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Ductal stenting in congenital heart disease with duct dependent pulmonary blood flow

Stent ductal en cardiopatía congénita con flujo pulmonar dependiente del ductus

To the Editor,

Stenting of ductus arteriosus in patients with congenital heart disease and duct-dependent pulmonary flow has a long history and validated outcomes.[1,2]

Our objective was to retrospectively review our experience of this technique. We collected cases of patients born between January 2008 and July 2019, who had been discussed at medical-surgical meetings and referred for ductal stenting based on their underlying heart defect, comorbidities, and ductal anatomy. A total of 32 neonates were included.

Table 1 shows the patients' characteristics; the most common defect was pulmonary atresia with intact ventricular septum (PAIVS), with 7 cases.

We provided prostaglandin E_1 at the dose required to achieve preprocedure saturations of 70% to 75%. For patients with possibly transient (days to at least a week) duct dependence (such as PAIVS, pulmonary stenosis following valvuloplasty, or Ebstein anomaly), we waited 2 to 3 weeks before performing the procedure; in all other cases, it was performed within the first 7 to 10 days.

The location and characteristics of the ductus (eg, tortuosity or peripheral pulmonary stenosis) are prognostic factors for procedural success and duration,[3] allowing a distinction to be made between complex and relatively noncomplex (normally-positioned and straight) cases.

It is not uncommon to find peripheral pulmonary stenosis (25%) due to the presence of ductal tissue in the branches, which in severe cases requires the placement of longer stents to fully cover the ductus and also treat the peripheral stenosis.[4]

Table 2 provides information on the procedure and follow-up. The procedure was always carried out under general anesthetic. In normally-positioned ductus, access was usually via the femoral artery using a 4-Fr long sheath for implantation. In other locations, access was via the femoral vein with transcardiac passage of a 5–6-Fr guide catheter or a 4-Fr long sheath, plus access via the carotid artery with a 4-Fr short sheath. Fluoroscopy and procedure times decreased with experience, and are currently very short, especially in noncomplex ductus cases; the difference in times between complex and noncomplex cases was not statistically significant, due to the sample size.

Our patients required a median 13 hours' intubation and 3 days' stay in the ICU after the procedure.

The overall success rate was 94% but rose to 100% for cases after 2014. The unsuccessful cases (2/32) corresponded to the initial phase of the series and were due to lack of guidewire stability in complex cases.

The stents used were coronary stents, with a median 1 stent per patient; the last 8 stents implanted were drug-eluting stents to reduce neointimal growth.

Table 1
Patient characteristics

Sex	
Female	14
Male	18
Birth weight, g	3090 (1375-3870)
Gestational age, wk	38 (30-40)
Age at catheterization, d	15 (4-165)
Heart disease	
PAIVS	7
DORV+PS	6
TA	6
PA+IVC	6
PS	3
TA+PS	3
Ebstein anomaly	1
Previous procedures	
Rashkind	2
PVP	8
Shunt	1
Scheduling	
Elective	26
Urgent	6
Initial Nakata index	129 (82-168)
Ductus	
Position	
Transverse	15
Isthmus	13
Brachiocephalic trunk	3
Left subclavian	1
Morphology	
Tortuous	18
Straight	14
Length	15 mm (9-26)
Diameter, mm	
Maximum	3,50 (1–5.5)
Minimum	1 (0.2-3)
Peripheral pulmonary stenosis	8

DORV, double outlet right ventricle; IVC, interventricular communication; PA, pulmonary atresia; PAIVS, pulmonary atresia with intact ventricular septum; PS, pulmonary stenosis; PVP, pulmonary valvuloplasty/valvulotomy; TA, tricuspid atresia; TF, tetralogy of Fallot.
Values are expressed as absolute number of cases or median (range).

