Renal Function Improvement Correlates with 3D Echocardiographic Findings: Effect of Surgical Mitral Valve Repair

Chenchen Wang

Shanghai Jiao Tong University Medical School Affiliated Ruijin Hospital

Wenbo Yang

Shanghai Jiao Tong University Medical School Affiliated Ruijin Hospital

Zhongwei Shi

Shanghai Jiao Tong University Medical School Affiliated Ruijin Hospital

Yuehua Fang (✉ fyh11372@rjh.com.cn)

Shanghai Jiao Tong University Medical School Affiliated Ruijin Hospital <https://orcid.org/0000-0002-7390-6693>

Research Article

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Abstract

Purpose

The relationship between surgical treatment of mitral regurgitation (MR) and renal function is not well described. We sought to evaluate renal function before and after surgical mitral valve repair (SMVR) in degenerative significant MR.

Methods

Patients with primary severe (4+) MR and preserved left ventricular ejection fraction (LVEF) who underwent surgical mitral valve repair, evaluated by a cutting-edge 3-dimensional (3D) echocardiographic probe were included in this study. Three CKD-EPI equations were used to calculate estimated glomerular filtration rate (eGFR) before surgery and before patients discharge. Forty patients with baseline lower mean eGFR were studied.

Results

Measurements substantiated statistically remarkable improvements in eGFR ($P < 0.001$), multivariable linear regression modeling demonstrated a strong association between increase of eGFR and reduction of MR ($P = 0.003$), reduction of pulmonary arterial systolic pressure (PASP, $P = 0.018$), as well as increase of forward stroke volume (FSV, $P = 0.02$), regardless of the reduction of LVEF, left ventricular global longitudinal strain (GLS) and left atrial ejection fraction (LAEF).

Conclusion

Renal function improves after surgical mitral valve repair in patients with degenerative severe mitral regurgitation and preserved LVEF, despite of cardiac functional worsening.

Introduction

Mitral regurgitation (MR) is the most frequent valvular heart disease in the general population, and the most common aetiology of organic MR is degenerative mitral valve (DMV) disease, which leads to mitral valve prolapse[1]. Surgical mitral valve repair (SMVR) is the preferred medical procedure for most of the severe DMV disease, with better short-term and long-term survival and quality of life than mitral valve replacement[2, 3]. Currently, the indication of SMVR is based on several clinical findings, such as heart failure symptoms, significant left atrial (LA) dilatation, poor left ventricular systolic function, recent atrial fibrillation onset or pulmonary arterial systolic pressure (PASP) ≥ 50 mm Hg, according to the American[4] and European practice guidelines [5]. However, renal function is not mentioned. The heart and kidneys are the organs tasked with maintaining internal homeostasis. MR may lead to significant hemodynamic

abnormalities, as a result, impair renal function[6]. It has been shown that even slight decrease of renal function after cardiac surgery is associated with a significant increase in mortality[7, 8]. What's more, relevant studies mostly focused on different stages of renal dysfunction significantly affect the prognosis of SMVR[9, 10], and acute kidney injury (AKI) after SMVR has been reported [11, 12]. Recently, evidence showed that renal function can be restored to some extent after percutaneous mitral valve repair[13] (PMVR). However, no studies have found improvement in renal function after SMVR. Based on the observations of our clinical works, we found a significant decrease of estimated glomerular filtration rate (eGFR) in severe DMV patients, and it will be improved after successful SMVR. Thus, we aimed to investigate the changes of eGFR before and after SMVR, trying to analyze the consequences and reasons behind.

Materials And Methods

Study Population

This retrospective analysis of a prospectively collected database was approved by an institutional review board of Ruijin Hospital affiliated to Shanghai Jiao Tong university school of medicine , and the need for individual consent was waived. Between April 2020 and July 2021, the medical records of 65 consecutive patients who received mitral valve repair in a single tertiary referral hospital were reviewed. We excluded patients who had undergone concomitant coronary artery bypass surgery (n = 11), tricuspid valve surgery (n = 4), or aortic valve surgery (n = 3), had left ventricular ejection fraction (LVEF) <60% (n = 6), or unsatisfactory imaging quality (n = 1). The final cohort comprised a total of 40 patients.

