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Determinants of delay between symptoms and hospital admission in 5978 patients with acute myocardial infarction

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The aim of this study was to analyse the influence of patient characteristics on delay between onset of symptoms and hospital admission (patient delay) in acute myocardial infarction, and especially to assess the impact of risk factors for acute myocardial infarction on patient delay.

A group of 6676 consecutive patients with enzyme-confirmed acute myocardial infarction, admitted alive to 27 Danish hospitals over a 26 month period from 1990 to 1992, were studied. Due to missing information on delay or in-hospital acute myocardial infarction 698 patients were excluded, leaving 5978 patients for analysis.

Mean patient delay was 9·1 h, median delay 3·25 h (5 to 95 percentiles: 0·67–40·0 h). Thirty-four percent were admitted within the first 2 h, 68% within 6 h and 81% within 12 h of onset of symptoms.

In multivariate logistic regression analysis, a greater than 2 h patient delay was independently associated with male gender (odds ratio (OR)=0·809, P=0·003), increased age (P=0·0001), diabetes mellitus (OR=1·269, P=0·03), left ventricular systolic function (wall motion index) (P=0·02), onset from midnight to 0600h (OR=1·434, P=0·0001), onset on a weekday (OR=0·862, P=0·04), history of angina pectoris (OR=1·198, P=0·02), chest pain as initial symptom (OR=1·293, P=0·02), ventricular fibrillation (OR=0·562, P=0·0001), ventricular tachycardia (OR=0·620, P=0·0001), Killip class ≥3 (OR=0·709 P=0·002), presence of ST elevation (OR=0·810, P=0·01) and ST depressions (OR=0·847, P=0·01). All these variables, except history of diabetes mellitus, angina pectoris, and chest pain as an initial symptom were also associated with a delay of more than 6 h.

Thrombolytic therapy was administered to 55·8% of patients admitted within 2 h of an acute myocardial infarction, 48·5% of patients admitted within 2–6 h, 31·5% of patients admitted after 6–12 h and 11·9% of patients arriving later than 12 h after start of symptoms.

Conclusion Patient delay continues to be disappointingly long. This also applies for patients at a high risk of acute myocardial infarction (notably those with a history of diabetes mellitus and angina pectoris).
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Key Words: Acute myocardial infarction, risk factors, delay, thrombolytic therapy.

Introduction

In recent decades, there has been a big drive to improve prognosis following an acute myocardial infarction, and it has been shown that in-hospital short- and long-term survival post-acute myocardial infarction can be significantly improved by early treatment with aspirin, β-blockers, thrombolysis and angiotensin-converting-enzyme inhibitors[1–3]. The optimal effect is achieved when the therapy is initiated as soon after onset of symptoms as possible[4–6]. Patient delay is defined as time from onset of symptoms until admission[7,8], but current strategies, focusing on early intervention in patients with acute myocardial infarction, have not been accompanied by a reduction in patient delay[9].

Public educational programmes aiming to reduce patient delay have had variable degrees of success[10–13], and in planning future information and intervention

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campaigns, it is important to assess the factors that significantly influence delay. It is especially important to identify patients at high risk of prolonged delay.

The intention of the current study was to identify risk factors of prolonged patient delay and to assess the impact of such risk factors in acute myocardial infarction on the magnitude of patient delay in a large cohort of consecutive acute myocardial infarction patients admitted to 27 Danish hospitals.

Methods

Patients

The study population consisted of 6676 consecutive patients, all admitted alive with an acute myocardial infarction. The definition of an acute myocardial infarction was presence of chest pain or electrocardiographic changes suggestive of infarction or ischaemia, and accompanied by an increase in cardiac enzymes to twice the upper normal value of that of the local hospital laboratory. Since the purpose of this study was to assess the importance of patient delay in patients outside hospital, infarctions occurring in hospital and infarctions where information concerning patient delay was not available were excluded.

Information was collected prospectively for the TRAndolapril Cardiac Evaluation study (TRACE), which was designed to evaluate the effect of trandolapril on mortality in patients with moderate to severe reduced left ventricular systolic function after acute myocardial infarction[14]. The TRACE study was approved by the scientific ethics committee, and the study population comprised those admitted to 27 Danish centres (seven university, seven county, and 13 community hospitals, all having complete regional uptake) from May 1990 until July 1992.

