Management of coronary artery disease

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Abstract. Coronary Artery Disease (CAD) is associated with significant morbidity and mortality, therefore it's important to early and accurate detection and appropriate management. Diagnosis of CAD include clinical examination, noninvasive techniques such as biochemical testing, a resting ECG, possibly ambulatory ECG monitoring, resting echocardiography, chest X-ray in selected patients; and catheterization. Managements of CAD patients include lifestyle modification, control of CAD risk factors, pharmacologic therapy, and patient education. Revascularization consists of percutaneous coronary angioplasty and coronary artery bypass grafting. Cardiac rehabilitation should be considered in all patients with CAD. This comprehensive review highlights strategies of management in patients with CAD.

1. Introduction

Coronary artery disease is the most common cause of death in cardiovascular disease. The rate of morbidity and mortality is high, the costs incurred for the treatment process are also very high, thus giving a bad impact on the welfare and quality of life both in patients, families, and health costs borne by the state. The proper management can reduce the number of losses. Therefore, here will be discussed about the appropriate treatment steps to deal with this coronary artery disease.

2. Definition

Stable coronary artery disease (SCAD) is generally characterized by episodesof reversible myocardial demand/supply mismatch, related to ischemia or hypoxia, which are usually inducible by exercise, emotion or other stress and reproducible but, which may also be occurring spontaneously.[1] Coronary artery disease most commonly caused by the inability of atherosclerotic coronary arteries to perfuse the heart due to partial or total occlusion of the coronary arteries.[2] SCAD also includes the stabilized, often asymptomatic, phases that follow an ACS.

SCAD has various clinical presentations that are associated with different underlying mechanisms that mainly include: plaque-related obstruction of epicardial arteries, focal or diffuse spasm of normal or plaque-diseased arteries, microvascular dysfunction and left ventricular dysfunction caused by prior acute myocardial necrosis and/or hibernation.[1]

3. Epidemiology

Angina pectoris is more prevalent in middle-aged women than men, probably due to the high prevalence of functional coronary artery disease such as microvascular angina in women. In contrast, angina pectoris is more prevalent in elderly men.[3,4]

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4. Natural History and Prognosis

Conventional risk factors for the development of CAD are hypertension, hypercholesterolemia, diabetes, sedentary lifestyle, obesity, smoking, and family history have an adverse influence on prognosis in those with established disease.[1]

Prognostic assessment is an important part of the management of patients with SCAD. On the one hand, it is important to reliably identify those patients with more severe forms of disease, who may have an improvement in outcome with more aggressive investigation and maybe intervention, including revascularization. On the other hand, it is also important to identify those patients with a less severe form of disease and a good prognosis, thereby avoiding unnecessary invasive and non-invasive tests and revascularization procedures. The prognosis in patients with SCAD is relatively benign with estimates of annual mortality rates in mixed population ranging from 1.2-2.4% with an annual incidence of cardiac death between 0.6 and 1.4%.[5-10]

5. Pathophysiology

Myocardial ischemia occurs when myocardial oxygen delivery cannot meet metabolic myocardial demand. Although this term is too simple, myocardial oxygen delivery largely determined by the carrying capacity of blood oxygen and coronary flow. In normal coronary arteries, coronary blood flow can increase three to five fold. This increase, termed coronary flow reserve, occurs mostly through decreased resistance in coronary microcirculation. Significant atherosclerotic plaquing in the epicardial coronary artery (> 75% cross-sectional region) results in a decrease in blood pressure throughout the stenotic lesions. Coronary arterioles dilate to compensate for the decrease in distal perfusion pressure, keeping coronary blood flow normal. Consequently, during exercise, however, coronary capacity the arterioles to widen further are finite, and the demand for myocardial oxygen immediately exceeds supply, resulting in ischemia, followed usually by angina.

