The Heart Surgery Forum #2007-1205
11 (2), 2008 [Epub April 2008]
doi: 10.1532/HSF98.20071205

Online address: http://cardenjennings.metapress.com

Diverticulum of the Left Ventricle: Etiology and Surgical Treatment

Antonis A. Pitsis, MD, FETCS, FESC,[1] Aikaterini N. Visouli, MD,[1] Timotheos G. Kelpis, MD,[1] Vlasis N. Ninios, MD,[1] Nikolaos Nikoloudakis, MD,[1] Fragiskos Parthenakis, MD, FESC,[2] Panos Vardas, MD, PhD[2]

[1]Thessaloniki Heart Institute, St Luke's Hospital, Thessaloniki, Greece; [2]University of Heraklion, Crete, Greece

ABSTRACT

Congenital left ventricular (LV) diverticula are rare findings, particularly when first diagnosed in adulthood. We describe successful surgical repair of an isolated congenital apical LV diverticulum associated with an abnormal submitral apparatus in a young adult who received his diagnosis following a peripheral embolism. We intraoperatively identified an abnormally originating belly of the posteromedial papillary muscle, which arose from the interventricular septum, causing partition of the LV cavity and forming an apical aneurysmal communicating chamber. This anatomy of abnormal papillary muscle bifurcation was possibly the mechanism for the development of the apical diverticulum and to the best of our knowledge has not previously been described. Reattachment of the abnormal belly to its normal position preserved mitral valve competence, and an endoventricular patch repair restored LV volume and geometry.

INTRODUCTION

Congenital diverticula, aneurysms, and double-chambered left ventricle (DCLV) are rare cardiac malformations and are included in the broader class of subdivisions of the LV cavity [Parthenakis 2005].

Since the first description in 1816, 411 cases of congenital aneurysms or diverticula have been reported and have been associated with other cardiac, vascular, or thoracoabdominal abnormalities in approximately 70% of cases [Ohlow 2006].

There are rare reports of mitral valve and papillary muscle anomalies associated with apical LV diverticula, but the specific anatomy in our case, an LV partition by an abnormally originating papillary muscle belly, to the best of our knowledge has not previously been described.

Received November 3, 2007; accepted January 3, 2008.

Correspondence: Antonis Pitsis, St Luke's Hospital, Panorama, 552 36, Thessaloniki, Greece; 30-2310-390550; fax: 30-2310-342055 (e-mail: apitsis@otenet.gr).

CASE REPORT

A 34-year-old man with a history of peripheral embolism was referred for surgical repair of an LV diverticulum. Investigation had revealed an apical diverticulum communicating and contracting simultaneously with the main LV chamber, a competent mitral valve, normal coronary arteries, and diverticulum perfusion. The patient received anticoagulation treatment until surgical correction [Parthenakis 2005].

An intraoperative transesophageal echocardiograph showed the hypokinetic, synchronously contracting apical diverticulum (Figure 1A) and a competent mitral valve. When the patient was on cardiopulmonary bypass, we identified the diverticulum as a flaccid area at the LV apex. We performed an apical ventriculotomy 3 cm to the left of the left anterior descending artery and entered the conical diverticulum. Its apex coincided with the LV apex, whereas its base consisted of a thick muscular band attached to the posterolateral LV wall and the posterior interventricular septum at approximately 3 cm from the apex. The free wall of the diverticulum was thin, and its neck consisted of the anterior interventricular septum, the anterior free edge of the muscular band, and the anterior LV wall, and had a diameter of 1 cm. Closer inspection revealed that the posteromedial papillary muscle had 2 separate bellies, one normally originating from the posterolateral wall and a second belly abnormally originating from the interventricular septum. The muscular band causing the LV partition consisted of this abnormal septal belly connecting to the normal belly beneath the origin of the tendinous cords (Figure 2A).

We detached the abnormal belly from the interventricular septum and reattached it at the posterolateral free wall close to the origin of the normal belly (Figures 2B and 2C). We sutured an oval-shaped bovine pericardial patch (6 × 9 cm) to exclude the diverticulum from the major LV cavity. We then oversewed the remnants of its free wall to cover the patch and ensure hemostasis. After discontinuing cardiopulmonary bypass, we confirmed normal LV volume and geometry and established the competence of the mitral valve (Figure 1B).

