Cardiometabolic Risk Factors and Atrial Fibrillation

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Atrial fibrillation (AF) is the most common arrhythmia worldwide; it is a significant risk factor for stroke and embolization, and has an impact on cardiac function. Despite its impact on morbidity and mortality, our understanding of the etiology and pathophysiology of this disease process is still incomplete. Over the past several decades, there has been evidence to suggest that AF has a significant correlation with metabolic syndrome (MetS). Furthermore, AF appears to be more closely related to specific components of MetS compared with others. This article provides an overview of the various components of MetS and their impact on AF.

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KEY WORDS

Atrial fibrillation • Metabolic syndrome • Obesity • Hypertension • Diabetes

Atrial fibrillation (AF) is the most common arrhythmia worldwide,[1] and is a potent risk factor for stroke and systemic embolization.[2] It currently affects approximately 1% of the world's population[3] and possesses a 25% lifetime risk in adults.[4]

The prevalence of AF increases with age and is uncommon in individuals under age 50 years. It has been estimated that approximately 70% of individuals with AF are between ages 65 and 85 years.[5] Furthermore, the prevalence of AF is more common in men than in women,[3,6] and in white populations than in black populations.[7]

In addition to the incremental risk of AF with advancing age, the risk of stroke with AF also increases with age. It has been shown that there is a steep increase in the risk of stroke in patients

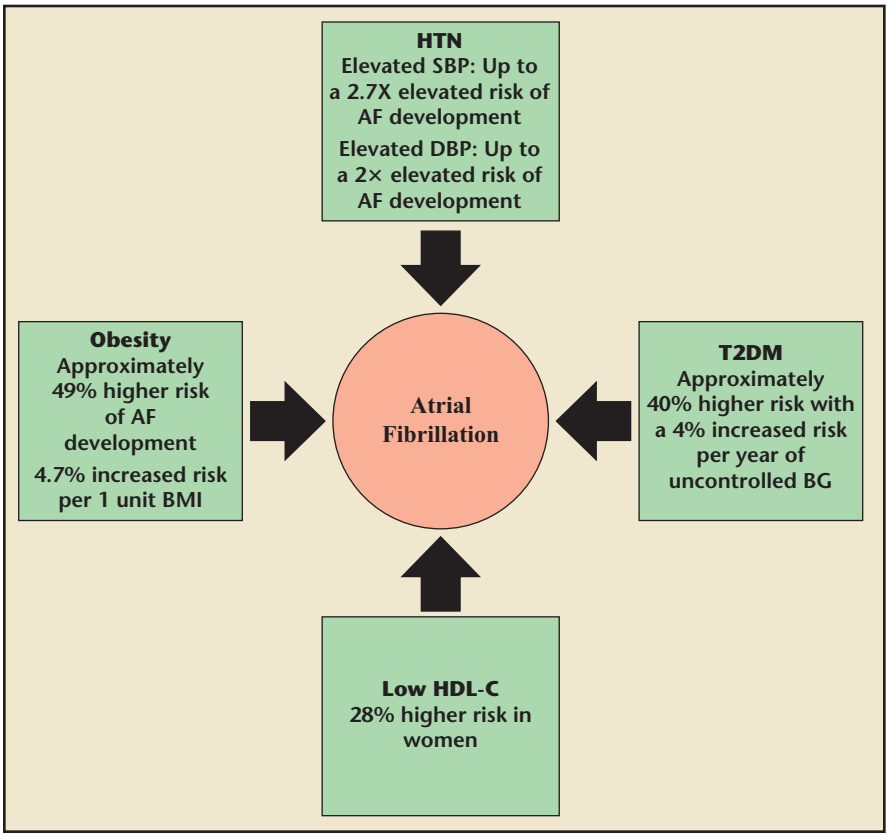

Figure 1. Metabolic risk factors for AF development. AF, atrial fibrillation; BG, blood glucose; BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; HTN, hypertension; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus.

