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Management of patients admitted with an Acute Coronary Syndrome in New Zealand: results of a comprehensive nationwide audit

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Abstract

Aims To audit all patients presenting to a New Zealand hospital with a myocardial infarction or unstable angina (an acute coronary syndrome [ACS]) over a 14-day period, to assess their number, presentation type and patient management during the hospital admission.

Methods We formed a group of clinicians to lead the local audit process with one representative for each hospital (n=36) that admitted ACS patients. A comprehensive data form was used to record individual patient information for patients admitted between 0000 hours on 13 May 2002 to 2400 hours on 26 May 2002.

Results 930 patients were admitted with a suspected or definite ACS: 11% with a ST-segment-elevation myocardial infarction (STEMI), 31% with a non-STEMI, 36% with unstable angina pectoris (UAP), and 22% with another cardiac or medical diagnosis. Cardiac investigations were limited: echocardiogram (20%), exercise treadmill test (20%), cardiac angiogram (21%).

In-hospital revascularisation rates were low for those patients with a definite presentation with an ACS (STEMI, non-STEMI, UAP, n=721). Percutaneous coronary intervention (PCI) rates were 13%, 8%, and 4%—with coronary artery bypass grafting (CABG) rates being 4%, 3%, and 4% respectively. The use of discharge medications of proven benefit was also generally low (n=695): aspirin (82%), clopidogrel (8%), beta-adrenergic blockers (63%), angiotensin converting enzyme (ACE) inhibitors (43%), and statins (55%).

Conclusions A collaborative group of clinicians has performed a nationwide audit of acute coronary syndrome patients, which has demonstrated low levels of investigations, evidence-based treatments, and revascularisation. There is a need for a comprehensive national strategy—particularly for continuing audit of the treatment of patients presenting with a suspected or definite acute coronary syndrome to a New Zealand hospital.

There is unequivocal evidence that certain treatments improve the outcome of patients presenting with an ACS.[1] International and local guidelines support intensive medical treatment, and for many patients early revascularisation, which is of proven benefit and shown to be cost-effective in high-risk patient groups.[2–6]

Comprehensive national surveys of ACS patients have previously been attempted in several countries, including Argentina[7] and Italy.[8] Other countries such as Britain[9] and the United States of America[10] have performed less complete surveys. In addition,

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some international trials and registries, such as the Global Registry of Acute Coronary Events (GRACE)[11] and the European Heart Survey[12] have attempted to compare the treatment of ACS patients between countries and regions.

In New Zealand, both the number and management of patients presenting with an ACS, is unknown, although an earlier Auckland-based study provided useful demographic and outcomes data from 1993.[13] Many clinicians are unable to optimally manage the ACS patients under their care due a limited provision of service and a relatively low level of funding.

In May 2001, the Cardiac Society of New Zealand supported a meeting, which invited representatives from all major New Zealand hospitals to discuss the appropriate management of patients with ACS. At this meeting, the need for a national audit was further developed and endorsed as an important aspect of the strategy to improve patient care. New Zealand has favourable characteristics to undertake a comprehensive national survey. There is a history of good collaboration in cardiovascular research, which is strengthened by the small specialist community across the country, and the personal contact between clinicians.

We therefore created a network of practicing clinicians representing every New Zealand hospital that admits ACS patients and performed a comprehensive National audit of the in-hospital management of all ACS patients across New Zealand. We chose to undertake the audit in a 2-week period in the autumn of 2002, to minimise the known influence of seasonal change on the numbers of ACS patients.[14]

Methods

Data collection—A network was created—consisting of one physician for every hospital in New Zealand that admitted ACS patients (n=36). Most centres also co-opted one or more research nurses or registrars to assist with data collection for the study.

The data collection form recorded patient demographics, initial and discharge diagnosis, medication use in hospital and at discharge, as well as investigations undertaken and invasive treatments received by patients. The inclusion criterion for the audit was ‘a patient admitted overnight with a suspected or definite acute coronary syndrome’.

A 2-week audit period was accepted as a compromise between the need to collect sufficient patient numbers to obtain an accurate representative cohort versus the ability of unfunded clinicians and nurses to collect the consecutive patient data. We collected data from 0000 hours on Monday 13th May to 2400 hours on Sunday 26th May 2002.

