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Migration of Prosthesis About Two Case Operated at the André Festoc Baba Ibrahima Diarra, Cardio-Vascular and Thoracic Surgeon

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1. Abstract

Atrial septal defect (ASD) and patent ductus arteriosus (PDA) are frequent congenital heart defects in children. AAC is defined by the persistence of a communication between the two atria, secondary to a defect in septal partitioning during embryogenesis, creating a left-to-right shunt between these two cavities. It accounts for 10% of congenital heart defects diagnosed at birth, and 30 to 40% in adulthood, with an estimated prevalence of 14 to 24 per 100,000 births. Patent ductus arteriosus (PDA) is defined as communication between the proximal descending aorta and the main pulmonary artery beyond three (3) months of extrauterine life, resulting in a continuous left-right shunt. Its frequency is estimated at 5 to 10%, occurring in 1/2500 to 1/5000 live births. We report two cases of Amplatzer device migration secondary to percutaneous closure of ASD and PDA.

1.1. Case 1: This is a 15-year-old patient with no previous history of heart failure who had a 33-mm CIA ostium secundum and an 08-mm PDA with a velocity of 3 m/s and dilated right cavities.

1.2. Case 2: This was a 2-year-old patient from a singleton pregnancy at term, diagnosed with a 07 mm PCA with dilatation of the left cavities on heart echo. Both patients were initially managed by cardiac catheterization for percutaneous closure of ostium secundum ASD and PDA. Immediate echocardiographic checks revealed intra-VG migration of the AIC closure device in the first case, and migration of the prosthesis into the pulmonary artery in the second. Cardiovascular surgical advice was sought, and the indication was given for surgical extraction under extracorporeal circulation, which was carried out successfully in both cases. Post-operative management was straightforward in the first case. In the second case, fever with convulsive seizures followed by hemodynamic instability and hypotension (40/20 mm hg) led to death 24 hours later.

2. Introduction

Atrial septal defect (ASD) is a frequent congenital heart disease accounting for almost 10% of heart disease diagnosed at birth and 30 to 40% in adulthood, with an estimated prevalence of 14 to 24 per 10,000 births [1]. It is defined as the persistence of communication between the two atria, secondary to a defect in septal partitioning during embryogenesis, creating a left-right shunt between these two cavities. Patent ductus arteriosus (PDA) is a communication between the proximal descending aorta and the main pulmonary artery beyond three (3) months of extra-uterine life, resulting in a continuous left-right shunt. It is a frequent congenital heart disease, accounting for 5 to 10% of all cardiopathies [3] and affecting around one in 1250 (0.08%) to one in 5000 (0.02%) newborns.

Surgical treatment has long been considered the gold standard in

the management of AIC and PCA. It is effective, but not without complications. Percutaneous closure has become the treatment of first choice for persistent patent ductus arteriosus (PDA) and ostium secundum atrial septal defects (ASD), with prostheses including the Amplatzer prosthesis. Complications may arise during the percutaneous closure procedure, such as device migration per or post immediate procedure, residual shunt, embolization, aortic or pulmonary protrusion, which are by far the most reported complications of procedural failure of percutaneous closure [16]. As percutaneous device retrieval is not possible in most cases, surgical intervention remains the last resort in the event of device migration for the survival of these patients. The aim of this study is to report on the use of surgery in two (2) cases of Amplatzer prosthesis migration during percutaneous closure of a ASDos and a persistent ductus arteriosus.

3. Observations

3.1. Case 1

A 15-year-old female with no previous history of cardiac disease was admitted to the cardiology department for ostium secundum atrial septal defect (ASD), which had been present since early childhood and was marked by exertional dyspnea and recurrent respiratory infections. Physical examination revealed a systolic murmur of intensity 3/6th max at the pulmonary focus, radiating to the back. Biological tests were normal. Chest X-ray revealed bilateral hilar overload and cardiomegaly at 0.54 (Figure 1). The electrocardiogram showed a regular sinus rhythm with incomplete right bundle-branch block without cavitary hypertrophy. Cardiac echocardiography noted a 33 mm atrial septal defect, associated with a 08 mm patent ductus arteriosus with velocity 3 m/s, dilated right cavities (dilated OD for 25 cm² surface area, dilated non-hypertrophied right ventricle TAPSE : 29 mm), moderate pulmonary valve stenosis with mean gradient: 45 mm hg, max gradient: 69 mm hg, with max velocity: 04 m/s, dilated VD-AP pathway, Right pulmonary artery: 16.5 mm, small left pulmonary artery: 11 mm, pulmonary valve opens in a dome shape. The indication for percutaneous closure of ASD and PDA was retained.

