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Case Report

Severe Aortic Stenosis and Proximal Left Anterior Descending Artery Stenosis: A Positron Emission Tomography/Computed Tomography Assessment Before and After Transcatheter Aortic Valve Implantation

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ABSTRACT

We report the case of an 82-year-old patient with symptomatic severe aortic stenosis and calcified proximal left anterior descending (LAD) artery stenosis who underwent a transfemoral transcatheter aortic valve implantation (TAVI) without complex percutaneous coronary intervention. Before TAVI, a positron emission tomography/computed tomography assessment confirmed a reduced global coronary flow reserve (CFR), more pronounced on the LAD territory. One month post-TAVI, a second positron emission tomography/computed tomography scan showed a normalization of the global CFR and more than a doubling in the LAD territory. This case illustrates that mechanisms other than vessel stenosis may play an important role in CFR in the setting of aortic stenosis.

RÉSUMÉ

Nous rapportons le cas d'un patient âgé de 82 ans, présentant une sténose aortique sévère symptomatique et une sténose calcifiée de la portion proximale de l'artère interventriculaire antérieure, qui a bénéficié d'une implantation valvulaire aortique par cathéter (IVAC) par voie transfémorale sans intervention coronarienne percutanée complexe. Avant l'IVAC, une tomographie par émission de positons/tomodensitométrie a confirmé une réduction de la réserve coronaire globale, réduction plus prononcée dans la région de l'artère interventriculaire antérieure. Un mois après l'IVAC, une seconde tomographie par émission de positons/tomodensitométrie a permis d'observer une normalisation de la réserve coronaire globale, qui avait plus que doublé dans la région de l'artère interventriculaire antérieure. Ce cas illustre le fait que dans les cas de sténose aortique, des mécanismes autres que la sténose vasculaire peuvent influer de façon importante la réserve coronaire.

Case Description

An 82-year-old diabetic patient, with prior percutaneous coronary intervention (PCI) on the right coronary artery (RCA) and left circumflex (LCX) artery, was admitted for New York Heart Association class II dyspnea and chest pain on exertion. A transthoracic echocardiogram showed severe aortic stenosis (AS) (mean gradient: 58 mm Hg, maximum velocity: 4.9 m/s, valve area: 0.6 cm^2, left ventricular ejection fraction 55%). Coronary angiogram revealed a 70% severely calcified proximal left anterior descending (LAD) artery lesion (Fig. 1) with a functional assessment by fractional flow reserve (FFR) at 0.83 just below the lesion and 0.77 distally in the vessel after injection of intracoronary adenosine (180 μg). A regadenoson stress test and resting ^{13}N-ammonia positron emission tomography/computed tomography (PET/CT) scan confirmed a reduced global coronary flow reserve (CFR), defined as the hyperemic to resting myocardial blood flow ratio, more pronounced on the LAD territory (global 0.99; LAD territory 0.87; LCX 1.17; RCA 1.18) (Fig. 2A).

After a discussion with the heart team, we decided to perform a transfemoral transcatheter aortic valve implantation (TAVI) without the complex PCI involving proximal LAD rotablation, nonetheless keeping in mind that post-TAVI PCI could still be performed, although probably more challenging.

The implantation of a 26-mm Evolut-R prosthesis (Medtronic, Minneapolis, MN) was uneventful. Discharge transthoracic echocardiogram revealed trace paravalvular leak and a mean gradient of 6 mm Hg. At 1 month, a second ^{13}N-ammonia PET/CT scan (same vasodilator used) showed a normalization of the global CFR and more than a doubling in

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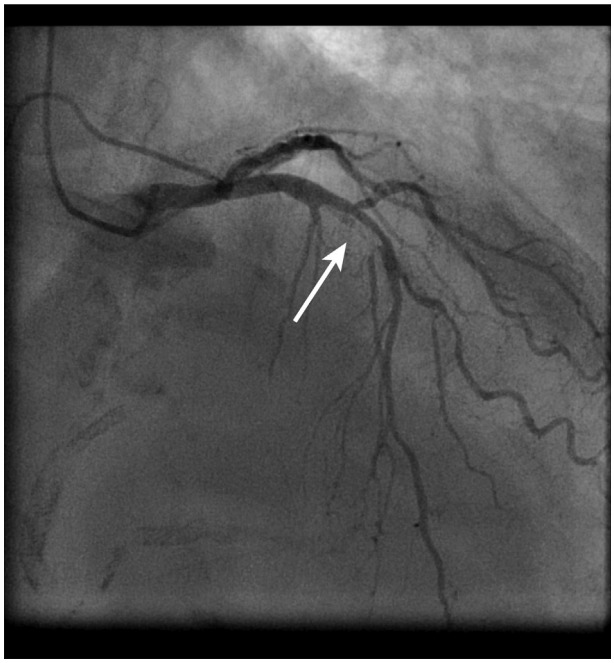

Figure 1. Coronary angiogram showing a 70% severely calcified proximal left anterior descending (LAD) artery lesion (**arrow**).

the LAD territory (global: 3.25, LAD territory: 2.71, LCX: 4.42, RCA: 3.29) (Fig. 2B).

At 1 year, the patient was in New York Heart Association class I and angina free with a normal and slightly improved left ventricular ejection fraction of 65%. There was no paravalvular leak, and the mean gradient was 5 mm Hg.

