Management of Diastolic Heart Failure

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Case Presentation
A 70 year-old lady with known history of uncontrolled hypertension for years, presented with bilateral ankle swelling and impaired exercise tolerance. Her serum N-terminal pro B-type natriuretic peptide (BNP) was increased. Echocardiogram showed normal left ventricular systolic function but there was presence of left ventricular hypertrophy with diastolic dysfunction. Her symptoms were controlled with diuretics and angiotensin receptor blocker. However, she stopped the treatment by herself after her symptoms had improved. She was finally admitted to the hospital because of acute pulmonary oedema.

Epidemiology
Patients with diastolic heart failure (DFH) are not uncommonly encountered by primary care physician. About 30% to 55% of patients with heart failure have preserved systolic function, often defined as left ventricular ejection fraction (LVEF) greater than 40 to 50%1-3. In Asia, due to the high prevalence of hypertension, 50% of patients with heart failure have normal systolic function4, 5 and the incidence of DHF will further increase due to the aging population. The mortality of DHF is about 5-8% per year6, 7, which is about half of that of systolic heart failure. In a recent community-based prospective cohort of patients with heart failure, more than half (55%) had preserved systolic function and the mortality of DFH (16% at 6 months) was shown to be comparable to that of systolic heart failure8. The morbidity in terms of reduction in quality of life and exercise tolerance, hospitalisation rates and health-care costs per person for both systolic and DHF are similar9.

Pathophysiology
The clinical manifestations and haemodynamic consequences of systolic and DFH are similar, although the primary pathophysiology mechanisms are different. DFH is caused by left ventricular diastolic dysfunction, leading to increased resistance to left ventricular filling and eventually resulting in heart failure syndrome. Hypertension, diabetes mellitus, and coronary artery disease are common conditions that predispose to the development of DFH. Impaired ventricular relaxation and increased ventricular stiffness are the underlying mechanisms causing diastolic dysfunction. Activation of the renin-angiotensin-aldosterone system plays an important role in the development of myocardial fibrosis and stiffness10, 11. Inhibition of renin-angiotensin-aldosterone system has been demonstrated to reduce myocardial stiffness and leads to regression of myocardial fibrosis12.

Diagnosis of DHF
According to ACC/AHA guidelines, the diagnosis of DHF is based on the clinical findings of heart failure with the findings of preserved LVEF and the absence of valvular abnormalities13. The European guidelines require the finding of evidence of diastolic dysfunction14. Echo-Doppler assessment is a convenient and effective way of assessing diastolic function. Echocardiogram can also exclude other specific conditions, such as hypertrophic cardiomyopathy, aortic stenosis, infiltrative cardiomyopathies and pericardial disease. BNP, a cardiac neurohormone released by the ventricles in response to volume expansion and pressure overload, has recently emerged as a marker for heart failure15. BNP is elevated in systolic and DHF, though more markedly elevated in systolic heart failure8. The level of BNP correlates with severity of diastolic dysfunction and is highest among those with a restrictive filling pattern15. Study in Hong Kong has shown that N-terminal pro BNP can help in the diagnosis of DHF16. Several studies report the use of BNP in the diagnosis of systolic and DHF in the primary care, urgent care, and emergency department settings17, 18. It can be used as a screening test to rule out heart failure due to its high negative predictive value.

Principles of Treatment
The current strategy for the management of DHF focuses on symptom relief and modification of underlying causes of DHF. Diuretic therapy is required in symptomatic patients but should be used cautiously, as excessive diuretics may decrease cardiac output and cause hypotension and renal failure. Compared with systolic heart failure, DHF patients require lower doses of diuretics and may tolerate their withdrawal without increasing heart failure symptoms19. Tachycardia is very poorly tolerated in DHF, and in the presence of atrial fibrillation, adequate control of heart rate by beta-blockers or calcium channel blockers and maintenance of sinus rhythm are beneficial. Reduction in heart rate...
may be associated with improved ventricular filling and haemodynamics. Non-pharmacological measures such as salt restriction, weight control and exercise have been shown to reduce symptoms in patients with DHF. For primary care physicians, hypertension is the most common underlying cause of diastolic dysfunction; therefore, aggressive management of hypertension is essential in the prevention and management of DHF. In patients with coronary artery disease, therapies to relieve myocardial ischaemia are also likely to be beneficial.

Specific drugs

It is likely that most of the proven drugs used in treating systolic heart failure (ACEI, angiotensin receptor blockers, beta-blockers, aldosterone antagonists) may also be beneficial in the treatment of DHF. However, evidence-based treatment strategies for DHF are limited. Two large randomised controlled trials have recently provided some evidence for the treatment of DHF. The Candesartan in Heart Failure - Assessment of Reduction in Mortality (CHARM) - Preserved study is a randomized placebo-controlled trial of candesartan (with a target dose of 32 mg daily) in 3023 patients with DHF, NYHA II-IV and controlled systolic dysfunction. The primary endpoint was a composite of death from cardiac causes, heart failure hospitalisation and need for haemodynamics. Non-pharmacological measures such as judicious use of diuretics, rate control of atrial fibrillation, and aggressive control of hypertension. Limited evidence from randomised controlled trials suggested that angiotensin receptor blockers and ACEI are beneficial. More evidence-based treatment strategies to enhance the care of this condition will be available when some of the on-going clinical trials are completed.

Conclusion

HF is common and may account for more than 50% of heart failure cases among the elderly. The principles of treatment include symptom relief by judicious use of diuretics, rate control of atrial fibrillation, and aggressive control of hypertension. Limited evidence from randomised controlled trials suggested that angiotensin receptor blockers and ACEI are beneficial. More evidence-based treatment strategies to enhance the care of this condition will be available when some of the on-going clinical trials are completed.

References