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Managing stable angina in primary care

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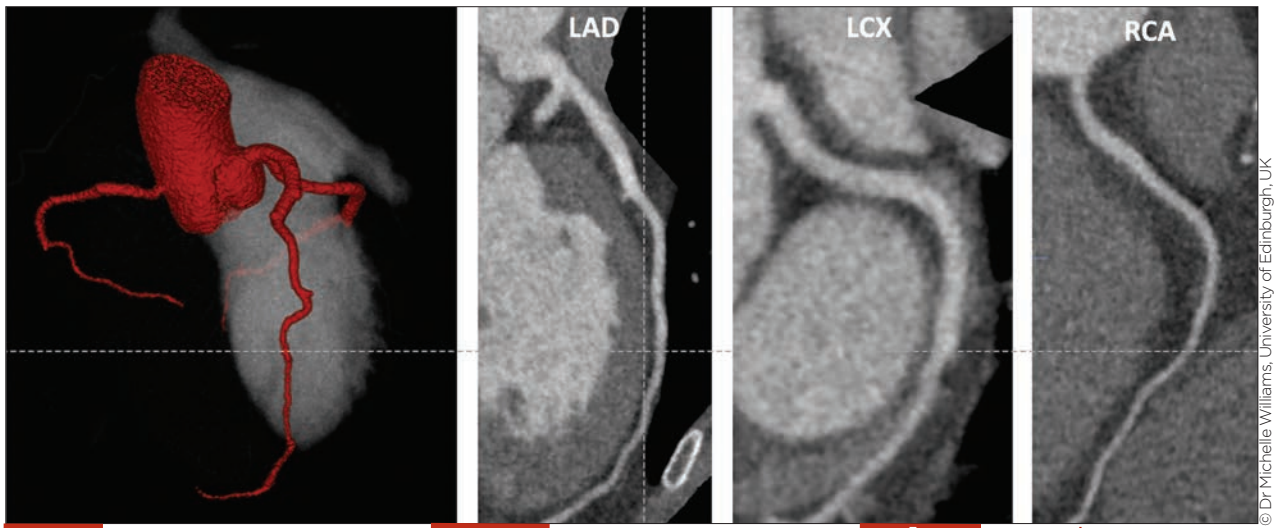

FIGURE 1
CTCA images showing normal coronary arteries in a 70-year-old man referred with suspected stable angina

LAD = left anterior descending artery; Lcx = left circumflex artery; RCA = right coronary artery

How should patients be assessed?

STABLE ANGINA IS ESTIMATED TO AFFECT MORE THAN 1.3 MILLION PEOPLE IN THE UK WITH almost 100,000 new cases diagnosed each year.[1] The prevalence of angina increases sharply with age. Around 50% of people diagnosed with ischaemic heart disease (IHD) present with stable angina as the first symptom.[1]

Patients with stable angina typically describe a predictable tightness or constriction of the chest brought on by physical exertion or emotional stress that may radiate to the neck and jaw, upper limbs, or less commonly to the back. It occurs as a result of myocardial ischaemia, most commonly due to atherosclerotic coronary artery disease (CAD). Although angina may also occur where there is an imbalance between myocardial oxygen supply and demand, e.g. hypertrophic cardiomyopathy or severe aortic stenosis, this article will focus mainly on the diagnosis and management of stable angina due to CAD.

Table 1

Chest pain characteristics to guide diagnosis of angina[2]

- A constricting discomfort in the front of the chest, or in the neck, shoulders, jaw, or arms
- Precipitated by physical exertion
- Relieved by rest or GTN within about five minutes

The presence of all three features is defined as typical angina, two of the three features as atypical angina and one or none of the features as non-anginal chest pain

What investigations should be carried out?

INITIAL ASSESSMENT

In many cases the history alone is sufficient to reach a diagnosis of stable angina.[2,3,4] NICE recommends the classification of chest pain into typical angina, atypical angina or non-anginal chest pain based on the presence of key features of the pain, to aid diagnosis and guide investigation and management, see table 1, left.[2]

The presence of all three features suggests a diagnosis of typical angina. The presence of two features is considered atypical angina and where one or none of the features are present the pain may be defined as non-anginal.[2] Stable angina is unlikely if the chest pain is continuous or prolonged, unrelated to exertion, is brought on by movement or inspiration, or is associated with other symptoms such as palpitations, dizziness, tingling or difficulty swallowing. Angina symptoms may be influenced by a number of factors including comorbidity (e.g. diabetes mellitus) and gender.

