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Usefulness of Cardiac Magnetic Resonance Delayed Enhancement technique in the assessment of the postinfarction scar size and the function of the left ventricle in patients after myocardial infarction, treated with Primary Percutaneous Coronary Intervention

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Summary

Background:

The aim of this study is to present usefulness of Cardiac Magnetic Resonance late enhancement technique in the assessment of postinfarction scar location, its precise size, and type, as well as in the evaluation of the left ventricular function (EF, EDV, ESV) in patients after myocardial infarction, treated with Primary Percutaneus Coronary Intervention (PPCI).

Material/Methods:

We analysed a group of 117 patients who underwent myocardial infarction, and were treated with PPCI. CMR with Delayed Enhancement sequence was performed after 6 months with the use of a 1.5 T Signa Excite scanner, surface Coil TORSOPA, and examination techniques including ECG – gated Gradient Echo Breath Hold, ECG – gated Gradient FIESTA Breath Hold, with segmented k-space acquisition and Delayed Enhancement (15 min after i.v. contrast administration). We assessed infarct size and left ventricular function.

Results:

A highly significant correlation was found between: EF and cardiac magnetic resonance infarct size (the bigger the infarct size, the smaller the EF), EDV and cardiac magnetic resonance infarct size (the bigger the infarct size, the higher the EDV), ESV and cardiac magnetic resonance infarct size (the bigger the infarct size, the higher the ESV).

Conclusions:

Cardiac Magnetic Resonance with Delayed Enhancement technique is an effective method to assessed the infarct size and the left ventricular function in patients after myocardial infarction, treated with PPCI.

Key words:

cardio MR • delayed enhancement • left ventricular infarct • postmyocardial infarction scar • left ventricular function parameters (EF, EDV, ESV)

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Background

Heart infarct is an irreversible necrosis of the cells of the heart muscle due to a longer ischaemia. In Poland, the incidence of acute myocardial infarction per year reaches 100 thousand people. Over 85% of the MI cases result from a recent clot occluding a coronary artery stenosed by

atheromatous plaques. Closure of the coronary vessel may also follow from: an acute inflammation in the course of vasculitis, injury or embolism within the coronary arteries. It may as well be the consequence of an increased blood viscosity, as in the case of patients with thrombocytosis or polycythemia vera, or with innate malformations of the coronary arteries [1].

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The absence of flow within the coronary artery, resulting from its occlusion, causes a disturbed perfusion of the heart muscle and diastolic dysfunction, leading to left ventricular systolic dysfunction that precedes the appearance of ECG changes and angina pectoris [2].

Acute ischaemia triggers a cascade of biochemical and pathophisiological changes of the heart muscle. Pathomorphologically recent MI may be diagnosed on the basis of the extension of wall necrosis. The transmural infarction involves the ventricular muscle in its full width, the subendocardial infarction regards only the most inner layer of the heart muscle, and the nontransmural infarction involves more than 25-75% of the wall thickness. In most of the cases, the ischaemia and infarction start at the subendocardial layer, then spread laterally and to the outside, towards the epicardium. The extension of the myocardial infarction is the most important prognostic factor and depends on: the mass of the heart muscle supplied by the occluded vessel, oxygen demand of the heart muscle within the pathological region, efficiency of the collateral vessels and reaction of the tissues to the processes modifying the ischaemia.

Functional changes of the left ventricle include: decreased cardiac output due to disturbed contractibility of the heart muscle at the early stage of MI. There is a decrease in contractile synchrosity of the muscle fibres, leading to hypokinesis (decreased contractility), akinesis (no contractility) or dyskinesis (aneurismal bulge of the wall - to the outside - during the systole). In the course of the myocardial infarction, the contractility of ventricles, and very often also the cardiac output, changes very fast. Approximately 30% of the cases of the transmural infarction are connected with a progress of wall thinning and dilatation of the ventricle within the necrotic wall. This situation may lead to the formation of a dyskinetic region or to thrombus resulting from blood retention in this part of the ventricle [1]. Aneurysms of the heart are formed in the course of infarct widening and the thinning of the myocardium. There are true and spurious aneurysms. The wall of the true aneurysm is made of all layers of the heart wall. True aneurysm means deformation of the left ventricle, both in the diastole and in systole. The wall of the aneurysm shows akinetic or dyskinetic movement during the systole. Spurious aneurysms are formed in the course of a fracture of the free left ventricular wall [2].

