**Brief Report**

Percutaneous Coronary Intervention for Iatrogenic Occlusion of the Circumflex Artery After Mitral Anuloplasty

Ramón Mantilla, a Juan J. Legarra, b Gonzalo Pradas, b Marisol Bravo, a Marcelo Sanmartín, a and Javier Goicolea a

aÁrea de Cardiología, Instituto Galego de Medicina Técnica, Hospital do Meixoeiro, Vigo, Pontevedra, Spain. bUnidad de Cirugía Cardíaca, Instituto Galego de Medicina Técnica, Hospital do Meixoeiro, Vigo, Pontevedra, Spain.

We describe a patient with obstruction of the dominant circumflex artery after surgical repair of the mitral valve, repaired successfully with percutaneous coronary intervention during the immediate postoperative period. We discuss the etiology, prevention and management of this complication with special emphasis on percutaneous intervention.

**Key words:** Occlusion. Coronary. Surgery. Mitral valve. Coronary angioplasty.

*Full English text available at: www.revespcardiol.org*

---

**INTRODUCTION**

When anatomically feasible, the technique of choice for severe mitral regurgitation (MR) is repair rather than replacement of the native valve.1,2 Preservation of the valvular structure and its mechanical relation to the left ventricle (LV) as well as a lower incidence of hemorrhagic complications associated with the absence of anticoagulation have a favorable impact on the patient’s prognosis.3,4 Despite the greater surgical complexity involved in mitral valve preservation and annuloplasty, only 2 cases of occlusion of the dominant circumflex artery—located in this case near the annulus—have been reported. We describe a case of postsurgical occlusion of the circumflex artery resolved by percutaneous coronary intervention (PCI). To our knowledge, this is the first time that PCI has been used in this context.

**CASE REPORT**

A 66-year-old male with symptomatic, chronic severe mitral regurgitation was referred to our unit for elective mitral valve repair. Preoperative echocardiography found severe MR caused by posterior leaflet prolapse. Coronary angiography revealed left dominance with no apparent lesions, and mild dilatation of the LV with evidence of severe MR on ventriculography. The presence of a severe central prolapse of the posterior leaflet, very elongated primary chordae tendineae, and rupture of the leaflet’s posterior and anterior secondary chordae was confirmed during surgery. A quadrangular resection of the posterior leaflet and mitral annuloplasty with a 28 mm Carpentier ring were performed. After extracorporeal circulation was terminated during sinus rhythm, transesophageal echocardiography revealed mild MR. In the intensive care unit the patient developed hypotension that was unresponsive to medication and intra-aortic balloon counterpulsation (IABP). Electrocardiogram revealed...
complete atrioventricular block and ST segment elevation of up to 6 mm in leads II, III, aVF, I, aVL, V₅, and V₆, suggestive of acute inferolateral myocardial infarction. Given the suspected occlusion of the dominant circumflex artery, an emergency cardiac catheterization was performed, revealing complete occlusion of the middle third of the dominant circumflex artery, near the Carpentier ring and after the emergence of a large marginal branch (Figure 1). Considering the patient’s critical situation, a percutaneous recanalization was performed by low-pressure (2 atm) balloon angiography. Complete expansion was achieved, although the elastic recoil of the dilated segment required insertion of a stent, which was implanted successfully. Migration of intraluminal material to the marginal branch resulting in compromised blood flow was observed. As a result, kissing balloon angioplasty was performed by simultaneously inflating 2 balloons inserted, respectively, into the marginal branch and the middle of the circumflex artery. Persistence of a significant occlusion in the marginal branch required the insertion of a second stent in this vessel. The final angiographic result was optimal (Figure 2). During the first 24 hours after the PCI, the electrocardiogram gradually normalized, hemodynamic stabilization was achieved, and both inotropic support and IABP were terminated. Serial echocardiography revealed a gradual recovery of global and segmental LV systolic function; at discharge, the patient presented mild inferior hypokinesis, contractility of the other segments was normal, the ejection fraction was 0.60, and the mitral valve showed no evidence of significant regurgitation. The electrocardiogram was in sinus rhythm with pathologic Q waves in leads II, III and AVF.

**DISCUSSION**

We present a case of iatrogenic occlusion of the dominant circumflex artery after mitral valve repair, resolved by PCI. There are few references to this complication in the literature. In most cases, this complication occurred during mitral valve replacement and was attributed to the inadvertent suture of the dominant or codominant circumflex artery. A left dominant circumflex artery is located, on average, closer to the mitral annulus than a codominant circumflex artery (4 mm compared to approximately 8 mm). In the only reported case of occlusion occurring after mitral valve repair, the complication was detected intraoperatively after transesophageal echocardiography revealed significant changes in contractility of the inferior, posterior, and lateral segments; as a result, extracorporeal circulation was restarted, the mitral annuloplasty ring was removed, the mitral valve was replaced with a mechanical prosthesis, and a saphenous vein was used to create a bypass to a marginal branch. In our case, after transesophageal echocardiography confirmed successful mitral valve repair with no anomalies in LV contractility, the patient was transferred to the intensive care unit, where he developed severe hypotension associated with ischemic changes in the electrocardiogram, causing us to suspect acute postsurgical occlusion of the circumflex artery. The timing of these events seemed to in-
dicate that the occlusion may have developed gradually, suggesting that the underlying cause was not a fixed mechanical occlusion caused by suture, but rather one of the following: subintimal hematoma; coronary artery spasm; deformation of the vessel as a result of surgery; or thrombotic occlusion due to laceration of the endothelium. This hypothesis is also supported by the fact that complete expansion of the injured segment was achieved through low-pressure, percutaneous coronary balloon angioplasty.

Even though preservation of the mitral repair and early revascularization was accomplished by PCI in our case, it is important to realize that if the underlying cause is the inadvertent suturing of the vessel, the result is unpredictable and the potential for revascularization by PCI is less. We believe that the emergence of the occlusion in the immediate postoperative period, as in our case, suggests mechanisms that may be resolved by PCI, whereas the best therapeutic option for intraoperative occlusions is undoubtedly a second intervention.

REFERENCES