CARDIOVASCULAR MEDICINE

Short and long term prognostic importance of regional dyskinesia versus akinesia in acute myocardial infarction

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Heart 2002;**87**:410–414

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Accepted 28 January 2002

Background: The prognostic importance of dyskinesia after acute myocardial infarction is unknown, and recommendations have been made that dyskinesia be included in calculations of wall motion index (WMI).
Objective: To determine whether it is necessary to distinguish between dyskinesia and akinesia when WMI is estimated for prognostic purposes following acute myocardial infarction.
Design: Multicentre prospective study.
Patients: 6676 consecutive patients, screened one to six days after acute myocardial infarction in 27 Danish hospitals.
Interventions: WMI was measured in 6232 patients, applying the nine segment model, scoring 3 for hyperkinesia, 2 for normokinesia, 1 for hypokinesia, 0 for akinesia, and –1 for dyskinesia. Calculation of WMI either included information on dyskinesia or excluded this information by giving dyskinesia the same score as akinesia.
Main outcome measures: Long term outcome (up to seven years) with respect to mortality.
Results: Dyskinesia occurred in 673 patients (10.8%). In multivariate analysis, WMI was an important prognostic factor, with a relative risk of 2.4 (95% confidence interval (CI), 2.2 to 2.7), while dyskinesia had no independent long term prognostic importance (relative risk 1.00; 95% CI, 0.89 to 1.12). For 30 day mortality dyskinesia had a relative risk of 1.23 (95% CI, 1.00 to 1.53) (p = 0.045).
Conclusions: Echocardiographic evaluation of left ventricular systolic function shortly after an acute myocardial infarct gives important prognostic information, but the presence of dyskinesia only has prognostic importance for the first 30 days.

The extent of damage to the left ventricle after acute myocardial infarction is important in prognosis and may be determined by echocardiography. By dividing the left ventricle into segments and scoring each of these segments as hyperkinetic, normokinetic, hypokinetic, akinetic, or dyskinetic, the average score—the wall motion index (WMI)—becomes an indicator of overall left ventricular systolic function. WMI is closely correlated with left ventricular ejection fraction determined by radionuclide cardiography.[1 2] Radionuclide cardiography is a reference method for estimating the left ventricular ejection fraction and includes both hyperkinesia and dyskinesia.

We have recently shown that hyperkinesia added to the WMI is a positive prognostic indicator,[3] while dyskinesia and akinesia are negative factors, reflecting deterioration in regional left ventricular systolic function. It is thought that dyskinesia may be associated with a worse prognosis than akinesia. Our aim in this study was to test whether the distinction between dyskinesia and akinesia is useful in evaluating prognosis after acute myocardial infarction using the WMI score.

METHODS

Patients

The patients in this study were screened (and subsequently one quarter were randomised) for the trandolapril cardiac evaluation (TRACE) study, which was designed to determine the effect of trandolapril on mortality in patients with a moderate to severe reduction in left ventricular systolic dysfunction after acute myocardial infarction.[4] A detailed description of the TRACE study and demographic information collected from the screened population has been reported previously.[5] Information was obtained prospectively for all patients with acute myocardial infarction in 27 hospitals in Denmark. Consecutive male and female patients over the age of 18 years with acute myocardial infarction were screened between day 1 and day 6 after the onset of symptoms. The diagnosis was confirmed by the combination of chest pain and ECG changes suggestive of infarction or ischaemia accompanied by an increase in cardiac enzymes to twice the upper normal value of the local hospital laboratory.

Between 1 May 1990 and 7 July 1992, 7001 consecutive episodes of acute myocardial infarction were evaluated in 6676 patients. In 444 of these, echocardiography was not done, either because the patient died or because of technical problems. The study population for this analysis thus consisted of 6232 patients.[1]

During screening, baseline data and information on complications experienced in hospital were collected in all patients, allowing a detailed description of the entire population, irrespective of whether they were subsequently entered into the TRACE study. One important item of data collected was the central personnel register (CPR) number. All Danish residents have a unique CPR number, and all deaths taking place within the country are recorded using this number. Mortality in all the screened patients was checked after at least five years by interrogating the CPR register. Survival

Abbreviations: D-WMI, dyskinetic wall motion index: information on dyskinesia included when calculating wall motion index; ND-NH-WMI, dyskinesia coded as akinesia and hyperkinesia replaced by normokinesia when calculating WMI; ND-WMI, non-dyskinetic wall motion index (that is, dyskinesia coded as akinesia); NH-WMI, wall motion index where hyperkinesia was replaced by normokinesia when doing the calculations; TIMI, thrombolysis in myocardial infarction study reflow grade; TRACE, trandolapril cardiac evaluation; WMI, wall motion index.