Cardiovascular risk factors

Along with a detailed assessment of the presenting symptoms and prior history of cardiovascular disease, risk factors which predispose to CAD should be evaluated, see table 2, p18. The likelihood of a diagnosis of angina increases with the number of cardiovascular risk factors present.

Patients with angina who smoke should be strongly advised to stop. Education, self-help materials, counselling and pharmacological support may be offered where appropriate to patients wishing to cease smoking. Blood pressure should be recorded and hypertension, if present, managed according to current UK guidelines.[5] Patients should be offered healthy eating advice, undertake an appropriate aerobic exercise regimen and, if obese, lose weight with a target BMI < 25 kg/m^2.

Baseline investigations

Patients presenting with a new diagnosis of suspected stable angina should have a number of blood tests performed at baseline, see table 3, p18, »

What are the management options?

and be considered for referral to secondary care. Specialist referral should also be considered for patients with known stable angina and a deterioration in symptoms, or those with limiting symptoms despite optimal drug therapy.

A resting 12-lead ECG is recommended in all patients with suspected angina. A normal result does not exclude the presence of CAD. An abnormal resting ECG increases the likelihood that the patient has underlying CAD and may also identify other conditions such as atrial fibrillation or structural heart disease.

Patients deemed to have non-cardiac chest pain should be reassured of this at an early stage and alternative diagnoses considered.[2,4] This reduces anxiety and distress, and prevents unnecessary hospital admissions and consultations.

FURTHER INVESTIGATION

Further investigation may be undertaken to confirm the presence of CAD, evaluate the burden of myocardial ischaemia, stratify future cardiac risk and identify those who may benefit from coronary revascularisation.

The choice of investigation will be determined by clinical presentation, pre-test probability, prior history of CAD, and local availability of these investigations, see table 4, p19.[2,3,4]

In general, anatomical investigations determine the presence of underlying CAD and identify individuals with a pattern of CAD that may benefit from revascularisation, while functional tests evaluate the burden of myocardial ischaemia and facilitate risk stratification.

Traditionally the exercise tolerance test (ETT) has played a key role in the investigation of patients with suspected stable angina. However, the sensitivity and specificity of the ETT in patients with stable angina is dependent on the pretest probability and limits its utility as a diagnostic tool.[2,4] A normal ETT may reassure many patients but it does not exclude a diagnosis of underlying CAD. In patients with an established diagnosis of CAD, the ETT remains a useful tool to assess ischaemic burden and prognosis.[4]

CT coronary angiography (CTCA) is a non-invasive procedure that evaluates the presence, location and severity of underlying CAD and has emerged as a valuable tool for the investigation of suspected angina, see figure 1, p17.

In a recent meta-analysis performed by NICE, the diagnostic accuracy of CTCA (sensitivity 96% and specificity 79%) was superior to contemporary functional or invasive testing in the detection of significant CAD. Taken together with limited cost-effectiveness data, NICE currently recommends CTCA as a first-line investigation in patients with a new diagnosis of suspected stable angina.[2] CTCA is particularly useful as a rule-out test where the index of suspicion of underlying CAD is low. The diagnostic accuracy of CTCA is reduced in patients with a high pretest probability, largely due to the presence of coronary calcification. In Scotland, SIGN recommends CTCA as a first-line investigation where a diagnosis of stable angina is suspected but not clear from the history alone.[4]

Importantly, there is now evidence that the use of CTCA to evaluate patients with suspected angina improves clinical outcomes. In a randomised trial of more than 4,000 patients referred to hospital with a suspected diagnosis of angina, the addition of CTCA to standard care increased diagnostic accuracy and permitted targeting of treatments and interventions to those patients who gained most benefit.[6] The use of CTCA reduced the need for further stress testing.[6] Although a short-term increase in the use of invasive coronary angiography was observed with CTCA, after five years' follow-up rates were similar between both cohorts.[7] Ultimately, this led to a 40% reduction in the risk of death from coronary heart disease or nonfatal myocardial infarction (MI) at 5 years; 2.3% vs. 3.9%, HR 0.59 (95% CI: 0.41-0.84; P = 0.004).[7]

