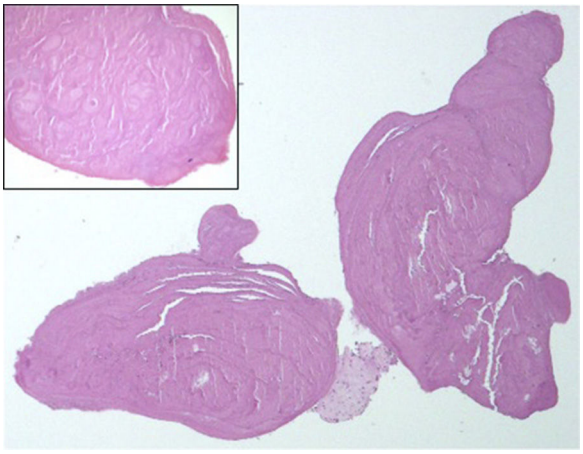

Figure 2. Microscopic image of 2 of the vegetations composed of acellular eosinophilic material (hematoxylin-eosin 40 X). The insert shows the characteristic fibrin strands with degenerated interwoven platelets and 1 trapped lymphocyte (hematoxylin-eosin, 400 X).

less than 1 mm, adhering to the atrial side of both leaflets of the mitral valve, were identified (Figure 1D). They were carefully resected with preservation of the valve. Culture of the masses was negative for microorganisms. Histologic examination of the specimen showed features compatible with nonbacterial thrombotic endocarditis (NBTE) (Figure 2).

The patient had a favorable, uncomplicated postsurgical course and was discharged 7 days after surgery without neurological sequelae and under oral anticoagulant therapy. At the 12 month follow-up, she is still asymptomatic with oral anticoagulation and with no mitral masses identified in subsequent transoesophageal echocardiography.

Nonbacterial thrombotic endocarditis is a disease characterized by endocardial vegetations primarily composed of a sterile matrix of platelets and fibrin that deposits on cardiac valves.[1] Nonbacterial thrombotic endocarditis is most commonly associated with neoplastic diseases, long-term illnesses, sepsis, and burns. While NBTE can be asymptomatic, up to 42% of patients can present with embolic events, especially affecting the cerebral arterial tree.[2] Other conditions, such as hyperhomocysteinemia due to mutations of the *MTHFR* gene, are a common cause of inherited prothrombotic states that can be associated with NBTE.[3]

Clinical diagnosis of NBTE is challenging and requires a high level of suspicion. Anticoagulation due to alterations in patients' coagulation status has been recommended by some authors.[4]

In our case, after excluding other etiologies, the increased clotting tendency of the patient during pregnancy and puerperium (also related to the appearance of preeclampsia) was thought to be the cause of NBTE. There are few cases in the literature that support this association and in those cases the diagnosis was conducted postmortem.[5] The detection of the *MTHFR* gene mutation is also important because it could be another factor increasing the risk of hypercoagulability in our patient, even when the values of homocysteinemia were normal (maybe due to diet).

Echocardiographic techniques such as transesophageal echocardiography have higher sensitivity (90%) for the diagnosis of intracardiac vegetations but a distinction between NBTE and infective endocarditis is not feasible. Furthermore, no clear morphological features of the NBTE vegetations assessed by imaging can predict embolic risk.[6]

To our knowledge, there are no guidelines for surgical intervention in patients with NBTE, but surgery has been recommended in patients presenting with acute cardiac failure and recurrent thromboembolism despite anticoagulation.[6]

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REFERENCES

1. Llenas J, Guerra J, Montes S, et al. Nonbacterial Thrombotic Endocarditis: Clinico-pathologic Study of a Necropsy Series. *Rev Esp Cardiol.* 2007;60:493–500.
2. López JA, Ross RS, Fishbein MC, et al. Nonbacterial thrombotic endocarditis: a review. *Am Heart J.* 1987;113:773–784.
3. Eldibany MM, Caprini JA. Hyperhomocysteinemia and thrombosis: an overview. *Arch Pathol Lab Med.* 2007;131:872–884.
4. Bessis D, Sotto A, Viard JP, et al. Trousseau's syndrome with nonbacterial thrombotic endocarditis: pathogenic role of antiphospholipid syndrome. *Am J Med.* 1995;98:511–513.
5. George J, Lamb J, Harriman D. Cerebral embolism due to non-bacterial thrombotic endocarditis following pregnancy. *J Neurol Neurosurg Psychiatry.* 1984;47:79–80.
6. Asopa S, Patel A, Khan O, et al. Non-bacterial thrombotic endocarditis. *Eur J Cardiothorac Surg.* 2007;32:696–701.

