

Clinical Factors Associated with Atrial Fibrillation in Congestive Heart Failure Patients Admitted to the University Teaching Hospital, Lusaka, Zambia

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Authors' contributions

This work was carried out in collaboration between all authors. Author FMG born the idea, author MJ designed the study, wrote the proposal and authors MJ, LK and CK corrected the data. Author MJ did the analysis, wrote the report and prepared the manuscript. Authors FMG and BA supervised the whole process. All authors read and approved the final manuscript.

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ABSTRACT

Introduction: Atrial fibrillation (AF) and Congestive Heart failure (CHF) have emerged as major global epidemics. Each of these conditions predisposes to the other, and their concomitant presence has additive adverse effects. This study examined the clinical factors associated with AF in CHF patients admitted to the University Teaching Hospital (UTH), Lusaka, Zambia.

Methods: This was a hospital-based cross-sectional study done in the admission wards of the UTH involving adult patients with the primary diagnosis of congestive heart failure. The data was collected from July 2014 to September 2014. A structured interview schedule was used to capture the socio-demographic and related historical data. Then all patients had a standard 12-lead ECG done on them to check for AF. Those participants with no AF on a standard 12-lead ECG had 24-hours ECG DR180+ Digital Recorder applied to try to pick-up paroxysmal AF. Finally all

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participants with AF were assessed for clinical factors (i.e. sex, age, BMI, smoking, excessive alcohol intake, hypertension, coronary artery disease, dilated cardiomyopathy, diabetes mellitus, and chronic lung disease). Pearson chi-square of independence of the data was used to analyze the data in SPSS[®] 20.0 to determine clinical factors of AF in CHF patients.

Results: A total of 49 patients were included in the study and 13 (26.5%) of them had AF, 7 diagnosed by standard ECG and 6 diagnosed by holter ambulatory ECG monitoring. The prevalence of AF in CHF was found to be strongly associated with age 65 years and above, obesity, smoking, excessive alcohol intake, hypertension, dilated cardiomyopathy, diabetes mellitus and chronic lung disease. These findings suggest the need for clinicians to consider full scale use of ambulatory ECG monitors in all CHF patients with the above conditions.

Keywords: ECG DR180+ digital recorder; smoking; cardiomyopathy; diabetes; lung disease.

1. INTRODUCTION

1.1 Background

Atrial fibrillation (AF) and Congestive Heart failure (CHF) have emerged as major global epidemics [1]. These two conditions share similar risk factors, frequently coexist, and have additive adverse effects when occurring in conjunction [2]. The risk factors include hypertension (HTN), coronary artery disease (CAD), structural heart disease (non-ischaemic, valvular), diabetes mellitus (DM), obesity and obstructive sleep apnoea [3]. The co-prevalence also increases with advancing age and each predicts/ compounds the course of the other [1,4].

There has been increasing evidence regarding the adverse role of AF in patients with CHF both in terms of morbidity as well as prognosis [1]. Most of the studies done have revealed that AF through the loss of organized atrial activity and absence of coordinated atrial mechanical function, is associated with clinical and hemodynamic deterioration which may predispose the patient to systemic thromboembolism and poorer prognosis [1]. Impaired contraction of the atria may cause blood stasis and the potential for thrombus formation, particularly in the left atrial appendage, especially in CHF as there is already presumed stagnation of blood [5].

The pathoetiological interplay between CHF and AF is complex. CHF predicts the development of AF and conversely AF predisposes to CHF [1]. The mechanisms, through which CHF provides arrhythmogenic atrial substrate include: elevated left-sided filling pressures, mitral regurgitation, atrial enlargement, interstitial fibrosis and electromechanical remodelling [4]; activation of

autonomic and renin-angiotensin axis; as well as changes in the intracellular calcium [5].

