REPARAÇÃO DE CIV POR AORTOTOMIA COMO CAUSA DE ENDOCARDITE VALVULAR AÓRTICA NATIVA

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Resumo

As comunicações interventriculares são a lesão congénita mais comummente diagnosticada em idade pediátrica mas representam apenas 10% dos defeitos cardíacos congénitos em idade adulta. Apesar da maioria das comunicações interventriculares encerrarem espontaneamente antes da idade adulta, muitas persistem predispondo a endocardite e outras complicações.

Apresentamos um caso de comunicação interventricular perimembranosa assintomática até à idade adulta, que complicou aos 53 anos com endocardite da válvula aórtica nativa associada a regurgitação aórtica severa, necessitando, por isso, de correção cirúrgica. Optamos pela correção cirúrgica implantando um retalho de pericárdio heterólogo através da aortotomia necessária para a substituição valvular aórtica (Figure 1 and 2). A cirurgia decorreu sem intercorrências. De referir apenas, no pós-operatório, a necessidade de implantação de um pacemaker permanente dado existência prévia de doença do nó sinusal. É de salientar a importância do papel da cirurgia na correção de comunicações interventriculares e a necessidade de escolher a abordagem cirúrgica mais apropriada, especialmente, quando existem lesões cardíacas concomitantes.

Abstract

Correction of adult-type VSD causing aortic valve endocarditis through aortotomy

Ventricular septal defects are the most common congenital abnormality diagnosed in children but account for only 10 percent of congenital heart defects in adults. Although many defects close spontaneously before adulthood, many others persist, predisposing to endocarditis, and other complications. Herein, we report a case of a known, asymptomatic, perimembranous ventricular septal defects that has complicated at 53 years of age with the need for surgery due to native aortic valve endocarditis and concomitant severe aortic regurgitation. We opted to surgically repair the ventricular septal defects with a pericardial patch through the necessary aortotomy used for aortic valve replacement (Figure 1 and 2). The surgery was straightforward. Postoperative course was only marked by the need of a permanent pacemaker implantation due to a sick sinus syndrome, which was diagnosed before the surgery. Thus, we emphasize the role of surgery in repairing ventricular septal defects and the importance of choosing the appropriate approach, especially when concomitant heart lesions are present.

CASE REPORT

A 53 year-old male with a known asymptomatic perimembranous ventricular septal defect (VSD) was admitted to the emergency department with fever and congestive left heart failure. Other than the known VSD, his past medical history included hypertension and dyslipidemia.

The patient underwent an echocardiogram which revealed a perimembranous restrictive VSD with a LV/RV maximum gradient of 124mmHg (figure1), infracentimetric ecogenic lesions of the aortic cusps suggestive of vegetations and severe aortic regurgitation; ventricular function was normal and there was moderate enlargement of the left chambers.

The diagnosis of the VSD complicated with native aortic endocarditis and severe aortic regurgitation having

