REVIEW ARTICLE

Atrial Fibrillation in Lusaka – Pathoaetiology, Pathophysiology and Clinical Management Challenges in Primary Care Settings

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ABSTRACT

Background*:* Atrial Fibrillation (AF) is the commonest sustained arrhythmia the world over and is associated with substantial morbidity and mortality. An excessive ventricular rate, a loss of atrial contraction and an irregular ventricular filling time are the hallmark of this condition and all have negative clinical consequences. AF can lead to HF and AF is reported in 10-50% of patients with HF. Presence of AF in HF is an independent risk factor for death. However, the prevalence of AF and its associated risk factors and/or complications in Zambia have not been elucidated. Modalities of investigations and treatment protocols for rate and rhythm control in AF remain unexplored. Although individuals with AF are at risk of death from multiple cardiovascular causes, stroke is the most feared of them all. However, it is not known how often or effectively oral anticoagulation is used in Zambia.

Methodology*:* A prospective, clinical registry captured data on patients presenting to UTH medical admission ward with an electrocardiographic (ECG) diagnosis of Atrial Fibrillation/Flutter. It captured the prevalence of emerging AF/flutter risk factors and co-morbidities and documented the way AF/flutter patients are managed, and the frequency of interventions. Consecutive patients, with documented AF (as their primary or secondary diagnosis) were identified for possible enrollment.

Results: In a period of just over 6 months, a total of 36 patients of AF were enrolled (15 male (42%)and 21 female(58%). They were all classified as patients with either permanent (91.7%) or persistent AF (8.3%). No candidates with paroxysmal AF were captured in this database. The ages ranged from 16 – 90 years. Predominant cardiac diagnoses included a) Valvular heart disease 26 (72.2%), b) Heart failure 22(61%) and C) Hypertensive Heart Disease 12(33%). Co-morbidities included previous stroke or transient ischemic attacks(TIAs) 8 (22.2%), dementia 1(2.8%), pericarditis 2(5.6%), emphysema 1(2.8%), diabetes mellitus 2(5.6%) and hyperthyroidism 1(2.8%). On presentation to the UTH the drugs the candidates were on included 1)Rate control medication (beta-blocker 2.8%, CCB (8.3%), Digoxin (66%) & Amiodarone (2.8%)), 2) Rhythm control medication (Amiodarone (2.8%),3) Anticoagulants (Warfarin (6.9%) and 4) anti-platelets (Aspirin 42.9%). Only pharmacological cardioversion was attempted in 5.6% patients. None were defibrillated.

Conclusions*:* AF is a common arrhythmia in primary care settings in Lusaka. A proportion of patients with symptoms of AF go undiagnosed due to a lack of ambulatory rhythm monitoring devices. Rheumatic Heart Disease, hypertensive heart disease and heart failure are major risk factor for AF. Treatment of AF is far from optimization as evidenced by the large proportion of patients not prescribed either rate control or rhythm control medication. The use of oral anticoagulation is also very low as is also the use of monitoring facilities for the International Normalised Ratio (INR) measurements.

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INTRODUCTION:

A recent multi-regional report has reiterated the fact that AF is not only the commonest sustained cardiac arrhythmia in the world but also a leading cause of stroke and is associated with increased mortality[1]. It has also been observed that there are wide regional variations in the risk factors, concomitant disease conditions and in the clinical management of patients with AF which has made it difficult to formulate universal guidelines for prevention and treatment.

AF is said to carry a four- to five-fold increased risk of stroke[2] and is associated with a high risk of cardiovascular (CV) events and hospitalization[3]. Patients with AF also suffer from a considerably impaired quality of life (QoL)[4]. Strokes associated with AF are said to be more severe than ischaemic strokes of other causes, and in consequence, the risk of death from AF-related stroke is doubled and the cost of care is said to increase 1.5-fold[2]. There is a significant cost to the optimal care of AF in terms of cost of treatment given and also in terms of man-hours lost due to absence from work and/or early retirement. It is important that this condition be identified in the primary care settings for early treatment and mitigation of complications arising from it. However, improving clinical outcomes requires an understanding of what is obtaining locally so as to inform on the adoption or adaptation of existing treatment protocols to standardize care.

Pathoaetiology

The precise mechanisms for the cause of AF are incompletely understood. AF and Atrial flutter are tachy-arrhythmias caused by re-entryand appear to require both an initiating event and a permissive atrial substrate. In many patients AF is due to a focal discharge at rapid rates and fibrillating conduction (heterogeneous conduction) through the atria. There is said to be fragmentation of the depolarization waves into multiple daughter wavelets that wander randomly throughout the atrium and rise to new wavelets that collide with each other and are mutually annihilated or give rise to new wavelets in a perpetual activity. There is therefore disorganized atrial depolarization without effective atrial contraction. On ECG there is evidence of "f" waves at a rate of 350-600/min, no P waves, irregular R-R intervals and/or functional A-V blocks (see Fuster V et al[5]).

