REFERENCES

1. Scirica BM, Braunwald E, Raz I, et al. Heart failure, saxagliptin, and diabetes mellitus: observations from the SAVOR-TIMI 53 randomized trial. *Circulation.* 2015;132:e198.
2. Galve E, Cordero A, Bertomeu-Martínez V, et al. Update in cardiology: vascular risk and cardiac rehabilitation. *Rev Esp Cardiol.* 2015;68:136–143.
3. Cebrián-Cuenca AM, Orozco-Beltrán D, Navarro-Pérez J, et al. Saxagliptin and risk of heart failure hospitalization: Concern or miscalculation? *Int J Cardiol.* 2016;220:573–574.
4. Fu AZ, Johnston SS, Ghannam A, et al. Association between hospitalization for heart failure and dipeptidyl peptidase 4 inhibitors in patients with type 2 diabetes: an observational study. *Diabetes Care.* 2016;39:726–734.
5. Raschi E, Poluzzi E, Koci A, et al. Dipeptidyl peptidase-4 inhibitors and heart failure: Analysis of spontaneous reports submitted to the FDA Adverse Event Reporting System. *Nutr Metab Cardiovasc Dis.* 2016;26:380–386.
6. Ikeda J, Kimoto N, Kitayama T, et al. Cardiac DPP-4 inhibition by saxagliptin ameliorates isoproterenol-induced myocardial remodeling and cardiac diastolic dysfunction in rats. *J Pharmacol Sci.* 2016;132:65–70.

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Percutaneous Mitral Repair With MitraClip in Patients Treated With Transcatheter Aortic Valve Implantation

Reparación mitral percutánea con MitraClip en pacientes tratados con implante percutáneo de válvula aórtica

To the Editor,

Up to 40% of patients treated with transcatheter aortic valve implantation (TAVI) have at least moderate mitral regurgitation (severe in 15.9%), and its persistence after prosthesis implantation (7.9% of patients) negatively affects prognosis.[1] In almost half of the patients, mitral regurgitation is reduced, mainly in patients with functional etiology, nondilated annulus, and noncalcified valves.[1,2] If the regurgitation is not reduced and the patient continues to have limiting symptoms, one option that has been proposed is the use of percutaneous repair techniques with the MitraClip device; there are already published case series of solution in Europe.[3] In Spain, the use of the MitraClip has become more widespread since 2011, mainly for patients with functional mitral regurgitation.[4,5] The technique requires adequate mitral valve anatomy to allow MitraClip implantation, although it has been proposed that the selection criteria be relaxed in centers with experience.[6] In patients with TAVI, the mitral annulus and/or mitral leaflets are often calcified, which can limit the treatment indication, and therefore the valve must be assessed in detail.

This study combines the experience of the first 5 cases of severe mitral regurgitation after percutaneous treatment with TAVI performed in 3 hospitals in Spain (2015 to 2016), with particular emphasis on patient selection.

Below we present the characteristics at baseline, those related to the previous TAVI, to MitraClip implantation, and at follow-up. Mitral regurgitation was present prior to TAVI in all 5 patients and persisted without reduction until MitraClip implantation, an average of 16.2 months later.

In patients 1, 2, and 5, and particularly in 5, the valve had abnormalities (degenerative etiology), which the clinician must take into account when deciding whether the procedure is indicated:

- The free edge of the anterior mitral valve must have at least 1 free 7-mm segment[6] that does not interfere with the TAVI. The presence of a previous dysfunctional bioprosthesis treated with TAVI (valve-in-valve), as in patient 1, did not present anatomical obstacles to treatment with MitraClip. Similarly, patient 3 had a double TAVI valve (both implanted in the same procedure because the first was too deep at 13 mm), although this did not interfere with the MitraClip device. The average depth of the valves was 7.25 mm.
- Calcification and flexibility of leaflets: in patients with calcified degenerative aortic stenosis, the calcification often extends to the posterior mitral annulus and to the "mitral-aortic curtain", as in patient 5 (Figure). Once again, at least 7 mm of noncalcified tissue is required in each of the valves to allow device implantation. In patient 5, the regurgitation jet originated on either side of the calcified area, and it was possible to implant the devices avoiding