All patients were screened 1–6 days after an acute myocardial infarction and all were aged over 18 years; 6637 were caucasians, six were blacks, 19 were orientals, five were of other racial extraction, and in nine their origin was unknown. At screening, a medical history was obtained, and an echocardiogram was recorded on videotape for evaluation of left ventricular systolic function, estimated as wall motion index. In-hospital complications were registered.

Patient delay

Patient delay was the interval between 'time of initial symptoms' until 'time of arrival in hospital'. Both time points were directly recorded by the investigators.

Patients were divided into three groups: group 1, delay ≤ 2 h; group 2, delay >2 h but ≤ 6 h; and group 3, delay >6 h. This subgrouping was based on reports which showed that a delay of less than 2 h, particularly in connection with thrombolytic therapy, influenced prognosis post-AMI[15,16]. At the time of the screening period, 6 h was generally accepted as the limit for administration of routine thrombolysis, although some centres administrated thrombolysis up to 24 h after onset of symptoms. The three groups were compared for clinical characteristics and short-term prognosis.

Mortality

Mortality data on 6673 patients were obtained after 24–50 months of observation on 15 July 1994, but there was no survival information for 28 non-Danish patients after discharge from hospital.

Statistical methods

Comparison of the three groups in relation to baseline characteristics, treatment and 6-day survival were made by chi-square tests. All tests of statistical significance were two-tailed, and P values of less than 0·05 were considered significant. A logistic multivariate regression analysis was used to examine the association of various patient-related factors with the extent of delay, while controlling for several potentially confounding factors. Two groups of analyses were performed: an analysis of factors in which a delay of more than 2 h was important, as compared to less than 2 h, and of more than 6 h as compared less than 6 h. All analyses were performed with SAS statistical package programs (SAS Institute, Cary, NC, U.S.A.).

Results

The characteristics of the 5978 patients with known delay, and the 698 patients excluded due to missing information concerning delay or in-hospital acute myocardial infarction, are listed in Table 1. Excluded patients were more frequently women, non-smokers, older than 65 years of age, and with an increased frequency of a history of hypertension, diabetes mellitus, angina pectoris, congestive heart failure, prior acute myocardial infarction and less often wall motion index $\geq 1{\cdot}3$, inferior Q-wave infarction, ventricular tachycardia/fibrillation, electrocardiographic ST-elevations and chest pain as an initial symptom. Excluded patients received thrombolytic therapy and aspirin less often and had a higher mortality in the first 6 days after the acute myocardial infarction.

The distribution of patient delay is shown in Fig. 1. Thirty-four percent of the patients were admitted within the first 2 h, 68% within the first 6 h and 81% within 12 h of onset of symptoms. The mean patient delay was 9·1 h, median 3·25 h, 5 to 95 percentiles (0·67–40)h.

Univariate analysis

Baseline characteristics associated with the extent of patient delay (Table 2) shows that women and patients

Table 1 Characteristics of included patients compared to excluded patients

Characteristics	Included patients (%) (n=5978)	Excluded patients (%) (n=698)	*P* value	
Male gender	67·9	63·3	0·013	
Age (years)				
<45	3·7	2·9	ns	
45, 1–55	12·3	7·2	<0·0001	
55, 1–65	23·3	22·1	ns	<0·01
65, 1–75	33·0	37·5	0·02	
>75	27·6	30·4	ns	
History				
Hypertension	22·2	26·1	0·02	
Diabetes mellitus	10·2	15·6	<0·001	
Prior AMI	22·7	28·9	<0·001	
Angina pectoris	36 0	44·0	<0·001	
Congestive heart failure	15·9	24·4	<0·001	
Smoker	52·4	43·8	<0·001	
Former smoker	21·7	23·8	ns	<0·001
Non smoker	25·9	32·4	<0·001	
BMI $\geq 27{\cdot}5\ kg \cdot m^{-2}$	27·1	26·9	ns	
Hyperlipidaemia	5·0	5·8	ns	
Symptoms				
Chest pain	89 6	79·1	<0·001	
Dyspnoea	25·9	27·9	ns	
VT	13·0	9·9	0·02	
VF	7·0	8·3	ns	
3rd degree AV block	4 1	4·6	ns	
Killip class ≥3	10·3	14·3	0·001	
ECG changes				
No changes	12·5	12·5	ns	
Non Q-wave	19·8	22 7	ns	
Anterior Q-wave	26·5	24·4	ns	<0·001
Inferior Q-wave	31·4	27 0	0·01	
Other, mixed	9·4	11·8	0·03	
Unknown	0·5	1·6	<0·001	
ST elevations	69·1	61·0	<0·001	
ST depressions	59·5	62·0	ns	
Signs of AMI	87·4	87·3	ns	
Q-wave infarctions	67·6	64·2	ns	
Wall motion index				
Not available	6·2	10·9	<0·001	
<0·8	4·7	7·6	0·001	
0·8–1·2	31·8	34·1	ns	<0 001
1·3–1·6	24·0	19·9	0·02	
>1·6	33·3	27·5	0·002	
Onset time				
0000 h–0600 h	21·0	—	—	
0600 h–1200 h	29·8	—	—	
1200 h–1800 h	25·2	—	—	
1800 h–2400 h	24·0	—	—	
Weekend	26·4	26·3	ns	
Treatment				
Thrombolysis	42·1	30·0	<0·001	
Aspirin	70·2	59·2	<0·001	
Vital status				
Alive on day 6	94·5	91·1	<0·001	

