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Relation of infarct site to 15 year prognosis in patients who survived for 28 days after a first myocardial infarction

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SUMMARY Six hundred and eighty four patients (629 men), all aged under 60 years, who had survived for 28 days after a first acute myocardial infarction were studied to determine the influence of the site of infarction on long term prognosis. The infarct site was not significantly related to age nor to extent of infarct at the time of the acute episode. Mechanical complications were more common in patients with anteroseptal infarctions, while atrioventricular conduction disturbances were more commonly found in those with inferior infarction. The site of infarction was not related to smoking habits or angina before the infarction or at 2 year follow up.

Life table methods did not show any relation between infarction site and morbidity or mortality either two years or 15 years after the initial infarction.

Studies of in-hospital and short term outcome in patients with myocardial infarction have shown that prognosis is poorer in anterior myocardial infarction than in inferior infarction, even when size of infarction is controlled for.[1–3]

The relation of site to long term prognosis has been less frequently examined, and although several studies have shown a worse long term outcome in anterior infarction[4–7] others have shown no difference.[8–10]

We examined the relation of site to 15 year morbidity and mortality in patients aged < 60 who survived for at least 28 days after a first acute myocardial infarction.

Patients and methods

This study is based on long term follow up study of two groups of patients who survived for at least 28 days after a first myocardial infarction when they were < 60. Between 1965 and 1975 inclusive only men were recruited; between 1978 and 1982 inclusive both sexes were included.

Myocardial infarction was diagnosed when there was typical cardiac pain at rest with abnormal Q waves or a twofold or greater increase in serially measured serum glutamic oxaloacetic acid transaminase, creatine kinase, and lactate dehydrogenase, or both.

The site of infarction was classified as anteroseptal if electrocardiographic changes were seen in some or all of leads V1–V4, anterolateral if changes were in some or all of leads V3–V6, combined anteroseptal and anterolateral with changes in V1–V6, or inferior if the changes were noted in II, III, and aVF with or without changes in V4–V6.

In-hospital complications were recorded according to a standard protocol which defined a total of 34 complications. Hemiblocks and premature atrial or ventricular contractions were not rated as complications because these had not been recorded with sufficient detail in some of the earliest cases admitted to the study.

The presence of angina of effort before infarction was recorded as were smoking habits before infarction. Chronic angina was regarded as being present before infarction only if it started three months or more before. Patients were classified as non-smokers if they had never smoked as many as one cigarette per day regularly, ex-smokers if they had smoked less than one cigarette per day for at least three months before infarction, and current smokers if they had regularly smoked at least one cigarette per day in the three months before admission.

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Table 1 *Relation of infarction site to age and to size and severity of infarction*

	ASMI + ALMI	*ASMI*	*ALMI*	*IMI*	*Total*
Total cases	27 (4·0%)	237 (34·5%)	55 (8·1%)	367 (53·5%)	686
Mean (SD) age (yr)	51·74 (6·65)	50·84 (6·71)	53·76 (5·18)	50·87 (6·60)	51·04 (6·55)
Kruskall-Wallis ANOVA, Age by site: p = 0·274					
Peak increase in SGOT (Iu/l):					
Mean	182·0	184·1	137·8	168·4	171·5
SD	130·7	121·9	80·8	122·7	120·1
No of cases*	19	180	46	292	537
Kruskal-Wallis ANOVA, SGOT by site: p = 0·154					
Prevalence of complications (%)	59·3	41·7	36·4	35·9	38·9
χ^2 = 7·0, df = 3, p = 0·071.					

*149 cases missing.
Infarct site: ASMI, anteroseptal; ALMI, anterolateral; IMI, inferior.

Angina and smoking habits were recorded at two year follow up outpatient visit. Primary cigar and pipe smokers and cigarette smokers who changed to cigar or pipe smoking after infarction were classified as current smokers.

We recorded the date and mode of death for those who died and the date that survivors were last seen. Vascular deaths were subdivided into reinfarction with shock, congestive heart failure, or sudden death (death within one hour of onset of terminal symptoms). The date of occurrence of any further acute episodes of coronary heart disease was noted. Acute episodes were defined as either reinfarction, as defined above, or unstable angina characterised by typical cardiac pain at rest and confirmed by serial ST or T wave changes in the electrocardiogram but with no increase or a less than a twofold increase in the concentration of cardiac enzymes.

