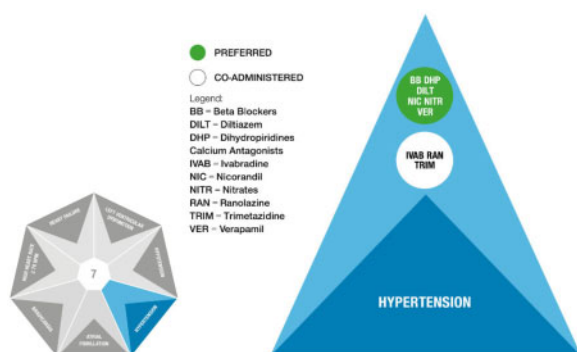


Angina due to post-myocardial infarction with associated hypertension

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A 52-year-old male patient was admitted to the emergency department with acute-onset chest pain lasting for several hours without relief. His medical history was not remarkable and he was not on any medication. He was a smoker (at least 10 cigarettes per day for 30 years), and he had a family history of coronary artery disease.

His electrocardiogram (ECG) (Figure 1) showed left bundle branch block (LBBB) with sinus rhythm and a heart rate of 72 b.p.m. On investigation, his blood chemistry was as follows: troponin I (TnI) 3.44 ng/mL (reference range 0-0.4), creatine kinase MB 1.6 ng/mL, and N-terminal pro-B-type natriuretic peptide 364 pg/dL. His bedside echocardiography demonstrated regional wall motion abnormality at the anterior wall with hypokinesia of other segments, ejection fraction was 50%, and mild mitral regurgitation was detected. The patient was admitted to the catheterization lab with still severe chest pain for a coronary angiography that was followed by a percutaneous coronary intervention on the left anterior descending artery.

Elective coronary intervention was planned for another significant lesion at the circumflex coronary artery that was not considered to be the culprit lesion. Non-significant lesions (<50% stenosis) were also found in the right coronary artery.

A few days after the first intervention, revascularization was completed with an elective intervention that was performed for a circumflex coronary artery lesion. He was then discharged on ticagrelor 90 mg twice, carvedilol 12.5 mg twice daily, ramipril/hydrochlorothiazide 5/12.5 mg, aspirin 100 mg, atorvastatin 40 mg, and pantoprazole 40 mg, and he was recommended to quit smoking. At the first follow-up visit on day 14, his ambulatory blood pressure was above the desired range and he complained of exercise-triggered angina episodes. His laboratory results were normal with only a significant increase in the uric acid level. At this stage, preventing angina episodes, lowering blood pressure, and reducing uric acid were necessary. To this end, the ramipril/hydrochlorothiazide was switched to perindopril/amlodipine 5/10 mg once daily.

Two weeks later, during the first-month follow-up visit, his resting heart rate was between 70 and 80 b.p.m. When he was carefully questioned, it was noted that he had restricted exercise capacity due to occurrence of chest pain. A 24-h rhythm Holter revealed sinus tachycardia episodes without any evidence of ventricular arrhythmias. His carvedilol dose was uptitrated to 25 mg twice daily.

Four months later, he was re-admitted to the emergency department with frequent stable angina attacks (i.e. 3-5 times per week). His electrocardiography showed an LBBB with sinus rhythm, a heart rate of 68 b.p.m. and a blood pressure of 125/85 mmHg. The echocardiography reconfirmed the akinesia, an ejection fraction of 50%, with mild mitral regurgitation. His cardiac troponin levels remained in the normal range and angina was relieved with sublingual short-acting nitrates. He was referred to an outpatient clinic for further investigations. A stress echocardiogram

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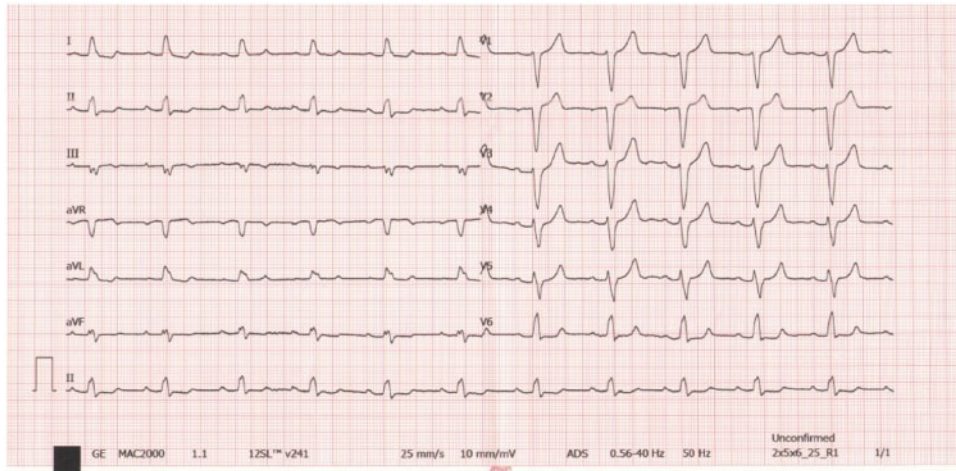


Figure 1 Electrocardiogram at the emergency department.