Echocardiographic Evaluation

Echocardiographic studies were performed using GE Vivid E95 system (GE Vingmed Ultrasound, Horten, Norway), equipped with a 1.4/2.8 MHz 4V transducer. Patients were studied in the left decubitus position. Measurements of the left ventricular (LV) and LA dimensions, LVEF , LA volume index (LAVI) , LA reservoir function (left atrial emptying fraction, LAEF), LA pump function (LAEF-active), LA conduit function (LAEF-passive), LA strain (LAS), transmitral early diastolic peak flow velocity (E), late diastolic peak flow velocity (A), peak early diastolic mitral annular velocity (e'), mean transmitral gradient (MTMG), PASP and left ventricular global longitudinal strain (GLS) were made in accordance with the current recommendations of the American Society of Echocardiography[14]. LVEF was measured using Simpson's biplane method (apical 4- and 2-chamber views) spanned the time interval between two consecutive Q waves, care was taken to obtain true apical images using real-time three-plane and standard anatomic landmarks in each view and not to foreshorten the left atrium, allowing a more reliable delineation of the LV endocardial border. For LAVI, LAEF, LAEF-active, LAEF-passive and LAS analysis, atrial endocardium was traced automatically during breath hold and with a stable electrocardiographic recording (show as **Figure 1 and 2**), satisfactory tracking throughout the cardiac cycle was checked with manual adjustments as needed. GLS was measured using the automated function imaging (AFI) technique with apical tri-plane images. Mitral regurgitation volume (Rvol) was measured by proximal isovelocity surface area (PISA) method

(show as **Figure 3**), and the forward stroke volume (FSV) was calculated with quantitative pulsed doppler method. MTMG was measured automatically by the tracing of mitral valve inflow pulsed doppler spectrum. The frame rate was set at 60 to 80 frames per second and three consecutive heart cycles were recorded and averaged. For patients with atrial fibrillation, five consecutive heart cycles were recorded and averaged. The analysis of all the images and videos recorded was performed off-line by a single experienced and independent echocardiographer, who was not involved in the image acquisition process and was blinded to the other echocardiographic parameters, using a commercially available semiautomated 3-dimensional software program (EchoPac, GE, Milwaukee, Wisconsin).

Plasma Creatinine and Cystatin C Level and Determination of Estimated GFR

The CKD-EPI equation was used to calculate eGFR, based on plasma creatinine and cystatin C measurements, before surgery and prior to patients discharge(at least one week after surgery). This formula is developed by Levey et al[15] and adjusted for each 1.73 m² of body surface area, and modified by Inker et al [16] measured by serum creatinine and cystatin C and both together:

CKD-EPI creatinine equation

Female ≤ 0.7 : $144 \times (\text{Scr}/0.7)^{-0.329} \times 0.993^{\text{Age}}$

Female > 0.7 : $144 \times (\text{Scr}/0.7)^{-1.209} \times 0.993^{\text{Age}}$

Male ≤ 0.9 : $141 \times (\text{Scr}/0.9)^{-0.411} \times 0.993^{\text{Age}}$

Male > 0.9 : $141 \times (\text{Scr}/0.9)^{-1.209} \times 0.993^{\text{Age}}$

CKD-EPI cystatin C equation

Female or male ≤ 0.8 : $133 \times (\text{Scys}/0.8)^{-0.499} \times 0.996^{\text{Age}}$ [$\times 0.932$ if female]

Female or male > 0.8 : $133 \times (\text{Scys}/0.8)^{-1.328} \times 0.996^{\text{Age}}$ [$\times 0.932$ if female]

