Chronic heart failure: epidemiology, investigation and management

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Abstract

Heart failure (HF) is a clinical syndrome characterized by dyspnoea, fatigue and fluid retention accompanied by objective evidence of cardiac dysfunction. The syndrome affects around 2% of the adult population, men more commonly than women (<80 years old), with the incidence and prevalence rising steeply with age. HF causes substantial morbidity and reduced life expectancy, and coronary artery disease accounts for two-thirds of cases in developed countries. Investigation is important to ascertain the diagnosis, identify the aetiology (which may be reversible) and give some indication of prognosis. The diagnosis of HF confers a significantly increased risk of hospital admission and death. Treatment has been revolutionized by large randomized controlled clinical trials studying the effects of antagonists of the renin–angiotensin–aldosterone, neutral endopeptidase and sympathetic nervous systems, and the effects of device therapy. Cardiac transplantation remains an option for patients who are severely symptomatic (and at high risk) despite optimal use of such therapy.

Keywords Cardiomyopathy; epidemiology; heart failure; investigation; MRCP; prognosis; treatment

Key points

- Heart failure (HF) is a syndrome with myriad causes; any reversible cause should be treated
- All patients with HF with reduced ejection fraction (HFrEF) should be treated with an angiotensin-converting enzyme inhibitor and (when euvolaemic) a β-adrenoceptor blocker
- Patients with severe left ventricular systolic dysfunction should also be given a mineralocorticoid receptor antagonist
- Sacubitril-valsartan is indicated for patients with HFrEF who remain symptomatic despite optimal treatment
- Device therapy is suitable for selected patients with HF: cardiac resynchronization therapy, implantable cardioverter-defibrillators and left ventricular assist devices
- Cardiac transplantation remains an option for those patients who worsen or fail to improve despite maximum disease-modifying therapy

Introduction

Heart failure (HF) is a clinical syndrome characterized by dyspnoea, fatigue and fluid retention accompanied by objective evidence of cardiac dysfunction. HF with reduced ejection fraction (HFrEF) is usually relatively easy to diagnose using a range of non-invasive methods. However, the diagnosis of HF with preserved ejection fraction (HFpEF) is more difficult, and while it is often attributed to diastolic dysfunction, abnormalities of systolic and diastolic function frequently coexist.

Epidemiology

Prevalence

Population-based studies suggest a prevalence of HF of 2–3%, increasing to 7% in elderly individuals. The prevalence of HFpEF has been estimated at 9.7/1000 (44% of total HF prevalence). The prevalence of chronic HF is projected to increase by 50% in the next 20 years because of:

- an ageing population
- improved survival from other cardiovascular diseases
- improved survival rates for HF itself.

Incidence

UK population data report an annual incidence of 0.12% in ages 55–64 years, rising to 1.2% in those aged >85 – equivalent to 63,000 new cases of HF each year. Median age at diagnosis is 76 years, with a higher incidence in men than women at all ages (M:F around 1.8:1).

Prognosis

Recent European data suggest that all-cause mortality rates at 1 year are 17% and 7% for HF patients who are hospitalized and ambulatory, respectively. Equivalent rates for hospital admission are 44% and 32% in these groups.[1]

Investigation

Investigation is important to ascertain the diagnosis, aetiology and prognosis. The most common investigations are as follows.

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Electrocardiography – mandatory; it is abnormal in >90% of individuals with HF. Common abnormalities include:

- **heart rate** – bradycardia (<60/minute) is common in patients taking a β-adrenoceptor antagonist and requires further investigation and management if associated with Mobitz type II or complete heart block. Tachycardia (>100/minute) is a possible cause, although more likely a consequence, of HF
- **atrial fibrillation (AF)** – this has a prevalence of 10–50% in HF
- **intraventricular conduction delay** – QRS duration >120 ms confers an increased risk of death. QRS>130 ms may identify patients for cardiac resynchronization therapy (CRT)
- **regional versus local changes** – this suggests underlying coronary artery disease
- **left ventricular hypertrophy** (e.g. secondary to hypertension).

Chest radiography – also mandatory:

- a normal chest X-ray does *not* exclude HF.
- cardiomegaly (cardiothoracic ratio >0.50), plus evidence of pulmonary congestion, suggests a cardiac abnormality.
- it can identify other causes of breathlessness.

