(Table 2). LV diastolic function estimated by transmitral E/A ratio was significantly impaired in diabetic patients compared with controls (Table 2).

Two- and 3D LV longitudinal mechanical function was increased in the controls compared with the other 2 groups. Two- and 3D LV circumferential, together with 2D LA longitudinal strain were decreased in the diabetic patients compared with controls. Two-dimensional LV radial strain did not differ between the 3 groups, whereas 3D LV radial strain was lower in diabetic patients than in controls (Table 2). Three-dimensional LV area strain was lower in the prediabetic and diabetic patients than in the controls (Table 2).

The multivariate regression analysis demonstrated that 2D LV mass index ($\beta = 0.38$; $P < .01$), 2D LA longitudinal strain ($\beta = -0.3$; $P = .01$) and 3D LV area strain ($\beta = -0.37$; $P < .01$) were independently associated with HbA_{1c} in the whole study population.

Our investigation revealed several new findings: *a)* 3D LV myocardial deformation in all directions is deteriorated in diabetic patients; *b)* prediabetic patients have decreased 2D LV longitudinal strain, as well as 3D longitudinal and area strain; *c)* 2D LA longitudinal strain is decreased in diabetic patients; and *d)* 2D and 3D parameters of left heart mechanics are independently associated with glycemic control, assessed by HbA_{1c}.

Recently published studies have shown that 2D LV and LA strain are strong predictors of cardiovascular morbidity and mortality in the general population and in diabetic patients. Ernande et al[2,4] showed that 2D longitudinal and radial functions of each LV segment are impaired in diabetic participants; Ng et al[3] found that only 2D longitudinal deformation was reduced in diabetic patients, whereas circumferential and radial strain were preserved. Ceyhan et al[5] have published the first report that persons with impaired glucose tolerance have decreased LV systolic strain and strain rate, as well as early diastolic strain rate compared with controls. Our results confirm previous findings and add a new piece of evidence: namely, we found that 2D LV circumferential strain, in addition to longitudinal function, is also deteriorated in diabetic patients. Additionally, 3D speckle tracking revealed that LV mechanical deformation is impaired in all directions, along with area strain, which represents a combination of longitudinal and circumferential strain. Our findings demonstrate that LV systolic function, together with diastolic function, is impaired long before the occurrence of cardiac symptoms.

Zhang et al[6] showed that well-controlled diabetic patients have a decreased 3D strain in all directions, which completely confirms our results. The authors also found that HbA_{1c} was independently associated with all components of 3D LV strain. Our model of stepwise multivariate regression was different, and revealed that 2D LV and LA longitudinal strain, as well as 3D LV area strain, were independently associated with HbA_{1c}.

Further analyses are necessary to evaluate the predictive value of 2D, and especially of 3D strain, on cardiovascular morbidity and mortality in prediabetic and diabetic patients.

Marijana Tadic,[a,][*] Sanja Ilic,[b] Cesare Cuspidi,[c] Vesna Kocijancic,[a] and Vera Celic[a,d]

[a]Division of Cardiology, University Clinical Hospital Center "Dr. Dragisa Misovic - Dedinje", Belgrade, Serbia
[b]Division of Endocrinology, University Clinical Hospital Center "Dr. Dragisa Misovic - Dedinje", Belgrade, Serbia
[c]Clinical Research Unit, University of Milan-Bicocca and Istituto Auxologico Italiano, Milano, Italy
[d]Faculty of Medicine, Belgrade, Serbia

[*]Corresponding author:
E-mail address: marijana_tadic@hotmail.com (M. Tadic).

REFERENCES

1. Rojo-Martínez G, Valdés S, Colomo N, Lucena MI, Gaztambide S, Gomis R, et al. Consumo de fármacos relacionados con el tratamiento de la diabetes mellitus y otros factores de riesgo cardiovascular en la población española. Estudio Di@bet.es. Rev Esp Cardiol. 2013;66:854–63.
2. Ernande L, Bergerot C, Rietzschel ER, de Buyzere ML, Thibault H, Pignonblanc PG, et al. Diastolic dysfunction in patients with type 2 diabetes mellitus: is it really the first marker of diabetic cardiomyopathy? J Am Soc Echocardiogr. 2011;24:1268–75.
3. Ng AC, Delgado V, Bertini M, van der Meer RW, Rijzewijk LJ, Shanks M, et al. Findings from left ventricular strain and strain rate imaging in asymptomatic patients with type 2 diabetes mellitus. Am J Cardiol. 2009;104:1398–401.
4. Ernande L, Rietzschel ER, Bergerot C, de Buyzere ML, Schnell F, Groisne L, et al. Impaired myocardial radial function in asymptomatic patients with type 2 diabetes mellitus: a speckle-tracking imaging study. J Am Soc Echocardiogr. 2010;23:1266–72.
5. Ceyhan K, Kadi H, Koç F, Celik A, Oztürk A, Onalan O. Longitudinal left ventricular function in normotensive prediabetics: a tissue Doppler and strain/strain rate echocardiography study. J Am Soc Echocardiogr. 2012;25:349–56.
6. Zhang X, Wei X, Liang Y, Liu M, Li C, Tang H. Differential changes of left ventricular myocardial deformation in diabetic patients with controlled and uncontrolled blood glucose: a three-dimensional speckle-tracking echocardiography-based study. J Am Soc Echocardiogr. 2013;26:499–506.