with AF, ranging from 1.5% at age 50 to 59 years to 23.5% at age 80 to 89 years.[8] Metabolic syndrome (MetS) is a collection of cardiometabolic risk factors that have been associated with a higher risk of developing atherosclerotic cardiovascular disease (Figure 1). These risk factors include central obesity, hypertension, insulin resistance, decreased high-density lipoprotein cholesterol (HDL-C), and hypertriglyceridemia (Table 1).[9] In the United States, the prevalence of MetS is almost 20% in adults and increases with age.[10] Over the years there have been multiple studies that have demonstrated an association between MetS and AF.[11-14]

AF and Obesity

In the United States, obesity is a disease of epidemic proportion and is a major independent risk factor for the development of cardiovascular disease.[15] Additionally, obesity has been associated with AF.[16,17] In fact, a large meta-analysis evaluated 16 studies that included 123,249 individuals to determine the role of obesity on the development of AF.[18] Obese individuals were found to have a 49% higher chance of developing AF when compared with individuals with normal body mass index (BMI).

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A large cohort of women, consisting of 34,309 participants in the Women's Health Study, were evaluated over a 12.9- ± 1.9-year follow-up period. The study aimed to identify the relationship between changes in BMI and AF.[19] During this period, 834 incidents of AF were observed. The study found that there was a linear correlation between BMI and AF risk, with a 4.7% (95% confidence interval [CI], 3.4-6.1; $P < .0001$) increase in risk with each kg/m^2. Furthermore, participants who became obese during the first 60 months had a 41% adjusted increase in risk of the development of AF ($P = .02$) compared with those maintaining BMI < 30 kg/m^2. It should be noted that this study used the classic 1997 World Health Organization classification for BMI,[20] which characterizes normal weight as BMI < 25 kg/m^2, overweight as 25.0 to 29.9 kg/m^2, and obese as > 30 kg/m^2.

Similar findings were found by Wang and colleagues[17] in 2004. Here, the authors observed a 4% increase in AF risk per 1-unit increase in BMI in men and in women. Furthermore, a large all-male Swedish study demonstrated that large body habitus, as well as weight gain from age 20 years to midlife, was independently associated with the development of AF.[16]

Another recently published Chinese study aimed to evaluate the association between AF and older Chinese individuals.[21] The study consisted of 5882 men and 14,548 women ≥ 50 years. After multivariate adjustment, BMI (adjusted odds ratio [OR] 1.06 per kg/m^2) and central obesity as assessed by waist circumference (adjusted OR 1.02/cm) were significant risk factors for the development of AF, suggesting that both overall obesity and central obesity are risk factors for AF.

Possible mechanisms for the increased prevalence of AF in obesity include left atrial enlargement, a known precursor of AF,[22] which

TABLE 1

Criteria for Diagnosing MetS

Diagnostic Criteria for MetS (Any 3 Criteria Below)	Defining Points
Elevated waist circumference	Men > 40 in Women > 35 in In Asian Americans Men > 35 in Women > 31 in
Elevated triglycerides	≥ 150 mg/dL OR Pharmacologic treatment for elevated triglycerides
Elevated blood pressure	≥ 130 mm Hg systolic blood pressure OR ≥ 85 mm Hg diastolic blood pressure OR Pharmacologic treatment for hypertension
Elevated fasting glucose	≥ 100 mg/dL OR Pharmacologic treatment for elevated blood glucose
Reduced HDL-C	Men < 40 mg/dL Women < 50 mg/dL OR Pharmacologic treatment for reduced HDL-C

HDL-C, high-density lipoprotein cholesterol; MetS, metabolic syndrome.

has also been associated with obesity.[23,24] Furthermore, data have also identified obesity as an independent predictor of ventricular diastolic dysfunction,[25] which in turn has been linked with AF.[26]

There is also evidence to suggest that obesity may be an independent predictor of procedural failure after catheter ablation of AF. Two recent studies demonstrated this point. The first study, published in 2010, consisted of 109 patients who underwent catheter ablation for AF.[27] Among the 34 patients with failed outcome, 5 (15%) had a normal BMI, whereas 14 (41%) were overweight and 15 (44%) were obese ($P = .04$); multivariate analysis demonstrated that BMI was an independent predictor of procedural failure. The second study published in 2013 showed similar results.[28] Of the 186 with AF who underwent catheter ablation, 47 (25.27%) experienced late recurrence of AF. They concluded that overweight/obesity (OR = 4.71; $P = .003$) was independently associated with AF recurrence after ablation.

