



ORIGINAL ARTICLE

Differences in clinical status and LV performance in post myocardial infarction patients with apical versus basal wall motion abnormalities

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Abstract: Introduction – Since apical rotation is a key determinant of left ventricular (LV) performance we hypothesized that the worse outcome observed in pts with anterior myocardial infarction (MI) may be, at least partly, a consequence of LV apical involvement. Methods – We analyzed retrospectively from our database 245 echocardiographic studies of pts with documented history of LV MI. Pts with either apical or basal akinetic segments, (between 2 and 5 segments) were included in our study and two groups were formed: 30 pts with apical and 30 pts with basal wall motion abnormalities (WMA). Results – A higher NYHA class was found in pts with apical WMA as compared to pts with basal WMA despite similar echocardiographic parameters of LV function. Wall motion score index (WMSI) was significantly higher in patients with apical dysfunction and emerged as the only predictor of functional NYHA class in the whole study population. No further statistical difference was observed between groups after correcting for WMSI. Conclusions – In our study the poorer functional status of post-MI patients with apical WMA as compared to patients with basal ischemic involvement was rather related to a more extensive ischemic area than to the actual location of akinetic segments.

Key words: echocardiography, myocardial infarction, left ventricular function, ischemic apical dysfunction

Rezumat: Introducere – Pornind de la premisa importanței apexului ventricular stâng (VS) în menținerea unei performanțe cardiace normale, ne-am propus să evaluăm impactul disfuncției apicale asupra statusului funcțional și al parametrilor ecocardiografici de funcție VS la pacienți cu infarct miocardic și afectare apicală comparativ cu pacienți cu afectarea segmentelor miocardice bazale. **Material și metodă** – Am evaluat retrospectiv examinări ecocardiografice aparținând unui număr de 254 de pacienți cu istoric de IM și am constituit două grupuri de studiu: 30 de pacienți cu segmente akinetice localizate apical și respectiv 30 de pacienți cu segmente akinetice bazale. **Rezultate** – Clasa funcțională NYHA a fost mai avansată în cazul pacienților cu afectare apicală în ciuda unor parametri ecocardiografici de funcție VS similari între grupuri. Scorul de cinetică segmentară (SCS), mai avansat în cazul pacienților cu disfuncție apicală, a fost predictor independent al clasei NYHA. După corectarea pentru SCS nu au mai existat diferențe semnificative între grupuri din perspectiva clasei NYHA. **Concluzii** – In studiul nostru clasa funcțională mai avansată a pacienților cu tulburare de cinetică apicală post IM comparativ cu pacienții cu tulburare de cinetică bazală s-a corelat mai degrabă cu extensia zonei afectate decât cu localizarea propriu-zisă a acesteia, în ciuda unor parametri similari de funcție VS.

Cuvinte cheie: ecocardiografie, infarct miocardic, funcție ventriculară stângă, disfuncție ischemică apicală

INTRODUCTION

Recent studies report a decrease in acute MI (MI) mortality in parallel with greater use of reperfusion therapy, primary percutaneous coronary intervention, modern antithrombotic therapy, as well as secondary prevention treatments. Still, mortality rate remains significant reaching 12% at 6 months after acute MI, which justifies the ongoing effort to improve quality of care for these patients¹. Thus, identifying prognostic

factors for a better risk stratification remains an utmost important goal in this setting.

Many previous studies have focused on the risk factors for adverse clinical outcome after acute MI. Age, history of prior MI, cardiovascular risk factors, cardiac biomarkers, Killip class, ejection fraction, infarct location, restrictive left ventricular (LV) filling pattern, left atrial volume, multivessel disease, renal dysfunction are among the parameters correlated with early and late mortality in this setting².

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Anterior location of MI has been identified by previous studies as a predictor of poor prognosis. Several studies have compared the clinical outcome of patients after anterior or inferior MI and even after adjusting for infarct size a higher incidence of congestive heart failure and cumulative cardiac mortality was observed in patients with anterior MI³⁻⁶.

Anterior MI may lead to LV apical dysfunction in a significant proportion of cases. Since apical rotation is the main determinant of torsion and, during diastole, apical recoil contributes to suction and ensures adequate LV filling at low pressure⁷, we hypothesized that the worse outcome in patients with anterior MI may be, at least partly, a consequence of LV apical involvement.