6. Diagnosis and Assessment

Diagnosis and assesment for SCAD includes clinical evaluation and identification for risk factor and additional investigation such as stress testing or coronary imaging to confirm the diagnosis of SCAD. Angina Pectoris can be classified into typical and atypical angina pectoris which definitions are summarized in table 1 below.

Table 1. Traditional clinical classification of chest pain[1].

Typical angina (definite)	Meets all three of the following characteristics: • substernal chest discomfort of characteristic quality and duration; • provoked by exertion or emotional stress; • relieved by rest and/or nitrates within minutes.
Atypical angina (probable)	Meets two of these characteristics.
Non-anginal chest pain	Lacks or meets only one or none of the characteristics.

The Canadian Cardiovascular Society classification which is widely used as a grading system for stable angina are divided into 4 class, angina pain at rest may occur in all grades of this classification as a manifestation of associated and superimposed coronary vasospasm. The class assigned is indicative of the maximum limitation while the patient may do better when the patient is healthy.

Non-invasive cardiac investigation include standard laboratory biochemical testing, a resting ECG, possibly ambulatory ECG monitoring, resting echocardiography and in selected patients a chest X-ray.

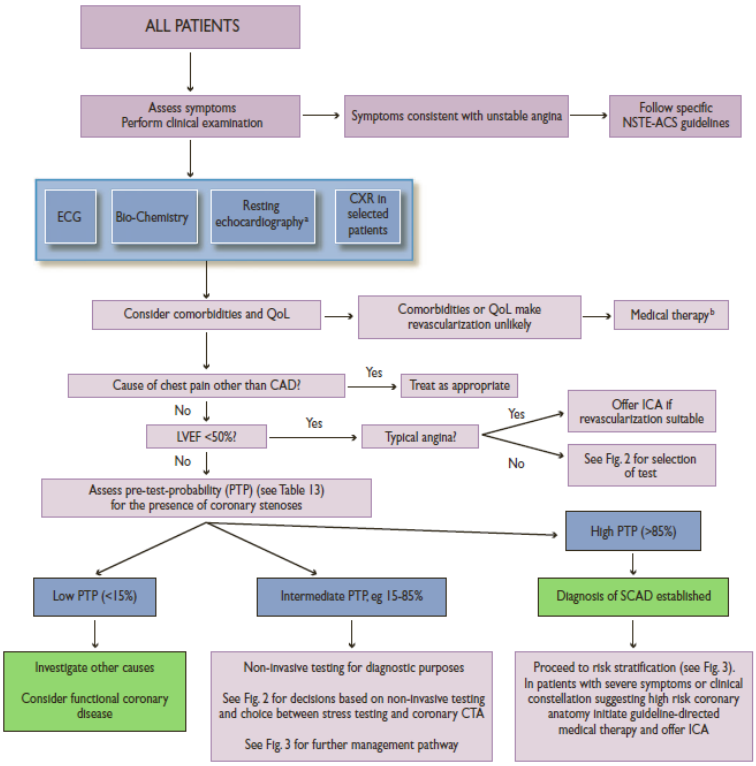


Figure 1. Initial diagnostic management of patients with suspected SCAD [1].

7. Lifestyle and Pharmacological Management

The aim of the management of SCAD is to reduce symptoms and improve prognosis. The management of CAD patients encompasses lifestyle modification, control of CAD risk factors, evidence-based pharmacological therapy and patient education.[11,12] Lifestyle recommendations include smoking cessation, a healthy diet, regular physical activity, weight and lipid management, also blood pressure and glucose control.[1]

Smoking is a strong and independent risk factor for CVD and all smoking must be avoided, smoking cessationis associated with a reduction in mortality of 36% after MI.[13,14] Healthy diet reduces CVD risk with The target of BMI is <25 kg/m^2, recommended dieat intake list in table 2 below.

