Case Reports

Tricuspid Valve Replacement in an HIV-Infected Patient

with Severe Tricuspid Regurgitation Secondary to Remote Endocarditis

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Surgical intervention for severe tricuspid regurgitation secondary to remote infective endocarditis has been infrequent, especially in patients also infected with the human immunodeficiency virus (HIV). We describe the case of a 62-year-old HIV-positive man, with a 24-year history of endocarditis caused by intravenous heroin use, who presented with severe tricuspid regurgitation. The patient was initially asymptomatic, was taking antiretroviral medications, and had a satisfactory CD4 count and an undetectable viral load, so we decided to manage the regurgitation conservatively. Two years later, he presented with biventricular heart failure and dyspnea. After surgical tricuspid valve replacement, his condition improved substantially. This case illustrates that HIV-infected patients with complex medical conditions can successfully undergo cardiac surgery. ***(Tex Heart Inst J 2016;43(6):514-6)***

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The introduction of antiretroviral medications has changed the course of human immunodeficiency virus (HIV) to that of a chronic disease. Now that HIV-infected patients are living longer, their risk of morbidity and death from cardiovascular disease has increased, and issues such as suitability for surgical intervention have become more important. We describe the case of a patient who underwent surgical intervention despite infection with HIV and years of severe tricuspid regurgitation (TR) caused by remote endocarditis.

Case Report

In 2010, a 62-year-old man was referred for cardiologic evaluation at the St. Luke's and Roosevelt Hospital HIV outpatient clinic. His medical history included atrial fibrillation, HIV infection, hypertension, intravenous heroin use, endocarditis in 1986, past alcohol use (sobriety since 2000), and past tobacco use (cessation in 2006). He reported no chest pain, dyspnea on exertion, or palpitations. His medications included warfarin, atenolol, lisinopril, and aspirin (81 mg), and the antiretroviral agents abacavir, lamivudine, atazanavir, and ritonavir. He had periodically undergone treatment for methadone abuse. On examination, he had an irregular heart rhythm and a systolic murmur ranging from grade 2/6 to 3/6 at the lower sternal border. He had clear lungs and no signs of peripheral edema or congestion.

A transthoracic echocardiogram (TTE) showed a flail anterior tricuspid valve leaflet, a severely dilated right atrium (RA) and right ventricle (RV), a hypokinetic RV, and severe TR (Fig. 1). The inferior vena cava was dilated; left ventricular (LV) dimensions and systolic function were normal. A transesophageal echocardiogram showed a patent foramen ovale and severe TR, with coaptation failure of the tricuspid valve leaflets secondary to complete destruction of that valve. No vegetations were seen. The patient began a Bruce-protocol treadmill test, but he experienced dyspnea and stopped after only 63 seconds. A Holter monitor showed atrial fibrillation with controlled ventricular rate. Cardiac catheterization revealed RA and RV systolic pressures of 24 mmHg each, a V wave of 24 mmHg, and no obstructive coronary artery disease. The severe TR precluded accurate measurement of pulmonary artery pressure.

After discussion with the patient and the cardiothoracic surgeon, we decided to manage the TR conservatively. The patient's poor exercise tolerance during treadmill