Haemodynamic stress, atrial ischaemia and inflammation have been shown to predispose to AF. Increased intra-atrial pressure, as may be caused by mitral or tricuspid valve disease and left ventricular dysfunction, causes atrial electrical and structural remodeling (atrial fibrosis and myocyte atrophy) that favourAF[6]. And so does systemic and pulmonary hypertension. The increase in intra-atrial pressure may contribute to the atrial ischaemia that leads to AF. AF has been seen in inflammatory conditions such as myocarditis and pericarditis which may be idiopathic or secondary to collagen vascular disease, infections, or cardiothoracic surgery[6].

Definite pathophysiological mechanisms are yet to be elucidated in non-cardiovascular respiratory conditions where AF has been observed. These include pulmonary embolism, pneumonia, lung cancer, and hypothermia. Endocrine disorders such as hyperthyroidism, diabetes, and phaeochromocytoma have also been associated with AF which has also been seen in drug and alcohol abuse, and neurologic disorders such as subarachnoid haemorrhage and stroke. Familial AF has also been associated with an increased risk of AF[7]. This occurrence has been occasionally associated with defined ion channel abnormalities, especially sodium channels[7].

Anatomical basis of AF pathoaetiology

The importance of focal pulmonary vein triggers has also been highlighted[6,8,9]. This has been the basis for surgical and ablation therapy for AF. The automatic focus theory and the multiple wavelet hypothesis appear to have the best supporting evidence for the occurrence.

Support for the automatic focus theory comes from several experimental models that show that AF persists only in isolated regions of atrial myocardium and indeed studies have demonstrated that a focal source of AF can be identified in humans and that isolation of this source can eliminate AF[6]. Although other foci have been shown in several locations within the atria, the pulmonary veins appear to be the most frequent source of these automatic foci. It is theorized that the heterogeneity of electrical conduction around the pulmonary veins promotes reentry and sustained AF as the cardiac muscle in the pulmonary

veins has active electrical properties differ from that of atrial myocytes. Therefore the initiating events may arise from pulmonary vein automatic triggers while the heterogeneity of conduction may provide the permissive atrial substrate for reentry.

The multiple wavelet hypothesisgarners its support from the observation that AF is common in cases of increased atrial mass[8]. This theory proposes that wave fronts propagating through the atria fractionate resulting in self-perpetuating "daughter wavelets." The refractory period, conduction velocity, and mass of atrial tissue determine the number of wavelets. The shortened atrial refractory period, delayed intra-atrial conduction and increased atrial mass increase the number of wavelets and promote sustained AF[8,9]. This provides a pathophysiological basis for therapeutic approaches in the management of AF.

Diagnosis of Atrial Fibrillation

Symptoms of AF are typically palpitations, chest pain, dizziness, breathlessness, sleep apnoea, and anxiety. Around a third of cases are asymptomatic[1] thus the importance of being aware of typical co-morbidities and associated risk factors.

Routine pulse-taking plays an important role in the detection of AF[10]. An irregularly irregular pulse should raise the suspicion of AF and necessitate an electrocardiogram (ECG) to diagnose it. It is advised that at least 30 seconds on a rhythm strip should be recorded for a diagnosis of AF[1].

The ECG, which can be in the form of a 12-lead ECG, bedside telemetry or ambulatory Holteror Event recordings, should be utilized to diagnose and/or describe the type of AF. It is described as Paroxysmal AF if the episodes terminate spontaneously within 7 days of onset, Persistent AF if the episodes do not terminate spontaneously but do convert with either electrical or pharmacological cardioversion, and Permanent AF if the episodes do not terminate either spontaneously or with electrical or chemical cardioversion, or where cardioversion had not been attempted. An echocardiogram is an important examination to perform to aid in the diagnosis of the underlying pathology as are basic laboratory investigations that may also be required to guide management that could reduce or eliminate the occurrence of further AF and / or improve the overall outcome of the patient[11].

The ECG findings in AF include a typically irregularly irregular ventricular rate at 110-160 beats/min with no discrete p waves which are replaced with irregular chaotic f waves. There may be aberrantly conducted beats after long-short R-R cycles (Ashman phenomenon) with pre-excitation. There may be other features of heart disease such as left ventricular hypertrophy, bundle-branch block, acute or prior MI.

