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Mitral valve repair for atrial functional mitral regurgitation in patients with chronic atrial fibrillation

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Abstract

OBJECTIVES: Atrial functional mitral regurgitation (MR) has been recently described in patients with chronic atrial fibrillation (AF). However, the results of surgical mitral valve (MV) repair for this type of MR have not been comprehensively reported. Our study aimed to address this deficiency.

METHODS: We retrospectively studied 10 chronic AF patients who underwent MV repair for atrial functional MR with normal left ventricular dimension and preserved left ventricular systolic function. All patients had chronic heart failure (HF) symptoms and at least one prior admission for HF complicated by severe MR.

RESULTS: Ring annuloplasty was performed in all patients; the median ring size was 26 mm (range, 26–30 mm). Concomitant tricuspid valve repair was undertaken in all patients. Preoperatively, left atrial (LA) diameter on the parasternal long-axis view, LA volume index and mitral annular diameter were 52 ± 9 mm, 72 ± 26 ml/m^2 and 33 ± 4 mm, respectively. There was no mortality and no re-admission due to HF during follow-up (range, 10–52 months). MR at the most recent examination was mild or improved in degree in all patients. The LA volume index decreased from the preoperative period, measuring 48 ± 17 ml/m^2 at the most recent period ($P = 0.03$). The New York Heart Association functional class dramatically improved from the preoperative period to the most recent period (from 3.0 ± 0.7 to 1.2 ± 0.4, $P < 0.0001$).

CONCLUSIONS: Our results suggest that MV repair leads to reductions in MR, LA size and HF symptoms, and that it may prevent future HF events in patients with atrial functional MR.

Keywords: Mitral regurgitation • Atrium • Atrial fibrillation

INTRODUCTION

Atrial fibrillation (AF) causes right and left atrial (LA) dilatation, leading to mitral and tricuspid annular dilatation in the absence of left ventricular (LV) dilatation and dysfunction [1–3]. Atrial dilatation has been reported to lead to atrial functional mitral regurgitation (MR) [3]. Atrial functional MR can cause advanced heart failure (HF) and is a potential target for intervention [3]. However, the benefits of surgical correction for atrial functional MR in terms of prognosis and symptom relief have not been well reported, because significant MR due to atrial dilatation without organic mitral valve (MV) changes is not incredibly common in AF patients [1, 2]. In the past several years, we have performed MV repair for atrial functional MR in a number of HF patients with chronic AF. This study aimed to present our surgical results in these patients.

MATERIALS AND METHODS

Patients

We retrospectively studied 10 HF patients with chronic AF who underwent MV repair for non-organic MR at Osaka City General Hospital from October 2008 to August 2014. All patients had the following: (i) chronic AF that had persisted for over 1 year; (ii) chronic, moderate or severe MR; (iii) chronic HF symptoms of at least New York Heart Association (NYHA) functional class 2 and (iv) at least one prior admission for acute decompensated HF complicated by severe MR. Patients with considerable organic abnormalities in the mitral leaflets or subvalvular structures, LV regional wall motion abnormality, LV dilatation (LV diastolic dimension greater than 56 mm for males and 50 mm for females) or an LV ejection fraction less than 56% were excluded from the study.

The limits for normal LV diastolic dimension and LV ejection fraction represent the mean values ± 2 SDs obtained from normal Japanese subjects [4]. We retrospectively studied the patients' clinical characteristics, symptoms and echocardiographic parameters before and after surgery.

The Institutional Review Board of Osaka City General Hospital approved the data analysis for this retrospective study and waived the need for patient consent.