Regular physical activity is associated with a decrease in CV morbidity and mortality in patients with established CAD. Patients with previous acute MI, CABG, percutaneous coronary intervention (PCI), stable angina pectoris or stable chronic heart failure should undergo moderate-to-vigorous intensity aerobic exercise training ≥3 times a week and for 30 min per session.[1]

Table 2. Recommended diet intake [1].

• Saturated fatty acids to account for <10% of total energy intake, through replacement by polyunsaturated fatty acids.
• Trans unsaturated fatty acids <1% of total energy intake.
• <5 g of salt per day.
• 30–45 g of fibre per day, from wholegrain products, fruits and vegetables.
• 200 g of fruit per day (2–3 servings).
• 200 g of vegetables per day (2–3 servings).
• Fish at least twice a week, one being oily fish.
• Consumption of alcoholic beverages should be limited to 2 glasses per day (20 g/day of alcohol) for men and 1 glass per day (10 g/day of alcohol) for non-pregnant women.

Dyslipidemia should be managed according to lipid guidelines with pharmacological and lifestyle intervention. The goals of treatment are LDL-C below 1.8 mmol/L (<70 mg/dL) or <50% LDL-C reduction when target level cannot be reached.[1]

Diabetes mellitus is a strong risk factor for CV complications, increases the risk of progression of coronary disease and should be managed carefully, with good control of glycated hemoglobin (HbA1c) to <7.0% (53 mmol/mol) generally and <6.5%–6.9% (48–52 mmol/mol) on an individual basis.[1]

There is sufficient evidence to recommend that systolic BP (SBP) be lowered to<140 mmHg and diastolic BP (DBP) to<90 mmHg in SCAD patients with hypertension. Based on current data, it may be prudent to recommend lowering SBP/DBP to values within therange 130–139/80–85 mmHg. BP targets in diabetes are recommended to be <140/85 mmHg.[1]

8. Pharmacological Management

The two aims of pharmacological management of stable CAD patients are to obtain relief of symptoms and to prevent CV events.[1]

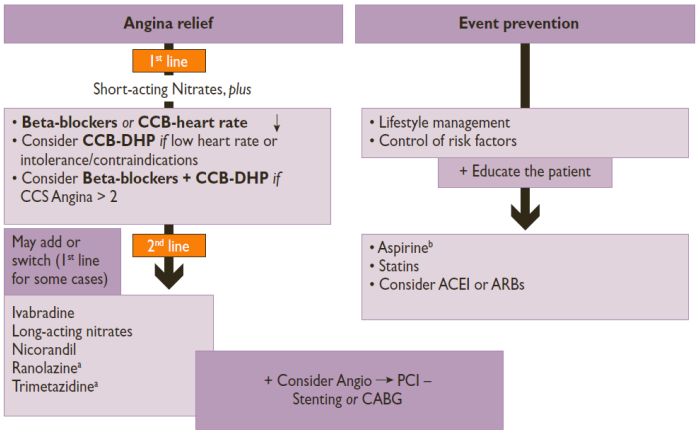


Figure 2. Medical management of patients with stable coronary artery disease. ACEI (angiotensin converting enzyme inhibitor); CABG (coronary artery bypass graft); CCB (calcium channel blockers); CCS (Canadian Cardiovascular Society); DHP (dihydropyridine); PCI (percutaneous coronary intervention). [a]Data for diabetics. [b]if intolerance, consider clopidogrel [12].

9. Revascularization

Advance in techniques, equipment, stent and adjuvant therapy have established PCI as a routine and safe procedure in patients with SCAD and suitable coronary anatomy. The decision to revascularize a

patient should be based on the presence of significant obstructive coronary artery stenosis, the amount of related ischaemia, and the expected benefit on prognosis and/ or symptom. Revascularization can also be considered as first-line treatment in the following situation: post-myocardial infarction angina/ischaemia, left ventricular dysfunction, multivessel disease and/or large ischaemic territory, left main stenosis.