Conversely, AF can lead to CHF through multiple adverse effects including loss of atrial systole, functional mitral/tricuspid regurgitation, tachycardiomyopathy, and reduced ventricular diastolic filling time [1]. Irregularity in the RR interval can also have a potentially deteriorating influence on cardiac output irrespective of the heart rate [6]. Moreover, deterioration of sinus rhythm in AF patients with CHF can lead to acute decompensation.

1.2 Clinical Factors Known to be Associated with AF in CHF

Body Mass Index [7] is said to be associated with AF and may impact on outcomes. This is probably due to its association with cardiovascular conditions like hypertension. The severity of heart failure as determined by the New York Heart Association (NYHA) classification has also been reported to be a factor in AF whose prevalence is said to increase with increased severity of the NYHA class [8].

Hypertension is implicated in the initiation and maintenance of AF through structural changes, neurohormonal activation, fibrosis, atherosclerosis seen in this condition [9]. Coronary artery disease is implicated in atrial fibrillation in that a partially blocked artery might cause an imbalance of nutrient flow to an area of downstream heart muscle causing ischemia [10]. Ischemia can cause electrical irritability in the ventricle leading to the initiation and perpetuation of atrial fibrillation [10].

Dilated Cardiomyopathy has been associated with occurrence of AF as well. Electrophysiological features associated with left atrial dilation in dilated cardiomyopathy include

shortening of the refractory period and prolongation of conduction time [11]. These alterations may both lead to development of multiple reentrant wave fronts starting and possibly perpetuating AF in dilated cardiomyopathy [11].

In diabetes mellitus both glucose and insulin disturbance may directly affect the myocardium in atrium and ventricle, leading to AF. Left ventricular (LV) hypertrophy has been associated with DM and abnormal glucose tolerance in several epidemiology studies and LV hypertrophy has been said to be a significant risk factor for AF. Analysis of the Framingham study subjects showed that LV mass increased with the worsening of glucose tolerance and the trend was more striking in women than in men. There was also a close relationship between insulin resistance and LV mass, as well as LV wall thickness, in women both with normal and abnormal glucose tolerance [12]. The supraventricular and ventricular arrhythmias are common in chronic obstructive lung disease. The reasons are thought to be due to hypoxia, hypercarbia, pulmonary hypertension, and

myocardial ischemia, which are easily provoked by this limited ventilatory condition [13].

Smoking and heavy alcohol intake are also factors in the occurrence of AF. Smoking may harm the heart through causing or aggravating endothelial dysfunction and atherosclerosis as well as causing cardiac rhythm disorders through the combined effects of nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons. Thus, smoking may change the myocardial substrate as well as action potentials, both processes that may provoke and/or facilitate AF [14]. Heavy alcohol drinking is described as the drinking of 5 or more glasses of alcohol on the same occasion on each of 5 or more days in the past 30 days [15]. It is understood that alcohol consumption acutely affects catecholamine release, causes metabolic acidosis and electrolyte disturbances, and increased oxidative distress [16]. In the long term, this results in myocardial fibrosis/dilatation, structural heart disease, metabolic disturbances, and increased sympathetic tone. The combination of these effects contributes to the increase in atrial arrhythmias including AF [16].

