creatine kinase-MB fraction was 79 μg/L. The remaining blood test results were normal. An echocardiogram showed normal anatomy of the coronary arteries, a moderately dilated left ventricle (end-diastolic diameter: 61 mm [Z-score=2.48]), an ejection fraction of 45% and moderate mitral failure. An MRI scan showed an ejection fraction of 50% with a normal end-diastolic volume. A delayed enhancement study showed a pattern of patchy subepicardial enhancement in the lateral wall. An area of increased signal intensity was visible in the T2-weighted MRI image, which was suggestive of edema (Fig. 2).

The patient’s course was favorable; his systolic function returned to normal with a decrease in markers of damage. The suspected diagnosis was acute myocarditis. At admission, the results of polymerase chain reaction of blood and nasopharyngeal aspirate were negative for viruses; therefore the causal agent was not identified.

The third patient was a 10-year-old girl who presented to the emergency department after experiencing 4 episodes of oppressive chest pain, radiating to her arm. Each episode lasted approximately 1 h. An echocardiogram showed moderate ST-elevation of 1 mm in II, III and aVF, with troponin T at 9.12 μg/L and creatine kinase-MB fraction at 272 μg/L. An echocardiogram showed normal coronary artery anatomy, a hypertrophic left ventricle (septum 10 mm [Z-score=2.77]; posterior wall 9 mm [Z-score=2.13]), which was not dilated, with an ejection fraction of 65%. T2-weighted MRI scan showed subepicardial areas of increased signal intensity in the free wall of the left ventricle. The delayed enhancement sequences showed generalized, subepicardial enhancement of the left ventricle, compatible with acute myocarditis. Polymerase chain reaction testing of blood and nasopharyngeal aspirate was negative for viruses. The patient’s course was favorable.

Precordial pain is a presentation of acute myocarditis and, although uncommon in children, should be included in the differential diagnosis.

The usefulness of MRI in these patients has been previously reported. The clinical course is usually favorable and the most commonly described causative agent is Parvovirus B19. In the cases reported here, the diagnostic utility of MRI should be emphasised as it allowed catheterization to be avoided in our patients. It should be performed as an emergency procedure and, if inconclusive, a coronary angiography should be conducted to rule out coronary disease.

Ferran Gran,a,* Amparo Castellote,b Laia Vega,a Dimpna Albert,a Queralt Ferrer,a and Joan Sanchez-De-Toledo c

aUnidad de Cardiología Pediátrica, Hospital Universitario de la Vall d’Hebron, Universidad Autónoma de Barcelona, Barcelona, Spain
bServicio de Radiología Pediátrica, Hospital Universitario de la Vall d’Hebron, Universidad Autónoma de Barcelona, Barcelona, Spain
cUnidad de Cuidados Intensivos Pediátricos, Hospital Universitario de la Vall d’Hebron, Universidad Autónoma de Barcelona, Barcelona, Spain

*Corresponding author:
E-mail address: fgran@vhebron.net (F. Gran).

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REFERENCES


Fenestration Closure After Fontan Surgery. Contributions of Percutaneous Interventionism

Cierra de fenestración tras la cirugía de Fontan. Aportaciones del intervencionismo percutáneo

To the Editor,

The Fontan procedure is the final step in surgery for patients with a single ventricle. The hemodynamic changes that occur after the procedure can have a negative impact on the immediate outcome due to a sudden increase in pulmonary artery pressure. Fenestration of the Fontan circuit during surgery is therefore a common procedure in high-risk patients, although systemic saturation may decrease as a result. The development of percutaneous implantation devices has enabled fenestration closure without the need for further surgery when hemodynamic conditions allow.2,3

Here, we analyze our experience of percutaneous closure of fenestrations in the extracardiac circuit after the Fontan procedure, taking into account the properties of the new occlusion devices available. In addition, we study the changes in pulmonary artery
Characteristics of the 13 Patients

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AoP, aortic pressure; AS, atrial shunt; D-TGA, double transposition of the great arteries; DVO, double right ventricular outflow; IC, functional class; HV, hypoplastic left ventricle; PA, pulmonary artery pressure; PS, patent foramen ovale; TA, tricuspid atresia; VS, ventricular septal defect.

The mean (standard deviation) duration of follow-up was 45 (41) months. No major complications were recorded during the procedures. Prior therapeutic catheterization had been performed in 8 of the 13 patients to close systemic-pulmonary collaterals. Of the 13 patients analyzed, 11 had systemic left ventricle and 2 systemic right ventricle (Table). None of the patients died during follow-up or were readmitted to hospital for cardiac causes.

In all cases, Amplatzer occluders were used. The device type was chosen in accordance with the postsurgical anatomy, which was assessed by computed tomography prior to the procedure. Multislice computed tomography was used to locate the site of fenestration and select the angiographic view that would best visualize the defect, sliced perpendicular to the largest diameter of the defect. Once catheterization had been performed, contrast was injected and the maximum diameter of fenestration was estimated, in accordance with the calibration marked with the catheter (Figure). Computed tomography and transesophageal echocardiography complemented these measures and contributed to better selection of the device size. In addition to the maximum diameter of the defect, the distance between the atrial chamber and the internal edge of the conduit was taken into account. The type of Amplatzer occluder was selected in accordance with the anatomical configuration. Devices for patent foramen ovale were used for narrower fenestration while those for ventricular shunts and patent ductus arteriosus were chosen for the wider ones (Table).

In 10 cases, the device for patent foramen ovale was used (18 mm in 9 patients and 25 mm in 1 patient). In 2 patients, a muscular ventricular shunt device was used, while in the remaining patient, a ductus closure device was chosen. Oxygen saturation increased significantly after closure of the fenestration (89% [3.6] vs 96% [2.0]; $P<.01$) without any evidence of a significant increase in pulmonary artery pressure (17 [3.6] mmHg vs 17.2 [3.9] mmHg).

Patients with congenital heart disease in the form of single ventricle circulation will need to undergo surgery several times during their lives. The development of devices placed by percutaneous procedures can help avoid surgical procedures in some of these patients. Closure of the fenestration is necessary due to the long-term harmful effects of chronic hypoxemia.

The experience in our hospital suggests that a multidisciplinary approach in these patients is essential when designing therapeutic strategies and establishing the timing of the interventions. The development of percutaneous implantation devices has allowed greater flexibility in surgery, which can be adapted to the...
hemodynamic conditions of each patient. Closure of the Fontan fenestration by catheterization is a safe and effective technique in these patients.

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Marta Santisteban,*a Manuel Pan,a Miguel Romero,a Jaime Casares,b Elena Gómez,c and José Suárez de Lezoa

aServicio de Cardiología, Hospital Universitario Reina Sofía, Córdoba, Spain
bServicio de Cirugía Cardiovascular, Hospital Universitario Reina Sofía, Córdoba, Spain
cServicio de Pediatría, Hospital Universitario Reina Sofía, Córdoba, Spain

*Corresponding author.
E-mail address: marta_santisteban@hotmail.com (M. Santisteban).
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REFERENCES


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