The indications for PCI and CABG in SCAD patients have clearly been defined by the recent recommendations on myocardial revascularization. Following figures show algorithms to help simplify the decision making process.

After revascularization, therapy and secondary prevention should be initiated during hospitalization, all revascularized patients receive a secondary prevention and be scheduled for follow-up visit, antiplatelet therapy that must be given is aspirin, for BMS DAPT is indicated at least 1 month, and 6 to 12 months after 2nd generation DES. In symptomatic patients, stress imaging is indicated rather than stress ECG, if low risk (<5% ischemic myocardium) optimal medical therapy is recommended, but if high risk (>10%) coronary angiography is recommended. Systematic control angiography, early or late after PCI is not recommended.

Table 3. Indications for revascularization of stable coronary artery disease patients on optimal medical therapy (adapted from ESC/EACTS 2010 Guidelines) [12].

Indication [a]	To improve prognosis:		To improve symptoms persistent on OMT:		
	Class [d]	Level [e]	Class [d]	Level [e]	Ref. [f]
A Heart Team approach to revascularization is recommended in patients with unprotected left main, 2–3 vessel disease, diabetes or comorbidities.	I	C	I	C	172, 426–428
Left main >50% diameter stenosis[b].	I	A	I	A	172
Any proximal LAD >50% diameter stenosis[b].	I	A	I	A	172
2–3 vessel disease with impaired LV function / CHF.	I	B	IIa	B	172
Single remaining vessel (>50% diameter stenosis[b]).	I	C	I	A	172
Proven large area of ischaemia (>10% LV[c])	I	B	I	B	172
Any significant stenosis with limiting symptoms or symptoms non responsive/intolerant to OMT.	NA	NA	I	A	172
Dyspnoea/cardiac heart failure with >10% ischaemia/viability[c] supplied by stenosis >50%.	IIb	B[429, 430]	IIa	B	172
No limiting symptoms with OMT in vessel other than left main or proximal LAD or single remaining vessel or vessel subtending area of ischaemia <10% of myocardium or with FFR ≥0.80.	III	A	III	C	23, 25, 172, 400

Recommendation for the type of revascularization (CABG or PCI) in patients with SCAD with suitable coronary anatomy for both procedures and low predicted surgical mortality

Recommendations according to extent of CAD	CABG		PCI		
	Class[a]	Level[b]	Class[a]	Level[b]	Ref[c]
One or two-vessel disease without proximal LAD stenosis.	IIb	C	I	C	
One-vessel disease with proximal LAD stenosis.	I	A	I	A	107,108,160, 161,178,179
Two-vessel disease with proximal LAD stenosis.	I	B	I	C	108,135,137
Left main disease with a SYNTAX score ≤ 22.	I	B	I	B	17,134,170
Left main disease with a SYNTAX score 23–32.	I	B	IIa	B	17
Left main disease with a SYNTAX score >32.	I	B	III	B	17
Three-vessel disease with a SYNTAX score ≤ 22.	I	A	I	B	17,157,175,176
Three-vessel disease with a SYNTAX score 23–32.	I	A	III	B	17,157,175,176
Three-vessel disease with a SYNTAX score >32.	I	A	III	B	17,157,175,176

Figure 3. Percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG) in stable coronary artery disease without left main coronary artery involvement [12].

10.Cardiac rehabilitation

A comprehensive risk reduction regimen integrated into comprehensive cardiac rehabilitation is recommended for patients with CAD.[1] Cardiac rehabilitation should be considered in all patients with CAD, including those with chronic angina. Cardiac-based rehabilitation exercises are effective in reducing total mortality and CV and hospital admissions. The evidence also shows beneficial benefits on health-related quality of life (QoL). In the selected subgroup, cardiac center-based rehabilitation may be substituted for home-based rehabilitation, which is not inferior. The participation of patients in cardiac rehabilitation remains too low, especially in women, elderly and socioeconomic loss, and may benefit from systematic referral.[13]

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