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STRATIFICATION OF CHRONIC ISCHEMIC HEART DISEASE DEPENDING ON DIAGNOSIS METHODS AND TREATMENT WAYS

Annotation: *Chronic stable angina in about 50% of all patients is usually caused by occlusion of at least one large epicardial coronary artery by an atheromatous plaque. Angina pectoris results from a mismatch in myocardial oxygen demand, leading to myocardial ischaemia. The indications for coronary revascularisation continue to evolve as scientific and technical advances improve both the results obtained with optimal drug therapy and the methods of revascularisation. A critical issue is the extent to which all forms of therapy are used appropriately based on guidelines and appropriateness criteria, especially with regard to cost and access to care. If symptoms and quality of life do not improve with drug therapy alone, an initial trial of drug therapy with the option to proceed to revascularisation is appropriate. Overall, these results support the recommendations and raise questions about the feasibility of coronary revascularisation, particularly percutaneous coronary intervention.*

Key words: *chronic heart failure, myocardial ischaemia, revascularisation, percutaneous coronary intervention, risk stratification.*

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Chronic stable angina in about 50 per cent of all of all patients is usually caused by occlusion of at least of at least one large epicardial coronary artery by an atheromatous plaque. Angina is caused by a mismatch in myocardial oxygen demand, which resulting in myocardial ischaemia. Angina is characterised by chest discomfort, heaviness or a feeling of pressure that can spread to the jaw, shoulder, back or arm and usually lasts for a few minutes. These symptoms usually occur as a result of physical exertion, emotional stress, cold or heavy meals and are relieved by by rest or nitroglycerin within a few minutes. The main clinical and angiographic predictors of survival in patients with CHD are (1) LV function, (2) the anatomical extent and severity of the coronary atherosclerosis, (3) severity of ischaemia, (4) rate and severity of angina pectoris or the presence of recent plaque rupture, and (5) the patient's general health status and noncoronary comorbidities. Other non-cardiovascular factors that may be determinants of overall mortality, including ethnicity, socioeconomic status, adherence to treatment, depression, and modification of risk factors are not considered in this article, but may nevertheless have a significant impact on prognosis impact on prognosis.

Despite the increasing reliance on noninvasive or invasive testing, history and physical examination are still helpful in assessing the severity of CHD. Pryor et al. [12] defined 11 clinical characteristics - typical angina, previous myocardial infarction, age, gender, duration of symptoms, hypertension, diabetes, hyperlipidaemia, smoking, carotid murmur and frequency of chest pain - and formulated a model using these characteristics to accurately assess the likelihood of severe disease in a patient. An easy-to-use 5-point cardiac risk scale was developed by Hubbard et al. [5] using male gender, typical angina pectoris,

history or electrocardiographic evidence of MI, diabetes and insulin use as risk factors to predict severe CHD at different ages.

Resting electrocardiography is helpful in risk stratification. The prognosis for patients with normal electrocardiography findings is usually excellent because normal electrocardiography findings imply normal LV function [17]. In contrast, such as Q wave abnormalities, ST-T changes, left ventricular hypertrophies, BLNPH, bifascicular block, second and third degree atrioventricular block, atrial fibrillation and ventricular arrhythmias are associated with poor prognosis [5, 18]. Left ventricular function is a major predictor of long-term survival in patients with CHD, and LV end-systolic volume has been shown to be the best indicator of survival after MI. Assessment of LV function, usually by echocardiography, is appropriate in patients with symptoms or signs of heart failure, a history of myocardial infarction or abnormal Q wave on electrocardiography.