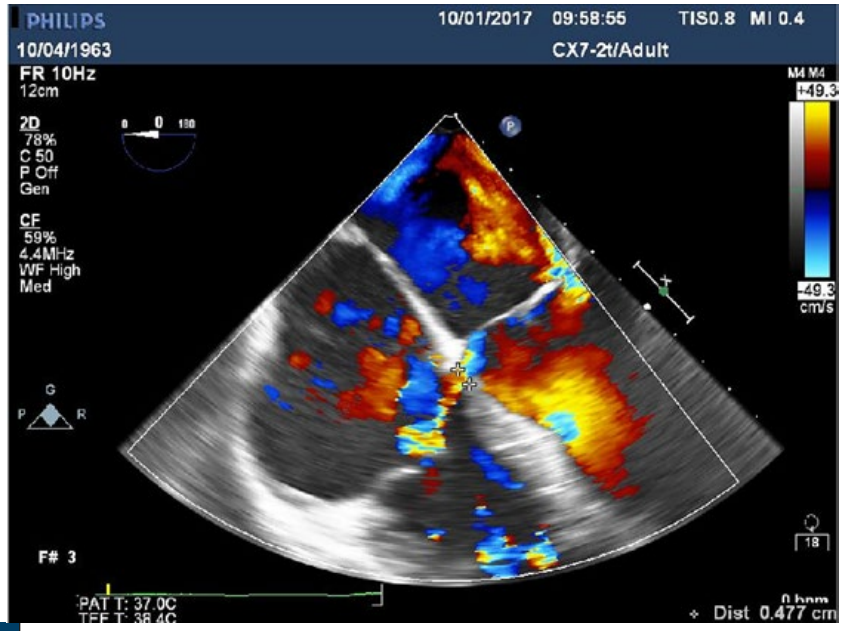

Figure 1 *Preoperative echocardiogram showing perimembranous VSD.*

thus indication for surgical repair. It is not known if the isolated VSD was complicated by "jet lesion infection" or if there was any associated regurgitation previously. As there weren't any criteria for urgent surgery, we opted to wait for two weeks before operating. While awaiting surgery hemocultures revealed *Granulicatella* adiacens bacteremia and thus targeted antibiotic therapy with ampicillin and gentamicin was initiated.

The operation was undertaken on cardiopulmonary bypass with general anesthesia. Intraoperative transesophageal echocardiography revealed severe aortic regurgitation and severe left ventricular dilation, which resulted in functional severe mitral and tricuspid regurgitation. Cardiopulmonary bypass was established with cannulation of the ascending aorta for arterial return and bi-caval cannulation for venous drainage in mild hypothermia (32ºC). Venting was performed through the right superior pulmonary vein. The aorta was cross-clamped and cold-blood cardioplegia delivered into the aortic root and coronary sinus. The procedure was performed with aortic replacement with a #23 ON-X® (On-X Life Technologies Inc®, Austin, TX, USA) mechanical prosthesis, mitral annuloplasty with a #34 Seguin® (St Jude Medical/Abbott, St Paul, MN, USA) ring, tricuspid annuloplasty with a #34 Contour 3D® (Medtronic, Minneapolis, MN, USA) ring and VSD closure with a heterologous pericardial patch.

After removal of the infected native aortic valve and preparation of the annulus, the heterologous pericardial path was implanted over the VSD using a continuous prolene 4.0 suture. Due to the anatomic region of the VSD and good exposure, we opted to surgically repair the defect through the aortotomy, avoiding ventriculotomy or partial deinsertion of tricuspid valve (figure 2 and 3). Mitral repair was performed through a left atriotomy. After closure of the left atrium, the aortic prosthetic valve was implanted over the annulus and the pericardial patch, using 14 ventricular pledget sutures. Right atriotomy was executed for tricuspid annuloplasty. No intra-operative complications were registered and cardiopulmonary bypass was discontinued with the need of temporary pacemaker but no significant inotropic support.

Intraoperative transesophageal echocardiogram showed no residual VSD, correctly implanted aortic prosthesis and only residual mitral and tricuspid regurgitation (figure 4).

The patient was extubated 12h after arriving at the intensive care unit and required no inotropic support. He revealed an extreme sinus bradycardia and junctional escape rhythm with the need for permanent pacemaker implantation.

The patient is still under hospitalization for continuation of antibiotic treatment. At 2 week follow-up the patient is asymptomatic and has an echocardiogram revealing normofunctioning aortic prosthesis, mitral and tricuspid valves with no residual VSD.