BMI=body mass index; AMI=acute myocardial infarction; VT=ventricular tachycardia; VF=ventricular fibrillation; AV=atrioventricular; ECG=electrocardiogram; ns=not significant; —=missing data.

older than 75 years, or patients with a history of diabetes mellitus, angina pectoris, congestive heart failure, chest pain and 'no changes' in the ECG were likely to experience a delay in their admission to hospital of more than 2 h after onset of symptoms. Patients with known hyperlipidaemia on admission were more frequently admitted within 2 h of onset, compared to more than 6 h. Patients with their first acute myocardial infarction, or onset of symptoms on a weekday were more frequently associated with a delay of more than 6 h

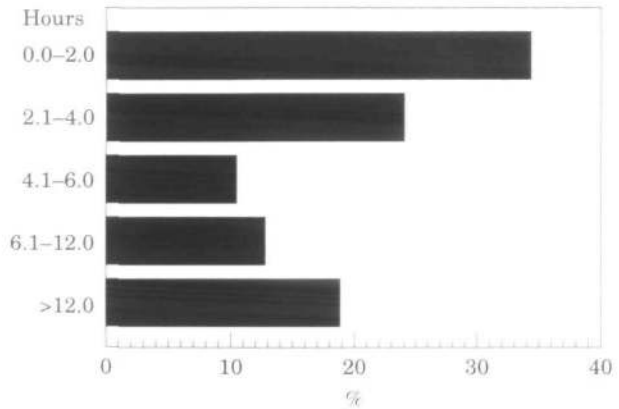

Figure 1 **Distribution of delay between onset of symptoms and admission to hospital in 5978 patients with enzyme confirmed AMI.**

compared to patients admitted during a weekend or with history of prior acute myocardial infarction. A delay less than 6 h was associated with smoking and inferior Q-wave infarctions. Aspirin treatment was less frequent in the group with a delay exceeding 6 h. Patient delay and ventricular fibrillation/tachycardia was inversely related (i.e. a shorter delay meant a higher frequency of malignant ventricular arrhythmias). The same relationship was seen with respect to 3rd-degree atrioventricular block, but was less prominent. The presence of ST-elevations, ST-depressions and Killip class ≥ 3 was associated with short delay. Not surprisingly, there was a strong inverse association between the frequency of prescribing thrombolysis and delay. Onset of symptoms from midnight to 0600h was associated with prolonged delay, whereas onset of symptoms from 0600h to noon was associated with short delay. Table 3 lists the median delay in subgroups of patients with well-known risk factors for acute myocardial infarction, and shows that age over 65 years, diabetes mellitus, angina pectoris and hyperlipidaemia are associated with prolonged patient delay, as much as 50 min for some of the risk factors.

Multivariate analysis

Table 4 specifies the risk of delays >2 h and >6 h when analysed by logistic regression analysis. Factors included in this analysis were those which had significant influences on delay in univariate analysis. A delay of more than 2 h showed a significant association with gender, increased age, history of diabetes mellitus, angina pectoris, initial acute myocardial infarction and chest pain as an initial symptom. Absence of ventricular tachycardia/fibrillation, ST-elevations and ST-depressions were associated with a delay of more than 2 h, as were declining wall motion index, Killip class <3, onset at night and during weekdays. A delay of more than 6 h shared characteristics similar to those when the delay was more than 2 h, except for angina pectoris, and chest pain, which were non-significant when the delay was more than 6 h. Increased maximal creatine kinase B was associated with a less than 6 h delay.