Echocardiography

All patients underwent transthoracic echocardiography (TTE) and transoesophageal echocardiography (TOE) in our echocardiography laboratory the day before the operation. MR grade was evaluated by measuring the colour Doppler jet area, using the Doppler-derived volumetric method or using the proximal isovelocity surface area method according to the previously published guidelines [5]. The LA antero-posterior systolic diameter was measured in the parasternal long-axis view, and the major axes of the LA were measured in the apical four-chamber view. The LA volume index (LAVI) was calculated using the prolate ellipse method [6]. Mitral geometric change was evaluated in mid-systole using the long-axis view of preoperative TOE (Fig. 1). The diameter of the mitral annulus was measured between A2 and P2 (antero-posterior diameter). The leaflet lengths of A2 and P2 were also measured. We measured the posterior leaflet tethering angle (α), the posterior leaflet atriogenic tethering angle (β), the anterior leaflet tethering angle (γ) and tenting height (t) in mid-systole. The posterior leaflet tethering angle (α) was defined as the angle comprising the annular line and the line drawn along the basal posterior leaflet. The posterior leaflet atriogenic tethering angle (β) was defined as the angle comprising the annular line and the line drawn between the posterior annulus and the tip of the posterior leaflet. The anterior leaflet tethering angle (γ) was defined as the angle comprising the annular line and the line drawn along the basal anterior leaflet. Tenting height was determined by the distance between the point of leaflet coaptation and the mitral annular line.

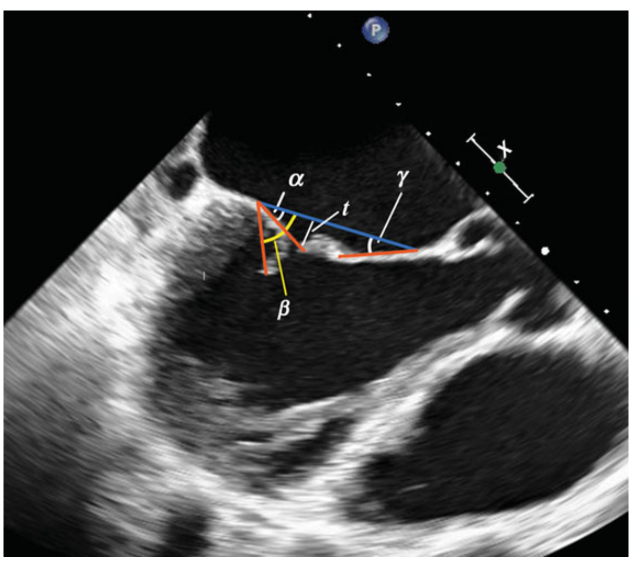

Figure 1: Preoperative transoesophageal echocardiography at a long-axis view in mid-systole. α shows the posterior leaflet tethering angle. β shows the posterior leaflet atriogenic tethering angle (yellow line). γ shows the anterior leaflet tethering angle. The blue line shows the antero-posterior diameter of the mitral annulus (A2–P2). Tenting height was determined by the distance between the point of leaflet coaptation and the mitral annular line (t).

Surgery

Cardiopulmonary bypass was established by ascending aortic cannulation and bicaval venous return through a median sternotomy. The MV was carefully observed via the trans-septal or left-sided atrial approach. All patients underwent MV annuloplasty. After sizing of the intercommissural distance and anterior MV size, an annuloplasty ring (Carpentier-Edwards Physio Ring or Physio Ring II; Edwards Lifesciences, Irvine, CA, USA) was implanted with 2-0 Ethibond sutures. If there was a large gap between the anterior and posterior mitral leaflets, anterior mitral leaflet chordal reconstruction was performed at the surgeon's discretion using artificial chordae and the loop technique [7, 8]. All patients underwent concomitant LA appendage closure and tricuspid annuloplasty with an MC3 ring (Edwards Lifesciences) or a Cosgrove-Edwards annuloplasty ring (Edwards Lifesciences).

Follow-up examination and management

Postoperatively, we followed the patients at our hospital and outpatient clinic as well as by telephone survey. Follow-up was completed by all patients and ranged in duration from 287 to 1566 days (10–52 months; median, 654 days). Postoperative echocardiographic follow-up was performed at our hospital 7–14 days after surgery and then every 3–6 months after discharge. The echocardiographic follow-up duration ranged from 8 to 52 months, with a median of 429 days.