DISCUSSION

Isolated ventricular septal defect (VSD) is the most common congenital heart defect, apart from bicuspid aortic valve, accounting for 30-40% of all congenital cardiac malformations.[1,2,3,4] It is generally diagnosed and, if necessary, treated in childhood. Although many defects close spontaneously before adulthood and many have insignificant left-to-right shunt remaining event-free throughout the patient's lifespan, many others persist and predispose to endocarditis, arrhythmias, heart failure, aortic/tricuspid regurgitation, double chambered right ventricle (DCRV) and/or pulmonary hypertension (PH).[1,2,3,4]

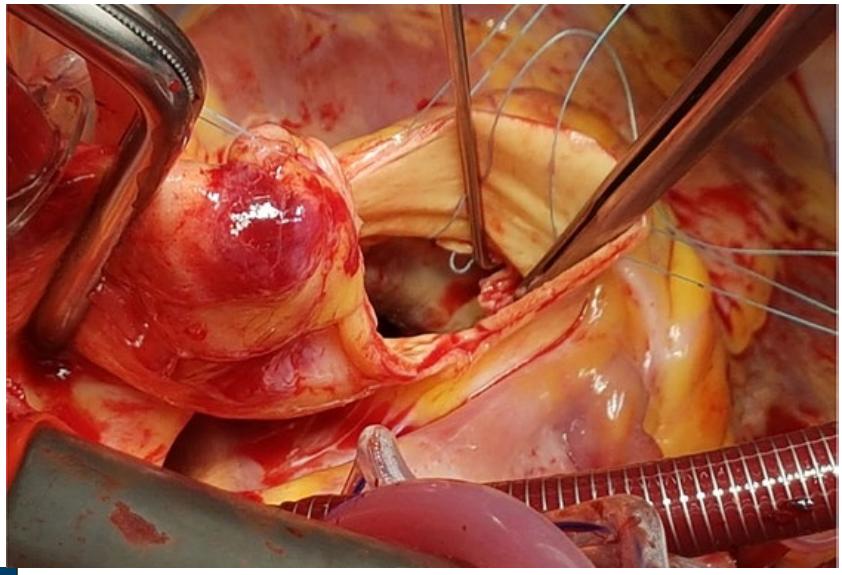

Figure 2 *Continuous suture for the implantation of a pericardial patch to repair a perimembranous VSD through an aortotomy.*

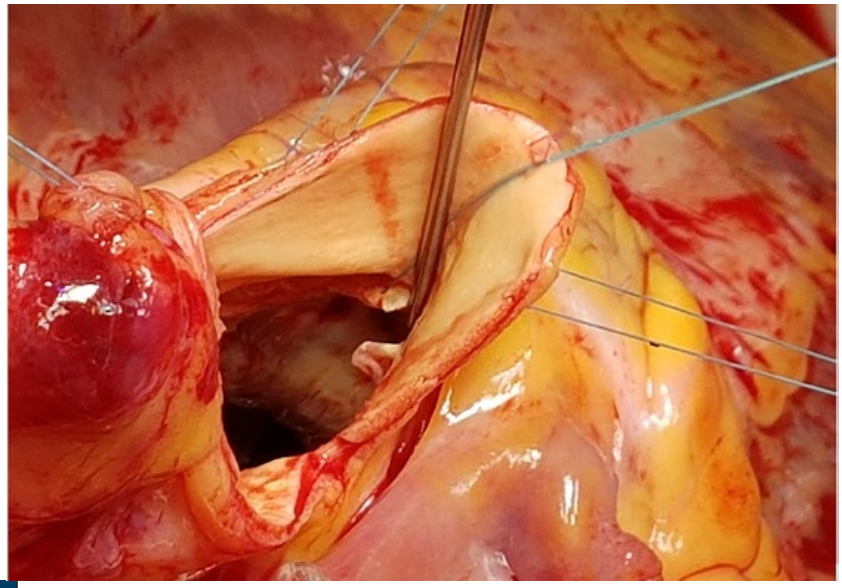

Figure 3 *Repair of a perimembranous VSD with a pericardial patch through an aortotomy.*

Four subgroups have been defined according to defect location: Infundibular/subarterial, membranous/perimembranous, inlet and muscular type.[1,2,3,4] The perimembranous type is the most common in adults (about 80% of all VSDs). Due to great variability in sizes, locations, presentation, natural history and complications, treatment is variable and challenging.[1,2,3,4]