Table 2

Common cardiovascular risk factors

- Age
- Male sex
- Ethnicity
- Family history of premature cardiovascular disease
- Hypertension
- Diabetes mellitus
- Hyperlipidaemia
- Presence of atherosclerotic disease in another vascular bed
- Previous history of ischaemic heart disease
- Chronic kidney disease
- Cigarette smoking
- Abdominal obesity
- Increased alcohol intake
- Sedentary lifestyle

Table 3

Baseline investigations in suspected angina

- Haemoglobin
- Creatinine or estimated glomerular filtration rate (eGFR)
- Fasting plasma glucose or HbA_{1C}
- Lipid profile
- Thyroid function

DRUG THERAPY

The objectives of pharmacological therapy in patients with stable angina are to prevent future vascular events and to alleviate symptoms of angina. It is important to explain to patients the diagnosis, why their medications have been prescribed and how to take them, as well as possible adverse effects and how to manage them.

Preventative therapy

Antiplatelet agents: There is clear evidence that aspirin reduces the risk of ischaemic events in patients with established CAD.[8] Aspirin should be prescribed, where possible, at a daily dose of 75-150 mg in all patients with stable angina. A recent meta-analysis has suggested that, at least for primary prevention, adjusting the daily aspirin dose according to the patient's bodyweight may be more effective at preventing cardiovascular events and avoiding unwanted side effects.[9] This hypothesis remains to be tested in a prospective randomised controlled trial in patients with stable CAD.

Clopidogrel may be considered as an alternative for patients with stable angina who are unable to take aspirin. In large-scale randomised trials in patients with stable atherosclerotic vascular disease, clopidogrel was marginally more effective than aspirin at reducing the combined endpoint of MI, ischaemic stroke or vascular death, while the addition of clopidogrel to long-term aspirin therapy did not reduce recurrent cardiac events.

Antiplatelet agents should be avoided in patients with active peptic ulceration or bleeding and used with caution in patients with bleeding disorders or a history of peptic ulcer disease.

Lipid-lowering therapy: There are robust data demonstrating that statins reduce the relative risk of death and MI in patients with IHD by as much as 40%, the derived clinical benefit being related to the reduction in LDL cholesterol.

NICE currently recommends initiating treatment with atorvastatin 80 mg once daily for the prevention of cardiovascular events in patients with stable angina.[10] A lower initial dose should be considered if there are potential drug interactions, a high risk of adverse effects or in light of patient preference.[10] The target for treatment is a 40% reduction in non HDL cholesterol.[10] If this is not achieved with initial treatment, review adherence to medication and lifestyle measures, and consider increasing the dose or switching to a more potent statin.

Although marked reductions in LDL cholesterol level may also be achieved by combining statin therapy and ezetimibe, a drug which reduces intestinal resorption of cholesterol, evidence of a clinical benefit for this combination in patients with stable CAD is still awaited.

Statins are contraindicated in active liver disease. A rare but significant side effect of statin therapy is reversible myositis, which is more common when a statin is used in combination with a fibrate or drugs that inhibit the hepatic enzyme cytochrome P450.

For patients intolerant of statin therapy, fibrates may offer a less effective alternative. Fibrates have a side-effect profile similar to that of statins, share a similar risk of myositis and should be avoided in moderate to severe renal impairment.

ACE inhibitors: The anti-ischaemic and mortality benefits of chronic ACE inhibition have been well documented in patients following MI with significant left ventricular dysfunction and in patients with diabetes mellitus.