Data collection and statistical analysis

Descriptive statistics used for categorical variables are reported as absolute values and percentages; continuous variables are shown as mean and SD. Continuous data were compared using the paired t-test. Statistical analysis was performed with StatView (SAS Institute, Cary, NC, USA). Statistically significant differences were assumed when P-values were less than 0.05.

RESULTS

Patients' preoperative and perioperative data

The patients' preoperative profiles are given in Table 1. All of the patients were treated with mitral annuloplasty. Two of the patients also underwent artificial chordae replacement with the loop technique. The patients' perioperative data are given in Table 2. None of the patients' clinical courses were complicated by bleeding necessitating re-exploration, systolic anterior motion of the mitral leaflets, deep sternal wound infection or acute renal failure requiring haemofiltration. The 30-day mortality rate was 0%. Two patients who had undergone a maze procedure temporally recovered sinus rhythm. However, all of the patients had persistent AF at the most recent follow-up.

Table 1: Patient profiles

Preoperative characteristics	$N = 10$
Sex (male/female)	5/5
Median age (years)	74 ± 5 (66–79)
BSA (m^2)	1.5 ± 0.2 (1.2–1.7)
Heart rate (bpm)	82 ± 25
Presenting symptom	10 (100%)
Comorbidities	
Systemic hypertension	4 (40%)
Diabetes mellitus	1 (10%)
Ischaemic heart disease	1 (10%)
Chronic renal failure	3 (30%)
COPD	1 (10%)
Drug therapy	
ACE or ARB	4 (40%)
Beta-blocker	5 (50%)
Anti-aldosterone	3 (30%)
Loop diuretics	8 (80%)
Digitalis	3 (30%)
NYHA	
II	2 (20%)
III	6 (60%)
IV	2 (20%)

BSA: body surface area; COPD: chronic obstructive pulmonary disease; ACE: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor antagonists; NYHA: New York Heart Association.

Table 2: Perioperative patient data

	$N = 10$
MAP	10 (100%)
Chordal replacement of loop	2 (20%)
TAP	10 (100%)
Maze	2 (20%)
Left atrial appendage closure	10 (100%)
Mitral annuloplasty ring size (mm)	
26	6 (60%)
28	2 (20%)
30	2 (20%)
Postoperative MAD (A2–P2) (length, mm)	24 ± 1.3

MAP: mitral annuloplasty; TAP: tricuspid annuloplasty; MAD: mitral annular diameter.

Valve-related morbidity and mortality

None of the patients had valve-related mortality in the follow-up period. Only 1 patient experienced valve-related morbidity: an embolism in the lower extremities 22 months after surgery. None of the patients developed a cerebral embolism or needed a reoperation.

Postoperative changes in New York Heart Association functional class

There was no re-hospitalization for HF during postoperative follow-up. Figure 2 shows patients' NYHA functional class before and after surgery. NYHA functional class was dramatically improved from the preoperative period to the most recent period (range, 10–58 months postoperatively; from 3.0 ± 0.7 to 1.2 ± 0.4, $P < 0.0001$).

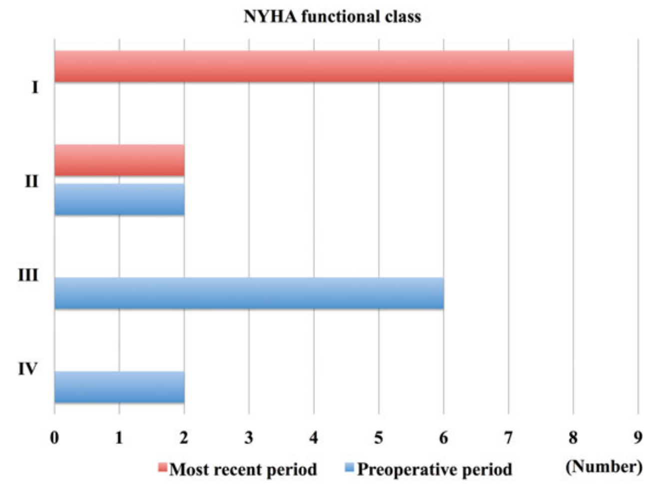

Figure 2: Changes in NYHA functional class before and after surgery. After surgery, NYHA functional class was dramatically improved (from 3.0 ± 0.7 to 1.2 ± 0.4, $P < 0.0001$). NYHA: New York Heart Association.

