Hidden caveats when diagnosing heart failure

AUSTIN N. MAY MB BS
GORDIAN FULDE MB BS, FRACS, FRCS(Ed), FRCS/FRCP, (A&E)Ed, FACEM

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.

A 63-year-old car sales representative, Mr DW, comes to see you, his GP, today along with his wife. He reports an insidious decline in his ability to swim 20 laps of the local olympic-sized swimming pool, which he has particularly noticed in the past six months. He has needed to interrupt his laps with a rest because of fatigue and dyspnoea, and on a number of occasions he has been unable to finish. He has no symptoms at rest and has not experienced any chest pain, palpitations or presyncope. He has no orthopnoea but volunteers that he has noticed some ankle swelling over the past four months. He acknowledges that he is overweight at 90 kg, but he has always been active, with which his wife agrees.

Mr DW normally sees you annually and has been stoic in reporting symptoms in the past. His medical history includes a recurrent meniscal knee injury, cholecystectomy and resected colonic polyps. He takes slow-release naproxen once daily with pantoprazole for gastric cover. He consumes up to three beers a day, with two alcohol-free days each week. He is a lifelong nonsmoker.

How do you begin this examination?

You ensure Mr DW is sitting comfortably and you measure his respiratory rate, which is normal. His pulse rate is 65 beats per minute and regular, and his blood pressure is 155/95 mmHg. His body mass index is calculated as 34 kg/m^2. There is no conjunctival pallor and no thyroid mass. Chest auscultation reveals dual heart sounds, no murmurs and normal vesicular breath sounds. His abdomen is soft with no organomegaly. He has no varicose veins and he has mild symmetrical pitting oedema of his ankles. You perform an ECG, which reveals sinus rhythm, normal axis and poor R wave progression (Figure 1). You also carry out some blood tests and a chest x-ray.

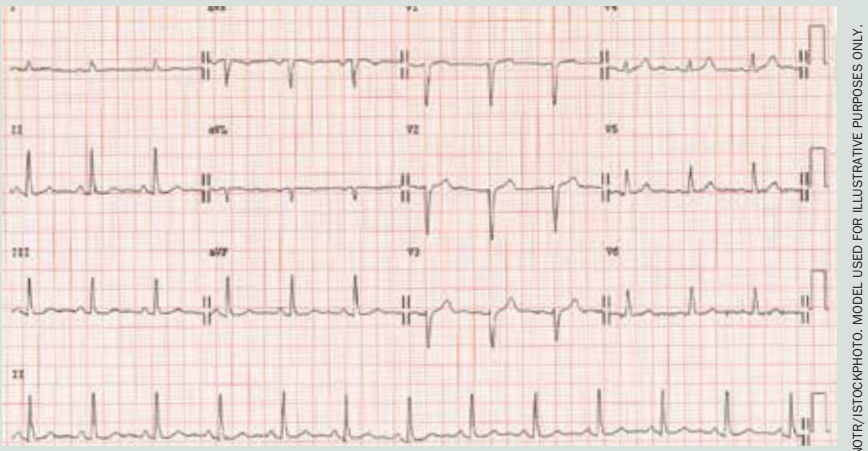
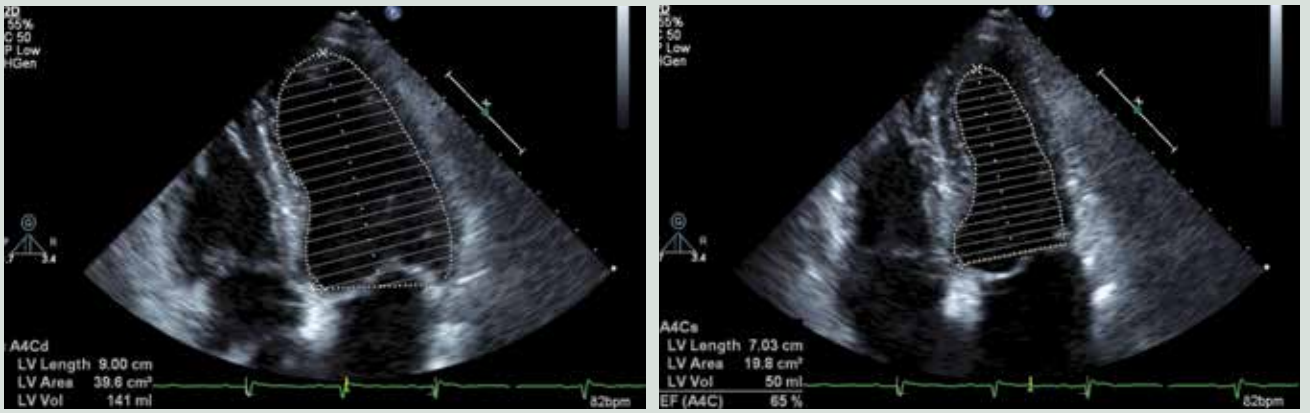


Figure 1. ECG showing sinus rhythm with normal axis and poor R wave progression.

