An assessment of regression of left ventricular hypertrophy following alcohol ablation of the interventricular septum in patients with hypertrophic cardiomyopathy with left ventricular outflow tract obstruction

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Abstract

Background: Hypertrophic obstructive cardiomyopathy (HOCM) is characterised by asymmetric myocardial hypertrophy, which is most pronounced in the interventricular septum (IVS) and is responsible for the dynamic obstruction of the left ventricular outflow tract (LVOT). Successful alcohol septal ablation (ASA) of the IVS allows to reduce the thickness of the parabasal part of the IVS myocardium and, in most cases, to permanently reduce the gradient in the LVOT.

Aim: To assess, using cardiac magnetic resonance imaging (MRI) and transthoracic echocardiography (TTE), the impact of gradient reduction in the LVOT on the type and severity of left ventricular (LV) remodelling.

Methods: The study included 30 patients (aged 56.9 ± 11.9 years) with HOCM and the mean peak gradient (PG) in the LVOT of 123 ± 33 mm Hg who underwent ASA. MRI measurements were performed before and at 6 months after ASA and TTE measurements were performed before, at 3 months and at 6 months after ASA.

Results: PG in the LVOT decreased to an average of 52 ± 37 mm Hg (p < 0.0001) at 3 months after ASA and to 37 ± 28 mm Hg (p < 0.0001) at 6 months after ASA. TTE revealed a decrease in IVS thickness outside the scar following ASA from 23.6 ± ± 3.5 mm to 19.3 ± 4.0 mm (p < 0.0001) and 19.4 ± 0.4 mm (p < 0.0001) at 3 and 6 months, respectively. There was also a decrease in lateral wall (PW) thickness from 15.9 ± 3.2 mm to 14.9 ± 2.9 mm (p = 0.046) and 14.16 ± 2.00 (p = 0.0065) at 3 and 6 months, respectively. MRI revealed a decrease in IVS thickness from 23.7 ± 2.8 mm to 18.04 ± 4.00 mm (p = 0.0001) at 6 months following ASA. We observed a regression of the PW hypertrophy from 13.2 ± 3.35 mm to 12.18 ± ± 2.4 mm (p = 0.0225). There was a decrease in IVS mass from 108.9 ± 20 g to 91.5 ± 29 g (p = 0.0006). There was a trend towards a decreased LV mass and LV mass excluding IVS mass at 6 months.

Conclusions: A significant decrease in PG in the LVOT is associated with a decrease in LV mass and with regression of LV hypertrophy outside the scar after ASA.

Key words: alcohol ablation, hypertrophic obstructive cardiomyopathy, regression of left ventricular hypertrophy

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INTRODUCTION

Hypertrophic obstructive cardiomyopathy (HOCM) is a genetic disorder of the myocardium [1, 2]. HOCM is characterise by asymmetric myocardial hypertrophy, which is most pronounced in the interventricular septum (IVS) and is responsible for the dynamic obstruction of the left ventricular outflow tract (LVOT) [3, 4]. Patients with heart failure who do not improve on optimal pharmacotherapy require invasive treatment: septal myectomy or alcohol septal ablation (ASA) [4, 5]. Approximately 80% of patients with LVOT obstruction requiring non-pharmacological treatment are patients eligible for ASA or the classical myectomy (Morrow procedure) [6–9]. The aim of the study was to prospectively assess, using cardiac magnetic resonance imaging (CMR) and transthoracic echocardiography (TTE), the impact of ASA on the type and severity of left ventricular (LV) remodelling.

METHODS
Patients
Thirty consecutive patients with HOCM who met the eligibility criteria for ASA were included in the study. Eligible patients were adults who failed to improve on optimal drug treatment, with a resting LVOT gradient of ≥ 50 mm Hg, with echocardiographically confirmed systolic anterior motion (SAM) of the anterior leaflet of the mitral valve towards the IVS and adhesion of the leaflet to the IVS during LV mid-systole, with a thickness of the myocardium of the parabasal part of the IVS of at least 18 mm and an anatomical arrangement of the coronary arteries enabling a safe and effective performance of ASA.