METHODS

We analyzed retrospectively from our database 245 echocardiographic studies of patients with documented history of LV MI (older than I year) referred for echocardiography to EUROECOLAB between January 2014 and May 2015. Patients with either apical or basal akinetic segments, (between 2 and 5 segments) were included in our study and two groups were formed on the basis of the wall motion abnormality location: Group I – apical, and Group 2 – basal wall motion abnormalities.

All echocardiographic studies were reviewed by a single observer blinded to clinical data and wall motion score index (WMSI) as well as parameters of systolic and diastolic function were assessed on stored images retrieved from the database. Clinical files of the included patients were reviewed for demographic data, cardiovascular risk factors, NYHA class, medication, age of MI. Coronary angiography performed during the hospitalization for MI was available in all patients. The most recent coronary angiography was considered in order to assess the extension of coronary artery disease in each patient.

Exclusion criteria were: severe valvular disorders, prosthetic valves, permanent atrial fibrillation and left bundle branch block.

For statistical analysis we used the SPSS software v. 19. Our results are expressed as mean±standard deviation or, where indicated, as absolute values or percentage.Variables were compared using T-test, ANOVA and Chi-squared test as appropriate. Statistical significance was fixed at a p value less or equal than 0.05. The contribution of multiple factors to a result was assessed by multivariate analyses.

RESULTS

The final study population included 60 patients $(60\pm9 \text{ yr})$ divided in two groups as previously mentioned.

Clinical characteristics of the two groups of patients are presented in Table I. There were no statistically significant differences between groups regarding age, gender, body surface area and values of systolic and diastolic blood pressures or cardiovascular risk factors (p<0.5 for all).

A statistically significant difference was found with regard to NYHA class, patients with anterior wall motion abnormalities (Group I) presenting a higher NYHA class as compared to patients with basal wall

Table I. Clinical characteristics of the study groups

	Apical dysfunction Group I	Basal dysfunction Group 2 (N=30)	р
	(N=30)		
Men, n(%)	24(80%)	28(93%)	0,12
Age (years)	62±9	59±8	0,21
NYHA class	2,07±0,7	1,57±0,8	0,02
Body surface area (m²)	1,9±0,2	2±0, I	0,11
Systolic blood pressure (mmHg)	I 38±25	128±10	0,17
Diastolic blood pressure (mmHg)	82±15	74±8	0,07
Heart rate (bpm)	64±8	69±10	0,12
Hypertension, n(%)	22(73%)	24(80%)	0,51
Diabetes, n(%)	8(26%)	10(33%)	0,57
Dyslipidemia, n(%)	21(70%)	23(76%)	0,53
Obesity, n(%)	9(30%)	I I (36%)	0,58
moking, n(%)	14(46%)	17(56%)	0,43
amily history, n(%)	2(6%)	l (3%)	0,55
NYHA = New York Heart Association			

	Apical dysfunction Group I (N=30)	Basal Dysfunction Group 2 (N=30)	р
LVEDV (ml/m ²)	67±14	64±11	0,43
LVESV (ml/m ²)	28±12	28±8	0,89
LVEF (%)	47,7±5	50±3	0,08
Cardiac index (I/min/m²)	2,5±0,7	2,5±0,6	0,88
S (septal) (cm/s)	6,5±1,2	7,2±1,3	0,09
S (lateral) (cm/s)	6,7±1,4	7,5±2	0,33
WMSI	1,6±0,2	I,4±0,I	<0,001
LV mass(g/m ²)	116±22	113±24	0,60
E (cm/s)	66±22	63±13	0,52
A (cm/s)	68±15	72±20	0,36
E/A	I,06±0,6	0,9±0,3	0,28
e' average(cm/s)	6,5±1,5	7,1±1,8	0,21
E/e'average	10,6±4,6	9,4±2,2	0,20
ETD (ms)	190±38	211±49	0,08
LA volume indexed (ml/m ²)	37±9	39±11	0,57
PASP	30±6	28±7	0,29

E = peak protodiastolic mitral flow velocity; A = peak telediastolic mitral flow velocity; e' = peak protodiastolic mitral annulus velocity; LVEF = LV ejection fraction; WMSI, LVEDV, LV end-diastolic volume; LVESV = LV end-systolic volume; ETD = E wave deceleration time; LA = left atrium; PASP = pulmonary artery systolic pressure; S = peak systolic velocity of the mitral annulus

motion abnormalities (Group 2) (2,07±0,7 vs 1,57±0,8; p=0.02).