CARDIOLOGY TODAY 2015; 5(2): 38-40

Dr May is a Cardiology Advanced Trainee at Gosford Hospital and Conjoint Associate Lecturer at the University of NSW, Sydney. Professor Fulde is Director of Emergency Medicine at St Vincent's Hospital; Associate Professor at the University of NSW; and Professor at the University of Notre Dame, Sydney, NSW.



Figures 2a and b. Transthoracic echocardiogram at end diastole (a, left) and end systole (b, right) showing normal left ventricular ejection fraction calculated using modified Simpson's rule.

Mr DW returns two days later for his test results. The results reveal a mildly elevated haemoglobin level of 155 g/L, normal creatinine level of 75 µmol/L and normal liver and thyroid function. There is no cardiomegaly or pulmonary oedema on chest x-ray. The lung fields are also clear with no evidence of hyperinflation.

What is your provisional diagnosis for this patient?

Your provisional diagnosis is new heart failure and so you arrange for Mr DW to have a transthoracic echocardiogram. The results show normal left ventricular size with left ventricular wall thickness on the upper limit of normal. Left ventricular systolic and diastolic function are normal and ejection fraction measures over 60% (Figures 2a and b). Right ventricular size and systolic function are normal. Aside from moderate tricuspid regurgitation, there is no significant valvular disease.

What do you tell the patient and his wife?

You discuss the test results with Mr DW and his wife. You explain that you would like to check Mr DW's blood pressure more frequently, and it is likely he will have to commence antihypertensive medication in the future. Although his symptoms suggest a clinical picture of heart failure, you explain that the echocardiogram findings are reassuring as they show that his heart pump function is normal. You know there is no indication to commence this patient on heart failure medications but you wonder if a diuretic would help his symptoms or if you are missing another explanation altogether.

How do you diagnose heart failure and what are the possible causes?

Heart failure is a clinical diagnosis and can be broadly separated into heart failure with reduced left ventricular ejection fraction and heart failure with preserved left ventricular ejection fraction (HFpEF).

There are many causes of reduced ejection fraction, divided into ischaemic (majority) and nonischaemic causes. These are diagnosed by the presence of symptoms, such as orthopnoea and exertional dyspnoea (using the New York Heart Association classification[1]) and signs such as raised jugular venous pressure and peripheral oedema, in combination with an imaging modality showing reduced ejection fraction.

A diagnosis of HFpEF, previously referred to as 'diastolic heart failure', can be made when clinical symptoms and signs are supported by additional echocardiographic information on left ventricular filling dynamics and elevated left ventricular diastolic pressures. HFpEF tends to affect women more frequently than men, and is usually preceded by years of systolic hypertension. Unfortunately, these patients have a devastating five-year mortality rate (approaching 60%) with no evidence that they respond to traditional medications for heart failure.[2] There is also considerable morbidity for affected patients, with a six-month hospitalisation rate of 50%.

Other cardiac conditions that can produce similar clinical presentations include hypertrophic cardiomyopathy, valvular heart disease, constrictive pericarditis, radiation-induced heart disease and tachyarrhythmias such as atrial fibrillation.

If the patient does not have heart failure or a cardiac cause for his presentation, what are the differential diagnoses?

There are some cases in which a patient's symptoms can be misdiagnosed as being due to heart failure. A meta-analysis of 22 studies found that the symptom of exertional dyspnoea had a low likelihood ratio of 1.3 of being due to heart failure (95% confidence interval, 1.2–1.4).[3] Instead, the explanation may be a number of comorbid conditions that are either overlooked or suboptimally treated (Box 1).

Noncardiac considerations for a decreased exercise tolerance are summarised below.

- ***Respiratory considerations.*** Elevated pulmonary pressures with or without a dilated right ventricle can cause exertional dyspnoea and peripheral oedema, and limit exercise tolerance. The causes of pulmonary hypertension are generally classified into five groups (Box 2).[4] In addition, chronic respiratory disease is a common cause of exertional dyspnoea. This includes chronic airways limitation (with and without bronchodilator reversibility), interstitial lung disease, occupational lung disease, obstructive sleep apnoea, sarcoidosis and malignancy.
- ***Haematology considerations.*** Impaired exercise tolerance is common in many haematological presentations, some of which can be identified on full blood count and blood film examination. For instance, measurement of haemoglobin and haematocrit levels will help identify anaemia and primary or secondary polycythaemia, whereas leukopenia will help identify underlying bone marrow disorders. Eosinophilia can be seen in both patients

