An irregular cause of heart failure

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Articles in this section use cases to illustrate the emergency management of patients presenting in general practice with cardiac problems. They are inspired by, but not based on, real patient situations.

Mrs FW is a 54-year-old nonsmoker who lives alone and presents for review after three months of increasing dyspnoea. She has a past history of hypertension and type 2 diabetes mellitus, for which her medications are ramipril, metformin, gliclazide and pioglitazone. She works in video surveillance security and is minimally active. She states the dyspnoea has recently interrupted her sleeping. She denies having leg swelling and chest pain. When asked, she recalls having intermittent palpitations over the past few years, which she attributes to stress. She has no history of travel or exposure to infections.

What does clinical examination show?

Mrs FW steps on the scales and weighs 94 kg (body mass index, 35 kg/m^2). Her heart rate is 141 beats per minute and blood pressure is 150/90 mmHg. Her saturations are normal on room air, with no evidence of increased work of breathing at rest. There is no conjunctival pallor or thyroid mass, and her jugular venous pressure is mildly elevated. On auscultation you suspect her heart sounds are not normal, with either a splitting of the second heart sound or something like a gallop rhythm. This makes it difficult to diagnose a murmur, but you suspect there is a pansystolic murmur at the apex. There is no pericardial rub. You identify medium pan-inspiratory crackles at both lung bases.

On examining her peripheries, you find no oedema or calf tenderness. You ask your practice nurse to perform an ECG, which shows atrial fibrillation (AF) with a ventricular rate of 134 beats per minute, with no evidence of ischaemia, bundle-branch block or pre-excitation (Figure 1).

What is the next step?

Both the heart failure and AF are new. You feel the quickest way to achieve adequate diuresis and rate control is to send Mrs FW to hospital. You explain to her that she will be able to receive a chest x-ray and transthoracic echocardiogram quickly in hospital. You call a nonurgent ambulance and phone the emergency medical officer to notify them of her impending arrival.

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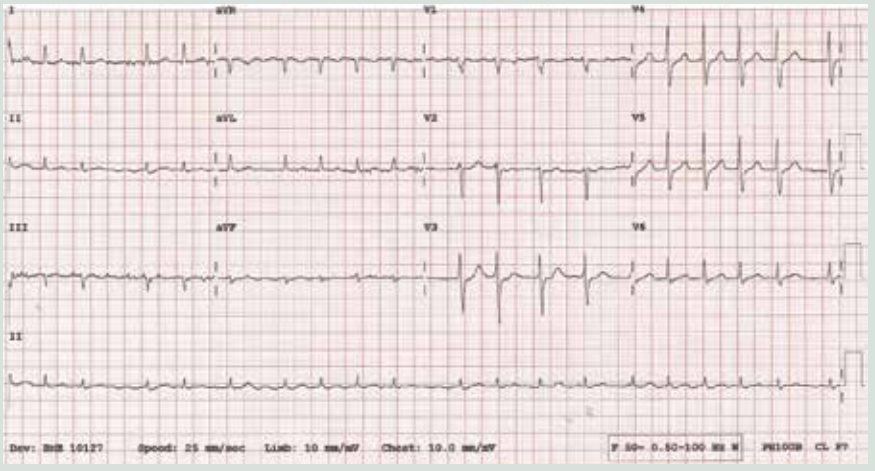

Figure 1. ECG showing atrial fibrillation with rapid ventricular response and heart rate of 134 beats per minute.

What happens in the emergency department?

On arrival at the emergency department Mrs FW is taken to an acute bed to receive cardiac monitoring. She has blood tests to assess for concurrent conditions and precipitants of AF including anaemia, infection, hypokalaemia, hypomagnesaemia and hyperthyroidism. These tests are often normal in this context, as they prove to be for Mrs FW. Her chest x-ray shows cardiomegaly with upper-lobe diversion and prominent vascular markings (consistent with left heart failure).

Initial pharmacotherapy is an immediate dose of intravenous furosemide (frusemide). On admission to the ward she is to be given regular oral furosemide to continue her diuresis, oral metoprolol (initially 25 mg twice daily and after confirming satisfactory blood pressure) to achieve gradual ventricular rate control, and therapeutic enoxaparin pending a decision on long-term anticoagulation. A transthoracic echocardiogram is ordered.

What does the echocardiogram show? What is the diagnosis?

The echocardiogram shows a moderately dilated left ventricle (internal diameter, 62 mm in diastole) with normal wall thickness and with moderate global impairment in systolic function (left ventricular ejection fraction [LVEF], 35%). There is mild mitral regurgitation that appears to be secondary to her dilated left ventricle. The left atrium is moderately dilated. There is no other valvular disease and right ventricular systolic function is normal. Her diagnoses are non-valvular AF with a CHA_2DS_2-VASc score of 4 (one point for each of heart failure, hypertension, diabetes and female sex), and moderate-to-severe cardiomyopathy.

What are the treatment priorities for Mrs FW?