The following patients were excluded: patients with changes in the mitral valve or in the mitral subvalvular apparatus responsible for LVOT obstruction and requiring surgical reconstruction and patients with LVOT obstruction at two levels and a severe mitral regurgitation.

Alcohol septal ablation
Alcohol septal ablation has been described previously [8, 10].

Transthoracic echocardiography
We recorded the parasternal view in the long axis of the LV, the transverse view at the level of the mitral valve, chordae tendineae and papillary muscles, and the apical two- and four-chamber view. We assessed LV dimensions (end-systolic and end-diastolic), left atrial dimensions, LV thickness at diastole (IVS thickness, posterior wall [PW] thickness, anterior wall thickness), obstruction site, magnitude of the gradient in the LVOT, magnitude of the mitral regurgitant jet, the presence of SAM, range of the hypertrophy, changes in the subvalvular apparatus, extent of the hypertrophy with the involvement of the middle segment of the LV and the possible presence of intraventricular obstruction.

Resting TEE (M-mode, 2D and Doppler), in accordance with the study protocol, was performed before ASA and at 3 and 6 months after ASA. Additional follow-up echocardiograms were obtained on the second day post-ASA and before discharge.

Cardiac magnetic resonance imaging
CMR scans were performed in patients in the supine position, using a MAGNETOM Avanto 1.5 T scanner from Siemens (Erlanger, Germany).

Acquisition of CMR images
The first acquisitions aiming to establish the appropriate long axes and the short axis of the heart were planned on the basis of a previous analysis of immobile tomographic cross-sections in three planes (frontal, sagittal and transverse planes) obtained in the half-Fourier acquisition single-shot turbo spin-echo (HASTE) sequence.

This was followed by acquisition of cine balanced steady-state free precession (bSSFP) images to assess the sizes and function of the cardiac ventricles in the end-expiratory phase of breathing. All the acquisitions were ECG-gated. The acquisitions were made in the long axis of the LV (2-, 3- and 4-chamber views) and in the short axis. At the end late gadolinium enhancement (LGE) images were acquired. For this purpose, after completing acquisition of the cine images a paramagnetic contrast agent (Gadovist, Bayer Schering Pharma AG, Berlin, Germany) was given intravenously at the dose of 0.1 mmol/kg. Ten to twenty minutes later LGE images were acquired using an ECG-gated IR-turbo flash gradient recall echo segmented inversion recovery sequence (IR flash with iPat) with an inversion time (TI) in the same cross-sections as the cine images.

Analysis of CMR images
All the cine CMR results were processed using MASS (Medis, Leiden, The Netherlands). The programme automatically determines the outlines of the endocardium (with the exclusion of the papillary muscles) and the epicardium at end-diastole and end-systole with the option of manual correction. LGE was defined as an area of the myocardium whose signal intensity corresponded to a full length at half maximum (FLHM).

The CRM scans were obtained before ASA and at 6 months after ASA.

