

Chronic heart failure

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Keywords: acute myocardial infarction, diabetes mellitus, heart failure, hypertension, obesity

Abstract

Heart failure is a global problem. It is estimated that approximately 38 million people worldwide experience heart failure, and this number is growing because of the ageing of nations' populations, but also because patients are rescued from death during an acute myocardial infarction, and later develop heart failure. The prevalence of heart failure is roughly 2% of the population, and up to 10% in people aged 75 years and older. It is also estimated that the lifetime risk of heart failure at the age of 40 years is approximately 20% (one in five) for both men and women. Heart failure is also increasing in low- and medium-income countries, probably because of changes in lifestyle which favour the development of obesity, hypertension and diabetes mellitus. The mortality of heart failure is worse than that associated with most cancers. Average mortality is roughly 50% within five years. However, evidence-based treatment did reduce mortality significantly. About two thirds of the economic burden of heart failure is owing to hospital admissions and re-admissions. Therefore, heart failure is also an expensive disease.

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Prof Nurs Today 2017;21(1):40-42

Introduction

Heart failure is a global problem. It is estimated that approximately 38 million people worldwide experience heart failure, and this number is growing because of the ageing of nations' populations, but also because patients are rescued from death during an acute myocardial infarction, and later develop heart failure.¹ The prevalence of heart failure is roughly 2% of the population, and up to 10% in people aged 75 years and older. It is also estimated that the lifetime risk of heart failure at the age of 40 years is approximately 20% (one in five) for both men and women. Heart failure is also increasing in low- and medium-income countries, probably because of changes in lifestyle which favour the development of obesity, hypertension and diabetes mellitus.² The mortality of heart failure is worse than that associated with most cancers. Average mortality is roughly 50% within five years. However, evidence-based treatment does not reduce mortality significantly. About two thirds of the economic burden of heart failure is owing to hospital admissions and re-admissions.³ Therefore, heart failure is also an expensive disease.

Pathophysiology

The first step in the development of heart failure is an injury to the myocardium, such as an ischaemic episode, left ventricular hypertrophy or injury due to a valvular problem. This myocardial injury leads to reduced cardiac output. The body responds by activating compensatory mechanisms [renin-angiotensin-aldosterone system (RAAS) and sympathetic stimulation] which restore the cardiac output. Unfortunately, the compensatory mechanisms are beneficial in the short term only, and damage the myocardium after time, causing other cellular and molecular reactions, such as apoptosis, remodelling and inflammatory cytokines, all which further damage the myocardium, and affect the whole circulation, e.g. vasoconstriction. Eventually, heart failure is the end result. The compensatory mechanisms are maladaptive in the long run. This concept has led to the modern treatment of heart failure by blocking the compensatory mechanisms with RAAS blockers [angiotensin-converting enzyme (ACE) inhibitors, angiotensin-receptor blockers (ARBs) and aldosterone antagonists], as well as beta blockers, which are associated with a reduction in morbidity and mortality.³

Clinical picture

The symptoms of heart failure are not very specific. Dyspnoea, effort intolerance and swelling of the limbs are associated with it, but can be caused by other conditions. Classical physical signs, such as elevated jugular venous pressure, bilateral lung crepitations and a third heart sound gallop, are more predictive, but none of them are 100% diagnostic. Therefore, a clinical diagnosis of heart failure can be missed.³

Co-morbid diseases are important as precipitants of heart failure. They also cause heart failure, contribute to exacerbations thereof, and to its progression, and ultimately to mortality and morbidity. Hypertension, coronary artery disease, diabetes mellitus, cardiac arrhythmias (e.g. atrial fibrillation), chronic obstructive pulmonary disease, cognitive dysfunction, depression, chronic kidney disease and arthritis are important co-morbidities.

Diagnostic tests

Diagnostic tests include the following:

- An electrocardiogram (ECG) is almost always abnormal (always some abnormality) in heart failure with reduced ejection fraction. A normal ECG should prompt an alternate diagnosis.
- A chest X-ray is important to exclude other diseases that cause dyspnoea, and it can be used to measure an enlarged cardiac size.
- Brain natriuretic peptide (BNP) and its precursor, N-terminal pro-brain natriuretic peptide (NT-proBNP), are useful for excluding heart failure (negative predictive) in patients with dyspnoea in whom the diagnosis of heart failure is uncertain. It has a predictive power of 90% in this situation (c-statistic 0.90). It is a useful rule-out test when normal. These peptides can be falsely raised by advanced age, the female sex and renal impairment, and are falsely lowered by obesity. Nevertheless, it is a useful blood test in the diagnosis of heart failure. The level of BNP/NT-proBNP is increasingly used to monitor the treatment of heart failure.
- Echocardiography is important as it can contribute to diagnosing the cause of heart failure, i.e. it can exclude pericardial disease. The lesson to be learnt from echocardiography is that heart failure can have a reduced ejection fraction (HFrEF), and also a normal (preserved) ejection fraction (HFpEF), and both are clinically identical, with the same symptoms and signs. Currently, HFrEF is used as treatment to successfully reduce mortality.

