Heart Failure

Author: Dr Steven Brady (ASH)

Topic Reviewers: Dr Marcus Ilton (RDH Cardiologist); Dave Corstorpan (RAN, Nyirripi Clinic)

In the last few years there has been the release of several highly detailed guidelines for the diagnosis and management of chronic heart failure. These have included guidelines from Australia[1], the USA[2] and Europe.[3] They give a detailed description of the clinical trials which have led to marked changes in the management of chronic heart failure, from one based on symptom control to one based on accurate diagnosis and a use of pharmacotherapy and neurohormonal control to improve survival.

The guidelines have very broad agreement about the primary therapies used for the management of chronic heart failure these being ACE inhibitors, beta-blockers, diuretics and digoxin and non-pharmacological measures. The guidelines also discuss in detail the approach to diagnosis of heart failure, which still remains quite problematic, particularly in a primary care setting.

The discussion here highlights the important areas that may be problematic to implement in a very scattered population with scarce resources.

Overview of diagnosis

Failure of heart to pump sufficient blood for metabolic requirements

The diagnosis of heart failure remains difficult. Surveys in primary care settings highlight the difficulties of diagnosis and the limited use of echocardiography.

Signs and symptoms of heart failure are not adequately sensitive and specific to confirm the diagnosis of heart failure in most circumstances. Some clinical signs, including a gallop rhythm and raised JVP, both confirm the diagnosis of heart failure and have implications for prognosis but are not reliable except in the hands of experienced clinicians.

Heart failure should, however, be suspected in anyone presenting with a history of new onset fatigue, oedema or breathlessness. This is particularly the case if the patient has a background of diabetes, chronic renal impairment, ischaemic heart disease, hypertension or rheumatic valvular disease.

Initial tests should include an ECG. A normal ECG makes the diagnosis of heart failure unlikely. A chest X-ray is valuable if able to be performed. Neither the ECG nor chest X-ray are sensitive or specific enough to form the sole basis of investigation.

Biochemical markers such as Brain Natriuretic Peptide may become available as a screening tool in primary care to enable the detection of patients with heart failure, especially where echo-cardiography is not easily available and may be suitable as an initial test[4,5,6,7] and to guide

therapy or further investigation. However, this is yet to be established in a primary care setting.

Echocardiography remains an essential part of the diagnosis of heart failure. Given the very high incidence of rheumatic valvular heart disease in the Northern Territory[8], whenever possible an echocardiogram should be performed to confirm the diagnosis.

Coronary artery disease is also very common in the Northern Territory. Given the high prevalence and the early age of onset of ischaemic heart disease in this population[9], coronary artery disease needs to be excluded as a cause of heart failure in people presenting with left ventricular systolic dysfunction, if no other cause is apparent. Exercise impairment, regional wall motion abnormalities and ECG abnormalities make interpretation of the exercise stress test and other non-invasive tests difficult. In the population with LV systolic impairment or an ideopathic dilated cardiomyopathy, coronary angiography is the preferred test to exclude significant ischaemic heart disease.[2] Patients with angina and heart failure should undergo coronary angiography as revascularisation of appropriate patients will prolong life and may result in improved LV function. Other patients with unexplained heart failure may also require angiography to exclude significant coronary artery disease, although the efficacy of revascularisation in improving symptoms and survival is less clear cut.[2]

Pharmacological treatment

Treatment

Treatment is based on the treatment of acute episodes of acute pulmonary oedema (or sudden cardiac decompensation), control of possible triggers of cardiac decompensation and maintenance therapy to maintain adequate cardiac function for usual daily activities and to reduce gradual decline in cardiac function.

The mainstays of pharmacological treatment are ACE inhibitors and beta-blockers to improve survival, decrease hospitalisations and diuretics and digoxin as symptomatic therapy. The aldosterone antagonist spironolactone has also been shown to improve survival in patients with severe (NYHA III & IV) heart failure.

