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DISSERTATION

Multislice Computed Tomography for Assessment of Reverse Remodeling of Mitral Valve and Left Ventricle after Surgical Repair of Acquired Posterior Left Ventricular Aneurysms Compared to Anterior Localization

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Meiner liebenswerten und mutigen Mutter Lydia Goldblat

The current manuscript contains concepts, references overview, methods descriptions and partial results of our studies that were recently published as an original article (1) and case reports (2, 3).

Contents

Abbreviations

ADA	aneurysm's defect area
AEDV	aneurysm's end-diastolic volume
AEDVI	aneurysm's end-diastolic volume index
AESV	aneurysm's end-systolic volume
AnAPMA	angle between mitral valve annulus and anterior papillary muscle head
AnPPMA	angle between mitral valve annulus and posterior papillary muscle head
AnAPMD	distance between mitral valve annulus and anterior papillary muscle head
AnPPMD	distance between mitral valve annulus and posterior papillary muscle head
APD	anteroposterior diameter/anteroposteriorer Diameter
BSA	body surface area
CABG	coronary artery bypass grafting
CBP	cardiopulmonary bypass
CD	coaptation distance
CMR	cardiac magnetic resonance
CT	computed tomography
ECG	electrocardiogram
ESVI	end-systolic volume index
ICD	intercommissural diameter
IKD	interkommissuraler Diameter
IMA	Interpapillarmuskelabstand
IMD	interpapillary muscle distance
KD	Kooaptationsdistanz

KF	Koaptationsfläche
LA	left atrium
LAVI	left atrial volume index
LV	left ventricle
LVEDD	left ventricular end-diastolic diameter
LVEDV	left ventricular end-diastolic volume
LVEDVI	left ventricular end-diastolic volume index
LVESV	left ventricular end-systolic volume
LVESVI	left ventricular end-systolic volume index
LVSI	left ventricular sphericity index
MI	myocardial infarction
MK	Mitralklappe
MKAF	Mitralklappenannulus-Fläche
MKSW	Mitralklappenschluss-Winkel
MR	mitral regurgitation
MSCT	multislice computed tomography/Multischicht-Computertomographie
MV	mitral valve
MVAA	mitral valve annulus area
MVCA	mitral valve closure angle
NYHA	New York Heart Association
SVI	stroke volume index
SVR	surgical ventricular repair
TA	tenting area

Figures

* Figures 1, 2, 3, 10 and 11 were already published in:

Solowjowa N, Penkalla A, Dandel M, Novikov A, Pasic M, Weng Y, Falk V, Knosalla C. Multislice computed tomography-guided surgical repair of acquired posterior left ventricular aneurysms: demonstration of mitral valve and left ventricular reverse remodelling.
Interact CardioVasc Thorac Surg 2016;23:383-90.

Tables

Zusammenfassung

Einleitung: Die Beteiligung der Mitralklappe (MK) stellt eine Herausforderung in der chirurgischen Therapie von Aneurysmen des linken Ventrikels (LV) dar, insbesondere der posterioren LV-Wand. Diese Arbeit analysiert die Möglichkeiten der Multischicht-Computertomographie (MSCT) für die Beurteilung des Mitralapparates und des LV als funktionalen Komplex für die Optimierung der chirurgischen Vorgehensweise bei LV-Aneurysmen, die Unterschiede zwischen der posterioren und der anterioren Lokalisation und die mittelfristigen Ergebnisse in beiden Gruppen.

Methodik: Eine chirurgische LV**-**Rekonstruktion (LVR) wurde bei 30 konsekutiven Patienten (m:w = 24:6, Alter median 66.0 Jahre; mean New York-Heart-Association (NYHA)-Klasse 2.98) mit posterioren (Gruppe 1) und 41 konsekutiven Patienten (m:w = 31:10, Alter median 57.7 Jahre; mean NYHA-Klasse 3.01) mit anterioren (Gruppe 2) LV-Aneurysmen durchgeführt. Enddiastolische und endsystolische Volumina des LV wurden zu Körperoberfläche indexiert (LVEDVI/LVESVI). Der MK-Apparat wurde durch Kooaptationsdistanz, Koaptationsfläche, MK-Schlusswinkel, MK-Annulusfläche, interkommissuralen/anteroposterioren MK-Annulusdiameter und Interpapillarmuskelabstand (KD, KF, MKSW, MKAF, IKD, APD, IMA) charakterisiert.

Ergebnisse: In Gruppe 1 und 2 betrugen die 30-Tage-Mortalität 10% und 0%, die 5-Jahre-Überlebensrate - 83.1% und 82.7%. In Gruppe 1 wurde nach chirurgischer LVR eine Reduktion des LVESVI von 110.6 ± 88.8 auf 50.2 ± 22.9 ml/m^2 ($p = 0.001$), in Gruppe 2 - von 118.6 ± 49.2 auf 63.6 ± 32.1 ml/m^2 ($p < 0.001$) erzielt. Die LV-Ejektionsfraktion (LVEF) stieg von 31.5 ± 15.1% auf 43.4 ± 9.9% ($p < 0.001$) in der Gruppe 1 und von 29.6 ± 9.1% auf 40.9 ± 10.3% ($p < 0.001$) in der Gruppe 2 an.

Beide Gruppen demonstrierten signifikant höhere Ausgangswerte der MKAF, KD und KF bei Patienten, die eine zusätzliche MK-Rekonstruktion oder Ersatz brauchten, Gruppe 2 zeigte zusätzlich eine höhere Prävalenz von begleitenden posterioren Myokardnarben. Die postoperative Reduktion der Mitralregurgitation (MR) nach LVR ohne MK-Chirurgie korrespondierte in der Gruppe 1 mit signifikanter Reduktion der IKD, APD, MKAF, KD, KF, MKSW und IMA; in der Gruppe 2 - mit leichter Reduktion der MVAA und signifikanter Reduktion der KD, KF und IMA.

Schlussfolgerung: Eine chirurgische LVR, unterstützt durch MSCT, führt bei LV-Aneurysmen beider Lokalisationen zu exzellenten mittelfristigen Ergebnissen durch

adäquate Volumenreduktion und funktionale Verbesserung. Die Patienten beider Gruppen, die eine zusätzliche MK-Chirurgie benötigten, zeigten fortgeschrittene Veränderungen der MK-Geometrie, assoziiert mit Dysfunktion der posterioren LV-Wand und des posterioren Papillarmuskels. Die postoperative Reduktion der moderaten begleitenden MR bei Patienten beider Gruppen ohne zusätzliche MK-Chirurgie korrespondierte mit einer Verbesserung der MK-Geometrie. Auf Basis der MSCT-Analyse schlagen wir einen Algorithmus für die chirurgische Planung bei posterioren LV-Aneurysmen vor.

Summary

Objectives: Involvement of the mitral valve (MV) apparatus represents a challenge in surgical ventricular repair (SVR), especially of posterior left ventricular (LV) aneurysms. Here we investigate the capability of MSCT for assessment of the MV/LV complex to optimize the surgical procedure for LV aneurysms, the surgically relevant differences between posterior and anterior localization, and the mid-term results of both groups.

Methods: Thirty consecutive patients (m:w = 24:6, median age 66.0 years; mean New York Heart Association (NYHA) class 2.98) with posterior LV aneurysm (group 1) and 41 consecutive patients (m:w = 31:10, median age 57.7 years; mean NYHA class 3.01) with anterior LV aneurysm (group 2) were operated upon. End-diastolic and end-systolic volumes of LV were indexed to body surface area (LVEDVI/LVESVI). The MV apparatus was characterized by coaptation distance, tenting area, MV closure angle, MV annulus area, intercommissural/anteroposterior MV annulus diameter and interpapillary muscle distance (CD/TA/MVCA/MVAA/ICD/APD/IMD).

Results: Groups 1 and 2 showed 30-day mortality of 10% and 0% and 5-year survival of 83.1% and 82.7% respectively. Reduction of LVESVI from 110.6 ± 88.8 to 50.2 ± 22.9 ml/m^2 ($p = 0.001$) in group 1 and from 118.6 ± 49.2 to 63.6 ± 32.1 ml/m^2 ($p < 0.001$) in group 2 was achieved after surgery. LV ejection fraction (LVEF) increased from 29.5 ± 15.1% to 43.4 ± 9.9% ($p < 0.001$) in group 1 and from 29.6 ± 9.1% to 40.9 ± 10.3% ($p < 0.001$) in group 2. Both groups showed significantly higher initial values of MVAA, CD and TA in patients who needed MV repair or replacement, while group 2 showed additionally a higher prevalence of concomitant posterior scars. Postoperative reduction of mitral regurgitation (MR) after SVR without additional MV surgery corresponded in group 1 with significant reduction in ICD, APD, MVAA, TA, CD, MVCA and IMD and in group 2 with a slight reduction of MVAA and significant reduction of TA, CD and IMD.

Conclusions: MSCT-guided SVR of LV aneurysms of both localizations allows excellent mid-term results to be achieved due to adequate volume reduction and functional improvement. The patients of both groups who needed additional mitral surgery demonstrated advanced changes in MV geometry associated with lesions of the posterior LV wall and posterior papillary muscle. Postoperative reduction of moderate MR without additional MV surgery corresponded with significant improvement

of MV geometry in both groups. Based on the MSCT assessment we propose an algorithm for surgical planning in posterior LV aneurysms.

1. INTRODUCTION

1.1. Pathophysiology

Surgical repair of posterior left ventricular (LV) aneurysms poses a technical challenge because of their localization in myocardial segments adjacent to the mitral valve (MV), and especially so in cases of involvement of the MV apparatus. In order to approach the aneurysm safely and to decide on the surgical strategy, the critical issue of reconstruction is resection of the aneurysm with preservation and restoration of the shape and function of the LV/MV complex, and – in cases of mitral apparatus involvement with severe mitral regurgitation (MR) – additional MV repair or replacement.

The underlying pathology in these cases is more frequently LV pseudoaneurysm than true aneurysm. While pseudoaneurysms are the result of acute free wall rupture – which as a complication of acute myocardial infarction (MI) has an incidence of 3.7% and causes 4% mortality (4) – and are contained by overlying adherent pericardium, true ventricular aneurysms present thinned dyskinetic areas of scarred myocardium. Whereas pseudoaneurysms require urgent surgical resection because of the danger of rupture, true aneurysms can be operated upon electively or sometimes treated conservatively.

Posterior and lateral localization is found in approximately 11% of all surgically treated true LV aneurysms, as reported by Jeganathan et al. (5) and Mickleborough et al. (6, 7). Due to the limited surgical experience in single institutions, literature on the planning of operative approaches and outcome of surgical repair of posterior LV aneurysms is sparse (3, 8-11).

Typical LV aneurysms of anterior localization occur as a consequence of acute transmural anterior MI, whereby the successful early reperfusion therapy and consistent application of β-blockers and angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers do not prevent LV remodeling. Savoye et al. reported a significant increase (> 20%) of end-diastolic volume after 1 year in 31% of adequately treated patients with anterior MI (12). The larger amount of infarcted segments requires a greater extent of shortening of remaining non-infarcted segments and means significantly higher mechanical stress for the residual viable LV myocardium. McKay et al. demonstrated that LV dilatation occurs when the asynergic LV circumference exceeds 40% (13). Dor et al. emphasized that the scarred LV wall in this context has to

be seen as the cause of left ventricular remodeling (14). White et al. demonstrated that end-systolic volume index (ESVI) of greater than 60 ml/m^2 marks a poor prognosis after MI (15). Therefore the postulated goal of the surgical treatment of anterior LV aneurysms is to diminish or exclude the scarred areas from the remaining contractile left ventricle with consequent reduction of LV wall stress, restoration of physiological LV volume and shape and improvement of LV function (14, 16). Surgical techniques of LV restoration in anterior aneurysms are aimed at exclusion of the scarred area and to configure the residual ventricle as accurately as possible so that it approaches physiological volume (14, 16-20).

1.2. Concomitant ischemic MR

Concomitant ischemic MR signifies an increased operative risk with higher perioperative morbidity and mortality in patients undergoing surgical ventricular repair (5, 21). Remodeling and distortion of the LV play a key role in the pathogenesis of ischemic MR, whereby posterolateral localization of MI and involvement of the posterior papillary muscle are associated with increased severity of ischemic MR (5, 21-23).

Emergence mechanisms of concomitant ischemic MR after anteroapical MI differ from those in posterolateral localization, because the papillary muscles, the load-bearing lateral and posterolateral wall and the myocardium between them as a rule are not affected, even in extremely large aneurysms (14). In the opinion of some authors ischemic MR in anteroapical aneurysms is more connected with secondary annular dilatation (14, 21). MV annular dilatation combined with loss of normal annular saddle shape was shown to play an important role for severity of MR after anterior MI (24, 25).

Yosefy et al. described inferoapical scar extension with consequent displacement of the papillary muscles as a further specific mechanism of MR after anteroapical MI (26). Deja et al. also demonstrated that those patients with advanced LV remodeling who show inferior extension of anterior MI and subsequent leaning of the posterior papillary muscle develop significant MR (24). Watanabe et al. demonstrated wider tethering of MV leaflets toward the LV in anterior MI compared with inferior MI, which shows localized tenting of the leaflet (27).

A stepwise algorithm was recently proposed by Jeganathan et al. for the planning of combined MV and ventricular reconstructive surgery in anterior LV aneurysms (5).