Since current guidelines state a class IIa indication for VSD closure after infective endocarditis and emphasize that repair of VSDs with aortic regurgitation should not be delayed even in asymptomatic patients, we opted to treat the VSD during surgery.[5,6,7,8] Transcatheter device VSD closure was never a treatment option as it is only a possibility in isolated uncomplicated VSDs.[5,6,7]

Even though recent studies evaluating the long-term outcome of adult patients with VSDs are scarce, isolated VSDs that underwent elective surgical closure seem to have quite good overall survival. Nevertheless surgical closure of ventricular septal defects has proven to be difficult, especially in patients with concomitant heart lesions.[6,7,8] The main complications during follow-up include: development of atrial arrhythmia, pacemaker implantation for high-degree AV block/complete heart block, some degree of LV outflow tract obstruction and even new onset endocarditis.[6,7,8] In this case, pacemaker implantation was necessary but previously sick sinous syndrome was already diagnosed. Nevertheless, permanent pacemaker implantation due to AV block remains a major concern after VSD closure mainly due to its' anatomic position and it's relation to the conduction bundle which lies just below the membranous septum. This

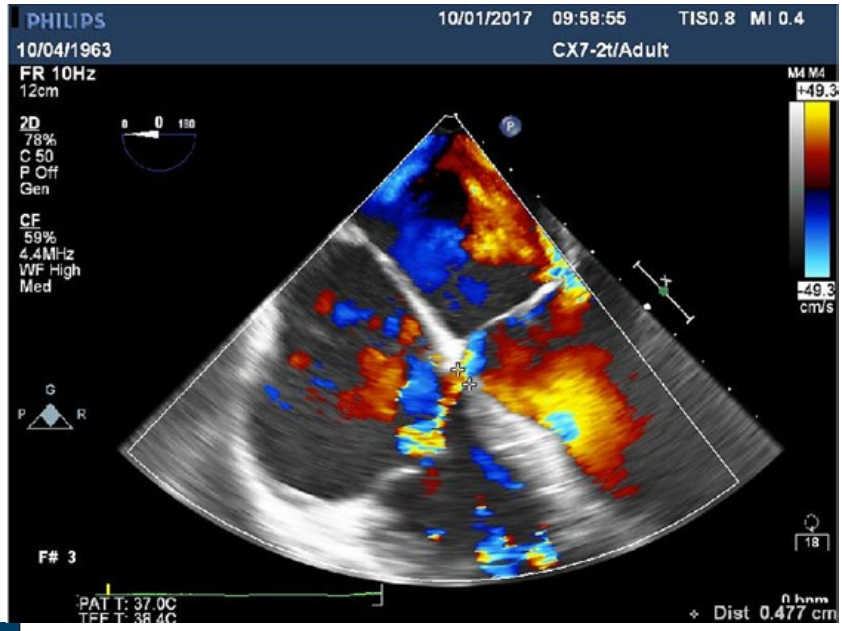

Figure 4 *Postoperative echocardiogram showing no residual VSD.*

particular constant location makes it common to induce block, particularly when treating the perimembranous type, using patch closure or ventriculotomy incisions.[6,7,8] The endocarditis itself could be another cause for the AV block, but I this case the infection was localized to the leaflets and thus infection should not be blamed for the conduction disturbance.

In summary, this case report illustrates a necessary, difficult, but successful surgery even though the very real possibilities of operative hazards were present. We conclude that the aortotomy pericardial patch implantation approach is a good solution for repairing VSDs, but also recognize that the choice for this or other approaches will depend mainly on the VSD's location, anatomy, severity and existence of concomitant lesions. We conclude that the aortotomy pericardial patch implantation approach is a good solution for repairing VSDs, but also recognize that the choice for this or other approaches will depend mainly on the VSD's location, anatomy, severity and existence of concomitant lesions.

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