pulmonary arterial hypertension (PAH) that precluded HTX consideration. Treatment with sildenafil citrate 20 mg TID was initiated and gradually up-titrated to 100 mg TID. After achieving target doses, a new right heart catheterization showed improved pulmonary resistance with reversible PAH. The pre-transplant study did not show any formal contraindication for HTX. Because of the patient's critical end-stage heart failure, young age, excellent response to combined antiretroviral therapy (cART) and lack of comorbidities, he was listed for HTX July 19, 2006. On September 7, 2007, the patient underwent successful elective orthotopic HTX.

The post-operative course was uncomplicated, standard induction was done with basiliximab 20 mg within 24 h of HTX and at 4 days post-transplant. Routine immunosuppression treatment with methylprednisolone, tacrolimus and mycophenolate mofetil was initiated the day of surgery. The dose of tacrolimus required to achieve the target level of 10-15 mg/dl was low during the first 6 months after transplantation (2 mg daily) and increased with time to the standard dose (6 mg daily). The cART was restarted on day 3 after HTX. Postoperatively, consistent improvement in PAH allowed the progressive withdrawal of sildenafil citrate until discontinuation.

It is now 3 years from HTX and the patient has improved remarkably. He has resumed work and leads an active life. Over this time, the patient had 1 episode, at 2 months after HTX, of acute rejection (International Society for Heart and Lung Transplantation grade 3A) requiring treatment with high-dose corticosteroids. Left heart catheterization at 1 year after HTX showed normal coronary arteries. He has not developed any focal or disseminated bacterial, viral, or fungal infection or any AIDS-defining disease. Weekly cytomegalovirus pp65 antigenemia testing was always negative. Under the cART regimen in addition to standard doses of immunosuppressant drugs, no pharmacokinetic interaction was detected. The CD4+ T-cell count evolution over time ranged between 201 and 754 cells/μL (Fig. 1).

The survival and quality of life in patients infected with HIV has dramatically changed since the introduction of cART in 1996. However, as HIV-1-related mortality decreases, cardiovascular diseases have become the primary cause of mortality in these patients. Antiretroviral regimes may cause dyslipemia, insulin resistance, and type 2 diabetes mellitus and have been associated to accelerated CAD. In this population, the incidence of CAD is increased 2-fold and the expected number of patients with end-stage heart failure secondary to ischemic heart disease is likely to increase. To our knowledge, ours is the first report of an HIV-infected heart recipient with ischemic cardiomyopathy. From the reported cases, 1 patient had advanced AIDS before HTX and survived only 3.5 years after. The other 5 transplant recipients, similar to our case, were on cART regimes and had undetectable virus load, no AIDS-related manifestations, and uncomplicated post-operative courses. All of these patients suffered non-ischemic dilated cardiomyopathy.

Although the first experiences with cardiac transplantation in very selected HIV-1-infected patients show very encouraging results, further reports and longer follow-ups are needed to elucidate possible HTX candidates when HIV infection coexists.

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**REFERENCES**


**Closure of Ventricular Septal Defect After Surgical Septal Myectomy by Hybrid Procedure in an Adult Patient**

**Cierre de comunicación interventricular tras miectomía septal quirúrgica mediante procedimiento híbrido en un paciente adulto**

To the Editor,

Iatrogenic ventricular septal defect (VSD) is a rare complication following septal myectomy that can cause a rapid postoperative hemodynamic deterioration. We report the case of a 76-year-old dyslipidemic, hypertensive woman who presented with a several-month history of progressive dyspnea. The diagnosis was severe degenerative aortic stenosis, severe hypertrophy of the basal septum (22 mm) with a subaortic gradient of 30 mmHg, and a mild double mitral valve lesion. On the basis of these findings and a EuroSCORE of 6, the decision was made to perform double valve replacement, mitral and aortic, with bioprostheses, as well as septal myectomy.