Following input from the 14 other local ethics committees across New Zealand, ethical approval was obtained from the North Health Ethics Committee. As an audit of current practice, individual patient consent was not required. The ethics committees encouraged the collection of patient names and National Health Index (NHI) numbers to assist with accurate data collection.

Data (including revascularisation procedures) from patients subsequently transferred to another institution are ‘attributed’ to their original admitting hospital. Patients readmitted within the 2 weeks have all admissions included in the data; they only represented a small % of the overall patient number. Ethnicity was self-reported at hospital admission.

All 36 hospitals had the facilities for assessing troponin levels. Five methods were used: troponin T [Roche] (16 hospitals), troponin I [Abbott] (13 hospitals), troponin I [Bayer] (3 hospitals), troponin I [Ortho] (1 hospital), ‘Rapid’ troponin T [Roche] (3 hospitals). In order to divide non-STEMI and unstable angina patients by means of a ‘positive’ troponin,[15] we defined ‘normal’ or ‘abnormal’ troponin levels using the ‘cut-off’ for ‘positive’ troponins as Troponin T [Roche] $\geq$0.03ug/L, Troponin I [Abbott] $\geq$0.4ug/L, Troponin I [Bayer] $\geq$0.2ug/L, Troponin I [Ortho] $\geq$0.08ug/L, ‘Rapid’ troponin T ‘positive’ [Roche].

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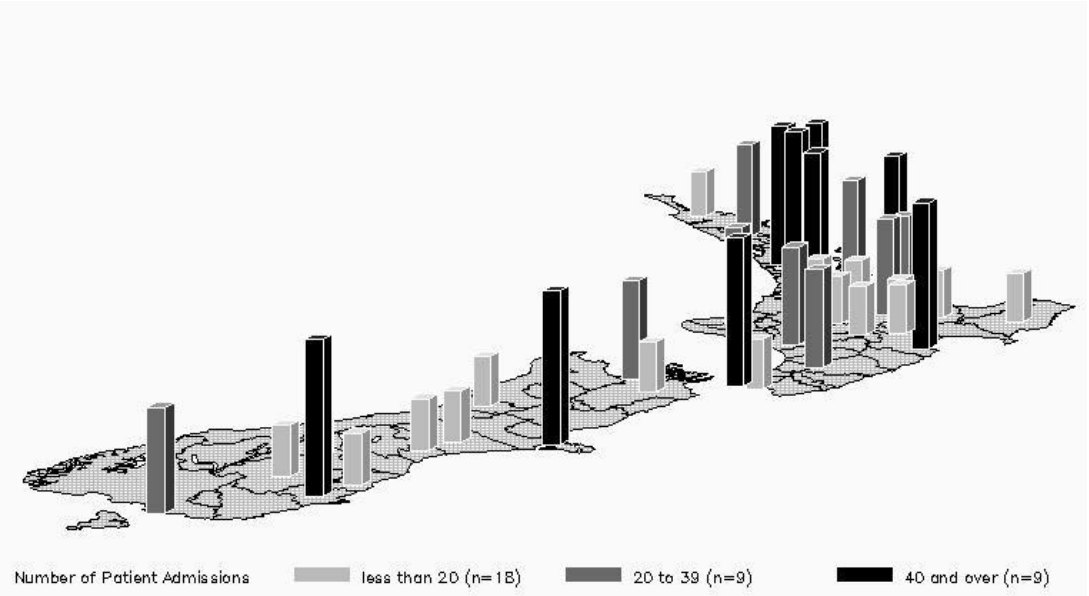
Hypertension and dyslipidaemia were defined as patients on treatment, or with a previous clinical diagnosis. Patients with diabetes mellitus were those on diet control, oral hypoglycaemic, or insulin treatment. Cardiogenic shock was defined as: a systolic blood pressure of <90mmHg for at least 30 minutes, or the need for supportive measures to maintain a systolic blood pressure of ≥90mmHg with end organ hypoperfusion.[16] Sustained ventricular tachycardia was defined as >30 seconds of ventricular tachycardia, or requiring electrical cardioversion.

Statistics—Continuous data are summarised as median and interquartile range. Differences in frequencies were tested using chi-squared procedures. All tests were two-tailed and a 5% significance level was used.