Treatment with an ACE inhibitor should be considered for patients with stable angina where left ventricular dysfunction or diabetes mellitus are present.[3]

Anti-anginal therapy

There are currently six major drug classes available in the UK for the treatment of stable angina: beta-blockers, calcium channel blockers (CCBs), nitrates, potassium channel activators, sinus node inhibitors and late sodium current inhibitors. Ideally, the dose of a single anti-anginal agent should be optimised before introducing combined therapy. There is little evidence to support the use of three anti-anginal drugs in combination. Patients with angina that is not adequately controlled with two agents should be considered for specialist referral. The major contraindications »

Table 4

Investigations in patients with suspected stable angina

Anatomical	Imaging modality	Advantages	Disadvantages
CT coronary angiography	Ionising radiation	● High sensitivity and specificity for diagnosis of CAD ● Rapid ● Well tolerated ● Feasible on most contemporary CT scanners	● Radiation ● Incidental findings ● Limited functional information outside a research setting ● Image quality dependent on patient factors
Invasive coronary angiography	Ionising radiation	● Considered gold standard for diagnosis of CAD ● Can be combined with invasive FFR assessment to obtain accurate functional information	● Radiation ● Invasive (small risk of CVA, MI and bleeding)
Functional			
Exercise tolerance test	Surface ECG	● Inexpensive ● Readily available ● Well validated for prognosis ● No radiation	● Poor sensitivity and specificity for diagnosis of CAD ● Dependent on the patient's exercise ability
Stress echocardiography	Ultrasound	● Uses existing resources ● Readily available ● Well validated ● Permits assessment of cardiac function, viability, valves and extra-cardiac structures	● Imaging quality dependent on patient factors ● Dependent on the patient's exercise ability (dobutamine can be used) ● Modest sensitivity and specificity for diagnosis of CAD
Myocardial perfusion scintigraphy	Ionising radiation	● Well validated for prognosis	● Radiation ● Modest sensitivity and specificity for diagnosis of CAD ● Dependent on the patient's exercise ability (adenosine or dobutamine can be used) ● High false-negative rate in balanced ischaemia
Stress perfusion CMR	Nonionising (magnetic field and radio waves)	● High sensitivity and specificity for diagnosis of CAD ● Provides detailed assessment of cardiac structure and function	● Expensive and resource dependent ● Time consuming ● Some patients have poor tolerance

Key: CAD = coronary artery disease; CMR = cardiac magnetic resonance; CT = computerised tomography; CVA = cerebrovascular accident; FFR = fractional flow reserve; MI = myocardial infarction

and cautions to anti-anginal therapy can be found in table 5, below.

Beta-blockers: There are substantial data to recommend that beta-blockers be considered as first-line therapy for symptomatic relief in patients with stable angina.[2,3,4] Evidence of a prognostic benefit in stable angina is limited, with only a single observational study reporting a mortality benefit in patients with stable CAD without previous MI or heart failure. Beta-blocker therapy should be titrated on an individual basis to the maximum tolerated dose required to control symptoms or a target resting heart rate of 55-60 beats per minute, whichever occurs first. Sudden withdrawal of beta blockade may cause a rebound tachycardia and hypertension, exacerbating angina, and should be avoided.

Absolute contraindications to beta-blocker use include severe bradycardia, second- or third-degree atrioventricular block, sick sinus syndrome, decompensated heart failure and severe asthma. Diabetes mellitus, chronic obstructive pulmonary disease and peripheral vascular disease are not contraindications.

Calcium channel blockers: The dihydropyridines (nifedipine, amlodipine and felodipine) act primarily on vascular smooth muscle to cause vasodilatation, while verapamil and diltiazem act preferentially on cardiac tissue to reduce