Late pathomorphological lesions in the course of a recent myocardial infarction involve the deposition of collagen, until the creation of the scar tissue. The process of scar formation ends approx. 7 weeks after MI [1]. Postinfarction scar is not contractible. When the heart beats, the scar size gets extended, which leads to the thinning of the heart wall.

The earliest possible revascularisation of the coronary vessels considerably decreases the infarct size.

The currently preferred method of patient management in acute myocardial infarctions is the primary percutaneous coronary intervention (PPCI) with stent implantation within the coronary artery that caused the infarct.

Diagnosis of MI bases on a typical history, characteristic changes in the ECG (electrocardiography) and changes of the profile of some specific serum enzymes.

According to the currently applying classification of acute coronary syndromes, patients with pain in the chest are divided into two groups, on the basis of the initial ECG recording: those with ST segment elevation (STEMI - ST segment elevation myocardial infarction) and without ST segment elevation (NSTEMI - Non-ST segment elevation myocardial infarction) [2]. Echocardiography helps in localisation of the ischaemic lesions within the heart wall. It also enables the evaluation of the global and segmental contractility of the left ventricle, when searching for the features of ischaemia or MI. The location and size of infarct can be determined on the basis of revealed disorders of segmental contractility, in the region supplied by the postinfarction artery. The evaluation of segmental contractility of the left ventricle is possible with ECG in about 90% of patients. The degree of contractility disorders depends on the fact whether the ischaemia of the LV wall is transmural or not, and on the existence of the collateral circulation [2].

The whole region of infarction, both at an early stage and after scar formation, may be visualised with Cardiac Magnetic Resonance with Delayed Enhancement technique [3–7]. Moreover, after the CMR examination with transverse (short axis) imaging of the left ventricle, in the sequences of gradient echo, and of steady-state free precession, we obtain all functional parameters of the left ventricle with the use of functional tests, so called cineMR, that reveal a high time-resolution. These parameters are then processed with a special computer program [8–11].

The aim of the work was to assess the usefulness of Cardio MR with DE technique in localisation and evaluation of the exact size and type of the postinfarction scar, as well as the left ventricular function.

Material and Methods

Patients with ECG and echocardiography (performed right after heart attack) results suggestive of the presence of the infarct area were qualified for heart MRI. Those patients underwent a successful coronary angioplasty with stent implantation within the first 6 hours from the moment of MI symptoms presentation.

The following exclusion criteria were applied: claustrophobia, being unable to lie down in supine position for about 45–90 minutes/e.g. severe degenerative lesions of the spine/, maximum weight exceeding 100 kg, pacemaker, insulin pomp or any other device applying medicines, neurostimulator or any other biostimulator, artificial valve (ventriculovenous or ventriculoperitoneal), any ferromagnetic elements within the body – both therapeutical and foreign bodies, patients with all kinds of arrhythmia and irregular heart rate, heart rate above 90/min or below 50/min, being unable to hold breath for about 25 seconds, infections within the upper airways connected with cough, and cough of other origin /allergic cough, difficulties with breathing and so on/, as well as being uncooperative.

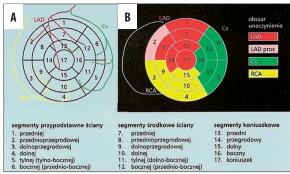


Figure 1. Left Ventricular Segmentation (using *bull-eye* diagram), defined by ASE, with the Main Coronary Artery routs (**A**) and with the assignment of segments to Coronary Arterial territories, signed by colours (**B**). (LAD — Left anterior descending, Cx — Circumflex coronary artery, RCA — Right coronary artery).

