High mortality rates in conservatively managed patients with acute coronary syndrome

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Abstract

Objectives. The revised diagnostic criteria for the acute coronary syndrome (ACS) have created the need for accurate and representative data on treatment and outcome for the three categories of ACS. Design. Consecutive patients admitted with a suspected ACS (n = 755) from February 1, 2003 to January 31, 2004 was registered and categorised into five diagnostic groups: 1) ST-elevation myocardial infarction (STEMI) (n = 126), 2) Non-ST-elevation myocardial infarction (NSTEMI) (n = 185), 3) Unstable angina pectoris (UAP) (n = 55), 4) Coronary heart disease (CHD) without ACS (n = 164) and 5) Non-coronary chest pain (n = 225). Results. All-cause one-year mortality rates were 20%, 32%, 7%, 10% and 3%, in patients with STEMI, NSTEMI, UAP, CHD without ACS and non-coronary chest pain, respectively. In patients with STEMI, 61% received immediate reperfusion therapy (ratio thrombolysis: primary PCI = 18:1). Only 3% of those with NSTEMI had PCI within two days. Conclusion. In this conservatively managed population of consecutive patients with ACS, the one-year mortality rate is significantly higher than seen in most registries and clinical trials.

Key words: Acute coronary syndrome, cohort studies, coronary disease, mortality, myocardial infarction, prognosis

The introduction of new diagnostic criteria for the acute coronary syndrome (ACS) in year 2000 has created a need for studies on prognosis according to the three categories of ACS: ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI) and unstable angina pectoris (UAP) (1). Importantly, studies in the “real world” may differ from those in clinical trial settings. The current criteria for acute myocardial infarction (AMI) are based upon a significant rise of cardiac markers in serial blood samples (1). Patients admitted with cardiac arrest and ischemic ECG changes will not necessarily qualify for the AMI diagnosis if not successfully resuscitated.

According to European guidelines patients with NSTEMI and UAP should be stabilised medically and evaluated for an interventional strategy within 48 hours, or at least during index hospitalisation (2). Patients with STEMI should be treated promptly with medical or mechanical reperfusion. Primary percutaneous coronary intervention (PCI) is the preferred treatment, given an experienced team can provide this procedure within 90 min after first medical contact (3). However, there is still a matter of discussion whether prompt thrombolysis may be of similar value in hospitals without PCI-facilities, since the documentation for superiority of transportation to such a laboratory is only based upon a meta-analysis (4). At present, there is apparently also an under use of drugs with prevention effects following ACS (5). Thus, both acute and long-term treatment will influence the prognosis of the three categories of ACS.

The aim of this prospective study was, in an unselected series of patients admitted to one hospital without PCI-facilities, to obtain mortality data from patients with a confirmed diagnosis of ACS subdivided into the three categories in relation to all
treatments provided in the acute phase and throughout one year. In addition, we aimed to find one-year mortality data for patients with or without established coronary heart disease who did not fulfil the criteria for ACS.

Patients and methods

During a one-year-period from February 1, 2003 to January 31, 2004 all patients with chest pain and a suspected ACS admitted to our hospital, were prospectively registered. The catchment population was 126,000 inhabitants. Our hospital is not a participant of the GRACE registry.

Patients were categorised into five groups: 1) STEMI, 2) NSTEMI, 3) UAP, 4) coronary heart disease (CHD) without ACS and 5) Non-coronary chest pain. STEMI, NSTEMI and UAP were defined according to ESC/ACC-consensus (1). Patients were diagnosed with AMI if presenting with typical symptoms and positive cardiac markers (troponin T \( \geq 0.1 \mu g/L \) or creatine kinase-myocardial band [CKMB] \( \leq 10 \mu g/L \)). The subtype of AMI was classified due to ECG findings. STEMI was present if persistent ST-segment elevation occurred in two adjacent leads (\( > 0.1 \) mV in limb leads, \( > 0.2 \) mV in V1–V3 and \( > 0.1 \) mV in V4–V6). Patients qualifying for AMI presenting without persistent ST-segment elevations were classified as NSTEMI. In the presence of left bundle branch block (LBBB), patients were categorised as STEMI if LBBB was presumed to be of recent onset, otherwise as NSTEMI. The diagnosis of UAP was based upon the clinical syndrome and ST-segment deviation or T-wave inversion on the resting ECG but negative cardiac markers, as applied in the CURE study (6).