Table 2
Procedure and outcomes

Implantation	
Femoral artery	
4-Fr long sheath	15
5-Fr guide catheter	2
Femoral vein	
5–6-Fr guide catheter	12
4-Fr long sheath	1
Carotid artery	
4-Fr short sheath	2
Number of stents	1 (1-4)
Type of stent	
Coronary, bare metal	24
Coronary, drug-eluting	8
Associated procedures	
Rashkind	2
Patent foramen ovale stent	1
Pulmonary valvuloplasty	2
Procedure time, min	
Overall	165 (42-296)
Since 2014	
Normal ductus	65 (51-77)
Complex ductus	106 (58-210)
Fluoroscopy time, min	
Overall	34 (8.38-79)
Since 2014	
Normal ductus	14 (12-14)
Complex ductus	26.2 (17-68)
SaO_2 (%)	
Initial saturation	77 (61-92)
Final saturation	92 (72-100)
Procedural success	30 (94%)
Complications	
End not covered	2
Stent thrombosis	1
Stent straightening	1
Intubation time, h	13.5 (0-96)
ICU time, d	3 (0-56)
Redilatations	
No. of redilatations	6
Days until stenting	145 (22-273)
Nakata index after stenting	256 (155-362)
Need for shunt	2
Time between stenting and first surgery, d	195 (82-539)
Stenting as final therapy	
Patients	5
Follow-up time, y	1-12
Patency	1/5

ICU, intensive care unit; SaO_2, oxygen saturation.
Values are expressed as absolute number of cases or median (range).

Initially, as thromboprophylaxis, we used enoxaparin for 48 hours, followed by aspirin. Currently, with drug-eluting stents, we use enoxaparin plus aspirin for 48 hours and then switch to aspirin plus clopidogrel.

The mortality rate was 0%. There were no significant vascular complications, and the prevalence of complications in the whole series was 13%, which decreased to 8% in procedures performed after 2014. Complications consisted of the following: need for recatheterization in the days after the procedure due to uncovered ductal ends (1 aortic and 1 pulmonary, the first 2 patients in the series), an early stent thrombosis not requiring additional flow (PAIVS with previous valvulotomy, with improvement in pulmonary flow), and 1 case of straightening of the aortic end of the stent, that interfered with the aortic wall and required surgical removal.

Surgical shunts were created in 2 patients with tricuspid atresia who underwent ductus stenting as neonates, because ductal flow remained insufficient, and Glenn procedure was ruled out or delayed. The first of these was in a premature conjoined twin, weighing 1375 g at birth, at 3 months after stenting (without previous stent dilatation). The second was in a patient who weighed 2060 g at birth, at 1.8 years (after maximal stent dilatation).

Six angioplasties were performed during the period analyzed: in 2 patients with comorbidities causing a progressive increase in pulmonary pressure and consequent reduction in ductal flow, the stents were dilated to adapt to the new hemodynamics; in another, with growth, the ductus had stretched due to traction resulting in the aortic end having incomplete coverage (angioplasty with stent); in the 3 others, during surgical planning, it was decided to postpone the procedure and instead to dilate to maintain saturations in the correct range while waiting.

The median time to surgery was 195 days in the 25 patients who underwent surgery; 5 patients had a stent as their final treatment (2 pulmonary stenosis, 3 PAIVS).

Presurgical pulmonary artery branch growth was favorable, increasing from a Nakata index of 123 to 256 mm^2/m^2; on bivariate analysis with the Student t-test, the P value was $< .01$.

In conclusion, stenting of ductus arteriosus in patients with congenital heart disease and duct-dependent pulmonary flow is a safe technique with a success rate in our series of 100% in all situations after 2014. The procedure is well tolerated by patients, has short intubation times and ICU stays, and allows good branch development and the option to adjust flow to the initial situation with subsequently dilatation if the surgical plan or hemodynamic situation changes. Our recent mortality and morbidity (0% and 8%) compare well with the reported rates from the international registry of surgical shunts (7.2% and 13.1%)[5] and are in line with publications from other authors.[6] Currently, the technical improvements and standardization of the procedure give reproducible results, allowing ductal stenting to be considered as a first-line option for all patients who have congenital heart disease with duct-dependent pulmonary flow and need for additional pulmonary flow.

Laura Marfil-Godoy,[a] Gerard Martí-Aguasca,[a] Queralt Ferrer-Menduiña,[b] Gemma Giralt-García,[b] and Pedro Betrián-Blasco[a,*]

[a]*Unidad de Hemodinámica Pediátrica, Hospital Universitario Materno-Infantil Vall d'Hebron, Universidad Autónoma, Barcelona, Spain*
[b]*Unidad de Cardiología Fetal y Neonatal, Hospital Universitario Materno-Infantil Vall d'Hebron, Universidad Autónoma, Barcelona, Spain*

[*] Corresponding author:
E-mail address: pedrobetrian@yahoo.es (P. Betrián-Blasco).