Finally, 117 patients (20 women and 97 men) in the age of 33–77 years (mean age of 55) were subjected to heart MRI in the sixth month following the infarction, due to a suspected (on the basis of ECG and echocardiography) postinfarction scar within the muscle of the left ventricle.

The examinations were performed with the use of MR SIGNA EXCITE 1.5 T GE system, with a superficial coil, TORSOPA, and in the following sequences: ECG – gated Gradient Echo Breath Hold for localisers, ECG – gated Gradient FIESTA Breath Hold, with segmented k-space acquisition and Delayed Enhancement – about 15 minutes after the intravenous administration of 20 ml of paramagnetic contrast.

The following parameters of examination were used:

- TR (Time to repeat) = 8 ms,
- TE (Time to echo) = 3.8 ms,
- TI (Time to inversion) = 160–300 ms/set for each patient individually,
- FOV (Field of view) 42×42 cm,
- FA (Flip angle) = $40-50^{\circ}$
- matrix 256×192
- · slice thickness 8 mm/interval 0 mm,
- NEX (Number of excitation) =2.

To visualise the whole left ventricle, from its base (i.e. from the level of the mitral valve) to its tip, the FIESTA sequence was used (a fast imaging method that carries out data acquisition in a steady state), with 9–15 slices, 8 mm each, without intervals between them. Every section included 15 phases of the heart rate, so the patient had to hold his/her breath for 10–17 seconds, depending on the speed of his/her heart rate (from 70/minute to 49/minute).

Acquisition for DE was performed during 15 minutes from the moment of intravenous contrast administration. During that time, the contrast medium gets completely washed out from a normal, healthy muscle, but remains in the regions made up of fibrous tissue mainly.

To obtain possibly the best transverse images of the left ventricle, the time to inversion (TI) was established for each patient individually (range from 160 s to 300 s), to



Figure 2. Tracing of postinfarction scar contours. Delayed enhancement perfusion transverse (short axis) imaging.

make sure that the normal, unchanged myocardium was black in T1 sequence of the MRI examination, i.e. the contrast medium was washed out completely, and that the scar tissue remained white (hyperintensive signal) and revealed a prolonged contrast presence.

DE sequence involved the same sections (in short axis) of the left heart ventricle, as the FIESTA sequence.

The morphology of the left ventricle was evaluated on the basis of images obtained in the FIESTA sequence. The functional parameters of the left ventricle were assessed after being processed with a highly specialised computer program, MASS Medis Plus. The following parameters of the left ventricle were measured: mass, wall thickness, dimensions of the heart chambers, EDV, ESV and enjection fraction (EF). The contractility of the heart muscle was assessed as well.

In order to locate the lesions, the left ventricle was divided into 17 segments (Figure 1).

Postinfarction scar was evaluated in the DE sequence. In this sequence, we may witness a prolonged retention of the contrast medium within the scar tissue. The location of the scar was evaluated on the basis of a division applied in echocardiography – i.e. the division of the LV muscle into 17 segments. Also the type of the scar was defined: subendocardial, nontransmural, transmural.

The total infarct size was measured after the delineation of the scar tissue (Figure 2), i.e. the region of retained contrast medium (regions of delayed enhancement) in DE sequence, in all layers of the transverse section of the left ventricular myocardium.

The analysis of correlations between the examined features (correlation between EF – ejection fraction, EDV – end-diastolic volume, ESV – end-systolic volume, and the size of the scar) was performed with the use of a program 'Statistica'. Pearson linear correlation coefficient was calculated in every case.

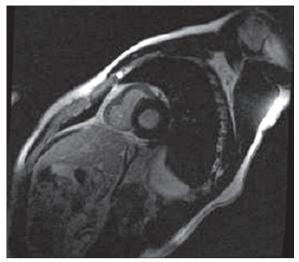


Figure 3. Normal left ventricle myocardium – Delayed enhancement perfusion short axis imaging.