Echocardiography is an important test to perform to evaluate for structural and functional lesions. This is more important in countries like Zambia where RHD valvular lesions are a common cause of AF. Transesophageal echocardiography (TEE) is preferred over transthoracic echocardiograph (TTE) as it is better able to detectleft atrial or atrial appendage thrombus.

The chest X-ray is useful in identifying lung or vascular pathology though it is usually normal. CT and MRI Scans are advised for the evaluation of atrial anatomy especially if ablation is planned. Images obtained can be utilized to create anatomic maps of the left atrium and pulmonary veins. More advanced imaging methods such as the preablation delayed-enhancement magnetic resonance imaging (DE-MRI) are said to be more useful.

Laboratory tests are done to uncover underlying disorders and/or for risk stratification. These include a full blood count (to exclude anaemia, infection), serum electrolytes and creatinine (to exclude electrolyte imbalance and renal dysfunction), Cardiac enzymes (Ck-MB, Troponin level (to exclude MI), B-natriuretic peptide (to evaluate for HF), Thyroid function tests (to exclude thyrotoxicosis, an important precipitant).

An exercise test may be required to exclude exercise-induced AF and/or myocardial ischemia while a six-minute walk may be used to evaluate for adequacy of rate control during therapy[12].

The EP studies are utilised to identify mechanisms of certain arrhythmias such as the wide-QRS tachycardia, precursor for AF and for locating sites for curative ablation or AV node ablation. These have now become routine investigations in well organized cardiac centres.

METHODOLOGY

The data were collected as part of a multi-centre registry on AF, and targeted patients presenting to the adult admission ward of the University Teaching Hospital (UTH). Demographic and clinical data were collected by interviews and review of consequent clinical records of patients enrolled. Informed written consent was obtained from the participants. Based on the clinical record and history, the AF was described as paroxysmal, persistent or permanent. The use of oral anticoagulation treatment was further explored in a subgroup of these patients. The 3 most recent INR values prior to the emergency department visit were collected for the patients on oral anticoagulation. In patients with at least two available INR results with a maximum of 63 days between consecutive INR values, the degree of INR control was to be calculated and presented as the time in therapeutic range.

RESULTS

Ten clinical characteristics for the primary analysis were defined namely; age, hypertension, diabetes mellitus, coronary artery disease, left ventricular dysfunction, heart failure, valvular heart disease, stroke/Transient Ischaemic Attack (TIA), obesity and alcohol use.

The clinical characteristics of patients enrolled are presented in Table 1. A total of 36 patients (15 male (42%) and 21 female (58%)) were enrolled with an average age of 45 (16-90) yrs. The average systolic blood pressure was 118 (90-160) mmHg and diastolic blood pressure of 77 (60-100) mmHg with an average pulse rate of 92 (72 - 132)/minute. Twelve (33.3%) of them had a history of hypertension but only 9 of them were on medication. The weight ranged between 34 – 75 kg. None of them could be described as obese.

History and Type of Atrial Fibrillation

Table 1: Patient Characteristics compared with presentation in the rest of Africa and the world(1)

	Zambia	Africa [1]	World [1]
Average Age (years)	45	51.2	65.4
Men (%)	41.7	46.9	52.8
Uncontrolled Hypertension (%)	47.8	53.8	62.0
Heart failure (HF) (%)	61.1	63.1	34.7
Rheumatic Heart Disease (RHD) (%)	52.8	21.5	11.6
Left ventricular hypertrophy (%)	69.4	21.7	38.1
Use of Aspirin (%)	42.9	27.2	36.2
Use of Warfarin (%)	6.9	15.9	23.3
Use of Amiodrone (%)	2.8	3.3	8.7
Use of Beta -blockers (%)	2.8	41.8	21.7

Twenty-two (61%) of the patients had prior history of AF, thus only 14 (39%) of them had the AF diagnosed for the first time on this admission. Of these, 33 (91.7%) had Permanent AF and 3 (8.3%) had Persistent AF, none could be described as paroxysmal AF based on the history although the AF spontaneously cardioverted during the admission period in 2 of the patients.

Co-morbid conditions in which Atrial Fibrillation was manifest:

Table 2 shows the different conditions in which AF was found in the patients. Most significant was the finding of Rheumatic Heart Disease (RHD) valvular lesions in 52.8% of the patients most of which were mitral valve related (75.0% mitral stenosis & 22.2% mitral incompetence). The other valves involved included the tricuspid valve (8.4%) and aortic valve (5.6%). Heart failure and Hypertension also played significant roles in the causation of AF. Heart failure was found in 22(61%) patients and Hypertensive Heart Disease in 12(33%) patients. Co-morbidities found in the study population included previous stroke or transient ischemic attacks(TIAs) in 8 (22.2%) patients, dementia1 (2.8%), pericarditis2 (5.6%), emphysema1(2.8%), diabetes mellitus2(5.6%) and hyperthyroidism1(2.8%).