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Transcatheter Device Closure of Aortopulmonary Window. Is There a Need for an Alternative Strategy to Surgery?

Cierre transcatéter de la ventana aortopulmonar. ¿Vale la pena un método de cierre alternativo a la cirugía?

To the Editor,

The aortopulmonary window (APW) is a defect resulting from incomplete separation of the walls of the main pulmonary artery and aorta at the conotruncal septum during early embryogenesis. It is an uncommon condition, accounting for 0.2% to 0.6% of all congenital heart diseases.[1]

APW has traditionally been treated by surgery. The first treatment by transcatheter closure was carried out in the 1990s using a double umbrella occluder system.[2] Since that time, reports of transcatheter APW treatment with these devices have emerged only sporadically.[3–5] An excessively wide diameter at the pulmonary or aortic ends makes transcatheter closure technically demanding, and therefore, this alternative option is little used.

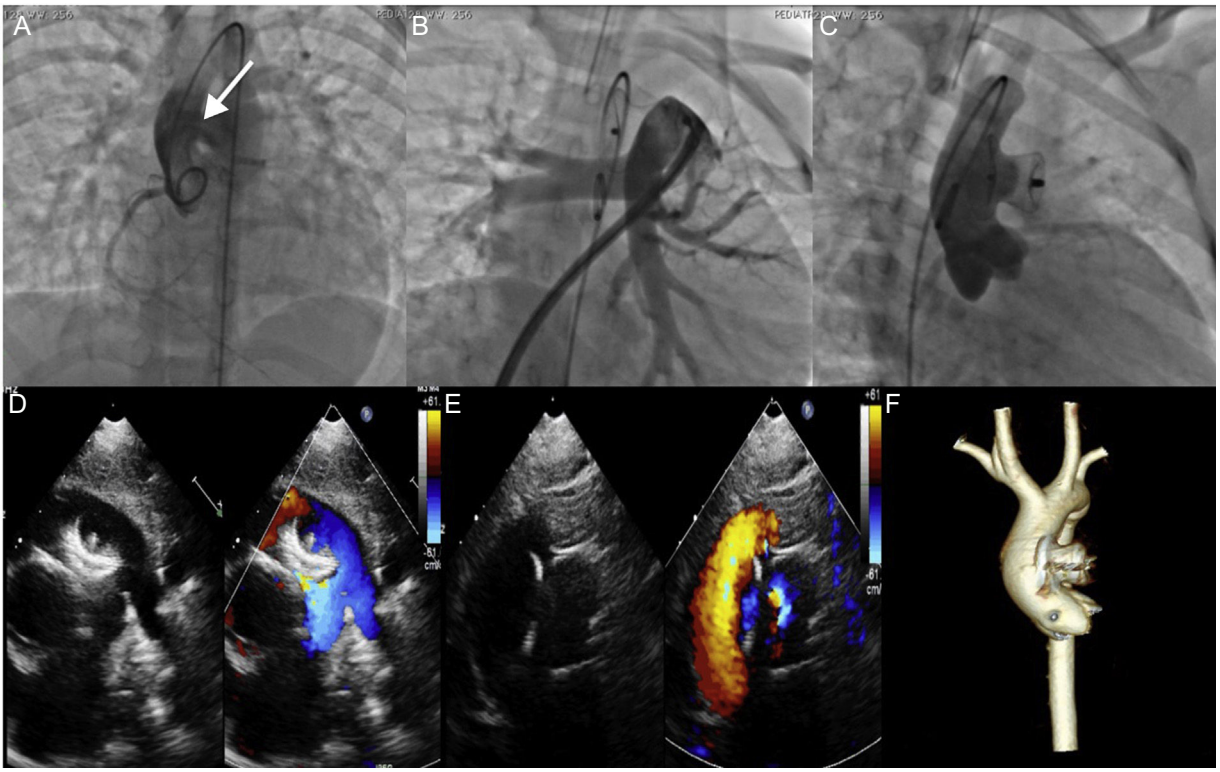

Figure. A. Aortography at the aortic root shows a large aortopulmonary window (arrow). B. Angiography of the pulmonary artery. C. Properly positioned occluder device, with no residual shunt. D and E. Echocardiogram shows slight protrusion of the device toward the main pulmonary artery and the retention disc in the ascending aorta, which does not generate a significant obstruction gradient. F. Computed tomography control shows a properly positioned occluder device.

