Invasive coronary revascularisation is better than conservative treatment in patients with acute coronary syndromes

New advances in interventional cardiology and anti-thrombotic pharmacological treatment have modified the risk:benefit ratio of percutaneous interventions in acute coronary syndromes without ST segment elevation. In general terms, invasive strategies have two main limitations. The first stems from the fact that the severity of a coronary stenosis is not an index of the likelihood of the development of clinical instability or future cardiovascular events. It is now well known that the main pathological mechanism of acute coronary syndromes is destabilisation of the atherosclerotic plaque, which leads to fissuring and the consequent exposure of the subendothelial matrix. The critical factors determining the clinical manifestations and prognosis of these syndromes are the duration and degree of the reduction in coronary flow caused by thrombosis (and its variable associations with superimposed vasoconstriction), the coexistence of collateral flows, and the microvascular embolisation of platelet aggregates and atherothrombotic material. Given the lack of suitable instruments for evaluating the degree of plaque vulnerability and predicting its prothrombotic response, angiographic data alone are insufficient.

The second and more practical limitation concerns the complications of percutaneous revascularisation: in addition to severe complications such as dissection, vascular damage and consequent platelet activation, percutaneous interventions contribute towards the onset of adverse events caused by the occlusion of collateral vessels and the embolisation of atherothrombotic material with varying degrees of microvascular deterioration. Recent studies have found a correlation between the incidence of myocardial necrosis (as indicated by post-intervention enzyme release) and that of long term mortality.

**Results of clinical trials**

It is therefore not surprising that the clinical data published before the introduction of the advances in these fields indicate the potentially negative effects of invasive treatment. The TIMI (thrombolysis in myocardial infarction) IIIB trial involved 1473 patients with unstable angina or non-Q wave acute myocardial infarction, but did not document any clinically meaningful difference in the incidence of the primary end point of death, myocardial infarction, and recurrent inducible ischaemia between the invasively and conservatively treated groups (16.2% vs 18.1%); however, the incidence of rehospitalisation at six weeks was 50% less in the patients who received early invasive treatment.

The OASIS (organisation to assess strategies for ischemic syndromes) registry of 8000 patients with unstable angina or non-Q wave acute myocardial infarction showed that the patients receiving invasive treatment had a lower incidence of refractory angina than those who were conservatively treated; however, although the mortality and reinfarction rates in the countries in which early diagnostic catheterisation and revascularisation were performed more often were similar to those observed in the countries in which it was less frequent, the stroke rate was higher.

The VANQWISH (Veteran Affairs non-Q wave infarction strategies in hospital) trial randomised 920 patients to invasive or conservative treatment within 1–3 days of a non-Q wave myocardial infarction. Cumulative all cause mortality was not significantly different between the two groups during long term follow up (death and non-fatal acute myocardial infarction was 29.9% in the invasive and 26.9% in the conservative arm, p = 0.35); however, the incidence of cardiac death and non-fatal myocardial infarction in the invasively treated group was 2–3 times higher at the time of hospital discharge and after 30 days, and remained higher throughout the first year.

The results of the recent FRISC II (fast revascularisation during instability in coronary artery disease) and TACTICS (treat angina with Aggrastat and determine cost of therapy or conservative strategy)-TIMI 18 trials of a modern antithrombotic treatment regimen added to extensive stenting procedures in the management of acute coronary syndromes without persistent ST segment elevation have now tilted the scales in favour of invasive strategies. In the FRISC II trial, after a period of stabilisation, the incidence of the composite end point of death or myocardial infarction at one year was lower in the 1222 patients assigned to invasive treatment than in the 1235 conservatively treated patients (10.4% vs 14.1%), with a 3.7% absolute and 26% relative risk reduction, although there was a higher rate of periprocedural myocardial infarction in the invasive group possibly because glycoprotein IIb/IIIa inhibitors were not used. Stenting alone has reduced the need for repeat percutaneous revascularisation and the risk of early acute mechanical occlusion by allowing better control over dissections and an increased lumen size, but has not reduced the incidence of acute periprocedural complications.

**Glycoprotein IIb/IIIa inhibitors**

A number of randomised trials have shown that the administration of glycoprotein IIb/IIIa inhibitors can protect against life threatening thrombosis, and that their use during stenting further reduces the adverse effects of revascularisation by synergistically combining the stent related lower incidence of reinterventions with an inhibitor related lower incidence of ischaemic events.

This synergistic benefit can be clearly seen in the results of the TACTICS-TIMI 18 trial, which confirmed the superiority of invasive over conservative strategies in reducing the incidence of the primary end point of death, myocardial infarction, and rehospitalisation at six months (15.9% vs 19.4%). The trial also showed that the use of tirofiban before revascularisation reduced the risk of acute periprocedural complications and improved the safety of the invasive treatment.

Waiting for the pharmacological stabilisation of acute coronary syndrome is no longer advisable. The exciting
prospect for the future is that we will be able to recognise plaques undergoing complications and thus have the possibility of adopting a more directed intervention.

GIANLUCA GONZI
PIERA ANGELICA MERLINI*
DIEGO ARDISSINO
Ospedale Maggiore di Parma,
University of Parma, Parma
*Division of Cardiology,
Ospedale Niguarda Ca’ Granda,
Milan, Italy

Correspondence to: Dr Diego Ardissino, Division of Cardiology, Ospedale Maggiore di Parma, Università degli Studi di Parma, Via Gramsci 14, 43100 Parma; cardiologia.parma@ao.pr.it


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M K DAVIES
A HOLLMAN

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GIANLUCA GONZI, PIERA ANGELICA MERLINI and DIEGO ARDISSINO

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