Table 1 Characteristics of 6232 patients without and with dyskinesia

	Without dyskinesia (n=5559)	With dyskinesia (n=673)	p Value
Median age (years) (5th and 95th centiles)	68 (46 to 84)	70 (51 to 85)	<0.01
Men (%)	69	65	0.07
Systemic hypertension (%)	23	22	0.82
Diabetes mellitus (%)	10	13	0.01
Previous AMI (%)	23	27	0.01
Previous angina (%)	36	39	0.19
Anterior Q wave AMI (%)	23	58	<0.01
Inferior Q wave AMI (%)	34	10	<0.01
Bundle branch block (%)	8	9	0.08
Patient delay (hours)*	3.00	3.75	<0.01
Thrombolytic treatment (%)	42	39	0.14
Ventricular fibrillation (%)	6	9	0.01
Atrial fibrillation (%)	19	31	<0.01
Congestive heart failure (%)	50	73	<0.01
Hyperkinesia (%)	10	24	<0.01
Dyskinetic WMI (D-WMI)†	1.5 (0.8 to 2.0)	1.0 (0.6 to 1.4)	<0.01
Non-dyskinetic WMI (ND-WMI)‡	1.5 (0.8 to 2.0)	1.1 (0.7 to 1.6)	<0.01
D-WMI distribution (%)			<0.01
<0.8	4	17	
0.8 to 1.2	24	61	
1.3 to 1.6	31	21	
>1.6	42	1	

*Patient delay is the time from start of symptoms until arrival at hospital.
†Where dyskinesia is included in the estimation of the wall motion index.
‡Where dyskinesia is not included in the estimation of the wall motion index.
AMI, acute myocardial infarction; WMI, wall motion index.

information for 28 non-Danish patients was censored at the time of discharge.

Echocardiography

At the time of screening an echocardiographic examination was recorded on videotape by the investigator and send to a core laboratory, where the WMI was calculated by two people using the nine segment model described by Heger *et al*.[6] Segmental scores and WMI were calculated as described by Berning and Steensgaard-Hansen.[7] Validation of this method has been reported previously.[1] In the model, dyskinesia is represented by a score of −1, akinesia 0, hypokinesia 1, normokinesia 2, and hyperkinesia 3. Dyskinesia is distinguished from akinesia because it is characterised by outward movement during systole. Hyperkinesia was considered present when movement of the myocardium was more pronounced in the effected area than in other areas with normal function. With this reverse scoring system, a normal left ventricle has a WMI of 2.0. A WMI of 1.2 corresponds to an ejection fraction of 35%.[8] The WMI calculated in this way contains specific information on dyskinesia (and hyperkinesia), and is referred to below as the dyskinetic wall motion index (D-WMI). As the database contained information on the motion of each segment, it was possible to eliminate the distinction between dyskinesia and akinesia, giving both a value 0, and then to recalculate the WMI. In this way a non-dyskinetic wall motion index (ND-WMI) was obtained.

Ethics

The TRACE study was registered with the Danish National Board of Health and the Danish Data Protection Agency, and was approved by all local ethics committees in Denmark. Patients were informed about the study both orally and in writing, before written consent was obtained.

