

Heart failure with preserved ejection fraction: Diagnosis and treatment.

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Introduction

Heart Failure with Preserved Ejection Fraction (HFpEF), also known as diastolic heart failure, has become an increasingly recognized clinical entity over the past few decades. Unlike heart failure with reduced ejection fraction (HFrEF), where the heart's pumping function is impaired, HFpEF is characterized by a normal or near-normal ejection fraction (EF), but the heart is unable to fill properly during diastole. This article explores the pathophysiology, diagnosis, and current treatment strategies for HFpEF. HFpEF is primarily a disorder of diastolic dysfunction, where the heart's ability to relax and fill during diastole is compromised. The condition is often associated with increased ventricular stiffness, which leads to impaired ventricular filling despite a normal systolic function. [1,2].

Chronic hypertension, aging, and diabetes mellitus cause fibrosis in the myocardium, increasing stiffness and reducing the heart's ability to stretch and accommodate incoming blood. Structural changes in the left ventricle, including thickening of the walls and a reduction in chamber size, contribute to diastolic dysfunction. In HFpEF, the endothelium (the inner lining of blood vessels) becomes dysfunctional, leading to impaired vasodilation and increased vascular resistance. This makes it harder for the heart to pump blood efficiently. There is growing evidence that chronic low-grade inflammation and oxidative stress play a role in the development and progression of HFpEF. HFpEF is often seen in patients with comorbidities such as obesity, chronic kidney disease, and atrial fibrillation, which exacerbate the disease process. [3,4].

Diagnosing HFpEF can be challenging because the symptoms overlap with other forms of heart failure and the ejection fraction is often preserved. However, several diagnostic tools and criteria are now used to identify this condition. Patients with HFpEF often present with symptoms such as shortness of breath, fatigue, and exercise intolerance. These symptoms are typically exacerbated by physical activity or fluid overload. The most commonly used imaging modality in diagnosing HFpEF, echocardiography can show preserved ejection fraction, left ventricular hypertrophy, and diastolic dysfunction. Diastolic indices such as E/A ratio (early diastolic filling velocity to late diastolic filling velocity) and deceleration time help in assessing the severity of diastolic dysfunction. Blood biomarkers like B-type natriuretic peptide (BNP) and its precursor NT-proBNP are often elevated in HFpEF, though not specific. These biomarkers help assess the

severity of heart failure. Magnetic resonance imaging (MRI) is useful in evaluating myocardial fibrosis, ventricular stiffness, and assessing any myocardial ischemia or scarring. [5,6].

Functional assessment through exercise testing, such as the six-minute walk test, can assess exercise capacity and provide insights into the severity of symptoms. In some cases, a biopsy of heart tissue may be done to confirm the diagnosis, especially in patients with atypical presentation or those with suspected infiltrative cardiomyopathies. While there is currently no cure for HFpEF, management focuses on symptom relief, improving quality of life, and preventing disease progression. Treatment strategies are typically individualized, considering the patient's comorbidities and the underlying causes of the condition. Current approaches include. Weight reduction, regular exercise, a low-sodium diet, and smoking cessation are essential in managing HFpEF. These measures help improve cardiovascular health and reduce the burden on the heart. [7,8].

Diuretics are used to relieve fluid overload, which can reduce symptoms like shortness of breath and edema. Loop diuretics, such as furosemide, are frequently used in HFpEF to manage congestion. Medications like spironolactone or eplerenone, which are used in HFrEF, have also been shown to have some benefit in HFpEF, particularly in patients with heart failure and hypertension. They help reduce fluid retention and may offer protective benefits in terms of fibrosis and remodeling. Since HFpEF is closely linked to comorbidities such as diabetes, atrial fibrillation, and obesity, managing these conditions is crucial. In particular, controlling diabetes with medications like SGLT2 inhibitors has shown promising results in improving outcomes in HFpEF. Many patients with HFpEF have atrial fibrillation (AF), which exacerbates heart failure symptoms. Rate control or rhythm control strategies, such as the use of beta-blockers or calcium channel blockers, may help stabilize heart rhythm and improve symptoms. Clinical trials are currently exploring new treatment options for HFpEF, including drugs targeting myocardial fibrosis, endothelial function, and inflammation. Medications like sacubitril/valsartan an angiotensin receptor-neprilysin inhibitor and others are being tested to determine their effectiveness in this population. [9,10].

Conclusion

Heart Failure with Preserved Ejection Fraction is a complex and increasingly prevalent form of heart failure, especially

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in elderly patients and those with comorbid conditions such as hypertension and diabetes. Though the management of HFpEF remains challenging, advances in diagnostics and therapeutic strategies continue to improve outcomes for patients. A multifaceted approach that includes managing comorbidities, improving lifestyle factors, and exploring novel pharmacological therapies offers hope for better management of this debilitating condition. As research progresses, more effective, targeted treatments may emerge, providing patients with improved quality of life and longer-term survival.

References

1. Olgac G. Antibiotics are not needed during tube thoracostomy for spontaneous pneumothorax: An observational case study. *J Cardiothorac Surg.* 2006;1:43.
2. Martin-Loeches I. Antibiotic prophylaxis in the ICU: To be or not to be administered for patients undergoing procedures? *Intensive Care Med.* 2020;46:364–367.
3. Henry M. Pleural Diseases Group. Standards of Care Committee. British Thoracic Society BTS guidelines for the management of spontaneous pneumothorax. *Thorax.* 2003;58:39–52.
4. Martin C. Antibio prophylaxis in surgery and interventional medicine (adult patients) Update 2017. *Anaesth Crit. Care Pain Med.* 2019;38:549–62.
5. Stewart A. Prophylactic antibiotics to reduce morbidity and mortality in newborn infants with intercostal catheters. *Cochrane Database Syst. Rev.* 2012;4:CD008173.
6. Gullestad L, Aukrust P, Ueland T, et al. Effect of high-versus low-dose angiotensin converting enzyme inhibition on cytokine levels in chronic heart failure. *J Am Coll Cardiol* 1999; 34:2061–2067.
7. Fliser D, Buchholz K, Haller H. Antiinflammatory effects of angiotensin II subtype 1 receptor blockade in hypertensive patients with microinflammation. *Circulation* 2004; 110:1103–1107.
8. Manabe S, Okura T, Watanabe S, et al. Effects of angiotensin II receptor blockade with valsartan on pro-inflammatory cytokines in patients with essential hypertension. *J Cardiovasc Pharmacol* 2005; 46:735–739.
9. Ohkubo T, Chapman N, Neal B, et al. Effects of an angiotensin-converting enzyme inhibitor-based regimen on pneumonia risk. *Am J Respir Crit Care Med* 2004; 169:1041–1045.
10. Okaishi K, Morimoto S, Fukuo K, et al. Reduction of risk of pneumonia associated with use of angiotensin I converting enzyme inhibitors in elderly inpatients. *Am J Hypertens* 1999; 12:778–783.