Table 5 specifies the variables expected to be known by the patient at the time of the infarction. A delay of more than 2 h showed a significant independent association with female gender, advanced age, history of diabetes mellitus, angina pectoris, chest pain, initial acute myocardial infarction, onset of symptoms at night and during weekdays. A delay of more than 6 h was associated with the same characteristics as the more than 2 h delay except for history of angina pectoris and presence of chest pain, which were non-significant at the more than 6 h delay.

Thrombolytic treatment was administered to 40·3% of the whole screened population. Admission to hospital within 2 h resulted in 55·8% of patients being given thrombolysis, from 2–6 h 48·5% were treated, from 6–12 h 31·5%, and after 12 h 11·9% received thrombolysis.

Table 6 specifies patient delay according to mortality at 6 days, 1 year, 2 years and 3 years, regardless of treatment and characteristics. Significantly longer delays were experienced in the group of patients who died 1 to 3 years post-acute myocardial infarction.

Discussion

The study examined 6676 consecutive acute myocardial infarction patients, which represented 20–25% of all patients hospitalized with an acute myocardial infarction in Denmark in that 2-year period and was therefore representative of the Danish acute myocardial infarction population as a whole. Distance to the nearest hospital is very short, and an emergency call for an ambulance will results in admission to an emergency room within a maximum of 45 min from all locations in Denmark. Several large studies have shown that the interval between onset of symptoms and administration of thrombolysis and β-blocking agents is extremely important for successful coronary artery reperfusion, subsequent improved left ventricular function and survival post-acute myocardial infarction[2–6,17–21].

Patients in our study tended to arrive late with a median patient delay of 3·25 h. Thus, in seeking medical attention, delay by the patient, the referring physician or general practitioner remains the major and most crucial component of delay in treating acute myocardial infarction. One important finding was that patients with certain well-known risk factors of acute myocardial infarction tend to have prolonged delay; i.e advanced age, history of diabetes mellitus and angina pectoris, while other known risk factors tend to make the patient arrive faster; i.e. being a male, and having experienced a prior acute myocardial infarction, whereas hypertension had no effect on patient delay. Time of onset of symptoms was found to have significant influence with increased delay associated with weekdays and nights.

Increased delay was not associated with early death, although long-term mortality was significantly

Table 2 Characteristics among those who experienced delay after onset of acute symptoms until admission to hospital in patients with acute myocardial infarction

Delay Characteristics	Group 1 0–2 h (%) (n=2039)	Group 2 2·1–6 h (%) (n=2049)	Group 3 >6 h (%) (n=1890)	*P* value	
Male gender	72·7	66·9	64·0	<0·001	
Age (years)					
<45	4·6	3·7	2·8	0·01	<0·0001
45, 1–55	15·0	10·9	11·0	<0·0001	
55, 1–65	25·9	22·1	21·6	<0·1	
65, 1–75	31·4	34·5	33·2	ns	
>75	23·1	28·8	31·4	<0·0001	
History					
Hypertension	21·5	23·0	22·1	ns	
Diabetes mellitus	8·4	10·8	11·6	<0·0003	
Prior AMI	23·0	24·3	20·7	0·03	
Angina pectoris	32·3	38·8	36·9	<0·001	
Congestive heart failure	13·8	18·0	15·9	<0·001	
Smoker	53·8	53·4	49·9	0·03	0·02
Former smoker	21·7	22·0	21·3	ns	
Non smoker	24·5	24·7	28·8	<0·01	
BMI $\geq 27{\cdot}5\ kg\ .\ m^{-2}$	27·2	27·5	26·6	ns	
Hyperlipidaemia	5·6	5·1	4·1	ns	
Symptoms					
Chest pain	87·6	91·3	89·7	<0·001	
Dyspnoea	26·0	26·6	25·1	ns	
VT	17·2	13·0	8·4	<0·001	
VF	10·0	5·9	5·1	<0·001	
3rd degree AV block	5·5	3·8	3·0	<0·01	
Killip class ≥3	11·5	10·0	9·1	0·04	
ECG changes					
No changes	11·1	12·9	13·6	ns	0·02
Non Q-wave	19·7	19·7	19·9	ns	
Anterior Q-wave	27·1	25·2	27·2	ns	
Inferior Q-wave	32·7	33·0	28·4	<0·01	
Other, mixed	9·2	8·8	10·2	ns	
Unknown	0·3	0·4	0·7	ns	
ST elevations	72·8	69 4	64·8	<0·001	
ST depressions	62·5	61·3	54·3	<0·001	
Signs of AMI	88·9	87·0	86·3	0·05	
Q-wave infarctions	69·1	67·2	66·3	ns	
Wall motion index					
Not available	6·0	6·6	5·9	ns	0·01
<0·8	4·0	5·3	4·9	ns	
0·8–1·2	31·6	29·1	35·0	<0·001	
1·3–1·6	24·4	24·7	22·8	ns	
>1·6	34·0	34·3	31·5	ns	
Onset time					
0000 h–0600 h	17·1	19·8	26·7	<0·001	0·001
0600 h–1200 h	31·4	32·6	25·0	<0·001	
1200 h–1800 h	27·4	24·1	23·9	0·02	
1800 h–2400 h	24·0	23·6	24·4	ns	
Weekend	28·2	27·5	23·1	<0·001	
Treatment					
Thrombolysis	55·8	48·5	19·8	<0·001	
Aspirin	73·8	72·6	63·7	<0·001	
Vital status					
Alive on day 6	95·2	93·9	94·3	ns	