Also, the mechanisms of recurrent ischemic MR after MV repair are closely linked with different types and factors of LV remodeling (28-30). In the attempt to decipher the characteristics of patients who developed recurrent MR, in the subanalysis of the recently published prospective trial from the Cardiothoracic Surgical Trials Network (31), Kron et al. identified the presence of basal inferior aneurysm/dyskinesis as an independent predictor of recurrent ischemic MR after MV repair (28).

1.3. Imaging techniques

In the course of time, various imaging modalities such as contrast ventriculography, radionuclide ventriculography, cardiac magnetic resonance (CMR) and transthoracic and transesophageal echocardiography have been used as approaches to analyze the characteristics of both true aneurysms and pseudoaneurysms and to plan the operative procedure. The advantages and disadvantages of each modality have been well described (32). Recent technical advances have brought not only the further development of the traditional three-dimensional imaging modalities echocardiography and cardiac magnetic resonance tomography but also impressive progress in cardiac multislice computed tomography.

The most important imaging modality for evaluating cardiac diseases remains transthoracic and transesophageal echocardiography with three-dimensional tools. It allows a comprehensive workup of the patients by assessing morphological abnormalities of the heart chambers, myocardium, valves and great vessels and grading the various diseases by integrating information from 2D or 3D echocardiography, color flow, and pulsed and continuous wave Doppler as well as quantitative Doppler flow measurements with the possibility of semiquantitative assessment of intracardial and transvalvular flows with evaluation of flow volumes and pressures. Echocardiography offers excellent time resolution, enabling very detailed analysis of the movement sequences of cardiac structures. Also, the limitations of echocardiography are commonly known: it requires exact geometric alignment of the structure of interest and depends highly on patient anatomical characteristics and the operator’s skills.

CMR imaging is now established as the gold standard for evaluating cardiac diseases, allowing comprehensive detailed assessment of myocardium including systolic left and right ventricular function, viability and scar imaging using gadolinium contrast agents

and diagnosis of myocardial ischemia with stress perfusion testing (33). Furthermore, CMR is distinctly superior to echocardiography in the detection of ventricular thrombus after MI (34). Valve morphology, movement and severity of valve dysfunction with blood flow quantification can also be exactly assessed with CMR (23, 35, 36). In particular, flow measurements with a phase-contrast pulse sequence show good correlation with Doppler echocardiographic findings. The main limitations of CMR continue to be its restricted availability and the lack of specific reimbursement. Arrhythmias that affect ECG gating, claustrophobia and implanted devices still represent relative contraindications for CMR.

The main established clinical application of cardiac multislice computed tomography (MSCT) is the evaluation of the coronary arteries for the diagnosis or exclusion of coronary artery stenosis (37-39). But each ECG-gated MSCT examination with its possibilities of unrestricted reconstruction of a primary three-dimensionally acquired dataset contains much more relevant imaging information beyond that on the coronary arteries alone. Recent technical advances in MSCT enable the detailed study of cardiac anatomy with free choice of view planes and ventricular functional assessment with very high accuracy (32). High spatial resolution of up to 0.4 mm and time resolution of up to 75 ms allow very detailed analysis of cardiac anatomy, including valves and their movement sequences. Modern MSCT software tools for analysis of volumes and of the cardiac chambers allow left and right ventricular functional assessment, based on exact true volume detection (32, 40).

In addition, myocardial function and segmental wall motion abnormalities can be assessed with MSCT with accuracy comparable to that of cardiac MRI (40). MSCT provides additional valuable tools such as delayed contrast enhancement for visualization of scar areas in patients with ischemic cardiomyopathy similar to late gadolinium enhancement with cardiac MRI (41). The visualization of older post-infarction scars is possible with MSCT by the tissue differentiation between myocardium and fat as a result of the fatty replacement of the fibrous myocardial scar (40, 42). MSCT also provides reliable identification of LV thrombus.

Anatomical valve features, thickening or calcification and structural changes of the subvalvular apparatus and other related structures can be simply recognized.

Several studies have demonstrated that morphology of a normal and diseased aortic and mitral valve as assessed with MSCT correlates at a high rate with morphological information obtained by echocardiography (35, 36, 43).

It also have been demonstrated that MSCT provides functional information regarding valvular disease severity. Planimetric measurements of maximal opening and maximal regurgitant orifice areas of the mitral and aortic valve have been shown to deliver data comparable with those derived from echocardiographic and Doppler measurements (44).

Moreover, MSCT provides a possibility to analyze the anatomical coherences of the heart, great vessels, airways and other structures of the chest.

The aforementioned advantages of MSCT, the possibilities to analyze the coherence of ventricular remodelling and subtle geometrical changes in the mitral valve apparatus, make it a useful tool for the analysis and planning of cardiac surgical and interventional approaches.

1.4. Aim of this study

This study aimed to investigate the potential of ECG-triggered MSCT for assessment of the mitral valve / left ventricle complex to optimize the surgical procedure for LV aneurysms, to find out the surgically relevant differences between posterior and anterior localization and to analyze our single-center early and mid-term results of MSCT-guided surgical reconstruction of LV aneurysms of both localizations.

2. METHODS

2.1. Study design

The diagnosis of LV true or pseudoaneurysm of posterior and anterior localization was made by echocardiography, computed tomography and angiography. The surgeons made the decision regarding the operative technique and approach, preparation of pericardium, aneurysm opening and patch size using mainly the MSCT data. Echocardiography and MSCT were repeated during the first postoperative week. The data evaluated before and after surgery were analyzed and compared retrospectively.

2.2. Study population

Data of all consecutive patients with coronary artery disease who underwent surgical ventricular repair (SVR) for LV aneurysms of posterior medial, posterior or posterior lateral localization in our hospital between February 2006 and May 2014 were retrospectively analyzed.

Additionally, data of 41 consecutive patients with coronary artery disease who underwent SVR for LV aneurysms of anterior localization in our hospital between January 2011 and May 2014 were retrospectively analyzed to compare the clinical outcome and changes in LV and MV function and geometry in both groups.

Written informed consent for surgery was obtained from all patients or their representatives. This study was approved by our Institutional Review Board with waiver of need for patient consent.

2.3. Operative procedures

All LV repairs were performed through a median sternotomy approach under cardiopulmonary bypass (CPB). The definite diagnosis of a true or pseudoaneurysm was made by direct surgical inspection with assessment of aneurysm localization, the presence of pericardial adhesions, myocardial disruption and thrombotic masses. Intraoperative transesophageal echocardiography was routinely performed to assess the adequacy of LV and MV repair. LV repair and MV repair or replacement were performed after coronary artery bypass grafting (CABG).

In posterior aneurysm localization the parameters of aneurysm localization and morphology, size of the aneurysm neck (ADA) and extension of the perfused pseudoaneurysm, obtained with MSCT preoperatively, as well as LV and aneurysm volumes, measured in MSCT, allowed exact estimation of the required volume reduction of the LV, assessment of the possibility of linear repair, and precise sizing of the Dacron patch for patch repair. Mitral surgery in this group was performed in patients with aneurysm defect area (ADA) larger than 20 sqcm, CT morphological signs of MV tethering and echocardiographically proven MR grade > 2.

Antero-apical ventricular repair was routinely performed using a modified Dor or McCarthy procedure with several Fontan sutures along the aneurysm perimeter without patch application, in order to exclude the aneurysm and restore LV geometry (45). This technique enables the effective exclusion of scarred areas, achieving the required target LV volume and reconstruction of the LV apex. Only one patient from this group, in whom the additional closure of a small ventricular septal defect after a huge antero-apical myocardial infarction was performed, needed a patch repair.

All surgeons had access to the complete preoperative MSCT assessment.

2.4. MSCT measurements

The following section is a revised and expanded version of the methods descriptions that were recently published in our original article (1) and case reports (2, 3).

2.5. Data acquisition

Contrast-enhanced ECG-triggered cardiac scanning was performed using a first- or second-generation dual-source (2 x 128-slice) scanner (Somatom Definition, Somatom Definition Flash, Siemens AG, Erlangen, Germany) with the following study protocol: tube voltage 100-120 kV, tube current 320 ref. mAs/rotation, rotation time 280 ms, slice collimation of 128 x 0.6 mm, temporal resolution 75 ms (not depending on heart rate), slice width 0.75 mm, reconstruction increment 0.4 mm, reconstruction kernel B30f. A total of 80 to 100 ml nonionic contrast medium (Imeron 400, Bracco, Altana Pharma, Constance, Germany) was usually administered via the antecubital/jugular vein at 4-5 ml/s. Automated peak enhancement detection in the left atrium was usually used to time the contrast bolus with data acquisition start at threshold level of 160 Hounsfield units. Scans were performed during an inspiratory breath-hold of 8 to 10 s. The electrocardiogram was recorded simultaneously to allow retrospective gating and reconstruction of the data at desired phases of the cardiac cycle. All images were transferred to a post-processing workstation for data analysis (syngo.via, Siemens AG).

2.6. Data analysis

To study the anatomy and geometry of the LV and MV apparatus, the data set was reconstructed with a slice thickness of 0.75 mm and reconstruction increment of 0.4 mm, starting in early systole (0% of cardiac cycle) and lasting to end diastole (90% of cardiac cycle) in steps of 10%.

LV volumes and systolic function were assessed using dedicated CT evaluation software (syngo Circulation, Siemens AG) and applying a 3D threshold segmentation algorithm. End diastole and end systole were estimated automatically and adjusted by the investigator if needed. Endocardial borders are traced semi-automatically and papillary muscles were regarded as being part of the LV cavity.

LV end-diastolic volume (LVEDV) and end-systolic volume (LVESV) were obtained (Fig. 1); left ventricular ejection fraction (LVEF) was calculated by the difference between LVESV and LVEDV divided by LVEDV.

Left atrium (LA) volume was calculated at end systole on the basis of planimetric measured LA area in two-chamber view (A1) and four-chamber view (A2) and LA-length (L) according to the simplified empiric formula (46):

$$(0.85 \times A1 \times A2)/L$$

The systolic and diastolic LV volumetric sphericity index (LVSI) was calculated on the basis of end-diastolic and end-systolic LV volume and LV long axis length in two-chamber view according to the empiric formula (47), (Fig. 2):

$$LVSI = LV\ Volume/LV\ long\ axis^{3} \times \pi/6$$

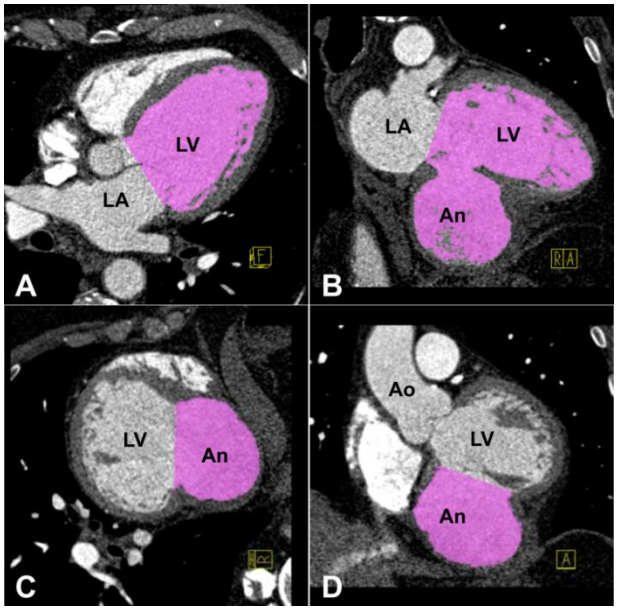


Figure 1. Assessment of left ventricular and aneurysmatic volume.
Panels A+B: MSCT reconstruction of four-chamber view and two-chamber view for semi-automatic measurement of LV volume including aneurysm in diastole (pink area). Panels C+D: short-axis view and two-chamber view for measurement of separate aneurysm volume in diastole (pink area). LA = left atrium, LV = left ventricle, An = aneurysm, Ao = aorta.

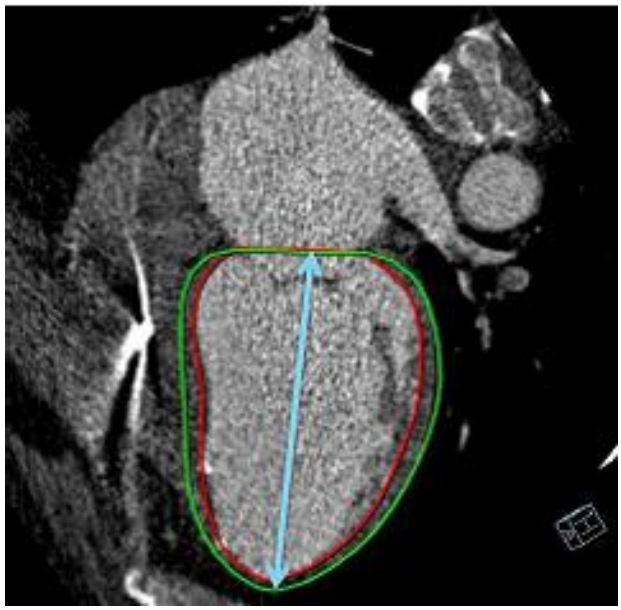


Figure 2. Calculation of the left ventricular volumetric sphericity index (LVSI).

Local wall motion abnormalities were estimated semiquantitatively using a usual 17-segment model with separate assessment of papillary muscles and the corresponding load bearing LV wall. All parameters obtained were indexed to body surface area (BSA).

Morphological characteristics of the aneurysm in terms of myocardial disruption, aneurysm defect area, apposition of thrombotic masses and pericardial effusion were evaluated.

Additionally, the aneurysm's end-systolic and end-diastolic volumes (AEDV and AESV) and end-diastolic aneurysm defect area (ADA) were estimated using the same software tool with manually defined 2D aneurysm borders to the remaining part of the left ventricle (Fig. 1).