CKD-EPI creatinine-cystatin C equation

Female $\text{Scr} \leq 0.7$

$\text{Scys} \leq 0.8$: $130 \times (\text{Scr}/0.7)^{-0.248} \times (\text{Scys}/0.8)^{-0.375} \times 0.995^{\text{Age}}$

$\text{Scys} > 0.8$: $130 \times (\text{Scr}/0.7)^{-0.248} \times (\text{Scys}/0.8)^{-0.711} \times 0.995^{\text{Age}}$

Female $\text{Scr} > 0.7$

$\text{Scys} \leq 0.8$: $130 \times (\text{Scr}/0.7)^{-0.601} \times (\text{Scys}/0.8)^{-0.375} \times 0.995^{\text{Age}}$

$\text{Scys} > 0.8$: $130 \times (\text{Scr}/0.7)^{-0.601} \times (\text{Scys}/0.8)^{-0.711} \times 0.995^{\text{Age}}$

Male Scr $\leq$ 0.9

Scys $\leq$ 0.8: $135 \times (\text{Scr}/0.9)^{-0.207} \times (\text{Scys}/0.8)^{-0.375} \times 0.995^{\text{Age}}$

Scys $>$ 0.8: $135 \times (\text{Scr}/0.9)^{-0.207} \times (\text{Scys}/0.8)^{-0.711} \times 0.995^{\text{Age}}$

Male Scr $>$ 0.9

Scys $\leq$ 0.8: $135 \times (\text{Scr}/0.9)^{-0.601} \times (\text{Scys}/0.8)^{-0.711} \times 0.995^{\text{Age}}$

Scys $>$ 0.8 $135 \times (\text{Scr}/0.9)^{-0.601} \times (\text{Scys}/0.8)^{-0.711} \times 0.995^{\text{Age}}$

Scr indicates serum creatinine, and Scys, serum cystatin C; (unit: mg/dL).

Statistical Analysis

The Kolmogorov–Smirnov test was used to determine the normal distribution for each variable. Continuous counts are presented as means and standard deviations (SDs), or medians (25th to 75th percentiles), where appropriate, based on whether a given dataset had a normal distribution or not. Categorical measures were summarized as proportions (%) . The data were evaluated using the t-test or MannWhitney test, where appropriate. Mean changes in eGFR between baseline and before discharge for the overall cohort were assessed using paired t tests. Paired echocardiographic core laboratory measurements were analyzed between baseline and prior to discharge, and multivariable linear regression modelling were fit to evaluate the association between changes of echocardiographic measurements and eGFR. All p values and confidence intervals (CIs) were calculated using 2-sided tests, and a p value <0.05 was used to declare statistical significance for each analysis. All statistics were performed using the standard statistical software package (SPSS 22.0, SPSS Inc, Chicago, Illinois).

Results

The baseline clinical characteristics of the overall study cohort (n=40) are shown in Table 1. Most of the patients were male, the average age is 59.4 years, and the average BMI is 24.4 kg/m². There were a high prevalence of hypertension and pulmonary hypertension, as well as a fraction of atrial fibrillation. Almost all of the patients had heart failure symptoms, that is 39(97.5%) patients had NYHA classification $\geq$ II. Half of the patients received antihypertensive therapy, however, the arterial systolic pressure still remained high(mean arterial systolic pressure 132.6 mm Hg). 9 patients (22.5%) received diuretic treatment, 4 patients underwent anticoagulant therapy, 2 patients took antidiabetic medication and 1 patient had statins. Most of the patients underwent SMVR through median sternotomy, only 6(15.0%) patients had minimally invasive thoracotomy.