Table 5

Contraindications and cautions with antianginal therapy

Drug class	Specific agent	Contraindication	Caution
Antiplatelet agents	Aspirin Clopidogrel	Active peptic ulcer disease (PUD) Active bleeding	Bleeding disorders; previous PUD Bleeding disorders; liver disease
Lipid-lowering therapy	Statins Fibrates	Acute or severe liver disease* Severe liver disease	Previous liver disease Renal impairment
ACE inhibitors		ACE inhibitor hypersensitivity; aortic stenosis; LV outflow tract obstruction; severe renal impairment; renal artery stenosis	Mild-moderate renal impairment; previous angioedema
Beta-blockers		Asthma; severe bradycardia; preexisting high-degree AV block; sick sinus syndrome; severe uncontrolled heart failure	First-degree AV block; severe peripheral vascular disease; coronary artery spasm
Calcium channel blockers	Cardiac specific (e.g. diltiazem/verapamil) Vasodilators (e.g. amlodipine)	Severe bradycardia; preexisting high-degree AV block; sick sinus syndrome; heart failure LV outflow tract obstruction	First-degree AV block; concomitant beta-blocker use (verapamil); severe aortic stenosis (verapamil)
Nitrates		Aortic or mitral stenosis; LV outflow tract obstruction; PDE-5 inhibitor use ≤ 24hr	
Potassium channel activator	Nicorandil	Cardiogenic shock; PDE-5 inhibitor use ≤ 24hr	Acute left ventricular failure
Sinus node inhibitor	Ivabradine	Heart rate < 70 bpm (heart rate on treatment < 50 bpm); sick sinus syndrome; preexisting high-degree AV block; acute myocardial infarction; severe hepatic insufficiency	Poorly controlled, severely limiting angina
Late sodium current inhibitor	Ranolazine	Concomitant use with potent inhibitors of cytochrome P450 3A4; moderate to severe hepatic impairment; severe renal impairment	Concomitant use with drugs that prolong QT interval; concomitant use with weak or moderate inhibitors or inducers of cytochrome P450 3A4; mild hepatic impairment; moderate renal impairment; congestive cardiac failure

*Serum concentrations of liver enzymes should be checked before, and within 1–3 months of, commencing statin therapy; treatment should be discontinued if serum transaminases rise to greater than 3 times the upper limit of the reference range

heart rate and myocardial contractility.

CCBs relieve symptoms of angina and improve exercise time as effectively as beta-blockers without conferring any prognostic benefit.[4] The choice of CCB will depend on comorbidity and drug interactions.

Rate-limiting CCBs (verapamil and diltiazem) are recommended as first line for symptomatic relief in patients with stable angina in whom beta-blockade is contraindicated or not tolerated.[3,4] Dihydropyridine CCBs should be considered for second-line therapy where breakthrough symptoms occur despite optimal beta-blockade.[3,4]

The dihydropyridine CCBs may also be effective in patients with Prinzmetal angina, a rare form of angina caused by coronary artery spasm that typically occurs at rest.

Rate-limiting CCBs are contraindicated in patients with heart failure, severe bradycardia or second- or third-degree atrioventricular block and should be used with extreme caution in combination with beta-blocker therapy. The dihydropyridine CCBs, amlodipine or felodipine, may be used safely in patients with angina and heart failure.

There are concerns about the safety of short-acting preparations of nifedipine in the treatment of angina, particularly in the setting of unstable coronary syndromes, and these should be avoided. Long-acting preparations of nifedipine may be used safely in the treatment of angina.

Nitrate therapy: In patients with stable angina, nitrates improve exercise tolerance and time to onset of ST depression during exercise testing.

Sublingual nitrates provide rapid relief of anginal symptoms, can be used prophylactically to prevent ischaemic symptoms during planned exercise, and should be offered to all patients.[3,4]

A meta-analysis comparing long-acting nitrates, beta-blockers and CCBs found no significant difference in efficacy in patients with stable angina. Long-acting nitrates should be considered as first-line therapy for patients in whom beta-blockers or CCBs are contraindicated, or as second-line therapy for breakthrough symptoms.

Tolerance is the major limitation to the use of nitrates and can be avoided by ensuring a daily 'nitrate-free period' of six to eight hours, either through twice daily eccentric (less than 12 hours apart) dosing or a long-acting, once daily preparation. Patients should be aware that they may experience an increase in symptoms of angina during the nitrate-free period and the timing of dosing should be chosen accordingly. The predominant side effect of nitrate preparations is self-liming headache which usually eases with regular use. Nitrates and phosphodiesterase inhibitors such as sildenafil should not be used within 24 hours of each other because of the risk of severe hypotension.

