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REVIEW

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Acute iatrogenic complications after mitral valve repair

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Abstract

Background and Aim of the Study: Mitral valve repair is the procedure of choice to correct mitral regurgitation. However, some dangerous iatrogenic complications can occur at the end of the procedure. Therefore, we sought to review the most frequent and clinically relevant acute iatrogenic complication following mitral valve repair.

Methods: A thorough review of the literature has been performed. Criteria for considering studies for this non-systematic review were as follows: observational and interventional studies investigating the acute iatrogenic complications following mitral valve repair, and essential review studies pertinent to the topic.

Results: The most frequent is the systolic anterior motion. Due to a systolic dislocation of the anterior leaflet toward the outflow tract, it causes both obstruction of the outflow tract and mitral regurgitation. Often it is due to excess of catecholamines or to reduced filling of the left ventricle but sometimes needs further surgical maneuvers, focused on moving posteriorly the coaptation line. It can be obtained by shortening the posterior leaflet or increasing the size of the ring or applying an Alfieri stitch to limit the movements of the anterior leaflet. Another complication, often underdiagnosed and potentially lethal, is the injury of the circumflex artery that happens at the level of the anterolateral commissure or P1 zone. Two mechanisms are involved. The first one is the direct injury of the artery by a stitch (roughly 25% of the patients present a distance artery-annulus <3 mm. The second one is the distortion of the artery, attracted toward the annulus by a misplaced stitch. The attraction causes kinking with stenosis of different degrees till functional occlusion. However, the artery has to be far from the annulus and the atrial tissue has to be stiff and resistant, as after an infective process, to move the circumflex artery toward the annulus without tearing. Positioning the stitches very close to the mitral leaflets in the dangerous area is the only prevention to the complication. The treatment in the operating theater is partial or total removal/reimplantation of the annular sutures or coronary artery bypass grafting to the circumflex area. If the injury is demonstrated only after coronary angiography, percutaneous revascularization can be attempted before further surgical treatment.

Conclusions: Acute iatrogenic complication after mitral repair exists and may compromize patient outcome. Raising awareness about these issues, the precautions to prevent them, and the manners of resolution is therefore mandatory.

KEYWORDS
cardiovascular pathology, clinical review, valve repair/replacement

1 | INTRODUCTION

Mitral valve (MV) repair is the procedure of choice for the treatment of severe mitral regurgitation (MR), particularly in degenerative disease with leaflet prolapse. Correction of the leaflets abnormalities enables to recover the valvular function and annular reshaping, by rings or bands, remodels the mitral annulus to adequate size and shape, enhancing the coaptation area and preventing further dilatation. As all surgical procedures, patients undergoing MV repair can experience severe complications before leaving the operative theater (OR).

Some of them are intrinsic to mitral disease and to its correction. Systolic anterior motion (SAM), that adds acute left ventricular (LV) obstruction to residual MR of variable grade, is, perhaps, the most common cause after MV repair for degenerative MR, as, in the case of ischemic or secondary MR, it virtually does not exist. Injury to the circumflex artery (CX) is an underestimated event, often lethal, that needs immediate recognition and treatment. Both these complications are directly related to MV repair, even if CX injury was first described after MV replacement.[1]

2 | SAM

MV repair can be accomplished in almost all cases of degenerative MV insufficiency (Type 2 according to Carpentier classification). Different techniques have been described and many surgeons documented excellent long-term results with different reparative options. It is important, however, not to face every operation in the same way. When considering, for example, flail leaflet pathology, in young patients with redundant myxomatous degenerative mitral leaflets the type of repair should not be equal to elderly patients with fibro-elastic deficiency.