Statistical analyses

The baseline characteristics of patients with and without dyskinesia were compared using the χ^2 test for discrete variables and the Kruskal–Wallis test for continuous variables. In the case of continuous variables, we give the median with 5th to 95th centiles. Survival curves were generated from Kaplan–Meier estimates, using the log-rank test for significance. Multivariate analyses and relative risk were calculated as a hazard ratio (relative risk), derived from the Cox proportional hazards regression model and using a backward selection procedure that initially included relevant baseline variables (referred to in table 1). For continuous variables the hazard ratio is given per unit. For the analyses of subgroups, estimates of hazard ratio (relative risk) and the associated 95% confidence intervals (CI) were generated using a Cox proportional hazard model. Interaction analysis was conducted by means of a likelihood ratio test. Calculations were performed with the SAS software (SAS Institute, Cary, North Carolina, USA). All tests of statistical significance were two tailed, and probability (p) values of < 0.05 were considered significant.

RESULTS

Dyskinesia occurred in at least one segment in the 673 patients in whom an echocardiogram was analysed (10.8%), and in 765 segments overall, giving an incidence per segment of 1.36%. In patients without previous infarction, the incidence was 10.3%, and in patients without any symptoms of previous ischaemic heart disease (n = 3304)—that is, previous acute myocardial infarction, angina pectoris, or congestive heart failure—the incidence was 9.8%. Dyskinesia occurred mainly in the apex of the left ventricle and was recorded in 23.4% of patients with anterior acute myocardial infarction and in only 69 patients (3.6%) with inferior acute myocardial infarction ($p < 0.01$). In patients without previous acute myocardial infarction, these figures were 23.9% and 2.9%, and in patients without previous ischaemic heart disease, 23.4% and 2.5%, respectively ($p < 0.001$).

The main differences between the baseline characteristics of patients with and without dyskinesia are shown in table 1. Patients with dyskinesia were older and generally had a higher prevalence of other important risk factors, both previous disease and complications during the index infarction. Patients with dyskinesia were admitted later than patients without dyskinesia, but there was no difference in the time to echocardiography in the two groups.

There was a trend for less dyskinesia in patients given thrombolysis: in those treated with thrombolysis (n = 2567),

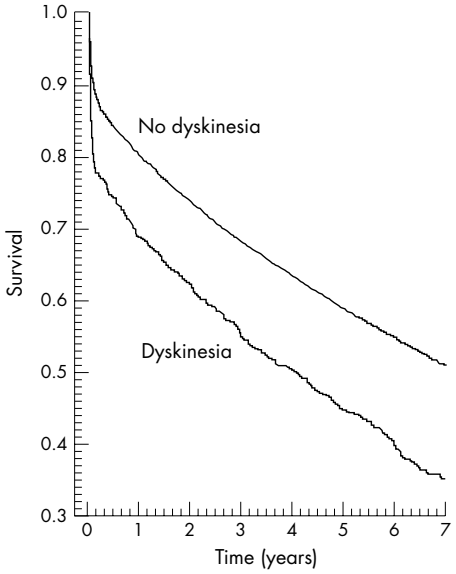

Figure 1 Survival curves for 6232 patients in relation to the presence of dyskinesia.

dyskinesia occurred in 10.2%; in those not treated with thrombolysis it occurred in 11.4% ($p = 0.143$). In patients without previous acute myocardial infarction the figures were 9.9% and 10.7%, respectively ($p = 0.40$), and in patients without known ischaemic heart disease, 9.3% and 10.4% ($p = 0.27$). Arrhythmias occurred more often in patients with dyskinesia (p~0.01). In patients without dyskinesia, median WMI was 1.5 (5th to 95th centile interval, 0.8 to 2.0). This was significantly different from the WMI (D-WMI) in patients who had dyskinesia (1.0, 0.6 to 1.4; $p < 0.0001$). If dyskinesia was excluded—that is, coded as akinesia—WMI (ND-WMI) was still significantly different from that in patients without dyskinesia (1.1; 0.7 to 1.6; $p < 0.0001$). Hyperkinesia was recorded in 24% of patients with dyskinesia and in 10.0% of those without ($p < 0.01$). If hyperkinesia was replaced by normokinesia when calculating WMI (NH-WMI), the NH-WMI for the dyskinetic group of patients was 1.0 (0.5 to 1.4), and for the non-dyskinetic group, 1.5 (0.8 to 2.0) ($p < 0.001$). If dyskinesia was excluded—that is, coded as akinesia—the wall motion index (ND-NH-WMI) was still significantly different from that in patients without dyskinesia (1.1, 0.6 to 1.4; $p < 0.0001$).