The postoperative course was indolent and the patient developed complete atrioventricular block that required implantation of a permanent pacemaker. Transthoracic and transesophageal echocardiography (TEE) led to the diagnosis of a VSD measuring approximately 5 mm following myectomy. This defect produced severe pulmonary hypertension (85 mmHg) with moderate-to-severe tricuspid insufficiency and a situation of low output (Fig. 1). Due to the progressive hemodynamic
deterioration and the fact that it was not possible to discontinue inotropic drugs or extubate the patient, the decision was made to close the VSD. Because of the high surgical risk so soon after a recent operation, together with the difficulty associated with a percutaneous intervention, we decided to perform a hybrid procedure. The TEE-guided procedure was carried out in the cardiac operating room. Median sternotomy was performed and, using an 18-gauge needle, a puncture was made through a purse-string suture placed in right ventricular free wall. A short (45 cm) 0.035” guidewire was then introduced through the needle.

Figure 1. A: transgastric view of the ventricular septal defect by transesophageal echocardiography. B: same image with color Doppler ultrasound.

Figure 2. A: passage of the guidewire through the ventricular septal defect following needle puncture of right ventricular free wall. B: position of the introducer after withdrawal of the guidewire. C: placement of the device in the defect with initial release of left disc. D: complete implantation of the device secured to the defect by the discs on either side of the two ventricles.
The guidewire was directed toward the VSD and through it. A 7-Fr introducer with the dilator was passed through the defect with utmost care in order to avoid perforating the left ventricular wall. Once the introducer was in place and the dilator withdrawn, a 6/4 Amplatzer Duct Occluder II (AGA Medical Corporation) was loaded into the delivery device and implanted into the VSD by means of the 7-Fr introducer, in accordance with the standard technique (Fig. 2). The result was satisfactory; there was a minimal residual shunt in the device and, at the end of the procedure, the pulmonary pressure had decreased from 85 mmHg to 40 mmHg. The patient was discharged with no complications.

This hybrid procedure has advantages over surgical closure in that it does not require cardiopulmonary bypass, a circumstance that reduces the risk of myocardial and neurological damage. Another advantage is the continuous monitoring by means of TEE, which enables us to assess and correct, in real time, the position of the device and possible interference with the atrioventricular valves.¹

With respect to percutaneous closure, the hybrid procedure offers several advantages such as the fact that it does not require arterial or venous access. Access to the defect and passage of the guidewire through it is simpler with the transventricular approach because the device penetrates at a right angle and the distance from the penetration site is short. This permits better control during delivery of the device, especially in tortuous VSD and those having irregular borders.¹ The technique employed in isolated percutaneous closure is complex; it requires access to right ventricle from left ventricle, the creation of an arteriovenous loop with the guidewire, and the use of a snare to trap the guidewire and achieve its externalization through the venous side, to ultimately introduce the delivery system and device into the arterial side from the venous side. In all these steps, difficulties often arise which, under conditions of severe hemodynamic deterioration, can prove fatal. Finally, should closure during the hybrid technique prove impossible or if serious complications develop, there always remains the possibility of immediately incorporating a pump and performing surgical closure.

This case illustrates the fact that a rare complication such as VSD following myectomy can be treated easily and successfully by means of a hybrid procedure, especially in high-risk patients. Moreover, it points out the importance of the collaboration between cardiac surgeons and interventional cardiologists, as well as the need for hybrid operating rooms.

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REFERENCE


Recurrent Tako Tsubo Related to Subclinical Hyperthyroidism

Síndrome de tako-tsubo recurrente asociado a hipertiroidismo subclínico

To the Editor,

We report the case of a 53-year-old woman referred to our hospital for a 2-h history of constrictive chest pains. She had no risk factors and no recent emotional stress. She only had a history of undifferentiated nasopharyngeal carcinoma 4 years ago treated by radiation and chemotherapy with total recovery. Clinical examination was normal, with a blood pressure of 129/83 mmHg and blood oxygenation of 98%, except for a sinus tachycardia of 109 beats per minute. The electrocardiogram (ECG) showed an accelerated sinus rhythm with infero-apico-lateral ST-segment elevation. A diagnosis of acute myocardial infarction was made and she was taken to the hemodynamic laboratory. The coronary angiography demonstrated nonobstructive coronary atheroma. The left ventricular angiography confirmed a severe impaired left ventricular systolic function with apical ballooning and elevated left ventricular end diastolic pressure of 32 mmHg (Fig. 1). She was assisted with intra-aortic balloon pump counterpulsation. The 6-h

Figure 1. Left ventriculography (right anterior oblique 30°) showing apical ballooning during systole.