

# Team-based management to improve real-world outcomes in heart failure

Matthew Ryan, BSc, MBChB, MRCP

Department of Cardiology, Guy's and St Thomas' NHS Foundation Trust, London, United Kingdom;  
Cardiovascular Division, King's College London, London, United Kingdom

Correspondence: Dr Matthew Ryan, The Rayne Institute, 4th Floor Lambeth Wing, St Thomas' Hospital,  
Westminster Bridge Road, London, SE1 7EH, UK  
E-mail: matthew.ryan@kcl.ac.uk

## Abstract

Adopting a team-based strategy in the management of heart failure has been demonstrated to improve patient outcomes through greater guideline adherence, better patient adherence, and closer monitoring of clinical status. We describe the case of a young patient who had repeated presentations with symptomatic heart failure, followed by disengagement from care. The cycle of deterioration was halted through the work of our multidisciplinary heart failure team. ■ *Heart Metab.* 2017;74:29-31

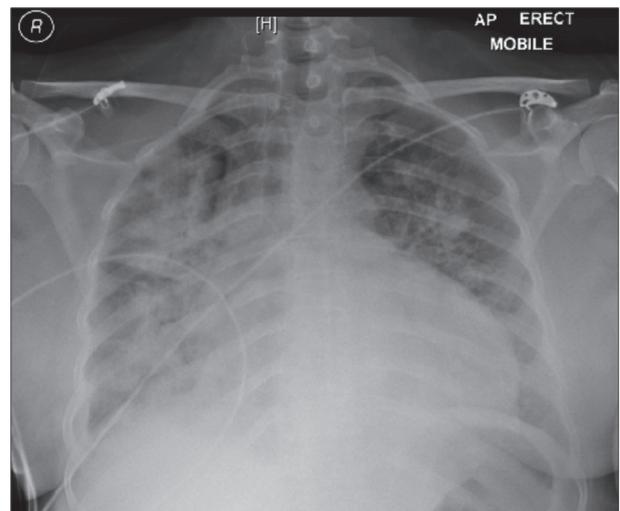
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## Case

A 47-year-old Ghanaian woman attended our emergency department with dyspnea at rest. Her symptoms had developed over a few hours, before which she reported feeling well, apart from a dry cough. There was no history of edema, chest pain, or syncope. Her past medical history was remarkable for hypertension, hypercholesterolemia, and recurrent urinary tract infections with a previous episode of acute kidney injury. Her hypertension was treated with once-daily amlodipine (10 mg) as monotherapy. She had been taking thrice-daily amoxicillin (500 mg), prescribed by her general practitioner for a presumed pneumonia and initiated 24 hours earlier. She smoked five cigarettes per day and consumed alcohol infrequently.

On initial assessment she was found to be in extremis, with a resting tachypnea of 40 breaths per minute and profound hypoxemia with oxygen saturations of 60% on air. Mean arterial pressure was elevated at 110 mm Hg. Clinical examination revealed bilateral end-in-

spiratory crackles, wheeze, and a third heart sound. She was euvolemic with no signs of systemic hypoperfusion. An anteroposterior chest radiograph (*Figure 1*) demonstrated pulmonary edema and marked cardio-



**Fig. 1** Chest radiograph demonstrating marked cardiomegaly and pulmonary edema.

**Abbreviation:** AP, anteroposterior.

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### Abbreviations

**BNP:** B-type natriuretic peptide; **ECG:** electrocardiogram; **EF:** ejection fraction; **GTN:** glyceryl trinitrate; **MDT:** multidisciplinary team; **MRI:** magnetic resonance imaging

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megaly, even considering the projection. A 12-lead electrocardiogram (ECG) confirmed sinus rhythm, with left axis deviation but no evidence of ischemia.

She was initially resuscitated in the emergency department with high-concentration oxygen, intravenous furosemide, and an intravenous infusion of glyceryl trinitrate (GTN). Broad spectrum antibiotics were administered to cover for superadded infection. Despite this, her oxygen saturation deteriorated, and she was sedated and intubated in the emergency department 4 hours after presentation. She was transferred to our intensive care unit for stabilization.

A review of her medical notes elicited important aspects of her case. She had presented to cardiology services on a number of occasions over the previous 6 years. The first of these presentations was an admission to hospital with atypical chest pain, where she was found to have an abnormal ECG (T-wave inversion in the anteroseptal leads) and a hypokinetic apical anterior left ventricle segment on echocardiography. An invasive coronary angiography performed at the time revealed unobstructed epicardial coronary arteries. The episodes of chest discomfort resolved, and she remained well at follow-up 12 months later, though she described intermittent episodes of dyspnea, which were thought at the time to represent panic attacks. Ventricular function had normalized on echocardiography. A review was offered a year later, but the patient declined to attend. She had attended hospital on one further occasion with a suspected lower respiratory tract infection, and an ECG performed at the time showed mild left ventricular dysfunction. Follow-up was arranged, but she missed a number of appointments. There were also concerns regarding adherence to her antihypertensive medication regime during this period.

Given the repeated presentations, we investigated her for alternative causes of flash pulmonary edema. Renal duplex ultrasound demonstrated normal flow in the renal arteries, and dynamic mitral regurgitation was felt to be unlikely given the normal valve morphology and unobstructed coronary arteries. A 24-hour urine collection excluded pheochromocytoma.