Potassium channel activators: Nicorandil acts primarily on vascular smooth muscle to induce coronary vasodilatation. Clinical data suggest that nicorandil and CCBs are equally effective at relieving angina. In a randomised trial of more than 5,000 patients with stable angina, nicorandil significantly reduced the primary composite endpoint of combined all cause mortality, cardiovascular events and unplanned hospitalisation.[11]

Nicorandil is contraindicated in cardiogenic shock and its major limiting side effects are hypotension and gastrointestinal (GI) ulceration that can occur at any point in the GI tract. In light of this side effect, the MHRA has recommended that nicorandil use be restricted to second-line therapy, behind beta-blocker and CCB therapy.[12]

Sinus node inhibitors: Ivabradine at recommended doses leads to a reduction in heart rate by around 10 beats per minute. The actions of ivabradine are restricted to sinus node tissue with no effects on cardiac conducting tissue in other areas, myocardial contractility or ventricular repolarisation. Patients must be in sinus rhythm for ivabradine to be effective.

In randomised, controlled trials in patients with chronic stable angina, ivabradine increased time to angina onset during exercise compared with placebo and demonstrated comparable efficacy to atenolol or amlodipine at improving exercise duration and reducing frequency of angina attacks. When used in addition to amlodipine in patients with angina, ivabradine improved exercise parameters compared with placebo.

In contrast to patients with heart failure, the use of ivabradine in stable angina does not confer any cardioprotective benefit and concerns have been raised about its use in poorly controlled angina. In a randomised trial enrolling more than 19,000 patients with stable CAD without heart failure, the addition of ivabradine to contemporary medical therapy had no effect on all cause mortality, cardiac death or non-fatal MI.[13] A prespecified subgroup analysis of patients with limiting angina, reported a small but significant increase in the combined risk of cardiovascular death or non-fatal MI with ivabradine use.[13]

Ivabradine is currently recommended as second-line treatment for patients with stable angina who are either intolerant of beta-blockade or in whom a rate-limiting CCB fails to achieve adequate heart rate control, and in combination with a beta-blocker in patients whose heart rate is inadequately controlled with an optimal beta-blocker dose.[3,4]

Ivabradine should not be initiated in patients with stable angina if the resting heart rate is < 70 beats per minute and should be withdrawn or the dose reduced if the heart rate on treatment falls below 50 beats per minute. Additional contraindications include evidence of significant conducting system disease (sick sinus syndrome, second- or third-degree heart block), severe hepatic insufficiency and acute MI. The major side effects include transient headache, dizziness and luminous phenomena. Patients with luminous phenomena typically describe a transient enhanced brightness in a limited area of the visual field. Fewer than 1 per cent of patients discontinue therapy as a result.

Late sodium current inhibitors:[3,4] Ranolazine inhibits the late inward sodium current in cardiac myocytes. When used in combination with existing antianginal therapy, ranolazine increased exercise time and reduced the frequency of anginal attacks but did not affect cardiovascular outcomes (all cause mortality, cardiac death, non-fatal MI).[14] Evidence of symptomatic benefit with ranolazine monotherapy is lacking.

Although currently licensed in the UK as adjunctive therapy in patients with stable angina, the Scottish Medicines Consortium has advised that ranolazine is not recommended for use in Scotland.

The major side effects with ranolazine are dizziness and GI, including nausea and constipation. It is metabolised predominantly through the cytochrome P450 system (3A4 and 2D6 enzymes) and is contraindicated in combination with other potent CYP3A4 inhibitors such as clarithromycin and grapefruit juice. In addition, ranolazine is known to increase the QT interval and care should be taken when used in combination with drugs known to prolong the QT interval. Ranolazine should be avoided in patients with moderate or severe hepatic impairment, severe renal impairment and in older people. »

key points

SELECTED BY

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Stable angina is estimated to affect more than 1.3 million people in the UK with almost 100,000 new cases diagnosed each year. The prevalence of angina increases sharply with age. Around 50% of people diagnosed with ischaemic heart disease (IHD) present with stable angina as the first symptom.

NICE recommends the classification of chest pain into typical angina, atypical angina or non-anginal chest pain based on the presence of key features: a constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arms; precipitated by physical exertion; relieved by rest or GTN within about 5 minutes. The presence of all three features suggests a diagnosis of typical angina, two features is considered atypical angina and where one or none of the features are present the pain is defined as non-anginal. The likelihood of a diagnosis of angina increases with the number of cardiovascular risk factors present.

