



A Prospective Update on Atrial Fibrillation Outcomes

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ABSTRACT

Studies have proved that the “impacts of AF and the disease linkages that are increasing have a significant impact on several aspects of practice”. Since the prevalence of common risk factors that put people at a greater risk of developing arrhythmias such as DM, HF, O, SA, HT, and CAD continues to rise, it is possible that current projections of the burden of AF are lower than what is actually the situation. Therefore, studies proved that “establishing strategies to reduce the risk of AA and its consequences is paramount in order to improve QOF, mortality rates, and resource utilization”. Thus, in this review, we have discussed the AF outcome.

Key words: AF, Mortality rate, Outcome, DM, HF, HT.

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INTRODUCTION

Studies have shown that “Atrial fibrillation (AF), is the most frequent persistent AA seen in clinical practice”. [1] Studies also shows that “as its incidence continues to rise, it is posing a greater challenge for healthcare professionals all over the globe”. [1] Studies have also shown that “between 0.4 and 1% of the general population and 10% of the population that is older than 80 years may acquire AF which results in a major worldwide burden of arrhythmia treatment and care for the repercussions of the condition” [2] As the prevalence of common risk factors that put people at a higher risk of developing arrhythmias such as DM, heart failure, obesity (O), sleep apnea(SA), hypertension(HT), and CAD continues to rise, it is possible that current projections of the burden of AF are lower than what is really the case. [3-5] Studies predict that “AF, as well as its repercussions and illness correlations, will have a significant influence on multiple facets of medical practice in the future. This will make preventive strategies to decrease the risk of AA and it is to enhance quality of life, reduce mortality rates, and limit the utilization of resources”. [6] A study that lasted 9.1±1.8 years looked at 343 people with atrial fibrillation and compared them to 2,945 people who did not have atrial fibrillation. The researchers found that AF increased the risk of serious cardiac events like death and stroke over the course of the CHADS2 score profile. [7] Based on these data, the notion that “AF is an independent risk factor for long-term adverse CVS events in the general population study that was carried out received support”. [8] A second study that made use of the INSTRINSIC RV database gave more evidence that lent support to this finding. [8] Furthermore, studies concluded that “those participants in this study of 1530 patients who had a history of AF had higher rates of hospitalization for heart failure (HF), mortality, and ICD shocks in comparison to those participants who did not have a history of AF. In addition, the onset of AF for the first time in people who had no history of the illness in the past was a significant factor that increased these risks”. [8] These data provide credibility to the hypothesis that AF is a discrete and independent risk factor for adverse outcomes. [6]

AF & HF

Studies have also concluded that “HF and AF are two common clinical conditions with the same risk factors. Although these disease states may occur independently of one another, it is more common to see them occurring together as a combined disease entity”. [9] The majority of patients who have both AF and HF had one condition before the other. This allows for an understanding of which disease predisposes the patient to the other disease. [9] In spite of this, the Framingham Heart Study discovered that simultaneous diagnosis of both heart failure and AF happened in one out of every five patients who suffered from both conditions. [3] Studies have also concluded that the “management of patients with AF and HF is difficult because these conditions exacerbate one another, affecting therapeutic options and hastening a never-ending cycle of complications, like RD, ICD shocks, stroke, dementia, a lower QOL, and even death”. [6] (Figure 1)