Statistical analysis
Continuous variables were expressed as means and standard deviations and categorical variables as percentages. The size of the study group (n = 30) was estimated at $\alpha = 0.05$ and $\beta = 0.8$ with the assumption of a 50% gradient reduction in the LVOT as assessed by TTE. The statistical analysis was performed with SAS version 9.2. The statistical significance be-
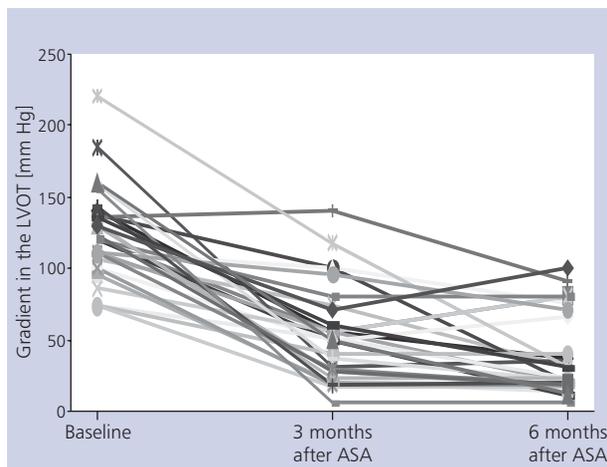
Figure 1. Peak gradient in the left ventricular outflow tract (LVOT) at baseline and at 3 and 6 months after alcohol septal ablation (ASA), as assessed by echocardiography
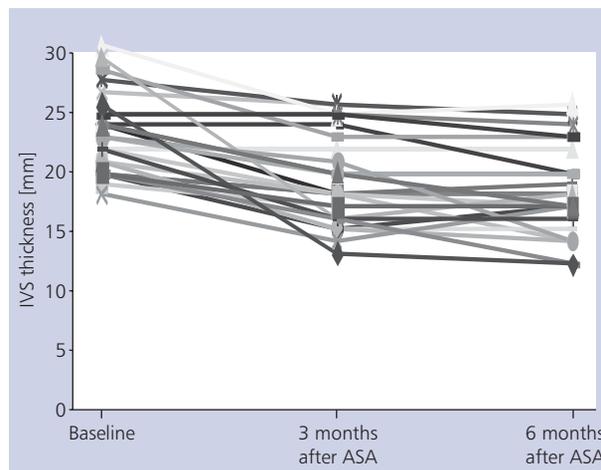

Figure 2. Thickness of the parabasal segment of the interventricular septum (IVS) at baseline and at 3 and 6 months after alcohol septal ablation (ASA), as assessed by transoesophageal echocardiography

tween specific time points was assessed with the paired t-Student test in cases of normally distributed data and with non-parametric tests (the sign test and the signed-rank test) in cases of data that did not meet the assumptions of the normal distribution. The normality of distribution was verified with the Shapiro-Wilk test.

The association between specific variables or their increments was defined using Pearson's linear correlation coefficient and linear regression coefficient if the distributions were found to be normal and using Spearman's correlation coefficient if the distributions were found not to be normal. The significance of the changes for categorical variables was assessed with the McNamara test after recoding into dichotomous variables. The statistical significance was adopted at 0.05.

RESULTS
Patients

This was a prospective study. We included 30 patients with HOCM aged 56.9 ± 11.9 years with the mean PG in the LVOT of 123 ± 33 mm Hg, who underwent ASA.

Alcohol septal ablation

During ASA absolute alcohol (mean volume 1.9 ± 0.5 mL) was administered into the first or second septal branch. As a result of ASA the PG in the LVOT decreased to an average of 55 ± 31 mm Hg directly after the procedure, 52 ± 37 mm Hg at 3 months after the procedure and 37 ± 28 mm Hg at 6 months after the procedure (Fig. 1).

At 6 months following ASA a significant reduction in the LVOT gradient was achieved in 26 patients (87%). Before ASA 7 patients were in the New York Heart Association (NYHA) functional class II and 23 patients were in the NYHA functio-

nal class III. After ASA 25 patients were in the NYHA functional class I–II and 5 in the NYHA functional class III.

Location and area of the infarct

The mean peak increase in creatine kinase MB fraction determined at 6 and 12 hours after ASA was 107 ± 55.9 and 96.9 ± 49 IU, respectively, and that of troponin I 33 ± 32.8 and 38.9 ± 37 ng/mL, respectively. The mean myocardial infarct (MI) size was 14.2 ± 4 g, which corresponded to $8 \pm 4\%$ of the total LV mass and $16 \pm 4.6\%$ of the IVS mass. The scar involved the entire IVS thickness in 12 patients, was limited to the left ventricular portion of the IVS in 4 patients and to the right ventricular portion in a further of 4 patients.