One of the reasons why these differences need to be considered is the possible occurrence of SAM. SAM is a complication of MV repair in which an anterior dislocation of the anterior mitral leaflet during systole occurs leading to the obstruction of the LV outflow tract and to mal-coaptation of the leaflets with varying degree of eccentric MR (directed toward the interatrial septum).[2] The incidence of SAM following MV repair varies from 1% to 10% according to different reports and definition used in the studies.[3,4]

Avoiding SAM is one of the goal of surgical repair of the MV. For this purpose, linking echocardiographic information to the type of repair is essential. Preoperative trans-esophageal echocardiography (TEE) helps predicting the risk of postoperative SAM. Besides the usual data regarding the severity of regurgitation, the regurgitant jet origin and direction, the presence of a flail leaflet, and the annular dimensions, TEE must advise surgeons about the risk of SAM particularly when abundant redundancy of the leaflets, hypertrophic interventricular septum, and anterior dislocation of the coaptation line during systole are observed. A distance between the coaptation point and the septum in systole (C-Septum distance) inferior to 25 mm, that usually occurs when the height of the posterior leaflet exceeds 25 mm (particularly in the median scallop P2), has been associated with an increased risk of SAM after repair[5] (Figure 1). This is a situation typically seen either in a young patient with a severe myxomatous disease, where the anterior/posterior leaflet ratio is close to 1, or in elderly patients with less abundant leaflet height but accentuated septum hypertrophy. The combination of a smaller LV end-systolic volume, a lower ratio of anterior to posterior leaflets heights, and the presence of bileaflet prolapse are associated with a high risk of SAM after separation from cardiopulmonary bypass (CPB).[4]

In these cumbersome anatomical circumstances, the goal of the correction has to create a coaptation line positioned posteriorly, toward the posterior annulus, having the anterior leaflet as much as possible extended in systole toward the posterior one. The height of the posterior leaflet needs to be reduced and this can be accomplished by resections of the prolapsing scallop (quadrangular, triangular, or any kind of resection) and, in case of >25 mm leaflet, by detaching the remaining posterior scallops from the annulus according to the sliding plasty technique described by Alain Carpentier.[6] When the height of the posterior scallops is extreme (>30 mm) or when there is a discrepancy between the height of the remaining scallops after resection and sliding, then shortening of the scallop can be performed by gently removing 5–10 mm of tissue from the posterior part of the scallop before suturing it back to the annulus. An alternative to resections and sliding plasty is to fold the posterior leaflet toward the posterior annulus with several stitches to reduce leaflet length and mobility.[7]

Lately, the "respect rather than resect" concept has been applied in the treatment of the prolapsing posterior leaflet.[8] In this technique, artificial chords (i.e., Gore-Tex®) are positioned from the papillary muscles to the free margin of the prolapsing leaflet. It is an excellent alternative to the resection technique. Both repairs for posterior mitral leaflet prolapse are associated with excellent results and

