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Case Report

Residual shunt and infective endocarditis after percutaneous device closure for patent ductus arteriosus

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Abstract: Percutaneous device closure has been proven as an effective treatment for some types of congenital heart diseases, such as atrial septal defect, ventricular septal defect, and patent ductus arteriosus (PDA). Despite being considered minimally invasive, some complications still occur after this procedure. A residual shunt and infective endocarditis are among the complications of percutaneous device closure. Unlike native endocarditis, this device-related infection is sometimes refractory to antibiotic therapy, and the neoendothelium covering the device also makes surgical procedures difficult. Herein, we report a new technique for removal of the infective closure device in a patient with PDA. We present the case of a 26-year-old woman with a residual shunt and infective endocarditis after inappropriate implantation of an atrial septal defect closure device in a PDA site. She was successfully treated with a surgical procedure and a new technique for removal of the closure device. This case emphasizes the importance of geometric conformance between the closure device and congenital heart defects. Surgery is recommended for patients with refractory device-related infection. Utilization of a cable and sheath method to fold the device is feasible in some patients.

Keywords: Percutaneous device closure, PDA, infective endocarditis, residual shunt

Introduction

Percutaneous device closure is a minimally invasive treatment for some types of congenital heart diseases, such as atrial septal defect (ASD), ventricular septal defect, and patent ductus arteriosus (PDA) [1]. Despite being minimally invasive, there are some unique risks associated with this method [2].

A residual shunt and infective endocarditis are among the complications of percutaneous device closure for a congenital heart defect. The closure device itself is considered an incremental risk factor for endocarditis [3, 4]. A geometric mismatch between the closure device and the defect may cause a residual shunt, and the resultant turbulent blood flow may also increase the risk of infective endocarditis [5]. Unlike native valve endocarditis, this device-related endocarditis is sometimes refractory to antibiotic therapy, and limited data are available about prevention, therapy, and prognosis [4]. Surgery is recommended in some cases [6]. The existence of neoendothelium covering the closure device imposes further difficulty on any surgical procedure.

Herein, we report the case of a patient with a residual shunt and infective endocarditis after percutaneous device closure for PDA. The patient recovered after open heart surgery.

Case report

A 26-year-old woman had been diagnosed with a cardiac murmur in childhood, but did not undergo treatment because of social reasons. In February 2015, a transthoracic echocardiogram (TTE) confirmed a diagnosis of PDA. She then underwent PDA closure with deployment of a device that was intended for use in ASD (Amplatzer Septal Occluder, AGA Medical Corporation, Minesota, USA). Although the postoperative TTE showed a residual shunt, she declined further intervention because of lack of symptoms.

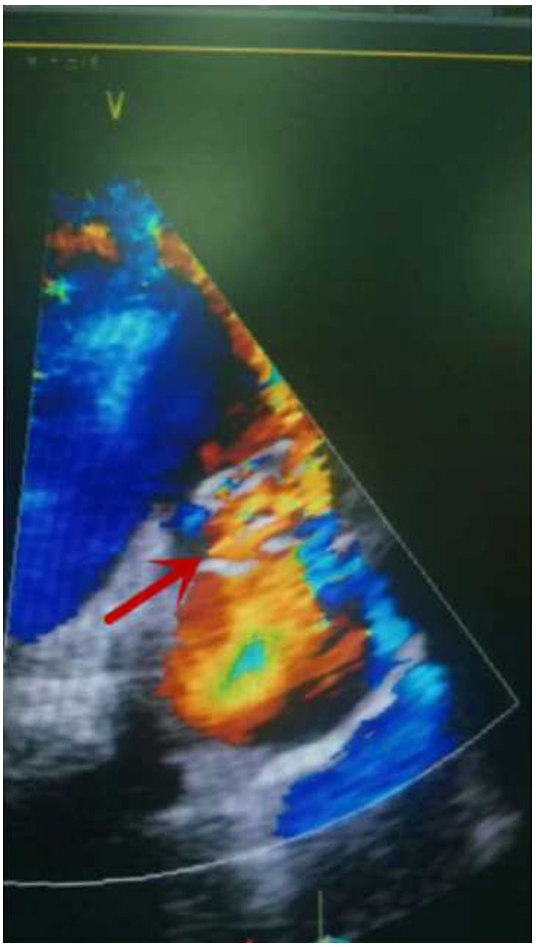

Figure 1. TTE showed a shunt between the aorta and the main pulmonary artery (Red arrow).