According to Norwegian consensus (7), negative cardiac markers in this respect, were compatible with a max troponin T \( < 0.10 \mu g/l \), and no attempts were made to subclassify UAP patients with troponin T \( < 0.03 \) vs. \( 0.03 - 0.09 \mu g/l \). All the ECGs were reviewed by an experienced physician (JEO) who also established the diagnosis, accordingly. 12-lead ECG was obtained for all patients at baseline (admission). In addition, continuous vector-ECG (MIDA) was used to monitor patients with established, or a strong suspicion of ACS at the coronary care unit.

The new diagnostic criteria for NSTEMI and STEMI require blood samples discriminating a rise in serum troponin T to the cut-off value of 0.1 \( \mu g/L \). Therefore, high-risk patients with cardiac arrest on admission who were not successfully resuscitated were excluded even if the ECG was indicative of, but not necessarily compatible with an AMI.

Patients with established coronary artery disease who did not fall into groups 1 – 3 were classified as CHD without ACS. Established coronary artery disease was defined as documented prior AMI, positive stress test, angiographic findings or prior PCI/coronary artery bypass grafting (CABG). Patients, who did not meet the criteria of groups 1 – 4, were categorised into group 5.

Baseline characteristics including co-morbidity were recorded during index hospitalisation. Medical treatment was registered at admission, during hospitalisation and at discharge. All referrals to coronary angiography were registered and discharge summaries from catheterisation laboratory collected. The proportion of patients who underwent revascularisation (PCI or CABG) was recorded.

Information about time of death was collected from a national registry (EDB Infobank). We have confirmed vital status for all patients admitted, regardless of follow-up status. Due to regulatory restrictions, cause of death was not available.

All patients with a confirmed diagnosis of ACS were asked to participate in the one-year follow-up study. Based on written consent, they were scheduled for clinic visits six and 12 months after the qualifying ACS.

The study was approved by the local ethics committee.

Statistical methods

Mann-Whitney test (non-parametric) was used for comparison of continuous data between different groups of patients. Proportions were analysed by \( \chi^2 \) test or Fischer’s exact test as appropriate. Two-tailed p-values below 0.05 were considered significant. Kaplan-Meier plots and Cox proportional hazards regression models were used to analyse the effects of STEMI, NSTEMI, UAP, CHD without ACS and non-coronary chest pain (reference variable) on patient survival. Diagnostic category was included as the first variable in the regression analysis; non-coronary chest pain \( = 0 \), CHD without ACS \( = 1 \), UAP \( = 2 \), STEMI \( = 3 \) and NSTEMI \( = 4 \), with non-coronary chest pain as the reference group. Variables associated with patient survival in the univariate analyses (\( p < 0.20 \)) were included in a multiple Cox regression analysis. The analyses were implemented using SPSS 12.0 (SPSS, Chicago, IL).

Results

Incidence figures and diagnostic categories

During the one-year period a total of 755 patients were admitted to our hospital with chest pain and managed for suspected ACS. Based upon ECG and
cardiac markers, 366 patients (48%) had an ACS, 164 (22%) were categorised as CHD without ACS and 225 (30%) as non-coronary chest pain (Figure 1).

Among patients with ACS, 51% had NSTEMI, 34% STEMI and 15% UAP. Clinical characteristics of patients in all five groups are shown in Table I. Patients with NSTEMI were older (p < 0.001), and had a higher prevalence of prior AMI (p < 0.001) as compared with STEMI patients. Patients with CHD without ACS had a higher frequency of cardiovascular premorbidity as compared with UAP patients.