We present our experience with transcatheter APW closure from July 2004 to December 2017. Six patients were referred for closure with an occluder device (Figure 1). Mean age was 4.3 years (1 month-15 years) and weight was 30 (3–45) kg. The median pulmonary artery systolic pressure was 50 (25–80) mmHg, with a pulmonary vascular resistance of 3.7 (2.1-4.5) WU and a left-to-right shunt (Qp:Qs) of 3:1 (1.5-4.56), reflecting unrestricted pulmonary flow through the defect. The defects considered for transcatheter closure were type 1 according to the classification of Mori et al.[6] The median diameter of the aortic orifice was 6.7 (2.31-14.9) mm. In all patients, the pulmonary arterial pressure decreased following closure with the device. Duration of hospital stay after the procedure was 3 (1–6) days (Table). During the first 24 hours, 2 patients experienced device embolization. Both patients underwent a new transcatheter procedure in which the device was retrieved and removed, and a larger one implanted to occlude the defect. Another child, treated during the first month of life, required placement of a second device 1 year later because of a considerable residual shunt. Median clinical follow-up was 6 (2–11) years. The estimated pulmonary artery systolic pressure on echocardiography was 28 (16-43) mmHg. There were no gradients indicative of obstruction at the aorta or pulmonary branches.

During the same period, APW was surgically treated in 7 pediatric patients: median age was 6.8 (1-25) years and weight 17.8 (7-54) kg. The defect was classified as type I in 2 patients, type II in 2 others, and type III in the remaining patients.[6] Transcatheter closure was not considered in those with a type I morphology because of the large size of the defects (> 20 mm). A bovine pericardial patch was used for closure in all patients. The mean duration of hospitalization following the procedure was 7 days.

Table
Transcatheter Closure of Aortopulmonary Window in Instituto Nacional de Cardiología Ignacio Chávez

Age,[a] months	Weight,[a] kg	Size of defect, mm	PASP, mmHg	Device, mm	Complications	Follow-up
9	6.1	2.3	25	ADO 5/4	None	Asymptomatic
180	45.0	4.1	30	ADO 10/8	None	Asymptomatic
19	8.4	4.4	30	ADO 8/6	None	Asymptomatic
0.7	3.1	5.8	80	ADO II 6/4	None	Residual shunt (closure with ADO II 4 × 4 mm)
55	15.0	8.4	80	ADO 14/12	Embolization[b]	Asymptomatic
37	14.2	14.9	60	Cera 16/18	Embolization[c]	Asymptomatic

ADO, Amplatzer persistent ductus arteriosus occluder device; ASO, Amplatzer atrial septal defect occluder device; PASP, pulmonary artery systolic pressure.
[a] Age and weight at the time of the procedure
[b] Definite closure with 6/18 mm Cera occluder
[c] Definite closure with 16 mm ASO occluder

With the advances in the development of devices for closure of various congenital heart defects, such as persistent ductus arteriosus and ventricular or atrial septal defects, it is surprising that transcatheter APW closure still plays a very small role. This approach has several attractive advantages: extracorporeal circulation is avoided during the procedure and the postoperative hospital stay is shorter. However, the excellent outcome achieved with surgery and the technical complexity of transcatheter closure are the 2 main reasons why most centers prefer surgical treatment. Regardless of these considerations, the candidates for transcatheter closure should have relatively small defects located at a point equidistant between the bifurcation of the pulmonary artery and the semilunar valves, and far from the left coronary artery ostium and the aortic valve; that is, type I defects according to the classification of Mori et al.[6] It is important to appropriately characterize the defect through the use of several angiographic views or even measurement balloons to precisely determine the dimensions of the window.[3–5] It is also a challenge to choose the appropriate device, and to date, there is no consensus on the choice of an optimal device for APW closure. Ductus arteriosus occluders tend to protrude toward the main pulmonary artery and carry a risk of obstructing the vessel, whereas atrial septal defect occluders are bulky for this purpose and may injure or obstruct the semilunar valves or left coronary ostium.[5] Trehan et al.[3] considered that the perimembranous ventricular septal defect occluder device may be the most appropriate. It has a relatively flat profile (waist diameter, 1.5 mm), which could cause less obstruction. Furthermore, because the discs are asymmetrical, they can be used to close an APW with a relative deficiency of one of the borders.