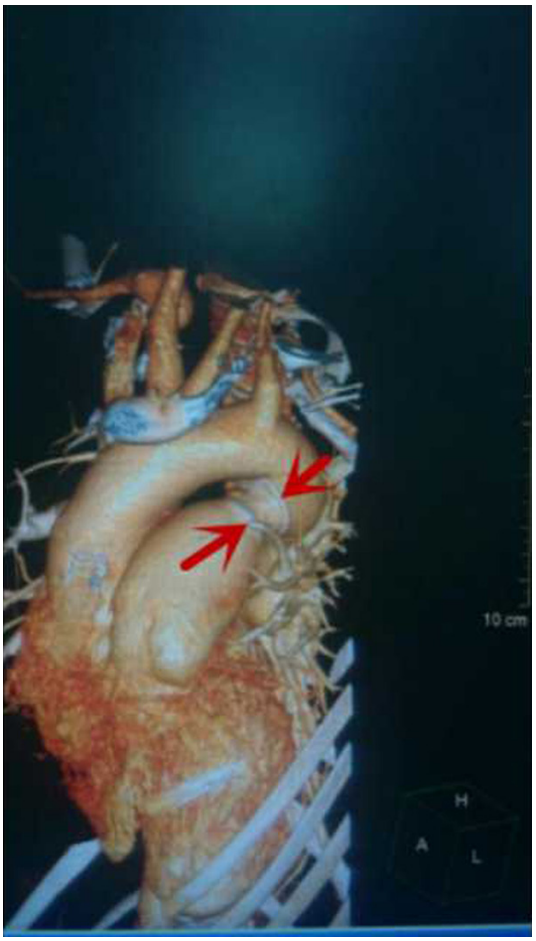

Figure 2. CT angiography showed the profile of the closure device (Red arrow).

Two months later, she developed a fever with a peak temperature of 42.1°C, along with cough, dyspnea, and fatigue. She was admitted to the same hospital where outpatient antibiotic treatment had been initiated for a probable diagnosis of pneumonia. The patient was transferred to our facility because of persistent fever in spite of treatment with intravenous antibiotics.

Two of three consecutive blood cultures yielded vancomycin-sensitive *Staphylococcus aureus*, thus establishing a baseline for antibiotic choice. Blood tests revealed a white blood cell count of 14.30×10^9/L, with neutrophils 72.1%, a C-reactive protein level of 81.3 mg/L, and an erythrocyte sedimentation rate of 65 mm/h, all of which were indicators of an intense inflammatory response. TTE demonstrated a continuous left-to-right shunt between the aorta and main pulmonary artery (**Figure 1**), and moderate aortic valve regurgitation, but no vegetation was found. Computed tomography angiography showed the profile of the closure device between the aorta and the main pulmonary artery (**Figure 2**). Given the history of percutaneous device closure for PDA, implantation of the closure device, prolonged fever, and the positive results of blood culture, a presumptive diagnosis of infective endocarditis was made, although the TTE failed to find any vegetations in the heart or on the closure device.

After 4 weeks of antibiotic therapy, she underwent surgery. The surgery was performed with the patient on cardiopulmonary bypass, with deep hypothermic circulatory arrest, which was established with cannulas in the innominate artery and superior and inferior vena cava. Main pulmonary arteriotomy revealed the closure device to be partially endothelialized on the artery side, without obvious vegetation. We used a cable to compress the closure device

