Angina due to diffuse coronary artery disease in a patient with heart failure

Luis Henrique Wolff Gowdak*

Heart Institute (InCor), Laboratory of Genetics and Molecular Cardiology, Av Dr Eneas de Carvalho Aguiar, 44 São Paulo, SP 05403-000, Brazil

A 64-year-old Caucasian female was referred to our Outpatient Clinic Center with a history of progressive, moderate angina (Canadian Cardiovascular Society Class II) and shortness of breath (New York Heart Association Class II) lasting for the past 3 months. Two years before, she had an acute myocardial infarction and underwent coronary bypass surgery (left internal mammary artery to left anterior descending artery + saphenous vein to the right coronary artery) at another facility. She had a long history of hypertension and hypercholesterolaemia, both irregularly treated. She works as a cook and is physically inactive.

On examination, she had a body mass index of 28.7 kg/m², a heart rate of 80 b.p.m., and a blood pressure (BP) of 132/82 mmHg. There was a mild holosystolic murmur (grade 2) best heard at the apex; fine bibasilar crackles were present as was pitting oedema in both legs. Blood glucose level was 108 mg/dL, HbA1c 6.1%, total cholesterol 191 mg/dL, low-density lipoprotein (LDL) 112 mg/dL, high-density lipoprotein 38 mg/dL, triglycerides 205 mg/dL, and a creatinine level of 0.9 mg/dL (glomerular filtration rate was 67 mL/min/1.73 m² as calculated by the Modification of Diet in Renal Disease Study equation). The resting ECG is shown in Figure 1 and a transthoracic echocardiogram (Figure 2) revealed a dilated left ventricle with an estimated ejection fraction of 28% (Teicholz), moderate left atrial enlargement, and mild mitral regurgitation.

She was on aspirin 100 mg once daily, enalapril 10 mg twice daily, carvedilol 12.5 mg twice daily, spironolactone 25 mg once daily, and atorvastatin 20 mg once daily.

At this stage, a diagnosis of stable angina in a patient with post-myocardial infarction heart failure was made. Based on the clinical diagnosis, how would you further investigate this patient?

Would you consider a functional, non-invasive assessment of her ischaemic burden? Would you prefer a non-invasive assessment of the coronary arteries by computed tomography angiography? Would you rather proceed immediately with an invasive angiography?

Although the patient was still not on optimal medical therapy for angina control, her history of recent, progressive symptoms and the impairment in the left ventricular function prompted our Heart Team to consider an invasive coronary angiography.

Meanwhile, medical treatment had to be optimized for better symptom control. The patient was strongly advised to lose weight, and, accordingly, nutritional counselling was recommended. Atorvastatin was increased to 80 mg daily in an attempt to achieve an LDL level < 50 mg/dL. Furosemide 40 mg once daily was added. However, the panel was divided between increasing the ACE inhibitor or the β-blocker dose—the main concern being the reduction in BP and, thus, tolerability. It was also proposed to switch the ACE inhibitor to the sacubitril/valsartan combination, but it was decided not to make this switch due to the risk of hypotension. Finally, we increased the dose of carvedilol to 25 mg twice daily.

One month later, she returned with the results of the coronary angiography (Figure 3). She mentioned a modest improvement in symptoms, especially the shortness of breath. She had lost about 2 kg. Angina was less frequent;
last week she had a disagreement with a co-worker and angina occurred at rest, but it was relieved with a short-acting nitrate. Her heart rate was down to 72 b.p.m. and BP to 122/72 mmHg.

Now we have to face the decision between further optimizing medical treatment or consider a myocardial revascularization procedure (percutaneous coronary intervention or redo coronary artery bypass grafting).

What would you do now?

The Heart Team convened again to discuss whether to proceed to coronary angioplasty of the obtuse marginal branch and the right coronary artery (chronic total occlusion). There was an overall consensus to first optimize medical treatment further trying to tackle both the coronary artery disease (for symptom control) and the presence of heart failure with reduced ejection fraction (for prognosis). Ivabradine 5 mg twice daily was added to her treatment. Ivabradine was selected because the heart rate was still above 70 b.p.m. on the maximally tolerated dosage of β-blocker.

The BEAUTIFUL trial¹ showed that, in patients with stable coronary artery disease with a heart rate above 70 b.p.m., in sinus rhythm, and a left ventricular ejection fraction below 40%, ivabradine on top of maximally tolerated therapy decreased the risk of hospitalization for fatal/non-fatal myocardial infarction by 36%, and the need of revascularization by 30%. The SHIFT trial,² targeting patients with severe left ventricular dysfunction like the one we are discussing here, showed that the addition of ivabradine on top of optimal medical therapy led to a significant 26% decrease in both the risk of hospital admissions for worsening heart failure and deaths due to heart failure. Antianginal agents with BP-lowering effects (such as dihydropyridine calcium channel antagonists or long-acting nitrates) should not be used or used with caution; drugs with myocardial depressant effects (verapamil or diltiazem) should also not be used in patients with left ventricular dysfunction.³

Trimetazidine has beneficial effects in patients with left ventricular dysfunction by decreasing the severity of angina and increasing left ventricular ejection fraction⁴ and could be an option if needed, although its impact on long-term prognosis is less well documented than for ivabradine.

One month later, the patient reported a significant improvement in the severity and frequency of her angina attacks. She was enrolled in a cardiac rehabilitation programme. Her vitals now were a heart rate of 64 b.p.m. and BP 120/68 mmHg. Ivabradine was titrated to 7.5 mg twice daily. When last seen, she was quite pleased with her treatment, having experienced no angina during her daily activities.

Funding

The authors didn’t receive any financial support in terms of honorarium by Servier for the articles.
Conflict of interest: L.H.W.G. has received honoraria from Servier for lecturing and consulting.

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