Heart failure

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Abstract

Heart failure is a clinical syndrome that can result from any structural or functional cardiac disorder that impairs the performance of the ventricle: either to eject blood (systolic dysfunction) [or reduced ejection function] or to fill with blood (preserved ejection fraction or diastolic heart failure).

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What is new?

About half of all clinical heart failure patients have preserved ejection fraction and this condition seems to be increasing in prevalence, especially with increasing age. The most effective therapy for this form of heart failure is currently unknown.

Comorbidities have a large influence on worse outcome in heart failure. Systemic hypertension, ischaemia of the ventricle, diabetes mellitus, chronic obstructive pulmonary disease, sleep apnoea, depression, anaemia and chronic renal disease add considerable complexities to diagnosis and management of heart failure.

Biochemical testing for heart failure

Measurement in plasma of brain natriuretic peptide or its precursor, N terminal proBNP (NT-proBNP) has aided in the diagnosis of heart failure. An elevated NT-proBNP is caused by stretching of cardiac myocytes and therefore the test cannot distinguish between left ventricular failure or right ventricular failure from other causes. A low BNP (or NT-proBNP) has a high negative predictive value, making it a useful rule-out test.

Stages of heart failure

There are four recognisable stages in the progression of heart failure.

Stage A: There are no symptoms or signs of heart failure. The heart has a normal structure and function, but the patient has *risk factors* for heart failure: hypertension, elevated cholesterol, diabetes, alcohol abuse, cardiotoxic drugs (e.g. chemotherapeutic drugs, cocaine, etc).

Stage B: There are no symptoms or signs but the heart of the patient has a structural abnormality (e.g. left ventricular hypertrophy, myocardial infarction, heart murmur, LV dilatation) or is functionally abnormal e.g. reduced ejection fraction, and diastolic dysfunction.

Stage C: There are now symptoms and signs of heart failure.

Stage D: These patients have progressed and still have marked/resistant symptoms despite maximal medical therapy.

Therapy for heart failure (What works?)

Physical activity is now recommended for all heart failure patients, except for those who are acutely decompensated.

Prevention of heart failure is possible through adequate blood pressure control in hypertensives, control of ischaemic heart disease occurrence and diabetes mellitus [*Stage A*]. Preventative treatment with an angiotensin-converting enzyme inhibitor (ACE-I) or angiotensin-receptor blocker (ARB) are given to individuals with high risk but normal ejection fraction (HOPE study; ONTARGET) or those with asymptomatic left ventricular dysfunction [*Stage B*]. Treatment post myocardial infarction with a beta-blocker [*Stage B*] can also slow progression of heart failure. For *Stage C* heart failure (symptoms and signs of heart failure),

There's no need to treat your patient's risk factors like different patients.

Hypertension and dyslipidaemia are relatively well-diagnosed, but only 9% of patients with both risk factors are controlled [1]

Caduet®:

- Targets 2 modifiable risk factors simultaneously, reducing risk of CV events [2]
- The coadministration of Norvasc® and Lipitor® results in significant reductions in non-fatal MI and fatal CHD [3]
- Simplifies treatment, which may lead to improved adherence and increased patient satisfaction [4]

☑ Hypertension
☑ Dyslipidaemia
= **Caduet®** amlodipine besylate/atorvastatin calcium

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neuro-hormonal inhibitions for patients with reduced ejection fraction has mortality benefits. ACE-inhibitors, ARBs, beta-blockers (bisoprolol, metoprolol), alpha-beta blockers (carvedilol) and aldosterone antagonists all are evidence-based therapies for mortality reduction. Cardiac resynchronisation therapy for heart failure with biventricular pacemakers with or without intracardiac defibrillators are recent additions to mortality reduction therapy. Isobide dinitrate plus hydralazime therapy have mortality benefits especially in African patients, but can be tried in others as well.

What does not reduce mortality? (What does not work?)

Statins given purely for heart failure do not reduce mortality. Digoxin, once the standard treatment, has not reduced mortality compared to placebo, in patients with low ejection fraction and in patients with normal ejection fraction. Digoxin is used to control atrial fibrillation should it occur in heart failure.

Calcium channel blockers also have no mortality benefit.

Inotropic therapy is associated with increased mortality.

What is the problem with treatment?

The majority of the randomised clinical trials in heart failure were done on patients with reduced ejection fraction. These patients have improved mortality on standard anti-neuro-hormonal therapy.

However, the therapy of heart failure patients with preserved ejection fraction has very little evidence. The few trials done in this condition have not shown the same mortality benefit. Much work needs to be done for patients with heart failure and preserved ejection fractions (almost half of all heart failure patients).

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