Categorical data were compared by the χ^2 test and differences in the distributions of metric variables between infarct topography groups were tested by the Kruskall-Wallis one-way analysis of variance by ranks. This latter test was chosen because both age and cardiac enzyme activities were not normally distributed. Life table methods were used to estimate survival functions and differences in survival experience between groups were tested by the logrank test. Because few patients were followed for more than 15 years, this study is confined to fifteen year life table morbidity and mortality rates.

Results

Six hundred and eighty six patients fulfilled the entry criteria. In two further cases left bundle branch block precluded identification of the infarction site and these patients were dropped from the analysis. Fifty five patients were women. Table 1 shows the relation between site and age, severity of infarction, and peak concentration of glutamic oxaloacetic acid transaminase. Patients with anterolateral infarctions showed the smallest rise in serial concentrations of glutamic oxaloacetic acid transaminase, and patients with other anterior infarctions showed the highest, but a one way analysis of variance did not reach statistical significance.

Table 2 *Relation of infarction site to smoking habits before and after infarction*

	ASMI + ALMI	*ASMI*	*ALMI*	*IMI*	*Total*
Preinfarction smoking habit:					
Never smoked	3 (11·1%)	23 (9·7%)	4 (7·3%)	37 (10·1%)	67 (9·8%)
Ex-smoker	5 (18·5%)	60 (25·3%)	13 (23·6%)	70 (19·1%)	148 (21·6%)
Current smoker	19 (70·4%)	154 (65·0%)	38 (69·1%)	260 (70·8%)	471 (68·7%)
		χ^2 = 4·0, df = 63, p = 0·686			
Smoking at 2 year follow up:					
Never smoked	1 (4·3%)	18 (9·0%)	3 (5·9%)	34 (10·9%)	56 (10·9%)
Ex-smoker	4 (17·4%)	46 (23·1%)	11 (21·6%)	56 (17·9%)	117 (20·0%)
Current stopped	11 (47·8%)	80 (40·2%)	20 (39·2%)	123 (39·3%)	234 (39·9%)
Current, smoking	6 (26·1%)	53 (26·6%)	16 (31·4%)	99 (31·6%)	174 (29·7%)
Ex, restarted	1 (4·3%)	2 (1·0%)	1 (2·0%)	1 (0·3%)	5 (0·9%)
Missing:					
Dead	3	22	3	26	54
Lost to follow up	0	11	1	15	27
No 2 yr follow up	2	7	1	12	22
		χ^2* = 5·37, df = 9, p = 0·801			

*Excludes ex-smokers who restarted (n = 5)
Infarct site: ASMI, anteroseptal; ALMI, anterolateral; IMI, inferior.

Table 3 *Relation of infarction site to pre-infarction and post-infarction angina*

	ASMI + ALMI	ASMI	ALMI	IMI	Total
Total cases	27 (4·0%)	237 (34·5%)	55 (8·1%)	367 (53·5%)	686
Preinfarction angina:					
Present (%)	18·5	18·4	22·2	18·8	18·9
Resolved 2 yr (%)	50·0	51·4	70·0	50·0	52·3
Absent (%)	81·5	80·3	77·8	80·9	21·7
Appeared 2 yr (%)	25·0	25·9	12·5	20·3	21·7
Relation of site to:					
Pre-infarction angina	p = 0·766 (NS)				
Post-infarction angina	p = 0·850 (NS)				
Change in angina	p = 0·764 (NS)				

Details of information on 2 year follow up missing from table 2. Infarct site: ASMI, anteroseptal; ALMI, anterolateral; IMI, inferior.

One or more complications were present in 38·9% of patients. There were complications in 59·3% of patients with infarctions extending through both anterior sites but the overall χ^2 value was of borderline significance (p = 0·071). Mechanical complications—left ventricular failure, cardiogenic shock and hypotension—showed a significant relation to site of infarction (p < 0·001); they were most common in combined anteroseptal and anterolateral infarcts (37·0%) and anteroseptal infarcts (19·0%), while anterolateral and inferior infarcts had similar rates (10·9% and 11·5% respectively). Atrioventricular blocks were significantly related to site, with 42 of 45 cases occurring in inferior infarction (p < 0·001). No other complications showed a significant relation to infarct site. The site of the infarct was not related to age.

Because of the influence of subsequent smoking habits on prognosis after myocardial infarction[8] and of the influence of pre-infarction or post-infarction angina on subsequent clinical course,[5] we examined the relation of these factors to infarct site. There was no association between site and either smoking habits on admission or change in smoking habits over two year follow up (table 2).