Similarly, data from the Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) trial[29] also demonstrated that higher BMIs were associated with a higher number of direct current cardioversions (DCCV; OR 1.017 per 1 kg/m^2 increased BMI; OR 1.088 per of 5 kg/m^2 increased BMI; OR 1.183 per 10 kg/m^2 increased BMI; $P = .006$ for each) and a higher likelihood of being in AF on follow-up (OR 1.020 per 1 kg/m^2 increased BMI; $P = .0283$; OR 1.104 per 5 kg/m^2 increased BMI; $P = .0283$; and OR 1.218, 95% per 10 kg/m^2 increased BMI; $P = .0283$).

Finally, there are very limited data to suggest that obesity and BMI may predict progression of paroxysmal AF to permanent AF among patients.[30] A study comprising 3248 individuals (mean age 71 ± 15 years; 54% men) diagnosed with paroxysmal AF determined that BMI independently predicted the progression to permanent AF (hazard ratio [HR] 1.04; $P < .0001$). Compared with normal BMI (18.5-24.9 kg/m^2), obesity (30-34.9 kg/m^2) and severe obesity (≥ 35 kg/m^2) were associated with increased risk for progression to permanent AF (HR 1.54 and 1.87, respectively). However, as mentioned earlier, the data that suggest this progression are very limited and further studies are needed to evaluate this correlation.

Despite the convincing evidence that obesity increases the prevalence and complexity of AF,[18] as with other aspects of cardiovascular diseases, including hypertension, coronary heart disease, and heart failure (Table 2), higher BMI and obesity have demonstrated an obesity paradox, because overweight and obese patients with various cardiovascular diseases seem to have a more favorable prognosis than do their lean counterparts.[31-34] This protective effect of a higher BMI appears to be an independent predictor of better clinical prognosis in AF.[35] Therefore, as noted with coronary heart disease and heart failure,[36,37] although overweight/

TABLE 2

Obesity Paradox in Cardiovascular and Noncardiovascular Patients

Cardiovascular
Coronary heart disease
Percutaneous revascularizations
Coronary artery bypass graft surgery
Treadmill referrals
Heart failure
Hypertension
Peripheral artery disease
Echocardiography referrals
Noncardiovascular
Elderly
End-stage renal disease and dialysis
Advanced cancers
Chronic obstructive lung disease
Rheumatoid arthritis
HIV/acquired immunodeficiency syndrome

Adapted from Lavie CJ et al.[31]

obese patients have more AF,[17,18] once AF is established, an obesity paradox is apparent, because overweight and obese patients with AF have a better clinical prognosis than do their lean counterparts with AF.[35]

AF and Hypertension

AF is the most common arrhythmia, and hypertension is the most common cardiovascular disorder. There have been many studies that have shown that patients with uncontrolled hypertension are at an increased risk for AF.[38]

A retrospective case-control study compared 55,412 incident AF cases with 216,400 control cases using logistic regression.[39] The study demonstrated that hypertension was indeed a risk factor for AF (relative risk [RR] 2.60). Similarly, 5331 Framingham Heart Study participants aged ≥ 35 years and initially free from AF were evaluated to determine the relationship between pulse pressure and incident AF.[40] During the 12-year follow-up, 698 participants developed AF (13.1%). After adjusting for variables such as age, sex, time-dependent change in mean arterial pressure, and known risk factors for AF, they demonstrated that pulse pressure was associated with increased risk for AF (HR, 1.26 per 20-mm Hg increment; 95% CI, 1.12-1.43; $P < .001$). They also found that systolic blood pressure (SBP) was related to AF (HR, 1.14 per 20-mm Hg increment; $P = .006$) whereas mean arterial pressure was unrelated (HR, 0.96 per 10-mm Hg increment; $P = .39$).