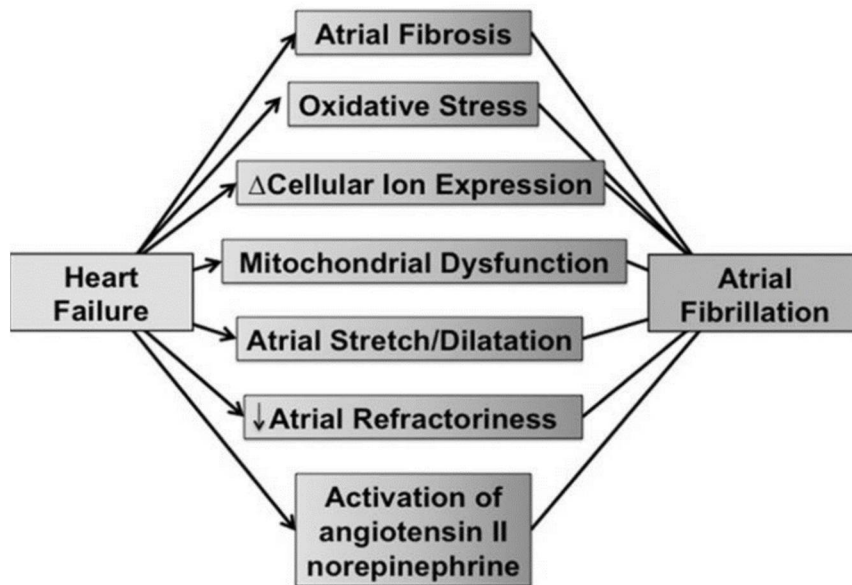


Figure 1: Micro & macro drivers of AF in patients with HF.[6]

Studies have further concluded that one of the clearer illustrations of AF's role as an independent factor in the development of CVS risk is the condition known as tachycardia-induced cardiomyopathy (TICM). One study concluded that outcomes were similar for patients who had new-onset AF and had rapid ventricular rates.[6] Furthermore, studies revealed that changes in hemodynamics may begin to occur as early as 24 hours with rapid ventricular pacing, and the development of end-stage HF can take anywhere from three to five weeks. However, studies have also concluded that if the AF was treated or RV pacing was administered, the MP was found to be reversible, and the patient made a full recovery in one to two weeks.[10] One study concluded that the reversibility of HF and the further temporary return of HF were seen once sinus rhythm was restored.[11] In one study, people with AF who were also found to have idiopathic dilated cardiomyopathy (IDCM) were given a rhythm management method. This raised the LVEF from 25% to 52% (median follow-up of 30 months). In another similar study, patients who had AF were found to have IDCM and underwent a rhythm management approach. The fact that the patients were monitored for a longer length of time (LLOT) Studies have further concluded that HF is a potentially serious consequence of AF, although its symptoms and severity may vary. Statistics in many studies have shown that between 15 and 20 percent of patients in the US who were diagnosed with AF underwent or developed HF at some point in the future. [1,3,9] Studies have also concluded that AF may be a consequence of VD, which in turn may be the result of either systolic or diastolic heart failure. Studies have further concluded that AF may be a consequence of ventricular dysfunction in the same manner that heart failure is a consequence of AF.[12] In other words, studies have also concluded that HF may be a consequence of AF. Studies have shown that those who have HF are at risk for about 4.5 and 5.9 times more of getting AF when compared to those who do not have HF. [13] Studies have further concluded that the findings of one study showed that 17.1% of patients who had recently been diagnosed with HF went on to acquire AF. [3] Studies have also concluded that the severity of HF is yet another factor that contributes to the overall risk. For example, according to one study, the occurrence of AF rises considerably when HF worsens, ranging from 10% in NYHA class II HF to 50% in NYHA class IV HF. This increase is due to the fact that AF affects the heart's electrical system.[13] Studies have further shown that , "changes in the function of cellular ion channels, changes in the expression of cellular ions, atrial stretch and enlargement, atrial fibrosis, and more SA have also been found to cause AF to start and keep going".[9] Additionally, studies have concluded that HF, which may alter cellular ion channel function, may be the root cause of all of these changes. [9,14,15,16,17,18,19,20] Studies have further concluded that these micro- and macro-anatomical changes may have a harmful effect on the manner in which the body reacts to both pharmacologic and nonpharmacologic treatment. Additionally, Studies said that they may raise the risk of adverse outcomes such as mortality and stroke. [9,21,22] Studies have also found that making the best use of medicines like angiotension receptor blockers and beta blockers may have a positive effect on the processes that cause HF, which may then lower the risk of AF.[23-24] Studies have also concluded that AF increases the risk of increased HF, ICD shocks, and mortality among patients who have implanted cardioverter defibrillator (ICD) failure, according to the INTRINSIC RV study. [8] Studies have further concluded that at the time of implant, 1356 out of 1530

patients in this study had no history of AF, which was the condition being studied. Additionally, studies have further concluded that these increased risks were seen across all age groups.

