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Brief Report

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Infective endocarditis developing early after percutaneous closure of a patent ductus arteriosus in a child using the Amplatzer Duct Occluder II

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Abstract

A 10-month-old girl underwent patent ductus arteriosus closure with an Amplatzer Duct Occluder II. After 1 week, she was admitted to our emergency room with tachypnoea, fatigue, and fever. Consecutive blood cultures yielded vancomycin-sensitive *Staphylococcus aureus*. The patient was already receiving vancomycin, but the fever did not respond to this treatment. The device was successfully removed via left lateral thoracotomy.

Persistence ductus arteriosus can trigger congestive heart failure, pulmonary hypertension, and bacterial endarteritis. In the pre-antibiotic era, patients with patent ductus arteriosus tended to die from infection rather than heart failure.[1] Given the development of diagnostic tools and treatments, the risk of endarteritis has been considerably reduced. Transcatheter patent ductus arteriosus closure using coils or other devices has become the first-choice treatment in most cardiac centres worldwide. Infective endocarditis developing after transcatheter or surgical patent ductus arteriosus closure is now very rare. Some authors have reported late endocarditis caused by residual leakage from percutaneously closed defects. However, the causes and frequency of endocarditis associated with patent ductus arteriosus closure remain unknown. We present a case of infective endocarditis that developed early after transcatheter closure of a patent ductus arteriosus using an Amplatzer Duct Occluder II device (St Jude Medical, Saint Paul, MN, USA).

Case

A 10-month-old girl underwent patent ductus arteriosus closure using the Amplatzer Duct Occluder II device. The device released in the appropriate position and no residual shunt was detected on the final angiogram. Transthoracic echocardiography was used to check the device 24 hours after the procedure and to ensure that the patent ductus arteriosus was completely closed. The patient was discharged 1 day after the procedure. After 1 week, she was admitted to our emergency department with tachypnoea, fatigue, and a fever (peak temperature of 41°C). The acute phase marker levels were significantly elevated (C-reactive protein, 19 mg/dL; erythrocyte sedimentation rate, 90 per hour; leucocytosis, 20,300 per mm^3). Three blood cultures were performed and the patient was hospitalised. Serial blood cultures were taken over the following few days. Empirical antibiotherapy (vancomycin and ceftriaxone) was started. The initial echocardiogram revealed that the patent ductus arteriosus device was in an appropriate position; it appeared as a bright object and the details of which were clearly visible (Fig 1). The fever persisted to day 4 after hospitalisation and consecutive blood cultures yielded *Staphylococcus aureus* in all seven blood cultures started during the first 3 days (three on day 1, two on day 2, and two on day 3). The modified Duke criteria[2] indicated "possible endocarditis"; our patient met one major criterion (microorganisms consistent with endocarditis in persistently positive blood cultures) and two minor criteria (a predisposing heart condition and fever). Antimicrobial susceptibility testing revealed methicillin-resistant *S. aureus* that was susceptible to vancomycin. Therefore, vancomycin was continued and ceftriaxone was stopped. Unfortunately, the fever did not abate and the acute phase reactant levels increased during the following few days. The general condition of the patient began to deteriorate, so she was transferred to the cardiovascular suite for surgical removal of the device. The operation was performed on day 7 of vancomycin antibiotherapy. The device was successfully removed (Fig 2), and the patent ductus arteriosus was ligated via left lateral thoracotomy without a cardiopulmonary bypass. The fever abated and her general condition improved immediately after the operation. Vancomycin antibiotherapy was continued for 4 weeks. The fever did not return and the clinical course of the