Compared to baseline follow-up CMR scans did not revealed any other new area of fibrosis other than the ASA scar. Due to the limitations associated with the necessity to implant a pacemaker or an automatic implantable cardioverter-defibrillator during the post-ASA observation 10 patients did not undergo a follow-up CRM scan at 6 months after the procedure.

Left ventricular remodelling as assessed by echocardiography

In the group of 30 patients, the parabasal IVS thickness following ASA decreased from 23.6 ± 3.5 mm to 19.4 ± 3 mm and 19.3 ± 4 mm at 3 and 6 months (Fig. 2). There was also a progressive decrease in lateral wall (PW) thickness at 3 and 6 months after ASA.

Six months after ASA there was an increase in end-diastolic LV dimension, a decrease in LVEF and a trend towards decreased anterior wall thickness, increased end-systolic LV dimension and decreased end-systolic LA size (Table 1).

Table 1. Echocardiographic parameters at baseline and at 3 and 6 months after alcohol septal ablation (ASA)

	Study population				
	Baseline	3 months after ASA	P	6 months after ASA	P
PG$_{max}$ [mm Hg]	122.7 ± 32.8	52.13 ± 37	0.0001	37.17 ± 28	0.0001
LVEF [%]	71.04 ± 7.6	71 ± 6.6	0.044	63.3 ± 8.5	0.037
LA [mm]	47.4 ± 6.4	45.9 ± 5.1	0.19	45.2 ± 5.4	0.06
LVdD [mm]	44.5 ± 5.7	46.1 ± 4.6	0.14	47 ± 5.3	0.046
LVsD [mm]	24.6 ± 4.7	25.3 ± 4	0.032	25.5 ± 5.5	0.29
IVSD [mm]	23.6 ± 3.5	19.4 ± 3	0.0001	19.3 ± 4	0.0001
PWD [mm]	15.9 ± 3.2	14.9 ± 2.9	0.046	14.2 ± 2	0.0065
AWD	17.6 ± 3.4	15.8 ± 2.4	0.09	15.3 ± 2.8	0.06

PG$_{max}$ — peak gradient; LVEF — left ventricular ejection fraction; LA — left atrium; LVdD — left ventricular end-diastolic dimension; LVsD — left ventricular end-systolic dimension; IVSD — interventricular thickness; PWD — posterior wall thickness; AWD — anterior wall thickness

Left ventricular remodelling as assessed by magnetic resonance imaging

The parabasal IVS thickness decreased from 23.7 ± 2.8 mm to 18.04 ± 4 mm at 6 months following ASA when measured at the typical site (Fig. 3) and to 13.3 ± 4.9 mm when measured at the level of the area subjected to ablation. We observed a regression of PW hypertrophy, an increase in the end-systolic dimension, an increase in end-diastolic dimension and a decrease in LVEF. There was also a trend towards a decrease in end-systolic LA dimension (Table 2).

Changes in left ventricular mass in patients following alcohol septal ablation as assessed by magnetic resonance imaging

The mean IVS mass decreased from 108.9 ± 20 g to 91.5 ± 29 g (–16%) at 6 months after ASA. There was a trend towards a decrease in the mean LV mass from 224.6 ± 59.3 g to 183.8 ± 77.9 g (–18%) at 6 months and a decrease in the mean LV mass excluding the IVS mass from 96.2 ± 34.6 g to 92.8 ± 60 g (–4%) at 6 months.

An assessment of the relationship between gradient reduction and the severity of remodelling and change in left ventricular function

CMR revealed a relationship between the change in the gradient in the LVOT as measured by TTE and the change in IVS thickness measured at the typical site (p = 0.0289) and at the level of post-ASA scar (p = 0.0001), and the change in IVS mass (p = 0.0185) and the change in LV mass minus IVS mass (p = 0.0432).