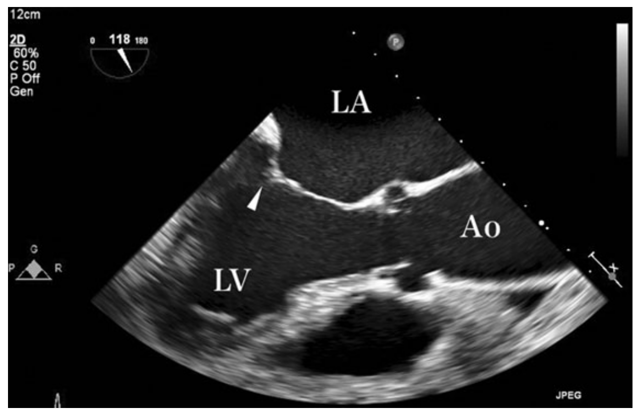

Figure 3: Preoperative transoesophageal echocardiography: long-axis view in mid-systole. The white arrowhead shows coaptation between the anterior and posterior leaflets. Mitral annular dilatation and LA dilatation are shown. LA: left atrium; LV: left ventricle; Ao: ascending aorta.

Perioperative transthoracic echocardiography data

A characteristic TOE image is shown in Fig. 3. In this figure, a dilated LA can be seen without LV dilatation; only a tiny coaptation is present between the anterior and posterior leaflets. The echocardiographic data for all of the cases are given in Table 3. All patients fell within the normal range of LV diastolic and systolic dimensions, and no patients had LV systolic dysfunction. The preoperative MR grade after medical treatment was moderate in 6 patients and severe in 4 patients. Mitral geometric change of preoperative TOE is given in Table 4. Dilatation of the mitral annulus (33 ± 4 mm), small size of the posterior leaflet (9.2 ± 2.6 mm) and high degree of posterior leaflet atriogenic tethering angle (48 ± 16°) are shown. Postoperative changes in TTE data relative to the preoperative values are shown in Fig. 4. LV diastolic dimension, LV systolic dimension and LV ejection fraction were not significantly changed from the preoperative period to the postoperative period (1–2 weeks after surgery) or the most recent period (the mean LV diastolic dimension was 45 ± 6 mm preoperatively, 45 ± 6 mm 1–2 weeks postoperatively and 46 ± 5 mm

Table 3: The echocardiographic data before surgery

Case	Dd (mm)	Ds (mm)	IVS (mm)	PW (mm)	EF (%)	LAD (mm)	LAVI (ml/m^2)	Estimated RVSP (mmHg)	MR (after medical treatment)
1	50	35	11	12	56	65	116	24	Severe
2	49	35	11	11	55	58	97	35	Moderate
3	52	34	13	13	63	60	105	51	Severe
4	50	35	6	7	57	53	61	23	Severe
5	31	18	10	10	74	50	52	46	Moderate
6	44	25	9	9	75	43	64	45	Moderate
7	45	25	12	9	76	57	72	43	Moderate
8	40	27	8	8	61	35	38	24	Moderate
9	44	27	11	12	69	46	45	26	Moderate
10	40	25	8	10	68	51	67	26	Severe
Mean	45	29	10	10	65	52	72	34	

Dd: diastolic dimension; Ds: systolic dimension; IVS: interventricular septum; PW: posterior wall; EF: ejection fraction; LAD: left atrial diameter at the long-axis parasternal view; LAVI: left atrial volume index; RVSP: right ventricular systolic pressure; MR: mitral regurgitation.