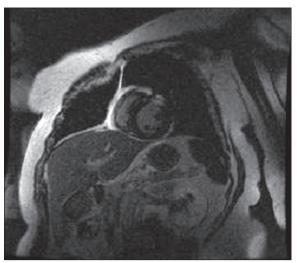


Figure 4. Small subendocardial postinfarction scar within the anterior segment of the left ventricle — a transverse (short axis) delayed enhancement perfusion imaging.

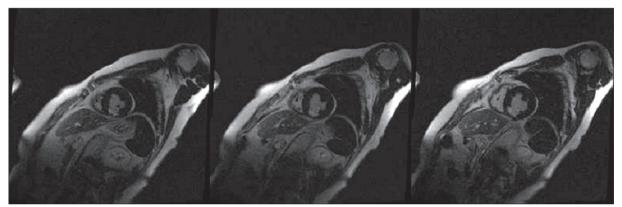


Figure 5. Small subendocardial postinfarction scar within the inferior segment of the left ventricle — a transverse (short axis) delayed enhancement perfusion imaging.

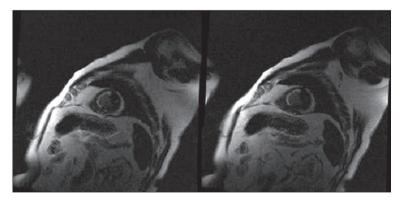


Figure 6. Subendocardial postinfarction scar within the inferoseptal, superoseptal and anterior segment of the left ventricle — a transverse (short axis) delayed enhancement perfusion imaging.

Results

One hundred and seventeen patients were subjected to examinations. In 9 cases (7.7%, 8 men and 1 woman), the postinfarction scar was not present, which was concluded from a diagnosed process of normal reconstruction of the myocardium within the MI site (Figure 3). The left ventricular enjection fraction in those patients ranged from 43.6% to 68.1% (mean value of 56.94%). In the remaining 108 cases (92.3%), we revealed the presence of the postinfarction scar

within the myocardium of the left ventricle. Its volume ranged from 1.07 to 52.15 ml (mean value of 15.30 ml).

Depending on the infarct size, we revealed: subendocardial infarctions (Figures 4–6) – the scar involved the most inner layer of the myocardium; transmural infarctions (Figures 7, 8) – the scar involved the whole muscle in its transverse dimension; and nontransmural infarctions (Figures 9–11) where the scar involved a larger area than in the case of subendocardial infarctions, but it did not

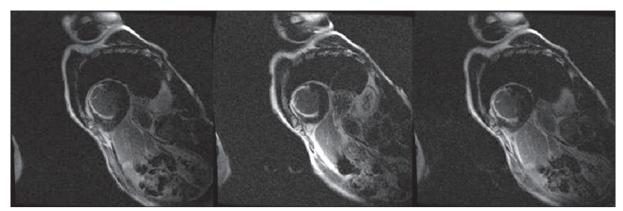


Figure 7. Vast transmural postinfarction scar of the inferoseptal, superoseptal and anterior segments of the left ventricle. Delayed enhancement perfusion transverse (short axis) imaging.

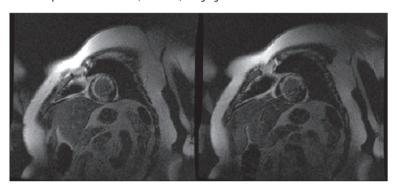


Figure 8. Vast transmural postinfarction scar of the inferior, inferoseptal, superoseptal, anterior and superolateral segments of the left ventricle. Delayed enhancement perfusion transverse (short axis) imaging.

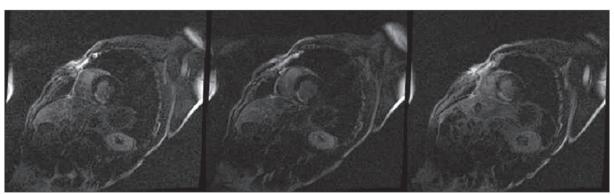


Figure 9. Nontransmural postinfarction scar of the inferoseptal and inferior segments of the left ventricle — Delayed enhancement perfusion transverse (short axis) imaging.

involve the whole wall – most of the time it was 25% to 75% of the pathologically changed wall.