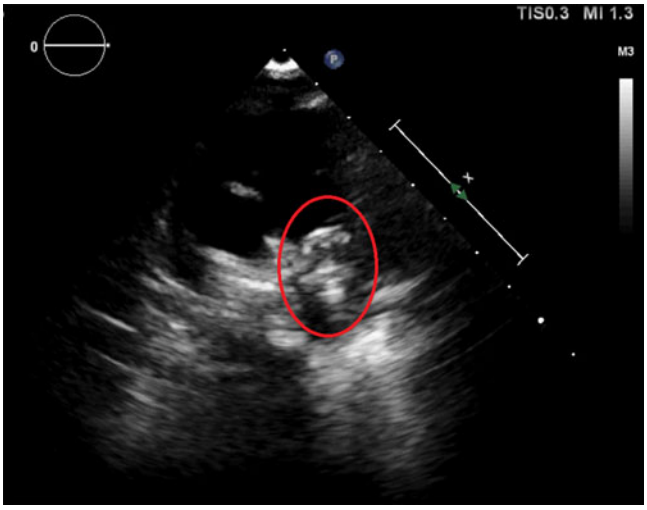

Figure 1. Parasternal short-axis echocardiographic view demonstrating the Amplatzer Duct Occluder II device is more hyperechogenic than normal (in red circle).

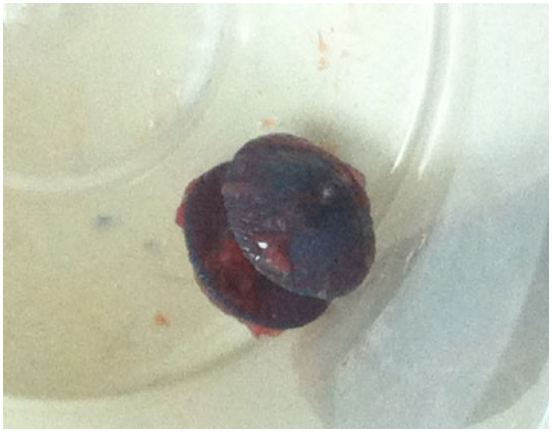

Figure 2. Amplatzer Duct Occluder II device that surgically removed.

patient was stable; the final echocardiogram revealed no residual shunt and she was discharged 4 weeks after the cardiac procedure.

Discussion

Infective endocarditis developing after device-mediated patent ductus arteriosus closure is rare. However, an early report on the natural course of patent ductus arteriosus emphasised that infective endocarditis/endarteritis is an important cause of mortality.[3] Thus, many centres prefer to close even small patent ductus arteriosus. Currently, percutaneous device-mediated closure is the first-line treatment. Although the operation is minimally invasive, complications including infective endocarditis, although rare, can develop. The infection may be acquired during device placement, and infection of the soft tissue surrounding the device can occur after placement.[4] Our most remarkable finding was that the device was clearly visible on echocardiogram when the patient visited the emergency room, which was probably attributable to inflammatory oedema in the surrounding tissue enhancing the ultrasound image. This information should be included in the text of the Duke criteria for diagnosing infective endocarditis; currently, endocarditis is often missed when diagnosis is carried out according to these criteria. In our case, although the clinical and laboratory findings indicated endocarditis, the Duke criteria indicated that this was only a possibility. Some reports have suggested that the Duke criteria are less sensitive in cases of prosthetic valve endocarditis or cardiac device infections.[5,6] In our opinion, the Duke criteria should be revised to cover patients with indwelling prosthetic materials.

Three case reports have described infective endocarditis developing during follow-up in patients with residual shunts after patent ductus arteriosus device implantation.[1,3,4] In contrast, our case exhibited no residual shunt on echocardiography. Device-related infections are usually resistant to antibiotherapy, unlike native endocarditis.[4,7] The therapeutic options for device-related endocarditis have received scant attention[8] and most patients heal after surgery. Nevertheless, the device may serve as the source of infection, so surgical removal is essential. Peng et al[9] suggested that inappropriate device implantation, associated with turbulent blood flow via a residual shunt, may injure the vascular endothelium. However, infective endocarditis does not develop in most patients with residual shunts.

We found no literature pertaining to the prevention of infective endocarditis associated with transcatheter device-mediated closure. However, the general guidelines for asepsis and antisepsis must be rigorously followed during interventional procedures. A 2015 guideline on intracardiac electronic device placement recommended that the air exchange rate in cardiac catheterisation rooms should be equal to that recommended for operating theatres, that is, 25 changes per hour rather than the currently recommended 15 changes per hour.[10]

In conclusion, infective endocarditis is a rare but very serious complication of percutaneous device-mediated closure of heart defects. The risk of endocarditis or endarteritis associated with patent ductus arteriosus closure devices remains unknown. Patients with early or late high fever should be carefully evaluated in terms of infective endocarditis.

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Conflicts of Interest. None.

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