The priorities in treatment are, firstly, to improve her symptoms of dyspnoea; secondly, to investigate and treat any underlying causes of AF; thirdly, to investigate and treat underlying causes of heart failure; and fourthly, to take steps to prevent systemic thromboembolism (particularly stroke).

Symptom control

The immediate goal in symptom control is to achieve diuresis. This involves a combination of pharmacotherapy (usually a loop diuretic such as furosemide) combined with fluid restriction. Monitoring of her diuresis is easily achieved by daily weight measurement using the same set of scales each morning. The second goal of symptom control is slowing Mrs FW's tachyarrhythmia. This often takes longer in hypervolaemic states, so oral rate-lowering medication is usually sufficient (and safer than intravenous therapy). As Mrs FW's ventricular rate improves her systolic function will likely improve and this will have a synergistic effect in achieving diuresis.

Investigating and treating new AF

Numerous risk factors may predispose a patient to AF. Some are modifiable, such as obesity, hypertension, heart failure, diabetes, thyroid disease and obstructive sleep apnoea. Others are nonmodifiable or less modifiable, such as advancing age, prior myocardial infarction, chronic lung disease and a genetic predisposition. Heart failure and AF often coexist; AF may be caused by heart failure, or AF with a rapid ventricular response may cause heart failure (a tachycardia-mediated cardiomyopathy).

Therapeutic intervention in AF can be via a rate-control or rhythm-control strategy. Earlier trials comparing medical therapy for rate and rhythm control in patients with AF showed no survival difference between the two strategies and more side effects in the rhythm-control arm.[1] However, a rhythm-control strategy is still often used for patients with significant symptoms such as palpitations, dyspnoea, dizziness or lethargy from their AF. The rhythm-control strategy aims to achieve and maintain sinus rhythm with the use of antiarrhythmics such as sotalol, flecainide or amiodarone. Direct current cardioversion (DCCV) is sometimes needed as well. In a case such as Mrs FW's, heart-failure symptoms will usually improve quite quickly with adequate rate control and diuresis; however, if this is not the case she may require intravenous amiodarone loading and/or DCCV to achieve sinus rhythm. It would be important to perform a transoesophageal echocardiogram to exclude the presence of atrial thrombus before either DCCV or chemical cardioversion, as they can both precipitate a stroke or peripheral embolism in patients who have not been adequately anticoagulated.

A further rhythm-control strategy in the long-term management of AF is catheter ablation (also called pulmonary vein isolation). In general, catheter ablation is performed in patients with symptoms from AF and without significant structural heart disease, although a recent trial showed that ablation was superior to amiodarone in achieving freedom from AF in patients with heart failure (LVEF, < 40%) as

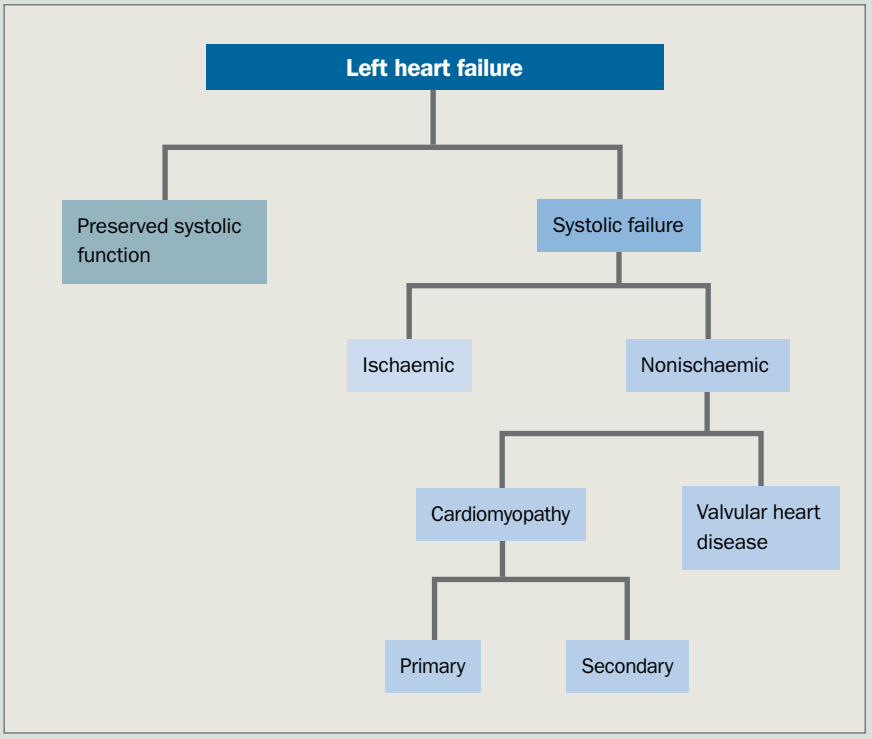

Figure 2. Classification of left heart failure.

well.[2] This randomised multicentre trial showed that ablation was associated with lower mortality (8% *vs* 18%), reduced hospitalisation, improved quality of life and exercise capacity and fewer complications compared with amiodarone. Patients and doctors should understand the risks of catheter ablation, which vary with the technique used but include a risk of stroke or transient ischaemic attack (one in 200 procedures), tamponade (one in 100 to 200) or a groin site complication (one in 50).[3]

Treatment of patients with AF should also include cardiovascular risk modification, which has been shown to reduce recurrent AF in patients undergoing catheter ablation.[4] Risk factor modification should include blood pressure control, weight loss, lipid management, glycaemic control, treating sleep-disordered breathing and smoking and alcohol management.