Subendocardial scar was found in 54 patients (50%). The volume of the scar ranged from 1.07 ml to 43.53 ml. Transmural scar was found in 7 individuals (6.5%): volume ranging from 1.85 ml to 50.30 ml, and in 18 cases (16.7%) the scar was nontransmural: volume ranging from 2.66 to 52.15 ml. In the remaining 29 patients (26.8%), we found scars of a mixed type (subendocardial, nontransmural and transmural).

On the basis of the 17-segment division, the location of the postinfarction scar was determined.

In 4 cases (3.7%), the scar was found within the lateral wall of the left ventricle, and the enjection fraction of the left ventricle ranged from 42.3% to 58.2% (mean 50.17%).

In 2 patients (1.8%) with EF of 51.8% and 56.3%, the scar was found within the anterior wall of the LV.

In 13 individuals (12%), the scar was revealed in the intraventricular septum, EF of the left ventricle ranged in here from 29.4% to 63.5% (mean 48.04%).

In 14 cases (13%), the scar involved the inferior wall, and the enjection fraction of the LV ranged from 42.6% to 63.8% (mean 48.2%).

In the remaining 75 cases (69.5%), there were extensive postinfarction scars involving more than one wall in

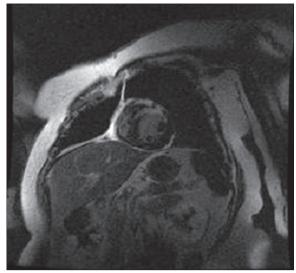


Figure 10. Nontransmural postinfarction scar of the superoseptal and anterior segments of the left ventricle. Delayed enhancement perfusion transverse (short axis) imaging. anterior and superolateral segments of the left ventricle. Delayed enhancement perfusion transverse (short axis) imaging.

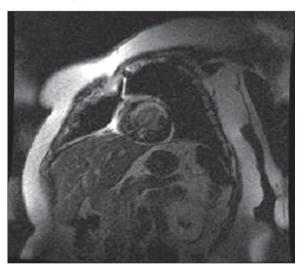


Figure 11. Nontransmural postinfarction scar of the superoseptal, anterior and superolateral segments of the left ventricle. Delayed enhancement perfusion transverse (short axis) imaging.

different segments. In that group, 4 patients (5.3%) had their scars within the inferior and lateral segments, 3 patients (4%) in anterior and lateral segments, 14 patients (18.7%) in septal and anterior segments, and 19 (25%) in septal and inferior segments of the left ventricle.

In 35 patients (47%), the infarction scars were found within 3 or more segments and walls of the LV. In this group of 75 patients, the enjection fraction ranged from 18.0% to 56.0% (mean 42.2%).

When analysing the relations between the location of the scar and of the papillary muscles, it was revealed that in 3 patients (2.8%) the scar involved the posterior papillary

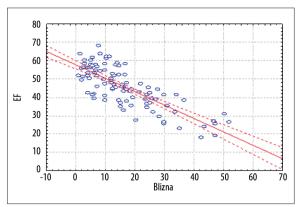


Figure 12. Correlation between postmyocardial scar formation size and EF: r=-0.7929. Confidence interval 0.95.

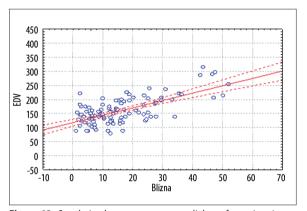


Figure 13. Correlation between postmyocardial scar formation size and EDV: r=0.65639. Confidence interval 0.95.