In the group of patients in whom ASA was successful and who achieved a gradient reduction below 50 mm Hg there was a relationship between the change of the gradient in the LVOT and the change in IVS thickness in TTE at 6 months (p = 0.0474).

In addition, there was a linear relationship between LV mass reduction as assessed by CMR at 6 months post-procedure and the regression in IVS hypertrophy measured by CMR
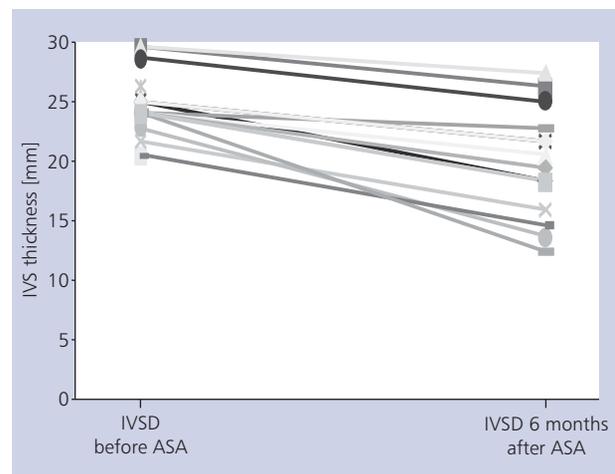

Figure 3. Thickness of the parabasal segment of the interventricular septum (IVS) at baseline and at 6 months after alcohol septal ablation (ASA), as assessed by cardiac magnetic resonance imaging; IVSD — interventricular thickness

at the typical site (p = 0.0005) and LV mass minus IVS mass as assessed by CMR at 6 months (p < 0.0001).

DISCUSSION

The most important finding of our study was the confirmation of the hypothesis that in addition to the reduction of gradient in the LVOT there was also a decrease in IVS thickness also outside the MI area and the free wall of the LV. The IVS decrease results from transmural MI of the parabasal segment of the IVS, while the remaining changes are a consequence of the post-MI LV remodelling lasting up to 6 or, as some authors believe, 12 months after ASA [11, 12].

The early stage of LV remodelling may be associated with a reduced expression of cardiac growth factors (CGFs). Nagueh et al. described a reduced expression of myocardial tumour

Table 2. Cardiac magnetic resonance results at baseline and 6 months after alcohol septal ablation (ASA)

	Study population		
	Baseline	**6 months after ASA**	**P**
LVEF [%]	74.2 ± 5.6	71.4 ± 5.6	0.0352
LV mass [g]	224.6 ± 59.3	183.8 ± 77.9	0.179
AO [mm]	33.4 ± 4.9	33.8 ± 3.8	0.45
LA [mm]	45.7 ± 8	43 ± 8	0.16
LVEDD [mm]	42.4 ± 7.4	46.3 ± 5.8	0.0225
LVESD [mm]	21.2 ± 4.5	27.6 ± 9.5	0.0319
IVSD [mm]	23.7 ± 2.8	18.04 ± 4	0.0001
PWD [mm]	13.2 ± 3.35	12.18 ± 2.4	0.0225
MASS_IVS [g]	108.9 ± 20.2	91.5 ± 28.9	0.006
MASS_rest [g]	96.2 ± 34.6	92.8 ± 60	0.379

LVEF — left ventricular ejection fraction; LV — left ventricle, LA — left atrium; LVEDD — left ventricular end-diastolic dimension; LVESD — left ventricular end-systolic dimension; IVSD — interventricular thickness; PWD — posterior wall thickness; MASS_IVS — interventricular septum mass; MASS_rest — left ventricular mass minus interventricular septum mass

necrosis factor-α (TNFα) in patients HOCM as early as 6 weeks following a significant reduction of the gradient in the LVOT as a result of ASA. The reduced TNFα level correlated with the reduction of cardiac myocyte hypertrophy and with the quantity of intercellular collagen [13, 14]. Our study was not designed to assess the reduced expression of the above factors.