Results

930 patients with a suspected or definite ACS were admitted to 36 New Zealand hospitals and enrolled in the ACS audit over the 14-day period (Figure 1). Thirty-six patients were readmitted—within the 2 weeks, 35 patients were admitted once and 1 patient was admitted twice (29 readmissions to the same hospital, and 7 to another hospital). Fifty-seven patients were transferred from their admitting hospital to another institution for further management (53 [93%] to an intervention centre). Over the 2 weeks, one hospital had no admissions, 9 hospitals admitted 40 or more patients, and one hospital admitted 131 patients.

Figure 1. New Zealand ACS hospitals (n=36) and patient numbers (n=930)



Patient demographics—The median age was 69.6 (IQR 58-78, range 21-102) years. Forty-two percent of patients were female, 81% Caucasian, 7% Maori, 2% Indian, 1% Pacific Islander, 1% Asian, and 5% were another ethnic group—and in 5% the ethnicity was unspecified. Baseline demographics are shown in Table 1.

Patient diagnoses—Using both the admission clinical diagnosis and the measurement of a positive troponin level, we found that 101 (11%) patients presented with a ST-segment-elevation myocardial infarction (STEMI), 287 (31%) with a non-STEMI,

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333 (36%) with unstable angina pectoris (UAP), and 209 (22%) patients with another cardiac or medical diagnosis.

Table 1. Baseline demographics (n=930)

Median age (IQR): 69.6 yrs (58–78 yrs)	
Gender (male)	535 (58%)
Ethnicity	
-Caucasian	753 (81.0%)
-Maori	62 (6.7%)
-Other groups	115 (12.4%)
Tobacco Smoker	
-Current	171 (18%)
-Previous	379 (41%)
-Never	347 (37%)
-Not reported	33 (4%)
Hypertension	442 (48%)
Diabetes mellitus	161 (17%)
Dyslipidaemia	326 (35%)
Prior MI	325 (35%)
Prior angiogram	257 (28%)
Prior PCI	105 (11%)
Prior CABG surgery	91 (9.8%)
Prior peripheral arterial disease	93 (10%)
Prior TIA/stroke	112 (12%)
Prior atrial fibrillation	126 (13%)

IQR: Interquartile range; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; TIA: Transient ischaemic attack.

Medical management—Medical treatments are shown in Table 2. Overall, 55% of STEMI patients received thrombolytic therapy. Of 77 STEMI patients who were admitted within 12 hours of symptom onset, 53 (69%) received thrombolytic therapy and 3 (3.9%) received primary PCI. Sixty-four percent of non-STEMI patients were treated with low molecular weight heparin (54% enoxaparin, 12% daltaparin) and 8.8% unfractionated heparin. A few patients received more than one type of heparin, 92 (32%) patients were not treated with any heparin. Three percent of non-STEMI patients received a glycoprotein 2b/3a inhibitor, and 13% clopidogrel therapy.

Cardiac Investigations—Investigations are listed in Tables 2 and 3. Of the 930 patient admissions, 184 (20%) underwent an echocardiogram, 190 (20%) received an exercise treadmill test, and 199 received (21%) a cardiac angiogram. 583 (63%) patients received neither an exercise treadmill test nor a cardiac angiogram.

Revascularisation—Of 721 ‘definite’ ACS patients (STEMI, non-STEMI, UAP), 159 (22%) patients underwent a cardiac angiogram, 50 (6.9%) patients received a PCI, and 25 (3.5%) patients received CABG (Tables 2 and 3).

Hospital outcomes—Thirty-eight (4%) patients died during their hospital admission: 14 (14%) of STEMI patients, 12 (2%) of non-STEMI/UAP patients, and 11 (5%) of ‘other cardiac or medical diagnosis’ patients. Twenty (2%) patients had a recurrent or subsequent myocardial infarction, and 144 (16%) had recurrent angina. Cardiogenic shock developed in 41 (4%) patients. Twenty (2%) patients received an intra-aortic

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balloon pump, 12 (1%) received a temporary pacemaker, and 2 patients received a permanent pacemaker. Six patients developed a stroke (5 non-haemorrhagic), and 13 (1%) sustained ventricular tachycardia. Only 1% of patients were enrolled in a research project whilst in hospital.