http://dx.doi.org/10.1016/j.rec.2014.07.019

Thrombosis of a Mechanical Tricuspid Valve Prosthesis Resolved With Fibrinolysis

Trombosis de prótesis tricuspídea mecánica resuelta con fibrinolisis

To the Editor,

Implantation of a mechanical prosthesis in the tricuspid position is uncommon because most of the patients with disease in this valve choose to undergo repair consisting of annuloplasty. When mechanical prostheses are implanted in the tricuspid position, the risk of thrombosis is greater than that associated with these devices in left chambers, as the flow is slower in the right chambers and the pressure is lower. Currently, there is little literature on prosthetic tricuspid valve thrombosis and its treatment.

We report the case of a 52-year-old woman, with congenital atrioventricular block, who had undergone implantation of a dual-chamber pacemaker in 1986 and developed a pressure ulcer that required deepening of the pocket in 1989. Lead dysfunction occurred in 1996, and a new system was implanted, but the leads from the previous system were not withdrawn. In March 2013, she was diagnosed with severe tricuspid stenosis due to leaflet fibrosis, secondary to the leads that passed through the valve, and a 27-mm St. Jude Masters double-disc mechanical prosthesis was implanted. During the follow-up period, while asymptomatic, she reported that she no longer could hear the sounds of the prosthesis, and auscultation detected an increase in the intensity of the murmur during tricuspid filling. Doppler ultrasound revealed an increase in the atrioventricular pressure gradient across the tricuspid valve, and 3-dimensional transesophageal echocardiography demonstrated the immobility of one of the leaflets produced by a
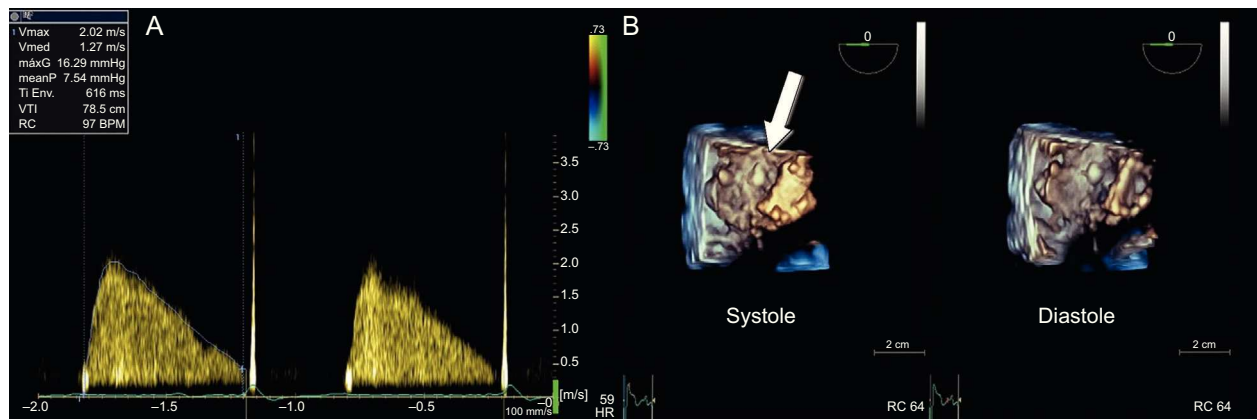
Figure 1. A: Doppler ultrasound at the level of the tricuspid valve prosthesis, showing an increase in the atrioventricular gradient with a decreased EF slope. B: 3-dimensional echocardiographic images at the level of the tricuspid valve prosthesis in systole and diastole, showing the immobility of one of the leaflets in systole produced by a thrombus (arrow).

thrombus (Figure 1). At that time, she was being treated with warfarin to maintain the International Normalized Ratio (INR) between 2.5 and 3.5. Nevertheless, her medical records show suboptimal anticoagulation levels weeks before the follow-up visit (INR 2.0-2.2).

The patient was admitted to the hospital and enoxaparin was added to the warfarin, at a dose of 1 mg/kg body weight/12 hours for 8 days. After this period, 3-dimensional transesophageal echocardiography was repeated and no changes were observed. Due to the risk of thrombus progression and its hemodynamic consequences, the decision was made to apply fibrinolysis, discontinue warfarin administration, and maintain enoxaparin. Once the INR was within normal range, 100 mg of tissue plasminogen activator was infused over 3 hours, with electrocardiographic monitoring and control of blood pressure and oxygen saturation; there were no complications.