New York Heart Association grading of symptoms in heart failure:

Class I: Cardiac disease, but ordinary activity causes no symptoms

Class II: Slight limitation, with ordinary activity causing symptoms

Class III: Marked limitation, with symptoms on less than ordinary activity

Class IV: Unable to carry on any activity without symptoms and may have symptoms at rest

Treatment of acute pulmonary oedema

Intravenous frusemide has been the mainstay of treatment for acute pulmonary oedema but has been subjected to few randomised controlled trials. One recent trial[10] randomised patients to high-dose intravenous nitrates and low-dose frusemide versus high-dose frusemide and low-dose intravenous nitrates. There were fewer requirements for mechanical ventilation and less progression to myocardial infarction in the group receiving high-dose nitrates, suggesting a benefit for the high-dose nitrates group. Whether this may be due to beneficial effects of the nitrates or deleterious effects of the frusemide is unclear.

In practice nitrates may be (are and should always be considered in normotensive or hypertensive patients with LVF) useful in treating acute pulmonary oedema.

Beta-blockers

Beta-blockers have been shown to improve survival in patients with mild to severe symptomatic heart failure and should be used in patients with LV systolic failure in the absence of any contraindication.[11] Patients with LV systolic dysfunction and no symptoms are likely to benefit from beta-blockers, but this has not been confirmed in clinical trials. Patients with known ischaemic heart disease should be on beta blockade (particularly if they have evidence of heart failure) and more widespread therapy with beta-blockers may decrease the incidence of LV failure.

The benefits of beta blockade occur slowly and may follow an initial decline in LV function and increase in symptoms. As a rule of thumb, LV function declines for a month after initiation of therapy and thereafter improves. Improvement in survival can be demonstrated within six months.

Patients must therefore be stable to begin therapy. Compliance is also important, as patients who stop and start therapy are more likely to experience harm than good. Carvidelol is licensed in Australia for the treatment of heart failure after specialist initiation and this would appear appropriate for the CARPA protocol. In stable patients this can be achieved with outpatient and outreach supervision (possibly over the phone) and does not require hospitalisation.

Overall long-term success with beta blockade is achieved in the same percentage of patients with diabetes and COPD as in those without, so these are not contraindications for use. One authority recommends avoiding use in patients with true asthma (usually have childhood symptoms), severe airways obstruction (FEV1<50%) or evidence of reversibility on pre- and post-bronchodilator spirometry. Patients with marked hypotension (SBP <90) and bradycardia should not be started on beta-blockers. Patients with borderline low blood pressures may require reduction in other therapies (particularly diuretics) to allow the introduction of beta blockade.

ACE inhibitors

ACE inhibitors have been shown to decrease progression and mortality in all stages of heart failure and also led to a reduction in the risk of heart failure. Widespread use in at-risk patients may reduce the numbers of patients developing heart failure.[12]

Angiotensin receptor blockers

Angiotensin receptor blockers (ARBs) remain second-line therapy behind ACE Inhibitors for the treatment of CCF.[1-3] Placebo-controlled trials have shown clinical and neurohormonal benefits. Trials comparing ARBs to ACE inhibitors have shown a trend to improved survival with ACE inhibitors.[3,13,14] The recent Val-Heft trial[15] compared valsarten or placebo to standard therapy for heart failure including beta-blockers and ACE inhibitors. No overall advantage on mortality was demonstrated. Valsarten improved mortality in patients receiving ACE inhibitors and beta-blockers but worsened mortality in patients receiving both of these drugs in post hoc analysis.

Therefore ARBs have a role in patients unable to take ACE Inhibitors because of angio-oedema or cough.[2] (Although the AMH defines ACEi-induced angioedema as a contraindication to using an ARB the American Heart

Association list this as a Class 1 indication for ARB. The low likelihood of angioedema and the life-saving nature of neurohormonal inhibition leads me to agree with the ACC/AHA, i.e. to recommend ARBs as the best alternative to ACEi if there is heart failure and angioedema or cough associated with use of ACEi.)

The advantage of combining ACE inhibitors and ARBs in patients unable to take beta-blockers will need to be confirmed in prospective trials.

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