Text

1. Causes of clinical presentations that may be mistaken for heart failure

- Respiratory causes
  - pulmonary hypertension
  - obstructive sleep apnoea
  - chronic airways limitation
  - asthma
  - interstitial lung disease
  - sarcoidosis
  - venous thromboembolism
- Haematology causes
  - anaemia of any cause
  - bone marrow disorder
  - myelodysplastic syndrome
  - myeloproliferative disorder
  - leukaemia
- Endocrine causes
  - hyper- or hypothyroidism
  - hyper- or hypocortisolism
  - obesity
- Renal causes
  - chronic renal failure
  - glomerulonephritis
- Gastroenterology causes
  - chronic liver disease
  - coeliac disease
- Neuromuscular disorders
  - myopathy
  - neuromuscular junction disorder (e.g. myaesthenia gravis, Lambert-Eaton myasthenic syndrome)
  - neuropathy

with haematological disorders and those without nonhaematological disorders (such as allergic disorders, connective tissue disorders and helminth infections). Other haematological disorders that can impair exercise tolerance include myelodysplastic syndrome, myeloproliferative disorders and leukaemias.

- ***Endocrine considerations.*** Metabolic causes of exertional dyspnoea include obesity, hyper- and hypothyroidism, hyper- and hypocortisolism, and syndrome of inappropriate antidiuretic hormone.
- ***Renal considerations.*** Many patients with chronic renal failure of any cause will notice exertional limitations. Additionally, both acute and chronic causes of renal failure can result in peripheral oedema.
- ***Gastroenterology considerations.*** Liver disease may result in decreased exercise tolerance and peripheral oedema. The main causes in developed countries are

2. Classification of pulmonary hypertension[4]

- Pulmonary arterial hypertension
- Pulmonary hypertension with left heart disease
- Pulmonary hypertension associated with lung diseases and/or hypoxaemia
- Pulmonary hypertension due to chronic thrombotic and/or embolic disease
- Miscellaneous causes of pulmonary hypertension

nonalcoholic fatty liver disease (NAFLD), alcoholic liver disease, viral hepatitis and haemochromatosis. NAFLD has an insidious course and can result from obesity (or anorexia), hyperlipidaemia and/or diabetes.

If other aetiologies are suspected, how would you investigate these?

If other aetiologies are suspected, it is important to investigate these before diagnosing heart failure. Initial investigations may include spirometry, chest x-ray, full blood count and assessment of renal, liver and thyroid function. If required, measurement of plasma brain natriuretic peptide levels can be helpful in supporting or excluding a diagnosis of heart failure. An echocardiogram and cardiopulmonary exercise testing can be extremely useful in guiding further investigation. If there is systolic dysfunction, a coronary circulation assessment will be required. If there is diastolic dysfunction, the aim is to optimise blood pressure control and use nonpharmacological strategies of diet, exercise and occasionally fluid restriction. If there is pulmonary hypertension, specialist referral should be considered. Further investigations may include a sleep study, ventilation–perfusion scan (or CT pulmonary angiography), high-resolution CT chest, transoesophageal echocardiogram (to examine for intracardiac shunt) and perhaps even a formal right heart catheterisation.

Management will depend on the outcomes of these investigations, with a view to reverse the underlying disorder if possible.

Your investigations suggest that Mr DW has elevated pulmonary pressure. The poor R wave progression on ECG and the moderate tricuspid regurgitation on echocardiogram are important clues. Basic spirometry is negative for an obstructive or restrictive defect. You enquire about sleep patterns and snoring, with the questions mainly directed to Mr DW's wife. She reports that he snores loudly, particularly on nights he has consumed alcohol. Mr DW reports experiencing daytime sleepiness at times, but there is no history of car accidents or near misses.

You arrange for Mr DW to undergo a sleep study the following week. The results of the study confirm that he has obstructive sleep apnoea with an apnoea–hypnoea index of 32 events per hour, consistent with a diagnosis of severe sleep apnoea (normal: 0–4 events per hour; mild: 5–14 events per hour; moderate: 15–29 events per hour; severe: >30 events per hour). You explain to Mr DW that sleep apnoea could be contributing to his high blood pressure and leg swelling. You tell him that If this is left untreated, he may develop right heart failure and he is at risk of a heart attack and stroke.

Outcome

Mr DW starts continuous positive airways pressure therapy, and a few weeks later he visits you to say he is grateful for your help and feels 're-energised'. He is also content to take an antihypertensive once a day, knowing that it may not be required in the long term if he achieves optimal control of his sleep apnoea and loses weight. You reinforce your advice that he should decrease his alcohol intake to help with his sleep apnoea, obesity and hypertension, but otherwise you are happy with his progress. **CT**

References

1. The Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 9th ed. Boston, MA: Little, Brown & Co; 1994: 253-256.
2. Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. N Engl J Med 2006; 355: 251-259.
3. Wang CS, FitzGerald JM, Schulzer M, Mak E, Ayas NT. Does this dyspneic patient in the emergency department have congestive heart failure? JAMA 2005; 294: 1944-1956.
4. Simonneau G, Robbins I, Beghetti M, et al. Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol 2009; 54(1 Suppl S): S43-S54.

COMPETING INTERESTS: None.