MV geometry was studied in the reconstructed mid-systolic phase, when the valve was closed. Using the two- und four-chamber view and the reconstructed LV short-axis view, a plane coaxial to the MV was reconstructed. At the level of the MV annulus, the annulus area was quantified by planimetry; anteroposterior and intercommissural diameters (APD, ICD) were measured on the level of MV segments A2/P2 and in the short axis, respectively (Fig. 3).

The corresponding three-chamber view representing the anteroposterior plane through the MV segments A2/P2 was used for the measurements of coaptation distance (CD) as the distance between the leaflet coaptation point and mitral annulus plane, tenting area (TA) as the area between the two mitral leaflets below the mitral annulus plane, and mitral valve closure angle (MVCA) as the angle between the leaflets at the coaptation point (Fig. 3).

Measurements of the submitral apparatus, including interpapillary muscle distance (IMD) and distances and angles between the MV annulus and anterior and posterior papillary muscle head (AnAPMD, AnPMD, AnAPMA, AnPPMA), were also performed in the reconstructed mid-systolic phase using the same planes. CT morphological characteristics of other elements of the submitral apparatus – chordae, papillary muscle – and load-bearing LV wall dysfunction were analyzed semi-qualitatively.

This minimum of measurements provides detailed analysis of LV and MV anatomy and geometry and the coherence of the mitral valve leaflets, annulus, subvalvular apparatus including papillary muscles and chords, left ventricle and left atrium. The severity of corresponding mitral regurgitation was estimated by echocardiography.

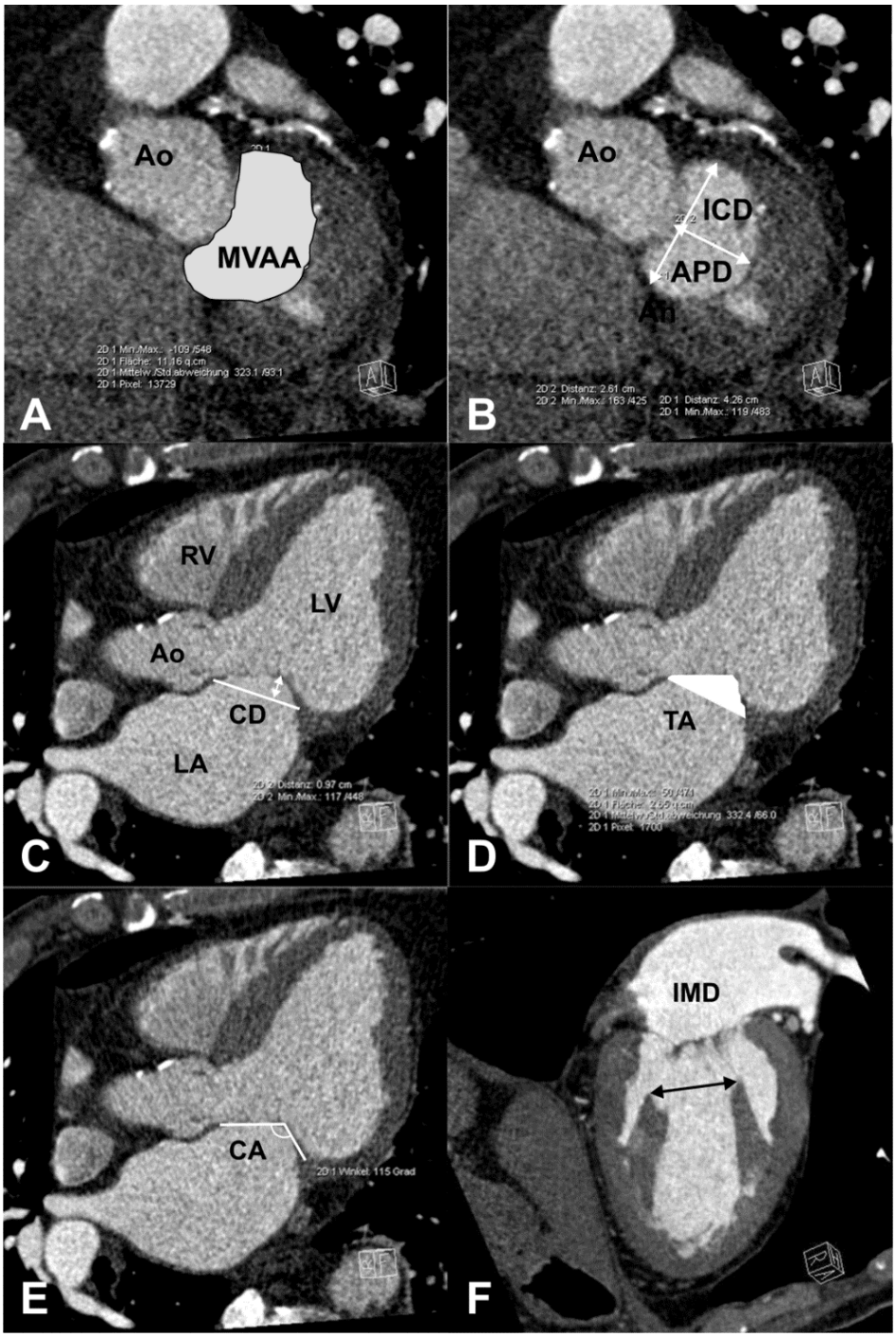


Figure 3. Geometric indices of mitral valve and subvalvular apparatus. Panels A+B: MSCT reconstruction of short-axis view at the level of the mitral annulus – mitral valve annulus area (MVAA), intercommissural (ICD) and anteroposterior diameter (APD). Panels C-E: three-chamber view as anteroposterior plane through the mitral valve segments A2/P2. Panel C: coaptation distance (CD) as the distance between the leaflet coaptation point and mitral annulus plane (A). Panel D: tenting area (TA) as the area between the two mitral leaflets under the mitral annulus plane. Panel E: mitral valve closure angle (MVCA) as the angle between the two leaflets at coaptation point (C). Panel F: two-chamber view – interpapillary muscle distance (IMD) as the distance between heads of the papillary muscles. MVAA = mitral valve annulus area, ICD = intercommissural diameter, APD = anteroposterior diameter, CD = coaptation distance, TA = tenting area, CA = closure angle, IMD = interpapillary muscle distance, LV = left ventricle, RV = right ventricle, LA = left atrium, Ao = aorta.

2.7. Statistical analysis

Continuous variables are presented as mean ± SD. Categorical variables are presented as numbers with percentages. Means were compared with the paired, two-tailed Student t-test. Actuarial survival curves were calculated using the Kaplan-Meier method. $P < 0.05$ was considered statistically significant. The data were analyzed with SPSS 20 (SPSS, Chicago, IL, USA).

3. RESULTS

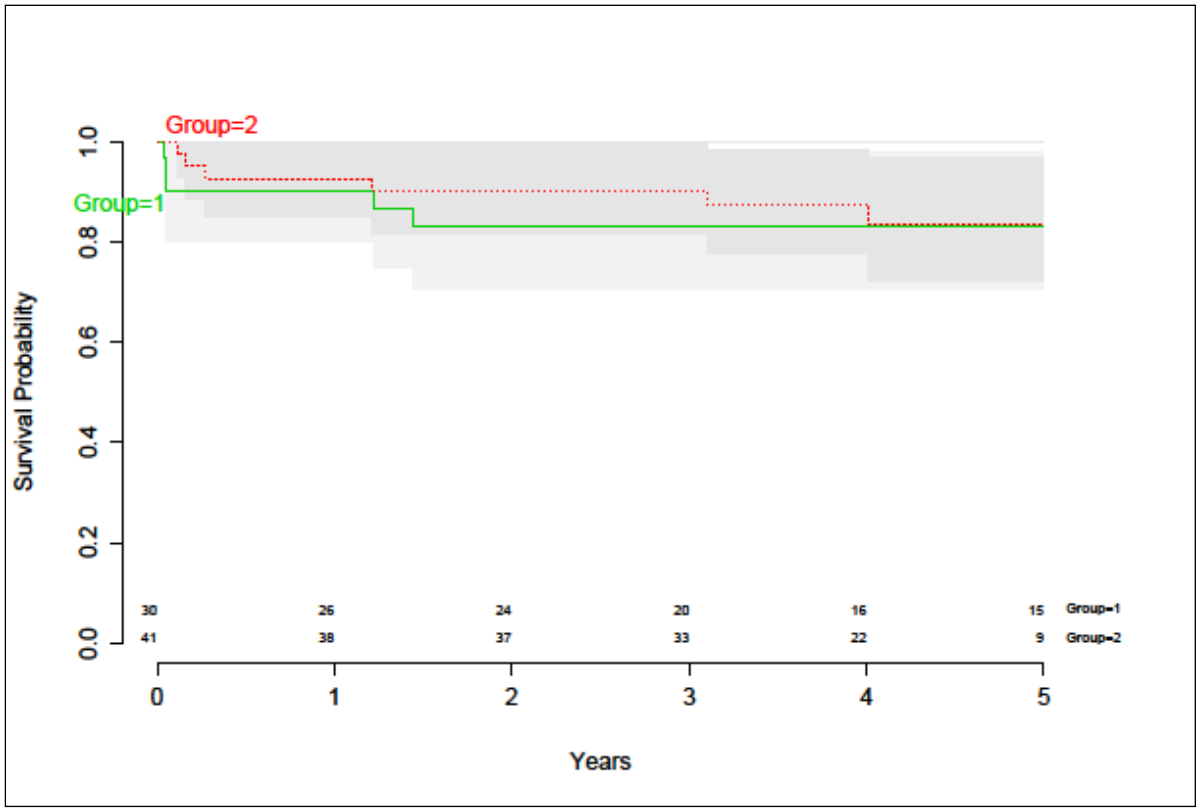
3.1. Clinical characteristics and outcomes

3.1.1. Group 1

Thirty patients with aneurysms of the posterior medial, posterior and posterior lateral LV wall due to myocardial infarction underwent surgical ventricular repair. The median age of the patients was 66 years (range 38-78 years). There were six (20%) female and 24 (80%) male patients. The mean preoperative New York Heart Association class (NYHA) was 2.98 ± 0.16. The mean preoperative LVEF, estimated with echocardiography, was 34.7 ± 10.2%. Seven patients had concomitant MR of grade ≥ 2. There were 17 patients with heart failure of NYHA class III and 8 patients in NYHA class IV. Preoperative patient characteristics are presented in Table 1. Overall 30-day mortality was 10% (3/30). One- and 5-year actuarial survival rates estimated by the Kaplan-Meier method were 86.5% and 83.1%, respectively (Fig. 4).

3.1.2. Group 2

Forty-one patients (group 2) with LV aneurysm of anterior localization due to myocardial infarction also underwent SVR. The median age of the patients was 57.7 years (range 33-75 years). There were ten (24.4%) female and 31 (75.6%) male patients. The mean preoperative NYHA class was 3.01 ± 0.11. The mean preoperative LVEF, estimated with echocardiography, was 29.2 ± 7.6%. Six of the patients of group 2 had concomitant MR of grade ≥ 2. There were 35 patients with heart failure of NYHA class III and 3 patients in NYHA class IV. Preoperative patient characteristics are also presented in Table 1. Overall 30-day mortality was 0% (0/41). One- and 5-year actuarial survival rates estimated by the Kaplan-Meier method were 95.1% and 82.7%, respectively (Fig. 4).



Group 1 vs. Group 2, p = 0.824					
Months	12	24	36	48	60
Group 1	86.5	83.1	83.1	83.1	83.1
Group 2	95.1	90.2	90.2	87	82.7

Figure 4. Group 1 + Group 2: Kaplan-Meier survival curve for the overall population.

Table 1. Baseline patient characteristics

	Group 1 (n = 30)	Group 2 (n = 41)	*P*-value
Median age (range), years	66.0 (38.0-78.0)	57.7 (33.0-75.0)	0.126
Female gender, n (%)	6 (20.0)	10 (24.4)	0.662
Diabetes mellitus, n (%)	11 (36.7)	10 (24.4)	0.376
Hypertension, n (%)	29 (96.7)	24 (58.5)	<0.001
Hypercholesterolemia, n (%)	26 (86.7)	27 (65.8)	0.046
Peripheral vascular disease, n (%)	1 (3.3)	4 (9.7)	0.296
History of renal failure, n (%)	4(13.3)	6 (14.6)	0.876
History of atrial fibrillation, n (%)	7 (23.3)	4 (9.7)	0.118
Previous cardiac surgery, n (%)	2 (6.0)	2 (4.9)	0.747
NYHA I–II, n (%)	5 (16.7)	3 (7.3)	0.220
NYHA III, n (%)	17 (56.7)	35 (85.4)	0.032
NYHA IV, n (%)	8 (26.7)	3 (7.3)	0.100
LVEF > 40%, n (%)	7 (23.3)	5 (12.2)	0.021
LVEF 20-40%, n (%)	20 (66.7)	30 (73.2)	0.063
LVEF < 20%, n (%)	3 (10.0)	6 (14.6)	0.916
Coronary lesions: Single vessel, n (%) Double vessel, n (%) Triple vessel, n (%)	 4 (13.3) 11 (36.7) 15 (50.0)	 10 (24.4) 8 (19.5) 23 (56.1)	 0.667 0.107 0.611
Mitral regurgitation ≥ 2+, n (%)	7 (23.3)	6 (14.6)	0.349

3.2. Procedural outcome

3.2.1. Group 1

For posterior ventricular reconstruction in the population of 30 patients patch ventriculoplasty was performed in 15 patients (50%) and linear repair in 15 (50%). Sixteen patients (53%) had associated myocardial revascularization. Six patients (20%) underwent concomitant MV surgery. MV repair was performed in three (10%) and MV replacement also in three (10%) patients. One patient needed reoperation due to secondary covered left ventricular perforation after 9 months. Three patients (10%) died in hospital due to septic shock (n = 2) or multiorgan failure (n = 1). All of them had combined surgery (1 = SVR + CABG, 2 = SVR, CABG + MV replacement). Operative and postoperative data are summarized in Table 2**.**

3.2.2. Group 2

For anterior ventricular reconstruction in the population of 41 patients patch ventriculoplasty was performed in 1 patient (2.4%) and linear repair (modified Dor procedure) in 40 (97.6%). Thirty-two patients (78%) had associated myocardial revascularization. Six patients of group 2 (14.6%) underwent concomitant MV surgery. Also in group 2 one patient needed reoperation due to secondary covered left ventricular perforation after 9 months. Hospital mortality in group 2 was 0%. Operative and postoperative data are also summarized in Table 2**.**

Table 2. Operative and postoperative data.