Normalization of the atrioventricular gradient was subsequently verified by Doppler ultrasound, and a 3-dimensional transesophageal echocardiogram confirmed the disappearance of the thrombus and the mobility of the 2 discs (Figure 2). At the time of discharge, she was asymptomatic.

Currently, the little available scientific evidence regarding the most effective treatment for prosthetic tricuspid valve thrombosis is based only on case series and expert recommendations. The resolution of tricuspid valve thrombosis achieved by intensifying anticoagulation therapy or adding low-molecular-weight heparin has been reported,[1] but the level of evidence is low because of the lack of randomized studies.

A recent review of 9 studies, which included 48 patients with prosthetic tricuspid valve thrombosis who received fibrinolytic therapy, demonstrated a success rate of 88%, partial success in 1 patient, and no effectiveness in 4, who underwent surgical replacement of the prosthetic valve.[2]

The administration of fibrinolytic therapy has been accepted as the treatment of choice for prosthetic tricuspid valve thrombosis in the latest guidelines on valve disease. It carries a IIa recommendation and level of evidence of B,[3] based on nonrandomized studies and on retrospective cohort studies. No systemic embolisms were documented, although small subclinical pulmonary emboli were observed, and surgery was reserved for those cases in which fibrinolysis had been unsuccessful.

There is no specific recommendation as to which is the fibrinolytic agent of choice, and the options include streptokinase, tenecteplase, urokinase, and recombinant tissue plasminogen activator; the latter is most widely used.[2,4]

Transesophageal echocardiography is the technique of choice for the confirmation of both the presence of a thrombus and its resolution,[5] but there are no data concerning the utility of 3-dimensional echocardiography. In the case described here, the 3-dimensional echocardiographic images clearly show both the presence and the resolution of the thrombus (video of the supplementary material).

Recurrences have been reported in nearly a third of the cases. Given the close relationship between tricuspid prosthetic valve thrombosis and suboptimal anticoagulation, it has been recommended that the INR be maintained between 3 and 4 and that
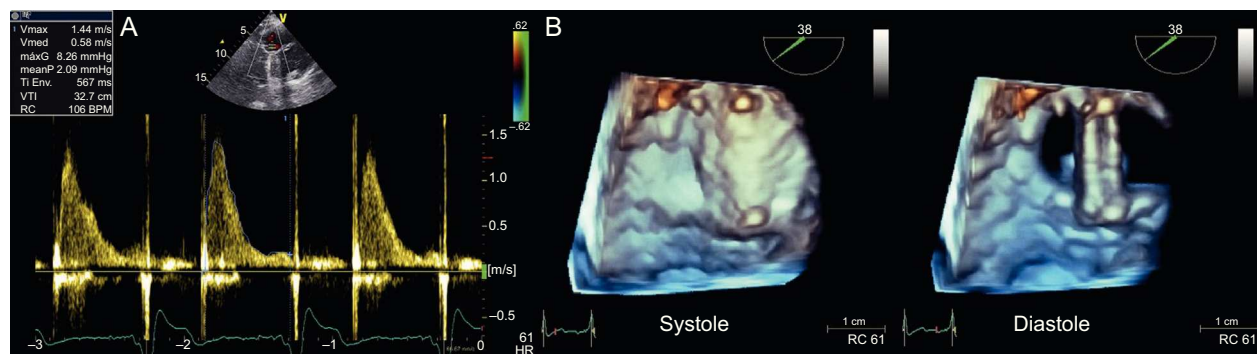

Figure 2. A: normalization of the tricuspid EF slope with normalization of the atrioventricular gradient. B: 3-dimensional echocardiogram at the level of the tricuspid valve prosthesis in systole and diastole, with complete aperture and closure of the 2 discs and disappearance of the thrombus.

acetylsalicylic acid or clopidogrel be included in the oral antic-oagulation therapy.[6]

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version available at doi:10.1016/j.rec.2014.07.018.

Andrés Betancur-Gutiérrez,* Julián Bayón-Fernández, Miguel Ángel García-Rodríguez, Abel García-Egido, Carmen Garrote-Coloma, and Felipe Fernández-Vázquez

Servicio de Cardiología, Complejo Asistencial Universitario de León, León, Spain

* Corresponding author:
E-mail address: afbetancur@gmail.com (A. Betancur-Gutiérrez).