Studies have further concluded that carvedilol, when compared to metoprolol, was shown to reduce the mortality impact of AF in HF patients. [23] Furthermore, studies have concluded that there is now an ongoing controversy about the mechanisms of benefit linked to the use of beta blocker treatment for patients with AF who also have HF. Studies have also concluded that the benefit may have been the result of effective rate regulation of rapid ventricular rates. [25] However, one study did research on intrinsic RV and concluded that the risk of AF mortality and HF was maintained throughout a broad HR stratum when compared to individuals who did not have AF. [26] In addition, studies have concluded that the use of ACE inhibitors, ARBs, and aldosterone blockers may have a positive impact on the treatment of AF and the outcomes in patients who have HF. Studies have also found that both drug-based and non-drug-based methods of treating AF in HF are complicated, and their effectiveness is lower than when used in healthy people. Sadly, the adoption of these medicines has only had a moderate impact on the long-term results of HF in the community over the course of the previous 20 years, particularly mortality. [27] Studies have also found that the vast majority of antiarrhythmic medications (AAD) increase mortality in HF via a variety of different pathways, which further compounds these community statistics. Studies also found that the updated guidelines from the AHA and the ACC list amiodarone and dofetilide as the first-choice alternatives for RR in HF. However, dofetilide is limited because many HF patients take diuretics and have renal failure at the same time.[28] Studies have also concluded that in the case of pharmaceutical failure or intolerance, catheter ablation is considered the second line of treatment. In the AF-CHF study, an aggressive pharmacologic rhythm management approach that included repeated cardioversions in order to preserve sinus rhythm over the long term in patients with HF did not have a beneficial impact on the rates of mortality, stroke, or worsening HF.[29]

Studies have also shown that “rate control strategies work better than AADs in the AF CHF trial. However, the neutral results could be changed with new and different rhythm control strategies that are safe, well-tolerated, and have long-lasting effects. These strategies would need to be long-lasting, safe, and effective”.[30] Even though some studies found that catheter ablation worked better in the CABA-CHF trial, cardiac resynchronization is still better for heart failure patients who develop AF in terms of lowering their risk of death. For instance, studies have also concluded that the MADIT CRT trial discovered that patients with HF who experienced atrial tachyarrhythmias had a decreased mortality rate when they got cardiac resynchronization treatment. This was shown to be the case when compared to patients who did not get treatment. [31]

Studies have also shown that even if the efficacy rates of CA in patients with HF are lower compared to those of patients who do not have HF, the outcomes are still quite favorable in comparison to those achieved with AADs. A lot of research has shown that catheter ablation results are a lot like PABA-CHF results. This suggests that an aggressive rhythm control strategy may help heart failure patients do better, as long as it works. In a recent meta-analysis of nine CA trials, the ejection fraction increased during long-term follow-up by an average of 11.1% (95% confidence interval: 7.1–15.2, P.002). This increase was seen in patients with AF who also had CHF. [32] Studies have also concluded that the outcomes that were reported with catheter ablation(CA) in conjunction with the generally negative outcomes that were reported with AADs Even if the efficacy rates of CA in patients with HF are lower compared to those of patients who do not have HF, the outcomes are still quite favorable in comparison to those achieved with AADs. A lot of the results from catheter ablation are similar to the results from PABA-CHF. This suggests that an aggressive rhythm control strategy may improve the outcomes of HF patients, as long as it works. In a recent meta-analysis of nine CA trials, the ejection fraction increased during long-term follow-up by an average of 11.1% (95% confidence interval: 7.1–15.2, P.002). This increase was seen in patients with AF who also had CHF. Studies have also found that the results of CA, along with the mostly bad results seen with AADs, suggest that patients who are a good candidate for the surgery should be thought of for CA as soon as possible as a way to treat AF.[6]

CONCLUSION

Studies have also concluded that the impact that AF has on the lives of patients is quite detrimental. When there is also the coexistence of other heart problems, this impact may be amplified even more. Although studies have proved that rhythm control strategies have been an interesting tool, they have mostly been ineffective in correcting or decreasing the adverse outcomes associated with AF. Addition to this, the difficulty of currently available AADs to maintain sinus rhythm and the presence of toxicities produced by the drug itself are the primary contributors to the failure of these medications to improve patients' chances of surviving cardiovascular disease. The first findings obtained via the use of ablation procedures are favorable and provide support to rhythm control strategies for the purpose of minimizing the risks

that are associated with atrial fibrillation over the long run. Despite this, the vast majority of the data originate from observational analysis or a very small number of randomized clinical trials. CA will ultimately play a role in defining the function of CA in the management of AF patients, but this will only happen once large-scale randomized prospective trials have been completed.

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