	Group 1 (n = 30)	Group 2 (n = 41)	*P*-value
Concomitant CABG, n (%)	16 (53.3)	32 (78.0)	0.028
No. of grafts performed, median (range)	1.0 (0-4)	3.0 (0-4)	0.003
Concomitant MV surgery, n (%)	6 (20.0)	6 (14.6)	0.551
Patch repair, n (%)	15 (50.0)	1 (2.4)	<0.001
Non-patch repair, n (%)	15 (50.0)	40 (97.6)	<0.001
CBP time, median (range), minutes	165.5 (57-505)	138.0 (46-266)	0.180
Cross-clamp time, median (range), minutes	84.8 (30-255)	144.0 (18-162)	0.937
Elective surgery, n (%)	13 (43.3)	41 (100.0)	<0.001
Myocardial infarction, n (%)	0 (0.0)	0 (0.0)	------
Stroke, n (%)	0 (0.0)	1 (2.4)	0.389
Sepsis, n (%)	7 (23.3)	0 (0.0)	0.001
Renal failure, n (%)	6 (20.0)	2 (4.9)	0.047
Hospital mortality, n (%)	3 (10.0)	0 (0.0)	0.039

3.3. Changes in LV geometry and function

3.3.1. Group 1

Postoperative echocardiographically measured LVEF had increased from 34.7 ± 10.2% to 40.2 ± 9.2% (p = 0.091) and mean LVEDD had decreased from 57.5 ± 6.9 mm to 52.7 ± 6.7 mm (p = 0.028).

LV volumetric parameters measured in MSCT in 24 patients were also significantly decreased. LVEDVI decreased from 151.2 ± 84.1 ml/m^2 to 85.7 ± 28.3 ml/m^2 (p = 0.001) and LVESVI from 110.6 ± 88.8 ml/m^2 to 50.2 ± 22.9 ml/m^2 (p < 0.001). There was a statistically significant increase in LVEF from 31.5 ± 15.1% to 43.4 ± 9.9%

(p = 0.001) after posterior SVR. The diastolic LVSI was significantly decreased from 0.60 ± 0.13 to 0.53 ± 0.07 (p = 0.007), showing reverse remodeling of the LV toward a more normal shape (for full comparison of LV parameters see Table 3a and Fig. 5,6,7).

3.3.2. Group 2

Postoperative echocardiographically measured LVEF had increased from 29.2 ± 7.6 to 35.2 ± 7.7% (p < 0.001) and mean LVEDD had decreased from 61.1 ± 8.6 to 58.6 ± 8.4 mm (p < 0.001).

LV volumetric parameters measured in MSCT in all patients were also significantly decreased. LVEDVI decreased from 164.4 ± 50.8 to 103.4 ± 34.5 ml/m^2 (p < 0.001) and LVESVI from 118.6 ± 49.2 to 63.6 ± 32.1 ml/m^2 (p < 0.001). There was a statistically significant increase in LVEF from 29.6 ± 9.1% to 40.9 ± 10.3% (p < 0.001) after anterior SVR. The diastolic LVSI was increased from 0.41 ± 0.08 to 0.57 ± 0.16 (p < 0.001), due to substantial shortening of the LV long axis in relation to LV volume reduction (for full comparison of LV parameters see Table 3b and Fig. 5,6,7).

Table 3a. Group 1: LV Dimensions measured in CT scans before and after ventricle restoration in 24 cases.

	Preoperative	Postoperative	*P*-value
LVEDD (echo), % (mean ± SD)	57.5 ± 6.9	52.7 ± 6.7	0.028
LVEF (echo), % (mean ± SD)	34.7 ± 10.2	40.4 ± 9.2	0.091
LVEDVI, ml/sqm (mean ± SD)	151.2 ± 84.1	85.7 ± 28.3	0.001
LVESVI, ml/sqm (mean ± SD)	110.6 ± 88.8	50.2 ± 22.9	0.001
SVI, ml/sqm (mean ± SD)	39.3 ± 12.2	35.2 ± 8.5	0.167
LVEF, % (mean ± SD)	31.5 ± 15.1	43.4 ± 9.9	0.001
LVSI diast. (mean ± SD)	0.60 ± 0.13	0.53 ± 0.07	0.007
LVSI syst. (mean ± SD)	0.56 ± 0.16	0.47 ± 0.09	0.011
LVEDVI, LVESVI = LV end-diastolic and end-systolic volume index; SVI = stroke volume index; LVEF = LV ejection fraction; LVSI = LV sphericity index, diastolic and systolic.			

Table 3b. Group 2: LV Dimensions measured in CT scans before and after ventricle restoration in 41 cases.

	Preoperative	Postoperative	*P*-value
LVEDD (echo), % (mean ± SD)	61.1 ± 8.6	58.6 ± 8.4	<0.001
LVEF (echo), % (mean ± SD)	29.2 ± 7.6	35.2 ± 7.7	<0.001
LVEDVI, ml/sqm (mean ± SD)	164.4 ± 50.8	103.4 ± 34.5	<0.001
LVESVI, ml/sqm (mean ± SD)	118.6 ± 49.2	63.6 ± 32.1	<0.001
SVI, ml/sqm (mean ± SD)	45.8 ± 11.4	39.8 ± 7.9	0.075
LVEF, % (mean ± SD)	29.6 ± 9.1	40.9 ± 10.3	<0.001
LVSI diast. (mean ± SD)	0.41 ± 0.08	0.57 ± 0.16	<0.001
LVSI syst. (mean ± SD)	0.33 ± 0.08	0.40 ± 0.16	<0.001
LAVI syst., ml/sqm (mean ± SD)	60.4 ± 19.6	52.2 ± 21.7	<0.001
LVEDVI, LVESVI = LV end-diastolic and end-systolic volume index; SVI = stroke volume index; LVEF = LV ejection fraction; LVSI = LV sphericity index, diastolic and systolic, LAVI = left atrial volume index, end-systolic.			

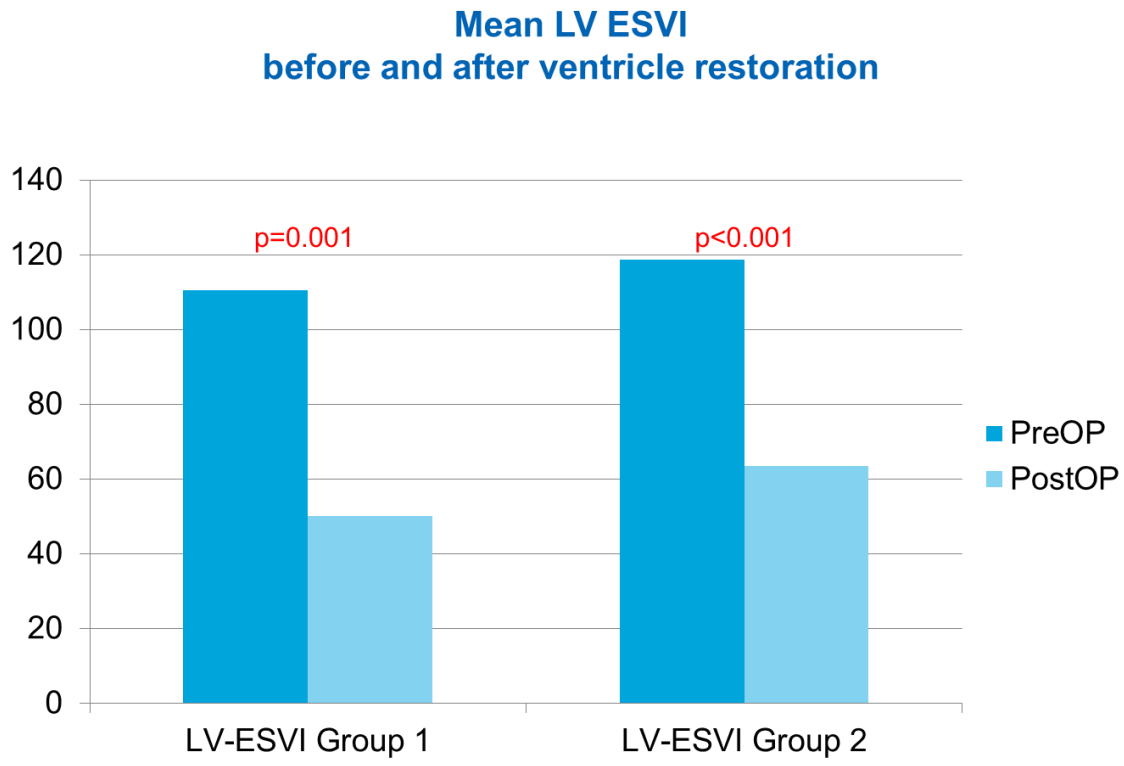


Figure 5. Mean LVESVI before and after ventricle restoration.

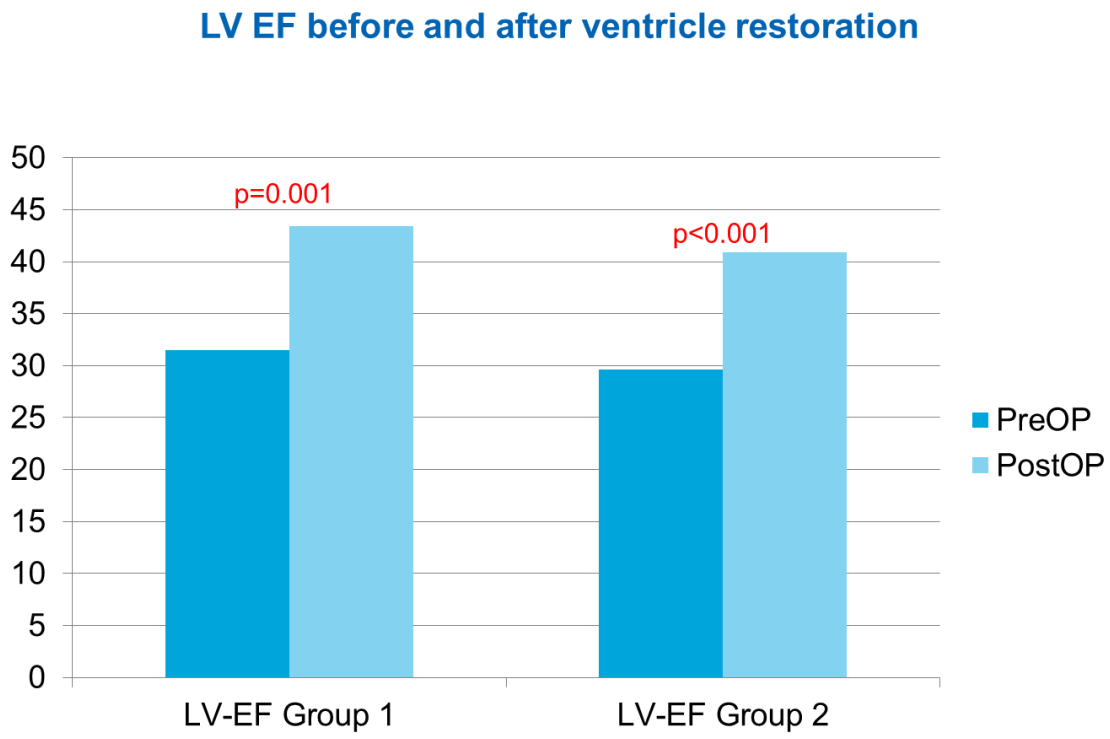


Figure 6. LVEF before and after ventricle restoration.