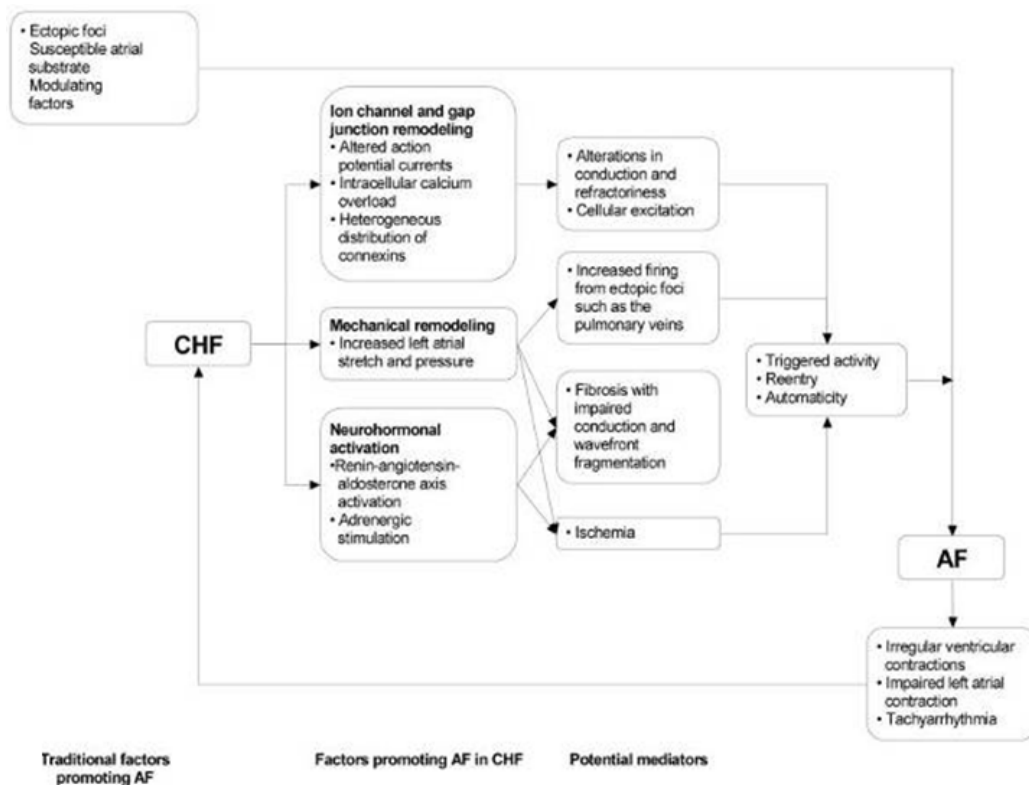


Fig. 1. Pathoetiological inter-relationship between AF and CHF

Source: Lubitz, Benjamin & Ellinor (2010)

2. MATERIALS AND METHODS

This was a hospital based cross-sectional study carried out in adult medical wards at the UTH, a tertiary health centre in Lusaka, Zambia. All known congestive heart failure patients aged 18 years and above who consented to take part in the study were included. However, CHF patients acute patients who were not able to get out of bed were excluded from the study.

2.1 Data Collection

A structured interview schedule was used to capture data on demographic characteristics, clinical factors and laboratory measurement results. The interview schedule was developed based on the World Health Organization (WHO) stepwise survey (STEPS) instrument [17]. The data on demographic and clinical factors were obtained by interview, review of medical records and anthropometric measurements.

The weight and height of the patients were measured using a ZT-160 adult weighing mechanical scale with a height rod (Wuxi Weigher Factory Co., Ltd, Zhejiang, China) whose values were used to compute the body mass index (BMI) taken as proportion of weight (in kilograms) and square height (in metres). Blood Pressure and pulse rate were measured on the left hand of the patient in a lying position using an Omron HEM 780 automated Blood Pressure machine (Omron HEALTHCARE Co. Ltd, Vietnam). A standard 12-lead Electrocardiogram (ECG) was done using Schiller AT-102 ECG machine on all participants to identify those with and without atrial fibrillation. Then those who had no atrial fibrillation on standard ECG had a holter monitor (DR180+ Digital Recorder, Northeast Monitoring Inc, USA) applied for 24 hours. Data was analysed using IBM®SPSS® version 20.0. The analyses included descriptive statistics and Pearson chi square of independence tests. A 95% confidence interval (CI) and *P*-value of < 0.05 were set.

3. RESULTS

3.1 Socio-demographic Data

Table 1 shows the socio-demographic characteristics of participants in the study. A total of 49 black African Congestive Heart Failure patients who met the inclusion criterion were enrolled into the study. There were almost equal number of men and women; 49% vs. 51% respectively. Most of the patients (42.9%) were

aged 65 years and above. The majority (53.1%) of the patients had a normal BMI (18.5 – 24.9). About 20.4% of the patients were tobacco smokers; and 30.6% of the patients were consumers of alcohol.