A resting 12-lead ECG is recommended for all patients with suspected angina. However, a normal result does not exclude the presence of underlying coronary artery disease (CAD). An abnormal resting ECG increases the likelihood that the patient has underlying CAD and may also identify other conditions such as atrial fibrillation or structural heart disease. In general, anatomical investigations, e.g. computerised tomography coronary angiography (CTCA), determine the presence of underlying CAD and identify individuals with a pattern of CAD that may benefit from revascularisation, while functional tests, e.g. exercise tolerance testing, evaluate the burden of myocardial ischaemia and facilitate risk stratification. In a randomised trial of more than 4,000 patients with a suspected diagnosis of angina, the addition of CTCA to standard care increased diagnostic accuracy and permitted targeting of therapy and interventions to those patients who gained most benefit.

There is clear evidence that aspirin reduces the risk of ischaemic events in patients with established CAD and it should be prescribed, where possible, for all patients with stable angina. There are robust data demonstrating that statins reduce the risk of death and MI in patients with IHD by 40%, and NICE currently recommends initiating treatment with atorvastatin 80 mg once daily. Treatment with an ACE inhibitor should be considered for patients with stable angina who have left ventricular dysfunction or diabetes mellitus.

There are currently six major drug classes available in the UK for the treatment of stable angina: beta-blockers, calcium channel blockers, nitrates, potassium channel activators, sinus node inhibitors and late sodium current inhibitors. Ideally, the dose of a single anti-anginal agent should be optimised before introducing combined therapy. There is little evidence to support the use of three anti-anginal drugs in combination. Beta-blockers should be used as first-line therapy, although evidence of a prognostic benefit in stable angina is limited.

CORONARY REVASCULARISATION

Coronary revascularisation can improve symptoms of angina and may offer prognostic benefit in those patients found to have high-risk coronary anatomy (left main stem stenosis or three vessel CAD). Revascularisation may be achieved by coronary artery bypass surgery or percutaneous coronary intervention usually with stent insertion. Patients with stable angina should be considered for coronary revascularisation where symptoms persist despite optimal medical therapy, where high risk coronary anatomy is identified, or in light of patient preference.

The recent ORBITA study confirmed existing evidence that in patients with a low burden of CAD, good exercise tolerance and minimal symptoms on optimal medical therapy, revascularisation with coronary stents confers no additional benefit.[15]

Factors influencing the choice of revascularisation technique include the burden and location of underlying CAD, the presence of left ventricular dysfunction or valvular heart disease, comorbidity including diabetes mellitus or renal dysfunction, and patient age.

Decision making should be patient-centred and informed by a multidisciplinary 'heart team' review involving cardiac surgeons and cardiologists.

REFRACTORY ANGINA

A small number of patients experience severe, limiting angina that is unresponsive to medical therapy and in whom revascularisation fails or is not available. Attendance at angina management programmes has been shown to improve both symptom control and exercise tolerance.

A number of therapeutic approaches have been developed to address refractory angina including enhanced external counter pulsation therapy, techniques such as nerve block, stellate ganglion block and sympathectomy, acupuncture and a stent to restrict coronary sinus blood flow.[16] The clinical data available for these techniques is currently insufficient to recommend their routine use in patients with stable, refractory angina.

CONCLUSION

Angina pectoris is a commonly encountered condition that is associated with significant morbidity and mortality. The clinical history plays a key role in diagnosis, aided by anatomical and functional testing where appropriate.

Treatment should focus on the prevention of ischaemic cardiac events and the satisfactory control of symptoms. Management should include patient education, risk factor identification and modification, drug therapy and, where appropriate, coronary revascularisation.

Competing interests

Dr Nicholas Cruden was chair of the guideline development group for *SIGN 151. Management of stable angina*, published in April 2018

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Useful information

The British Cardiac Patients Association
www.bcpa.uk

British Heart Foundation
www.bhf.org.uk

Chest Heart and Stroke Scotland
www.chss.org.uk