Table 2. The cardiac and co-morbid conditions in which AF was manifest

Condition	Prevalence (n)	Prevalence (%)
Congenital Heart Disease		
- Ebstein anomaly	1	2.8
Heart failure	22	61.1
Cardiac surgery	6	16.7
Rheumatic Heart Disease	19	52.8
- Mitralregurgitation	8	22.2
- Mitral stenosis	15	75
- Aortic regurgitation	1	2.8
- Aortic stenosis	1	8.4
- Tricuspid regurgitation	3	8.4
- Valvuloplasty	4	11.1
Hypertensive Heart Disease	12	33.3
LVH	25	69.4
Stroke/Transient Ischemic Attack	8	22.2
Dementia	1	2.8
Pericarditis	2	5.6
Emphysema	1	2.8
Diabetes mellitus	2	5.6
Hyperthyroidism	1	2.8

Management of Atrial Fibrillation

A. Rate and rhythm control

The treatment given to the different patients of AF prior to admission is indicated in table 3. This was indicative of the drugs the patients were given in the primary care setting where they were attended to prior to the acute admission. The rate control medication included beta-blockers1(2.8%), Calcium Channel Blockers 3(8.3%), Digoxin (66%) & Amiodarone 1(2.8%)). The beta-blocker prescribed for the lone patient in this database was atenolol, while calcium channel blockers included verapamil in 1 patient and diltiazem in 2 patients. Digoxin was used in a larger population which included mostly patients with heart failure. Amiodarone was given to just 1 patient in this database. Amiodarone can be said to have been the only rhythm control medication given in this database. No other rhythm control medications were documented.

Table 3: Medication given to the patients of AF in primary care

Drug	Prevalence (n)	Prevalence (n)
Digoxin	24	66.7
Amiodarone	1	2.8
Diuretics	26	72.2
Angiotensin Converting Enzyme Inhibitors	11	30.6
Calcium Channel blockers	3	8.3
Betablockers	1	2.8
Anticoagulants/Antiplatelets		
- Warfarin	2	6.9
- Aspirin	15	42.9

B. Anti-thrombotic medication

Less than half 21 (42.9%) of the patients with AF were on aspirin. None were on clopidogrel. Only 03(6.9%) patients had ever been on the vitamin K antagonist oral anticoagulant (OAC), warfarin, with varied follow-up procedures. Only 2 of them had 2 or more INR results.

The other co-morbid conditions were also being treated accordingly eg heart failure, diabetes, hypertension etc.

DISCUSSION

AF is a common but rather complex condition that requires multiple interventions being pursued in parallel for optimal results[1].While AF is said to be commoner in the older age group, the Zambian average age for AF was 45 years. This age is much younger than the average of 52.1 years recorded for the rest of Africa and 65 years reported for the whole world (Table 1). This calls for concerted effort to prevent and/or adequately manage it to mitigate its effects on morbidity and mortality.

Control of ventricular rate, and/or control of ventricular rhythm and antithrombotic therapy, with adequate therapy of concomitant cardiac diseases are all essential for the management of AF. While it is preferable to have set treatment protocols and management guidelines, like many other serious clinical conditions, AF requires customized management[13]. Needless to say that a newly diagnosed AF needs thorough investigations to establish the pathoaetiology and to determine whether it is a primary or secondary event. There are currently no set treatment guidelines/protocols for AF at the UTH.

Heart failure and Atrial Fibrillation

Heart failure (HF) and AF are said to be "twins". While HF may predispose to the development of AF, AF predisposes to HF[29]. The pathophysiology through which HF predisposes to an arrhythmogenic atrial substrate include elevated left sided-filling pressures, mitral regurgitation, atrial enlargement, interstitial fibrosis and electromechanical remodelling[30].The activation of the autonomic system and renin-angiotensin axis in HF also contribute to the pathophysiological mechanisms[31].0n the other hand, AF may also precipitate or exacerbate heart failure. The increase in resting heart rate and the exaggerated heart rate response to exercise result in shorter diastolic filling time, leading to a reduction in cardiac output[32]. This is worsened by the irregularity of the ventricular response. The loss of effective atrial contractile function also contributes, even more importantly in patients with diastolic dysfunction[33]. AF may therefore worsen heart failure in individuals who are dependent on the atrial component of the cardiac output. Furthermore, development of new AF in a failing heart may pose a significant metabolic demand on the myocardial tissue until compensatory metabolic pathways are upregulated[34]. Thus the development of AF in heart disease predicts heart failure and is associated with a worsened New York Heart Association Heart Failure classification. Those with hypertensive heart disease and those with valvular heart disease are particularly at high risk for developing heart failure when

AF occurs. In addition, AF may cause tachycardia-mediated cardiomyopathy if adequate rate control is not established.