The extension of coronary artery disease according to the most recent coronary angiography available in each patient showed no statistically significant differences between groups. Moreover, no significant differences between groups were observed regarding renal function, medication or age of MI.

Echocardiographic parameters of systolic and diastolic function

No statistically significant differences were found between the two groups with regard to LV ejection fraction and echocardiographically estimated cardiac index. Still, WMSI was significantly higher in patients with apical dysfunction $(1,6\pm0,2 \text{ vs } 1,4\pm0,1; p<0.001)$.

The echocardiographic parameters of LV diastolic function were similar between groups (Table 2).

Correlates of NYHA class in the studied groups

When evaluating the impact of clinical and echocardiographic parameters on NYHA class within the whole study population, we found a significant correlation between the NYHA class index and: LV ejection fraction (p=0.02), WMSI (p=0.02), LV mass index (p=0.03) and diastolic parameters: peak E (p=0.01), E/A ratio (p=0.001), and E/E' ratio (p<0,001). With multivariate analysis the WMSI remained the only predictor of functional NYHA class.

In order to correct for the influence of WMSI study on functional status we matched participants according to WMSI and two subgroups were formed: a subgroup of 20 patients with apical wall motion abnormality and a subgroup of 18 patients with basal wall motion abnormality (Table 3).

There was no further statistical difference observed between the two groups with similar WMSI and similar

	Apical dysfunction (N=20)	Basal dysfunction (N=18)	р
Men n(%)	18(90%)	18 (100%)	0,16
Age (years)	63±8	59±10	0,21
NYHA class	I,9±0,8	1,56±0,9	0,25
Body surface area (m²)	I,9±0,I	2±0, I	0,21
Systolic blood pressure (mmHg)	143±29	128±11	0,15
Diastolic blood pressure (mmHg)	86±14	74±7	0,01
Heart rate (bpm)	63±8	69±10	0,14
NYHA = New York Heart Association			

Table 4. Echocardiographic variables in the two	groups of patients with comparable WMSI (WMSI)
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	Apical dysfunction (N=20)	Basal dysfunction (N=18)	Р
LVEDV(ml/m ²)	70±15	66±11	0,37
LVESV (ml/m ²)	28±14	30±8	0,58
LVEF (%)	49±6	48±4	0,65
Cardiac index (l/min/m ²)	2,6±0,7	2,5±0,5	0,64
Peak S (septal) (cm/s)	6,6±1,4	7,2±1,3	0,24
Peal S (lateral) (cm/s)	6,4±1,3	7,5±2	0,25
WMSI	I,5±0,I	1,4±0,07	0,09
LV mass (g/m ²)	115±23	120±24	0,47
Peak E (cm/s)	69±22	62±13	0,28
Peak A (cm/s)	68±15	69±21	0,91
E/A	I,08±0,5	0,96±0,4	0,50
Peak E' (cm/s)	6,6±1,4	7,1±1,9	0,46
E/E'	,2±5,	9,4±2,5	0,20
TD (ms)	194±39	209±53	0,32
LA volume (ml/m ²)	37±8	38±10	0,77
PASP	32±7	26±8	0,18

E = peak protodiastolic mitral flow velocity; A = peak telediastolic mitral flow velocity; e' = peak protodiastolic mitral annulus velocity; LVEF = LV ejection fraction; WMSI, WMSI; LVEDV = LV end-diastolic volume; LVESV = LV end-systolic volume; ETD = E wave deceleration time; LA = left atrium; PASP = pulmonary artery systolic pressure; S = peak systolic velocity of the mitral annulus

echocardiographic parameters of systolic and diastolic function with regard to NYHA class (p=0.25) despite the different location of wall motion abnormalities (Table 4).

The main correlates of NYHA class within this group were: peak E (p=0,008), E/A ratio (p=0,001), E/E' ratio (p=0,00). Multivariate analysis retains E/E' ratio (p=0.01) as predictor of NYHA functional class.