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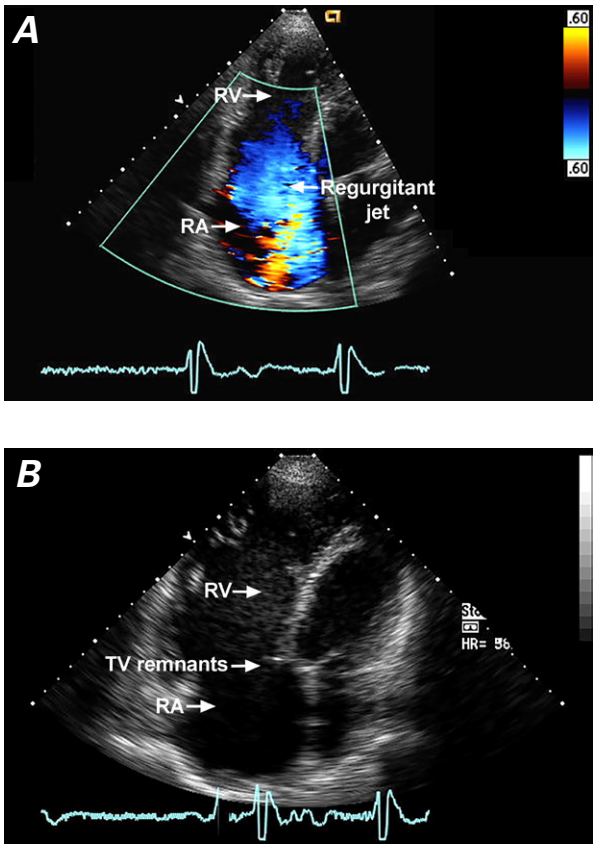

Fig. 1 *Preoperative transthoracic echocardiograms.* ***A****) Four-chamber view (color-flow Doppler mode) shows severe tricuspid regurgitation.* ***B****) Four-chamber view shows tricuspid valve (TV) remnants and marked right ventricular and right atrial dilation.*

RA = right atrium; RV = right ventricle

testing was of concern; however, his condition remained stable for some time. During outpatient visits, he mentioned no symptoms, reported physical activity, and had no signs of fluid overload on physical examination.

In July 2012, the patient, now 64 years old, reported dyspnea at rest. Examination revealed bilateral pedal edema, mild-to-moderate elevation in jugular venous pressure, and crackles in both lung bases. A TTE showed deteriorated LV systolic function (ejection fraction, 0.35) and LV dimensions at upper limits of normal (end-diastolic diameter, 4.1 cm; and end-systolic, 3.3 cm). The RV compressed the LV and deformed the septum consequent to RV pressure overload. Results of cardiac catheterization confirmed the LV ejection fraction of 0.35 and a mean RA pressure of 18 mmHg; the V wave on the RA tracing approximated the RV systolic pressure of 36 mmHg. Because of the patient's severe TR, the catheter could not be advanced into the RV.

The patient's declining functional status and right-sided heart failure caused by TR prompted the decision to replace his tricuspid valve. Of note, the patient's CD4 count was 505 cells/mm^3, and his HIV-1 RNA level was <50 copies/mL (undetectable). A #33 Biocor® porcine valve (St. Jude Medical, Inc.; St. Paul, Minn) was placed; in addition, defibrillation restored sinus rhythm. Because of the large atrial diameter and biventricular failure, a maze procedure was not attempted. The patient recovered uneventfully and was discharged from the hospital on postoperative day 6. At a clinic visit in September 2012, he had no signs of congestion and said that he had climbed 6 flights of stairs without chest pain or dyspnea.

In November 2012, a TTE showed good function of the prosthesis and no significant regurgitation or stenosis (Fig. 2). The patient's LV ejection fraction was 0.60, and his pulmonary artery systolic pressure was normal. The RV was somewhat less dilated and remained severely hypokinetic. The patient was clinically monitored; in August 2013, he completed over 5 min of a Bruce protocol test. The mean gradient across the tricuspid valve was 3 mmHg. The patient considered himself to be cured and was eventually lost to follow-up.