Overall survival curves according to the presence or absence of dyskinesia are shown in fig 1. Patients with dyskinesia had a 30 day mortality of 19.2%, and a one, three, and five year mortality of 31.3%, 44.9%, and 54.9%, respectively. In patients without dyskinesia the corresponding figures were 9.4%, 19.9%, 32.2%, and 41.2%. In univariate analyses the relative risk associated with dyskinesia was 1.6 (1.4 to 1.7) ($p = 0.0001$).

This apparently adverse prognosis associated with dyskinesia was completely explained by patients with dyskinesia having more depressed left ventricular systolic function: in a multivariate model including dyskinesia and ND-WMI, only ND-WMI had prognostic importance. In this analysis, the relative risk of dyskinesia was 1.00 (0.90 to 1.11), and that of ND-WMI, 4.1 (3.8 to 4.6).

The results of multivariate backward selection analyses where dyskinesia was forced into the model, after including all variables that were either of prognostic importance in univariate analyses or showed significant differences between the groups, are shown in table 2. Dyskinesia did not have any long term prognostic significance. The relative risk of ND-WMI was 2.5—equal to that obtained in a multivariate analyses including D-WMI and the same set of variables. If hyperkinesia was replaced by normokinesia when calculating WMI (NH-WMI), dyskinesia did not have prognostic importance in multivariate analyses (relative risk 0.99, 0.88 to 1.11; $p = 0.85$).

Table 2 Relative risk of death after acute myocardial infarction: results of multivariate analyses with non-dyskinetic wall motion index (ND-WMI)

	Relative risk (95% CI)	p Value
Dyskinesia	1.00 (0.89 to 1.12)	1.00
Age*	1.05 (1.04 to 1.05)	<0.01
Systemic hypertension	1.2 (1.1 to 1.3)	<0.01
Diabetes mellitus	1.4 (1.2 to 1.6)	<0.01
Previous angina pectoris	1.2 (1.1 to 1.3)	<0.01
Anterior Q wave AMI	0.9 (0.8 to 1.0)	<0.01
Bundle branch block	1.2 (1.0 to 1.3)	0.01
Patient delay †	1.003 (1.001 to 1.005)	<0.01
Thrombolysis	0.7 (0.7 to 0.8)	<0.01
Atrial fibrillation	1.3 (1.2 to 1.4)	<0.01
Ventricular fibrillation	1.7 (1.4 to 1.9)	<0.01
Congestive heart failure	1.7 (1.6 to 1.9)	<0.01
Non-dyskinetic WMI*	2.4 (2.2 to 2.7)	<0.01

*Relative risk is per unit for continuous variables.
†Patient delay is time from start of symptoms until arrival at hospital.
AMI, acute myocardial infarction; CI, confidence interval; WMI, wall motion index.

Table 3 Relative risk of dyskinesia in selected subgroups: results from multivariate analyses including non-dyskinetic wall motion index (ND-WMI)

Subgroup	Relative risk of dyskinesia (95% CI)
ND-WMI <0.8	1.0 (0.8 to 1.4)
ND-WMI 0.8 to 1.2	0.9 (0.8 to 1.1)
ND-WMI >1.2 to 1.6	1.1 (0.9 to 1.4)
ND-WMI >1.6	1.2 (0.5 to 2.7)
Anterior Q wave AMI	1.00 (0.85 to 1.18)
Non-anterior Q wave AMI	0.97 (0.83 to 1.14)
No previous AMI	0.96 (0.85 to 1.10)
Previous AMI	0.99 (0.81 to 1.21)
Systemic hypertension	0.98 (0.79 to 1.22)
No systemic hypertension	0.97 (0.86 to 1.11)
Diabetes mellitus	1.07 (0.81 to 1.42)
No diabetes mellitus	0.99 (0.88 to 1.12)
Thrombolytic treatment	0.94 (0.77 to 1.14)
No thrombolytic treatment	1.01 (0.89 to 1.16)

AMI, acute myocardial infarction; CI, confidence interval.