Table 2. Treatments and investigations of STEMI, Non-STEMI, and UAP patients

	STEMI	Non-STEMI	UAP	P**
N	101 (11%)	287 (31%)	333 (36%)	
Treatments in hospital				
Thrombolytic therapy	56 (55%)			
Primary PCI	3 (3.0%)			
Enoxaparin	33 (34%)	156 (54%)	126 (39%)	<0.001[a]
Daltaparin	6 (5.9%)	33 (12%)	39 (12%)	0.23
UF heparin	28 (28%)	25 (8.8%)	22 (6.6%)	<0.001[b]
No heparin***	40 (40%)	92 (32%)	159 (48%)	0.0011[a]
Tirofiban	5 (5.0%)	6 (2.1%)	1 (0.3%)	0.0046[c]
Eptifibatide	2 (2.0%)	2 (0.7%)	1 (0.3%)	0.20
Abciximab	1 (1%)	0	0	0.05
Aspirin	87 (88%)	228 (79%)	268 (81%)	0.22
Clopidogrel	14 (14%)	35 (13%)	21 (6.2%)	0.015[d]
Investigations in hospital				
Chest X-ray	89 (89%)	265 (92%)	269 (81%)	0.0001[e]
Echocardiogram	35 (35%)	61 (22%)	54 (16%)	0.0003[b]
Exercise test	18 (18%)	52 (18%)	86 (26%)	0.04[e]
Angiogram	31 (31%)	71 (35%)	57 (16%)	0.006[d]
No ETT/Angio	57 (57%)	180 (63%)	203 (63%)	0.51
No Echo/Angio	50 (50%)	178 (62%)	241 (72%)	<0.0001[e]
PCI	13 (13%)	24 (8.4%)	13 (3.9%)	0.004[d]
CABG surgery	4 (4.0%)	8 (2.8%)	13 (3.9%)	0.72
In-hospital deaths	14 (14%)	10 (3.5%)	2 (0.9%)	<0.0001[f]
Discharge medications (n=721-26 deaths: n=695)				
Aspirin	77 (89%)	228 (83%)	266 (80%)	0.78
Clopidogrel	14 (14%)	26 (9.5%)	17 (5.1%)	0.01[g]
Beta-blockers	68 (76%)	177 (63%)	193 (59%)	0.22
ACE-inhibitors	43 (51%)	127 (45%)	128 (39%)	0.33
Statins	58 (67%)	153 (55%)	172 (52%)	0.59
Fibrates	0	7 (2.5%)	9 (2.4%)	0.26

**Compares STEMI/Non-STEMI/UAP Post hoc tests.

[a]UAP different from Non- STEMI, Non- STEMI different from STEMI.

[b]STEMI different from Non- STEMI and UAP.

[c]STEMI different from UAP, Non- STEMI different from UAP.

[d]UAP different from Non-STEMI and STEMI.

[e]UAP and Non-STEMI different.

[f]All different.

[g]UAP and STEMI different.

***Neither enoxaparin, daltaparin, nor UF heparin.

PCI: Percutaneous coronary intervention; UF: Unfractionated; CABG: Coronary artery bypass grafting; Angio: Angiogram; ETT: Exercise treadmill test; UAP: Unstable angina pectoris; ACE: Angiotensin converting enzyme.

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Clinicians from 13 hospitals, which admitted ACS patients exclusively to their coronary care unit (CCU), were confident of collecting all ACS patient admissions (n=202). 10 hospitals that admitted patients with an ACS to either a CCU or to a medical ward were able to fully enrol these patients into the audit (n=320). 4 hospitals, which admitted patients to either the CCU or to a medical ward, were able to enrol all CCU patients and most of the medical ward patients (n=172), estimating that 2–5% of medical ward patients (6 patients) were missed. 9 hospitals, which admitted ACS patients to both the CCU and the medical ward, were able to enrol all CCU patients (n=266) but none of the medical ward patients, and missed an estimated 5 to 30% of patients (37 patients). Hence, an estimated total of 43 ACS patients (4%) were admitted to a medical ward over the 2 weeks, and not included nor further considered in this audit.