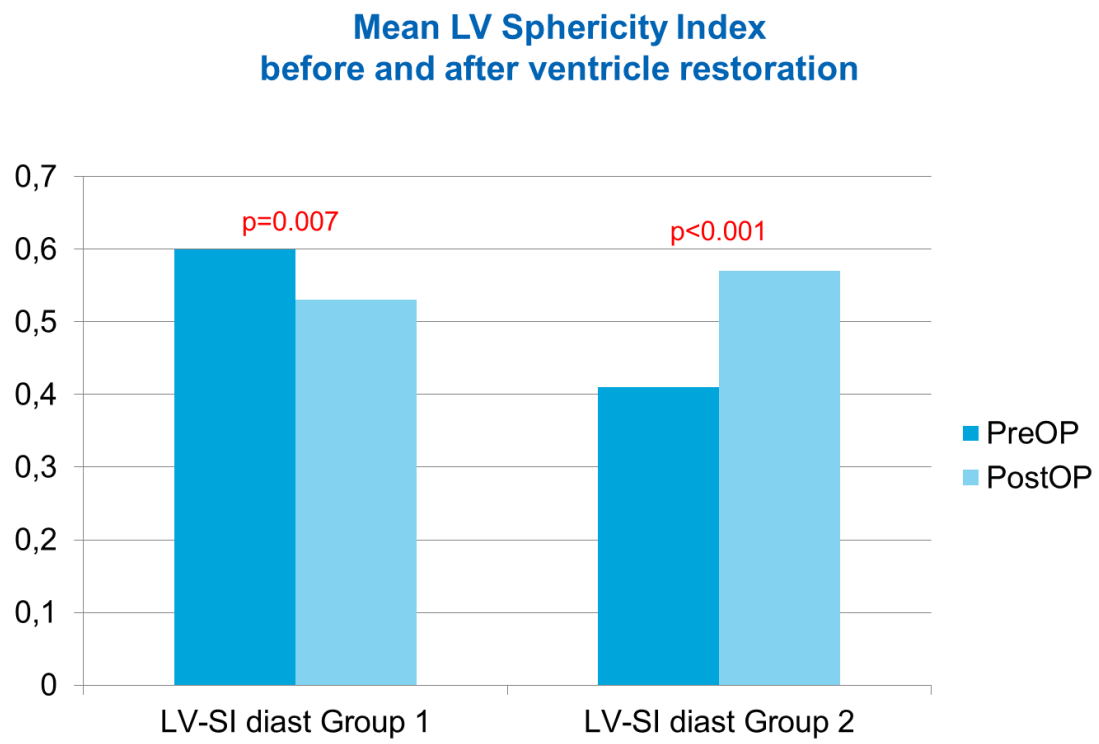


Figure 7. Mean LV Sphericity Index before and after ventricle restoration.

3.4. LV aneurysm characteristics before ventricular restoration

3.4.1. Group 1

Aneurysms were localized in posterior myocardial segments in 9 patients, in posterior medial segments in 7 patients and in posterior lateral segments in 8. Aneurysm morphology was analyzed using such criteria as myocardial disruption, thrombotic apposition, pericardial adhesion and pericardial effusion, based on 4D reconstruction of MSCT data. LV pseudoaneurysm according to these criteria was suspected in 19 patients (79%) and verified intraoperatively in 17 patients (71%). ADA was smaller than 10 sqcm in 9 patients, 10 to 20 sqcm in 9 patients and larger than 20 sqcm in 6 patients (5 of these 6 patients needed MV surgery). The ADA was used to estimate the required patch size and as one of the criteria to select patients needing MV surgery.

Preoperative aneurysm end-diastolic volume index (AEDVI) showed a systolic increase of more than 10 ml/m^2 only in five patients with AEDVI higher than 42.9 ml/m^2, demonstrating volume shift during systole of potential adverse hemodynamic significance. LV aneurysm characteristics are summarized in Tables 4a and 4b.

Table 4a. Group 1: LV Aneurysm characteristics evaluated in CT scans before ventricle restoration in 24 cases.

	n	%
Localization: - Posterior - Posterior medial - Lateral	 9 7 8	 38 29 33
Suspected myocardial disruption	19	79
Verified myocardial disruption	17	71
Suspected true aneurysm	5	21
Verified true aneurysm	7	29
Aneurysm defect area diast., sqcm: < 10 10–20 > 20	 9 9 6	 38 38 25

Table 4b. Group 1: LV Aneurysm dimensions evaluated in CT scans before ventricle restoration in 24 cases (*one patient who had only wall necrosis with small perforation without aneurysm formation needed a patch repair).

	Median	(Range)
ADA diast., sqcm	17.3	(0*–57)
ADA syst., sqcm	16.0	(0*–75)
AEDV, ml	55.0	(0*–507)
AESV, ml	59.0	(0*–505)
AEDVI, ml/sqm	32.4	(0*–260)
AESVI, ml/sqm	30.1	(0*–259)
ADA diast., ADA syst. = aneurysm defect area end-diastolic and end-systolic; AEDV, AESV = aneurysm end-diastolic and end-systolic volume; AEDVI, AESVI = aneurysm end-diastolic and end-systolic volume index.		

3.4.2. Group 2

Aneurysms were localized in antero-apical myocardial segments in 16 patients (39%), in antero-apical segments with involvement of the basal septum in 8 patients (19.5%), in antero-apical segments combined with posterior scar in 11 patients (26.8%) and in antero-apical segments with involvement of both basal septum and posterior scar in 6 patients (14.6%). Aneurysm morphology was analyzed using such criteria as myocardial thinning, myocardial disruption, thrombotic apposition and wall calcification, based on 4D reconstruction of MSCT data. Thrombotic apposition was seen in 9 patients (22%), myocardial disruption in 3 patients (7.3%) and aneurysm calcification in 3 patients (7.3%). LV aneurysm characteristics are summarized in Table 4c.

Table 4c. Group 2: LV Aneurysm characteristics evaluated in CT scans before ventricle restoration in 41 cases.

	n	%
Scar localization: - Antero-apical - Antero-apical + septal - Antero-apical + posterior - Antero-apical + septal + posterior	 16 8 11 6	 39 19.5 26.8 14.6
Thrombotic apposition	9	22
Myocardial disruption	3	7.3
Calcification	3	7.3

3.5. Mitral geometry and function

3.5.1. Group 1

In our series only six patients (20% of the whole population) needed concomitant MV surgery; all of them received a preoperative MSCT assessment.

Postoperative reduction of MR (from grade 0.84 ± 0.15 to 0.25 ± 0.09, p = 0.003) in 18 patients from the MSCT analysis group (n = 24) without concomitant MV surgery corresponded with a significant improvement of MV geometry: a significant reduction in ICD, APD, MVAA and TA, shortening of leaflet CD and flattening of MVCA. Measurements of the submitral apparatus that characterize MV tethering showed significant changes in such parameters as IMD from 33.4 ± 6.3 to 28.9 ± 6.0 mm (p < 0.001) and in AnAPMD and AnPPMD (anterior and posterior papillary muscle tethering length) from 23.6 ± 4.1 to 21.8 ± 3.7 mm, p = 0.027) and from 27.1 ± 3.9 to 23.9 ± 6.8 mm, p = 0.033). The complete data are reported in Table 5a.

Table 5a. Group 1: CT measurements of MV and submitral apparatus dimensions before and after LV restoration in 18 patients without mitral surgery.

	Preoperative	Postoperative	*P*-value
MR, degree (mean ± SD)	0.84 ± 0.15	0.25 ± 0.09	0.003
ICD, mm (mean ± SD)	39.3 ± 6.0	35.3 ± 4.6	0.034
APD, mm (mean ± SD)	25.9 ± 4.8	23.3 ± 3.6	0.010
MVAA, sqcm (mean ± SD)	8.7 ± 1.5	8.1 ± 1.4	0.026
CD, mm (mean ± SD)	10.1 ± 2.0	8.6 ± 2.0	0.011
TA, sqcm (mean ± SD)	1.9 ± 0.5	1.6 ± 0.4	0.003
MVCA, angular degree (mean ± SD)	100.3 ± 14.4°	111.5 ± 10.2°	0.059
CSD, mm (mean ± SD)	32.6 ± 4.2	29.8 ± 5.2	<0.001
IMD, mm (mean ± SD)	33.4 ± 6.3	28.9 ± 6.0	< 0.001
AnAPMD, mm (mean ± SD)	23.6 ± 4.1	21.8 ± 3.7	0.027
AnPPMD, mm (mean ± SD)	27.1 ± 3.9	23.9 ± 6.8	0.033
AnAPMA, angular degree (mean ± SD)	83.4 ± 9.6°	84.2 ± 12.4°	0.765
AnPPMA, angular degree (mean ± SD)	84.7 ± 7.2°	80.7 ± 7.8°	0.086
MR = mitral regurgitation; ICD and APD = intercommissural and anteroposterior MV annulus diameter; MVAA = MV annulus area; CD = coaptation distance; TA = tenting area, MVCA = MV closure angle; CSD = coaptation-to-septum distance; IMD = interpapillary muscle distance, AnAPMD, AnPPMD = distance between MV annulus and anterior and posterior papillary muscle head (papillary muscle tethering length); AnAPMA, AnPPMA = angle between MV annulus and anterior and posterior papillary muscle head (papillary muscle anterior and posterior angle).			

The preoperatively measured MVAA, TA and CD were significantly higher in six patients who needed MV repair/replacement (MVAA 10.7 ± 1.7 vs. 8.7 ± 1.5 scsm, p = 0.013; TA 3.1 ± 1.4 vs. 1.9 ± 0.5 mm, p = 0.009; CD 12.7 ± 2.6 vs. 10.1 ± 2.0 mm, p = 0.020), representing parameters potentially predictive for the necessity of concomitant MV surgery. Volumetric parameters of the LV and aneurysm as well as aneurysm localization showed no significant differences between the SVR and SVR + MV repair/replacement groups, whereas ADA was markedly higher in the SVR + MV repair/replacement group. Patients who needed MV surgery showed also substantially higher prevalence of involvement of the posterior papillary muscle (83% vs.44%) and posterior load bearing wall (100% vs.72%) (Fig. 8). The comparison of clinical variables between these two groups shows higher prevalence of patients with severe MR, LVEF < 20%, NYHA IV symptoms and high hospital mortality in the SVR + MV repair/replacement group, however without statistical significance (Tables 6a-1, 6b-1, 6c-1).

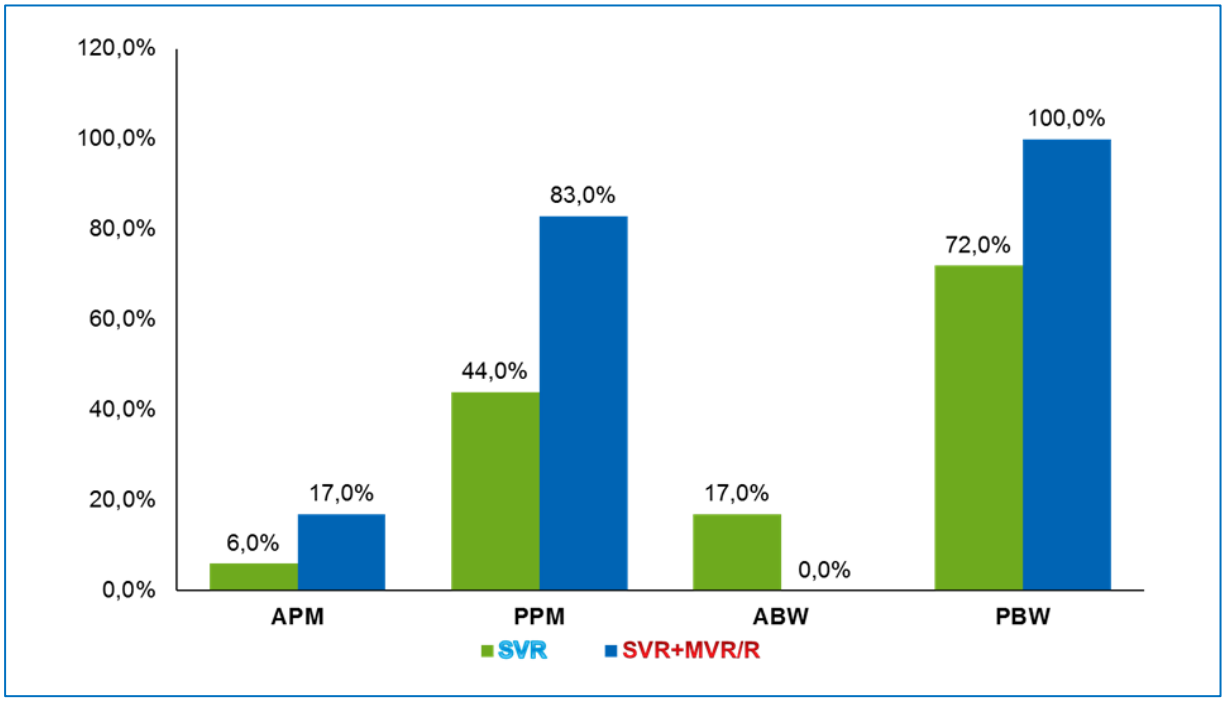


Figure 8. Group 1: Motion abnormalities of papillary muscles and load bearing LV wall – SVR vs. SVR + MV repair/replacement.

Table 6a-1. Group 1: Clinical variables among patients with SVR and SVR+MV repair/replacement.

Variable	SVR (n = 18)	SVR + MV repair/replacement (n = 6)	*P*-value
Median age, years	64	61	0.586
Elective admission, n (%)	7 (36)	4 (67)	0.155
NYHA IV symptoms, n (%)	2 (12)	2 (33)	0.482
EF < 20%, n (%)	1 (6)	1 (17)	0.462
Mitral regurgitation > 2+, n (%)	1 (6)	6 (100)	<0.05
Previous cardiac surgery, n (%)	2 (12)	0	1.0
Hospital mortality, n (%)	0 (0)	2 (33)	0.059

Table 6a-2. Group 2: Clinical variables among patients with SVR and SVR+MV repair/replacement.

Variable	SVR (n = 35)	SVR + MV repair/replacement (n = 6)	*P*-value
Median age, years	58.3	62.1	0.385
Elective admission, n (%)	35 (100)	6 (100)	n.s.
NYHA IV symptoms, n (%)	2 (5.7)	0 (0)	<0.05
EF < 20%, n (%)	4 (11.4)	2 (33.3)	<0.05
Mitral regurgitation ≥ 2+, n (%)	0 (0)	6 (100)	<0.05
Previous cardiac surgery, n (%)	2 (5.7)	0 (0)	0.548
Hospital mortality, n (%)	0 (0)	0 (0)	n.s.