The association between elevated blood pressure and AF may also extend to patients with blood pressure in the upper range of normal. A recently published Norwegian study examined the long-term impact of upper-normal blood pressure on incident AF in a population of middle-aged men.[41] From 1972 to 1975, 2014 healthy Norwegian men were included in a prospective cardiovascular survey including standardized blood pressure measurements. During the 35-year follow-up period, 270 men were documented with AF. The authors observed that men with baseline SBP ≥ 140 mm Hg had a 1.60-fold risk of developing AF whereas men with upper-normal SBP 128 to 138 mm Hg had a 1.50-fold risk of AF, respectively, when compared with men with SBP < 128 mm Hg. Furthermore, baseline diastolic blood pressure (DBP) ≥ 80 mm Hg increased the risk of incident AF 1.79-fold compared with DBP < 80 mm Hg.

This relationship between elevated BP and AF was also observed among a large cohort of initially, apparently healthy women. Here, a total of 34,221 women participating in the Women's Health Study were prospectively followed for incident AF over a 12.4-year period.[42] Multivariable-adjusted HR for SBP categories (< 120, 120-129, 130-139, 140-159, and ≥ 160 mm Hg) were 1.0, 1.00, 1.28, 1.56, and 2.74 (P for trend < .0001). Adjusted HR for DBP categories (< 65, 65-74, 75-84, 85-89, 90-94, and ≥ 95 mm Hg) were 1.0, 1.17, 1.18, 1.53, 1.35, and 2.15 (P for trend = .004), suggesting that blood pressure was a

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significant predictor of incident AF, with SBP being a better predictor than DBP.

The exact mechanism relating AF and hypertension is currently unclear. Over the past few years, there has been compelling evidence to support the role of renin-angiotensin aldosterone system (RAAS) as a causative factor of AF likely through atrial remodeling.[43] As a result, there have been numerous studies that evaluated the beneficial effects of RAAS-blocking agents in the prevention and management of AF.[44,45]

A meta-analysis involving a total of 23 randomized controlled trials with 87,048 patients was evaluated for this purpose.[46] The study concluded that RAAS inhibition reduced the OR for AF by 33% ($P < .00001$). However, it should be noted that there was substantial heterogeneity among trials. RAAS inhibition was found to be effective in patients with heart failure, hypertension, and left ventricular hypertrophy, but not in postmyocardial infarction patients. Furthermore, RAAS inhibition, when administered in addition to antiarrhythmic drugs, including amiodarone, further reduced the odds for AF recurrence after DCCV by 45% ($P = .01$) and in patients on medical therapy by 63% ($P < .00001$).

Another meta-analysis of eight randomized controlled trials included 2323 patients[47] and demonstrated that angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) significantly reduced the incidence of recurrent AF (RR 0.611; $P = .003$). Further evaluation showed that the RR for recurrent AF for studies using ARBs was 0.643 ($P = .023$) and 0.54 for studies using ACE inhibitors ($P = .002$).

Despite this, there have been yet other studies that have not demonstrated any beneficial effect of ACE inhibitors and ARBs in the prevention of AF. A small meta-analysis of four ACE inhibitor studies comprising 355 patients and six ARB studies comprising 4040 patients was analyzed.[48] The pooled ACE inhibitor data did demonstrate a statistically significant effect in preventing AF recurrences. However, the studies were individually very small and did not have a strong follow-up algorithm to recognize AF episodes. However, pooled ARB data did not show any effect in preventing AF recurrences (RR 0.90; $P = .24$).

