Coronary Obstruction During Transcatheter Aortic Valve Replacement: Related to Calcification or Thrombus?

Obstrucción coronaria durante el implante percutáneo de válvula aórtica: ¿relacionada con calcificación o con trombo?

To the Editor,

We read with great interest the recently published article by Mori Junco et al. This is a very interesting case successfully treated with thrombus aspiration and balloon dilatation. However, some issues require further discussion.

Although coronary thromboembolism during transcatheter aortic valve replacement (TAVI) is a rarely encountered complication, it may lead to life-threatening problems particularly due to acute myocardial infarction. The optimal treatment is percutaneous coronary intervention with angioplasty and stenting if necessary. Percutaneous coronary intervention may be a lifesaving strategy, especially in thromboembolism at the proximal coronary arterial tree. In the present case, a 74-year-old man had an acute myocardial infarction due to distal left main coronary thromboembolism during TAVI. TAVI is an alternative and effective treatment in patients with severe symptomatic aortic stenosis and high surgical risk.2,3 Even though TAVI is a less invasive treatment than surgical aortic valve replacement, some serious complications may develop. Some of these are aortic rupture,4,5 early aortic valve thrombosis, and peripheral and neurological embolization of aortic valve calcium following TAVI. In the present case, the 74-year-old man had a predisposition to thrombus formation related to hypertension, type 2 diabetes mellitus, diabetic nephropathy, and chronic alcoholic liver disease. Moreover, the authors state that 2 drug eluting stents had previously been implanted in the left anterior descending artery. Regarding drug eluting stent implantation, we wonder whether or not the patient took dual antiplatelet therapy and unfractionated heparin before the TAVI procedure. We strongly believe that thrombus to the distal left main artery may be closely related to antiplatelet and anticoagulant treatment in the present case.

On the other hand, calcifications extending to the ascending aorta or left ventricular outflow tract may lead to some complications such as systemic embolism, aortic annulus rupture, or coronary embolism. In addition, a prosthetic valve during the implantation procedure may also damage and obstruct the ostium of the left main artery.6–9 Lastly, laser aortic annulus and aortic root may be damaged iatrogenically at various levels during balloon inflation and valve implantation. Therefore, the landing zone of the valve and the direction of the annular calcification are very important to predict these complications.

Besides thrombus formation, residual calcification related to the natural valve is also a potential etiologic factor for embolism. Because of the potential catastrophic complications of TAVI,
patients should be closely followed-up by echocardiography during and after the procedure. On echocardiography, detection of thrombus formation on the implanted aortic valve is essential after TAVI and interventional cardiologist should promptly start antplatelet and antithrombotic treatment after TAVI in those patients.

In conclusion, it is very important to be aware of this catastrophic event and all interventional cardiologists should take all preventive actions such as placing a guiding catheter in the left coronary ostium.

Cengiz Ozturk,* Ali Osman Yildirim, Mustafa Demir, and Sevket Balta

Department of Cardiology, Çalhane Military Medical Academy,
School of Medicine, Etlik-Ankara, Turkey

* Corresponding author:
E-mail address: drcengizozturk@yahoo.com.tr (C. Ozturk).
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Obstrucción coronaria durante el implante percutáneo de válvula aórtica: ¿relacionada con calcificación o con trombo? Respuesta

To the Editor,

We appreciate the comments of Ozturk et al on our publication entitled, “Coronary Thromboembolism During Transcatheter Aortic Valve Replacement”.

The case in question concerned a patient with high thromboembolic risk who received 2 drug-eluting stents in the anterior descending artery during coronary angiography 1 month before the valve replacement procedure. The patient received dual antiplatelet therapy after the angioplasty and, later, unfractionated heparin during the transcatheter aortic valve implantation to maintain an activated clotting time between 200 and 250 s. Thus, the patient received complete antithrombotic therapy despite a high risk of bleeding (hypertension, diabetic nephropathy, and chronic alcoholic liver disease). We do not know if there were fluctuations in the anticoagulation level during the procedure. After the implant, the patient continued to receive dual antiplatelet therapy. Although coronary thromboembolic complications during aortic valve implantation are infrequent (around 1%), they can be catastrophic and thus require urgent treatment.

Calcifications in the left ventricular outflow tract, the native aortic valve itself, and the ascending aorta can lead to certain complications. 1–6 Another possible complication to bear in mind is coronary embolism of calcified material. In our patient, the presence of an intracoronary thrombus was revealed using a thromboaspiration device. Because this device is designed to remove intravascular thrombi, it helped to achieve optimal myocardial perfusion.

Cases such as this one reinforce the need for adequate procedure-related antithrombotic therapy, in addition to transesophageal echocardiography monitoring to permit early detection of complications requiring urgent management, as well as for other uses.

Ricardo Mori Junco,a,b Francisco Domínguez Melcón,a,b and Mar Moreno Yangüela,a

aServicio de Cardiología. Hospital Universitario La Paz, Madrid, Spain
bUnidad de Imagen Cardíaca, Servicio de Cardiología, Hospital Universitario La Paz, Madrid, Spain

*Corresponding author:
E-mail address: ricardomori22@gmail.com (R. Mori Junco).
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