Table 1
Baseline Clinical Characteristics of the Overall Study Cohort

Demographic data	Patients(n=40)
Age (years)	59.4±13.3
Gender, male (%)	27 (67.5)
Heart rate (beat/min)	81.6±10.9
Diabetes mellitus (%)	4 (10.0)
Hypertension (%)	24 (60.0)
Arterial systolic pressure (mm Hg)	132.6±17.0
Arterial diastolic pressure (mm Hg)	75.4±12.8
Atrial fibrillation (%)	5 (12.5)
Pulmonary hypertension (%)	21 (52.5)
BMI (kg/m ²)	24.4±2.7
BSA (m ²)	1.72±0.14
Medical therapy	13(32.5)
Angiotensin-converting enzyme inhibitors	3(7.5)
or angiotensin receptor blockers	4(10.0)
β Blockers	9(22.5)
Calcium channel blockers	1(2.5)
diuretics	4(10.0)
Statins	2(5.0)
Anticoagulants	
antidiabetics	
NYHA classification	1(2.5)
I	24(60.0)
II	14(35.0)
III	1(2.5)
IV	

BMI, body mass index; BSA, body surface area; NYHA classification, New York Heart Association classification.

Demographic data	Patients(n=40)
mini-thoracotomy (%)	6 (15.0)
BMI, body mass index; BSA, body surface area; NYHA classification, New York Heart Association classification.	

Preoperatively, all the patients had MR grade 4+ by quantitative assessment by the echocardiography core laboratory: the mean regurgitant volume is 68.7ml and the regurgitant fraction is 58.2%. Preoperative and postoperative echocardiographic measurements of the overall study cohort are shown in Table 2, almost all the parameters listed were changed significantly after surgery. Quantitative analysis of MR regurgitant volume and MR regurgitant fraction showed a significant reduction in both measurements after SMVR ($P<0.001$), and all the classifications of Rvol were $\leq 1+$ (range: 0-25.8ml). The measurements of cardiac chambers and volume, including LVEDD, LVESD, LAD and LAVI had decreased significantly ($P<0.01$). Interestingly, the cardiac functional parameters as for LAEF, LAS, LVEF and GLS had declined significantly after surgery, but LAEF-passive and LAEF-active separately remained relatively stable. LV diastolic measurements increased significantly including E velocity, E/A, E/e' sep, E/e' lat and E/e' avg, the same to MTMG. Pulmonary arterial systolic pressure decreased significantly after the procedure ($P=0.005$).

Table 2
Echocardiographic Measurements of the Overall Study Cohort

Parameter	Before surgery	After surgery	P value
RVol (ml)	68.7±20.5	7.0±7.1	<0.001
RF (%)	58.2±0.9	10.1±0.9	<0.001
LVEDD (mm)	57.9±5.5	50.1±5.7	<0.001
LVESD (mm)	34(32 to 37)	32 (30 to 34.8)	0.002
LVEF (%)	69 (67.3 to 72.8)	63 (60 to 68)	<0.001
FSV (ml)	48.0±10.0	59.6±16.7	<0.001
GLS (%)	-18.3 (-20.3 to -16.0)	-12.7 (-14.2 to -10.4)	<0.001
LAD (mm)	47.5 (44 to 52)	43 (40 to 46)	<0.001
LAVI (ml/m ²)	59.5 (46.4 to 81.8)	47.7 (36.1 to 64.1)	<0.001
LAEF (%)	39.9±10.9	32.5±8.9	<0.001
LAEF-active (%)	18.7±9.4	15.2±11.4	0.094
LAEF-passive(%)	20.5±8.3	18.4±9.4	0.295
LAS (%)	12.5±8.1	8.8±6.6	0.008
E (cm/s)	125.7±28.9	140.2±35.4	0.045
A (cm/s)	62.9±29.4	72.4±50.8	0.158
E/A	1.6 (1.25 to 2.2)	1.3 (1.0 to 1.7)	0.016
e' sep (cm/s)	7.9±2.4	6.4±1.7	0.001
e' lat (cm/s)	12.1±3.4	9.1±2.5	<0.001
E/e' sep	14.6 (11.8 to 21.5)	20.7 (17.2 to 29.2)	0.002
E/e' lat	11.1±3.9	16.0±5.7	<0.001
E/e' avg	14.4±0.9	19.7±1.0	<0.001
PASP (mm Hg)	38 (34 to 52.8)	33.5 (30 to 40.8)	0.005
MTMG(mm Hg)	1.9±0.8	3.1±1.3	<0.001
e' sep, septal peak early diastolic mitral annular velocity; e' lat, lateral peak early diastolic mitral annular velocity; E/e' avg, average value of the sum of E/e' sep and E/e' lat.			