REFERENCES

1. Shin HS, Kim KH, Lee HS, Choi HM, Lee SA, Park EA, et al. Organized prosthetic tricuspid valve thrombosis treated successfully with medical treatment. J Cardiovasc Ultrasound. 2013;21:192–6.
2. Huang G, Schaff H, Sundt T, Rahimtoola S. Treatment of obstructive thrombosed prosthetic heart valve. J Am Coll Cardiol. 2013;62:1731–6.
3. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, Guyton RA, et al. 2014 AHA/ACC guidelines for the management of patients with valvular heart disease. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63: e57–185.
4. Yi Zang D, Lozier J, Chang R, Sachdev, Chen M, Audibert J, et al. Case study and review: treatment of tricuspid prosthetic valve thrombosis. Int J Cardiol. 2012;162:14–9.
5. Tong A, Roudaut R, Ozkan M, Sagie A, Shahid M, Pontes S, et al. Transesophageal echocardiography improves risk assessment of thrombolysis of prosthetic valve thrombosis: results of the international PRO-TEE Registry. J Am Coll Cardiol. 2004;43:77–84.
6. Whitlock RP, Sun JC, Fremes SE, Rubens FD, Teoh KH. American College of Chest Physicians. Antithrombotic and thrombolytic therapy for valvular disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012; 141:e576S–600S.

Coronary Angioplasty With Catheter Extension Following Transcatheter Aortic Valve Implantation

Angioplastia coronaria con extensión de catéter tras implante transcatéter de prótesis aórtica

To the Editor,

Transcatheter aortic valve implantation has experienced exponential growth in recent years.[1] Aortic stenosis is frequently associated with coronary artery disease,[2] and the performance of a coronary angiogram prior to implantation is indispensable. The need for preimplantation angioplasty is a matter of debate.[3] In any case, given the increase in the rate of successful implantation and in the survival of these patients, it is becoming more common to treat patients with coronary artery disease who have received these prostheses.

The CoreValve® self-expanding prosthesis (Medtronic) is designed for placement above and below the origin of the coronary arteries. The purpose of this design is to ensure the patency of the coronary ostia. Its configuration in open cells theoretically allows intubation of the coronary arteries after implantation. On occasion, intubation of the coronary arteries can be difficult, since the imaging techniques usually employed do not have sufficient temporal and spatial resolution to determine the position of the cells with respect to the origin of the coronary arteries.

We present the case of a 75-year-old man who underwent implantation of a 26-mm CoreValve® prosthesis to treat symptomatic severe aortic stenosis and was at high surgical risk. Coronary angiography performed prior to implantation revealed no evidence of angiographically significant stenosis (Figure 1). Two years after implantation, the patient was admitted to the hospital with non-ST segment elevation acute coronary syndrome. Coronary angiography revealed 90% stenosis in the proximal circumflex artery (Figure 1), and the decision was made to perform angioplasty.

Left coronary artery was nonselectively intubated from a right radial approach through the cells of the prosthesis using a 4.0 6 Fr EBU guiding catheter, after attempting intubation unsuccessfully
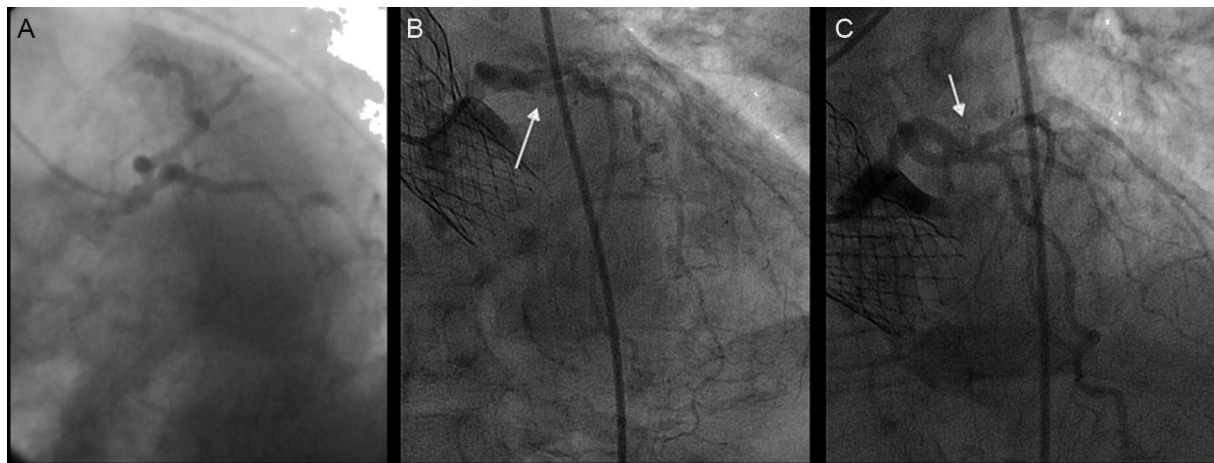

Figure 1. A: Coronary angiography prior to transcatheter aortic valve implantation. B and C: Angiographically significant stenosis in proximal circumflex artery.