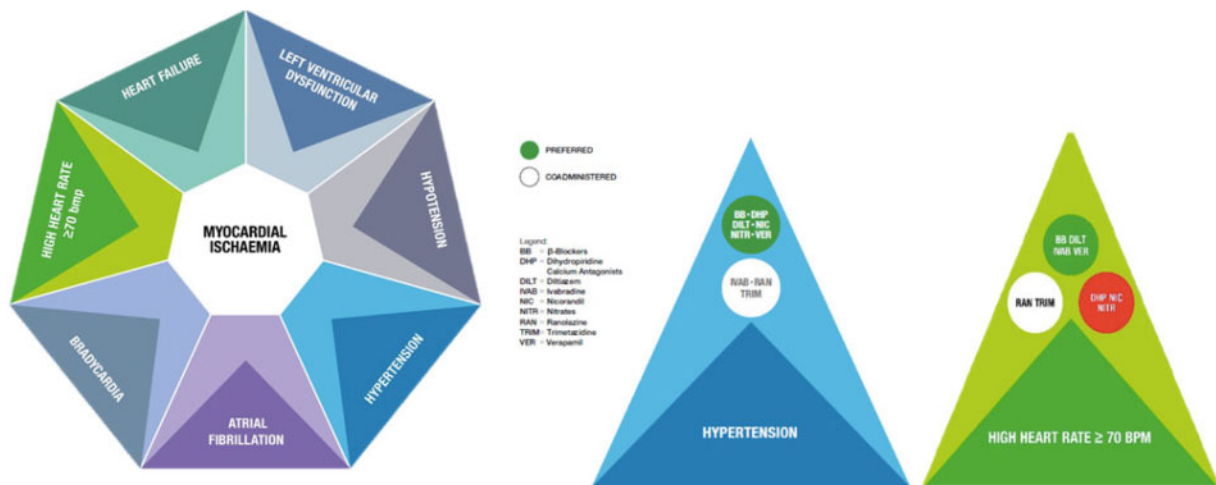


Figure 2 The choice of anti-ischaemic drugs according to blood pressure, heart rate, and comorbidities.³

was performed, and the small akinetic anterior wall motion abnormality did not change during the test. There was no newly developed wall motion abnormality. The stress echocardiogram pointed out the presence of scar tissue on the anterior wall, thus not requiring an additional percutaneous intervention. Due to recurrent stable angina attacks, it was decided to add trimetazidine 35 mg trim. 35 mg and 375 mg ranolazine twice daily to his treatment.

One year later, he remained asymptomatic under this treatment.

Discussion

This case demonstrated the complexity of managing stable angina and its close link to the unsustainability of the disease. The main issue is to understand the underlying causes of stable angina and to establish a strategy to prevent angina episodes and the relative ischaemic burden. The combination of lifestyle modifications, anti-ischaemic

treatment, and controlling risk factors and revascularization, when possible, is the approach recommended by the current guidelines.¹

The goal of anti-ischaemic therapy is two-fold: relieve the symptoms and prevent cardiovascular events. The determinants of this therapy depend on the patient's heart rate, blood pressure, renal function, and, of course, on the underlying pathophysiology of angina. First-line treatment of stable angina for patients after a myocardial infarction has always been considered with beta-blockers. By reducing heart rate, they facilitate perfusion of ischaemic areas by prolonging diastole and increasing the vascular resistance in non-ischaemic areas. In addition, beta-blockers reduce the oxygen consumption of the heart. In clinical trials, when given after a myocardial infarction along with symptomatic relief, beta-blockers achieved a 30% risk reduction in cardiovascular death and myocardial infarction.² Beta-blockers can be combined with dihydropyridines to control angina and hypertension, as in our case (Figure 2).

Also, short-acting nitrates offer coronary arteriolar and venous vasodilatation, which are the basis of symptomatic relief of effort angina. After optimizing heart rate and blood pressure, trimetazidine (35 mg twice daily) added to beta-blockade, if the patient has refractory angina, improves anaerobic metabolism induced by effort myocardial ischaemia and reduces episodes of stable angina in patients already receiving one or more antianginal drug.

The decision to revascularize a patient should be made in the presence of significant/residual obstructive coronary artery stenosis, the amount of related ischaemia, and the expected benefit on prognosis and/or symptoms. In addition to this, if physicians adjust the medical therapy by considering the 'diamond approach', it will be possible to prevent the unnecessary invasive procedures limiting the interventions to acute coronary syndromes only.

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