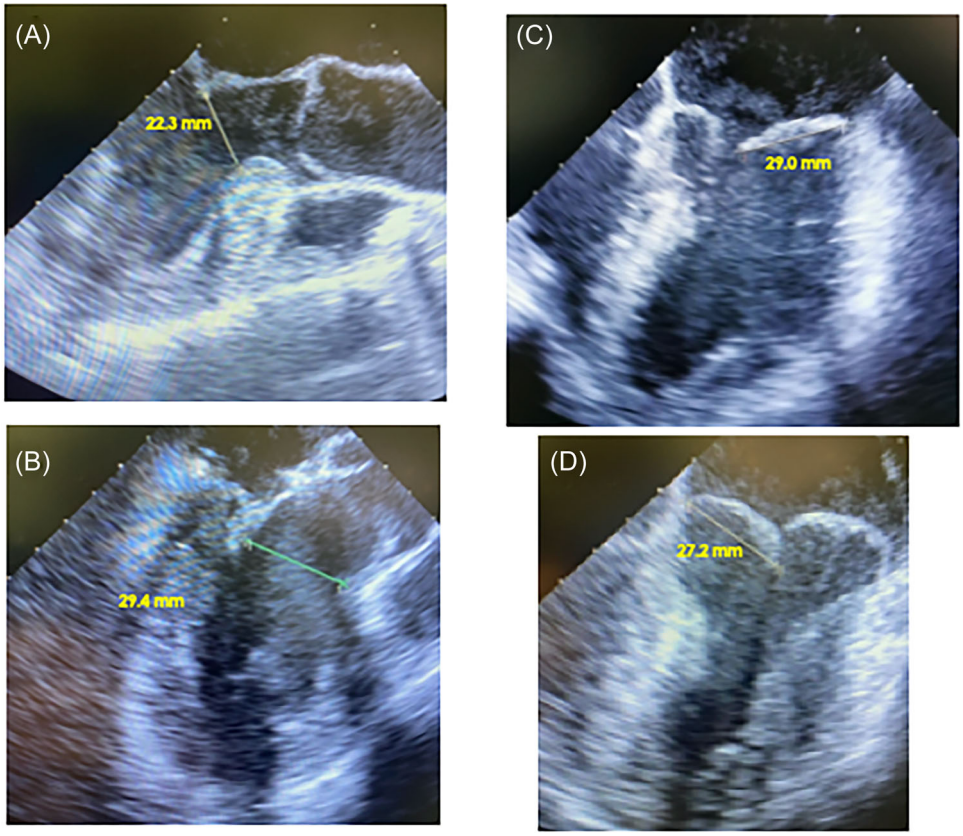

FIGURE 1 (A) Preoperative measure of a C-Septum distance inferior to 25 mm. (B) C-Septum distance following a successful repair via posterior mitral leaflet resection, sliding, and ring annuloplasty. (C) Anterior mitral leaflet measure. (D) Posterior mitral leaflet measure (i.e., slight underestimation: 2.72 cm is the measure of the chord whereas the arc—"true leaflet" is indeed longer). The anterior/posterior leaflet ratio is thus close to 1.

appear comparable in the early postoperative course.[9] However, in an anatomical situation at risk of developing SAM following repair, the "loop technique" should be avoided because it would facilitate anterior displacement of the coaptation line, and resection is preferred instead.

Placing an annular ring is a key element for long-lasting MV repair. In fact, in Type II degenerative MR the valve almost invariably takes a circumferential shape, and the ring serves to restore normal intercommissural and septo-lateral diameter recreating the normal elliptical shape. However, when the risk of SAM exists, the choice of the ring is crucial. Placing a small complete ring (<34 mm) in a large and redundant myxomatous valve may favor anterior displacement of the coaptation line leading to SAM. If the "loop technique" is preferred over resection of the prolapsing posterior scallop, then a large rather than small ring should be considered. The ring should have the only goal of recreating an elliptical shape rather than forcing coaptation, and with this in mind, an open ring can also be used.

In most cases, SAM occurring in the operating room, observed when the patient has been weaned from CPB, can be successfully treated by increasing the LV filling volume with fluids, removing any inotropic drug used to come off bypass, and reducing heart rate with beta-blockers. Very seldom this strategy is insufficient, and a decision needs to be taken to correct the anomaly. In this situation, the surgeon faces difficult moments: the patient has already gone through a certain amount of time on CPB and cardioplegic arrest, another mitral repair attempt must be resolutive without the risk of another early failure and a third pump run. In this scenario, surgeons can correct the problem using a larger ring, decreasing posterior leaflet height, or adding an Alfieri stitch.[4,10]

3 | CX LESION

MV repair can be associated with the danger of CX injury (direct damage by sutures), due to the close spatial relationship between the CX and the mitral annulus. The artery is involved in the great majority of the cases in its proximal portion, close to the anterolateral commissure or P1, where the distance between the artery and the annulus is the shortest.[11] Patients with larger CX diameter seem to be more exposed to this complication, as the distance to the annulus reduces.[12] Another mechanism is CX distortion, where the CX is attracted toward the annulus when a misplaced stitch is tied causing severe flow reduction till functional

