Non-Q wave myocardial infarction management strategies

See page 2014 for the article to which this Editorial refers

In this issue, Heggunje and colleagues\(^1\) dissect out from the VANQWISH Trial patients with non-Q wave myocardial infarction who had a prior myocardial infarction and compare outcomes to those who did not have a prior myocardial infarction. They evaluated the trial primary end-point of death or myocardial infarction at 1 and 12 months as well as the initial randomized treatment strategy. The bottom line conclusions of their investigation indicate that a prior myocardial infarction identifies a moderately high risk subset of non-Q wave myocardial infarction patients whose outcomes are similar regardless of whether they were randomized to an invasive or non-invasive treatment strategy. Patients who did not have a previous myocardial infarction and are experiencing their first non-Q wave myocardial infarction seem to fare better with a conservative or ischaemia-guided approach during the first post-infarction year.

Several points must be considered when trying to make clinical decisions about management of patients with non-Q wave myocardial infarction.

Firstly, one must recognize that patients with non-Q wave myocardial infarction are not a homogeneous population. Coronary pathology can vary from single vessel coronary disease, e.g. 70% stenosis of the posterior descending coronary artery to multivessel disease with high grade stenoses in all vessels. Ventricular function may also vary from normal to abnormal. ECG changes can be extensive or minimal. Some patients will have indicators of inflammation (C-reactive protein), some will release creatine kinase, troponin I, and others will have no evidence of inflammation, release only troponin I and have normal creatine kinase serum levels. Current data available in the literature suggests that those who have these markers of inflammation and necrosis fare less well than those who do not.

The type of coronary pathology in these patients also plays a role in decision making about which therapy is best for the individual patient. All who evaluate these patients with coronary angiography are not only assessing single, double or triple vessel disease, but rather that a detailed assessment of the coronary pathology is the rule, e.g. percent stenosis, presence or absence of serial stenosis in the same vessel, proximal vs distal stenoses, chronic vs acute stenoses, long vs short stenoses, TIMI grade coronary blood flow, and presence or absence of collaterals.

In the VANQWISH trial as in every other trial comparing medical therapy to interventional therapy, one has to be concerned about entry criteria bias. It is highly unlikely that patients with high grade proximal left anterior descending coronary artery stenoses were randomized to non-revascularization therapy in any clinical trial. Investigator bias generally will exclude these patients and refer them for angioplasty or coronary bypass surgery.

Medical therapy in patients assigned non-interventional strategies may not have been optimum. In the year 2000, cardiologists strongly recommend that patients receive appropriately high dose antianginal drugs, antiplatelet drugs, lipid lowering agents to National Cholesterol Education Program guidelines, angiotensin converting enzyme inhibitors, beta-blockers, and in addition, smoking cessation, blood pressure control to Joint National Committee guidelines, weight reduction, programmed physical exercise and diabetes control as recommended by the American Diabetes Association. Although patients in the VANQWISH trial did have these therapies recommended, they were not protocol driven, nor have they been protocol driven in any clinical trials of which I am aware.

Another point that has to be considered relates to whether angioplasty was performed optimally. In the year 2000, optimal angioplasty generally consists of the use of stents, glycoprotein IIb/IIIa receptor blockers, protocol driven use of other platelet blockers, aspirin, and even possible radiation therapy.

Finally, in those patients assigned to coronary bypass surgery, one needs to be reassured that all patients had complete revascularization, and had as many arterial conduits as possible. It is highly
unlikely that any of these patients had beating heart surgery.

It has always been my position that judgement must be used to make clinical decisions about management strategies in the individual patient. Everyone who has taken care of patients with non-Q wave myocardial infarction will recognize that not every patient needs emergent coronary angiography.

The cardiovascular community needs to recognize that conservative and invasive management should not be considered as competing strategies but rather as complementary approaches to management. Which treatment strategy is appropriate for non-Q wave myocardial infarction patients depends on the severity of symptoms, the extent of the provoked myocardial ischaemia, the coronary artery pathology, the status of left ventricular function, and the presence or absence of co-morbid disease.

Patients who might benefit from early angiography and revascularization are those who have refractory angina despite aggressive maximum medical therapy, including antiplatelet and antithrombotic therapy, patients with known multivessel coronary disease and left ventricular dysfunction, patients with suspicion of left main coronary artery stenosis, and patients who have had previous angioplasty and for whom there is concern about restenosis or patients who had previous coronary bypass surgery in whom there is concern about graft failure.

Detailed information about the ECG, presence or absence of persistent ischaemia, creatine kinase Mb, troponin I, C-reactive protein, left ventricular function and coronary pathology is required in order to make rational clinical decisions about which therapy is appropriate for the individual patient. It makes good sense to be concerned about any patient who has had a myocardial infarction prior to the current non-Q myocardial infarction. In this instance the total amount of infarcted myocardium will generally be greater than if no previous infarction occurred and that in itself is important prognostic information.

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Reference

How hot is inflammation in acute coronary syndrome?

See page 2026 for the article to which this Editorial refers

Platelet aggregation is considered a major causal component in arterial thrombosis. Inhibition of platelet aggregation leads to the acute and chronic protection of patients presenting with atherothrombotic diseases[1]. In the secondary prevention of ischaemic heart disease aspirin, established for more than 100 years as an analgesic and antipyretic, has been known since the 1980s as a most cost-effective antiplatelet agent. Following myocardial infarction, aspirin reduces death and recurrent infarction by 20%. In patients with unstable angina, myocardial infarction can be significantly reduced with aspirin (by 30%) at very low cost and with a high margin of safety. Patients with coronary bypass grafts[2], those undergoing coronary angioplasty[3] and those with stable angina[4] also significantly benefit from aspirin.

Recently, two trials with an oral antiplatelet drug sibraﬁban (platelet glycoprotein IIb/IIIa receptor blocker) for the secondary prevention of coronary artery disease have been presented[5,6]. The overall outcome is that this drug causes harm: not only more bleeding, but more ischaemic events. The possible mechanisms[7] are discussed, but no deﬁnitive answer is provided.

Both Symphony trials give a unique insight into the protective properties of aspirin, since they contained randomly assigned treatment arms without aspirin, unlike all other recent trials evaluating new anti-thrombotic strategies, where all patients received aspirin. In the Symphony trials, mortality at 90 days (Table 1) is significantly increased by 30% in the