She had undergone percutaneous ASD and PDA closures with Amplatzer prostheses, as well as dilation of the pulmonary stenosis via a right femoral venous approach, under transthoracic and transesophageal ultrasound control in the interventional cardiology unit. After successful dilation of the pulmonary stenosis and closure of the ductus arteriosus, immediate post-procedure echo cardiography revealed intra-ventricular left migration of the inter-atrial septal defect closure device (Figures 2, 3, 4). The patient was operated on under extracorporeal circulation after aorto-bicaval cannulation. After aortic clamping and exclusion of the vena cava, cardiac arrest was achieved by antegrade blood cardioplegia. A right atriotomy was performed to explore the atria. This revealed a 15 mm atrial septal defect and the presence of an Amplatzer prosthesis in the left ventricle (Figures 5-6). We therefore proceeded with its extraction (Figures 7-8) and closure of the septal defect with a patch of heterologous pericardium using 2 hemi-splices of 5/0 prolene (Figure 9). We then closed the right atriotomy with a double 5/0 prolene overjet after purging the right and left cavities. Aortic clamping took 58 minutes and extracorporeal circulation 81 minutes. The patient spent 3 days in the intensive care unit and 6 days in the post-resuscitation unit, for a total hospital stay of 9 days. No complications were observed.

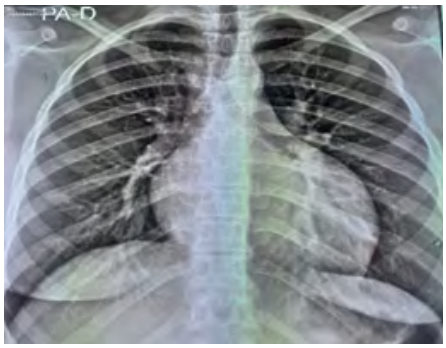

Figure 1: Chest X-ray showing bilateral hilar overload and cardiomegaly at 0.54

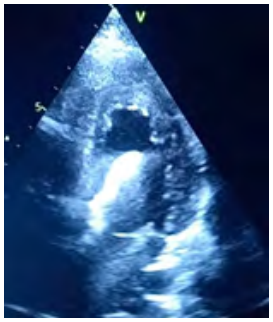

Figure 2: Apical section 4C: showing a left ventricular device

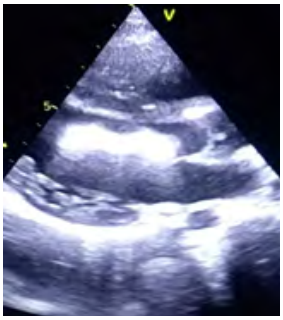

Figure 3: Long-axis PS section: left ventricular device

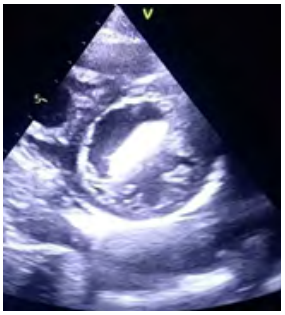

Figure 4: Short-axis PS section: showing a left ventricular device

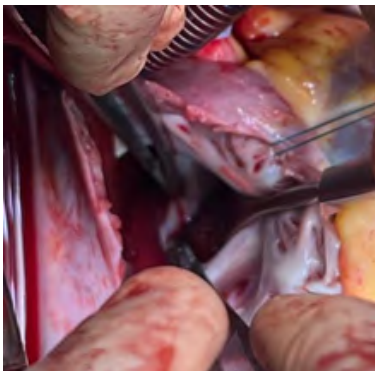

Figure 5: demonstration of an ASD and a left ventricular device

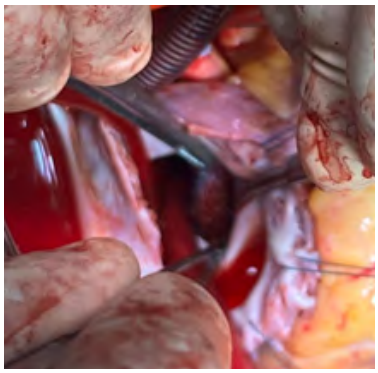

Figure 6: Demonstration of the Amplatzer prosthesis in the left ventricle

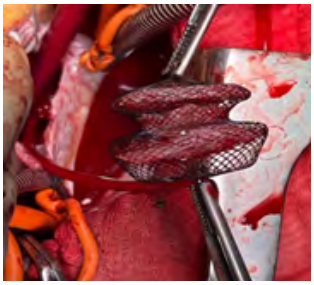

Figure 7: Removed Amplatzer prosthesis

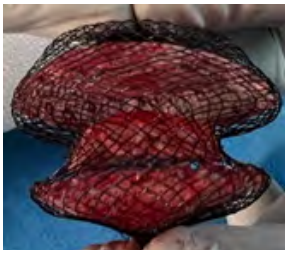

Figure 8: Amplatzer prosthesis in large format

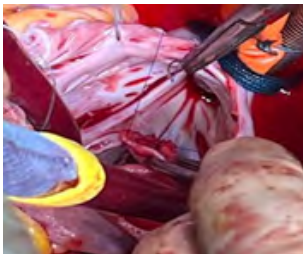

Figure 9: Closure of ASD bone with a patch of heterologous pericardium

3.2. Case 2

A 2-year-old patient from a singleton pregnancy at term, followed for persistent ductus arteriosus. Her symptoms included recurrent respiratory infections and dyspnoea on feeding, which had been present since birth. Physical examination revealed a left subclavicular tremor and a left subclavicular systolo-diastolic murmur of intensity 5/6. Cardiac ultrasound revealed a 7 mm patent ductus arteriosus (PDA) with dilatation of the left cavities (Figures 10; 11). Initially managed by the cardiac catheterization center for percutaneous closure, the immediate post-procedure echocardiogram had noted a device migration into the pulmonary artery with a patent ductus arteriosus. She was urgently referred to us for removal of the prosthesis and surgical closure of the ductus arteriosus.