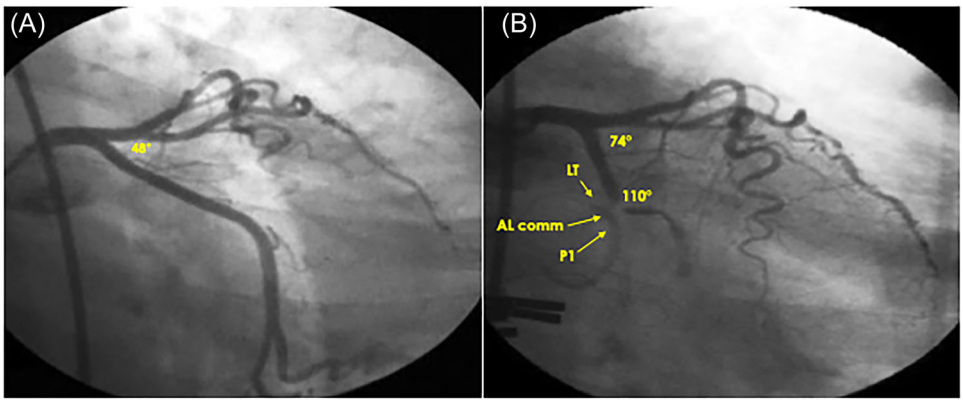

FIGURE 2 Coronary angiography. (A) Preoperatively, the angle between the CX and the LAD was narrow (48°), anticipating a course of the artery far from the mitral annulus. (B) Before discharge, the SVG to OM was well functioning (not shown). The angle CX-LAD increased to 74° and the CX itself was severely distorted (angle 110°). This was possible because the CX was far from the mitral annulus. This patient had a previous endocarditis, which could have caused atrial wall stiffening. From Calafiore et al.,[13] with permission. CX, circumflex artery; LAD, left anterior descending artery; OM, obstruse marginal artery; SVG, saphenous vein graft.

occlusion (Figure 2). In this case, the CX has to be far from the annulus, but other contributing factors have to be present, as the atrial wall has to be enough stiff and resistant to move the CX toward the annulus without tearing.

The spatial relationship between the CX and the mitral annulus has been widely studied. Most of the reports showed that the CX is closer to the annulus in presence of left dominance or codominance, but, according to Levisman,[14] independently from the dominance, the proximal CX can be as close as 1 mm to the mitral annulus at the level of the anterolateral commissure. These data were confirmed by Caruso et al.,[15] who found that 66.7% of the patients considered at high risk (mean distance CX-annulus <3 mm) had right dominance. However, Kishimoto et al.[16] found that the distance CX-annulus is 3 mm or less in 25% of the patients overall, but in presence of a left coronary dominance, the percentage rose to 75%, whereas the prevalence was more or less similar in patients with left co-dominance (26%) or right dominance (17%).

This complication is potentially lethal and immediate recognition and treatment are mandatory to avoid the sequelae of a lateral acute myocardial infarction. Surgeons must suspect CX injury or kinking in presence of ventricular arrhythmias, difficult weaning from CPB, inferolateral hypo- or akinesia, or electrocardiograph (EKG) signs of myocardial ischemia. Better information can be provided by direct echocardiographic analysis of the CX course, where the presence of aliasing or no flow is able to make evident the lesion.[17] When there is no suspicion in OR, in the Intensive Care Unit arrhythmias, EKG changes or hemodynamic instability are indications for urgent coronary catheterization, as angioplasty can immediately restore the flow. If the point of injury cannot be crossed, urgent surgery is mandatory. It consists in direct revascularization of the occluded vessel or in partial/total removal and reimplantation of the ring, in the area of the anterolateral commissure and P1, passing the stitches as close as possible to the leaflet. Not always the consequences are immediately evident, but the complication can be diagnosed even after days,[18] months,[19,20] or years.[21,22] In a review of the literature, where cases of MV replacement were included, 7% of the patients had a diagnosis after 30 days from surgery.

The prevalence of iatrogenic lesions of the CX during MV repair is not well known. Only a few papers checked systematically the preoperative relationships between the CX and the annulus, reporting the results and the surgical outcome. Caruso et al.,[15] in 95 consecutive patients, showed that in 25% of the patients the distance CX-annulus was <3 mm. In these patients, the stitches were not passed in the dangerous area. Nevertheless, one patient (1.1%) experienced CX obstruction, immediately corrected replacing a rigid ring with a flexible one.[23] Ender et al. used TEE to visualize the CX in 110 cases. Three patients (2.7%) experienced CX injury and successfully underwent surgical or percutaneous correction.[17] In other similar series the prevalence was 1.9%[24] and 1.8%,[25] higher than reported in other experiences (0.15%[26] to 0.3%[18]).