Similarly, there was no association between site and either pre-infarction angina or change in anginal symptoms over two year follow up (table 3). Prognosis was assessed by life table methods over a fifteen year period. There were 340 patients available for calculation of five year event rates, 211 at ten years, and 60 at 15 years.

Table 4 shows the life table rates for 15 year endpoints in the study group as a whole and within the subgroups for infarct site. Patients with combined anteroseptal and anterolateral infarctions were excluded from morbidity and mortality calculations because there were so few. Life table mortality was 61·4% at fifteen years, with a 33·1% rate of non-fatal acute episodes of coronary heart disease. At fifteen year follow up coronary heart disease mortality was 56·6%. There was no difference in event rates between the groups with different infarct sites. Several further analyses were carried out to examine differences in prognosis in relation to site of infarction. A cause specific mortality analysis was performed to test for higher rates of sudden death or of fatal reinfarction with other causes controlled. Neither sudden death nor fatal reinfarction was significantly more common in any topographical group. Among patients experiencing a subsequent episode of coronary heart disease, there was no association between site and type of episode (infarction or unstable angina). We also examined mortality and morbidity in the first two years after discharge to determine whether early prognosis was related to site. Once again, no significant relation emerged between site and event rates.

Discussion

Long term prognosis after myocardial infarction has been related to several variables, including age,[1 7 10] sex,[1 10] hypertension,[7] smoking,[8] diabetes,[7] previous

Table 4 *Relation of site to 15 year prognosis (%)*

	ASMI	ALMI	IMI	Total	p
15 year mortality	60·7 (5·1)	68·1 (11·4)	60·3 (4·2)	61·4 (3·0)	0·739
15 year non-fatal acute CHD	37·7 (5·1)	28·4 (9·2)	31·3 (3·9)	33·1 (2·8)	0·509
15 year fatal CHD	59·4 (5·9)	59·2 (14·1)	54·6 (4·7)	56·6 (3·6)	0·492
15 year sudden death	36·6 (5·9)	45·0 (17·8)	31·1 (4·3)	34·1 (3·42)	0·405

Results are mean (SEM). Infarct site: ASMI, anteroseptal; ALMI, anterolateral; IMI, inferior. CHD, coronary heart disease.

history of acute coronary heart disease,[4 5 7] pre-infarction angina,[5 7] infarct size,[1 7 9] cardiomegaly,[10] cardiac failure at the time of infarction,[1 4 5] ejection fraction,[5 9] and arrhythmias, both atrial[7] and ventricular,[4 5 7] and the extent of coronary artery disease.[5]

Although site of infarction seems to be important in determining in-hospital prognosis,[2] even allowing for the larger size of anterior infarction,[3] its effect on long term prognosis is less clear.[4-7] In part this disparity may result from the inclusion in other studies of patients with previous infarction[4] and those who did not survive the in-hospital phase of their illness. To our knowledge, ours is the longest follow up study of the relation of infarct site to subsequent outcome. It is, furthermore, a useful study of the natural course of the disease, since few of the patients underwent coronary artery bypass surgery or routine treatment with β blockade after infarction.

Risk factors of patients with anterior and inferior infarctions were identical, which is consistent with the findings of Taylor *et al*, who noted the same extent of coronary disease in patients with myocardial infarction irrespective of the site of the infarct.[9] Necrosis of the left ventricular muscle rather than the thinner right ventricular muscle and complications such as acute regional dilatation, left ventricular aneurysm, and myocardial rupture seem to be more common with anterior infarction than inferior infarction. This may account for the larger enzymatically estimated infarct size in anterior infarction reported by some[3] and for the higher in-hospital mortality of patients with anterior infarction. Even among 28-day survivors, patients with anterior infarctions had higher rates of mechanical complications.

Furthermore, we were unable to show a difference in the pattern of subsequent episodes of acute coronary heart disease for up to 15 years after myocardial infarction that was related to site of infarction. Not only were total mortality rates the same, but the subsequent prevalence of angina and the incidence of both fatal and non-fatal episodes of acute coronary heart disease were also similar. Sudden death rates were also no different.

Although a difference in mortality might be expected on the basis of differing infarct sizes, and could be explained by more left ventricular damage in anterior infarction, once healing has taken place and the groups with a poorer prognosis have been removed by their higher early death rate, infarct site seems unimportant in determining outcome after a first episode of acute coronary heart disease.

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