DISCUSSIONS

The main results of our study are: (1) A poorer functional status in patients with apical vs basal wall motion abnormalities despite similar LVEF and cardiac index was associated in our study with a higher WMSI and thus to a more extensive LV wall abnormality, rather than with actual location of the MI. (2) LV apical dysfunction was not associated in our study with worse echocardiographic parameters of LV diastolic function in Group I as compared to Group 2. (3) WMSI emerged as a predictor of NYHA class in the whole group of post-MI patients. (4) No further difference with regard to NYHA class index was found between groups after matching for both WMSI and LVEF irrespective of the location of wall motion abnormalities. E/E' ratio emerged as a predictor of functional class within this population.

Previous studies have identified the anterior location of MI as an indicator of poor prognosis³⁻⁶. A study led by Hands et al, which included 398 patients with anterior MI and 391 patients with inferior MI, concluded that anterior location is an indicator of poor prognosis, in both the short and the long term, independently of the size of infarction as evaluated by the levels of peak creatine kinase measured in the acute phase⁵. The authors mentioned as possible explanations for this finding the qualitative differences between the anterior and inferior walls, the higher risk of dilatation and thinning of the infarcted zone associated with anterior location, as well as the limitations of assessing the extent of damage to the left ventricle via the level of peak creatine kinase considering that patients with an inferior MI can also associate a right ventricular lesion which could have contributed to the rise in creatine kinase. Stone et al obtained similar results after matching patients for size of infarction and found an increased mortality, a higher incidente of congestive heart failure and a lower ejection fraction among patients with an anterior MI4. More recent studies did not provide further insights on this topic.

Anterior location of MI may be associated with LV apical dysfunction. Previous studies reported LV apical rotation as the main determinant of systolic torsion whereas apical relaxation during diastole is responsible for a rapid decrease in intraventricular pressure gradient, thus contributing to suction and ensuring the ventricular filling at low pressures⁷. A study conducted by Opdahl et al underlined the major contribution of apical rotation to the systolic torsion of the left ventricle and also proposed apical rotation, as evaluated by speckle tracking echocardiography, as an index of ventricular torsion⁸. Data regarding the clinical impact of LV apical dysfunction in patients with anterior MI is scarce. Diminished LV torsion and prolonged untwisting was reported by Takeuchi et al in 30 patients with anterior MI and abnormal LV systolic function as a result of reduced apical rotation⁹.

Our study revealed a statistically significant difference regarding the functional status expressed as NYHA class index between the two main study groups. Thus, a higher NYHA class index was found in patients with apical wall motion abnormality compared to the patients with basal wall motion abnormality despite similar LVEF and cardiac index. There was no statistical difference between groups regarding echocardiographic parameters of diastolic function although higher values for the E/A ratio, shorter deceleration time of E wave velocity and higher average E/E' ratio were found in the apical dysfunction group.

Among the echocardiographic parameters of systolic function a statistically significant difference between groups was noted with regard to the WMSI despite similar LVEF and ecocardiographically assessed cardiac index. Thus, patients with anterior MI presented a more extensive wall motion abnormality. Moreover the WMSI was independent predictor of NYHA class index in the whole study population. This tendency of anterior MI to be more extensive than inferior MI was already described by Thanavaro et al in a study that demonstrated the independent prognostic value of anterior infarction location after correction for infarction size assessed by the level of cardiac enzymes10. In our study, after correction for WMSI there was no statistically significant difference between patients with apical versus basal wall motion abnormalities with regard to NYHA class index or echocardiographic parameters of LV performance.

LIMITATIONS

Despite the large number of echocardiographic studies that have been retrospectively evaluated to test our hypothesis, the number of cases included in the present analyses was restricted due to the inclusion criteria requiring a specific location and extension of wall motion abnormalities. Thus, further confirmation of our results in a larger number of patients would be required in order to increase statistical accuracy and draw conclusions over the whole population with MI.

Clinical implications

The prognostic significance of ischemic LV apical involvement in patients with MI have never been studied. However, since the haemodynamic importance of normal LV apical function with regard to an efficient LV performance is well known it would be of interest to retest our hypothesis in a prospective study including a larger population in an attempt of improving the clinical outcome of post MI patients by more refined risk stratification.

CONCLUSIONS

In our study the poorer functional status of post-MI patients with apical wall motion abnormality as compared to patients with basal ischemic involvement was rather related to a more extensive ischemic area than to the actual location of akinetic segments and to apical dysfunction. Moreover, WMSI was the main predictor of functional class in our study population.

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