Baseline and postoperative serum creatinine and cystatin C measurements are shown in Table 3. Compared to preoperative measurements, they were both decreased significantly. Based on their values,

eGFR were calculated from three different CKD-EPI formulas. Results were consistent among three different calculations, namely postoperative mean eGFR had increased significantly. For CKD-EPI creatinine equation, the mean eGFR preoperatively were 83.7 ± 26.5 mL/min/1.73 m², compared with 91.0 ± 27.9 mL/min/1.73 m² of the postoperative measurements ($P < 0.001$). For CKD-EPI cystatin C equation, the preoperative mean eGFR were 70.5 ± 25.1 mL/min/1.73 m², compared with 78.2 ± 26.5 mL/min/1.73 m² of the postoperative measurements ($P = 0.001$). For CKD-EPI creatinine-cystatin C equation, the mean eGFR had increased from 75.3 ± 24.6 mL/min/1.73 m² preoperatively to 83.3 ± 27.2 mL/min/1.73 m² postoperatively ($P < 0.001$).

Table 3
Serum Creatinine and Cystatin C Concentration as well as eGFR Calculated with
Three Methods of the Overall Study Cohort.

CKD-EPI equation	Before surgery	After surgery	P value
serum creatinine (mg/dL)	0.90(0.79 to 1.03)	0.84(0.73 to 0.96)	<0.001
serum cystatin C (mg/dL)	1.08(0.93 to 1.30)	0.96(0.86 to 1.15)	0.001
Scr(mL/min/1.73 m ²)	83.7 ± 26.5	91.0 ± 27.9	<0.001
Scys(mL/min/1.73 m ²)	70.5 ± 25.1	78.2 ± 26.5	0.001
Scr-Scys(mL/min/1.73 m ²)	75.3 ± 24.6	83.3 ± 27.2	<0.001

Multivariable linear regression modelling was made to investigate the relationship between changes of eGFR and echocardiographic measurements. Changes of eGFR was calculated by the difference value of postoperative and preoperative calculations, based on the CKD-EPI creatinine-cystatin C equation.

Changes of different echocardiographic parameters were calculated by each of the difference value of postoperative and preoperative measurements.

The results are shown in Table 4. Data shows that several echocardiographic parameters were associated with the increase of eGFR, including Rvol, FSV and PASP. Partial regression coefficients (β) indicate that smaller the difference value of Rvol, bigger the increase of eGFR, in other words, reduction of Rvol is consistent with increase of eGFR. It is the same situation about PASP, reduction of PASP is consistent with increase of eGFR. Moreover, FSV is in positive correlation with increase of eGFR.

Table 4
Multivariable linear regression model between
changes of eGFR and echocardiographic
measurements

variables	β	95% CI	P value
Rvol	-0.204	(-0.336, -0.071)	0.004
FSV	0.186	(0.030, 0.341)	0.021
PASP	-0.215	(-0.412, 0.018)	0.034
LASr	-0.057	(-0.308, 0.194)	0.644
LAVI	-0.048	(-0.230, 0.134)	0.594
LAEF	0.138	(-0.163, 0.440)	0.355
LVEF	0.017	(-0.394, 0.429)	0.932
LVEDD	-0.242	(-0.938, 0.454)	0.482
LVESD	0.140	(-0.785, 1.064)	0.759
GLS	0.062	(-0.249, 0.373)	0.686
MTMG	0.838	(-0.729, 2.406)	0.283

Discussion

The main findings of this study are (1) renal function could improve after successful SMVR; and (2) increase of forward stroke volume, reduction of regurgitant volume, and decrease of pulmonary arterial systolic pressure are associated factors for the increase of renal function.