Table 3. Investigations and invasive treatments

Procedure	All patients (n=930)	'Definite' ACS patients* (n=721)
Chest X-ray	794 (85%)	623 (86%)
- Pulmonary oedema **	96 (10%)	76 (11%)
Echocardiogram	184 (20%)	150 (21%)
Exercise treadmill test	190 (20%)	156 (22%)
Cardiac angiogram	199 (21%)	159 (22%)
Exercise test and cardiac angiogram	42 (4.5%)	38 (5.3%)
Exercise test or cardiac angiogram	347 (37%)	277 (38%
Neither exercise test or cardiac angiogram	583 (63%)	444 (62%)
PCI	69 (7.3%)	50 (6.9%)
CABG	35 (3.8%)	25 (3.5%)

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; *Patients with STEMI (n=101, non-STEMI (n=287), and unstable angina pectoris (n=333); **Physician/Radiologist assessment.

Discussion

The major strength of this study has been the demonstration that a collaborative group of clinicians can perform a prospective, comprehensive audit of patients admitted to 36 hospitals in New Zealand with an ACS. Our approach has allowed clinicians to collect the data, which we believe has resulted in an accurate collection. It also allows clinicians to collectively have responsibility for the data. By benchmarking current practice, the ACS audit will enable physicians to examine the provision of equitable and good practice throughout the country. Moreover, the infrastructure created will facilitate future audit with the goal of assessing temporal trends and improving patient outcomes.

We identified 930 patients admitted with presentations of STEMI (11%), non-STEMI (31%), UAP (36%), and other cardiac or medical diagnosis (22%). The hospital management and outcomes of these patients throughout New Zealand had not previously been known.

In-hospital investigations: For the entire cohort, the use of a chest X-ray following presentation with a suspected or definite ACS was 85%. The use of an

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echocardiogram (20%), exercise treadmill test (20%), or cardiac angiogram (21%) was low.

For the STEMI and non-STEMI patients (n=388), with myocardial damage and at the highest risk, the use of echocardiography (25%) or angiography (26%) was low, with 59% receiving neither as a method of assessing left ventricular systolic function, which is important in risk stratification.[17]

Furthermore, for the same group, the use of an exercise treadmill test (18%) or a cardiac angiogram (26%) as methods of risk assessment was also low—with 61% of patients not receiving either test. These levels of investigation contrast with the recommendations of local and international guidelines,[2–6] which recommend that all STEMI and non-STEMI patients be considered for assessment in these ways.

International comparisons—Previous international ACS patient cohorts[7–12] have selected different patient populations, which limits the ability to compare these studies with the New Zealand ACS Audit. The GRACE registry[11,18] is probably the most appropriate comparator for our audit, although the methods used to enrol patients were similar, but not identical. It was not designed as a comprehensive national survey, rather as a collection of 95 hospitals in 14 countries in North and South America, Europe, Australia, and New Zealand (2 sites) which has allowed comparisons to be made between countries.

Patients entered in the GRACE registry had to be admitted with an ACS as the presumptive diagnosis and to have $\geq$1 of the following: electrocardiographic changes consistent with ACS, serial increases in serum biochemical markers of cardiac necrosis, and/or documentation of coronary artery disease.[18]

Reperfusion therapy, heparin, and platelet inhibitor use—In the current audit, 77 STEMI patients were admitted within 12 hours of symptom onset and had the most to gain from reperfusion: 53 (69%) received thrombolytic therapy and 3 (3.9%) were treated with PCI. In the widely spread population of New Zealand, this finding was expected as 24-hour cover for primary PCI was routinely available at only 1 centre in New Zealand, with the 4 other public interventional centres offering it to a variable extent. The overall level of thrombolytic therapy for STEMI patients (55%) is consistent with data from GRACE where thrombolytic therapy use was 47%—although primary PCI use was higher in GRACE at 18%,[19] compared to 3% in the New Zealand ACS Audit.

Some non-reperfused patients are likely to have contraindications to treatment (although we did not specifically record this)—but some patients will probably have missed an opportunity for reperfusion, which has also been reported from GRACE.[20] Further study of this issue would be helpful.