There has also been evidence to suggest that there is no benefit of ACE inhibitors or ARBs on the secondary prevention of AF after ablation of chronic persistent AF.[49] One hundred thirty nine patients with chronic persistent AF who underwent radiofrequency ablation were evaluated for AF-free survival with and without the use of ACE inhibitors or ARBs.[50] During the follow-up period of 14.6 ± 8.9 months after AF ablation, AF-free survival in the ACE inhibitor/ARB group did not significantly differ from the non-ACE inhibitor/ARB group ($P = .339$). Similarly, another small study included 234 patients with drug-resistant paroxysmal or persistent AF who either underwent LASSO®-guided segmental pulmonary vein isolation or a CARTO®-guided left atrial circumferential ablation (LASSO and CARTO are manufactured by Biosense-Webster, Diamond Bar, CA).[51] After a median follow-up of 12.7 months, 64% of patients with paroxysmal and 45% of patients with persistent AF were free of AF; ACE inhibitor or ARB use (HR 1.12; $P = .59$) did not significantly influence ablation outcome as assessed by Cox regression analysis.

The role of ACE inhibitors and ARBs to prevent AF is currently controversial. Some studies suggest benefits,[52] whereas others dispute this.[48] Post hoc analyses of two large hypertension trials (the Losartan Intervention For End Point Reduction in Hypertension [LIFE] trial[53] and the Valsartan Antihypertensive Long-Term Use Evaluation [VALUE] trial)[54] demonstrated a preventative effect of ARBs on new-onset AF. Conversely, outcomes from other large trials (the Angiotensin II-Antagonist in Paroxysmal Atrial Fibrillation [ANTIPAF] trial[55] and the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico-Atrial Fibrillation [GISSI-AF] trial)[56] have shown no benefit in the prevention of recurrent AF. Thus, although the use of ACE inhibitors in the prevention of AF recurrence is reasonable, the use of ARBs for this purpose likely provides little to no benefit. Furthermore, most of the post hoc analyses that did show a beneficial effect of ARBs on reducing the rate of AF recurrence were not primarily designed to demonstrate this, whereas the trials that failed to show any benefit of ARBs in the prevention of recurrent AF were specifically designed to do so.

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AF and Type 2 Diabetes Mellitus

The prevalence of insulin resistance and type 2 diabetes mellitus (T2DM) is increasing, and there has been evidence to suggest that it may be associated with AF. Possible mechanisms for this include obesity and central obesity, as well as

likely impaired intra- and interatrial synchronicity associated with insulin resistance.[57] However, over the past few years, this relationship has been controversial and various studies have demonstrated conflicting results.

The Atherosclerosis Risk in Communities (ARIC) study obtained detailed medical histories from 13,025 participants and classified them as having no T2DM, prediabetes, or T2DM.[58] During a mean follow-up of 14.5 years, the study demonstrated that there was a significant increase in the risk of AF among patients with T2DM (HR 1.35). Among patients with prediabetes or those with undiagnosed T2DM, there was no indication of an increased risk of AF compared with those without T2DM. In addition to this, there was a positive linear association observed between glycosylated hemoglobin (HbA_{1c}) and the risk of AF in patients with and without diagnosed T2DM. These findings were similar among both white and black subjects.

Similarly, a meta-analysis of seven prospective cohort studies and four case-control studies included data on 108,703 cases of AF in approximately 1.68 million subjects.[59] The study demonstrated that patients with T2DM had an approximately 40% greater risk of AF compared with patients without T2DM ($P < .001$).

What duration of T2DM is necessary to pose a risk for the development of AF? A population-based case-control study of approximately 3600 participants suggested that persistent, uncontrolled T2DM (based on HbA_{1c} levels) might pose a cumulative risk on the initiation of AF.[60] Among the patients with AF (1410 patients), 252 (17.9%) had T2DM compared with 311 (14.1%) of the control subjects (2203 patients). The adjusted OR for AF was 1.40 for people with T2DM compared with those without T2DM. It was also observed that the risk of developing AF was 3% higher for each additional year of persistent T2DM. Furthermore, the study demonstrated that, compared with patients without T2DM, the OR for AF among those patients with T2DM increased with increasing HbA_{1c} levels, suggesting that strict long-term glucose control may play a significant role in decreasing incidence of new-onset AF.