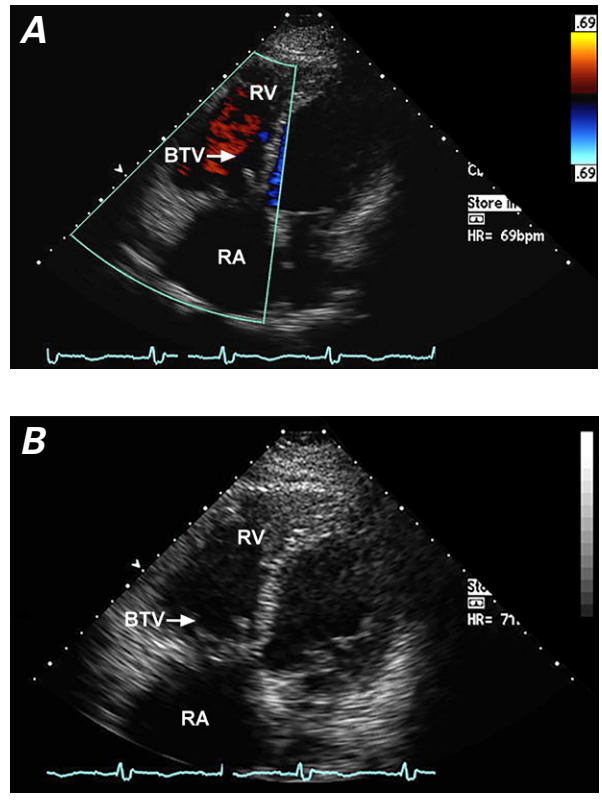

Fig. 2 *Postoperative transthoracic echocardiograms.* ***A****) Four-chamber view (color-flow Doppler mode) shows the bioprosthetic tricuspid valve (BTV) and less right atrial and right ventricular dilation.* ***B****) Four-chamber view shows improved right ventricular function.*

RA = right atrium; RV = right ventricle

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Discussion

Intervention for symptomatic, severe TR is a class IIa recommendation in the 2014 and 2006 American Heart Association/American College of Cardiology guidelines for the management of valvular disease.[1,2] Nevertheless, little information is available about intervention when long-standing primary TR is caused by endocarditis. In a retrospective study of 33 patients who underwent surgical repair or replacement for isolated tricuspid valve endocarditis,[3] most did well and had improved functional status. Fourteen of the 33 patients had used intravenous drugs. The authors gave no information about HIV status, when endocarditis was first diagnosed, or duration of TR.[3]

Antiretroviral therapy can lengthen HIV-infected patients' lifespans to ages when cardiovascular disease is more prevalent. However, options for surgical treatment (coronary artery bypass grafting and valve surgery) were often thought to be limited by the HIV infection. Patients with virologic suppression and satisfactory CD4 counts are now being considered as operative candidates. Using data from the Nationwide Inpatient Sample of patients infected with HIV from 2000 to 2010, Polanco and colleagues[4] showed that cardiac surgeries in this population doubled from 0.1% to 0.2% (P <0.001), with 1,239 cases during the study period. Of the operations, 36% were for valve surgery; however, they did not report the number of tricuspid valve surgeries. Endocarditis caused the valvular abnormality in 21.3% of cases. The mortality rate in valve operations was 4.7% overall, 1.3% in patients without active disease or HIV-related symptoms, and 7.7% in patients with acquired immune deficiency syndrome or therapy for HIV-related disease.[4] Other data from the same database revealed that HIV-infected patients were not at increased risk of inpatient death but were more likely to receive blood transfusions and have postoperative complications.[5] In 308 HIV-infected patients who underwent various surgical procedures in China, 196 had postoperative infectious complications, and low CD4 count was associated with infection development.[6] To our knowledge, no authors have defined a lower-cutoff or optimal CD4 count for HIV-infected patients in whom cardiac surgery is contemplated.

Despite our patient's poor exercise tolerance, he initially reported no symptoms. In addition, because of sparse evidence to support tricuspid valve replacement in an HIV-infected patient, we decided not to perform surgery immediately. When his clinical status and LV systolic function deteriorated, we replaced his tricuspid valve, which resulted in marked improvement.

This patient might not have been considered for surgical intervention in previous years. As more patients living with HIV successfully undergo cardiac and noncardiac surgeries, further research will help to identify characteristics that might predict good outcomes.

Acknowledgments

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