Overall, we found that there was a low level (68%) of heparin use (unfractionated or low molecular weight) for non-STEMI patients. Patients not treated would be unable to benefit from an estimated 47% reduction in death or myocardial infarction.[21]

Although there will certainly be a small group of non-STEMI patients with a clear contraindication to the use of heparin, these treatment levels are lower than expected and may be an area where clinicians can improve their medical treatment. Again, further study of this issue would be helpful. All 36 hospitals had access to heparins—although not all hospitals had access to enoxaparin, which is specified as preferable to

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unfractionated heparin by the Australia and New Zealand, and the United States Guidelines.[2,6]

There was a very low use of intravenous glycoprotein 2b/3a inhibitors (3%) for the management of non-STEMI patients, despite the recommendations of international and local guidelines, and despite the expected 9% reduction in death or myocardial infarction.[22]

Many hospitals do not have glycoprotein 2b/3a inhibitor drugs available for clinicians to use, a result of local pharmaceutical policies. In addition, some clinicians try to 'target' glycoprotein 2b/3a inhibitor drugs to particularly 'high-risk' patients, such as those with elevated troponins and diabetes mellitus.[22]

Medically treated patients were also largely unable to benefit from the use of clopidogrel (used in 13%)—despite a 20% reduction in cardiovascular death, myocardial infarction or stroke demonstrated in similarly treated patients in the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial.[23] Patients receiving a PCI were the only group to routinely access clopidogrel.

In-hospital revascularisation—Overall, revascularisation was undertaken in 17% of STEMI, 11% of non-STEMI and 8% of UAP patients. We found that PCI during the hospital admission was performed in 13%, 8%, and 4% of patients and CABG in 4%, 3% and 4% of patients, from each group. This intervention level is low compared to figures from the GRACE Registry,[11] which reported PCI rates as being 40%, 28%, 18%, and CABG surgery rates being 4%, 10% and 5% (Table 4 and Figure 2).

Table 4. Cardiac interventions by baseline condition: comparison with the GRACE Registry[11]

	Patients					
	STEMI		Non-STEMI		UAP	
	GRACE n=3419	NZACS n=101	GRACE n=2893	NZACS n=287	GRACE 4393	NZACS n=333
Angiogram	55%	31%	53%	25%	42%	17%
PCI	40%	13%	28%	8.4%	18%	3.9%
CABG surgery	4%	4%	10%	2.8%	5%	3.9%

STEMI: ST-segment-elevation myocardial infarction; UAP: Unstable angina pectoris; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.

Indeed, many STEMI, non-STEMI and UAP patients will potentially benefit if treated with revascularisation,[2–6,24–27]—and the low levels found in the current audit are a cause of concern.