BMI=body mass index; AMI=acute myocardial infarction; ECG=electrocardiogram; VT=ventricular tachycardia; VF=ventricular fibrillation; AV=atrioventricular; ns=not significant.

associated with increased patient delay (Table 6). The median delay of 3·25 h found in our study is comparable to others, who have reported median delays ranging from 1·7 to 8 h[7,9–11,13,16,22–37]. The great variation in patient delay is probably due to differences in the populations studied and in the registration of onset of

Table 3 The magnitude of patient delay, in hours, estimated by single risk factors for AMI

Parameter	Median patient delay (5 to 95 percentiles)		n	Median patient delay (5 to 95 percentiles)		n	*P* values
Male vs female gender	3	(0·58–36·3)	4061	3·75	(0·75–47·48)	1916	<0·001
Age; >65 vs <65 years	3·58	(0·75–42·33)	3628	2·75	(0·58–36·68)	2350	<0·001
AMI; prior vs initial	3	(0·58–39·25)	1353	3·33	(0·67–40)	4607	0·03
BMI ($kg \cdot m^{-2}$) >27·5 vs <27·5	3·20	(0·75–43·75)	1619	3·25	(0·67–38·50)	4359	0·79
Diabetes mellitus ±	4	(0·67–48)	610	3·17	(0·67–38·7)	5360	0·001
Hypertension ±	3·25	(0·67–40·5)	1325	3·33	(0·75–37·67)	4641	0·40
Angina pectoris ±	3·5	(0·75–34)	2149	3·08	(0·6–44·5)	3819	0·01
Hyperlipidaemia ±	3·25	(0·67–40·5)	295	3	(0·58–26·5)	5644	0·04
Congestive heart failure ±	3·42	(0·75–46)	947	3·25	(0·67–38·92)	5016	0·12
Smoker ±	3·08	(0·67–35·75)	3051	3 5	(0·67–44·67)	2771	0·01

vs=Versus; AMI=acute myocardial infarction; BMI=body mass index; n=number of patients; ± =present/absent.

Table 4 Multivariate adjusted likelihood of delaying admission to the hospital by >2 h and >6 h