Patients without evidence of CHD (group 5) were younger (Median age 60 vs. 72 years, p < 0.001), included more women (49% vs. 37%, p = 0.002) than those with CHD, and had a significantly lower prevalence of previously diagnosed cardiovascular disease (Table I).

Data for mortality are complete, but among the 366 patients with ACS, 42 were not considered eligible for follow-up due to dementia and social

Figure 1. Flow-chart demonstrating number of patients and mortality in the different diagnostic categories.

Table I. Baseline characteristics, according to different categories of patients presenting with chest pain

|                | STEMI<sup>a</sup> (n=126) | NSTEMI<sup>b</sup> (n=185) | UAP<sup>c</sup> (n=55) | CHD without ACS (n=164) | Non-coronary chest pain (n=225) | p-value |
|----------------|-----------------------------|-----------------------------|-------------------------|--------------------------|---------------------------------|---------|
| Age (years)    | 69 (55 – 80)                | 76 (64 – 84)                | 69 (60 – 77)            | 70 (59 – 81)             | 60 (48 – 75)                    | < 0.001 |
| Male sex       | 81 (64%)                    | 115 (62%)                   | 30 (55%)                | 108 (66%)                | 114 (51%)                       | 0.015   |
| Medical history|                             |                             |                         |                          |                                 |         |
| Diabetes       | 12 (10%)                    | 27 (15%)                    | 8 (15%)                 | 21 (13%)                 | 21 (9%)                         | 0.421   |
| Prior AMI<sup>e</sup> | 14 (11%)                  | 57 (31%)                    | 19 (35%)                | 118 (72%)                | 0 (0%)                          | < 0.001 |
| Stroke         | 4 (3%)                      | 17 (9%)                     | 9 (16%)                 | 2 (1%)                   | 4 (2%)                          | 0.007   |
| CABG<sup>f</sup> | 5 (4%)                      | 17 (9%)                     | 9 (16%)                 | 56 (34%)                 | 1 (0%)                          | < 0.001 |
| PCI<sup>g</sup> | 4 (3%)                      | 9 (5%)                      | 4 (7%)                  | 35 (21%)                 | 0 (0%)                          | < 0.001 |

Categorical data are presented as n (%) and continuous data as median (inter-quartile range).
<sup>a</sup>ST-elevation myocardial infarction. <sup>b</sup>Non-ST-elevation myocardial infarction. CAD: coronary artery disease. <sup>c</sup>Unstable angina pectoris. <sup>d</sup>Coronary heart disease. <sup>e</sup>Acute myocardial infarction. <sup>f</sup>Coronary artery bypass grafting. <sup>g</sup>Percutaneous coronary intervention.

Statistics: Kruskal-Wallis or χ² tests as appropriate.
reasons, and 65 of those who were asked for informed consent declined to participate. In addition, 35 patients died during the index hospitalisation.

Reperfusion therapy

A total of 73 (58%) patients with STEMI were treated with thrombolysis (tenecteplase + enoxaparin), of whom 36 had pre-hospital treatment. In addition, four patients underwent primary PCI. Thus, the proportion of patients receiving immediate reperfusion therapy in this group was 61% (77/126). Nine patients were transported to rescue PCI after unsuccessful thrombolysis, and one patient had facilitated PCI (tenecteplase).

The cumulative incidence of CABG and PCI for the three diagnostic categories of ACS is shown in Figure 2. During the first two days 14% of patients with STEMI and 3% of those with NSTEMI were treated with PCI. The one-year cumulative revascularisation rates were 54%, 28% and 37% in patients with STEMI, NSTEMI and UAP, respectively.

