First, while it is quite normal in surgical procedures to repair several valves in a single operation, in interventional cardiology this aspect may generate some controversy. Longer duration of the procedure is associated with an increased risk of complications associated with vascular access,[2] the need for a longer anesthetic,[3] increased use of ionizing radiation, and more transesophageal ultrasound time.[4]

Second, with currently-available medical treatment, the short-term mortality from TR, even in its most severe forms, is low, especially in patients who maintain good functional status.[5]

Third, the 2 valvulopathies are closely dependent. It has been demonstrated that correction of MR, independently of the technique, significantly reduces left ventricular filling pressures and pulmonary hypertension.[6] In the case of the MitraClip, between a third and a half of patients have a significant reduction in TR grade following MR repair.[7,8] It is therefore more than reasonable to wait and see the results before planning a second procedure, especially in cases with little dilatation of the tricuspid annulus and a structurally normal valve.

Fourth, the use of several devices in a single procedure implies a higher financial burden, which means rigorous selection of appropriate candidates is essential, especially since a high percentage of cases of TR improve after MR repair.

Last, there is little experience of the benefit of performing both repair procedures in combination. Only one study has indicated that a certain survival benefit could be obtained, but there were many limitations to its design, and it compared mitral repair alone against simultaneous repair of both valves, but not against staged repair.[9]

The percutaneous repair of TR represents a major advance and hope, particularly for patients who are not candidates for surgery.[10–14] Although nobody would question combined repair in a surgical procedure, in percutaneous procedures this is more controversial. A randomized study is needed to compare simultaneous repair of both valves against a staged approach based on the results on TR.

CONFLICTS OF INTEREST

Á. Sánchez-Recalde is associate editor of *Revista Española de Cardiología;* the journal's established editorial procedure to ensure impartial management of the manuscript has been followed.

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Available online 17 October 2020

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https://doi.org/10.1016/j.rec.2020.08.017
1885-5857/

Simultaneous percutaneous repair in mitral and tricuspid regurgitation: step by step. Response

Reparación percutánea simultánea en la insuficiencia mitral y tricuspídea: paso a paso. Respuesta

To the Editor,

First of all, we would like to thank Sánchez Vega et al. for the interest shown in our recent article. The field of combined interventional cardiology for mitral and tricuspid regurgitation (TR) is in its early stages, and the scientific evidence is still not sufficiently strong. Therefore, it is only natural that questions arise about these complex procedures. Nonetheless, some arguments support combined repair in certain cases. The presence of severe TR at the time of mitral repair has been reported to be associated with poorer prognosis, even in the short-term.[1,2] Furthermore, although TR is theoretically significantly reduced after mitral repair, this outcome is seen in only 15% to 40% of patients.[3] Consequently, a number of patients could experience clinical deterioration due to residual TR. Factors such as annular dilatation, degree of TR, right-sided dysfunction and dilatation, or the presence of congestive symptoms may indicate that the TR will not improve and that there is a risk of adverse events during follow-up. Once the decision has been made to treat both valves, combined treatment appears to

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have advantages. It is safe and feasible, it shortens the duration of the procedure compared with 2 deferred procedures, and it lowers the risks associated with a new vascular access, another hospital stay, and repeat general anesthesia in patients already at high risk. Randomized studies would be ideal, but definitive conclusions might not be reached, given the heterogeneity of the condition. In the interim, our decisions should be guided by the clinical judgment and resources of the interventional cardiology team.

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Available online 21 October 2020

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https://doi.org/10.1016/j.rec.2020.09.018
1885-5857/

Prothrombosis in times of COVID-19

Protrombosis en tiempos de la COVID-19

To the Editor,

We read with special interest the recent article in *Revista Española de Cardiología* by Rey et al.[1] on a patient with acute coronary syndrome (ACS) and simultaneous thromboses in 2 coronary arteries in the context of coronavirus disease 2019 (COVID-19). We would like to report a case with similar characteristics, in which the patient was a 55-year-old man with a history of hypertension, type 2 diabetes mellitus, former smoker (30 pack-years), chronic obstructive pulmonary disease defined as Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage 2 A, and no known history of heart disease. Our patient presented to the Emergency Department with a history lasting several days of orthopnea and dry cough, describing dyspnea and squeezing chest pain that had lasted some days but had completely disappeared by the time he arrived.

Electrocardiography revealed newly appeared left bundle-branch block (LBBB) that did not meet the Sgarbossa criteria. However, myocardial injury markers were elevated (high-sensitivity troponin T of 220 ng/L followed by 333 ng/L at 3 h, with dynamics consistent with ACS). Point-of-care echocardiography showed severe left ventricular dysfunction with akinesia of the anterior, lateral, and posterior segments, inferior hypokinesia, and asynchronous contractility due to LBBB. Chest radiography revealed interstitial pattern and bilateral alveolar infiltrates, consistent with COVID-19 infection (figure 1). Two PCR tests for COVID-19 were negative.

The patient was admitted to the intensive care unit, and medical treatment was started for ACS along with diuretic, vasodilator, and inotropic therapy. Coronary angiography showed a severe lesion in the obtuse marginal artery, which was revascularized with a drug-eluting stent.

Once in the ward, the patient underwent comprehensive echocardiography showing severe systolic dysfunction due to general hypokinesia with considerable asynchrony (left ventricular ejection fraction by biplane Simpson's method, 29%), with no other findings of interest. Subsequent cardiac magnetic resonance imaging showed 2 foci of late transmural enhancement in the sequences: 1 in the short axis view, in the mid anterolateral and lower medial segments, and 1 in the 2-chamber long axis view, in the anterior basal and lower medial segments, which would be consistent with simultaneous acute infarctions, given that the edema sequences showed infarction in these regions (figure 2).

After the patient was stabilized, a decision was made to discharge him to home with medical treatment for ACS and heart failure with reduced left ventricular ejection fraction.

Outpatient follow-up with the heart failure unit of our hospital showed that the patient currently has no cardiovascular symptoms and has partially recovered left ventricular ejection fraction (40%) after the disappearance of LBBB. Because the admission radiograph was consistent with COVID-19 and there were transmural infarctions in several territories, a decision was made to perform a new PCR test for COVID-19 and serology testing by enzyme-linked immunosorbent assay (ELISA), which was positive for immunoglobulin G (IgG).

Coronaviruses and the remaining microorganisms causing acute respiratory infections are known to be key triggers for the development of ACS.[2] The most common complications related to this virus are acute respiratory distress syndrome, secondary infections, and shock,[2] but a number of case reports have been published on arterial thromboses secondary to the virus, including the article mentioned,[1] possibly due to the virus-induced hypercoagulable state secondary, among other factors, to the cytokine storm or blood stasis caused by immobilization.[3]

The case we describe is similar to that reported in King et al.,[1] except for the difference that coronary angiogram showed a severe lesion in only 1 epicardial blood vessel, but subsequent cardiac

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