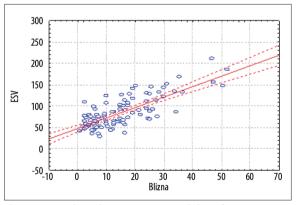


Figure 14. Correlation between postmyocardial scar formation size and ESV: r=0.78455. Confidence interval 0.95.

muscle, in 13cases (12%) – the anterior papillary muscle, and in 4 patients (3.7%) – both papillary muscles of the left ventricle. In the remaining 88 patients (81.5%), there were normal papillary muscles of the left ventricle – not involved with postinfarction scarring.

When analysing the value of EF in the whole examined group of 17 patients, the value of the EF was found out to be approx. 48.08% (from 18.0% to 63.8%). The reference value amounted to $69\pm6\%$ (8).

The enjection fraction exceeding 60% was found in 14 patients (12%); EF of 50-59% in 36 patients (30.8%), EF of 40-49% in 35 patients (29.9%), EF of 30-39% in 21 patients (17.9%), EF of 20-29% in 9 patients (7.7%), and EF below 20% in 2 patients (1.7%).

The end-diastolic volume of the left ventricle in the group of 117 patients amounted to approximately 164.46 ml (from 85.31 ml to 335.5 ml). The reference value: 160±29 ml (8).

Normal values were found in 94 patients (80.4%), slightly increased (range from 190 to 210 ml) in 8 patients (6.8%) – from 195.94 ml to 207.15 ml, and significantly increased (over 210 ml) – from 211.56 ml to 335.5 ml! in 15 patients (12.8%).

The end-systolic value of the left ventricle in the group of 117 patients amounted to 94.31 ml on average (from 35.01 ml to 250.01 ml). The normal reference value amounted to 50 ± 16 ml (8).

Normal values – from 35.01 ml to 64.92 ml were found in 57 patients (48.8%), slightly increased (range from 70 ml to 90 ml) in 17 patients (14.5%) – from 74.50 ml to 89.41 ml, increased (range from 90 ml to 150 ml) in 33 individuals (28.2%) – from 90.17 to 148.25 ml, and significantly increased (over 150 ml) – from 152.29 ml to 250.01 ml! in 10 (8.5%) patients.

Every patient underwent evaluation of the contractility disorders within the LV walls. The following contractile disorders were found: hypokinesis regarding one or two postinfarction segments of the left ventricle – in 62 patients (53%), hypokinesis within more than two segments with segmental akinesis – in 2 cases (1.7%), hypokinesis in more than two segments, with segmental dyskinesis – in 24 patients (20.5%), as well as hypokinesis, within more than two infarction segments, with segmental akinesis and dyskinesis – in 7 patients (6%).

In 22 patients (18.8%), a normal contractility of the left ventricular muscle was found.

We examined interrelations between the scar size and different parameters of the LV (EF, EDV, ESV). A significant reverse correlation was found between the scar size and the value of the left ventricular enjection fraction – the bigger the scar size, the lower the enjection fraction, which was equal to worse prognosis.

The end-diastolic and end-systolic volumes were increasing with the increasing size of the scar within the left ventricle.

The statistical analysis showed a very high reverse correlation between the scar size and EF (Figure 12). Pearson linear correlation coefficient amounted to -07929. There was a high correlation between the scar size and EDV (r=0.65639) (Figure 13) and between the scar size and ESV (r=0.78455). These correlations were statistically significant, with significance level of 0.05 (Figure 14). The size of the scar and its transmurality significantly influenced the abnormal systolic function of the left ventricle, resulting in slight hypokinesis (especially in patients

with subendocardial scars) or akinesis (mainly in patients with larger nontransmural scars), as well as dyskinesis (in patients with transmural scars).

Discussion

Myocardium evaluation in patients with ischaemic heart disease has posed a challenge to clinicians for many years. Alive myocardium may fully regain its normal function after a course of reperfusive treatment (PPCI in most cases). The region of the heart muscle that underwent necrosis or scarring is not able to regain its normal contractile function. That is why it is very important to be able to evaluate the heart muscle, and not to put the patient at unnecessary risk connected with coronary revascularisation in detected necrosis or postinfarction scar [12].