Investigating and treating new heart failure

Systolic heart failure is broadly divided into ischaemic and nonischaemic causes. The classification of left heart failure is described in Figure 2. A nonurgent coronary assessment should be performed (such as a CT coronary angiogram, an invasive coronary angiogram or a myocardial perfusion scan). Mrs FW's angiogram results are normal, indicating she has a nonischaemic cardiomyopathy. There is no evidence of valvular heart disease on her echocardiogram and a careful history reveals that her cardiomyopathy is not familial or secondary to exposures such as alcohol, chemotherapy or infection. Given Mrs FW's presentation with AF and rapid ventricular response she is diagnosed with tachycardia-induced cardiomyopathy secondary to occult AF (a moderately enlarged left atrium suggests the AF may have been present for some time).

Standard pharmacotherapies for systolic heart failure that have been shown to reduce mortality include an ACE inhibitor or angiotensin–neprilysin inhibition (which has recently been approved for use in Australia in the form of sacubitril–valsartan), a heart failure-specific beta-blocker and an aldosterone antagonist. Further therapies may include ivabradine for patients in sinus rhythm intolerant of beta-blockers and without activity-limiting angina, an implantable cardioverter-defibrillator for primary prevention of ventricular arrhythmias in patients with LVEF less than 35%, and cardiac resynchronisation therapy (also called a biventricular pacemaker/defibrillator) in patients with severe cardiomyopathy in sinus rhythm and with left bundle branch block. Mrs FW is to continue on her dose of ACE inhibitor and metoprolol is changed to bisoprolol, which is a heart failure beta-blocker (along with carvedilol, nebivolol and metoprolol succinate). For the desired therapeutic benefits, it is very important to uptitrate heart failure medications on a weekly or second-weekly basis to achieve the maximal doses tolerated by the patient according to symptoms, heart rate and blood pressure. In addition, Mrs FW's pioglitazone is replaced with a dipeptidyl peptidase 4 (DPP-4) inhibitor, as the thiazolidinedione class (pioglitazone and rosiglitazone) may worsen heart failure.

Deciding whether long-term anticoagulation is required

Based on her CHA_2DS_2-VASc score, Mrs FW has an annual stroke risk of between 4.8 and 7.8%.[5] European guidelines suggest anticoagulation in patients with AF and a CHA_2DS_2-VASc score of 1 or more, and no anticoagulation in patients with a CHA_2DS_2-VASc score of zero.[6] Importantly, a second risk factor must be present for female sex to contribute one point to the CHA_2DS_2-VASc score. Aspirin is no longer used for stroke prevention in patients with AF, but is still used in those with cerebrovascular disease (stroke or transient ischaemic attack) without AF, or patients with AF who refuse any anticoagulant. The CHA_2DS_2-VASc score does not apply to patients with valvular AF, meaning they have either mitral stenosis or a mechanical valve replacement, as these patients require warfarin anticoagulation regardless of their score.

Do I choose warfarin or a NOAC?

The nonvitamin K antagonist oral anticoagulants (NOACs) are either superior or noninferior to warfarin for stroke prevention in patients with nonvalvular AF,[7] like Mrs FW; however, renal function must be assessed, as all NOACs are at least partially renally excreted. The main benefit of choosing a NOAC over warfarin is a

lower rate of intracranial bleeding, but benefits also include improved compliance, greater time spent in the therapeutic window, no need for INR checks and reduced drug–drug interactions. For these reasons, Mrs FW is commenced on a NOAC. The three NOACs available in Australia are dabigatran, rivaroxaban and apixaban, and selecting between them frequently depends on patient factors. For example, apixaban may be preferred in patients with risk of gastrointestinal bleeding, rivaroxaban may be preferred in patients not willing to adhere to a twice-daily regimen, and high-dose dabigatran may be preferred in secondary prevention for patients with very high embolic risk.

Outcome: Mrs FW's symptoms improved dramatically and she returned to work two weeks after her admission. Her condition had been stabilised on maximal doses of heart failure therapy, she had tolerated a NOAC without major bleeding problems and had achieved a modest 5% weight loss with dietary intervention. She continued to have symptoms from AF despite a trial of antiarrhythmic therapy, so she underwent a catheter ablation procedure and has not had any recurrence of AF since. At her most recent follow up six months later she was in sinus rhythm with mildly impaired left ventricular systolic function (LVEF, 50%) and had no further symptoms of heart failure or AF.

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COMPETING INTERESTS: None.