Adjunctive medical treatment in ACS

Medication during index hospitalisation, at discharge and after six and 12 months is shown in Table II. Aspirin, statins and beta-blockers were used by the majority of STEMI patients throughout the study period, but to a lesser degree given to patients with NSTEMI and UAP. Clopidogrel was given to 75% of NSTEMI and 62% of UAP patients during hospitalisation but only to 56% and 35% on discharge, respectively. Angiotensin converting enzyme inhibitors (ACE-I) was only prescribed to 30 – 40% of all ACS patients during one-year follow-up.

Mortality

The Kaplan-Meier estimates of patient survival are shown in Figure 3. The mortality rates during the first 30-days were 17%, 11% and 2% for NSTEMI, STEMI and CHD without ACS, respectively, whereas no patients with non-coronary chest pain or UAP died during the first month. The one-year mortality rate was 20% among patients with STEMI, 32% in patients with NSTEMI, 7% in patients with UAP, 10% in patients with CHD without ACS and 3% among patients with non-coronary chest pain.

Variables predicting mortality at follow-up according to proportional hazards regression analyses are presented in Table III. After adjustment for age, gender, diabetes and prior AMI the all-cause mortality hazard ratios (using “patients with non-coronary chest pain” as reference) were quite similar between NSTEMI (HR 5.6, 95% CI 2.4 – 13.8, p < 0.001) and STEMI (HR 5.7, 95% CI 2.3 – 14.0, p < 0.001). Further, the apparently small difference in unadjusted mortality risk between patients with UAP (HR 2.8, 95% CI 0.8 – 9.9, p = 0.112) and CHD without ACS (HR 3.8, 95% CI 1.5 – 9.6, p = 0.006) was no longer present, and not significantly different from those hospitalised for non-coronary chest pain, following multiple regression analysis.

Patients with CHD without ACS who died during one-year follow-up were significantly older compared with the survivors in this group (median age 82 vs 67 years, p < 0.001) and all the deceased were
subjected to severe comorbidity (cancer, heart failure, severe atherosclerosis).

**Discussion**

**Mortality**

The primary aim of this study was to evaluate the prognosis of consecutive patients with ACS diagnosed using the ESC/ACC criteria (1). The one-year mortality rates were considerably higher than those observed in clinical trials (4,8–10) and observational surveys (11,12), but consistent with the findings in a recent Danish “confirmative prognostic factor study” (13).

A different selection of patients may partly explain our high mortality figures. Infarction registries are based upon voluntarily participating hospitals and