Table 4: Each parameter of mitral geometry in the preoperative transoesophageal echocardiography

Case	MAD (mm)	Leaflet length		*t* (mm)	Anterior leaflet	Posterior leaflet	
		A2 (mm)	P2 (mm)		γ (°)	α (°)	β (°)
1	35	31	7.9	4.9	20	26	63
2	34	30	8.5	4.4	21	36	56
3	31	28	9.1	6.4	21	45	78
4	38	32	11	3.0	20	25	51
5	33	24	13	3.3	12	16	25
6	33	27	12	4.0	15	16	30
7	39	29	10	3.4	21	17	41
8	27	21	6.3	3.3	16	12	37
9	25	20	5.5	2.0	19	26	45
10	34	28	7.8	3.4	23	18	52
Mean	33	27	9.2	3.8	19	24	48

MAD: mitral annular diameter; *t*: tenting height; α: posterior leaflet tethering angle; β: posterior leaflet atriogenic tethering angle; γ: anterior leaflet tethering angle.

at the most recent period; the mean LV systolic dimension was 29 ± 6 mm preoperatively, 31 ± 2 mm 1–2 weeks postoperatively and 30 ± 3 mm at the most recent period; the mean LV ejection fraction was 65 ± 8% preoperatively, 59 ± 6% 1–2 weeks postoperatively and 64 ± 5% at the most recent period; Fig. 4A).

In contrast, the LAVI significantly decreased from the preoperative period (72 ± 26 ml/m^2) to the postoperative period (1–2 weeks after surgery; 49 ± 19 ml/m^2) and the most recent period (48 ± 17 ml/m^2; $P < 0.03$; Fig. 4B). The mean mitral pressure gradient was 3.8 ± 1.4 mmHg 1–2 weeks after surgery and 3.8 ± 1.3 mmHg at the most recent period. In all patients, postoperative MR was mild or improved 1–2 weeks after surgery and at the most recent period. In all patients, the degree of tricuspid regurgitation was mild or improved 1–2 weeks after surgery and at the most recent period.

DISCUSSION

We examined chronic AF patients treated with MV repair for non-organic MR with LA dilatation but without significant LV dilatation or systolic dysfunction. Our results showed that MV repair led to a reduction in MR, produced symptom relief and was effective in preventing subsequent HF events in these patients. We observed decreases in LA size after MV repair, which were maintained at the most recent period even if sinus rhythm was not restored.

AF has an estimated prevalence of ~1.6% among adults aged ≥40 years in Japan. The prevalence of AF gradually increases with advancing age, from 0.2% at 40–59 years to 1.5% at 60–79 years and 2.8% at ≥80 years [9]. One study found a 6.4% incidence rate of functional MR in patients initially presenting with AF [3]. Therefore, it is likely that MR is much more common in AF patients than originally thought. Because the number of AF patients will continue to increase with ageing of the population, we presume that the number of patients who require mitral surgery, such as those in our present study, will increase in the near future.

In the guidelines for the surgical and interventional treatment of valvular heart disease (JCS 2012), there are separate surgical indications for organic MR and functional MR. In cases of organic MR, surgery is only indicated for severe MR. In cases of functional MR, surgery can be indicated for moderate or severe MR if the patient needs coronary artery bypass grafting or has symptoms related to HF. However, there are no recommendations regarding