The patient had an uneventful recovery and remains asymptomatic. He received anticoagulation therapy for 3 months. An echocardiographic evaluation at 9 months postoperatively revealed normal LV shape and contractility and showed no mitral regurgitation (MR).

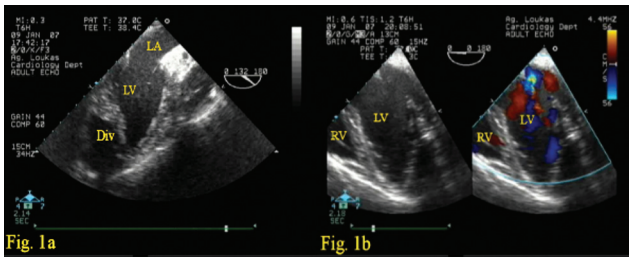

Figure 1. Intraoperative transesophageal echocardiography. A, Midesophageal long-axis prerepair view showing the apical diverticulum (Div) with narrow communication with the major left ventricular (LV) cavity. B, Four-chamber postrepair view showing restored LV volume and geometry. LA indicates left atrium; RV, right ventricle.

DISCUSSION

Although the terms *congenital diverticulum* and *aneurysm* have been used interchangeably, congenital muscular diverticula are protrusions of the ventricular wall that include all 3 cardiac layers, a narrow neck, and contractility, whereas aneurysms have wide communication with the ventricle. Congenital diverticula can be asymptomatic or may cause systemic embolization, valvular regurgitation, heart failure, ventricular tachycardia, ventricular wall rupture, or sudden cardiac death [Parthenakis 2005; Ohlow 2006].

There are rare reports of apical LV diverticula, aneurysms, and DCLV affecting the mitral valve and the submitral apparatus, but the nature of papillary muscle involvement with LV apical lesions has not been clearly defined. Although MR is common in subannular diverticula, apical diverticula are rarely associated with MR [Gueron 1976; Mardini 1984; Yoshizumi 2005].

In the first report of 2 cases of apical diverticula associated with MR [Gueron 1976], the authors commented that papillary muscle involvement with the diverticula could explain the MR. In a reported case of a congenital apical diverticulum [Mardini 1984], the associated incompetence of a dysplastic, fibrotic, and myxomatous mitral valve was treated by mitral valve replacement. In another reported case involving a severely symptomatic infant with an apical diverticulum [Yoshizumi 2005], the regurgitation of the mitral valve, which had a normal appearance, was attributed to the involvement of the diverticulum with the papillary muscles, to which it was closely located. The diverticulum was excluded via a Dor procedure, which improved the orientation of the papillary muscles, and an additional central double-orifice mitral valve repair was required to treat the MR.

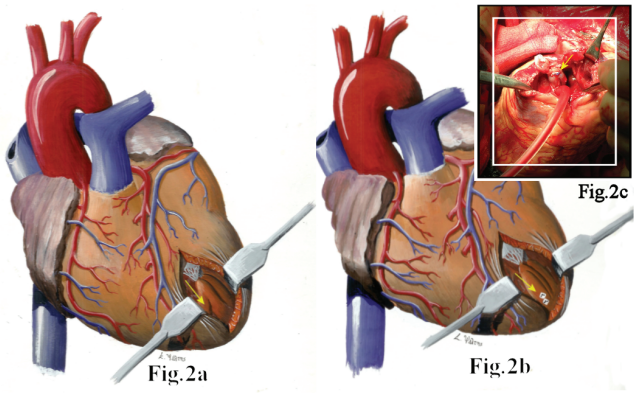

Figure 2. Anatomy and repair of the diverticulum. A, The abnormal septal belly of the posteromedial papillary muscle (arrow) arising from the interventricular septum and connecting to the normally originating belly beneath the tendinous cords. B, Reattached septal belly. Arrow indicates the pledgeted sutures used for reattachment. C, Reattachment of the abnormal septal belly to its normal position on the posterolateral free left ventricular wall (intraoperative photograph).