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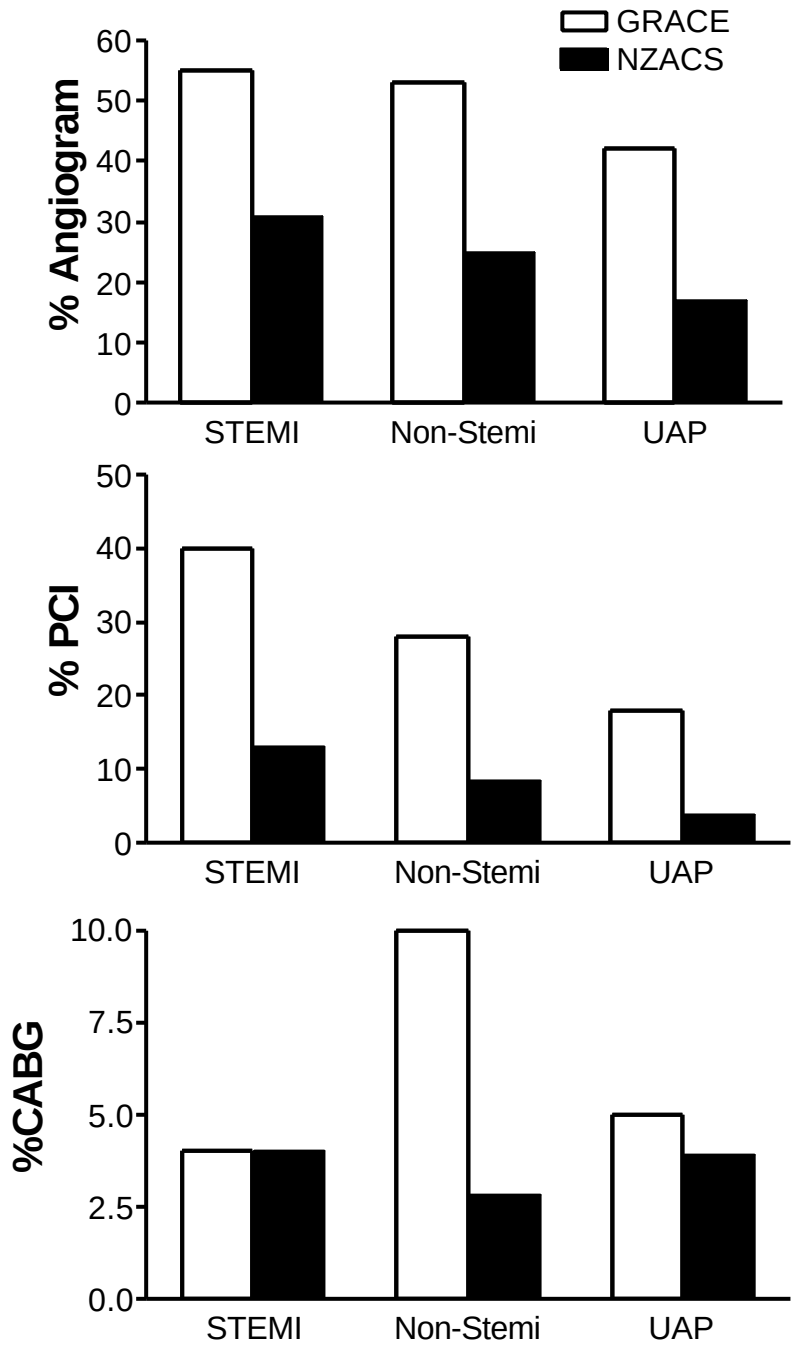
Figure 2. Cardiac interventions by baseline condition: comparison with the GRACE Registry[11]



Discharge medications—Patients (without clear contraindications), and discharged following a presentation with an ACS, should be routinely prescribed aspirin—resulting in a reduction in vascular events of about 25%[28] and clopidogrel with a 20% reduction for those with a non-STEMI presentation.[23]

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In addition, a beta-blocker, with a reduction in risk of death of around 20%[29], and a statin, with a reduction in risk of death of approximately 30% in 5 years,[30,31] should be given to these patients. Furthermore, an angiotensin converting enzyme (ACE) inhibitor should be given acutely for all patients with anterior MIs, second or subsequent MIs, and for those patients with heart failure and an ejection fraction of <40%—resulting in a reduction of the risk of death of 25%.[32]

ACE-inhibitors should also be prescribed for all patients with coronary or other vascular disease, due to their role in preventing vascular events.[33,34] In addition, other medications may also be appropriate for some sub-groups of these patients.

For STEMI/non-STEMI/UAP patients, the use of various therapies at discharge (May 2002) was generally lower than the rates reported in patients enrolled in GRACE from April 1999 to December 2000[11]: aspirin (80–89% vs 90–95%), beta-blockers (59–76% vs 75–81%), ACE inhibitors (39–51% vs 50–57%), and statins (52–67% vs 37–51%), respectively.

Unfortunately we did not record the presence of contraindications for the use of secondary prevention therapies. Nevertheless, only half of New Zealand ACS Audit patients received a statin—a sub-optimal level,[30,31] which in New Zealand has been partly due to PHARMACs previous funding restrictions, resulting in a low level of statin use across the community.[35]

The results of the Heart Protection Study[36] showed a benefit for vascular patients (individuals with prior coronary, cerebral or peripheral vascular events, or with diabetes mellitus or hypertension, at high-risk of developing events) with a cholesterol level of 3.5mmol/L and above, and implied that all vascular patients should be considered for statin therapy after presentation with an ACS.

Although there are Australia and New Zealand guidelines for the management of patients with ACS (endorsed by the Cardiac Society of Australia and New Zealand), it is recognised that it would be of value to have local guidelines addressing some of the unique aspects of the New Zealand health scene where there are restrictions to funding and the availability of various therapies. These local, guidelines are being developed with the help of the Cardiac Society and the New Zealand Guidelines Group.