Patients with severe MR who develop an LVEF $\leq 60\%$ have already developed LV systolic dysfunction[17, 18], the whole study cohort comprises LVEF $>60\%$, and the mean global longitudinal strain is -18.3% , which is in the normal range better than the lower limit of -16.7% in men and -17.8% in women[19], also indicates a normal LV systolic function before surgery, thus there is a high likelihood of successful and durable SMVR. Whereas, left-sided chambers dilated along with LV diastolic function impaired: the mean E value is 125.7 cm/s, because patients with severe MR have a dominant early filling due to increased diastolic flow across the mitral valve (E velocity is usually ≥ 1.2 m/s)[20]; E/e' avg is 14.4, which is a sign of LV diastolic dysfunction[21]. MR increased left atrial preload and pressure, and the left atrium is extremely sensitive to sustained volume and pressure overload secondary to increased LV filling pressures[22], as a result LA function damage ensued: LAEF, LAEF-passive, LAEF-active which separately represent LA reservoir, conduit and pump functions decreased beyond the normal range, LA global longitudinal strain decreased significantly as well. In addition, the stepwise backward effects of loss in LA functional properties lead to a reduction in lung vessel compliance and vascular remodelling, triggering pulmonary hypertension[23].

Initiated by Carpentier in 1983, SMVR has been validated as the optimal treatment to manage DMV disease with indications for surgery, aiming to preserve mitral valve function and facilitate cardiac reverse remodeling[24, 25]. Our results show that an overall successful SMVR were achieved, with Rvol decreased to $\leq 1+$ and mean MTMG maintained in 3.1 mm Hg, which was comparable with those in previous studies[25, 26]. Mean MTMG increased because of mitral annuloplasty, mainly the cause of relatively small orifice. Also, cardiac adverse remodeling were reversed, as a result of the correction of fundamental significant MR. Noteworthy, LA and LV functional parameters declined, probably the reasons of significant hemodynamic (load and pressure) changes and cardiac injury during the operation.

As mentioned before, MR could result in significant hemodynamic turbulence and damage renal function[11], consequences will be serious if the magnitude of regurgitant volume is severe. There is a concept called “regurgitation begets regurgitation”[4], which is the initial MR causes LV dilatation, generating further destruction to the valve apparatus, resulting in more severe MR and further LV dilatation, thus initiating a perpetual cycle of ever-increasing LV volumes and MR. In view of the 4+ Rvol and heart failure symptoms, ever-increasing damage to renal function had already appeared before SMVR. The mean eGFR calculated with CKD-EPI creatinine-cystatin C equation is 75.3 mL/min/1.73 m², which is of more than 16% decline compared to the lower limiting value of 90 mL/min/1.73 m².