Table 1. Socio-demographic characteristics of CHF patients recruited (N=49)

Variable	Frequency	Per cent
Sex		
Female	25	51
Male	24	49
Age		
35 - 44 Years	2	4.1
45 - 54 Years	10	20.4
55 - 64 Years	16	32.7
65 Years and above	21	42.9
Body mass index		
18.5 - 24.9	26	53.1
25 - 29.9	11	22.4
30 and above	12	24.5
Smoking		
No	39	79.6
Yes	10	20.4
Alcohol consumption		
No	34	69.4
Yes	15	30.6

3.2 Clinical Factors Data

Table 2 shows the clinical characteristics of the CHF patients included in the study. The majority of the patients (81.6%) were in the New York Heart failure Association (NYHA) class IV; 26.5% of the patients had hypertension; 18.4% had dilated cardiomyopathy; 14.3% had chronic lung disease; 14.3% had diabetes mellitus; and 6.1% had coronary artery disease.

3.3 Electrodiagnosis of Atrial Fibrillation in CHF Patients

Fig. 2 shows the electrographic modality utilised to diagnose AF. Standard 12-lead ECG showed that 7 (14.3%) participants had atrial fibrillation. The ambulatory ECG monitor revealed atrial fibrillation in another 6 (12.2%) patients, giving a combined prevalence of AF of 26.5% in this study population.

3.4 Association between AF in CHF and the Socio-Demographic Characteristics

Using Pearson chi-square of independence test, the association between atrial fibrillation in congestive heart failure patients and the

socio-demographic characteristics. The results obtained are presented in Table 3.

Table 3 shows the cross tabulations of AF by the socio-demographic factors. The incidence of AF was higher in males 8 (33.3%) than in the females 5 (20.0%) although no statistical significance was noted ($p>0.05$). The presence of AF in CHF patients increased with age from 4 (25%) below 65 years to 9 (42.9%) in those above 65 years. Furthermore, the incidence of AF increased with the increase in the BMI from 3 (27.3%) in the overweight to 10 (83.3) in the obese. 7 (70%) of the 10 smokers in CHF had AF and Fisher's exact test showed that there is a statistically significant association between atrial fibrillation in congestive heart failure and smoking. The majority 12 (80.0%) of the patients who reported taking alcohol had atrial fibrillation.

Table 2. Clinical characteristics of CHF patients (N=49)

Variable	Frequency	Per cent
NYHA class		
Class III	9	18.4
Class IV	40	81.6
Hypertension		
No	36	73.5
Yes	13	26.5
Coronary artery disease		
No	46	93.9
Yes	3	6.1
Dilated cardiomyopathy		
No	40	81.6
Yes	9	18.4
Diabetes mellitus		
No	42	85.7
Yes	7	14.3
Chronic lung disease		
No	42	85.7
Yes	7	14.3

3.5 Association between AF in CHF and the Identified Clinical Factors

Table 4 above shows the Pearson chi-square of independence test of AF in CHF by the clinical factors. While 11 (27.5%) of the 40 patients in NYHA IV had AF, only 2 (22.2%) of the 9 patients in NYHA III had AF. However, this difference did not attain statistical difference. Of the 13 hypertensive patients in the study population, 11 (84.6%) had AF. Only 3 patients were reported to have coronary artery disease. And of these, 2 (66.6%) had AF. Of the nine (9) patients who had Dilated Cardiomyopathy,

7 (77.8%) had AF. Six (6, 85.6%) of the seven patients with diabetes mellitus and similar proportion with chronic lung disease had AF. The results showed that there was a statistically significant association between AF in CHF and hypertension, dilated cardiomyopathy, diabetes mellitus as well as chronic lung disease.