Exercise electrocardiography is recommended as the first choice for all patients with moderate to high probability of CHD, except those who cannot exercise or have electrocardiographic abnormalities that make interpretation difficult, or those for whom the information is unlikely to influence treatment. Risk should also be stratified for patients with chronic CHD who have a marked change in the severity of cardiac symptoms by exercise electrocardiography. A useful tool for calculating risk is the Duke treadmill score [6], which includes exercise tolerance, ST segment deviation and angina pectoris as major risk factors. The score is calculated using the following formula: exercise time in minutes - (5 × maximum ST-segment deviation in millimetres) - (4 × angina index [0, no pain; 1, angina; and 2, angina caused by discontinuation of treatment]. Other determinants of risk factors include extensive and prolonged ST-segment depression, transient ST-segment elevation, abnormal heart rate recovery, and delayed systolic blood pressure response to exercise [1, 4, 7].

The increasing value of imaging tests as an initial testing method compared with electrocardiography with load sports, [2, 8, 10] but they are the first choice in patients with electrocardiographic abnormalities that do not allow interpretation of load measurements, or in patients taking digoxin. Imaging studies can provide additional information on the extent, severity and localisation of myocardial danger; assessing the extent of irreversible scar tissue and LV function. Imaging studies with stress imaging are also indicated to assess the functional consequences of coronary artery lesions when planning PCI [3, 4, 5]. Coronary angiography, which helps to differentiate risk in patients based on the extent and localisation of atherosclerosis, is indicated in patients with high-risk criteria for non-invasive testing, patients with angina signs and symptoms of congestive heart failure, patients with sudden cardiac arrest or serious ventricular arrhythmias as the first test in patients with Canadian Society of Cardiology (CCS) class III or IV angina despite drug therapy. Coronary angiography is acceptable in patients with CCS class I or II angina who are intolerant of medications, whose lifestyle is still compromised by these symptoms, who have LV dysfunction, or whose risk status remains uncertain after noninvasive testing. The extent and severity of coronary atherosclerotic disease and LV dysfunction detected by cardiac catheterisation are the most powerful predictors of long-term outcome [12,14,16]. Additional risk stratification is provided by the severity of obstruction and its location, with proximal lesions predicting reduced survival. Quantification of the extent of coronary heart disease, including nonobstructive lesions, also contributes to risk stratification [17].

CT coronary artery calcium scanning is a screening tool that has no role in patients with established CHD. In addition, the specificity of coronary artery calcium assessment for obstructive coronary artery lesions is low. Although CT coronary angiography shows promise for noninvasive detection of obstructive CHD in major epicardial arteries, it is still limited by the large number of false-positive results (up to 50% for severe calcification and coronary stents), specific patient selection (heart rate should be regular and <70 beats/min; the patient should hold his/her breath for 15 seconds), and high-dose irradiation. Magnetic resonance imaging can be used to visualise stress perfusion or visualise wall motion during stress, as well as non-invasive coronary angiography. Most prosthetic heart valves and vascular stents are compatible with MRI; however, MRI cannot be used in the presence of certain

implanted metal objects or medical devices such as pacemakers or implantable cardioverter-defibrillators. However, electronic rhythm management and other cardiovascular devices are currently being developed that may be compatible with MRI [13, 16]. The treatment of CHD has two main goals: to reduce symptoms and ischaemia, and to prevent MI and death. These are governed by different mechanisms: symptoms and ischaemia, inadequate oxygen supply/consumption ratio (usually due to coronary atherosclerosis); MI and death, usually due to unstable rupture of coronary artery plaque. Treatment is critical for all patients with CHD. The first step is to identify and treat any comorbidities that may provoke angina, either by increasing myocardial oxygen demand or by reducing the amount of oxygen delivered to the myocardium (e.g. heart failure, lung disease or anaemia). The second step is to manage CHD risk factors and prevent MI through lifestyle modification and pharmacological treatment. Recognition of the importance of optimal drug therapy (OMT) is changing the management of patients both in patients undergoing coronary revascularisation and those treated conservatively. Optimal drug therapy remains the cornerstone of the management of all patients with CHD because it is logical, relatively inexpensive, and undoubtedly effective in improving long-term outcomes. The challenge is to realise these measures in all patients with CHD [1, 3, 8].