Discussion

The AS degenerative process is similar to arterial atherosclerosis. Not surprisingly, AS is commonly associated with coronary artery disease (CAD). In the UK TAVI registry, 45.7% of the patients had concomitant CAD (1-vessel disease in 21.1%, 2-vessel disease in 11.5%, and 3-vessel disease in 13.2%). In the randomized **Su**rgical **R**eplacement and **T**ranscatheter **A**ortic **V**alve **I**mplantation (SURTAVI) trial including intermediate-risk patients, 20% of them required PCI before TAVI.

The **F**ractional Flow Reserve vs **A**ngiography for **M**ultivessel **E**valuation (FAME) trials have demonstrated the benefit of FFR to improve clinical outcomes in patients with stable CAD. Nonetheless, patients with severe AS were excluded. Pesarini et al.[1] studied FFR changes immediately after TAVI and found only marginal changes that did not change the indication for treatment in 94% of lesions, although all

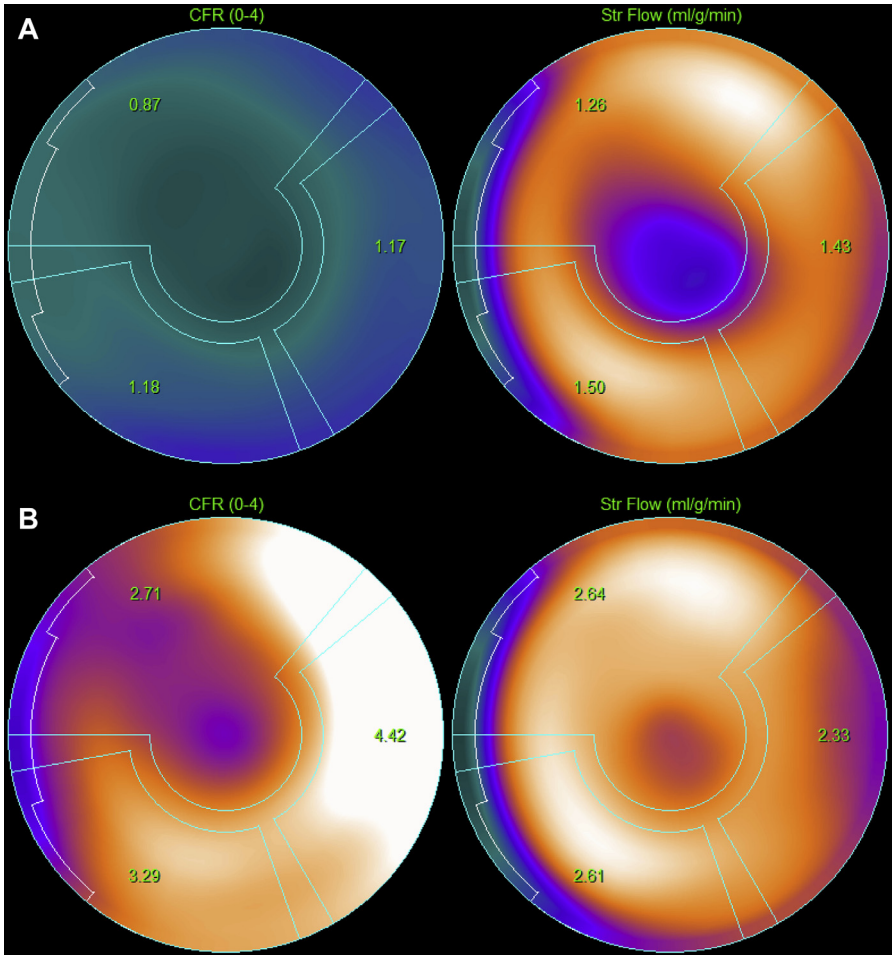

Figure 2. Coronary blood flow assessed by positron emission tomography/computed tomography **(**PET/CT): coronary flow reserve (to the left) and hyperemic stress blood flow (to the right) before transcatheter aortic valve implantation (TAVI) (**A**) and after TAVI (**B**).

significant lesions worsened and all nonsignificant lesions improved after TAVI.[1] Likewise, Ahmad et al.[2] found an overall significant decrease of FFR immediately after TAVI, whereas instantaneous wave-free ratio values remained unchanged, offering a more robust afterload-independent assessment.[2] An abrupt increase of the systolic component of the coronary flow after TAVI, due to increased aortic root flow and decreased left ventricular systolic pressure, was postulated as the main reason behind FFR (systolic-diastolic parameter) and instantaneous wave-free ratio (diastolic parameter) discrepancies.

On the other hand, myocardial stress PET/CT is considered to be the most accurate noninvasive method to assess ischemia and has a prognostic value in predicting adverse outcomes.[3] By means of rest-hyperemic myocardial PET/CT studies, Rajappan et al.[4] demonstrated that CFR is impaired in patients with AS with angiographically normal coronary arteries and attributed this finding to the pressure overload and the extra-vessel compression rather than the structural changes (remodeling) of the intramural coronary arteries and small vessels.[4] The same authors demonstrated the increase of CFR after surgical aortic valve replacement in patients without CAD. Of note, the CFR increase was better correlated with the aortic valve area and the diastolic perfusion time rather than left ventricular hypertrophy, further supporting the evidence of subendocardial mechanical compression.[5]

Conclusion

In alignment with these studies, our patient exhibited a decreased global CFR before TAVI that was more pronounced in the stenotic vessel territory and was subsequently increased globally and in the LAD territory. This case illustrates that mechanisms other than vessel stenosis may play an important role in CFR in the setting of AS.

Disclosures

The authors have no conflicts of interest to disclose.

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