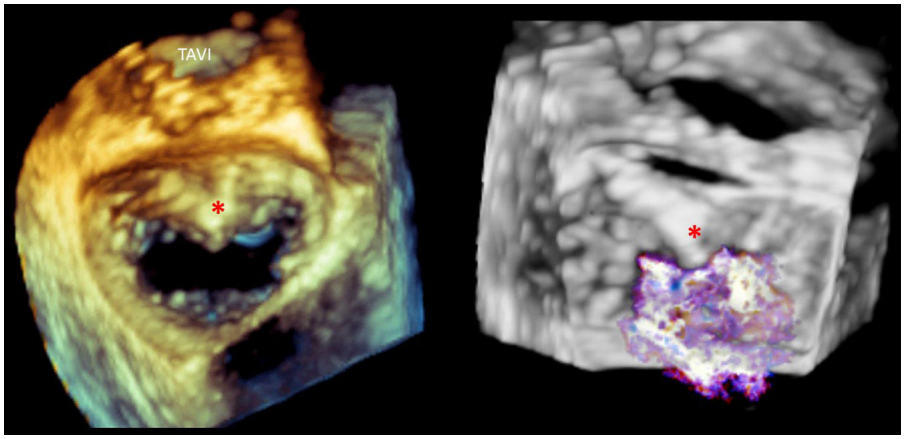

Figure. Three-dimensional transesophageal echocardiogram with a view of the mitral valve from the left atrium in patient 5. The asterisk indicates an area of calcification along the mitral-aortic curtain. The regurgitation originates mainly on either side of the calcified area. TAVI, transcatheter aortic valve implantation.

this (Figure). The presence of calcified areas is only compatible with treatment with MitraClip if there is a good valve area in general (> 4 cm^2) and the rest of the valve is sufficiently flexible.
- Valve area: ideally, the available area should be > 4 cm^2, although a smaller area can be tolerated (up to 3 cm^2) provided the valves are flexible and not thickened or calcified. In the patient series presented, the average valve area was 4.14 cm^2, and it was possible to implant 2 MitraClip devices without producing stenosis in 4 of the 5 patients.

The outcomes of MitraClip implantation were successful in both reducing mitral regurgitation and producing clinical improvement; mean follow-up was 9.6 months. All patients had an initial improvement in functional class to at least grade II, although at follow-up, 3 patients with severe ventricular dysfunction had deteriorated in functional status. In one of them, mitral regurgitation progressed to grade II-III.

In conclusion, MitraClip may be an option for patients with mitral regurgitation after TAVI, although complex valvular anatomies can be expected; therefore, it is advisable to perform the treatment in centers with a high patient volume. Registries are needed to define the clinical profile of the patients that may benefit most from this treatment.

CONFLICTS OF INTEREST

F. Carrasco-Chinchilla, R. Estévez-Loureiro, and X. Freixa declare a possible conflict of interest due to collaboration with Abbott vascular.

Fernando Carrasco-Chinchilla,[a,*] Rodrigo Estévez-Loureiro,[b] Leire Andraka,[c] Dabit Arzamendi,[d] Xavier Freixa,[e] and José Suárez de Lezo[f]

[a]*Departamento de Hemodinámica e Imagen Cardiaca, Hospital Virgen de la Victoria, Málaga, Spain*

[b]*Departamento de Hemodinámica, Complejo Asistencial Universitario de León, León, Spain*

[c]*Departamento de Hemodinámica, Hospital de Basurto, Bilbao, Vizcaya, Spain*

[d]*Departamento de Hemodinámica, Hospital de Sant Pau i la Santa Creu, Barcelona, Spain*

[e]*Departamento de Hemodinámica, Hospital Clínic, Barcelona, Spain*

[f]*Departamento de Hemodinámica, Hospital Reina Sofía, Córdoba, Spain*

[*] Corresponding author:
E-mail address: fernandocarrascochinchilla@gmail.com
(F. Carrasco-Chinchilla).