Stages of heart failure

A useful concept is to think of heart failure as developing and progressing in stages.⁴

Stage A

People with no symptoms and no signs of heart failure have a normal heart both structurally and functionally in stage A. They do have risk factors for heart failure, such as hypertension, diabetes mellitus, hypercholesterolaemia, a family history of cardiomyopathy, and the use of toxins, i.e. alcohol, chemotherapeutic drugs and cocaine. Treatment of the risk factor may reduce the risk of progressing to stage B. For instance, the treatment of hypertension can reduce the relative risk of heart failure development by almost 50%.

Stage B

People have an abnormal cardiac structure [previous myocardial infarction, valvular problem (heart murmur) or left ventricular hypertrophy] in stage B. They may also have functional abnormality in the form of an elevated BNP/NT-proBNP level. People in stage B are still asymptomatic, and do not have signs of heart failure. These people have already progressed from stage A to B, and even with treatment, they will not return to stage A.

Stage C

Patients have progressed to the clinical picture of heart failure, with the symptoms and signs thereof, in this stage.

Stage D

Stage D implies heart failure which did not respond to treatment.

This staging system is useful in showing how heart failure is progressive and advances from stage A to B to C to D, and that prevention in the early stages (A and B) may reduce the risk of progression to clinical heart failure (C and D). There is evidence that treatment during stage B with a RAAS blocker or a beta blocker may reduce the risk of progression to stage C.

Management of heart failure

There are at least five critical issues in the management of heart failure.

Symptomatic treatment

The most critical treatment with which to reduce the symptoms of heart failure is the use of loop diuretics to reduce congestion in the lungs and limbs. Furosemide (Lasix®) is commonly used with doses administered according to the severity of oedema. Physical activity is recommended for all stable patients as it improves well-being. Salt restriction is recommended, but the evidence for this is limited. Low-quality data suggest that nutrients, such as coenzyme Q10 and micronutrients, may be of benefit. Psychosocial support and the treatment of depression are becoming important issues.

Reduce mortality

All patients with heart failure should receive a RAAS blocker (ACE inhibitor or ARB),⁵ together with a specific type of beta blocker (bisoprolol, metoprolol and carvedilol), as some beta blockers do not reduce mortality and some have never been tested. The ACE inhibitor should be started at a low dose, and gradually increased over weeks until the full dose is reached, e.g. enalapril 2.5 mg twice daily for a few days, and then 5 mg twice daily for as long as it takes for the blood pressure to stabilise. Thereafter, the dose must be increased to 10 mg twice daily, and even to 20 mg twice a day after several months.

Many people start to add the beta blocker even before the maximum dose of the ACE inhibitor has been reached. Some specific patients may also be placed on aldosterone antagonists (e.g. in cases of heart failure after an acute myocardial infarction), or on a combination of hydralazine plus Isordil® (isodinitrate), especially in black patients and in those who are still symptomatic after the administration of a full-dose ACE inhibitor and beta blocker. Recently, a combination of a neprilysin inhibitor and valsartan (ARB) reduced mortality more than an ACE inhibitor (enalapril). This is a new development in the treatment of heart failure, and if confirmed, may change the treatment of heart failure completely. The serum levels of urea and potassium should be regularly measured.

Intracardiac defibrillator implantation has also been shown to reduce mortality in appropriate patients, i.e. those with an arrhythmia. The implantation of biventricular pacemakers has been shown to reduce mortality significantly in many clinical trials, and should be considered. The significant mortality reduction with these drugs has only been shown in HFrEF. Currently, no specific therapy can reduce mortality significantly in HFpEF, and it is important to treat the comorbidities, especially hypertension, which may impact on mortality.

Find the cause and treat it

Important issues are to find those patients who may benefit from surgery, i.e. coronary artery bypass grafting, valve replacement and pericardectomy. Testing for other treatable causes of heart failure is also important, e.g. sarcoidosis.

Find the precipitant and co-morbid disease and treat it

Conditions such as anaemia, abnormal thyroid function, electrolyte disturbances, renal dysfunction and infection need to be identified and treated. Elevated blood pressure is often present and may be the precipitant.

Prevention

Prevention in stages A and B is important in reducing the development of heart failure (stage C), and in averting another acute exacerbation episode of heart failure. It is important to ensure that patients are on the correct treatment, and that the doses of the drugs are adequate, as this may reduce the risk of acute exacerbation of heart failure.

Conclusion

Heart failure is a common deadly condition. The correct treatment can reduce morbidity and mortality.

Prevention of heart failure is important. The focus must be on stages A and B to reduce progression to symptomatic heart failure (stage C).

Inhibition of the RAAS with an ACE inhibitor, and ARB, when the patient is ACE intolerant, is critical in the treatment of heart failure.

The addition of beta blockers to the ACE inhibitor is also critical.

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