The real problem is the time from CX lesion to revascularization, from which the extension of lateral infarction and the clinical outcome depends. When the complication is treated before leaving the OR, results are uniformly good, but when the treatment is performed after coronary angiography, the mortality is around 10%, but the consequences on the LV function can be serious. Coutinho et al. reported six cases where only one case was treated in OR successfully. All the other cases had coronary angiography and delayed treatment. One patient was transplanted after 10 days, three were discharged with a low ejection fraction (one had a redo 2 years later due to severe MR and huge posteroinferior aneurysm) and one was reoperated on after 5 years for severe MR with depressed LV function.

4 | COMMENT

Acute iatrogenic complications of MV repair depend on the techniques applied by the surgeons to correct the valvular lesions (SAM) and on the position of the stitches used to implant a ring or band to reshape the annulus (CX injury).

SAM is specific of the correction of degenerative MR, while CX injury can happen every time stitches are passed close to the mitral annulus. SAM has been widely studied and many strategies have been suggested to prevent or correct it. However, even if surgeons are aware of this possibility, the prevalence of SAM has remained more or less the same on the last decades, being 9.1% in 1994,[27] 8.4% in 2007,[28] and 8.1% in 2017.[29] In the most recent experience,[4] the prevalence was 13%, but, after adequate surgical or medical treatment, still 3.7% of the patients with MV repair were discharged with SAM.

CX injury is surely less frequent, but possibly underdiagnosed, but it has to be suspected any time there is difficult weaning from CPB or there are signs of an ischemic event. However, it is not part of the surgeons' mentality that the necessity to have a preoperative diagnosis on the CX relationships with the annulus. The mechanism of injury, moreover, is not always the same. A short distance between the CX and the annulus exposes the danger of passing a stitch through the artery, whereas attracting the CX toward the annulus can happen independently from the CX position. It is evident that to cause occlusion or severe stenosis by attraction, the CX has to be far from the annulus, as in the case shown in Figure 2. Intraoperative echocardiographic evaluation of the CX flow is the most helpful tool we have to diagnose the complication independently from the mechanism and to promptly react to avoid or to limit a dangerous perioperative myocardial infarction.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Danielson GK, Cooper E, Tweeddale DN. Circumflex coronary artery injury during mitral valve replacement. *Ann Thorac Surg*. 1967;4:53-59.
2. Mihaileanu S, Marino JP, Chauvaud S, et al. Left ventricular outflow obstruction after mitral valve repair (Carpentier's technique). Proposed mechanisms of disease. *Circulation*. 1988;78:I78-I84.
3. Lee KS, Stewart WJ, Lever HM, Underwood PL, Cosgrove DM. Mechanism of outflow tract obstruction causing failed mitral valve repair. Anterior displacement of leaflet coaptation. *Circulation*. 1993;88:II24-II29.
4. Ashikhmina E, Schaff HV, Daly RC, et al. Risk factors and progression of systolic anterior motion after mitral valve repair. *J Thorac Cardiovasc Surg*. 2021;162:567-577.
5. Maslow AD, Regan MM, Haering JM, Johnson RG, Levine RA. Echocardiographic predictors of left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve after mitral valve reconstruction for myxomatous valve disease. *J Am Coll Cardiol*. 1999;34:2096-2104.
6. Jebara VA, Mihaileanu S, Acar C, et al. Left ventricular outflow tract obstruction after mitral valve repair. Results of the sliding leaflet technique. *Circulation*. 1993;88:II30-II34.
7. Calafiore AM, Di Mauro M, Actis-Dato G, et al. Longitudinal plication of the posterior leaflet in myxomatous disease of the mitral valve. *Ann Thorac Surg*. 2006;81:1909-1910.
8. Perier P, Hohenberger W, Lakew F, et al. Toward a new paradigm for the reconstruction of posterior leaflet prolapse: midterm results of the "respect rather than resect" approach. *Ann Thorac Surg*. 2008;86:718-725.
9. Falk V, Seeburger J, Czesla M, et al. How does the use of polytetrafluoroethylene neochordae for posterior mitral valve prolapse (loop technique) compare with leaflet resection? A prospective randomized trial. *J Thorac Cardiovasc Surg*. 2008;136:1205.
10. Mascagni R, Al Attar N, Lamarra M, et al. Edge-to-edge technique to treat post-mitral valve repair systolic anterior motion and left ventricular outflow tract obstruction. *Ann Thorac Surg*. 2005;79:471-473.
11. Fabian B, Osadczuk A, Barany L, Baksa G, Racz G, Ruttkay T. Real 3D visualization of the circumflex artery surrounding the mitral annulus. *Thorac Cardiovasc Surg*. 2022;70:87-92.
12. Torres CS, Sanders JVS, Martins de Brito H, et al. Anatomical relationship between mitral valve annulus and circumflex artery and its surgical implications. *Morphologie*. 2020;104:182-186.
13. Calafiore AM, Iacò AL, Varone E, Bosco P, Di Mauro M. Distortion of the proximal circumflex artery during mitral valve repair. *J Card Surg*. 2010;25:163-165.
14. Levisman JA. Systolic anterior motion of the mitral valve due to hypovolemia and anemia. *Chest*. 1976;70:687-688.
15. Caruso V, Shah U, Sabry H, Birdi I. Mitral valve annulus and circumflex artery: in vivo study of anatomical zones. *JTCVS Tech*. 2020;4:122-129.
16. Kishimoto N, Takahashi Y, Fujii H, et al. Computed tomography to identify risk factors for left circumflex artery injury during mitral surgery. *Eur J Cardiothorac Surg*. 2022;61:675-683.
17. Ender J, Selbach M, Borger MA, et al. Echocardiographic identification of iatrogenic injury of the circumflex artery during minimally invasive mitral valve repair. *Ann Thorac Surg*. 2010;89:1866-1872.
18. Coutinho GF, Leite F, Antunes MJ. Circumflex artery injury during mitral valve repair: not well known, perhaps not so infrequent-lessons learned from a 6-case experience. *J Thorac Cardiovasc Surg*. 2017;154:1613-1620.
19. Ziadi J, Mleyhi S, Denguir R, Khayati A. Iatrogenic occlusion of the circumflex artery and left ventricle pseudoaneurysm after mitral annuloplasty. *J Cardiol Cases*. 2014;9:104-105.
20. Hiltrop N, Bennett J, Desmet W. Circumflex coronary artery injury after mitral valve surgery: a report of four cases and comprehensive review of the literature. *Catheter Cardiovasc Interv*. 2017;89:78-92.
21. Busu T, Alqahtani F, Kawsara A, Hijazi M, Alkhouli M. Iatrogenic circumflex artery stenosis following mitral valve repair. *Cureus*. 2017;9:e1680.
22. Sunagawa O, Nakamura M, Hokama R, Miyara T, Taba Y, Touma T. A case of percutaneous coronary intervention for treatment of iatrogenic chronic total occlusion of the left circumflex artery after mitral valve repair. *Cardiovasc Interv Ther*. 2017;32:146-150.