Modern medical and revascularisation treatments enhance patient outcomes[2–6] and many have been shown to be 'cost-effective'. These include the use of statin therapy following a MI,[37-39] and the use of an invasive revascularisation strategy for non-STEMI patients.[40–42] It should be emphasised how cost-**ineffective** it is to **not** have adequate facilities available for use, or for them not to be used. This point must be emphasised to politicians, health administrators, PHARMAC, physicians and the public of New Zealand.

Study limitations include the fact that a short audit may produce some chance findings and bias in patient selection. Furthermore, we were reliant on local investigators to check the accuracy of individual patient data, without there being a central system of review. Nevertheless, this audit has revealed deficiencies with the delivery of optimal management for ACS patients, with a low level of service provision for this high-risk group. These data show that few hospitals in New Zealand are practising evidence-based medicine according to local and international guidelines.[43] It is probable that

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this situation results, at least in part, from both a limited central coordination of clinical service as well as a lack of local support services.

Conclusions—There appear to be deficiencies in the availability and use of modern medicines, as well as inadequate facilities for appropriate investigations and revascularisation of patients with acute coronary syndromes. Continuing audit and feedback to local clinicians will probably help to improve the clinical service. Furthermore, open, informed public debate needs to be encouraged as to the level of service provision available in all regions of New Zealand. We believe that there is an urgent need to develop a comprehensive nation-wide strategy for patients presenting to a New Zealand hospital with an ACS.

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Auckland/Northland (North Island)

Kawakawa Hospital, Dr P Burgoyne, Ms S August (4), **Whangarei Hospital,** Dr B Wong, Ms K O'Keefe (30), **North Shore Hospital**, **Auckland**, Dr H Hart, Ms J Wickham (66), **Auckland Hospital,** Dr C Ellis#*, Mr G Gamble*, Ms W Benjamin (48), **Mercy Private Hospital**, **Auckland**, Dr T Clarke, (4), **Green Lane Hospital**, **Auckland**, Assoc Prof J French*, Prof H White*, Ms B Williams (26), **Ascot Private Hospital, Auckland,** Dr A Maslowski, (0), **Middlemore Hospital, Auckland,** Dr A Ko, Dr M Lund, Dr H Oettli (40).

Waikato/Central North Island

Thames Hospital, Dr J Lennane, Dr Aftabuzzaman (23), **Tauranga Hospital,** Dr J Tisch, Dr G Porter, Ms V Watts, Ms J Braid (48), **Waikato Hospital**, **Hamilton,** Dr G Devlin*, Ms D Penney (63), **Whakatane Hospital,** Dr E Edwards, Ms D Garner (13), **Rotorua Hospital**, Dr K Logan, Ms A Morley (26), **Tokoroa Hospital,** Dr P Reeve, Dr F Kanan (1), **Te Kuiti Hospital,** Dr P Reeve, Dr J Pusupati (2), **Taupo Hospital,** Dr A Ludbrook (11), **Gisborne Hospital,** Dr F Aitcheson, Ms K Weytmans (7), **Tauramunui Hospital**, Dr P Reeve, Dr R Shepherd (1), **New Plymouth Hospital,** Dr I Ternouth (28).

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Wellington/Southern North Island

Hastings Hospital, Dr R Luke, Ms J Mackenzie (66), **Wanganui Hospital,** Dr T Thompson, Ms K Olsen (30), **Palmerston North Hospital**, Dr R Shameem (27), **Masterton Hospital,** Dr T Matthews, Ms K Lee (12), **Hutt Hospital,** Dr S Mann*, Ms A Cuthbert (19), **Wellington Hospital**, Dr P Matsis*, Ms D Middlemitch, Ms B Scott (50), **Wakefield Private Hospital, Wellington**, Dr M Abernethy (2), **Nelson Hospital,** Dr A Hamer, Ms R Price (21), **Blenheim Hospital**, Dr M Heynike, Ms M Udy (16).

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Dunedin, Otago (South Island)

Oamaru Hospital, Dr P Curzon (4)**, Dunstan Hospital Clyde,** Dr G Nixon, Ms S Meaden (9), **Dunedin Hospital**, Dr MJ Williams*, Ms M McLelland (42), **Invercargill Hospital,** Dr C Renner, Dr A Maloney (23).

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