4. DISCUSSION

Atrial fibrillation is said to be the most common arrhythmia seen in clinical practice and is responsible for significant morbidity [18]. The presence of AF is said to confer a five-fold increased risk of stroke [19], a significantly increased risk of dementia [20] and an almost two-fold increased risk of death [21]. The clinical consequences of AF are derived from the loss of organized atrial activity and absence of coordinated atrial mechanical function. Impaired contraction of the atria may cause blood stasis and the potential for thrombus formation, particularly in the left atrial appendage, with a resultant risk of stroke. This risk of stroke is said to be increased in patients with CHF [22]. The concomitant presence of AF and CHF identifies individuals with a higher risk for death than with either condition alone [2].

4.1 Prevalence of Atrial Fibrillation in Congestive Heart Failure

The prevalence of AF in the CHF patients admitted to UTH during the period of the study was 26.5%. This prevalence was quite high; though almost half of the patients in this group were missed by routine ECG. Indeed this underlines the recommendations that came out of the Cryptogenic Stroke and Underlying Atrial Fibrillation (CRYSTAL-AF) trial [23] and the 30-Day Cardiac Event Monitor Belt for Recording Atrial Fibrillation After Cerebral Ischemic Event (EMBRACE) trial [24] which demonstrated the effectiveness of extended cardiac monitoring. This demonstrates the need for use of ambulatory diagnostic equipment such as ECG Holter monitors and the insertable cardiac monitors (ICM) in the diagnostic investigations for arrhythmias. With prolonged monitoring we may have obtained a higher yield of individuals with AF. However, the prevalence rate recorded on this study is similar to the 30% prevalence rate reported in the Acute Decompensated Heart Failure National Registry [25] in the United States in 2005. The high prevalence rate may be attributed partially to the advancing age of the Zambian population [26] and/or increase in

prevalence of the non-communicable diseases [27].

4.2 Socio-demographic Characteristics of the Patients

Although, we did not find any statistical difference ($X^2= 1.12$, $p= 0.291$) in the prevalence of AF in CHF between males and females, the majority 8 (61.5%) of patients with AF in CHF were males. Among the male CHF patients, the prevalence of AF was higher (33.3%) compared to 20% among the female CHF patients. Similarly, Lloyd-Jones AM et al. [28] reported that AF after the age of 40 in the United States was 26% for men, and 23% for women and Humphries KH et al. [29] also reported that in all age groups, men have a higher incidence of AF than women. It is postulated that this may be so because males are more exposed to other risk factors for AF like smoking and excessive alcohol intake [3]. However, although women have a lower incidence of AF, studies have shown a worse outcome and a higher rate of recurrence after cardioversion [21,30].

The study also revealed that age 65 years and above was statistically ($X^2= 5.03$, $p< 0.05$) associated with AF in CHF. This result was similar to what was reported by Psaty BM et al. [31] and Nazario B [3]. Advancing age is implicated in the development of AF probably because pre-existing alterations, such as autonomic dysbalance, degenerative tissue changes and fibrosis, can provide an electrophysiological and morphological substrate, which increases the likelihood of AF. In

particular, alterations of the interstitial matrix in atrial tissue seem to be significant contributory factors [32].

The majority 26 (53.1%) of the patients in the study had a normal body mass index (18.5 – 24.9) (Table 2). Of the 13 (26.5%) patients who had AF, the majority 10 (76.9%) were obese and 3 (23.1%) were overweight. No case was found among the participants with a normal body mass index. The study also revealed that body mass index is significantly ($X^2= 22.59$, $p<0.001$) associated with AF in CHF. Similarly, Guilian L et al. [7] and Overvad TF et al. [33] reported that obesity is associated with the development of AF and may impact AF-related outcomes. However, it is worth noting that it is very difficult to calculate body mass index in CHF patients because of the exaggerated patient’s weight resulting from fluid retention.

