Infective endocarditis following coil occlusion of perimembranous ventricular septal defect with the Nit-Occlud(®) Le device

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ABSTRACT

The Nitinol coil system was recently developed by "PFM" specifically for the transcatheter occlusion of ventricular septal defects (VSD). The device consists of a coil fitted with polyester fibers designated for the closure of perimembranous defects with an aneurysmal septum and some muscular VSDs. We report a case of fatal acute infective endocarditis 10 days following the procedure.

Keywords: Infective endocarditis, PFM coil, ventricular septal defect

INTRODUCTION

Ventricular septal defect (VSD) is among the most common forms of congenital heart defects.[1] The conventional treatment has been the surgical closure of these defects which carries a minimal risk of morbidity and mortality.[2] The most serious complication of this surgery is a complete atrioventricular block that has been reported in 0.7-3.1% of cases.[3,4] A comparative study between surgical and transcatheter closure of VSD showed that the transcatheter closure is less invasive, has a shorter recovery time and requires less hospital stay time. The complication rates of both methods were comparable.[5]

The Nitinol coil system was recently developed by PFM specifically for transcatheter occlusion of VSDs. The device consists of a coil fitted with polyester fibers designated for the closure of perimembranous defects with aneurysmal septum and cone-shaped muscular defects. Due to the flexible design of Nit-Occlud® Lê (PFM Medical), no permanent heart block has been reported so far.[6] Instances of endocarditis after device closure of VSDs are extremely rare. No instances of endocarditis following PFM coil closure have been reported.[7,8]

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Website: www.annalspc.com

DOI: 10.4103/0974-2069.171355

CASE REPORT

A 10-year-old female patient presented with perimembranous VSD measuring 5 mm, no aortic regurgitation, mild left ventricular dilatation, and no pulmonary hypertension. She was scheduled for device closure after her parents reported breathlessness on exertion and were advised that the defect was unlikely to close spontaneously at her age. She also had a small patent ductus arteriosus which was scheduled for intervention at a later date.

The patient underwent device closure using a 10 × 6 "PFM NiT-Occlud" device under general anesthesia following procedural guidelines.[6] New hardware was used as the hospital ceased its re-sterilization policy in 2011.

She was given 50 mg/Kg of cefatoxime before the procedure and a similar dose 12 h later. The procedure was successful with no residual shunt, intraoperative or postoperative complications.

Ten days later, the patient developed a persistent fever of up to 40°C which failed to respond to a 1-week

How to cite this article: El-Sisi AM, Menaissy YM, Bekheet SA. Infective endocarditis following coil occlusion of perimembranous ventricular septal defect with the Nit-Occlud(®) Le device. Ann Pediatr Card 2016;9:59-61.

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course of co-amoxyclav. The patient was admitted to the pediatric ward for additional investigation. The temperature was 39°C, heart rate was 140/m, respiratory rate was 30/min, and blood pressure was 100/60 mm Hg on admission. A new systolic murmur (3/6) was detected in the lower left sternal border with no organomegaly, no splinter hemorrhages or Roth's spots detected. Laboratory investigations showed an elevated erythrocyte sedimentation rate of 120 mm/h, C-reactive protein of 91 mg/dl, a white blood cell count of 28,000/mm^3 and normal platelet count as well as hemoglobin of 6 g% for which she received blood transfusion. Three sets of blood cultures were taken, and all grew *Pseudomonas aeuroginosa*.

Echocardiography revealed vegetation of 5 mm × 6 mm attached to the tricuspid valve, a flail tricuspid valve, and severe tricuspid regurgitation. It also showed moderate to severe dilatation of the right ventricle, but no pulmonary hypertension. The VSD was completely closed by the coil with no residual shunt. The patient was diagnosed with infective endocarditis and started empirically on the sensitivity results showed resistance to ceftazidime and sensitive to ciprofloxacin. Vancomycin was continued due to the high incidence of methicillin-resistant *Staphylococcus aureus* in the unit. She showed no improvement for 3 weeks and continued to develop intermittent fever; blood tests showed persistent leucocytosis, a high sedimentation rate, and high C-reactive protein.

The patient then underwent surgical intervention on cardiopulmonary bypass. Opening the right atrium revealed a destroyed tricuspid valve with vegetations and necrotic tissues around the coil [Figure 1]. The coil was removed, and the VSD was closed by an autologous pericardial patch using a continuous prolene suture [Figure 2]. The tricuspid valve could not be repaired and was replaced with a size 31 tissue valve [Figure 3]. The bypass and cross clamp time were 35 and 12 min, respectively. The patient developed heart block immediately and was paced, however, she continued to deteriorate with heart failure and ongoing sepsis. She required high doses of inotropes (adrenaline, dopamine, and dobutamine) and was continued on antibiotics. Cultures obtained from surgical specimens were negative. She died 2 days after surgery.