The approach was via a vertical median sternotomy. After closure of the ductus arteriosus, the extracorporeal circulation was set up between an aortic cannula and two vena cava cannulae. The ductus arteriosus was approximately 15 mm wide. Opening the pulmonary artery trunk (PAT) revealed a prosthesis (Figures 12, 13), which was extracted (Figure 14). The postoperative course was marked by fever with convulsive seizures, followed by hemodynamic instability with hypotension (40/20 mm hg) and death 24 hours postoperatively.

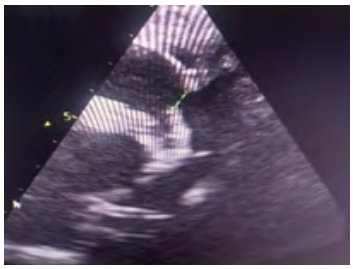

Figure 10: Supra-sternal section: showing a PDA

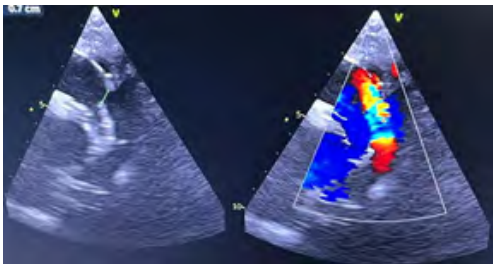

Figure 11: Short-axis para-sternal section showing PDA in two-dimensional mode

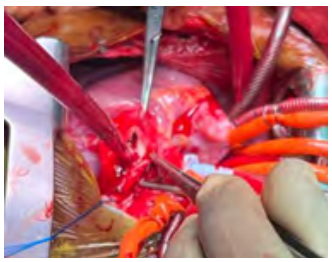

Figure 12: Pulmonary artery incision.

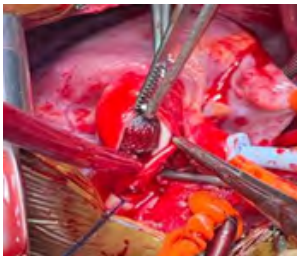

Figure 13: Demonstration of a prosthesis in the pulmonary artery trunk.

Figure 14: Amplatzer prosthesis extracted from the pulmonary artery

4. Discussion

Interatrial septal defect and persistent ductus arteriosus are congenital heart defects representing respectively 10% and 5-10% of all congenital heart defects [4]. In the past, surgery was the only therapeutic option. Since the first attempts at percutaneous closure, respectively in 1976 by King [2] and Mills, and in 1967 by Portsmann [21], percutaneous closure of ASD and PDA has become an alternative to surgery. Transcatheter treatment modalities have become common worldwide for the closure of ASD and PDA [1,6]. The main advantages are the absence of surgical trauma and adverse effects of extracorporeal circulation, shorter hospital stay, rapid resumption of normal life, and better aesthetic results [6,7]. Several types of prosthesis appeared in the 1980s and were the subject of clinical studies in the United States and Europe without proving their effectiveness. The Amplatzer prosthesis rapidly supplanted all others when it was first used in 1997, for a number of reasons: ease of use, easy retrieval of the prosthesis in the event of malpositioning, very low rate of residual shunts due to its self-centering nature, and very low complication rate due to its circular, non-contouring shape [17]. The Amplatzer prosthesis is self-extending and self-centering, the latter giving it great stability since the central part acts as a stent through the ASD [18]. It is now estimated that around 80% of bone ASD can be closed with the Amplatzer prosthesis. However, this figure only applies to adolescents and adults [19]. In children, the ASD is very wide, and the size of the interatrial septum must be taken into account, with the risk of obstruction of neighboring structures (VSD, coronary sinus, pulmonary veins) severely limiting the indications [18].