The second stage involves remodelling that is not only associated with organisation of the post-ASA scar but also with a permanent change in the haemodynamic environment. Already in 2000, Faber, in an analysis published in *The Heart* summarising long-term outcomes of ASA in the first 25 patients, noticed regression of hypertrophy as assessed by TTE (M-mode) in the parabasal portion of the PW following a successful reduction of gradient in the LVOT [15]. A year later Mazur, based on an analysis of a group of 26 patients who were assessed with TTE one and two years after ASA, described regression of not only IVS hypertrophy but also of the inferior and anterolateral LV wall hypertrophy [16]. A similar observation, involving 9 patients who had undergone ASA, also on the basis of TTE, was published by Dimitrov in 2002 [17]. In a 6-month follow-up, in a group of patients who had undergone a successful ASA, the author observed regression of hypertrophy of the PW of the LV.

According to the hypothesis put forward by Marian, LV hypertrophy in HCM is a compensatory process, secondary to the reduced contractility resulting from a genetic mutation [18]. The abnormal structure of sarcomere proteins, which are responsible for generating the power of contraction, leads to decreased contractility. The abnormal contractility of cardiac myocytes contributes to their chronic loading. This mechanism, as well as the increased stiffness of the myocardium, contribute to the increases pressures in the LV and the increased loading that induces hypertrophy [18].

In the group of patients with HCM and LVOT obstruction, the increased systolic pressure in the LV adds to the load, and therefore an additional factor affecting LV thickness [19, 20].

As regards more recent studies, those by Van Dockum's group are particularly interesting. Studies using CMR concern a 30-day, followed by 6-month follow-up of a group of 29 patients with HOCM managed by ASA. In the study published in 2004, the authors point to the reduction in the thickness and mass of the IVS and of the remaining walls of the LV as early as 30 days post-ASA [21]. A continuation of this very interesting analysis is the publication by these same authors, supplemented by CMR scans at 6 months, in which the previously observed regression of LV wall hypertrophy was confirmed. The paper points to the correlation between the degree of LV mass reduction and the reduction of the gradient in the LVOT and the location of post-ASA scar. Based on CMR the authors conclude that LV hypertrophy in patients with HOCM is partially reversible and hence at least partially associated with LV loading and not exclusively with a genetic background [22].

Yoan et al. arrived at an opposite conclusion in their paper published a few years ago. They used CMR to assess post-ASA scars and LV remodelling in a group of 52 patients one week and one year post procedure [23]. While the authors did achieve a significant reduction in LV mass at one week and at one year, they failed to observe any changes in the thickness of the other walls of the LV other than the IVS.

The above bibliographical data confirm the significance of the discussion on LV remodelling. Our results support the hypothesis assuming the regression of LV hypertrophy following a successful gradient reduction in the LVOT.

The significant reduction of the gradient in the LVOT was associated with a reduction in mass and regression in IVS hy-

pertrophy outside the post-procedural scar and with a reduction in the mass of the remaining LV walls after exclusion of IVS mass.

Our results confirm that a massive LV hypertrophy in the course of HOCM is not only determined by genetic factors but is also associated with myocardial strain due to elevated systolic pressure in the LV. The strain component of the hypertrophy may be evidenced by the significant regression of hypertrophy at the post-ASA scar site, as seen in TTE and CMR, but also by the regression of hypertrophy in the middle part of the IVS and in the PW, and by the trend towards a decreased thickness of the anterior wall.

Limitations of the study

This was an open-label study and the assessment of the TTE and CMR parameters was unblinded. Due to ethical reasons randomisation in this patient population was not an option.

The relatively small size of the study population was sufficient to confirm the hypothesis related to the strain component of hypertrophy in the course of HOCM. It was, however, insufficient to obtain statistical significance with respect to certain important variables. We merely observed a trend towards total LV mass reduction or the decrease in LA dimensions.

