Arrhythmias and Heart Failure

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KEYWORDS

• Heart failure • Arrhythmia • Atrial fibrillation • Ventricular tachyarrhythmia

KEY POINTS

- Atrial fibrillation and ventricular tachyarrhythmias are frequently seen in patients with heart failure, and complicate the management of such patients.
- Both types of arrhythmia lead to increased patient morbidity and mortality.
- Many randomized studies have been performed in patients with these conditions and heart failure, and these have helped to guide clinicians in designing optimal treatment strategies.

Heart failure (HF) is a highly prevalent disorder, afflicting approximately 6 million individuals in the United States with an incidence of 10 per 1000 population after the age of 65 years.[1] Nearly 300,000 Americans are diagnosed with heart failure annually and, although overall survival has improved over time, the mortality remains high, as approximately 50% of patients die within 5 years from initial diagnosis.[2] The 2 most common arrhythmias dealt with in HF patients are atrial fibrillation (AF) and ventricular arrhythmias such as ventricular tachycardia (VT) and ventricular fibrillation (VF). These disorders cause considerable mortality and often prove to be challenging issues to manage.

ATRIAL FIBRILLATION

Prevalence

AF is by far the most common arrhythmia in North America, affecting an estimated 2.3 million people. In the last 20 years, hospital admissions attributable to AF have increased by 66%.[3] It is estimated that the prevalence of AF will increase by 2.5-fold by the end of the year 2050 and will affect 5.6 million Americans.[4]

Association Between Heart Failure and Atrial Fibrillation

AF and HF are thought to perpetuate each other. Both share common risk factors, such as:

- Advanced age
- Hypertension
- Diabetes mellitus
- Coronary artery disease
- Heart disease

Evidence also exists of a more complex relationship between the two that may be independent of mutually predisposing factors.

HF is the strongest predictor for the development of AF, with up to a 6-fold increase in risk seen in the Framingham study.[5] In the Framingham Heart Study, 1470 subjects developed either new AF or HF from 1948 to 1995, with 383 (26%) developing both.[6] In addition, lifetime risks for developing AF in men and in women who are 40 years or older is 1 in 6 in patients without congestive HF or myocardial infarction (MI), and 1 in 4 in the same population when patients with congestive HF or prior MI are included.[7]

The reported prevalence of AF in series of moderate HF patients ranges from 13% to 27%.[8–12] It has also been noted that prevalence of AF in patients with HF increases in parallel with the severity of HF, ranging from 5% in patients with mild HF, 10% to 26% among patients with moderate HF, and up to 50% patients with severe HF.[13] A study by Deedwania and Lardizabal[14] showed an almost identical parallel association. The prevalence of AF in the setting of preexisting HF increases in patients with worsening HF class: 4% at functional

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class I, 27% at functional class II and III, and 50% at functional class IV.

Middlekauff and colleagues[8] found that patients with advanced HF and AF had a significantly reduced 1-year survival when compared with patients in sinus rhythm. However, AF seems to be a stronger predictor of negative outcome in patients with mild to moderate HF in comparison with patients with severe HF. A study by Corell and colleagues[15] found that the presence of AF in patients with HF was associated with increased morbidity and mortality in patients with better cardiac function.

Atrial Fibrillation Precipitating Heart Failure

AF may directly facilitate the development and/or progression of HF in several ways. First, the increase in resting heart rate and an exaggerated heart-rate response to exercise results in shorter diastolic filling times, which lead to a reduction in cardiac output. The irregular ventricular response leads to a reduction in left ventricular filling during short cycles. In addition, the loss of an effective atrial contraction is a contributing factor, as the contribution of left atrial systole in left ventricular filling can be up to 50%.[16,17]

The onset of AF is often accompanied by cardiac decompensation and deterioration in functional class in patients with HF. In a study by Pozzoli and colleagues,[18] 344 patients with previously diagnosed HF, initially in sinus rhythm, were found to have worsening of New York Heart Association (NYHA) functional class, peak oxygen consumption, and cardiac index, as well as increased mitral and tricuspid regurgitation and cardiac chamber dimensions, corresponding to the onset of AF.

Restoration of sinus rhythm improves cardiac output, exercise capacity, and maximal oxygen consumption.[19] In the Valsartan in Acute Myocardial Trial (VALIANT) of 14,703 patients with acute MI complicated by HF, AF was associated with greater long-term mortality and morbidity.[19]

In the Trandolapril Cardiac Evaluation (TRACE) study, long-term mortality was found to be increased in all groups of patients in AF except those with the most advanced disease.[20]

The presence of AF, while serving as a marker for outcomes, may not necessarily directly affect the prognosis of patients with HF. For example, post hoc analysis of data from the Veterans Affairs Vasodilator Heart Failure Trials (V-HeFT),[9] which enrolled more than 1300 patients with mild to moderate HF, found that the rates of mortality, hospitalization, and other adverse events in patients with AF were no different from those in sinus rhythm. Similarly, in severe HF, AF has not been shown to be independently associated with adverse outcomes in the Prospective Randomized Study of Ibopamine on Mortality and Efficacy (PRIME) study.[21] Ahmed and Perry[22] found that among 944 elderly patients hospitalized with HF, onset of new AF carried a significantly higher risk of death when compared with patients with no AF or those in chronic AF. In fact, more than 80% of patients hospitalized with HF and found to have new-onset AF died within 4 years of discharge, compared with only 61% to 66% of those without AF or with persistent AF, respectively.

A post hoc analysis of data from the Carvedilol Or Metoprolol European Trial (COMET), which enrolled more than 3000 patients with symptomatic systolic HF, showed that patients who were in AF on baseline electrocardiogram had significantly higher risks of all-cause and cardiovascular mortality and hospitalization rates over a 5-year period. However, after adjustment for patient-related variables (eg, age and gender), the presence of AF at baseline was no longer independently associated with mortality. Serial electrocardiography was performed throughout the COMET follow-up period to screen for subsequent development of AF. Of the nearly 2500 patients who were in sinus rhythm at baseline, 580 developed new-onset AF during the study. In this subset of patients, new-onset AF was an independent predictor of subsequent all-cause mortality, and remained so regardless of treatment and changes in functional class over time.[23] In summary, although preexisting chronic AF has not been definitively shown to independently affect the rates of mortality or morbidity in patients with HF, the onset of new AF is certainly associated with adverse outcomes in chronic HF.

Pathophysiology

Under normal circumstances, atrial systole may contribute up to 25% of cardiac output. In the setting of ventricular dysfunction, atrial contribution to the total cardiac output may be 50%. The onset of AF abolishes the “atrial kick,” leading to a reduction in cardiac output, and peak oxygen uptake in exercise tolerance.[16,17] Rapid ventricular rates during periods of uncontrolled AF lead to inadequate ventricular filling forward blood flow. An irregular ventricular response, even if independent of heart rate, causes a decrease in cardiac output, increased pulmonary wedge pressures, and elevation of right atrial pressures.[24–27]

AF also plays a direct role in the development of a cardiomyopathy, namely a tachycardia-induced