Case Report

Stenting of a Stenotic Aortic Homograft in Pulmonary Position After the Ross Procedure

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Pulmonary autograft replacement of the aortic valve (Ross procedure) has potential advantages, with favorable rates of survival and freedom from reoperation. The procedure itself, however, involves insertion of a homograft in the pulmonary position. The development of severe homograft stenosis is an uncommon but clinically important complication. We report the case of a young female who developed a symptomatic homograft stenosis a year after she underwent the Ross procedure. The lesion was stented successfully and the homograft's patency, together with a markedly improved pressure gradient, was still maintained eight months after percutaneous stenting.

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The challenge to provide a suitable replacement for a diseased human heart valve has led to an ongoing process. The pulmonary autograft has emerged as an attractive alternative substitute for aortic valve replacement, with promising hemodynamic and clinical results.[1] The autograft is non-thrombogenic, thus not requiring anticoagulation, and the risk of endocarditis is low. Moreover, in children and adolescents there is evidence that the autograft grows in parallel with the patient. This surgical method was originally introduced in 1967 by Donald Ross, who implanted a pulmonary autograft in the subcoronary position.[2] The Ross procedure entails replacing the transplanted pulmonary valve and adjacent main pulmonary artery with a valved homograft conduit to reestablish right ventricular-pulmonary artery continuity. The long-term function of cryopreserved homograft conduits determines late morbidity and the need for reoperation. Evolving homograft stenosis complicating the Ross procedure is a rare problem with significant clinical consequences. Keeping in mind the well known advantages of the percutaneous procedure, stenting of a stenosed homograft instead of reoperation is a real challenge.

Case presentation

A 30-year-old female attended her follow-up appointment nine months after she had undergone the Ross procedure because of severe congenital aortic stenosis (bicuspid valve). A cryopreserved aortic homograft was used to replace the pulmonary valve and adjacent main pulmonary artery after the replacement of the diseased aortic valve by a pulmonary autograft. Six months after the operation she started complaining of worsening dyspnea, palpitations and fatigue. On the echocardiogram an increased systolic pressure gradient of 35 mmHg was first noticed at that time.

On physical examination a right ventricular heave was easily palpated and a loud (5/6) systolic murmur was audible at the left upper sternal border. These findings were definitely absent before and right after the Ross procedure. Her electrocar-

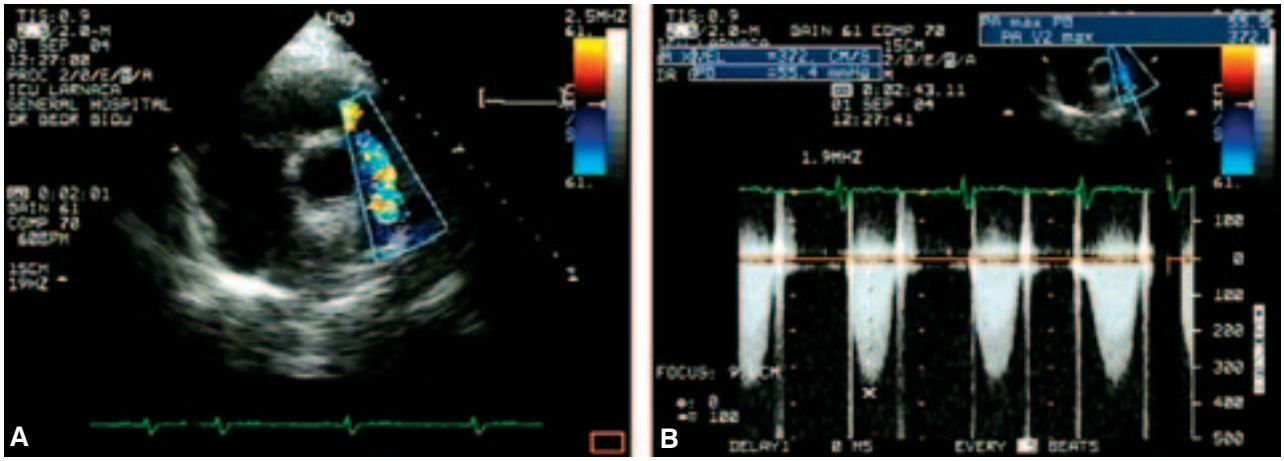

Figure 1. Transthoracic echocardiogram color-2D (A) and continuous-wave Doppler (B) in short axis parasternal view showing turbulent flow within the tubular part of the homograft and significantly increased flow velocity, which was compatible with a peak systolic gradient of 55 mmHg.

diogram fulfilled the criteria for right ventricular hypertrophy with right axis deviation.

The transthoracic echocardiogram revealed turbulent flow within the homograft. Continuous-wave Doppler (CWD) detected a significantly increased flow velocity with a maximal systolic pressure gradient of 55 mmHg (Figure 1). The right heart cavities were mildly dilated and moderate tricuspid regurgitation was detected. The subcostal view showed right ventricular hypertrophy (right ventricular thickness during diastole was 10 mm).