Blood tests –

- **urea and electrolytes** – HF is commonly associated with renal impairment and electrolyte disturbance; hyponatraemia indicates an adverse prognosis
- **full blood count** – anaemia is common in HF and associated with adverse prognosis
- **ferritin and iron studies** – haemochromatosis is an uncommon but reversible cause of HF. Iron deficiency is important to identify because intravenous iron supplementation may be appropriate (and of objective benefit)
- **liver function tests** – these can be abnormal in the presence of hepatic congestion
- **thyroid function tests** – thyrotoxicosis can cause left ventricular systolic dysfunction (LVSD) with or without AF, and hypothyroidism can accelerate coronary artery disease
- **urate** – this is often elevated in patients taking diuretics; hyperuricaemia is associated with an adverse prognosis
- **Brain natriuretic peptide** (BNP; also N-terminal prohormone of BNP (NT-proBNP)) – this is useful in diagnosis (strong negative predictive value) (Figure 1) and determining prognosis.

Echocardiography – an accessible and non-invasive diagnostic test that can identify (and in some instances quantify) the following aspects of cardiac function:

- left ventricular dimensions and systolic function
- regional or localized wall abnormalities, suggestive of underlying coronary artery disease
- valve function
- right ventricular function
- estimation of pulmonary artery pressure
- presence or absence of a pericardial effusion
- markers of diastolic dysfunction.

Cardiac magnetic resonance – the gold standard for measurement of cardiac volumes and left ventricular mass. It helps to characterize cardiac tissue and identify areas of infarction or infiltration.

Other investigations may be useful:

- **coronary angiography** – coronary artery disease is a potentially reversible cause of cardiac dysfunction. The STICHES trial has suggested that surgical revascularization in HF with minimal chest pain offers a prognostic advantage over medical therapy alone over the longer term (around 10 years)
- **cardiopulmonary exercise testing** to quantify peak VO_2 (a useful prognostic marker)
- **genetic testing**
- **right heart catheterization** if candidacy for heart transplantation is being considered
- **endomyocardial biopsy** if an infiltrative or rapidly progressive myocarditic process is a possibility.

Therapeutic options

The medical treatment of chronic HF has been revolutionized by large randomized controlled clinical trials studying the effects of antagonists of the renin–angiotensin–aldosterone, neutral endopeptidase and sympathetic nervous systems (Figure 2). Once established, HF is usually associated with poor prognosis and disabling symptoms, so therapy aims to reduce mortality and improve morbidity.

Primary prevention

It is of paramount importance to realize that prevention is better than cure. Treatment is recommended for patients with hypertension and with, or at high risk of, coronary artery disease, to delay or prevent the onset of HF. Treatment of other risk factors such as obesity and diabetes mellitus should be considered. In type 2 diabetes mellitus, sodium-dependent glucose transport protein-2 (SGLT2) inhibitors such as empagliflozin can delay the onset of HF and thus warrant specific consideration. In the setting of asymptomatic LVSD, angiotensin-converting enzyme (ACE) inhibitors are indicated, with the addition of a β-adrenoceptor blocker where the aetiology is ischaemic.

Disease-modifying therapy

ACE inhibitors

These are first-line agents that should be given to all patients with LVSD, whether symptomatic or not, combined with a diuretic if there is evidence of cardiac decompensation. In large clinical trials, ACE inhibitors achieve an average 20–25% relative risk reduction in morbidity and mortality. Their use is mandatory unless there is a firm contraindication – such as significant renal disease, angioedema or ACE inhibitor-induced cough – or the patient is taking sacubitril-valsartan (see below). Drugs with proven efficacy in clinical trials are enalapril, captopril, ramipril, lisinopril and trandolapril.

β-adrenoceptor antagonists

There is unequivocal evidence that the β-blockers bisoprolol, carvedilol and sustained-release metoprolol provide both a significant mortality and long-term symptomatic benefit in patients with all grades of HF, and in LVSD after myocardial infarction (MI). It is imperative that β-blockers are started only when patients are euvolaemic, and then up-titrated slowly – 'start low, go slow'. Chronic obstructive airways disease without airways

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