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Echocardiographic findings in critical patients with COVID-19

Hallazgos ecocardiográficos en pacientes críticos por COVID-19

To the Editor,

In the first cases of coronavirus disease 2019 (COVID-19) described in China, acute myocardial injury was identified as being associated with a worse prognosis.[1] The etiology of this myocardial injury is not entirely clear, but it could be related to the processes of microvascular damage, myocarditis, hypoxemia, cytokine-mediated injury, or even stress cardiomyopathy.[2,3] However, diagnosis of myocardial injury has mostly been based on raised biomarkers in the absence of cardiac imaging. In this study, we describe the echocardiographic findings of 37 consecutive patients admitted to the intensive care unit (ICU) with acute respiratory distress syndrome secondary to COVID-19.

This was a prospective, single-center study of consecutive patients with COVID-19, confirmed on polymerase chain reaction testing, who were admitted to the ICU due to acute respiratory distress syndrome. The patients were divided into 2 groups based on whether their left ventricular ejection fraction (LVEF) was greater or less than 50%. In patients with reduced function, the severity of the reduction was estimated qualitatively as mild (40%-49%) moderate (30%-39%) or severe (< 30%). Values of high-sensitivity troponin T, N-terminal pro-brain natriuretic peptide, C-reactive protein, and ferritin were considered inflammatory biomarkers, and their peak levels were recorded and compared between the 2 groups. Echocardiography was performed with a handheld ultrasound (Vscan, General Electrics), with visual assessment of right and left ventricular function on 2-, 3-, and 4-chamber views, to minimize patient exposure. The presence of regional wall motion abnormalities, whether they had coronary or noncoronary distribution, and the presence of pericardial effusion were also assessed. Continuous variables are described as median [interquartile range] or mean ± standard deviation and were compared using the Mann-Whitney U test or Student t test depending on the normality of the distribution of the data. Categorical variables are described as percentage and were compared using the Fisher or chi-square test. Data collection was approved by the ethics committee of our institution.

Table 1
Baseline characteristics of the 37 patients with COVID-19 admitted to the ICU due to acute respiratory distress syndrome

Variable	Total (n = 37)	LVEF < 50% (n = 6)	LVEF ≥ 50% (n = 31)	*P*
Age, y	67.6 [59.6-70.5]	69.6 [68.3-70.8]	65.8 [57.7-70.5]	.117
Male	34 (91.9)	5 (83.3)	29 (93.6)	.421
Ischemic heart disease	2 (5.4)	0	2 (6.5)	.999
Previous systolic dysfunction	0	0	0	.999
Chronic kidney disease	1 (2.7)	0	1 (3.2)	0.999
Chronic lung disease	8 (21.6)	2 (33.3)	6 (19.4)	0.591
ACE-I	17 (45.9)	3 (50)	14 (45.2)	0.999
PaO_2/FIO_2	107.5 [78-125]	99 [85-109]	110 [78-133]	.4225
Biomarkers				
High-sensitivity troponin T (ng/mL)	31.1 [21-103]	210 [28-326]	30.9 [20-81]	.0698
NT-proBNP (pg/mL)	1.367 [766-4.868]	3.023.5 [1.174-7.714]	1.367 [742-4.868]	.5365
CRP (mg/L)	275.5 [187-370]	263 [186-435]	277 [188-361]	.9831
Ferritin (ng/mL)	1.505.5 [663-3.055.6]	1.676.5 [681-3.223]	1.505.5 [583-2.888]	.8318
Echocardiographic findings				
LVEF (%)	55.9 ± 8.9	40.8 ± 3.8	58.9 ± 6.2	.0001
Regional wall motion abnormalities	3 (8.1)	3 (50)	0	.003
Depressed RV systolic function	3 (8.1)	2 (33.3)	1 (3.2)	.015
RV dilation	3 (8.1)	1 (16.7)	2 (6.5)	.425
Pericardial effusion	4 (10.8)	2 (33.3)	2 (6.45)	.055

ACE-I, angiotensin-converting enzyme inhibitors; CRP, C-reactive protein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide; PaO_2/FIO_2, ratio of arterial oxygen partial pressure to fractional inspired oxygen; RV, right ventricle.
Values are expressed as No. (%), mean ± SD or median [interquartile range].