Magnetic resonance angiography (MRA) confirmed the diagnosis of significant homograft stenosis. The narrowing was mainly due to a 70% reduction in luminal diameter of the tubular part of the homograft.

The patient was referred for right heart catheterization and angiography. This showed a significant stenosis of the tubular part of the homograft, with a pull-back systolic gradient of 55 mmHg between the bifurcation of the main pulmonary artery and the valve of the homograft. Direct stenting of the grafted main pulmonary artery was performed with a satisfactory final angiographic result (Figures 2 and 3). A combination of aspirin (150 mg daily) and Plavix (75 mg daily) was

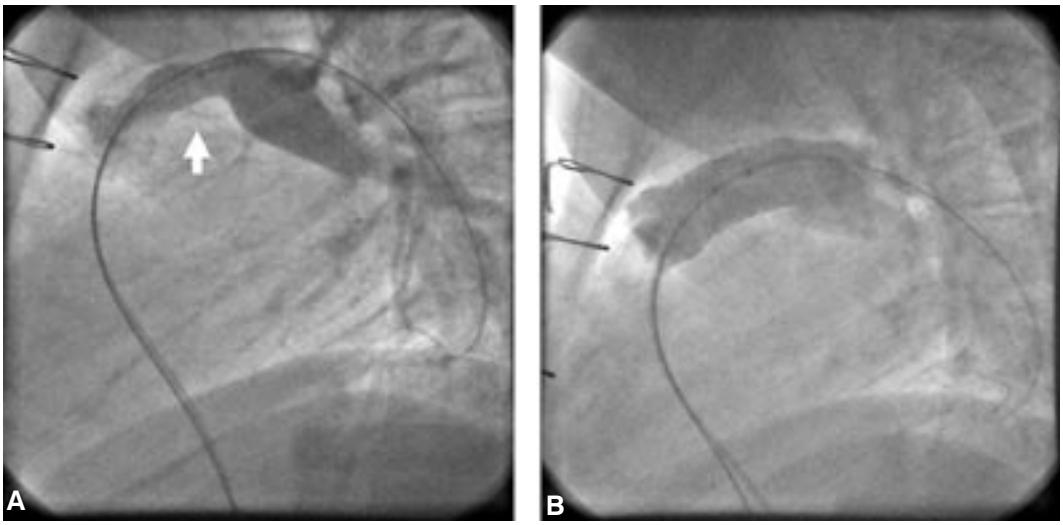

Figure 2. Angiography (LAO 90°, Caudal 0°) of the aortic homograft in pulmonary position before stenting (A) revealing significant stenosis of the tubular part of the homograft (white arrow), and after stenting (B) with obvious patency of the homograft.

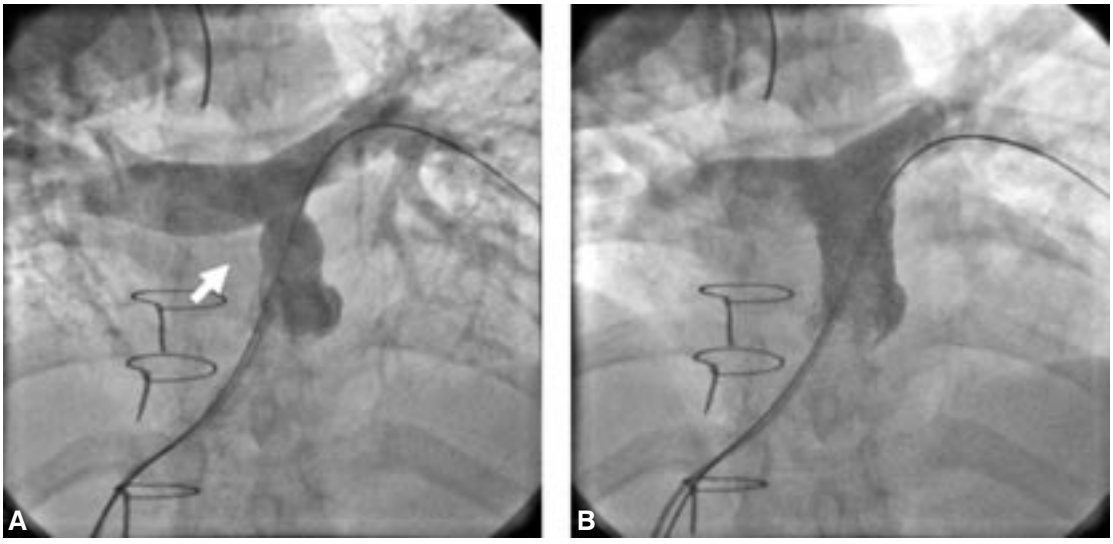

Figure 3. Angiography (LAO 0°, Cranial 30°) before (A) and after stenting of the homograft (B).

used for one month after stenting, followed by continuous administration of aspirin.

A series of echocardiograms performed three, six and nine months after stent implantation confirmed the patency of the homograft; the maximal systolic pressure gradient with CWD had decreased to 27 mmHg (Figure 4). Mild to moderate (2+/4) pulmonary incompetence was also noticed. The patient's symptoms were markedly improved.