The study did not assess changes in CGFs or TNFá that could affect the early phase of LV remodelling directly following ASA. However, from the perspective of time and experience arising from the work on the project, we believe that future studies of groups with HOCM should also include assessments of these parameters. Their involvement in the stimulation of cardiac myocyte hypertrophy and in the presence and quantity of intercellular collagen in the LV wall in patients with HCM could be an interesting supplement of the present studies.

CONCLUSIONS

In conclusion, a successful ASA of the parabasal segment of the IVS leads to LV remodelling with a significant decrease in IVS mass and thickness also outside the post-ASA scar site and PW.

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Ocena regresji przerostu mięśnia lewej komory po zabiegu ablacji alkoholowej przegrody międzykomorowej u pacjentów z kardiomiopatią przerostową z zawężaniem drogi odpływu lewej komory

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Streszczenie

Wstęp: Cechą charakterystyczną kardiomiopatii przerostowej zawężającej (HOCM) jest odpowiedzialny za dynamiczne zawężanie w drodze odpływu lewej komory (LVOT) asymetryczny przerost mięśnia, najbardziej nasilony w obrębie przegrody międzykomorowej (IVS). Skuteczny zabieg ablacji alkoholowej (ASA) IVS pozwala na redukcję grubości przypodstawnej części mięśnia przegrody i w większości przypadków trwałe obniżenie gradientu w LVOT.

Cel: Celem pracy była ocena za pomocą badań rezonansu magnetycznego (MRI) i ECHO serca (TTE) wpływu redukcji gradientu w LVOT na rodzaj i stopień przebudowy ścian lewej komory (LV).

Metody: Do projektu włączono 30 pacjentów (wiek: 56,9 ± 11,9 roku) z HOCM, z gradientem maksymalnym (PG) w LVOT średnio 123 ± 33 mm Hg, u których przeprowadzono ASA. Pomiary MRI wykonano przed ASA i po 6 miesiącach od zabiegu, pomiary TTE przed ASA oraz po 3 i po 6 miesiącach od zabiegu.

Wyniki: PG w LVOT obniżono średnio do 52 ± 37 mm Hg (p < 0,0001) 3 miesiące po ASA i do 37 ± 28 mm Hg (p < 0,0001) 6 miesięcy po ASA. W TTE grubość IVS poza blizną po ASA zmniejszyła się z 23,6 ± 3,5 mm do 19,3 ± 4 mm (p < 0,0001) i 19,4 ± 0,4 mm (p < 0,0001) po 3 i 6 miesiącach. Grubość ściany bocznej (PW) zmniejszyła się z 15,9 ± 3,2 mm do 14,9 ± 2,9 (p = 0,046) i 14,16 ± 2 (p = 0,0065) mm po 3 i 6 miesiącach od ASA. Obserwowano trend w kierunku zmniejszenia grubości ściany przedniej. W MRI grubość IVS zmalała z 23,7 ± 2,8 mm do 18,04 ± 4 mm (p = 0,0001) 6 miesięcy po ASA. Obserwowano regresję przerostu mięśnia PW z 13,2 ± 3,35 mm do 12,18 ± 2,4 mm (p = 0,0225). Masa IVS zmniejszyła się z 108,9 ± 20 g do 91,5 ± 29 g (p = 0,0006). Obserwowano trend w kierunku zmniejszenia masy mięśnia LV i masy LV z wyłączeniem masy IVS po 6 miesiącach.

Wnioski: Istotne obniżenie gradientu w LVOT wiąże się ze zmniejszeniem masy i regresją przerostu ścian LV poza miejscem objętym blizną po zabiegu ASA.

Słowa kluczowe: ablacja alkoholowa, kadiomiopatia przerostowa zawężająca, regresja przerostu lewej komory

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