Perspectives by a position statement on atrial fibrillation in acute heart failure a: Mechanisms and therapeutic approaches

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Abstract

The co-existence of atrial fibrillation (AF) and acute heart failure (AHF) is frequently reported and can exacerbate either or both of them. Their combination leads to increased morbidity and mortality. Although there has been a lack of studies on the prevalence and significance, as well as the treatment, of AF in patients with AHF, a position statement from the Acute Cardiovascular Care Association and European Heart Rhythm Association has recently reviewed the latest evidence on AF in the setting of AHF. The purpose of this paper is to briefly overview the crucial aspects of this consensus document. *(Anatol J Cardiol 2020; 23: 308-11)*

Keywords: atrial fibrillation, acute heart failure, rate control, rhythm control, catheter ablation, pacing, bleeding risk, anticoagulation

Introduction

Atrial fibrillation (AF) and acute heart failure (AHF) are frequent clinical situations. They usually co-exist and can exacerbate each other. Their combination leads to increased morbidity and mortality in patients. However, there has been a lack of studies on the prevalence and importance, as well as the therapeutic management, of AF in AHF. The management of AF in AHF requires a multidisciplinary team-based approach because it involves crucial aspects such as the treatment of underlying disease(s), along with identification and treatment of potentially correctable causes, precipitating factors, and anticoagulation. A position statement from the Acute Cardiovascular Care Association (ACCA) and European Heart Rhythm Association (EHRA) has recently overviewed the therapeutic management of AF in the setting of AHF (1). The purpose of this paper is to summarize the interplay between AF and AHF, treatment principles, and crucial aspects forwarded by this position statement.

Interplay between AF and AHF

The new position statement comprehensively overviewed the interplay between AF and AHF (1). It is essential to remember that AF affects about one-third of patients presenting with AHF. AF and AHF share common risk factors, have similar etiologies, and can exacerbate each other. Their combination leads to an increased morbidity and mortality in patients. The development of AF during AHF is a multifactorial pathophysiological process. The causative relationship between them is both reciprocal and variable (1). AF can induce electrical and hemodynamic deterioration and can cause tachycardia-mediated cardiomyopathy, further resulting in HF (2). By inducing a rapid ventricular response and altering left ventricular (LV) diastolic function, AF can also cause HF symptoms, even, in patients with normal LV systolic function (2-4). The presence of AF or HF increases the risk of the other condition, with AHF being the strongest risk factor for the development of AF. Similarly, AF precipitates and exacerbates LV dysfunction, giving rise to AF-induced cardiomyopathy (5, 6). Furthermore, AF begets AHF, and additional cardiac disorders such as hypertensive, coronary, or valvular heart diseases are often the underlying conditions for both AF and AHF. Accordingly, the causal therapy of the underlying cardiac disorders has an utmost importance in all patients with AF and AHF (3, 7).

How to treat AF in AHF?

General measures

The main recommendations of new position statement are in agreement with the current European Society of Cardiology (ESC) guidelines on AF and HF (1, 3, 5). It is essential to remem-

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ber that all reversible causes of AF and AHF need proper identification and correction, whenever it is possible. Moreover, it is also important to diagnose and treat acute coronary syndrome upon its presence. The identification of underlying cause usually requires a comprehensive evaluation of medical history, physical examination, electrocardiography (ECG), basic laboratory tests, and echocardiography (3, 4).

AHF is a syndrome that requires the effective collaboration of cardiologists, emergency physicians, intensivists and other healthcare providers in delivering urgent aid/care to the patients (6, 8). Non-invasive monitoring including pulse oximetry, arterial blood pressure, respiratory rate, and continuous ECG should be initiated within minutes of first medical contact. The presence of hemodynamic instability should be treated with urgent cardioversion (3, 4). Medical treatment should be initiated based on the arterial blood pressure and/or the degree of congestion. Importantly, intravenous administration of amiodarone should be considered for the rapid control of ventricular rate in patients with AHF and AF (6). Intravenous digoxin may be considered as an option if the rate control is not obtained by amiodarone alone.

The new position statement supports the use of ABC (Atrial fibrillation Better Care) pathway for the management of AF in AHF as follows (1):

- 'A' Avoid stroke with Anticoagulation.
- 'B' Better symptom management with patient-centered decisions on the rate or rhythm control.
- 'C' Cardiovascular risk and comorbidity management, including lifestyle changes.

An ABC pathway compliant management approach has been associated with a reduced risk of adverse outcomes, shorter length of stays in hospital, and reduced healthcare costs (9).

Rate control

The new position statement strongly emphasizes that the underlying causes of elevated heart rate, such as ischemia, infection, anemia, and pulmonary embolism, should be properly evaluated and treated before commencing rate control (1, 3). The choice of drug and target heart rate will depend on the patient's characteristics, symptoms, LV function, and hemodynamics. Intravenous amiodarone may be considered in the patients with AHF (3). If amiodarone fails to control the heart rate, then digitalis (digoxin or digitoxin) may be added for the acute control of ventricular rate; however, there is a controversy regarding digoxin use in AF or HF. Beta blockers are often and calcium channel blockers are always contraindicated in the acute situation (3, 10). The optimal heart rate target for patients with AHF has not been investigated yet, but a heart rate of less than 100 bpm guided by hemodynamic and symptomatic improvement is considered optimal heart rate. Urgent cardioversion should be considered in unstable patients.

Rhythm control

The new position statement provides a detailed description of rhythm control in AF with AHF (1). It states that rhythm control may be a preferred option in AF associated with AHF (1). Initially, rate control should be instituted; however, urgent cardioversion may be considered in unstable patients. If rhythm control is indicated, then all antiarrhythmics drugs (AADs) are contraindicated except for amiodarone (3). Furthermore, amiodarone slows the heart rate, which may contribute in improving the hemodynamic situation (11).