Abnormal mitral valves were reported [Papagiannis 2001] in all 4 autopsied cases of congenital LV aneurysms (apical or involving most of the LV free wall), along with short, thin, or absent papillary muscles.

DCLV separated by an abnormal muscle bundle has rarely been reported in the literature. In one case of DCLV, a large submitral ridge obstructing the LV inlet and outlet was located in series with the mitral apparatus [Albertucci 1994]. In another case, a 13-year-old girl with DCLV underwent both surgical resection of the hypertrophic floating mass and mitral valve replacement [Dogan 2004].

In our patient, LV partition due to the abnormal septal posteromedial papillary muscle belly could have led to diverticulum formation, and although several theories about the etiology and pathogenesis of congenital diverticula have been described [Parthenakis 2005; Ohlow 2006], this report is the first in the literature, to the best of our knowledge, to identify such a possible triggering mechanism for the development of this rare pathology. We concluded the lesion to be congenital because of the absence of ischemia, trauma, inflammation, and cardiomyopathy [Ohlow 2006] and the nature of the anatomic findings. No concomitant cardiac or extracardiac lesions were found.

Surgical correction of the lesion, which had caused a life-threatening complication, was the treatment of choice. The free wall of the diverticulum was dilated, hypokinetic, and thin. Its filling and emptying was slow, indicating blood stagnation and predisposing to thrombus formation. Apart from the continuous risk of thromboembolism, further dilatation and decreasing wall thickness could have led to LV dysfunction and failure, or even rupture and sudden cardiac death.

Despite the presence of an abnormally originating septal belly, the mitral valve was competent. The identification of the nature of the band causing the LV partition led to preservation of the submitral apparatus and mitral valve competence. Reattachment instead of excision of the abnormal septal belly prevented weakening of the papillary muscle and preserved durable mitral competence. Surgical ventricular remodeling instead of diverticulum excision preserved LV volume and geometry, thus avoiding the risk of diastolic heart failure.

Apical diverticula may be associated with mitral valve abnormalities even in the absence of mitral regurgitation. During surgical correction of apical diverticula, a careful

evaluation of the mitral valve, the subvalvar apparatus, and their relationships to the lesion is needed, and proper orientation of the papillary muscles must be secured to repair existing MR or to avoid inducing MR.

ACKNOWLEDGMENT

We thank Dr. Emmanuel Psaltis for designing the figures.

REFERENCES

Albertucci M, Lang RM, Karp RB, Marcus RH, Follman D. 1994. Double-chambered left ventricle: submitral accessory valvular tissue causing inlet and outlet obstruction. J Am Soc Echocardiogr 7:67-71.

Dogan OF, Alehan D, Duman U. 2004. Successful surgical management of a double-chambered left ventricle in a 13-year-old girl: a report of a rare case. Heart Surg Forum 7:E198-200.

Gueron M, Hirsch M, Opschitzer I, Mogel P. 1976. Left ventricular diverticulum and mitral incompetence in asymptomatic children. Circulation 53:181-6.

Mardini MK. 1984. Congenital diverticulum of the left ventricle: report of two unusual cases. Br Heart J 51:321-6.

Ohlow MA. 2006. Congenital left ventricular aneurysms and diverticula: definition, pathophysiology, clinical relevance and treatment. Cardiology 106:63-72.

Papagiannis J, Van Praagh R, Schwint O, et al. 2001. Congenital left ventricular aneurysm: clinical, imaging, pathologic, and surgical findings in seven new cases. Am Heart J 141:491-9.

Parthenakis FI, Kochiadakis GE, Patrianakos AP, et al. 2005. Peripheral arterial embolism due to a left ventricular diverticulum in a young adult. Chest 127:1452-4.

Yoshizumi K, Ishino K, Kawada M, Fujisawa E, Ohstuki S, Sano S. 2005. Dor procedure for left ventricular diverticulum with mitral regurgitation in an infant. Ann Thorac Surg 80:332-3.