Until recently, the diagnosis of the myocardial infarction based solely on a typical history, characteristic ECG changes and changes in the profile of specific serum enzymes.

Additionally, echocardiography was helpful in localisation of the ischaemic focus within the wall of the heart muscle. However, it was not giving a clear answer to the question whether the postinfarction region of the myocardium underwent a: normal remodelling, necrosis or scarring.

The whole postinfarction region may be visualised with the DE technique in Cardio MR examination which allows for determination of the presence of the postinfarction scar, its location and range.

Timo et al. [13] performed Cardio MR examination in 22 patients, after 5 days and after about 5 months from the MI incident. These patients were treated with PPCE and stent implantation. Every examination was carried out with the use of a 1.5T scanner. FIESTA sequence was used to evaluate the volume and contractility of the walls of the LV, and T1 IR GE sequence with DE at 10–20 minutes after intravenous contrast administration, to evaluate the postinfarction scar.

In the studied group of patients, 15 cases (70%) had a postinfarct scar within the anterior segment, and 7 persons (30%) within the inferior or lateral segment.

The authors found a significant increase in EF – from $48\pm11\%$ in the first examination, to $55\pm9\%$ in the follow-up study, an increase in EDV: from 160 ± 44 ml to 172 ± 55 ml and insignificant differences within ESV – from 84 ± 4 ml to 81 ± 4 ml. In this group, contractility disorders of the LV walls were found in patients with a CMR-detected nontransmural scar, involving more than 75% of the wall thickness, and in patients with transmural scars.

Rasmus Sejersten Ripa et al. [14] performed CMRs in 58 patients, right after their incidence of heart attack and PPCI treatment, and then after 1 month and 6 months. The examinations at 6 months revealed a significant increase in EF, as compared to both previously performed CMR examinations – from $52.9\pm13.4\%$ to $61\pm11.9\%$, an increase in EDV – from 128 ± 35 ml to 140 ± 35 ml, and insignificant ESV differences – from 63 ± 34 to 57 ± 30 ml. With the use of the DE sequence, the authors found a significant decrease in the infarct size

in the examination performed 1 month after the baseline examination (right after the myocardial infarction). They did not reveal any significant difference in the infarct size when comparing the results of the examination performed after one month and the one carried out after 6 months.

Some authors – Gerber et al. [15] – believe that the best period for the follow-up CMR study is 7 months after the heart attack. Others – Taylor et al. [16] think this is 3 months.

In our material, where the patients had their CMR examination performed after 6 months from the day of their myocardial infarction, it was possible to detect the process of scar formation. Basing on our studies and experiences of other authors [12–16], we believe that the time of 3–7 months from the moment of MI is optimal for the evaluation of the postinfarction scar, as no significant differences in the scar size were revealed in that period, because the strongest remodelling of the ischaemic myocardium can be observed during the first month from the PCI procedure [14].

All authors of the articles ([12–16], and others) revealed a high correlation between the infarct size or the scar size and the disorders of LV contractility, as we did. No regions of abnormal contractility were found if the scar was not present.

However, Rasmus Sejersten Ripa et al. [14] point out to the fact that the objective CMR results in patients after MI not always correlate with subjective feelings of the patients. It is worth remembering. It should also be underscored that when the infarct is complicated by aneurysm, it is possible then to define (with the use of the DE sequence) the range of the postinfarction aneurysm, and its type: true or spurious, and to evaluate the proportion of the scar within the aneurysm.

Conclusions

The MRI examination with Delayed Enhancement technique evaluated the presence, precise location and size of the infarction scar. Moreover, the examination allowed for evaluation of the character of muscle 'remodelling': scar formation – subendocardial, nontransmural, transmural or unchanged (normal) structure of the heart muscle. The statistical analysis revealed a very high reverse correlation between the scar size and EF. There is also a high correlation between the scar size and EDV, and between the scar size and ESV.

Cardio MR is currently an optimal diagnostic method in the detection and evaluation of the postinfarction scars.

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