The indications for coronary revascularisation continue to evolve as scientific and technical advances improve both OMT outcomes and revascularisation techniques. A critical issue is the extent to which all forms of therapy are appropriately utilised based on guidelines and feasibility criteria, particularly in relation to cost and access to care [1, 7]. The benefits of coronary revascularisation in reducing the incidence of heart attacks and death have been widely recognised in the context of acute coronary syndromes with ST-segment elevation MI and non-ST-segment elevation MI. However, the benefits of revascularisation therapy for patients with chronic stable angina pectoris with respect to the ‘hard’ endpoints of death and MI are much more controversial. In high-risk patients, even in the setting of chronic stable angina, coronary revascularisation is generally considered beneficial and indeed has been recognised as revolutionary in the treatment of CHD over the past 30 years. Moreover, there is a long history of neutral studies comparing coronary revascularisation with drug therapy in low-risk patients with chronic stable angina. Possible reasons for these neutral results include inadequate sample size and low event rates in this low-risk population. The earliest trials of coronary revascularisation, particularly coronary artery bypass grafting (CABG), compared with drug therapy in patients with chronic stable angina pectoris were conducted in the 1970s and 1980s [9, 11, 12, 13]. Despite significant advances in drug therapy (especially antiaggregant and hypolipidaemic therapy) and surgical techniques, the overall findings of these trials and related registry studies remain relevant today. Symptomatic relief was better with ACS; however, there was no overall difference in survival or absence of MI with ACS compared with medical therapy, except in high-risk patients based on underlying left-sided disease, multivessel lesion plus LV dysfunction, and severe angina. Revascularisation also appears to increase survival in patients with postinfarction angina pectoris.

In a subsequent series of studies conducted in the 1990s and 2000s, revascularisation, particularly percutaneous balloon angioplasty, was compared with drug therapy in patients with stable CHD. The most important information taken from these trials is that balloon angioplasty was associated with further symptomatic relief compared with drug therapy alone, but had no significant effect on the hard endpoints of MI and death, even though crossover from drug therapy to revascularisation was frequent (up to 50%). Subsequent trials comparing drug therapy and PCI with stenting were again neutral, and a recent meta-analysis summarising 20-year trials of PCI in patients with non-acute CHD found no benefit of PCI for death or MI compared with drug therapy [9, 11, 12, 13]. A recent meta-analysis demonstrated a benefit of PCI with respect to mortality, but this analysis had several limitations, the most important of which was the inclusion of patients who had undergone MI. The COURAGE study included 2287 patients with coronary artery stenosis greater than 70% in at least one proximal epicardial coronary artery and evidence of myocardial ischaemia on exercise test or electrocardiography at rest or patients with at least one coronary artery stenosis of at least 80% and classic angina without provocation testing. For the primary outcome, the composite of death and nonfatal MI, no statistical difference was found

between the two groups after a mean follow-up period of 4.6 years. The incidence of angina pectoris was consistently lower in the PCI group than in the medical therapy group during follow-up but was no longer statistically significant after 5 years. The incidence of subsequent revascularisation was also lower in the PCI group. The BARI-2D study, which examined 2368 patients with type 2 diabetes mellitus and CHD, 82% of whom had stable mild to moderate angina and 18% of whom had positive stress test results, further confirmed that there was no significant difference in survival between patients. However, diabetic patients who underwent ACS (but not PCI) compared with drug therapy alone had significantly fewer major cardiac events, mainly due to a reduction in non-fatal MI [15].

Thus, in the absence of symptoms or ischaemia, revascularisation is not indicated because lesions that may be future 'culprits' for subsequent MI or death cannot be identified at present using current techniques. Finding the location of future plaque ruptures or erosions leading to MI (so-called vulnerable plaques) is an important area of cardiovascular research and has the potential to radically change the way IBS is diagnosed and treated. If symptoms and quality of life do not improve with drug therapy alone, an initial trial of drug therapy with the possibility of progression to revascularisation is appropriate.

Overall, these results support the recommendations and raise questions about the feasibility of coronary revascularisation, particularly PCI.

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