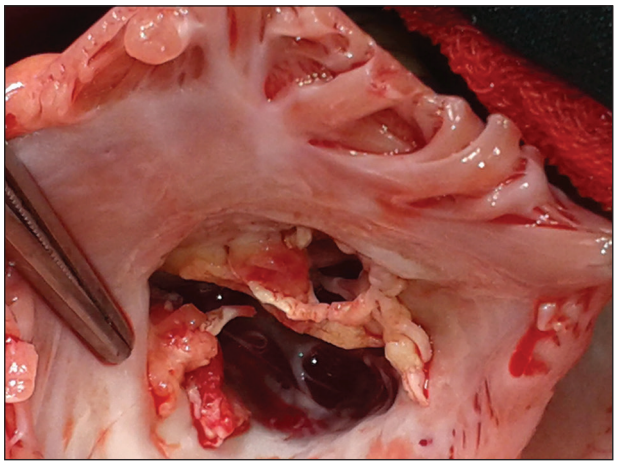

Figure 1: Destroyed tricuspid valve with vegetations and necrotic tissues

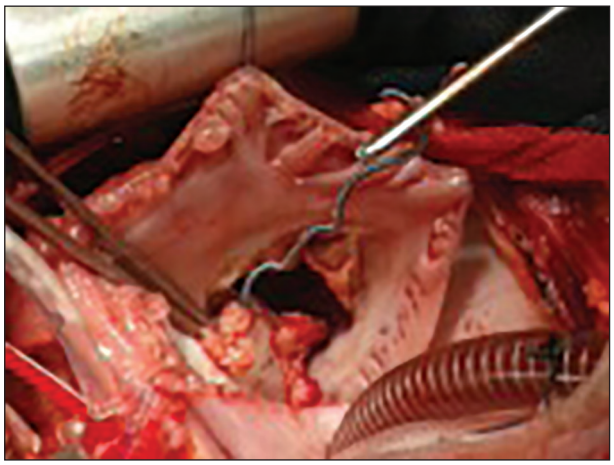

Figure 2: PFM coil removed

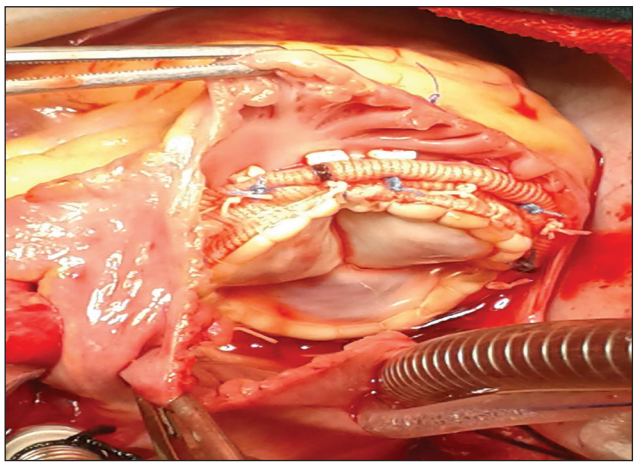

Figure 3: Tissue valve implanted in the tricuspid position

DISCUSSION

Transcatheter closure of VSDs was reported to be a safe and effective method, with a success rate of 130 out of 137 (94.8%) patients, using various devices.[9]

The main complications include conduction disturbances, device embolization, aortic regurgitation, tricuspid regurgitation, hemolysis, and residual shunts.[9] Despite the presence of a foreign body, endocarditis is considered a rare complication; only a few cases with Amplatzer-device associated endocarditis have been reported in children after closure of atrial

septal defects and two cases were reported after VSD closure.[6,7] To the best of our knowledge, this is the first report of a case that contracted aggressive *P. aeurogenosa* endocarditis after successful closure by a PFM Nit-Occlud device, complicated by severe tricuspid valve insufficiency and necessitating cardiac surgery. The patient also had patent ductus arteriosus, however, the vegetation and the infection were related to the region of the VSD device.

Device infection can occur in two ways: Through the introduction of microbes during implantation or due to the seeding of microorganisms after the procedure. After device implantation, it takes up to 6 months for complete neo-endothelialization. Any transient bacteremia during this time can potentially cause seeding of the device material with the microorganisms. The organism growth in our patient (*P. aeuroginosa*) is typical of nosocomial infection, indicating that the seeding likely occurred during the procedure. This stresses the importance of meticulous asepsis during the procedure and perhaps also suggests the need for infective endocarditis prophylaxis in such patients, for at least 6 months after device implantation. Sterilization was not achieved through antibiotics based on culture and sensitivity results, as evidenced by the large vegetation and presence of the foreign body.

CONCLUSION

To summarize, endocarditis is a rare but potentially fatal complication of device closure. A sterile environment and appropriate perioperative doses of antibiotic prophylaxis are paramount. Any fever within 1-2 weeks after an intervention should be taken as endocarditis unless proven otherwise. The families of patients should be instructed to immediately seek the emergency room when the fever does not subside within 2 days.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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