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REFERENCES

1. Cortés C, Amat-Santos IJ, Nombela-Franco L, et al. Mitral regurgitation aftertranscatheter aortic valve replacement: prognosis, imaging predictors, and potential management. *JACC Cardiovasc Interv.* 2016;9:1603–1614.
2. Hekimian G, Detaint D, Messika-Zeitoun D, et al. Mitral regurgitation in patients referred for transcatheter aortic valve implantation using the Edwards-SAPIEN prosthesis: mechanisms and early postprocedural changes. *J Am Soc Echocardiogr.* 2012;25:160–165.
3. Kische S, D'Ancona G, Paranskaya L, et al. Staged total percutaneous treatment of aortic valve pathology and mitral regurgitation: institutional experience. *Catheter Cardiovasc Interv.* 2013;82:E552–E563.
4. Carrasco-Chinchilla F, Arzamendi D, Romero M, et al. Initial experience of percutaneous treatment of mitral regurgitation with Mitraclip® therapy in Spain. *Rev Esp Cardiol.* 2014;67:1007–1012.
5. Estévez-Loureiro R, Arzamendi D, Carrasco-Chinchilla F, et al. Usefulness of MitraClip for the treatment of mitral regurgitation secondary to failed surgical annuloplasty. *Rev Esp Cardiol.* 2016;69:446–448.
6. Li C-H, Arzamendi D, Carreras F. Papel de las técnicas de imagen en el tratamiento percutáneo de la insuficiencia mitral. *Rev Esp Cardiol.* 2016;69:421–436.

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Impact of Autoimmune Disease on the Management and Prognosis of Acute Coronary Syndrome

Impacto de las enfermedades autoinmunitarias en el tratamiento y el pronóstico del síndrome coronario agudo

To the Editor,

Patients with autoimmune disease have worse short-term prognosis after acute coronary syndrome (ACS).[1–3] Studies are needed in Spain to analyze the possible reasons for this and to determine long-term prognosis after discharge from hospital.

This was a retrospective observational study of patients admitted to a tertiary hospital for ACS between January 2011 and February 2016. The study was conducted according to the tenets of the Declaration of Helsinki and was approved by the ethics committee of the hospital.

The primary objective was to determine the prognostic influence of autoimmune disease on all-cause death, type 3 to 5 major bleeding according to the Bleeding Academic Research Consortium classification,[4] and a composite endpoint of nonfatal acute myocardial infarction and stroke. The primary objective was assessed in patients who were alive after at least 1 year of follow-up (n = 1742). Events were recorded by telephone contact or extracted from medical records. The secondary objective was to determine the characteristics, presentation, and treatment of ACS in patients with and without autoimmune disease. For this objective, the overall population was analyzed (n = 2236).

The effect of autoimmune disease was calculated by Cox regression with adjustment for age, diabetes mellitus, atrial fibrillation, peripheral vascular disease, cerebrovascular disease, neoplasms, chronic obstructive pulmonary disease, Killip score ≥ 2 on admission, heart rate, systolic blood pressure, hemoglobin, troponin T, glomerular filtration rate, left main coronary artery disease and/or 3-vessel coronary artery disease, and ventricular function. The cumulative incidence of events was estimated with the Kaplan-Meier method and was compared using the log-rank test.

Among patients with ACS, 74 had autoimmune disease (prevalence of 3.3%). Of these, the most prevalent were rheumatoid arthritis (24 patients), spondyloarthritis (14 patients), and inflammatory bowel disease (10 patients). The median duration of autoimmune disease was 14 years (interquartile range, 4-14 years). Seventy percent of the patients were receiving corticosteroid treatment, 50% disease-modifying therapy/immunosuppressants, 22% anti-inflammatory agents, and 8% biological therapy.

There was a higher prevalence of atrial fibrillation and obstructive pulmonary disease in patients with autoimmune