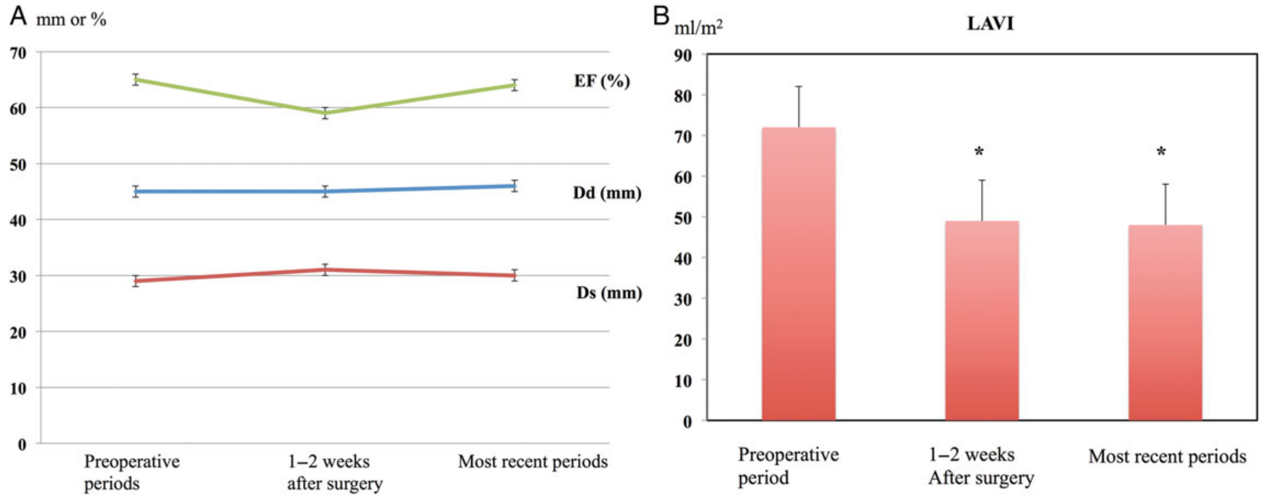

Figure 4: (**A**) Changes in Dd, Ds and EF before and after surgery are shown; there were no significant changes in these parameters from the preoperative period to the postoperative period (1–2 weeks after surgery) or the most recent period. Dd: left ventricular diastolic dimension; Ds: left ventricular systolic dimension; EF: ejection fraction. (**B**) Changes in the LAVI before and after surgery are shown. The LAVI significantly decreased from the preoperative period to the postoperative period (1–2 weeks after surgery) and the most recent period. *$P < 0.03$, comparison between the preoperative value and the value 7–14 days after surgery or at the most recent period. LAVI: left atrium volume index.

AF or LA dilatation in these guidelines; essentially no attention has been paid to surgical interventions for HF caused by atrial functional MR. Therefore, we focused on this matter in this study. In our study, 6 patients were classified with moderate MR after medical treatment, although all of them had experienced at least one prior admission for acute decompensated HF complicated by severe MR. After surgery, none of the patients in our series required readmission for worsened HF. Therefore, we conclude that MV repair was effective and acceptable in preventing HF events in patients with atrial functional MR, even those with a moderate grade of preoperative MR.

Several studies have previously shown that a dilated LA, particularly as a result of AF, is associated with mitral annular dilatation [10]. MR due to mitral dilatation induced by LA dilatation in AF patients is called atrial functional MR. It is distinguished from MR due to secondary leaflet restriction associated with LV dilatation and systolic dysfunction [2, 11]. Vohra *et al*. [12] reported the surgical results of 20 patients with MR secondary to AF. However, the mean LV diastolic dimension was 56 mm in these patients, which was larger than the mean LV diastolic dimension in our cases, and 7 patients had LV dysfunction in their report. Therefore, we consider it uncertain as to whether the MR in their report was purely caused by AF-induced mitral annular dilatation.

Whether annular dilatation can be the sole cause of MR remains controversial. Otsuji *et al*. demonstrated that isolated annular dilatation does not usually cause significant MR in AF patients [10, 13, 14]. However, Kihara *et al*. [1], Kilic *et al*. [2] and Ring *et al*. [15] showed that significant MR in AF patients can be caused by mitral annular dilatation alone, although this was infrequent. In a recent study, Silbiger *et al*. [16] hypothesized that enlargement of the LA has the potential to increase mitral leaflet tethering and to worsen MR through a mechanism unrelated to LV remodelling, which these authors termed atriogenic leaflet tethering.

With advanced LA enlargement, the posterior wall of the LA extends behind the basal posterior wall of the LV, and the posterior mitral annulus must be displaced onto the crest of the LV inlet. Backward LA enlargement also leads to inward bending of the basal posterior LV towards the LV outflow tract [17]. As a result, the tip of the posterior mitral leaflet is tethered towards the posterior LV. The posterior leaflet then curves and overlies the muscle layer of the basal posterior LV, and its movement becomes restricted. Such functional restriction of the posterior leaflet has traditionally been called 'hamstringing of the posterior cusp' [18], which may be the same mechanism as atriogenic tethering. Annular dilatation and atriogenic tethering of the posterior leaflet can result in reduction of coaptation, and worsen MR.