23. Caruso V, Sabry H, Birdi I. Dramatic resolution of an immediate postoperative distortion of the circumflex artery during mitral valve surgery. *J Card Surg*. 2020;35:1135-1137.
24. Miura K, Komiya T, Shimamoto T, Matsuo T. How far is the left circumflex coronary artery from the mitral annulus? *Gen Thorac Cardiovasc Surg*. 2020;68:1447-1452.
25. Aybek T, Risteski P, Miskovic A, et al. Seven years' experience with suture annuloplasty for mitral valve repair. *J Thorac Cardiovasc Surg*. 2006;131:99-106.
26. Bargagna M, Trumello C, Sala A, et al. Left circumflex artery injury after mitral valve surgery: an algorithm management proposal. *Ann Thorac Surg*. 2021;111:899-904.
27. Grossi EA, Steinberg BM, LeBoutillier M 3rd, et al. Decreasing incidence of systolic anterior motion after mitral valve reconstruction. *Circulation*. 1994;90:II195-II197.
28. Brown ML, Abel MD, Click RL, et al. Systolic anterior motion after mitral valve repair: is surgical intervention necessary? *J Thorac Cardiovasc Surg*. 2007;133:136-143.
29. Denti P, Pozzoli A, Geretto A, et al. Systolic anterior motion after mitral valve repair: a predictive computational model. *Interact Cardiovasc Thorac Surg*. 2017;25:513-519.

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