As mentioned earlier, despite the evidence that demonstrates a correlation between T2DM and AF, there is yet more evidence that shows no association between the two. A subset from the Framingham Heart Study examined the association between insulin resistance and incident AF using multivariate Cox proportional hazards regression analysis and adjusting for the established AF risk.[61] Of the 3023 eligible participants, 279 participants developed AF (9.3%) within ≤ 10 years of follow-up. Using multivariate modeling, the study demonstrated that insulin resistance was not significantly associated with incident AF ($P = .34$).

Another study evaluated 1739 subjects (798 men, 941 women) for the prevalence of AF in patients with T2DM and hypertension.[62] Here, patients were categorized as those with only hypertension (n = 597), those with both hypertension and T2DM (n = 171), and those with only T2DM (n = 147). The study showed that the adjusted ORs were 0.7 among the patients with hypertension only, 3.3 among those patients with hypertension and T2DM, and 2.0 among patients with T2DM only. This suggests no statistically significant association between T2DM and AF.

Finally, there is evidence to suggest that the presence of T2DM is an independent risk factor for AF recurrence after successful DCCV. A retrospective analysis evaluated 289 patients who had a successful DCCV.[63] Of the 289 patients, 42 (14.5%) were known to have T2DM compared with 247 (85.5%) without. Of the group without T2DM, 66.8% (165 of 247) remained in sinus rhythm versus 45.2% (19 of 42) of the group with T2DM ($P = .005$). Using binary logistic regression analysis, the study demonstrated that the presence of T2DM ($P = .019$) is an independent risk factor for recurrence of AF after DCCV. However, it is important to note that this study had a small sample size with unequal distribution. Further, larger studies are necessary to evaluate this correlation.

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AF and Dyslipidemia

Dyslipidemia is a prevalent growing problem in the United States and is a known risk factor for cardiovascular disease.[64] There is a small amount of controversial data that suggests that there may be a relationship between dyslipidemia and AF, or, more specifically, low HDL-C levels and incidence of AF.

A Japanese study published in 2011 evaluated 28,449 individuals without AF at baseline among the general population using annual health examinations. The purpose of this study was to assess the association between the baseline lipid profile and the risk of new-onset AF.[65] The study demonstrated that low levels of HDL-C were associated with the development of AF

in women (HR 2.86) but not in men (HR 1.35). Furthermore, they concluded that women had a 28% higher risk of AF with each 10% decrease in HDL-C. Unfortunately, the study was unable to demonstrate any significant correlation between triglyceride levels and AF. Similarly, the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) also demonstrated that AF was significantly more prevalent among individuals with HDL-C $<$ 35 mg/dL ($P < .01$).[66]

Furthermore, there is some evidence to suggest that statin therapy is associated with a lower incidence and recurrence of AF. However, this relationship is controversial at best and needs further assessment. A meta-analysis of 20 studies evaluated 23,577 patients and aimed to determine whether statins reduced the risk of AF. Results from the study showed that statin therapy was associated with a significantly decreased risk of AF when compared with the control group (OR 0.49; $P < .00001$).[67] In addition, this beneficial effect was observed in atorvastatin and simvastatin subgroups, but was not noted in the pravastatin and rosuvastatin subgroups. Another smaller study of 84 patients demonstrated that atorvastatin, a lipophilic statin, was more effective in preventing recurrence of paroxysmal AF, as well as conversion to permanent AF, than pravastatin, a hydrophilic statin.[68] Yet another study consisting of 65 patients (29 in the atorvastatin group and 36 in the simvastatin group) determined that the unadjusted OR of having an AF recurrence for patients on atorvastatin versus those on simvastatin was 0.31 ($P = .02$).[69]