Our patients in the present study also had LA dilatation and mitral annular dilatation. Furthermore, our patients had another unique mitral characteristic, which was a small size of the posterior leaflet with atriogenic leaflet tethering. Fernandes *et al*. [19] reported that the mean P2 height in the normal heart was 12 mm. Therefore, our patients appeared to have a relatively small posterior leaflet, with ∼9 mm of P2 height. Gelsomino *et al*. [20] reported that the normal leaflet tethering angles were 24 ± 3° and 34.4 ± 5° at the anterior and posterior leaflets, respectively. The anterior leaflet tethering angle in our patients (∼20°) is similar to previous data [20]. The posterior leaflet tethering angle in our patients (∼20° or slightly higher) is smaller than that found in previous studies [20]. In contrast, the posterior leaflet atriogenic angle was large. A large difference between the posterior leaflet tethering angle and posterior leaflet atriogenic tethering angle in our patients indicates posterior leaflet bending, which can be translated to atriogenic tethering or hamstringing of the posterior leaflet.

Our study has several limitations. First, it was retrospective and involved a small number of patients. Secondly, we performed the maze procedure in only a few patients, because the success rate and benefits of this procedure are still unknown in patients with chronic AF and advanced LA dilatation. However, MV repair led to decreases in LA size after surgery that were maintained through the most recent period even when sinus rhythm was not restored. These issues should be addressed by future prospective studies containing larger numbers of patients and comparing various therapeutic options. Thirdly, the echocardiographic evaluation of MR and systolic MV morphology in a single beat may be inaccurate in AF patients; to mitigate this, we were careful to select a beat occurring after two serial beats with average RR intervals [21].

ORIGINAL ARTICLE

Finally, because it is difficult to accurately evaluate LV diastolic function in AF patients [22], we did not necessarily evaluate this as a possible cause of HF in our AF patients with preserved LV ejection fraction.

In summary, our results suggest that MV repair leads to reductions in MR, LA size and HF symptoms, and that it may prevent future HF events in patients with atrial functional MR.

Conflict of interest: none declared.

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eComment. Atrial mitral regurgitation: a new paradigm to understand

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I have read with great interest the article by Takahashi *et al.* [1]. This article is particulary relevant in the context of atrial fibrillation (AF) as generator of 'functional' mitral regurgitation (MR). The concept of a large left atrium related to mitral annulus enlargement is not new. This was previously reported in 1996 [2]. What is relatively new is the idea that AF may cause MR itself. Atrial MR means MR with Carpentier's type I (dilated annulus with normal leaflet motion), and normal anatomic findings (functional MR without tenting nor tethering effects on the mitral leaflets). Atrial MR is different to functional MR because the left ventricle is almost normal in dimensions in most of the cases. In addition, there is no left ventricular remodelling. Gertz *et al.* [3] have clearly shown an important reduction in MR after AF was treated and normal sinus rhythm was regained. Vohra *et al.* have demonstrated similar results utilizing only mitral annuloplasty [4]. Atrial MR seems to be directly related to a process of annular dilation. The question is why the left atrium is so great that even mitral annular dilation allows functional MR on a 'normal' mitral valve with no organic dammage. Surprisingly, all 10 patients reported by Takahashi *et al.* were non-organic MR [1]. By contrast, the strong relationship between AF and rheumatic mitral valve disease is well known [5]. Atrial tissue in mitral valve disease is thicker, harder and atrial fibrosis is present. In fact, atrial fibrosis is mainly responsible for AF in these cases. Rheumatic mitral valve dammage explains the great impact on left atrial size. In these current cases, the Cox-maze procedure is indicated for AF treatment in addition to mitral valve procedure. At this point, the discussion becomes even more interesting. According to data obtained by several authors [1-4], the only mitral valve annulus dilation is a strong predictor for atrial MR in AF. Whether or not mitral valve ring annuloplasty is enough to treat atrial MR remains to be elucidated by larger follow-ups.

Conflict of interest: none declared.

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