We report the case of a 20-year-old woman who had percutaneous closure of a patent ductus arteriosus (PDA) with a 17-mm Rashkind occluder when she was 4 years of age. A small residual shunt was noted on color-flow Doppler echocardiography immediately after the procedure and at 6-month follow-up. Because of the lack of hemodynamic complications, no additional intervention was performed.

Sixteen years later, she presented with recurrent bilateral bronchopneumonia that required antibiotic treatment. Chest radiography during the third episode showed a right bronchopneumonia (Figure 1). At that time, new treatment with amoxicillin and clavulanic acid was given for 10 days, but fever persisted despite this course of antibiotics.

Results of the physical examination showed blood pressure of 110/70 mm Hg, pulse of 92 bpm, temperature of 38.5°C, and pale appearance. There were no other examination findings except a continuous murmur below the left clavicle.

Laboratory workup revealed C-reactive protein 152 mg/L, leukocytes 17 300/mm³, hemoglobin 12.3 g/dL, and hematocrit 35%, which suggested persistent infection. Six hemocultures were positive for *Streptococcus acidominimus*.

A hypothesis of infection at the occluder site was confirmed by transthoracic and transesophageal echocardiograms, which showed a 5×8-mm vegetation on the left pulmonary artery with attachment to the Rashkind occluder (Figure 2; online-only Data Supplement Movie I) and another vegetation on the main pulmonary artery (online-only Data Supplement Movie II) along the persistent left-to-right shunt (online-only Data Supplement Movie III). Furthermore, a chest computed tomography...
scan showed septic emboli in both lung fields (Figure 3). An incidental finding was made of a filling defect in the proximal left pulmonary artery, closely related to the occluder device (Figure 4).

The origin of this infection was not found. Descaling had been performed 4 months earlier with antibiotic prophylaxis with 2 g of amoxicillin. Results of a dental checkup performed during hospitalization were normal.

Intravenous antibiotic treatment consisted of amoxicillin and gentamycin for 2 weeks, then amoxicillin alone for 2 more weeks. After 1 week of treatment, C-reactive protein decreased to normal levels. A follow-up transesophageal echocardiogram was performed, which showed no regression of either lesion. Surgical removal of the vegetation and concomitant ligation of the PDA were undertaken. Subsequent culture of the Rashkind device yielded no bacterial growth.

It is widely agreed that PDAs with evidence of left-sided heart volume overload should be closed, but less evidence exists concerning the management of nonsignificant residual shunts after percutaneous occluder implantation. To the best of our knowledge, this is the first case of late endocarditis reported after percutaneous PDA occlusion with a residual shunt. The pathophysiology is proposed to be the same as that of native endocarditis, with endothelial damage resulting from mechanical lesions provoked by turbulent blood flow across the PDA, but the risk is clearly increased by the exogenous prosthesis. Even though native PDA endocarditis is well known, there are limited data on device-related endocarditis. Previous animal studies revealed no increased risk of endocarditis between device-implanted subjects and control subjects after injection with bacteria-contaminated serum.

The prevalence of a residual shunt after PDA percutaneous occlusion has been reported in 14% of patients after 6 months. This case reports the first infective endocarditis on a PDA occluder with residual shunt and highlights the need for long-term follow-up in case of residual shunt. Even though the risk of endocarditis requires further evaluation, implantation of a second device is a satisfactory means of abolishing persistent residual flow and should be considered if flow persists beyond 6 months.
Disclosures

None.

References

Residual Shunt After Ductus Arteriosus Occluder Implantation Complicated by Late Endocarditis
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