Characteristics	Odds ratio	>2 h 5 to 95 percentiles	*P* values	Odds ratio	>6 h 5 to 95 percentiles	*P* values
Male gender	0·809	0·703–0·931	0·003	0·848	0·735–0·979	0·02
Age	1·018	1·011–1·024	0·0001	1·009	1·003–1·016	0·006
History						
Diabetes mellitus	1·269	1·018–1·584	0·03	1·165	0·938–1·447	ns
Prior AMI	0·779	0·658–0·922	0·004	0·702	0·586–0·839	0·0001
Angina pectoris	1·198	1·034–1·388	0·02	1·068	0·918–1·242	ns
congestive heart failure	1·037	0·849–1·268	ns	0·824	0·670–1·013	ns
Smoker	1 139	0·996–1·302	ns	0·940	0·818–1·081	ns
Symptoms						
Chest pain	1·293	1·044–1·600	0·02	1·086	0·860–1·371	ns
Ventricular fibrillation	0·562	0·428–0·739	0·0001	0·591	0·413–0·847	0·004
Ventricular tachycardia	0·620	0·512–0·751	0·0001	0·507	0·397–0·647	0·0001
3rd degree AV block	0·820	0·591–1·139	ns	1·011	0·692–1·476	ns
Killip class ≥3	0·709	0·567–0·886	0·002	0·638	0·496–0·820	0·0005
ECG changes						
ST elevations	0·810	0·690–0·951	0·01	0·827	0·702–0·975	0·02
ST depressions	0·847	0·725–0·963	0·01	0·753	0·659–0·860	0·0001
Q-wave AMI	1·032	0·883–1·205	ns	1·029	0·876–1·210	ns
Wall motion index	0·817	0·686–0·973	0·02	0·610	0·508–0·734	0·0001
Creatine kinase B	1·000	0·999–1·000	ns	0·997	0·996–0·998	0·0001
Onset time						
0000 h–0600 h	1·434	1·227–1·676	0·0001	1·693	1·456–1·969	0·0001
Weekend	0·862	0·750–0·991	0·04	0·799	0·687–0·930	0·004

Odds ratio of age is per increasing year, of wall motion index it is per increasing unit and of creatine kinase B it is per unit of maximal creatine kinase B; AMI=acute myocardial infarction; ns=non-significant; AV=atrioventricular; ECG=electrocardiogram.

symptoms. Criteria for acute myocardial infarction differ and some studies included patients admitted with a suspicion of acute myocardial infarction. The number of subjects was often relatively low; in 13 of 22 studies it was less than 250.

Our finding that delay is influenced by gender, age and diabetes mellitus is consistent with other studies[9,16,22,30], but in our population a first acute myocardial infarction, time of day, angina pectoris and chest pain as an initial symptom are also significantly associated with prolonged patient delay. The last two characteristics and diabetes mellitus were only associated with a more than 2 h delay, but not a more than 6 h delay. Severity of the infarction, expressed as Q-wave infarction, and 3rd-degree atrioventricular conduction block, did not seem to result in the earlier arrival of the patient at hospital, but Killip class ≥3, ST-elevations or ST-depressions were strongly associated with short delay. Hypotension and cardiogenic shock were found more often in patients arriving within 2 h of onset of symptoms, which is consistent with the findings of Maynard *et al.*[27]. In contrast to others[16,22,30] we found no association between patient delay and hypertension, or with a history of congestive heart failure. Prevalence of hypertension in our population is lower than reported in the above studies, and could in part explain the differing results with respect to hypertension. Smoking was found by Turi *et al.*[16] to be associated with shorter delay, whereas our data suggest no independent association between smoking and delay. An association

Table 5 Multivariate adjusted likelihood of delaying admission to hospital by >2 h and >6 h in relation to medical history and chest pain

Characteristics	Odds ratio	>2 h 5 to 95 percentiles	*P* value	Odds ratio	>6 h 5 to 95 percentiles	*P* value
Male gender	0·798	0·700–0·900	0·0008	0·824	0·728–0·931	0·002
Age	1·018	1·013–1·024	0·0001	1·013	1·007–1·019	0·0001
History						
Diabetes mellitus	1·313	1·080–1·596	0·006	1·250	1·040–1·503	0·02
Prior AMI	0·845	0·728–0·980	0·03	0·791	0·679–0·921	0·003
Angina pectoris	1·198	1·050–1·366	0·007	1·066	0·935–1·214	ns
Hypertension	0·998	0·867–1·138	ns	0·928	0·809–1·065	ns
Congestive heart failure	1·089	0·915–1·297	ns	0·881	0·742–1·047	ns
Smoker	1·118	0·991–1·261	ns	0·950	0·841–1·073	ns
Symptoms						
Chest pain	1·380	1·150–1·656	0·0005	1·030	0·852–1·245	ns
Onset time						
0000 h–0600 h	1·469	1·275–1·693	0·0001	1·652	1·446–1·886	0·0001
Weekend	0·854	0·754–0·967	0·01	0·770	0·676–0·878	0·0001

Odds ratio of age is per increasing year; AMI=acute myocardial infarction; ns=non-significant.