The accurate evaluation of renal function is difficult, especially in those patients undergoing cardiac surgery. To guarantee a more reliable result, we calculated glomerular filtration rate with three dependable CKD-EPI equations, the results were consistent among three methods and increased significantly after surgery. The most widely used calculations are based on serum creatinine. Serum creatinine is freely filtered and not re-absorbed, but partly secreted into the tubules from the peritubular capillaries. Hence, more creatinine is cleared by the kidneys than by glomerular filtration alone. In contrast, cystatin C is a cysteine protease inhibitor produced by all the internal nucleated cells, which is freely filtered at the glomerulus, and not secreted by the tubules. While muscle mass varies considerably even among healthy persons, humans have essentially the same number of nucleated cells per body surface area unit. Therefore, cystatin C level is relatively independent of body mass, diet, drugs, infections or inflammatory states. Generally speaking, the CKD-EPI creatinine equation overestimates the CKD-EPI cystatin C equation by approximately 15–20%[27]. With regard to our results, the former overestimates the latter by approximately 18.7% (mean value 83.7 mL/min/1.73 m² VS 70.5 mL/min/1.73 m²) preoperatively and 16.4% (mean value 91.0 mL/min/1.73 m² VS 78.2 mL/min/1.73 m²) postoperatively. Nevertheless, the most precise formula is the combined creatinine–cystatin C equation, with the best performance in practical application[16]. At least seven days after SMVR, the hemodynamic conditions kept relatively stable, then we investigate the correlation between changes of eGFR and changes of echocardiographic parameters, with the CKD-EPI creatinine–cystatin C equation.

The multivariable linear regression model demonstrated that the increase of eGFR were attributed to reduction of regurgitant volume, increase of forward stroke volume and decline of pulmonary arterial systolic pressure, despite of LA and LV functional worsening. Firstly, correction of mitral valve regurgitation redressed the hemodynamic turbulence, which remedied the damage to renal function.

Secondly, increase of FSV improved the perfusion to kidneys, thus increased the glomerular filtration. Last but not least, decline of pulmonary arterial systolic pressure. The pulmonary vasculature is normally a low-pressure, low impedance system, which creates a low afterload on the right ventricle (RV), increase of PASP impair RV function and result in increase in right atrial pressure, which can cause renal dysfunction as a result of venous congestion[6]. Sustained elevation of LA pressure caused by mitral valve regurgitation passively transmitted backward into the pulmonary veins, which can result in disruption of the vulnerable alveolar-capillary complex, in a process known as alveolar capillary stress failure, leading to capillary leakage and pulmonary edema, thus elevation of PASP[28]. In the initial phases, this lesion is reversible after correction of the root cause[29]. Hence, successful SMVR decreased Rvol, reduced PASP, improved renal function.

Although renal function improved a lot, the evaluated glomerular filtration rate was still less than normal. When should we take actions to restore a normal renal function still remains to be studied.

This study has several limitations. The number of patients was small, partly can be attributed to the strict study criteria. In addition, the time interval between preoperative and postoperative examinations is very short, the main purpose of this study was to evaluate eGFR and echocardiographic parameters before and after SMVR in patients with severe DMV diseases, long-term results were beyond the scope of our investigation, and long-term follow-up study is underway. Nevertheless, large studies are needed to confirm our results, and the prognostic implications of eGFR after SMVR should be demonstrated with a longer clinical follow-up.

Conclusion

Patients with preserved LV function who undergo successful SMVR for degenerative mitral insufficiency might benefit from improvement of the existing declined eGFR, which are associated with correction of significant MR, increased forward stroke volume and decreased pulmonary arterial systolic pressure .

Declarations

Funding

None.

Conflict of interest

The authors declare that they have no conflict of interest.

Ethics approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration

and its later amendments or comparable ethical standards. The study was approved by the Ethics Committee of Ruijin Hospital, Shanghai Jiao tong University School of Medicine.

Availability of data and material

The datasets supporting the results of this article are included within the article.

Author contributions:

YF, CW and ZS conceived and designed the study. CW and WY collected the data and performed analysis. CW drafted the manuscript. All authors critically reviewed data analysis and the manuscript. All authors read and approved the final manuscript.

Disclosures

The authors have no conflicts of interest to declare that are relevant to the content of this article.

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Figures

Figure 1

Preoperative measurements of the LA volume and strain with 3D echocardiography

Figure 2

Postoperative measurements of the LA volume and strain with 3D echocardiography

Figure 3

Preoperative calculation of mitral regurgitation with the PISA method. a: the effective regurgitant orifice; b: tracing of the regurgitant jet and calculation of Rvol