## Table II. Medication during one-year follow-up according to different categories of acute coronary syndrome.

|                     | STEMI\(^a\) n = 126 | NSTEMI\(^b\) n = 185 | UAP\(^c\) | P-value n = 55 |
|---------------------|----------------------|-----------------------|-----------|----------------|
| **Medication during index hospitalisation** |                      |                       |           |                |
| Aspirin             | 116 (92%)            | 152 (82%)             | 47 (86%)  | 0.046          |
| Warfarin            | 16 (13%)             | 23 (12%)              | 8 (15%)   | 0.917          |
| Clopidogrel         | 37 (29%)             | 138 (75%)             | 34 (62%)  | < 0.001        |
| CCB\(^d\)           | 7 (6%)               | 40 (22%)              | 16 (29%)  | < 0.001        |
| Beta-blocker        | 117 (93%)            | 159 (86%)             | 45 (82%)  | 0.067          |
| Statin              | 106 (84%)            | 118 (64%)             | 32 (58%)  | < 0.001        |
| ACE-I\(^e\)         | 55 (44%)             | 63 (34%)              | 14 (26%)  | 0.046          |
| ARB\(^f\)           | 7 (6%)               | 15 (8%)               | 7 (13%)   | 0.256          |
| **Medication at discharge** |                    |                       |           |                |
| Aspirin             | 100 (86%)            | 122 (76%)             | 41 (75%)  | 0.080          |
| Warfarin            | 14 (12%)             | 22 (14%)              | 7 (13%)   | 0.918          |
| Clopidogrel         | 20 (17%)             | 90 (56%)              | 19 (35%)  | < 0.001        |
| CCB\(^d\)           | 4 (3%)               | 29 (18%)              | 12 (22%)  | < 0.001        |
| Beta-blocker        | 107 (92%)            | 138 (86%)             | 45 (82%)  | 0.119          |
| Statin              | 97 (84%)             | 106 (66%)             | 34 (62%)  | 0.001          |
| ACE-I\(^e\)         | 52 (45%)             | 56 (35%)              | 14 (26%)  | 0.039          |
| ARB\(^f\)           | 4 (3%)               | 11 (7%)               | 5 (9%)    | 0.271          |
| **Medication at 6 months** |                    |                       |           |                |
| Aspirin             | 68 (87%)             | 64 (74%)              | 24 (63%)  | 0.011          |
| Warfarin            | 8 (10%)              | 14 (16%)              | 10 (26%)  | 0.084          |
| Clopidogrel         | 40 (51%)             | 46 (54%)              | 11 (29%)  | 0.032          |
| CCB\(^d\)           | 7 (9%)               | 18 (21%)              | 14 (37%)  | 0.002          |
| Beta-blocker        | 74 (95%)             | 74 (86%)              | 28 (74%)  | 0.006          |
| Statin              | 70 (90%)             | 69 (80%)              | 27 (71%)  | 0.039          |
| ACE-I\(^e\)         | 34 (44%)             | 30 (35%)              | 12 (32%)  | 0.343          |
| ARB\(^f\)           | 6 (8%)               | 10 (12%)              | 8 (21%)   | 0.119          |
| **Medication at 12 months** |                   |                       |           |                |
| Aspirin             | 66 (89%)             | 54 (68%)              | 22 (63%)  | 0.002          |
| Warfarin            | 7 (10%)              | 14 (18%)              | 9 (26%)   | 0.082          |
| Clopidogrel         | 32 (43%)             | 33 (42%)              | 10 (29%)  | 0.311          |
| CCB\(^d\)           | 4 (5%)               | 14 (18%)              | 15 (43%)  | < 0.001        |
| Beta-blocker        | 68 (92%)             | 66 (84%)              | 27 (77%)  | 0.096          |
| Statin              | 67 (91%)             | 65 (82%)              | 25 (71%)  | 0.040          |
| ACE-I\(^e\)         | 30 (41%)             | 34 (43%)              | 12 (34%)  | 0.709          |
| ARB\(^f\)           | 8 (11%)              | 12 (15%)              | 8 (23%)   | 0.255          |

Data are presented as n (%).

\(^a\)ST-elevation myocardial infarction. \(^b\)Non-ST-elevation myocardial infarction. \(^c\)Unstable angina pectoris. \(^d\)Calcium channel blocker. \(^e\)Angiotensin converting enzyme inhibitor. \(^f\)Angiotensin receptor blocker.

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![Figure 3. Kaplan-Meier estimates of patient survival for the five diagnostic categories. Day 0 is day of admission to the emergency room.](image-url)
reported cases. The results in such registries are therefore biased towards better than average practices (12). Furthermore, the registries comprise a mixture of hospitals with and without interventional facilities. Additionally, a Swedish study on patients treated with thrombolysis demonstrated that patients included in a clinical trial had at better prognosis than those not enrolled (14). This difference was explained by selection of less critically ill patients to the trial. Large scale clinical trials with numerous inclusion and exclusion criteria are clearly hampered by selection and information bias. Finally, none of these trials or the observational surveys includes all patients admitted with chest pain without ACS. Since these patients represent a large group, it is of interest to characterise their prognosis in comparison to those with an ACS diagnosis.

The mortality data are not only influenced by the inclusion criteria, but also by the completeness of follow-up. Our mortality data are robust, since vital status was confirmed for all patients, irrespective of clinical follow-up.