Discussion

The Ross procedure provides continued excellent hemodynamics in the aortic position, and from a hemodynamic standpoint, it is possibly the ideal operation for this valve.[1] By the mid-1980s, cryopreserved homografts were considered by many surgeons to be the valve conduit of choice for replacement of the pulmonary valve and adjacent artery, because they have technical and hemodynamic advantages over alternative prosthetic valved conduits.[3,4] As the Ross procedure has gained popularity, the use of the pulmonary homograft to replace the pulmonary valve in this patient population has also risen. The pulmonary homograft appears to be more durable than the aortic homograft, which was used in our case in the pulmonary

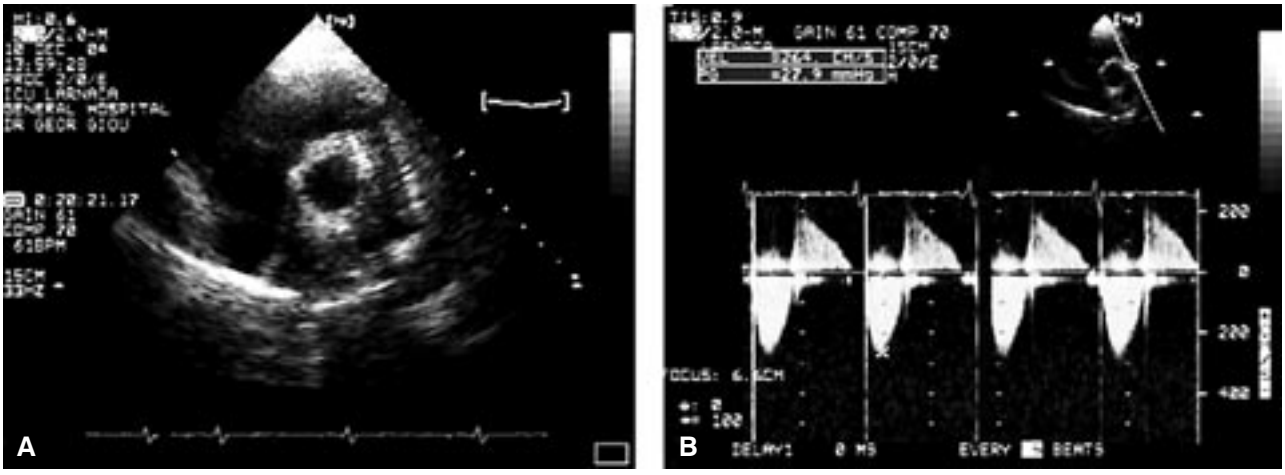

Figure 4. Transthoracic echocardiogram 2D- (A) and continuous-wave Doppler(B) in short axis parasternal view after stenting of the homograft, confirming significant improvement in the homograft's lumen and an impressive decrease in peak systolic gradient across it.

position. One study[5] showed that freedom from homograft dysfunction at 8 years was 80% for pulmonary homografts versus 56% for aortic homografts.

Carr-White et al demonstrated that in patients who undergo the Ross procedure, evolving pulmonary homograft stenosis is an uncommon but clinically important problem.[6] Freedom from any pulmonary homograft stenosis at 7-year follow-up was 79.7%, with instantaneous hazard falling to zero after 4 years. Freedom from reoperation at 7 years was 96.7%. Multivariate analysis of patient-, surgery-, and homograft-related variables did not reveal any significant risk factors for development of pulmonary homograft stenosis. The operative and histological findings, in combination with MRA images, suggested that the underlying mechanism of homograft failure was predominantly inflammatory in nature, as granulomatous tissue was seen encircling the homograft. This reaction may be immunologically mediated in a manner similar to that seen in chronic reaction and leads to extrinsic compression and/or shrinkage.[7] Carr-White et al highlighted the fact that follow up of pulmonary homograft function is critically important in the first two years after surgery, and where possible, those with echocardiographic gradients should undergo MRA.[6]

Experience in stenting of stenosed homografts is limited worldwide.[8,9] Despite this we referred our patient for a percutaneous intervention, recognizing the well-known advantages of this non-surgical interventional procedure in treating lesions of other major arteries and respecting the patient's will. There is no experience in stenting of the valvar part of calcified and fibrotic homografts. As the risk of major complications, including pulmonary artery perforation and severe pulmonary valve incompetence post stenting is high, reoperation remains the treatment of choice regarding this type of old and severely diseased valved homograft.

Taking into consideration the low flow across the pulmonary artery and the risk of stent thrombosis after stenting of other major arteries, a dual antiplatelet therapy consisting of aspirin and clopidogrel was recommended for at least one month after stenting of the homograft.

In conclusion, stenosis of the homograft can be a significant problem following the Ross procedure. The predominant mechanism is inflammatory, although the precise details are unclear. Follow up of homograft function is critically important in the first two years after surgery. Direct stenting of the homograft lesion instead of reoperation is an attractive alternative but the long-term results are still unpredictable.

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