The quality of care in acute coronary syndromes


The Euro Heart Survey — Acute Coronary Syndromes (EHS–ACS)[1] and the Global Registry of Acute Coronary Events (GRACE)[2] presented in this issue, provide insight into the practice of cardiology in different hospital settings, and in different countries. EHS–ACS recorded prospectively 14 271 patients admitted with chest pain, with a subsequently documented acute coronary syndrome (ACS) in 10 484 (73%). GRACE identified in a prospective or retrospective manner, 11 543 patients with a final diagnosis of myocardial infarction or unstable angina i.e. an acute coronary syndrome in 10 709 patients (93%).

In both studies, this admission diagnosis of non-ST elevation ACS was more frequent than evolving myocardial infarction with ST-elevation. In those patients with ST-elevation, 56% (EHS–ACS) and 65% (GRACE) received reperfusion therapy. The latter figure (65%) is a summation of thrombolytic agents (47%) and primary PCI (18%) assuming no overlap among these therapies. An analysis as to why reperfusion therapy was not provided is presented in EHS–ACS, identifying late arrival (how late?), resolution of the initial ST elevation and advanced age (how old?), and unavailability of such therapy (3-7%) as reasons not to offer this therapy. It is amazing indeed that in some hospitals facilities (fibrinolytic drugs) for life-saving reperfusion therapy were not immediately available and we can only hope that the physicians and hospital administrators will have improved this in the mean time. The other reasons for not giving reperfusion therapy should be studied in more detail, since it is likely that a significant proportion of those patients might have benefited from either fibrinolysis or direct PCI, in spite of relatively late arrival or advanced age. EHS–ACS also analysed the time delay between hospital arrival and initiation of fibrinolytic therapy (mean 40 min) or PCI (93 min). Again, this gives much room for improvement and challenges each hospital to carefully review their procedures for these patients.

In both EHS–ACS and GRACE about half of all patients underwent diagnostic coronary angiography, PCI was conducted in 40% of patients admitted with ST elevation and in about a quarter of patients without initial ST elevation. This includes patients with ST elevation undergoing primary PCI. Rates of bypass surgery were low: 3–10%. It is striking that both studies indicated that revascularization was carried out frequently because it was ‘routine practice’: or because facilities were available. Practice guidelines recommend systematic risk stratification and revascularization in patients with high risk characteristics, such as recurrent ischaemia or elevated troponin levels, but it is not clear whether these guidelines are followed in clinical practice. In any case, availability is a major factor in the decision to perform or not offer revascularization.

Medical therapy in hospital includes aspirin in over 90% of patients, appropriate use of unfractionated or low-molecular-weight heparin, but low use of glycoprotein IIb/IIIa receptor blockers when compared to guideline recommendations. Therapy at discharge includes aspirin and/or ticlopidine/clopidogrel in over 90% of patients, ACE-inhibitors (EHS–ACS 56%, GRACE 55%), β-blockers (EHS–ACS 73%, GRACE 71%) and statins (EHS–ACS 53%, GRACE 47%). These figures are appropriately higher than in the recent Euro Heart Survey secondary prevention (Euro Aspire II), indicating an increasing awareness of the need for preventive medication in most patients with coronary artery disease. However, yet again, further improvement is required, since the proportion of patients discharged on statin therapy, in particular, remains too low.

The data summarized above show important similarities between the two studies. This supports the validity of the findings, and strengthens the need for further improvement of medical care. However, there are some important differences, which make it difficult to compare outcome in specific patient groups. In particular, the terminology applied for the initial, or admission diagnoses, and final diagnoses differ. The EHS–ACS uses the term ACS with or without ST elevation as initial diagnoses, and Q wave MI, non-Q MI and unstable angina as discharge diagnoses. In contrast, GRACE initially describes patients as having unstable angina or myocardial infarction, and uses ST elevation MI and non-ST elevation MI as discharge diagnoses. This is confusing and calls for standardization! In fact, such standardization is provided by the joint ESC–ACC task force report on the definition of myocardial infarction. The terminology of EHS–ACS is in agreement with these recommendations and it is regrettable that GRACE did not follow the same.

The Euro Heart Survey–ACS and the GRACE registry provide important and reliable information on the quality of care in acute coronary syndromes, and identify areas for further improvement of such
quality. Most patients receive reperfusion therapy, oral antiplatelet agents, and anticoagulant therapy as appropriate. However, intravenous therapy with glycoprotein IIb/IIIa receptor blockers as well as β-blockers, statins and probably ACE inhibitors are underused. Furthermore, the coronary revascularization procedures are not used as often as recommended in recent guidelines. In particular, access to such procedures should be improved for patients initially treated in hospitals without facilities for invasive investigation and therapy.

Repeated surveys and ongoing registries indicate that adherence to guidelines does improve over time, and that this is associated with improved patient outcome (Fig. 1). Continuing surveys and registries are essential to reassess the quality of care at regular intervals. This may be greatly facilitated by integration of clinical data registration systems and data collection for national and international registries and surveys[3].

At this moment, the financial support for these surveys and registries comes from the European Society of Cardiology, a few national heart foundations and pharmaceutical and medical device industries. We should be grateful for this industrial support. But, this carries the risk that these studies may be limited to areas of significant industrial financial interest, including acute coronary syndromes, heart failure and preventive therapy. Independent support through health care providers, national health authorities and/or the European Union would be appropriate to ensure ongoing assessment of the quality of care in cardiology.

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References


Thrombin inhibitors in acute coronary artery disease

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Ischaemic heart disease is the most common cause of death worldwide. Following disruption of an atherosclerotic plaque, the de-endothelialized vascular surface is exposed to flowing blood. The coagulation system is activated, leading to thrombin generation. Thrombin generation is a powerful platelet agonist that contributes to platelet activation and recruitment, and catalyzes the formation of fibrin. This dynamic process of platelet–fibrin thrombus underlies the pathogenesis of the acute coronary syndromes. Therapies designed to interrupt this thrombotic process include antiplatelet and anticoagulant agents.

Unfractionated heparin remains a widely used anticoagulant, and is endorsed by the major treatment guidelines published on both sides of the Atlantic[1,2]. However, the antithrombin effects of unfractionated heparin are limited for numerous reasons: heparin inhibits thrombin indirectly (requiring an interaction with antithrombin III); unfractionated heparin is bound by circulating plasma proteins, resulting in unpredictable bioavailability and difficulties in maintenance of a stable therapeutic range; the residual thrombus contains active thrombin bound to fibrin, which is stoichiometrically poorly accessible to the large heparin–antithrombin III complex; the platelet rich arterial thrombus releases large amounts of platelet factor 4, which inhibits heparin; fibrin II monomer formed by the action of thrombin on fibrinogen may inhibit thrombin; heparin may cause immunologically mediated thrombocytopenia. Numerous clinical studies have described an increase in thrombotic events and coagulation markers following cessation of heparin infusion (‘rebound’). It is not altogether surprising therefore that the available (but under-powered) clinical trial data fail to provide conclusive evidence demonstrating benefit from the addition of unfractionated heparin in addition to aspirin.

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