The decision to initiate AAD aims to control symptoms. There is no significant effect on mortality than rate control in chronic HF. Safety considerations should be based on the choice of the AAD, and symptom burden must be weighed against potential side effects. The current guidelines for the maintenance of sinus rhythm in patients with AF and HF recommend either dofetilide or amiodarone; however, dofetilide is not approved in Europe (1). Dronedarone, class IC antiarrhythmics (flecainide and propafenone), and sotalol are not recommended in AHF. A shorter duration of AAD therapy seems reasonable in reducing the risk of side effects, especially in patients who are deemed at an increased risk of side effects or in patients with a low risk of recurrent AF.

The optimization of concomitant cardiovascular risk factors and diseases has been shown to reduce the symptom burden of AF and facilitate the maintenance of sinus rhythm. This includes weight reduction, blood pressure control, HF treatment, increasing cardiorespiratory fitness, treatment of obstructive sleep apnea, and other measures (3, 12).

The results of Catheter Ablation for Atrial Fibrillation with Heart Failure trial showed that catheter ablation for AF in patients with chronic HF was associated with a better prognosis (1, 13). As compared with rate or rhythm control, catheter ablation was associated with a significantly lower rate of death from any cause or hospitalization for worsening HF (13). In the ablation group, 63% of patients were in sinus rhythm at 60 months as compared with 22% of patients in the medical therapy group, which suggests that maintenance of sinus rhythm is beneficial when it is achieved without the use of AADs.

Stroke prevention

The new position statement delivers several key messages regarding stroke risk assessment and prevention (1). It is clearly recommended that individual stroke risk is dynamic and must be re-assessed on every clinical visit (3, 14). Current AF guidelines recommend stroke risk assessment by using the clinical risk factor-based CHA_2DS_2-VASc score (3, 7). In patients with AF, HF is an independent risk factor for thromboembolism and mortality (3). The "C" in CHA_2DS_2-VASc refers to the recent or ongoing decompensated HF or LV dysfunction on cardiac imaging irrespective of symptoms, in line with the increasing body of evidence showing similar thromboembolic risks in patients with both AF and HF (15, 16).

Any antithrombotic drug confers a risk of bleeding. Thus, as a part of clinical practice, there is a need of bleeding risk assessment. Many bleeding risk assessment are available in the medical files (3, 7, 17). The HAS-BLED score is most validated tool and is

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applicable on patients receiving non-antithrombotic therapy, antiplatelet drugs, and OAC [whether a Vitamin K antagonist (VKA) or NOAC]. A high HAS-BLED score per se is not an excuse to withhold OAC, as such patients derive even greater net clinical benefit while balancing the reduction of ischemic stroke against the potential for serious bleeding. Modifiable bleeding risk factors should be addressed, and patients who are still at a 'high risk' should be scheduled for an early review and a follow-up (3, 7).

Anticoagulation should be considered for all patients with AHF, which is complicated by AF, as well as in patients with permanent AF and incident AHF. Anticoagulation should be provided to the patients with AF having a reduced LV ejection fraction, together with cardioversion, if there are signs of severely compromised hemodynamics and normalization of fluid balance with diuretics.

In the very acute phase, parenteral anticoagulants (heparin or LMWH) are the preferred drugs. The early initiation of the anticoagulation therapy also allows for a safe cardioversion in case of hemodynamic instability. When prompt rhythm control is required, periprocedural heparin can be used. Postprocedural anticoagulation should be continued for four weeks with further long-term anticoagulation therapy. Additionally, bridging from heparin to VKA is required until INR is the range of 2.0–3.0. Once the patient has been stabilized, OACs should be started. OACs are, in fact, effective in preventing cardioembolic stroke and systemic embolism in patients with AF and are associated with a significantly higher reduction of disability and mortality than antiplatelet agents (3, 5).

Chronic OAC is recommended in patients with AF who are at a higher risk of stroke or systemic embolism according to their CHA_2DS_2-VASc scores (≥2 for men and ≥3 for women), as recommended by the ESC guidelines (3). For patients with a single non-sex CHA_2DS_2-VASc score risk factor (1 for men and 2 for women), anticoagulation may be considered, but not all risk factors carry equal weight, and the paroxysmal nature of the AF may be considered (18). However, the risk of stroke in AF and HF is high. Decompensated HF is one of the components of the CHA_2DS_2-VASc score and is a strong indicator of OAC (3, 7).

For long-term treatment, NOACs are preferred over VKAs when patients are eligible for both. To further support this recommendation, it should also be considered that patients with HF who are receiving VKA treatment are at a higher risk for reduced TTR than patients without HF (19), with the consequent potentially limited efficacy and safety of such drugs. Based on the subgroup analyses of pivotal trials, the NOACs appear to be safe and effective in both patients with and without HF (20).

Conclusion

The new position statement of ACCA and EHRA comprehensively overviews the therapeutic management of AF in the setting of AHF. It recommends that the management and treatment of AF in AHF should be a multidisciplinary teamwork process starting from the pre-hospital stage. All reversible and underlying causes of AF and AHF need proper identification and correction. Urgent cardioversion should be considered in hemodynamically unstable patients. For rate control, amiodarone and digoxin can be used as a viable option to slow heart rate. If rhythm control is indicated, then electrical cardioversion or amiodarone may be used. For the maintenance of sinus rhythm, amiodarone may be used with a caution of side effects. Stroke prevention with OACs remains a mainstay of treatment in patients with AF and AHF. Once the patient is stabilized, the default choice is to offer stroke prevention (which is OAC, with well-managed VKA or preferably, a NOAC).

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