Table 6b-1. Group 1: CT-morphological variables among patients with SVR and SVR + MV repair/replacement, (measurements given as mean ± SD).

	SVR (n = 18)	SVR + MV repair/replacement (n = 6)	*P*-value
LVEDVI, ml/sqm	149.3 ± 89.5	156.3 ± 75.0	0.856
LVESVI, ml/sqm	110.8 ± 95.7	110.2 ± 75.4	0.988
AEDVI, ml/sqm	44.6 ± 66.8	67.0 ± 38.3	0.452
AESVI, ml/sqm	47.8 ± 68.9	71.5 ± 46.1	0.449
ADA D, sqsm	17.5 ± 14.8	25.4 ± 18.9	0.307
ADA S, sqcm	18.5 ± 18.4	23.6 ± 17.6	0.560
MVAA, sqcm	8.7 ± 1.5	10.7 ± 1.7	0.013
CD, mm	10.1 ± 2.0	12.7 ± 2.6	0.020
TA, sqcm	1.9 ± 0.5	3.1 ± 1.4	0.009
MVCA, angular degree	100.3 ± 14.4	104.0 ± 12.0	0.586
IMD, mm	33.4 ± 6.3	37.1 ± 9.6	0.287
LVEDVI, LVESVI = LV end-diastolic and end-systolic volume index; LVEF = LV ejection fraction; AEDVI, AESVI = aneurysm end-diastolic and end-systolic volume index; ADA D, ADA S = aneurysm defect area, end-diastolic and end-systolic; MVAA = MV annulus area; CD = coaptation distance; TA = tenting area, MVCA = MV closure angle; IMD = interpapillary muscle distance.			

Table 6b-2. Group 2: CT-morphological variables among patients with SVR and SVR + MV repair/replacement, (measurements given as mean ± SD).

	SVR (n = 35)	SVR + MV repair/replacement (n = 6)	*P*-value
LVEDVI, ml/sqm	163.5 ± 51.2	169.6 ± 52.4	0.787
LVESVI, ml/sqm	116.4 ± 49.3	131.1 ± 51.7	0.508
SI diast. (mean ± SD)	0.41 ± 0.08	0.44 ± 0.05	0.512
SI syst. (mean ± SD)	0.33 ± 0.08	0.37 ± 0.07	0.212
LAVI syst., ml/sqm (mean ± SD)	57.7 ± 15.8	76.1 ± 32.2	0.032
MVAA, sqcm	9.4 ± 1.9	12.9 ± 2.9	<0.001
CD, mm	8.5 ± 2.0	11.1 ± 3.3	0.013
TA, sqcm	1.6 ± 0.6	2.7 ± 1.2	0.001
MVCA, angular degree	111.4 ± 15.5	106.8 ± 9.7	0.488
IMD, mm	36.0 ± 5.3	39.5 ± 6.2	0.157
LVEDVI, LVESVI = LV end-diastolic and end-systolic volume index; LVEF = LV ejection fraction; SI = sphericity index, diastolic and systolic; LAVI = left atrial volume index end-systolic; MVAA = MV annulus area; CD = coaptation distance; TA = tenting area, MVCA = MV closure angle; IMD = interpapillary muscle distance.			

Table 6c-1. Group 1: Preoperative qualitative CT-morphological characteristics of aneurysm localization and submitral apparatus among patients with SVR and SVR + MV repair/replacement.

	SVR (n = 18)	SVR + MV repair/replacement (n = 6)
Chordae rupture, n (%)	0 (0)	0 (0)
APM dysfunction, n (%)	1 (6)	1 (17)
PPM dysfunction, n (%)	8 (44)	5 (83)
LVAW dysfunction, n (%)	3 (17)	0 (0)
LVPW dysfunction, n (%)	13 (72)	6 (100)
Aneurysm localization: posterior, n (%) posterior medial, n (%) lateral, n (%)	 9 (50) 4 (22) 5 (28)	 1 (17) 2 (33) 3 (50)
APM, PPM = anterior and posterior papillary muscle; LVAW, LVPW = LV anterior and posterior load bearing wall.		

Table 6c-2. Group 2: Preoperative qualitative CT-morphological characteristics of aneurysm localization and submitral apparatus among patients with SVR and SVR + MV repair/replacement.

	SVR (n = 35)	SVR + MV repair/replacement (n = 6)
Chordae rupture, n (%)	0 (0)	0 (0)
APM dysfunction, n (%)	4 (11.4)	2 (33.3)
PPM dysfunction, n (%)	15 (42.9)	4 (66.6)
LVAW dysfunction, n (%)	9 (25.7)	2 (33.3)
LVPW dysfunction, n (%)	15 (42.9)	5 (83.3)
Aneurysm (scar) localization, n (%): Antero-apical Antero-apical + septal Antero-apical + posterior Antero-apical + septal + posterior	 15 (42.9) 7 (20.0) 7 (20.0) 6 (17.1)	 1 (16.7) 1 (16.7) 4 (66.6) 0 (0.0)
APM, PPM = anterior and posterior papillary muscle; LVAW, LVPW = LV anterior and posterior load bearing wall.		

3.5.2. Group 2

Also in this series six of the patients (14.6%) needed concomitant MV surgery.

Postoperative reduction of MR (from grade 0.79 ± 0.39 to 0.37 ± 0.40, $p < 0.001$) in 35 without concomitant MV surgery corresponded with improvement of MV geometry, whereby the changes in mitral annulus diameter (ICD, APD) were not significant and reduction of MV annulus area (MVAA) was only 6.7%, however with statistical significance (from 9.4 ± 1.9 to 8.8 ± 1.5 mm, $p = 0.007$). Significant improvement of MV geometry concerned much more the coaptation parameters of the mitral leaflets with significant reduction in TA (from 1.6 ± 0.6 to 1.2 ± 0.3 sqcm, $p < 0.001$), shortening of leaflet CD (from 8.5 ± 2.0 to 6.6 ± 1.4 mm, $p < 0.001$) and flattening of MVCA (from 111.4 ± 15.5° to 119.5 ± 13.0°, $p < 0.001$). Measurements of the submitral apparatus that characterize MV tethering showed significant changes in such parameters as IMD from 36.0 ± 5.3 to 32.5 ± 4.5 mm, $p < 0.001$). There were no significant changes in anterior and posterior papillary muscle tethering length (AnAPMD from 24.3 ± 4.8 to 23.4 ± 3.8 mm, $p = 0.21$ and AnPPMD from 26.1 ± 3.9 to 25.7 ± 2.7mm, $p = 0.33$), but in anterior and posterior papillary muscle tethering angles (AnAPMA from 97.1 ± 10.3° to 90.8 ± 10.0°, $p = 0.001$ and AnPPMA from 81.7 ± 10.5° to 79.5 ± 8.5°, $p = 0.040$). The complete data are reported in Table 5b.

Table 5b. Group 2: CT measurements of MV and submitral apparatus dimensions before and after LV restoration in 35 patients without mitral surgery.

	Preoperative	Postoperative	*P*-value
MR, degree (mean ± SD)	0.79 ± 0.39	0.37 ± 0.4	< 0.001
ICD, mm (mean ± SD)	40.1 ± 3.5	39.0 ± 3.1	0.067
APD, mm (mean ± SD)	25.1 ± 4.0	24.0 ± 3.5	0.055
MVAA, sqcm (mean ± SD)	9.4 ± 1.9	8.8 ± 1.5	0.007
CD, mm (mean ± SD)	8.5 ± 2.0	6.6 ± 1.4	<0.001
TA, sqcm (mean ± SD)	1.6 ± 0.6	1.2 ± 0.3	<0.001
MVCA, angular degree (mean ± SD)	111.4 ± 15.5°	119.5 ± 13.0°	<0.001
CSD, mm (mean ± SD)	34.7 ± 5.7	33.4 ± 5.5	0.065
IMD, mm (mean ± SD)	36.0 ± 5.3	32.5 ± 4.5	<0.001
AnAPMD, mm (mean ± SD)	24.3 ± 4.8	23.4 ± 3.8	0.210
AnPPMD, mm (mean ± SD)	26.1 ± 3.9	25.7 ± 2.7	0.334
AnAPMA, angular degree (mean ± SD)	97.1 ± 10.3°	90.8 ± 10.0°	0.001
AnPPMA, angular degree (mean ± SD)	81.7 ± 10.5°	79.4 ± 8.5°	0.040

MR = mitral regurgitation; ICD and APD = intercommissural and anteroposterior MV annulus diameter; MVAA = MV annulus area; CD = coaptation distance; TA = tenting area, MVCA = MV closure angle; CSD = coaptation-to-septum distance; IMD = interpapillary muscle distance, AnAPMD, AnPPMD = distance between MV annulus and anterior and posterior papillary muscle head (papillary muscle tethering length); AnAPMA, AnPPMA = angle between MV annulus and anterior and posterior papillary muscle head (papillary muscle anterior and posterior angle).

The preoperatively measured MVAA, CD and TA were significantly higher in six patients who needed MV repair/replacement (MVAA 12.9 ± 2.9 vs. 9.4 ± 1.9, $p < 0.001$; TA 2.7 ± 1.2 vs. 1.6 ± 0.6, $p = 0.001$; CD 11.1 ± 3.3 vs. 8.5 ± 2.0, $p = 0.013$), representing parameters potentially predictive for the necessity of concomitant MV surgery. Volumetric parameters of the LV showed no significant differences between the SVR and SVR+MV repair/replacement groups, whereas LA-VI (left atrial volume index) was markedly higher in the SVR + MV repair/replacement group (LAVI 76.1 ± 32.2 vs. 57.7 ± 15.8, $p = 0.032$). Patients of group 2 who needed MV surgery showed substantially higher prevalence of additional scars of posterior localization (66.6% vs.20%) and involvement of the posterior papillary muscle (66.6% vs.42.9%) (Fig. 9).

The comparison of clinical variables between these two groups shows higher prevalence of patients with severe MR and LVEF < 20% in the SVR + MV repair/replacement group, however without statistical significance (Tables 6a-2, 6b-2, 6c-2).

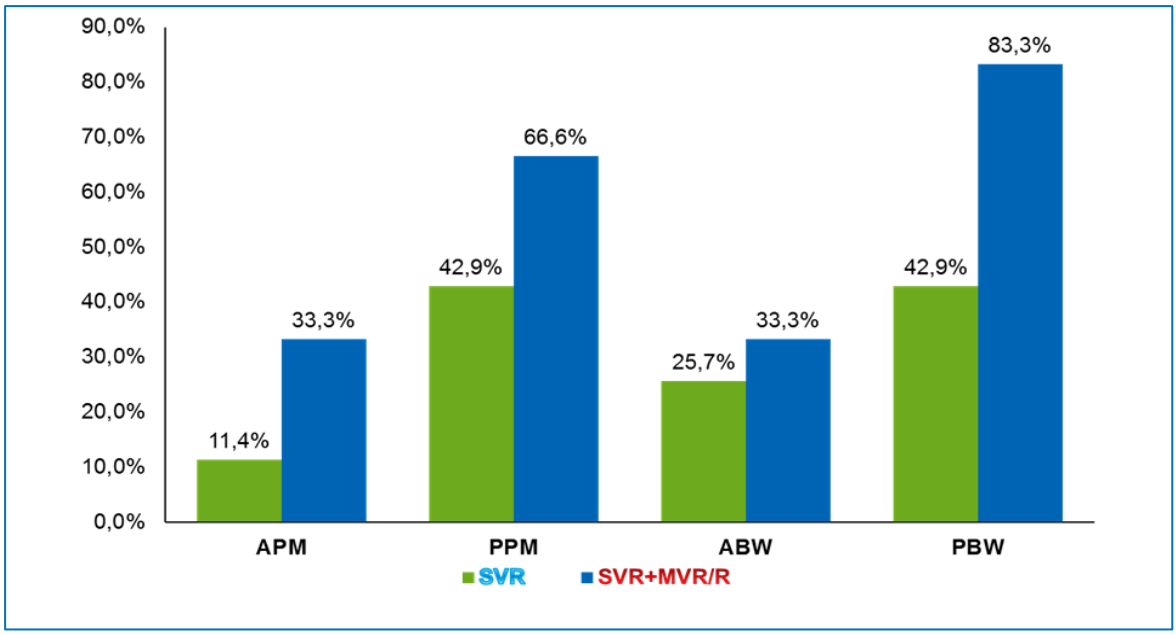


Figure 9. Group 2: Motion abnormalities of papillary muscles and load bearing LV wall - SVR vs. SVR + MV repair/replacement.

3.6. Linear vs. patch repair

3.6.1. Group 1

Patch ventriculoplasty was performed in 11 (46%) and linear repair in 13 (54%) of 24 analyzed cases. The differences between these two groups in patient age (61 vs. 65 years, p = 0.22), presence of NYHA IV symptoms (27% vs. 8%, p = 0.51) or severe impairment of LV function (18% vs. 8%, p = 1.0) and hospital mortality (9% vs.8%, p = 1.0) were not statistically significant. The differences in CT morphological variables – LV and aneurysm volumes, aneurysm defect area and localization and geometrical parameters of the MV and submitral apparatus – did not reach statistical significance (Tables 7a-7c).

Table 7a. Group 1: Clinical variables among patients with linear and patch repair.