There were 10 (20.4%) patients who were smokers in the study, and 7 (70%) of them had AF compared to 6 (15.4%) among the 39 non-smokers ($X^2= 9.54$, $p<0.01$). This result is in agreement with what was reported by Heeringa J et al. [34] and Chamberlain AM et al. [35] who reported a more than two-fold increased risk of AF attributed to current smoking. Smoking may harm the heart through causing or aggravating endothelial dysfunction and atherosclerosis as well as causing cardiac rhythm disorders through the combined effects of nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons [14]. Thus, smoking may change the myocardial substrate as well as action potentials; of which both processes may provoke and facilitate AF.

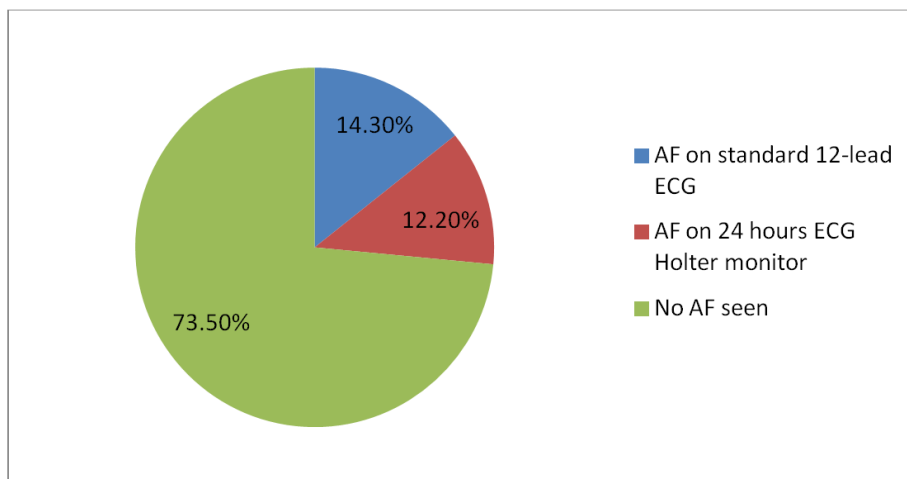


Fig. 2. Electrodiagnosis of atrial fibrillation (N=49)

Table 3. Atrial fibrillation by the socio-demographic factors

Clinical factor	Atrial fibrillation		X ²	P-value
	No AF seen	AF seen		
	N (%)	N (%)		
Sex^a				
Female	20 (80.0)	5 (20.0)	1.12	NS
Male	16 (66.7)	8 (33.3)		
Age^a				
35 - 44 Years	2 (100.0)	0 (0.0)	5.03	<0.05 [*]
45 - 54 Years	10 (100.0)	0 (0.0)		
55 - 64 Years	12 (75.0)	4 (25.0)		
65 Years and above	12 (57.1)	9 (42.9)		
Body mass index^a				
18.5 - 24.9	26 (100.0)	0 (0.0)	22.59	<0.001 [*]
25 - 29.9	8 (72.7)	3 (27.3)		
30 and above	2 (16.7)	10 (83.3)		
Smoking^a				
No	33 (84.6)	6 (15.4)	9.54	<0.01 [*]
Yes	3 (30.0)	7 (70.0)		
Alcohol intake^a				
No	33 (97.1)	1 (2.9)	27.88	<0.001 [*]
Yes	3 (20.0)	12 (80.0)		