According to the two scientific societies, the European Society of Cardiology (ESC) and the American Society of Cardiology (ACC/AHA), percutaneous closure is to be ruled out in the presence of an asymptomatic bone ASD <5 mm, a wide bone ASD > 40 mm, edges < 5 mm (except for the anterosuperior border, the absence of which does not contraindicate percutaneous closure), complex non-oval anatomical shapes, multiple defects, the presence of other anomalies requiring surgical correction, such as abnormal pulmonary venous return, and the presence of fixed PAH [2, 4, 5]. With regard to PDA, the interventional technique is contraindicated in the following forms: PCA with pulmonary arterial hypertension (PAH), duct diameter greater than 10 mm, prematurity, children under 5 kg, tubular and long Krichenko type E duct, and endocarditis with persistent ductus arteriosus [13]. Other situations, such as femoral venous thrombosis, sepsis, recurrent pulmonary infections, any severe infection less than 1 month old, and the presence of thrombi-intracardia also contraindicate closure by Amplatzer prosthesis [20]. The procedural success rate described ranged from 85 to 100% [8-10], comparable to that found by Abid et al [7], with 94.3% procedural success and 5.7% procedural failure.

However, despite the advantages of this technique and a promising success rate, complications are not negligible. Pascal A. Berdat et al [6] report recourse to surgery in 8% of cases having benefited from percutaneous closure. The rate of serious complications requiring urgent surgical intervention (malposition or migration of the prosthesis, cardiac perforation) is around 1% in large series [21, 22,23]. Intra- and post-procedural accidents are not uncommon, with device migration the most frequently described complication (0.55% to 1.1%) [4,7]. Chessa et al [8] report a 3.5% rate of embolization/malposition and a 2.6% rate of arrhythmia (series of 417 patients). It occurs either immediately after the procedure or late in the procedure, and is discovered on echocardiographic monitoring in the absence of symptoms. Otherwise, symptomatology depends on the site of device migration: right heart chambers, pulmonary arteries, left heart chambers, aorta or one of its collaterals. Several factors predisposing to this complication are incriminated: a large defect, a large or small device, defect edges < 5mm, narrowness of the OG. Baldino et al [9] report the following locations (OG in 24.6% of cases, aorta in 18.4% and right ventricle in 16.7%). ABID et al [7] noted immediate procedural failure in three 3 of 53 patients, with migration of the Amplatzer prosthesis in two cases (3%) and persistence of a large residual shunt in one case (1%), necessitating recourse to surgical treatment. A case of secondary migration of the Amplatzer into the left ventricle as a short-term complication in a patient with a 22 mm ASD closed by a prosthesis that appeared stable at the time of implantation, the Amplatzer was urgently removed surgically. The ASD was closed at the same time [7].

Noureddine et al [12] reported a case of tricuspid valve obstruction due to migration of the Amplatzer device, revealed by severe ventricular arrhythmia. Concerning procedural failure of the persistent ductus arteriosus, Gribaa et al [10] recorded 11 complications in their study: 04 cases of protrusion into the aortic isthmus, 03 cases of protrusion into the pulmonary artery, one case of inguinal hematoma, three (3) cases of prosthesis migration, including two migrations into the PA respectively in a 16-month-old patient without

recourse to surgery and in a 3-month-old infant weighing 6 kg with a type C duct, one case of Amplatzer ADO II aortic migration into the aorta removed by lasso.

Some authors have reported cases of migration with both ADO II and ADO II AS, with a frequency of up to 6.7% [11]. According to the report by Faella and Hijazi [14], 15 patients out of 316, or 4.7%, experienced complications, including one case of sudden death after the procedure, one case of hemolysis, one case of surgical closure and one case of device misplacement. Shyam et al [24] reported two cases (1.05%) of migration of the Amplatzer device into the pulmonary artery, in a 1.6 kg child and a 04 kg child respectively, after 24 hours of the procedure. In the first case, the device was retrieved using a transcatheter approach with a 4-French sheath. In the second case, the device was retrieved 189 days later during a residual shunt obstruction procedure.

5. Conclusion

The choice of prosthesis for percutaneous closure must be adapted to the clinical characteristics of each patient, but also to the anatomy of the canal and ASD, to avoid complications.

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