Echocardiography was repeated during intensive care. The left ventricle was found to be dilated and globally hypokinetic with an ejection fraction (EF) of 30%. The right ventricle's function appeared well preserved and there were no significant valvular lesions or pericardial effusion. Her hypertension was controlled and she had a brisk diuresis. This led to a rapid improvement in gas exchange, permitting successful extubation after 12 hours. Guideline-directed heart failure therapy (angiotensin-converting enzyme [ACE] inhibitor and  $\beta$ -adrenoceptor blocker) was initiated and uptitrated quickly to maximum tolerated doses, along with a small dose of loop diuretic. Amlodipine was discontinued. She was discharged 8 days after admission.

After discharge, the patient's ongoing monitoring was supervised through our heart failure multidisciplinary team (MDT). She received support from our community heart failure nurses, including regular contact and extensive education and encouragement to adhere to her medication regime. At an early outpatient review with her heart failure consultant, she was asymptomatic and her blood pressure was well controlled. A cardiac magnetic resonance imaging (MRI) scan showed persistent left ventricle dilatation but an improvement in EF, measured at 45%. No myocardial scar or right ventricular dysfunction was seen. MRI aortography again excluded renal artery stenosis and coarctation of the aorta. A formal diagnosis of dilated cardiomyopathy was made, and she will remain under follow-up with review by the MDT where necessary.

### Heart failure in the real world

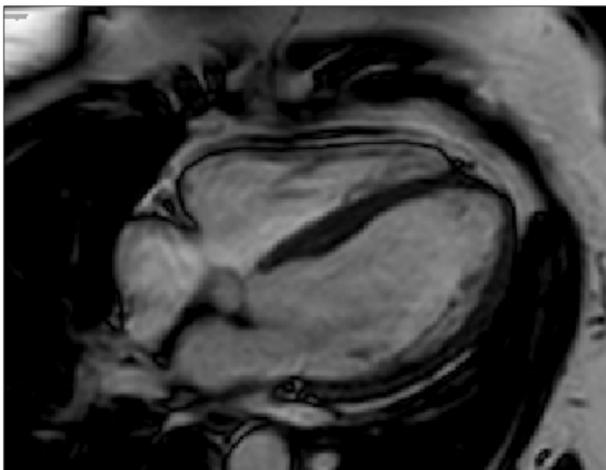
Managing heart failure in real clinical practice often feels a world away from the apparent perfection achieved in clinical trials. Optimally titrated doses and robust follow-up find themselves replaced with challenging adherence issues and missed opportunities to optimize care. This case highlights a number of these issues, as well as strategies that can be adopted with the aim of improving outcomes.

As in this case, the real-world diagnosis of heart failure is often delayed but apparent in retrospect. This is particularly true for younger patients, who with a greater physiological reserve will adapt to a certain level of dysfunction until a "tipping point" is reached and florid symptoms develop. It is not uncommon for episodes of dyspnea to be diagnosed as panic attacks or lower respiratory tract infections. Chest pain, though seen in-

frequently, may result from microvascular dysfunction in early dilated cardiomyopathy.<sup>1</sup> The combination of chest pain, normal coronary arteries, and an abnormal ECG merits consideration of detailed investigation<sup>2</sup> and close follow-up even if initial investigation is unremarkable. Other nonspecific but important symptoms in this demographic are generalized fatigue, exertional dyspnea despite a relatively preserved exercise capacity, and upper abdominal fullness. It is not infrequent to see new referrals in our heart failure clinic where pulmonary edema or cardiac chamber dilatation are detected on an upper abdominal computed tomography scan performed for chronic abdominal pain.

The transient nature of these symptoms further challenges clinicians, as the patient will often look well at the time of clinical assessment. Many clinicians will have a low index of suspicion of heart failure in relatively young patients with transient symptoms, meaning that opportunities to diagnose are missed. Under a busy clinical workload, how can we ensure that critical time is not lost due to diagnostic delay? The key components must be a greater awareness and consideration of the condition, coupled with the use of B-type natriuretic peptide (BNP) screening tests. The use of BNP measures to make timely diagnoses prevents hospitalizations and critical care admissions of the sort seen in this case, in turn significantly reducing the cost of care.<sup>3</sup> There is evidence that even in mildly symptomatic, well-compensated patients, the use of disease-modifying therapy is strongly associated with positive clinical outcomes.<sup>4</sup>

It is notable that this patient presented with significant hypertension. This is a positive prognostic



**Fig. 2** Cardiac magnetic resonance image in the apical four-chamber view, demonstrating left ventricular dilatation. The right ventricle is of normal size, and there was no evidence of late gadolinium enhancement to suggest a cardiomyopathic process.

sign in patients with systolic heart failure, predicting a good treatment response.<sup>5</sup> Hypertensive patients have greater capacity to tolerate vasodilation and therefore uptitration of medication to optimal doses.

Even with a prompt diagnosis and the initiation of gold-standard pharmacological therapy, a positive outcome will still not be achieved if the patient does not engage with treatment. This is the true real-world problem of managing heart failure. Reported rates of nonadherence vary widely, but average around 30%, with associated costs in both rehospitalization and outcomes.<sup>6</sup> Attendance at follow-up for disease surveillance is equally important, though less widely investigated and reported than medication adherence. Missed reviews result in suboptimal therapy, and adverse outcomes go unnoticed.

These factors contribute significantly to the difference in mortality observed between randomized trials and real-world registries. Trial patients derive significant benefit from often-mandated guideline-directed medical therapy, structured follow-up, and close monitoring for adverse outcomes. There can be no denying that adopting these processes in the real world is challenging, but there is good evidence that such integrated management can break a cycle of repeated hospitalization and improve clinical outcomes.<sup>7</sup> ■

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