Management of congestive heart failure: How well are we doing?

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See related article page 284

We are making great progress in controlling the epidemic of coronary artery disease that plagued much of the last century. Incidence has declined, and treatment of acute coronary artery disease has resulted in a greatly increased life expectancy. However, improved treatment may have reduced immediate mortality, but it often leaves patients with chronic myocardial dysfunction. This is an important factor contributing to the increased incidence of congestive heart failure (CHF), the cardiovascular condition most rapidly on the rise.

CHF currently affects an estimated 200 000 to 300 000 Canadians.[1] The morbidity and mortality associated with this condition are substantial. In the most severely affected patients the 1-year death rate can be as high as 40%.[2] In all affected patients the 6-year death rate ranges from 65% to 80%.[3] Furthermore, because the incidence of CHF increases with age and our elderly population is growing, we can expect a heavier burden in the future.

Guidelines have been established for the evaluation and treatment of CHF.[4] These recommendations include assessment for the underlying cause, determination of left ventricular function to distinguish between diastolic and systolic dysfunction, and recommendations for the use of medications. For patients with CHF, particularly those with left ventricular dysfunction, there is overwhelming evidence that appropriate management can alleviate symptoms and enhance survival.

Transthoracic Doppler 2-dimensional echocardiography is a particularly helpful diagnostic tool to determine which patients have left ventricular dysfunction. Once a diagnosis of systolic dysfunction is established, the next step involves patient education and the introduction of drug therapy. Patient education aims to achieve lifestyle modifications, such as proper diet (including sodium and fluid restriction), weight loss and a program of graduated exercise. In the appropriate context, these activities in conjunction with drug therapy have been shown to improve outcome.[5]

Drug therapy includes the use of time-tested medications, such as diuretics and digitalis, and the addition of newer agents, such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, β-blockers and spironolactone. The cornerstone of medical management of CHF is the ACE inhibitor class of drugs. When used with conventional therapy (diuretics and digoxin), ACE inhibitors have been clearly shown in randomized trials to improve survival among symptomatic patients who have documented left ventricular dysfunction.[6,7]

Despite these incontrovertible data, several reports have shown that ACE inhibitors are frequently underused and, when prescribed, often underdosed.[8–10] The reasons for this are multifactorial. In one study, patients followed by cardiologists were more likely to be prescribed an ACE inhibitor than were those managed by primary care physicians, in part because the primary care physicians were less likely to perform imaging procedures that document left ventricular dysfunction.[11] In another study, patients with renal failure were less likely than other patients to be prescribed an ACE inhibitor, for fear of worsening renal function.[9] Significant worsening of renal failure is rare and may be due to bilateral renal artery stenosis. ACE inhibitors are not contraindicated in renal failure unless it is associated with elevated potassium levels (greater than 5.0 mmol/L). Nonetheless, these drugs should be used with caution. Patients with renal failure require careful monitoring of renal function and electrolyte levels beginning as early as 2 weeks after initiation of ACE inhibitor treatment. Studies also reveal that older patients appear less likely than younger patients to be prescribed an ACE inhibitor, mainly because of concern about adverse drug effects.[8,9] Aside from renal failure, the main adverse effects are angioedema, which is rare (occurring in less than 1% of treated patients), and dry cough, which is more common (occurring in about 5%–10% of patients). Neither of these adverse effects has been shown to occur more often in older patients.

Nonetheless, regardless of patient and physician characteristics, ACE inhibitors are frequently underdosed relative to doses used successfully in clinical trials. This has been ascribed to concern over another adverse effect, hypotension.[12] In clinical studies asymptomatic hypotension did not prevent use of ACE inhibitors at target doses as long as there was no evidence of end-organ (e.g., kidney) dysfunction.[13] As a general rule, asymptomatic systolic blood pressure values over 80 mm Hg are acceptable. When hypotension becomes problematic, decreasing the diuretic dose may help to raise the blood pressure. Of interest, one study showed that optimized dosing aided by the inclusion of a pharmacist in the multidisciplinary team managing CHF translated into reduced rates of clinical events.[14]

In this issue of *CMAJ* (page 284) Evette Weil and Jack Tu[15] report on the management of CHF in a large teaching hospital in Toronto, as assessed by a retrospective review of the charts of 200 patients. Their findings are encouraging. Close to 90% of the patients had left ventricular function assessment by echocardiography. Almost 90% of the patients who were deemed ideal candidates for treatment with ACE inhibitors were prescribed these medications. These 2 figures exceed proportions reported in series from the United States and elsewhere.[8,9]

In contrast, Weil and Tu found that only 23% of the patients considered ideal candidates received doses of ACE inhibitors used in clinical trials. As suggested by the authors, underdosing of these drugs may have resulted from patient characteristics (e.g., age and concomitant diseases) or from physician concern regarding adverse effects. Another possible explanation comes from the recognition that titration of ACE inhibitors is frequently performed over weeks in an outpatient setting. Because the patients in this report were admitted to hospital, appropriate doses may have been subsequently realized on outpatient follow-up. It would be interesting to learn whether the underdosing reported by Weil and Tu continued through time or whether it was rectified by appropriate titration of the medication.

It will be important to build on the authors' observations to include not only the use of medications, such as ACE inhibitors, digoxin and β-blockers, but also nonpharmacologic aspects of this complex clinical problem. Nonpharmacologic causes of hospital readmission include gaps in treatment plan and follow-up after discharge from hospital as well as lack of patient education. Therefore, in patients who are admitted to hospital with CHF, we need to consider more than the assessment of underlying cause, the determination of left ventricular function and the initiation of drug therapy. We need to educate patients to understand their medical condition so that they know when it is appropriate to come to the hospital and why it is important to adhere to the treatment plan, be it pharmacologic intervention or lifestyle modifications.

Although the findings reported by Weil and Tu are promising, are they representative of all centres across Canada? Are the rates of adherence to CHF management guidelines the same in teaching and nonteaching hospitals? What about patients followed in specialty clinics? In order to provide patients with the best possible management of their CHF, we must continue to educate the health care team managing this condition of the importance of educating patients, encouraging lifestyle modifications and optimizing medical therapy. Outcomes-based research will be needed to determine the effect of the various interventions on patient outcomes, economic factors and delivery of health care services. With proper study of how to manage CHF and how this affects patients and our health care system, we should be able to meet the increased prevalence of CHF with rational and cost-effective solutions.

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