The prognostic importance of dyskinesia in the various subgroups is shown in table 3. Dyskinesia did not add prognostic information at any level of WMI. As dyskinesia mainly occurred in anterior Q wave infarction, multivariate analyses were repeated in the patients with an anterior infarct ($n = 2445$). Again dyskinesia did not add prognostic information. Dyskinesia did not add any long term prognostic information in patients treated with or without thrombolysis. This was also the case in the "first infarct" and "no previous ischaemic heart disease" subgroups.

As the multivariate analysis reflected a time period of up to seven years, we could have overlooked the possibility that dyskinesia was a marker of early mortality. To test for the short term prognostic importance of dyskinesia, multivariate models were constructed looking only at death occurring up to 30 days after the infarct. In univariate analysis, dyskinesia was associated with a relative risk of 2.2 (1.8 to 2.7). The results of multivariate analyses including ND-WMI are shown in table 4. The factors that had prognostic importance for the first 30 days differed slightly from those found important for long term prognosis, though the only major difference was the

Table 4 Relative risk of 30 day mortality after acute myocardial infarction: results of multivariate analyses with non-dyskinetic wall motion index (ND-WMI)

	Relative risk (95% CI)	p Value
Dyskinesia	1.23 (1.00 to 1.53)	0.045
Age*	1.04 (1.03 to 1.05)	<0.01
Systemic hypertension	1.2 (1.0 to 1.5)	0.03
Previous AMI	0.8 (0.6 to 0.9)	<0.01
Patient delay†	1.009 (1.005 to 1.014)	<0.01
Thrombolysis	0.8 (0.6 to 1.0)	0.02
Congestive heart failure	2.9 (2.2 to 3.8)	<0.01
Ventricular fibrillation	3.6 (2.9 to 4.4)	<0.01
Atrial fibrillation	1.2 (1.0 to 1.5)	0.02
Non-dyskinetic WMI*	3.7 (2.9 to 4.8)	<0.01

*Relative risk is per unit for continuous variables.
†Patient delay is time from start of symptoms until arrival at hospital.
AMI, acute myocardial infarction; CI, confidence interval; WMI, wall motion index.

relative risk of ventricular fibrillation, which was 3.6 in the short term and 1.7 in the long term. Dyskinesia had independent prognostic importance for the 30 day mortality, though this was lost if it was included in the wall motion index (D-WMI). The relative risk of dyskinesia then became 1.09 (0.88 to 1.35), and that of D-WMI 3.7 (2.9 to 4.8). If hyperkinesia was excluded from the wall motion index (NH-WMI), dyskinesia did not have prognostic importance in relation to the 30 day mortality (relative risk 1.21, 0.98 to 1.49; p = 0.08). Dyskinesia did not have prognostic importance for the time interval 30 days to seven years (relative risk 0.90, 0.79 to 1.02; p = 0.09) after including wall motion index (ND-WMI).

DISCUSSION

The principal finding of this study is that left ventricular systolic function measured by the wall motion index shortly after acute myocardial infarction is a powerful predictor of long term mortality; however, no useful prognostic information was obtained by distinguishing between akinesia and dyskinesia as components of the WMI. Intuitively, dyskinesia would be expected to result in a worse prognosis as it leads to a reduction in the left ventricular ejection fraction. Removing dyskinesia from the WMI by changing it to akinesia should have shown that dyskinesia had independent prognostic importance. However, this was found only in multivariate analysis of 30 day mortality. Dyskinesia had no prognostic value for long term mortality. Thus dyskinesia is a weak prognostic factor that extinguishes rapidly over time after acute myocardial infarction.

Variables describing regional left ventricular dysfunction and used for prognostic purposes include akinesia, dyskinesia, and aneurysm formation. These variables are related, but describe different facets of dysfunction. In contrast to akinesia, dyskinesia is characterised by outward movement of a wall segment during systole. Defined in this way dyskinesia includes aneurysm formation; aneurysms, however, show bulging during diastole as well, and a thinner myocardium.[9] Infarct expansion is a definite outward bulging and alteration in the curvature of the infarcted left ventricular segment, with hinge points persisting during both systole and diastole.[9] These phenomena and their relation to long term prognosis need to be considered in the light of any improvements resulting from revascularisation procedures.