In selected patients, percutaneous APW closure can be considered a viable, effective procedure. Nonetheless, the potential risks should be considered, such as device embolization and residual shunts. Because of its excellent results, surgical management remains the method of choice for treating these defects.

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REFERENCES

1. Kutsche LM, Van Mierop LHS. Anatomy and pathogenesis of aorticopulmonary septal defect. *AmJ Cardiol.* 1987;59:443–447.
2. Stamato T, Benson L, Smallhorn J, Freedom R. Transcatheter closure of an aortopulmonary window with a modified double umbrella occluder system. *Cathet Cardiovasc Diagn.* 1995;35:165–167.
3. Trehan V, Nigam A, Tyagi S. Percutaneous closure of nonrestrictive aortopulmonary window in three infants. *Cathet Cardiovasc Interven.* 2008;71:405–441.
4. Richens T, Wilson N. Amplatzer device closure of a residual aortopulmonary window. *Cathet Cardiovasc Interven.* 2000;50:431–433.
5. Naik G, Chandra S, Shenoy A, et al. Transcatheter closure of aortopulmonary window using Amplatzer device. *Cathet Cardiovasc Interven.* 2003;59:402–405.
6. Mori K, Ando M, Takao A, Ishikawa S, Imai Y. Distal type of aortopulmonary window: report of 4 cases. *Br Heart J.* 1978;40:681–689.

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Tako-tsubo Cardiomyopathy Complicated With Cardiac Tamponade and Cardiogenic Shock

Síndrome de tako-tsubo *complicado con taponamiento cardiaco y* shock *cardiogénico*

To the Editor,

Although clinical progress is favorable in most patients with tako-tsubo syndrome with resolution of ventricular dysfunction, complications leading to shock sometimes occur.[1]

We present the case of an 83-year-old woman, with no medical history of interest, who consulted for a 12-hour history of oppressive central chest pain with no clear triggering factor. An electrocardiogram showed ST-segment elevation in V_3-V_6, DII, DIII, and aVF (Figure 1A). An ST-segment elevation acute coronary syndrome was suspected, and a loading dose of dual antiplatelet therapy was administered (300 mg aspirin and 600 mg clopidogrel). Emergent coronary angiography showed no significant lesions in the epicardial arteries (Figure 1C). The study was completed with ventriculography, which showed apical akinesia (Figure 1D), and transthoracic echocardiography, which depicted akinesia of the left ventricular middle and apical segments (Figure 1E). Left ventricular ejection fraction was 35%, with hypercontraction of the basal segments, and the mitral valve showed a systolic anterior movement (SAM), without significant flow acceleration in the left ventricular outflow tract (LVOT), and a mild (< 10 mm), circumferential pericardial effusion (PE).

The patient was hemodynamically stable, showed no evidence of heart failure, and had a minimal troponin I elevation (peak 0.823 ng/mL); nonetheless, at 24 hours she began to show hemodynamic deterioration. Transthoracic echocardiography detected an increase in the dynamic LVOT obstruction[2] and progression of the PE (18 mm in the right ventricular free wall) (Figure 2A), with no echocardiographic signs of cardiac tamponade. Based on these findings, fluid therapy was increased and phenylephrine and esmolol infusion was started. These measures led to a decrease in the dynamic LVOT obstruction and improvement of the patient's hemodynamic status. In light of the PE increase, cardiac computed tomography was performed, but there was no evidence of cardiac rupture. However, an aneurysmal dilatation of the left ventricular apical region was detected, with preserved myocardial thickness and a thrombus adhering to the inferoapical segment (Figure 2B).

In the following hours, the patient's clinical course was unfavorable, with the development of severe cardiogenic shock (hypotension, anuria, elevated lactate), worsening of the dynamic LVOT obstruction (Figure 2C), progression of PE (21 mm), and evidence of tamponade (partial collapse of the right chambers in diastole and a change in the transtricuspid flow > 50%).