	Linear repair (n = 13)	Patch repair (n = 11)	*P*-value
Median age (range), years	65	61	0.368
Elective admission, n (%)	7 (54%)	4 (36%)	0.106
NYHA IV symptoms, n (%)	1 (8%)	3 (27%)	0.510
LVEF < 20%, n (%)	1 (8%)	2 (18%)	1.0
Previous cardiac surgery, n (%)	1 (8%)	1 (9%)	1.0
Hospital mortality, n (%)	1 (8%)	1 (9%)	1.0

Table 7b. Group 1: Preoperative CT-morphological variables among patients with linear and patch repair.

	Linear repair (n = 13)	Patch repair (n = 11)	*P*-value
LVEDVI, ml/sqm	153.4 ± 96.8	148.5 ± 70.9	0.893
LVESVI, ml/sqm	114.7 ± 103.4	105.6 ± 72.7	0.811
LVEF, %	32.1 ± 13.5	30.6 ± 17.6	0.827
AEDVI, ml/sqm	50.7 ± 71.8	50.7 ± 46.8	0.998
AESVI, ml/sqm	53.3 ± 73.3	55.4 ± 53.1	0.938
ADA D, sqsm	20.6 ± 16.6	18.3 ± 15.6	0.736
ADA S, sqcm	22.0 ± 20.6	16.9 ± 14.4	0.490
MVAA, sqcm	8.8 ± 1.8	9.3 ± 2.1	0.584
CD, mm	10.1 ± 2.1	11.4 ± 2.6	0.207
TA, sqcm	2.0 ± 0.6	2.3±1.3	0.465
MVCA, angular degree	104.1 ± 13.8	98.0 ± 13.3	0.305
IMD, mm	32.8 ± 5.8	36.4 ± 8.7	0.273
LVEDVI, LVESVI = LV end-diastolic and end-systolic volume index; LVEF = LV ejection fraction; AEDVI, AESVI = aneurysm end-diastolic and end-systolic volume index; ADA D, ADA S = aneurysm defect area, end-diastolic and end-systolic; MVAA = MV annulus area; CD = coaptation distance; TA = tenting area, MVCA = MV closure angle; IMD = interpapillary muscle distance.			

Table 7c. Group 1: Preoperative qualitative CT-morphological characteristics of aneurysm and submitral apparatus in 24 patients with linear and patch repair.

	Linear repair (n = 13)	Patch repair (n = 11)
Morphological characteristics:		
Chordae rupture , n (%)	0 (0)	0 (0)
APM dysfunction , n (%)	0 (0)	2 (18)
PPM dysfunction , n (%)	7 (54)	6 (55)
LV AW dysfunction , n (%)	2 (15)	1 (9)
LV PW dysfunction, n (%)	10 (77)	10 (91)
Aneurysm localization:		
Posterior , n (%)	4 (1)	5 (46)
Posterior medial, n (%)	6 (46)	1 (9)
Lateral, n (%)	3 (23)	5 (46)
APM, PPM = anterior and posterior papillary muscle; LV AW, LV PW = LV anterior and posterior load bearing wall.		

3.6.2. Group 2

Patch ventriculoplasty was performed in only one patient with anteroapical LV aneurysm, in whom the additional closure of a small ventricle septum defect after a huge anteroapical myocardial infarction was needed. In all other patients of group 2 the standard modified McCarthy procedure with several direct Fontan sutures along the aneurysm perimeter without patch application was performed.

4. DISCUSSION

SVR is currently considered an effective therapeutic strategy in the management of patients with ischemic heart failure.

Surgical procedures for the more common anteroapical LV aneurysms are well developed, studied and standardized (7, 16, 18). Posterior LV aneurysms are much more heterogeneous because of the variability of their localization, from posterior medial to posterior lateral, and of their morphological presentation, from acute wall necrosis with a very small defect to pseudoaneurysms with adherence to the pericardium, to true scar aneurysms. Our institutional results show that the majority in this mixed group are patients with pseudoaneurysms (71%), which require prompt surgical intervention because of potential danger of rupture or thrombotic complications (1). Restoration of LV shape and function and preservation of the mitral geometry need to be achieved in such an operation. This task requires quick and at the same time precise planning using all available diagnostic tools before surgery.

4.1. Diagnostic testing

Accurate assessment of the MV, LV and aneurysm morphology and their spatial relations is key for successful surgical ventricular restoration (18). The questions that should be answered with the available imaging modalities concern the localization and size of aneurysm, size of basic wall defect, spatial relationship to and potential involvement of the mitral apparatus, morphological features of the aneurysm in the proper sense of true or pseudoaneurysm, assessment of global LV function, MR and potential adverse hemodynamic effects of the aneurysm.

The most widely available imaging modality remains echocardiography, but its utility depends highly on the patient’s anatomy and the operator’s skill. CMR is now

considered the ‘gold standard’ technique for preoperative non-invasive diagnostic evaluation of ventricular aneurysms (18, 48). Localization, depth and extension of the myocardial scar, asynergic areas, ventricular wall disruption and thrombus formation can be excellently recognized by CMR. However, implanted pacemaker devices or acute cardiac decompensation with the need of full intensive care equipment restrict the feasibility of CMR.

MSCT without technical contraindications, with short examination time and the possibility of unrestricted reconstructions of primarily acquired three-dimensional data, has become increasingly used. Former studies (32, 35, 49) demonstrated that MSCT is a valuable tool to study LV and MV geometry and function. Delgado’s group demonstrated very high suitability of MSCT for studying MV anatomy and provided a concept of geometric indices of clinical interest for the functional MV/LV complex (35).

For our study we developed a combined measurement protocol comprising LV volumetric and functional parameters, geometric parameters of the mitral apparatus, scar localization, morphological characteristics and volumetric assessment of the aneurysm. Precise analysis of LV and MV dimensions and reliable distinction between true and pseudoaneurysm was possible, based on CT data. Complementary to MSCT we used echocardiography for the evaluation of myocardial viability and severity of MR pre- and postoperatively.

4.2. Surgical approaches and results

As stated above, restoration of LV shape and function with preservation of the mitral geometry is the aim of surgical treatment in posterior LV aneurysms.

The parameters of aneurysm localization and morphology, size of the aneurysm neck (ADA) and extension of the perfused pseudoaneurysm allow precise planning of the operative steps. LV and aneurysm volumes, measured in MSCT, allow exact estimation of the required volume reduction of the LV, the possibility of linear repair, and precise sizing of the Dacron patch for patch repair.

Therapeutic aims in antero-apical LV aneurysms are to stop remodeling of the LV through the exclusion of scar areas, to reduce the volume and thus to reduce LV wall stress and restore elliptical LV geometry.

Efficient LV volume reduction toward a physiological range of LVESVI of < 60-70 ml/m^2 is essential to improve survival after surgical repair in both localizations.

Di Donato et al. (50) showed that postoperative LVESVI of > 60 ml/m^2 is an independent predictor of mortality for both aneurysm localizations. The latest analysis of the STICH trial data (51) shows that a statistically significant reduction in mortality was achieved in those patients attaining an LVESVI of less than 70 ml/m^2 in antero-apical localization. In our study group 1 the LVESVI was markedly reduced from 110.6 ± 88.8 ml/m^2 to 50.2 ± 22.9 ml/m^2 ($p = 0.001$), accompanied by a statistically significant increase in LVEF from 31.5 ± 15.1% to 43.4 ± 9.9% ($p = 0.001$) and improvement of LV shape, with a significant decrease of diastolic LV sphericity index from 0.60 ± 0.13 to 0.53 ± 0.07 ($p = 0.007$), showing reverse remodeling of the LV toward more normal shape. In our study group 2 the LVESVI was also markedly reduced from 118.6 ± 49.2 to 63.6 ± 32.1 ml/m^2 ($p < 0.001$), also accompanied by a statistically significant increase in LVEF from 29.6 ± 9.1% to 40.9 ± 10.3% ($p < 0.001$). The postoperative increase in diastolic LV sphericity index from 0.41 ± 0.08 to 0.57 ± 0.16 ($p < 0.001$) in patients with antero-apical aneurysm localization can probably be explained by significant shortening of the LV long axis after aneurysm exclusion.

Concomitant correction of severe MR is an essential part of the surgical procedure in both aneurysm localizations for the following reasons: MR is highly relevant for hemodynamic stability immediately after surgery, for severity of postoperative heart failure and for clinical prognosis and survival (5, 52, 53). Severe MR was present in 7 patients of group 1 (23% of the whole population). Six of them (20%) needed concomitant mitral repair or replacement; this subgroup shows a markedly higher quota of NYHA IV symptoms (33%), LVEF < 20% (17%) and hospital mortality (33%). We identified CT morphological variables of potential predictive relevance for the necessity of concomitant mitral surgery in patients with posterior LV aneurysm – ADA, MVAA, CD and TA – which differ with statistical significance between patients who needed SVR alone and those who needed SVR with concomitant MV surgery in posterior aneurysm localization. Similar data were obtained for anterior aneurysm localization, showing the role of MV annulus remodeling and leaflet tethering in severe MR.

A remarkable result of data analysis for posterior as well as for anterior aneurysm localization is that SVR alone led to a reduction in mild MR (from grade 0.84 ± 0.15 to 0.25 ± 0.09, $p = 0.003$ in group 1), with concomitant improvement in MV geometry as shown by the statistically significant reduction of mitral annulus diameters, mitral

annulus area and tenting area, shortening of coaptation distance, flattening of MV closure angle and reduction of interpapillary muscle distance (Table 5a). These changes reveal a geometric remodeling effect toward a more physiologic shape of the MV by SVR alone through improved LV geometry, realignment of papillary muscles and reduced wall tension.

We therefore propose an MSCT-based algorithm for structured planning of the surgical procedure in posterior LV aneurysms (Fig. 10) that allows not only identification of the mechanism of severe mitral regurgitation as related to LV distortion due to aneurysm but also performance of the LV repair applying an undersized patch and consequent approximation of papillary muscles with recovery of mitral function (Fig. 11).

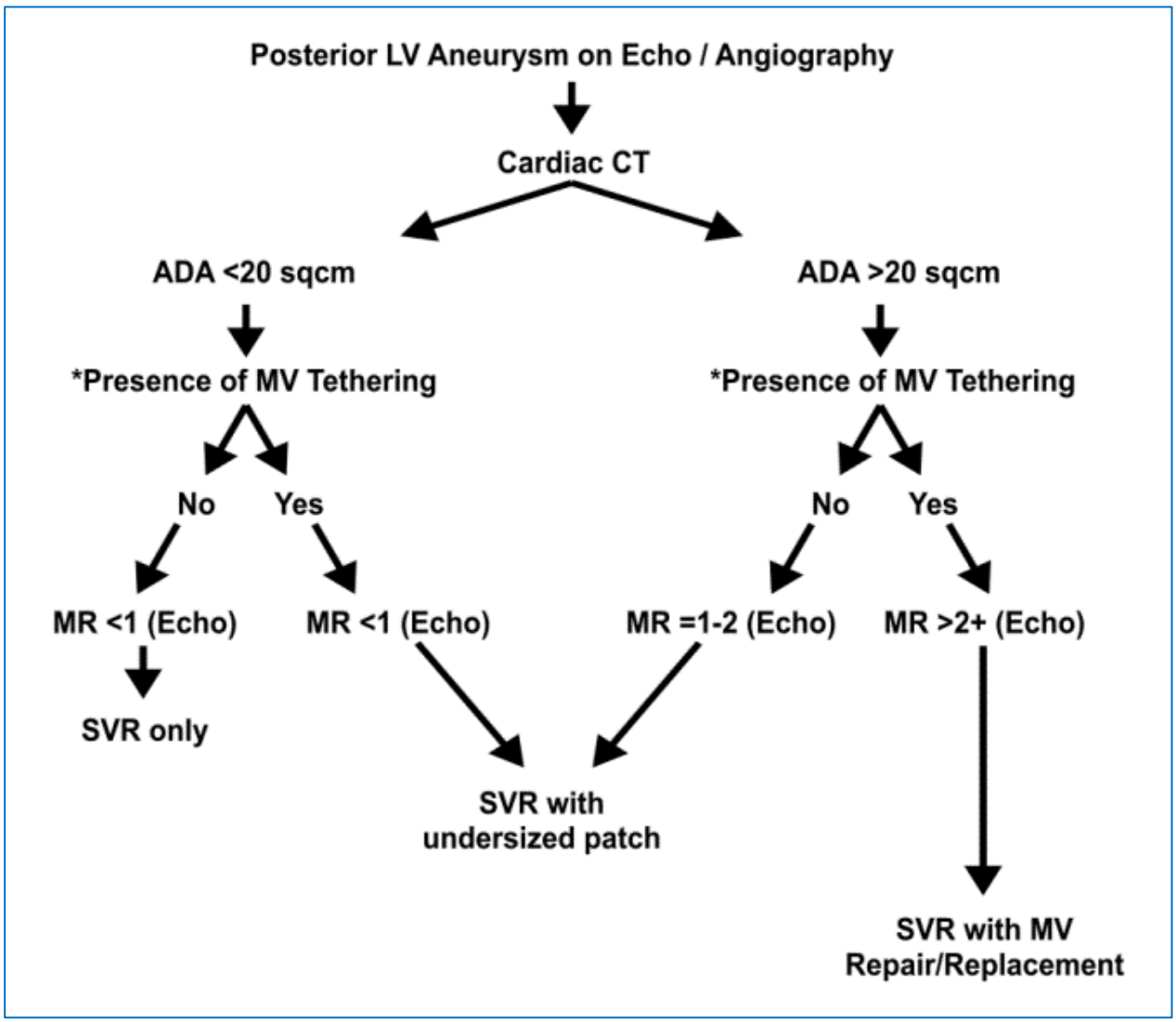


Figure 10. Algorithm for MSCT-guided treatment of posterior submitral LV aneurysm.
Left ventricular distortion: aneurysm defect area (ADA) > 20 sqcm; mitral valve tethering: annulus area > 10 sqcm, tenting area > 2.5 sqcm, coaptation distance > 11 mm, interpapillary distance > 35 mm.