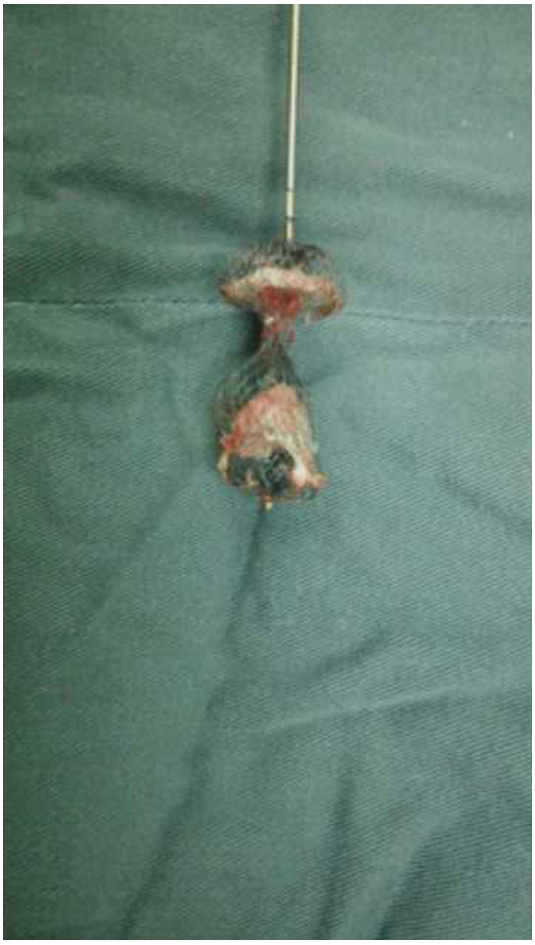

Figure 3. The closure device and the device used to fold it in surgery.

into a special sheath that was designed to release the device during open-chest management of congenital heart defects (**Figure 3**); we then debrided and excised the abutting friable tissue, and closed the PDA with an autologous pericardial patch. We evaluated the aortic valve through the aortotomy, and found mild-moderate prolapse of the non-coronary leaflet. Accordingly, folding and fixing of the non-coronary leaflet to the commissure were performed. An intraoperative transesophageal echocardiogram revealed no residual shunt through the ductus arteriosus and only mild aortic valve regurgitation. Tissue cultures adjacent to the device yielded vancomycin-sensitive *S. aureus*, which remained the baseline for intravenous antibiotic choice. A postoperative TTE revealed no residual shunt through the ductus arteriosus and mild-moderate aortic regurgitation; the blood culture yielded a negative result. The patient was discharged without complications 4 weeks after surgery.

Discussion

Congenital heart disease is commonly seen in newborns [7]. Traditional treatment requires sternotomy and cardiopulmonary bypass, both of which are associated with complications. Percutaneous device closure offers a minimally invasive option, and satisfactory outcomes have been reported [1]. However, associated complications include device embolism, device thrombosis, cardiac erosion, tamponade, and endocarditis [2]. Meanwhile, a residual shunt is observed after implantation of the closure device in some patients. After percutaneous PDA closure, the reported prevalence of a residual shunt is about 14% [8]. Geometric conformance between the closure device and the cardiac defect is essential for a successful procedure. Lack of geometric adaptation may account for the occurrence of a residual shunt in some cases [9, 10]. In this case, a closure device designed for an ASD was used to close a PDA, with an inevitable mismatch between the device and defect. This inappropriate implanted closure device, accompanied by resultant turbulent blood flow, may have caused injury to the vascular endothelium. Furthermore, the closure device itself, as an exogenous prosthesis, is considered an incremental risk factor for endocarditis [3, 4].

Limited literature is available concerning the therapeutic options for closure device-related endocarditis. Some patients heal with surgery [4, 6], but recovery after antibiotics alone is also reported [11]. In our opinion, given that the closure device itself may act as a source of infection, surgical removal is essential in the treatment of device-related endocarditis, especially in cases with a dynamic significant residual shunt or intractable infection (when the offending agent is *S. aureus* or a fastidious organism). In all cases, an adequate course of appropriate intravenous antibiotics is the basis of treatment.

Removing a mismatched closure device is often a challenge. In our case, the closure device used was designed for an ASD, with two surfaces stuck in the main pulmonary artery and aorta, respectively. We first attempted to fold the closure device using a sheath catheter,

which is designed to release the closure device during open-chest management of congenital heart defects, and it worked. If this had failed, we would have had to excise the two surfaces of the closure device with the abutting tissues through incisions in the main pulmonary artery and aortic arch, followed by repair of the great vessels. In some cases, replacement of the aortic arch would also be required. We partially attribute the success of our cable and sheath technique to the incomplete endothelialization of the device surface. It is also feasible to remove the closure device after complete endothelialization if this is preceded by removal of the neoendothelium.

To the best of our knowledge, device-related infectious endocarditis after percutaneous device closure for congenital heart disease is rare. There are few reports about the use of closure devices for ASD in a PDA site that was later complicated by a residual shunt and late infective endocarditis. This case is a good example of closure device-related complications and the therapeutic options.

In conclusion, we emphasize the importance of geometric conformance between the closure device and the defect. Surgery is recommended for patients with refractory device-related infection. Utilization of the cable and sheath method to fold the device is feasible in some patients.

Disclosure of conflict of interest

None.

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