004 using a mixed technique with an Amplatzer Duct Occluder (ADO) associated to coils, being the first one described in the literature. (2) Since then, other authors have performed percutaneous closure with stents or fluid embolization with N-butyl cyanoacrylate with positive outcomes in adult patients.

Although congenital isolated arterio-arterial mammary artery-to-pulmonary artery fistula is a rare condition, percutaneous closure can be useful in pediatric patients as demonstrated in our case.

Conflicts of interest

None declared.

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

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REFERENCES

1. Alsidawi S, Abdalla M, Arif I, López-Candales A. Congenital anastomosis between left anterior mammary and pulmonary vasculatures. *Am J Med Sci*. 2013;345:158-9. <http://doi.org/cc6h>
2. Burchell HB, Clagett OT. The clinical syndrome associated with pulmonary arteriovenous fistulas including a case report of a surgical cure. *Am Heart J* 1947;34:151-62. <http://doi.org/b2vwwb>
3. Peter AA, Ferreira AC, Zelnick K, Sangosanya A, Chirinos J, de Marchena E. Internal mammary artery to pulmonary vasculature fistula--case series. *Int J Cardiol* 2006;108:135-8. <http://doi.org/cnd-mqk>
4. Geyik S, Yavuz K, Keller FS. Unusual systemic artery to pulmonary artery malformation without evidence of systemic disease, trauma or surgery. *Cardiovasc Intervent Radiol* 2006;29:897-901. <http://doi.org/bpnc8c>
5. Fernández FJ, Montes PM, Alcívar J, Rodrigo D, Barrenetxea JI, Gotxi R. Percutaneous closure of complex fistula between the internal mammary artery and a lobar branch of a pulmonary artery. *Rev Esp Cardiol* 2004;57:585-8. <http://doi.org/cc6j>
6. Nakai M, Ikoma A, Sato H, Minamiguchi H, Sonomura T. Transcatheter Embolization of an Internal Mammary Artery-to-Pulmonary Artery Fistula Using N-Butyl Cyanoacrylate and Temporary Dual-Balloon Occlusion. *J Vasc Interv Radiol* 2017;28:156-7. <http://doi.org/cc6k>

REV ARGENT CARDIOL 2017;85:449-450. <http://dx.doi.org/10.7775/rac.v85.i5.10290>