In previous reports of patients with transmural anterior acute myocardial infarction, dyskinesia has been found in 21.5%[10], 25%,[11] and up to 35% of cases,[12] and in 1% of segments.[13] The incidence of aneurysm formation is 12.4% in necropsy studies.[14] The low incidence of dyskinesia in the present study may partly reflect the fact that the study was performed after generalised use of thrombolytic treatment had become the norm. It may also be relevant that on average echocardiography was performed three days after the infarct, at a time when dilatation of the left ventricle is not yet complete[15 16] and when infarction expansion is still progressing. Interpretation of the type of infarct related wall motion in the days following acute myocardial infarction is further complicated by the fact that low sympathetic drive may improve function and high sympathetic drive decrease it (by increasing myocardial ischaemia), as with dobutamine stimulation.[17–19] Assuming a similarity between the echocardiographic observations during the first days after acute myocardial infarction and during dobutamine stimulation, some regions may show improved wall motion during the acute phase.[20] Also, the echocardiographic pattern of an akinetic segment at rest may become dyskinetic during peak stress. As both akinesia and dyskinesia most probably represent infarcted myocardium, motion in this area may be determined by the function of myocardium remote from the infarct site. Thus a hyperkinetic area may change an akinetic area to a dyskinetic area mechanically.

In this study we observed dyskinesia with the same frequency in patients treated with thrombolysis or not. In previous studies, patients treated with thrombolysis did not have a significantly lower rate of dyskinesia, but there was slightly better wall motion of the infarct areas if complete or near complete reperfusion (TIMI grade 3 flow) was obtained by thrombolysis,[21 22] especially if collateral supply was present.[23 24] However, in one study a relation between thrombolytic treatment (or high TIMI flow) and the size of the infarct was already present from day 2 to 3.[25] The reason for this difference may be that measurement of the perimeter of the infarct is a more sensitive marker of dyskinesia than echocardiographic estimation.

The decreasing systolic function of the left ventricle during the first days or weeks after acute myocardial infarction, reflected in the decrease in left ventricular ejection fraction or increase in left ventricular end systolic volume, is mainly caused by the disappearance of hyperkinesia.[21] Echocardiographic demonstration of changes in the infarct area within the first hours and days following symptoms shows that 66% of initially hypokinetic segments remain unchanged up to 10 days after the infarct and most of the rest improve.[26] In contrast to this is the finding that anterior segments with dyskinesia have an 83% risk of aneurysm formation within one year.[10] In the present study we drew no distinction between dyskinesia and aneurysm (as echocardiography was done on average three days after the infarct). Previously, formation of an aneurysm[10] and infarction expansion[9] have been shown to be of prognostic importance. As dyskinesia does not apparently have any long term prognostic significance it is possible that the majority of our patients did not develop an aneurysm. Our finding of only short term prognostic importance for dyskinesia could be explained by the formation of aneurysms in some patients, with their dismal short term prognosis,[12] and a larger group without progression to aneurysm formation.

Conclusions

Dyskinesia and akinesia represent infarcted myocardium, and they are thus important factors in prognosis. However, when used in the wall motion index score for determining prognosis, a distinction between these two phenomena does not seem to be justified. This is in contrast to the opposite end of the scoring system, where a distinction between hyperkinesia and normokinesia is advantageous.

ACKNOWLEDGEMENT

The TRACE study was supported by a grant from Knoll AG, Ludwigshafen, Germany and Roussel Uclaf, Paris, France.

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IMAGES IN CARDIOLOGY

Pyoderma gangrenosum presented as a refractory wound infection following permanent pacemaker implantation

Pyoderma gangrenosum (PG) is a rare painful ulcerative cutaneous disease of unknown aetiology. An underlying systemic disorder is associated in up to 50% of cases. We present a case of PG which developed after permanent pacemaker implantation in an 85 year old woman with a background history of monoclonal gammopathy and polymyalgia rheumatica. This was initially treated as a severely infected pacemaker site, which led to extraction of her pacemaker system. Her wound only started to heal after she was treated with systemic steroids.

PG has been reported following various surgeries but to the best of our knowledge none has been reported following permanent pacemaker implant. Such diagnosis should be born in mind as it may avoid unnecessary pacemaker system extraction, a lesson we have now learned.

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