**Indicates significant p-value at p < 0.05*

Table 4. Atrial fibrillation by the clinical factors

Clinical factor	Atrial fibrillation		X ²	P-value
	No AF seen	AF seen		
	N (%)	N (%)		
NYHA class^a				
Class III	7 (77.8)	2 (22.2)	0.00	NS
Class IV	29 (72.5)	11 (27.5)		
Hypertension^a				
No	34 (94.4)	2 (5.6)	26.71	<0.001
Yes	2 (15.4)	11 (84.6)		
Coronary artery disease^a				
No	35 (76.1)	11 (23.9)	0.90	NS
Yes	1 (33.3)	2 (66.7)		
Dilated cardiomyopathy^a				
No	34 (85.0)	6 (15.0)	11.81	<0.001
Yes	2 (22.2)	7 (77.8)		
Diabetes mellitus^a				
No	35 (83.3)	7 (16.7)	11.35	<0.001
Yes	1 (14.3)	6 (85.7)		
Chronic lung disease^a				
No	35 (83.3)	7 (16.7)	11.35	<0.001
Yes	1 (14.3)	6 (85.7)		

*^aFisher's Exact Test. *Indicates significant p-value at p < 0.05*

Compared to non-consumers of alcohol where only 1 (2.9%) patient had AF, the majority 12 (80%) of the consumers of alcohol in CHF had AF (X²= 27.88, p= <0.001). Similarly several case-control studies [36,37,30], reported significantly higher odds of developing AF among heavier drinkers. Furthermore, the risk of developing AF is said to increase with increasing levels of alcohol consumption [38]. There has

been much controversy over the exact mechanism by which alcohol induces AF. Mukamal KJ, et al. [38] postulated that alcohol-induced atrial arrhythmias were related to intramyocardial catecholamine release in response to the toxic effects of acetaldehyde. Other studies [39,40], have suggested that an increase in sympathetic reaction could be related to the production of AF based on the increased

density of beta-adrenergic receptors in lymphocytes. Balbão CE et al. [16] proposed multiple mechanisms for the acute and long-term consumption of alcohol resulting in AF. Probably a combination of these effects contributes to the increase in atrial arrhythmias.

4.3 Clinical Factors Associated with AF

The patients in this study were in severe CHF (NYHA classes III/IV). Although, there was no statistical difference ($X^2 = 0.00$, $p = 1.000$) between the two groups, there were more AF cases in NYHA class IV compared to the cases in NYHA class III. Findings from previous studies [8,41-44] have also revealed that the prevalence of AF increases significantly with the increase/severity in the NYHA class. Our small sample size may have influenced the results in this study.

Slightly over a quarter 13 (26.5%) of the patients had hypertension in this study population. The majority 11 (84.6%) of these patients had AF compared to the non hypertensive group where only 2 (5.6%) of 36 patients had AF. Hypertension was strongly associated ($X^2 = 26.71$, $p < 0.001$) with AF in CHF. Similar reports have affirmed this finding [31,45]. Untreated or suboptimally treated hypertension leads to the development of Left Ventricular Hypertrophy (LVH), which is one of the most important expressions of subclinical organ damage, and is an independent risk factor for cardiovascular events, including the development of AF. In the presence of LVH, left ventricular compliance is reduced, left ventricular stiffness and filling pressure increase, coronary flow reserve is decreased, wall stress is increased and there is activation of the sympathetic nervous system and of the renin-angiotensin-aldosterone system. In the atria, proliferation and differentiation of fibroblasts into myofibroblasts and enhanced connective tissue deposition and fibrosis are the hallmarks of this process. Structural remodelling results in electrical dissociation between muscle bundles and in local conduction heterogeneities facilitating the initiation and perpetuation of AF. This electroanatomical substrate permits multiple small re-entrant circuits that can stabilize the arrhythmia. Over time tissue remodelling promotes and maintains AF by changing the fundamental properties of the atria [9].

In this cohort only 3 (6.1%) of the patients had coronary artery disease and out of these 2 (66.8%) had AF. Thakkar S & Bagarhatta R [20]

reported that transient ischemic attack as may be found in coronary artery disease is a risk factor for AF. However, in these patients, systolic heart failure may be more important than atrial ischemia in causing AF [10]. Nevertheless, significant stenosis in the proximal right coronary artery and the circumflex artery prior to the takeoff of the atrial branches increase the likelihood of AF [46].