As observed in myocardial infarction registries (5,12) and the study of Terkelsen et al. (13), patients with NSTEMI are older, with more advanced disease than those with STEMI. Accordingly, when these differences were adjusted for in a multivariate analysis, the hazard ratios for mortality in patients with STEMI and NSTEMI were quite similar and nearly 4-fold higher than in patients with UAP. The findings underline the prognostic importance of myocardial necrosis (as reflected by elevated troponins) in patients with ACS, since patients with UAP had no rise of troponins, but all had ST-depression and/or negative T-waves in their ECG. It is possible that a lower cut-off value for troponin T would have diminished the number of patients with UAP and increased the NSTEMI population with a "dilution" effect showing a lower mortality. But as shown in the recent survey by Hjortshøj et al. (15), there is a considerable difference in the use of cut-off points for troponins in Scandinavia. Denmark, Iceland and Norway favour the 0.1 µg/l level, whereas both Sweden and Finland apply levels around 0.03 to 0.05 µg/l. Such different practice will obviously influence mortality data among the different categories of ACS.

The one-year mortality rates in patients with UAP and patients with CHD without ACS were comparable/similar in our study. The latter group included patients with recent onset angina. However, patients with established CHD, when hospitalised for acute chest pain, have a similar prognosis to those with UAP in spite of the fact that they had neither a rise of troponins nor new ECG changes compatible with ischemia. Criteria for proceeding to coronary angiography should probably not differ between these two groups. An explanation for the similar mortality rates in these two groups could be that patients without ECG changes or rise in troponins had more cardiovascular comorbidity at baseline.

Interesting information was obtained from the group without any evidence of CHD. They were younger and had a higher female representation than those with established CHD. The one-year mortality was 3%, nearly twice as high as the placebo groups of ACTION trial comprising patients with established stable CHD (16). This is a small difference in absolute terms, but the finding supports the notion that patients admitted to hospital for non-coronary chest pain might have a more severe prognosis than the population in general. Regrettably, this study could not elucidate the causes of death in this group, but the numbers were small and the mortality data in this group must be interpreted with caution.

**Under use of reperfusion therapy**

Sixty-one percent of patients with STEMI received immediate reperfusion therapy (ratio 18: 1 for

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**Table III. Hazard ratios (HR) of death in the different categories of patients with chest pain during the one-year follow-up.**

| Category                              | Univariate Cox regression analysis | Multiple Cox regression analysis |
|---------------------------------------|-----------------------------------|---------------------------------|
|                                       | HR      | 95% CI       | p-value | HR      | 95% CI       | p-value |
| Non-coronary chest pain               | 1.00    | NA            | 0.20    | 1.00    | NA            | 0.20    |
| CHD without ACS                       | 3.77    | 1.48 – 9.64  | 0.006   | 1.49    | 0.55 – 4.07  | 0.438   |
| Unstable angina pectoris              | 2.79    | 0.79 – 9.89  | 0.112   | 1.54    | 0.43 – 5.58  | 0.508   |
| ST-elevation MI                       | 8.36    | 3.43 – 20.38 | < 0.001 | 5.70    | 2.32 – 13.98 | < 0.001 |
| Non-ST-elevation MI                   | 14.77   | 6.38 – 34.20 | < 0.001 | 5.75    | 2.40 – 13.79 | < 0.001 |
| Age                                   | 1.08    | 1.06 – 1.10  | < 0.001 | 1.08    | 1.06 – 1.10  | < 0.001 |
| Male sex                              | 1.41    | 0.95 – 2.09  | 0.089   | 1.99    | 1.30 – 3.03  | 0.002   |
| Diabetes mellitus                     | 1.46    | 0.88 – 2.42  | 0.140   | 0.81    | 0.48 – 1.37  | 0.437   |
| Prior AMI                             | 2.39    | 1.64 – 3.47  | < 0.001 | 1.76    | 1.14 – 2.71  | 0.011   |
| Stroke                                | 1.50    | 0.66 – 3.42  | 0.334   | NA      | NA            | NA      |