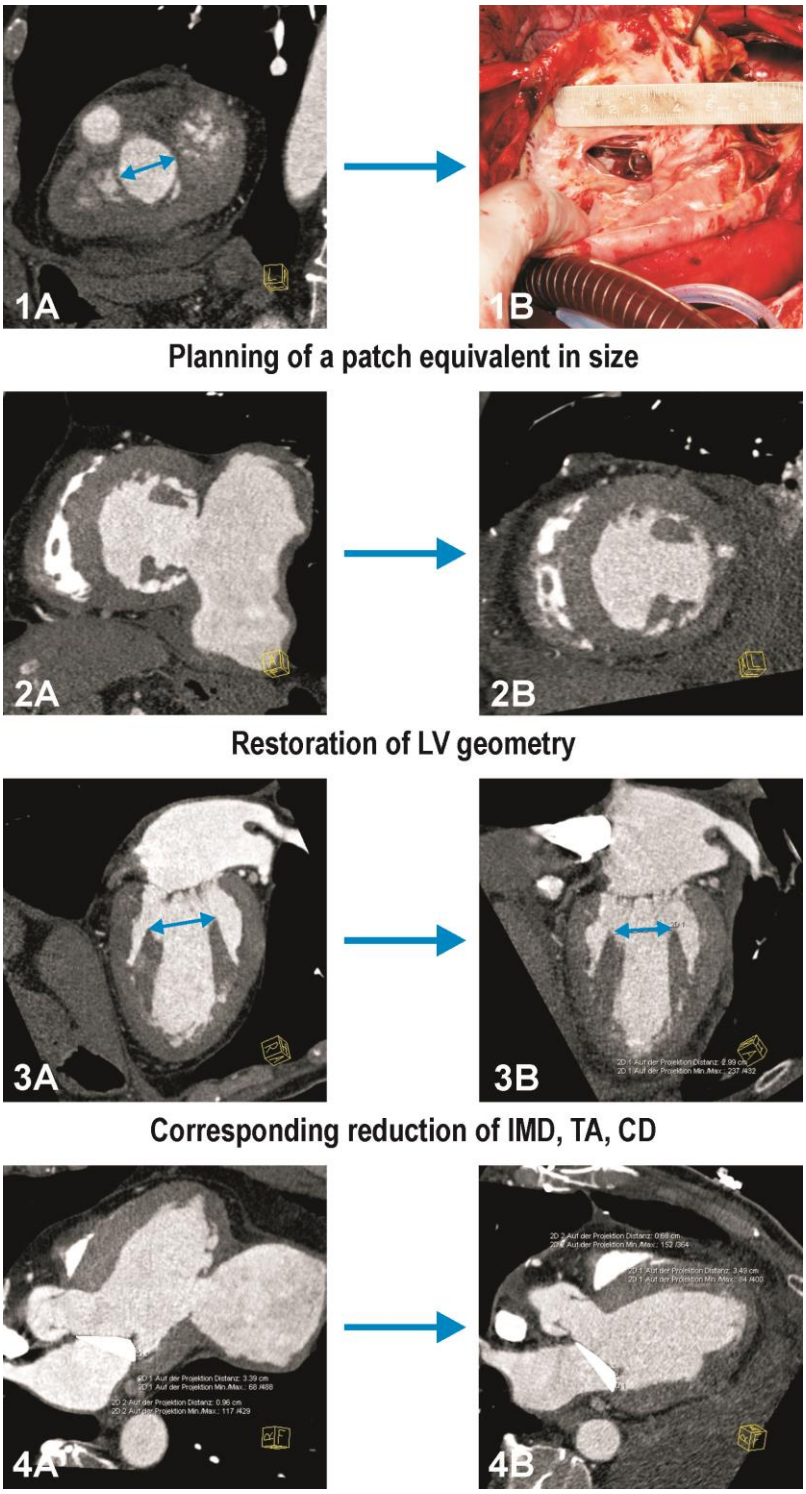


Figure 11. MSCT-guided surgical repair of submitral LV aneurysm according to the algorithm proposed.

(1A) MSCT reconstruction of a plane depicting aneurysm defect, estimation of defect area (ADA = 8.2 qcm) and planning of a patch equivalent in size to preserve the MV geometry, (1B) intraoperative view of the same defect with correlating dimensions. (2) MSCT reconstruction of short-axis view at the level of the papillary muscles: (2A) before surgery with demonstration of LV distortion and slight MV tethering due to aneurysm defect, (2B) after surgery with restoration of LV geometry. 3A and B and 4A and B show the corresponding reduction of the interpapillary muscle distance (IMD from 32 to 26 mm), tenting area (TA from 1.9 to 1.7 qcm) and coaptation distance (CD from 9.4 to 6.4 mm) of the MV with disappearance of moderate MR without additional mitral surgery. This case was reported previously (22) and demonstrates successful application of the MSCT-guided algorithm for the treatment of submitral LV aneurysm presented in this study.

Patients with anterior aneurysm localization (group 2) also showed a reduction in mild MR (from grade 0.79 ± 0.39 to 0.37 ± 0.4, $p < 0.001$) with concomitant changes in MV geometry after SVR without concomitant mitral surgery – a slight reduction of MVAA and significant reduction of TA, CD and IMD (Table 5b). These changes demonstrate that exclusion of scar areas that are not closely related to the submitral apparatus also affects the MV geometry toward normalization solely through reduction of LV volumes and improvement of LV geometry.

4.3. Surgical outcome

In the first group three patients died in hospital, accounting exclusively for the overall 30-day mortality of 10%. All of them died due to septic shock or multiorgan failure and all of them had had combined surgery (1 = SVR + CABG, 2 = SVR + CABG + MV replacement). Thirty-day mortality in the second group was 0%. This difference in early mortality can be explained through the higher number of urgent operations in group 1. Jeganathan et al. (5) reported hospital mortality of 13.3% in patients who underwent combined SVR and MV surgery, 27% of whom had an aneurysm with posterior localization. His group identified NYHA class IV symptoms, preoperative atrial fibrillation, previous cardiac surgery and presence of ischemic MR as significant risk factors for increased operative mortality.

One- and 5-year actuarial survival rates in our study group 1 were 86.5% and 83.1% and one- and 5-year actuarial survival rates in the group 2 were 95.1% and 82.7%, respectively. Mickleborough et al. (7) reported 1-, 5- and 10-year actuarial survival of 92%, 82% and 62% in patients who underwent SVR for anterior and posterior LV aneurysms. Dor et al. (18) found hospital mortality rates of 8.1% and 4.8% and 5-year survival rates of 82% and 85% in early and late series of patients who underwent SVR for both localizations, partially combined with mitral surgery. Garatti et al. reported 30-day mortality of 8.3% in patients after SVR for anterior aneurysm and 5.4% after SVR for posterior aneurysm, with 5-year survival of 80% and 75% and 10-year survival of 60% and 58%, respectively (52). Our institutional hospital mortality and survival rates are absolutely comparable with those of other groups, although our patient collective presumably had substantially higher morbidity and despite the larger proportion of pseudoaneurysms in posterior localization.

4.4. Limitations

It is to be acknowledged that the current study has certain limitations. First, by its nature it is subject to the restrictions of a retrospective study of prospectively collected data sets. Second, the study cohorts are relatively small. However, to the best of our knowledge this study presents the second largest single center experience of posterior LV aneurysm repair and the largest analysing MSCT in such cases.

5. CONCLUSIONS

MSCT represents an efficient diagnostic tool for the assessment of the MV/LV complex. With valuable information about aneurysm localization, its extent and its effect on LV and MV geometry, obtained with MSCT, the reconstruction of LV aneurysms of both posterior and anterior localizations can be performed with good early and mid-term results due to adequate volume reduction and functional improvement, evaluated with MSCT.

The patients who need additional mitral surgery demonstrated advanced changes in MV geometry associated with lesions of the posterior LV wall and posterior papillary muscle in both groups. Postoperative reduction of moderate MR in patients of both groups without additional MV surgery corresponded with reverse remodeling effect on the MV apparatus with significant improvement of MV geometry. MSCT provides, therefore, predictive parameters for necessity of concomitant mitral surgery.

We propose an MSCT-based algorithm that allows identification of the mechanism of severe mitral regurgitation as related to LV distortion in posterior aneurysm localization and performance of the LV repair applying an undersized patch to improve the MV geometry.

Furthermore, this study reports one of the largest series of patients undergoing SVR for aneurysm of the posterior LV wall, in which a surgical procedure was accompanied with systematic analysis of morphological and functional data.

The MSCT imaging approach described here can be successfully used to design new strategies for the interventional and surgical treatment of LV and MV pathology.

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8. List of Publications

Book Chapters

1. Hetzer R, Solowjowa N, Kukucka M, Knosalla C, Röttgen R.
   Correction of aortic valve incompetence combined with ascending aortic aneurysm by relocation of the aortic valve plane through a short-length aortic graft replacement.
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1. Solowjowa N, Penkalla A, Dandel M, Novikov A, Pasic M, Weng Y, Falk V, Knosalla C.
   Multislice computed tomography-guided surgical repair of acquired posterior left ventricular aneurysms: demonstration of mitral valve and left ventricular reverse remodelling.
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European Journal of Echocardiography 2000;1(Suppl 2):S53.

Awards

"Poster Award Silver" for the Poster Presentation:

Solowjowa N.
Potential of multi-slice computed tomography in assessment of mitral valve and left ventricular reverse remodeling after surgical repair.
Cardiac MRI & CT Clinical Update 2013,
Cannes, France, 19.-21.04.2013.

"Poster Award Bronze" for the Poster Presentation:

Solowjowa N.
MSCT-guided Planning and Implementation of a Surgical Procedure in Submitral Left Ventricular Aneurysms with Demonstration of Mitral Valve and Left Ventricular Reshaping.
Cardiac MRI&CT Clinical Update 2015,
Cannes, France, 16.-18.04.2015.

9. Affidavit

Eidesstattliche Versicherung

„Ich, Natalia Solowjowa, versichere an Eides statt durch meine eigenhändige Unterschrift, dass ich die vorgelegte Dissertation mit dem Thema:

Multislice Computed Tomography for Assessment of Reverse Remodeling of Mitral Valve and Left Ventricle after Surgical Repair of Acquired Posterior Left Ventricular Aneurysms Compared to Anterior Localization

selbstständig und ohne nicht offengelegte Hilfe Dritter verfasst und keine anderen als die angegebenen Quellen und Hilfsmittel genutzt habe.
Alle Stellen, die wörtlich oder dem Sinne nach auf Publikationen oder Vorträgen anderer Autoren beruhen, sind als solche in korrekter Zitierung (siehe „Uniform Requirements for Manuscripts (URM)“ des ICMJE -*www.**icmje**.org*) kenntlich gemacht. Die Abschnitte zu Methodik (insbesondere praktische Arbeiten, Laborbestimmungen, statistische Aufarbeitung) und Resultaten (insbesondere Abbildungen, Graphiken und Tabellen) entsprechen den URM (s.o) und werden von mir verantwortet.
Meine Anteile an etwaigen Publikationen zu dieser Dissertation entsprechen denen, die in der untenstehenden gemeinsamen Erklärung mit dem Betreuer, angegeben sind. Sämtliche Publikationen, die aus dieser Dissertation hervorgegangen sind und bei denen ich Autor bin, entsprechen den URM (s.o) und werden von mir verantwortet.

Die Bedeutung dieser eidesstattlichen Versicherung und die strafrechtlichen Folgen einer unwahren eidesstattlichen Versicherung (§156,161 des Strafgesetzbuches) sind mir bekannt und bewusst.“

Datum Unterschrift

Anteilserklärung an etwaigen erfolgten Publikationen

Natalia Solowjowa hatte folgenden Anteil an den folgenden Publikationen:

Publikation 1:
Solowjowa N, Penkalla A, Dandel M, Novikov A, Pasic M, Weng Y, Falk V, Knosalla C. Multislice computed tomography-guided surgical repair of acquired posterior left Ventricular aneurysms: demonstration of mitral valve and left ventricular reverse remodeling.
Interactive Cardiovascular and Thoracic Surgery 2016;23(3):383-90

Beitrag im Einzelnen:
- Ausarbeitung der Methodik
- Erfassung von Daten
- Analyse und Interpretation der Daten
- Anfertigung von Bildmaterial
- Verfassen des Manuskripts

Publikation 2:
Penkalla A, Solowjowa N, Dandel M, Knosalla C.
Giant true inferoposterior left ventricular aneurysm presenting with heart failure: insights from multimodality imaging.
European Journal of Cardio-thoracic Surgery 2014;46(2):333.

Beitrag im Einzelnen:
- Ausarbeitung der Methodik
- Analyse und Interpretation der Daten
- Anfertigung von Bildmaterial

Publikation 3:
Saito T, Solowjowa N, Hetzer R, Knosalla C.
Giant pseudoaneurysm on left ventricular posterolateral wall with an orifice between papillary muscles.
Interactive Cardiovascular and Thoracic Surgery 2014;19(5):869-71.

Beitrag im Einzelnen:
- Ausarbeitung der Methodik
- Analyse und Interpretation der Daten
- Anfertigung von Bildmaterial

Unterschrift des Doktoranden/der Doktorandin

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