Only 9 (18.4%) of the patients had dilated cardiomyopathy and the majority 7 (77.8%) of these patients had AF. Dilated cardiomyopathy in CHF was strongly ($X^2 = 11.81$, $p = 0.001$) associated with AF. Similar findings have been reported [47,48]. Luchsinger JA & Steinberg JS [49] also reported that Tachycardia-induced cardiomyopathy may be a more common mechanism of LV dysfunction in patients with atrial arrhythmia. Electrophysiological features associated with left atrial dilation in dilated cardiomyopathy include shortening of the refractory period and prolongation of conduction time [11]. Both these alterations may lead to development of multiple reentrant wave fronts starting and possibly perpetuating AF in dilated cardiomyopathy [11].

Diabetes mellitus has been implicated in the initiation and perpetuation of AF [50,51]. In this study 6 (85.7%) of 7 diabetic patients with CHF had AF. Diabetes was strongly ($X^2 = 11.35$, $p < 0.001$) associated with AF in CHF. Both dysglycemia and insulin disturbance can directly affect the myocardium in atrium and ventricle, by causing atrial and ventricular hypertrophy leading to AF [12]. In the animal model of diabetes mellitus, the occurrence of AF was enhanced by adrenergic activation. The heterogeneous increase in sympathetic innervations has proved to be associated with the promotion of AF in several studies [52,53].

6(85.7%) of 7 patients with chronic lung disease in CHF had AF. There was a strong association ($X^2 = 11.35$, $p < 0.001$). Impaired pulmonary function has been described as an independent risk factor for AF [54]. Indeed, FEV₁%, which represents the severity of airway obstruction, was associated with chronic AF [55] and the greater the pulmonary function impairment, the greater the co-existence with AF. Atrial fibrillation in chronic lung disease is thought to result from changes in blood gases, abnormalities in pulmonary functions, and hemodynamic changes resulting from pulmonary hypertension [56] as well as structural remodelling. Hypoxemia and

hypercapnia are associated with over-compensatory fluctuations in autonomic tone, intrathoracic pressures and cardiac haemodynamics, with possible atrial stretch and remodeling, each of which could lead to AF, particularly when hypercapnia causes a significant decrease in pH values [57]. Morphological abnormalities associated with chronic obstructive pulmonary disease (COPD) include signs of right atrial enlargement, and right ventricular hypertrophy. Structural remodeling results in an electrical dissociation between muscle bundles and local conduction heterogeneities, facilitating the initiation and perpetuation of AF. This electro-anatomical substrate allows multiple small re-entrant circuits that may trigger the arrhythmia [57].

5. CONCLUSION

This study objectively evaluated clinical factors associated with AF in CHF patients admitted to UTH, Lusaka, Zambia. AF is quite common in CHF and strongly associated with obesity, smoking, excessive alcohol intake, hypertension, dilated cardiomyopathy, diabetes mellitus and chronic lung disease. AF at UTH is often diagnosed by routine ECG examination, in the course of investigating and/or managing other cardiovascular disorders. However, the ambulatory ECG monitor for 24 hours captured almost as many cases as were missed by the routine ECG.

This study highlights the importance of electrocardiographic evaluation of patients with chronic heart failure and enlightens the physicians to be more vigilant in searching for AF in particular subpopulations. These findings will guide the physicians in risk stratification and in initiating appropriate treatment for prevention and control of AF in CHF thus enhancing the physicians' clinical practice.

ETHICAL APPROVAL

Ethical clearance was obtained from ERES CONVERGE IRB (Reference number 2014-Mar-003) and permission was also sort from UTH Management and the department of Medicine before starting data collection. Participation in this study was voluntary, with participants free to withdraw from the study at any time. Authors further declare that all procedures used to collect data were normal routine procedures done in the routine care of patients, within the patient's natural environment, and nothing was done to

the discomfort of the patient. Patients found to have atrial fibrillation were referred to the cardiologist for treatment.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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