However, as stated earlier, this correlation between statin use and lower AF incidence is controversial. In a meta-analysis of 13 short-term trials involving 4414 patients, statin treatment seemed to reduce the odds of an episode of AF by 39% ($P < .001$).[70] However, there was significant heterogeneity ($P < .001$) between the trials. In contrast, among 22 longer-term and mostly larger trials of 105,791 patients, statin treatment was not associated with any significant reduction in AF (OR 0.95; $P = .24$).[70]

AF Prevention Through Potential Improvements in Cardiometabolic Parameters

As mentioned, increased BMI and waist circumference are associated with an increased risk of AF development. In fact, obese individuals had a 49% increase in risk of AF development,[18] with an approximately 4% increased risk per 1 unit of BMI increase,[17] when compared with individuals with normal BMI. It would seem reasonable to counsel patients on the importance of maintaining a BMI $<$ 30 kg/m^2, preferably $<$ 25 kg/m^2.[16-19,21]

Elevated SBP, DBP, and pulse pressure have been associated with an increased risk in the development of AF. An SBP $<$ 129 mm Hg appears to be ideal and is associated with the least amount of risk in the development of AF.[41,42] Furthermore, caregivers should aim for a DBP $<$ 80 mm Hg, although a DBP $<$ 65 mm Hg appears to be associated with the smallest risk of new-onset AF.[41,42]

Elevated SBP, DBP, and pulse pressure have been associated with an increased risk in the development of AF.

Strict glucose control among patients with T2DM may decrease the risk of new-onset AF.[58,59] An $HbA_{1c} < 7$ should be the goal of therapy among these patients.[60] However, caregivers should be aware that this correlation between T2DM and AF is currently still controversial and a subject of continued debate among experts.

Finally, although still controversial, there is evidence to suggest that low HDL-C levels ($<$ 35 mg/dL) may be associated with an increased risk of AF.[66] Although it may be important to advise patients with low HDL-C to engage in lifestyle modifications to increase HDL-C (exercise, diet, niacin), there is no strong evidence to support this. Furthermore, it should be noted that, although the use of statins in the prevention of AF has shown benefit in a few studies,[68,69] the role of this class of medication in the prevention of AF is still controversial.[70] Finally, it is important to remember that the current evidence linking DLP and AF pertains to low levels of HDL-C. There is insufficient evidence linking triglycerides and low-density lipoprotein cholesterol to AF.

Conclusions

AF is the most common arrhythmia worldwide and has a significant impact on morbidity and mortality. Although our understanding of the pathophysiology and etiology of AF is still incomplete, there are certain conditions that may be directly or indirectly related to AF. Over the past few decades, there has been substantial evidence to provide a direct correlation between MetS and AF. Although some of the components of MetS are not as strongly related to AF as others, there is evidence to suggest that all of the individual components of MetS do have some impact on the incidence or recurrence of AF. Further studies are needed to improve our understanding of AF and the impact of the

various components of MetS on this disease process, as well as potential interventions directed at various cardiometabolic parameters for the primary and secondary prevention of AF. ■

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MAIN POINTS

- Atrial fibrillation (AF) is the most common arrhythmia worldwide; it is a significant risk factor for stroke and embolization, and has an impact on cardiac function. In addition to the incremental risk of AF with advancing age, the risk of stroke with AF also increases with age.
- Despite the convincing evidence that obesity increases the prevalence and complexity of AF, as with other aspects of cardiovascular diseases, higher body mass index and obesity have demonstrated an obesity paradox, because overweight and obese patients with various cardiovascular diseases seem to have a more favorable prognosis than do their lean counterparts.
- The role of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers to prevent AF is currently controversial. Some studies suggest benefits, whereas others dispute this.
- Over the past few decades, there has been substantial evidence to provide a direct correlation between the metabolic syndrome (MetS) and AF. Although some of the components of MetS are not as strongly related to AF as others, there is evidence to suggest that all of the individual components of MetS do have some impact on the incidence or recurrence of AF.

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