Table 6 Delay in patients alive or death at 6 days, 1 year, 2 years and 3 years

		Number (patients)	Mean delay (h)	Median delay (5 to 95 percentiles) (h)	*P* value
	Alive	5646	9·16	3 25 (0·67–41·67)	
6 days	Dead	328	8·12	3·51 (0·83–34·42)	0·09
	Excluded	4			
	Alive	4628	8·47	3·00 (0·67–36·50)	
1 year	Dead	1327	11·38	4·00 (0·75–49 00)	<0·001
	Excluded	23			
	Alive	4250	8·42	3·00 (0·67–35·50)	
2 years	Dead	1705	10·84	4·00 (0·75–48·50)	<0·001
	Excluded	23			
	Alive	2037	8·95	3·00 (0·67–39·00)	
3 years	Dead	1983	10·60	3·83 (0·75–48·50)	<0·001
	Excluded	1958			

between diabetes mellitus and silent myocardial infarction is well-known[8], but whether diabetes mellitus is associated with weaker symptoms or a slower onset of symptoms is unknown. Such an association would explain our finding of longer delay in patients having diabetes mellitus. Angina pectoris was only associated with a more than 2 h delay but not with a more than 6 h delay, which may be due to the initial difficulty in discriminating between pain of severe angina pectoris or acute myocardial infarction. Our finding that delay is longest on weekdays is in contrast to the findings of Tjoe and Luria[24]. Trent *et al.*[38] found a correlation between reduced left ventricular function (expressed as left ventricular stroke distance on admission), and short delay, but we found no such correlation. Importantly, Trent *et al.* estimated stroke distance immediately after arrival of the patient, whereas wall motion index was estimated 1–6 days after the infarction. The association of Killip class ≥ 3 with short delay supports the possibility that the severity of the acute myocardial infarction tends to shorten delay[38].

The distribution of utilization of thrombolysis in our study is in agreement with data from the National Registry of Myocardial Infarction, which registered demographic, procedural, and outcome data from a large cohort of patients with acute myocardial infarction in the United States from 1990 to 1993[37]. Not surprisingly, patients presenting for medical aid soon after initial symptoms are significantly more likely to receive thrombolysis. In a study by Goldberg *et al.*[39] the corresponding figures are: delay <2 h 27·3%, from 2–6 h 17·1% and more than 6 h 4·9%. The finding of Moss *et al.*[22] that delay is longest during daytime hours, cannot be confirmed by our study, which showed that delay is significantly longer from midnight to 0600h. Increased maximal creatine kinase B shows an association with a delay of less than 6 h. This is in agreement with an earlier study by Rawles *et al.*[40], which showed an inverse association between patient delay and maximal serum aspartate aminotransferase.

Previous studies of broad educational campaigns have focused on the urgency of prompt admission, with

diverging results[7,11–13]. Ridker *et al.*[29] found that patients who were very well informed had a significantly shorter delay. The results of our investigation may help in directing attention to those patients with the highest risk of excessive delay.

An important result of this investigation is the discovery that large groups of patients who experience excessive delay are already in contact with the medical profession; i.e. patients with diabetes mellitus or angina pectoris, and to some extent people older than 65 years. Such patients need to receive periodic instruction regarding the possible meaning of a change in symptoms, in particular of chest pain at rest, and other symptoms of acute myocardial infarction. Patients with angina should be encouraged to seek medical aid promptly (i.e. within 15 min) if sublingual nitrates fail to give rapid relief of their chest pain. Periodic instruction on how to obtain immediate emergency care both day and night is needed. At discharge from hospital a formal teaching programme may be useful. An educational drive aimed at these easily identifiable groups should be rewarding.

Limitations of the study

We were able to study total patient delay, but not several important components: decision time, time that could not be unaccounted for and transportation time[22]. Another limitation of the study is that patients who died out of hospital or those dying without verified elevated coronary enzymes were not included.

Conclusion

This study of a large proportion of Danish patients admitted alive with an acute myocardial infarction demonstrates that patient delay in Denmark is disappointingly long despite an efficient transport system. Most importantly, critical patient groups already in contact with the medical profession and who are promising targets for an educational drive have a relatively increased delay.

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