*Coronary heart disease. Acute coronary syndrome. Not addressed. The results of univariate and multiple proportional hazards regression analyses are shown. Variables associated with death in the univariate analyses (p < 0.20) were included in the multiple regression model.
thrombolysis vs. primary PCI). This proportion is relatively low but includes patients ineligible for immediate reperfusion because of extended time delays beyond 12 hours. Nevertheless, the findings do not differ from the LEVEREM post-MI study in Norway where 64% received immediate reperfusion (17). In a report by Carruthers et al. the reperfusion rate varied between 71% in UK and 59% multinational (12), not unlike what was found by Eagle et al. where 30% did not receive immediate reperfusion therapy (18). In the Danish study on unselected ACS patients (13), only 55% of all the STEMI patients received acute reperfusion therapy, but 70% underwent this treatment if symptom duration was 12 hours or less. No information is given on the number of primary PCIs versus thrombolytic therapies performed.

Only 14% of STEMI patients and 3% of those with NSTEMI underwent PCI during the first two days. It is remarkable that the cumulative rate of revascularisation at Day 14 and one year is higher in those with UAP than in those with NSTEMI. This observation may reflect that NSTEMI patients were older and subjected to more comorbidity including heart failure, making them less prone to be admitted to coronary angiography and subsequent revascularisation.

The co-operation with the PCI centre at Rikshospitalet University Hospital in Oslo is close, and we had established consensus criteria for transferring patients for coronary angiography. These criteria for submitting patients for coronary angiography were based upon risk stratification according to the European guidelines for NSTEMI (2) and STEMI (3) at the time of the study period.

In view of the mortality figures in the present study, and most recent guidelines and metaanalyses (3,4), our treatment policy has changed. From September 2005 we have sent patients with STEMI and symptom duration 3–12 hours directly to the National Hospital in Oslo (100 km) for primary PCI. All with NSTEMI and UAP are referred for coronary angiography within 24–48 hours after admission.

Suboptimal adjunctive medical treatment

Despite efforts to implement European guidelines for medical secondary prevention from 2003 (19), there was an apparent under use of such medications in the present study.

An explanation for aspirin being less prescribed among patients with NSTEMI and UAP may be the common use of warfarin in patients with atrial fibrillation.

A surprising finding was the under use of medical secondary prevention treatment in patients with NSTEMI and UAP compared with STEMI patients. The observed drop in prescription rate of clopidogrel on hospital discharge was possibly related to reimbursement, since the authorities of Norway did not, at that time, approve clopidogrel prescription without a specific application for each patient. The prescription rate of statins to patients with NSTEMI was lower than among those with STEMI (82% vs. 91%, respectively), but quite similar to the RITA 3 trial where 80% received statins after five years (20). This may be explained by the fact that NSTEMI patients were older and sicker.

Study limitations

Our survey did not include all patients with an ACS in the region during the time of the study. This may have created some bias towards more favourable results, because only patients who survive long enough to have meaningful determinations of a rise in cardiac markers will be included. Despite the fact that patients who died in the emergency room were excluded, our mortality rates were considerably higher than in most previously published trials and registry data.

The early revascularisation rate was very low, and we did not find it appropriate to implement this variable in the survival analyses due to the obvious selection bias and the difficulty in correcting for all known and unknown confounding factors. Similarly we did not collect sufficient data to calculate the GRACE risk score (21), and recognise that this would have been a valuable supplement to the multivariate analyses.

Conclusions

ACS diagnosed according to the ESC/ACC criteria is a far from benign condition in unselected conservatively managed patients admitted to a teaching hospital without PCI facilities. One-year mortality rates were higher than in most myocardial infarction registries or randomised clinical trials, but similar to an observational survey performed at one single hospital in Denmark. Apart from patient selection, under use of invasive therapy and drugs with proven prophylactic effects are factors that may explain these high mortality figures. The evidence suggests that current practice probably needs to be changed into a more aggressive approach to invasive procedures and secondary prevention.
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