Management of AF therefore requires optimal attention being paid to the management of HF. However, the use of angiotensin converting enzyme inhibitors (ACEi) was low in this cohort of patients, as was the use of third generation beta-blockers, the two groups of medication that are said to be the hallmark of HF management. Only 11 (30.6%) of patients in this cohort were on ACEi and none were on the third generation beta-blockers for the treatment of heart failure.

Hypertension and Atrial Fibrillation

While 12 (33.3%) patientshad raised blood pressure at the time of admission, only 9 of them were on any anti-hypertensive medication. However, the presence of LVH in 69.4% of the participants indicates a greater prevalence of possible poorly controlled chronic hypertensive states. Among the patients on antihypertensive therapy only 52.2% had BP in the normtensive range. This indicates a 47.8% failure to control hypertension in this study population. While this compares well with what is obtaining in the rest of Africa on average (Table 1), it underlines a great need for treatment guidelines and protocols for hypertension control to be adjuvant to the ones being advocated for in this paper.

Rheumatic Heart Disease (RHD) and Atrial Fibrillation

The prevalence of rheumatic valvular heart disease was quite high in our sample (Table 1). At a prevalence of 52.8% this is far in excess of the African average of 21.5% documented by **Oldgren J et al**[1]and even higher than the national average for India which has been classified as "the worst affected"[1]. This is a disease that is preventable and thus calls for active engagement of the public health practitioners and capacity building of the health workers dealing with children to have competencies for adequate treatment of streptococcal sore throats and early detection of rheumatic fever. Discovering RHD at this late stage pauses additional clinical management challenges as the valvular involvement becomes a serious risk factor for both HF and AF and the treatment options include costly surgery.

Atrial Fibrillation and Thromboembolism

The Framingham Heart Study[35] has shown a strong association between AF and thromboembolic events. In this study nearly a quarter (22.2%) of the patients sampled had a history of having suffered stroke. This is very similar to the findings in this study. We described 8 (22.2%) of patients in this study population as having either suffered stroke or transient ischeamic attack. This is a worrisome occurrence and needs particular attention. The risk of stroke from AF is said to be greater when the arrhythmia lasts longer than 24 hours thus bringing a major clinical management concern to the health workers in the primary care settings. Disruption of normal atrial electromechanical function in AF leads to blood stasis. This, in turn, can lead to development of thrombus, most commonly in the left atrial appendage. Dislodgement or fragmentation of a clot can then lead to embolic phenomena, including stroke[35].

The risk of stroke is said to be directly related to stroke risk factors as described by the CHADS2 score and independent of the pattern of AF (i.e. paroxysmal, persistent, or permanent).Meaning all types of AF are thrombogenic

Electrodiagnosis of Atrial Fibrillation at UTH

Specific recognition of the arrhythmia is of utmost importance for purpose of optimal clinical care and treatment as stated above. The importance of clinical evaluation of the pulse cannot be overemphasized. An irregularly irregular pulse should provide a very high degree of suspicion for the diagnosis of AF. However, AF is diagnosed on electrocardiography. There is, therefore, need to have electrocardiographic equipment available in all clinical areas. At the time of this study only the routine ECG monitor was available in UTH and was done on all the patients in this database. The ECG not only demonstrated the typical ECG pattern of AF but also revealed underlying pathophysiological diagnoses or resultant complications of adjuvant pathologies. However, the routine 12 lead ECG is not very sensitive as paroxysmal AF may be missed if the recording is made when the sinus rhythm is re-established. The Holter ECG or Events Monitor would be the preferred piece of

equipment to use. This facility is not yet available at the UTH. This may explain the absence of the category of Paroxysmal AF in this database.

Management of Atrial Fibrillation

Rate and Rhythm Control.

The overall goal of rate and rhythm control is to improve symptoms and clinical outcomes[14,15,16]. Ventricular rate is an important factor in the pathophysiology. Factors that influence atrio-ventricular (AV) conduction, both intrinsic and extrinsic, are said to be the main determinants of ventricular rate response in AF. Intrinsic AV nodal conduction properties are therefore principal as are the dromotropic influences of the autonomic nerves on AV nodal activity.

Rate control medication is encouraged in patients with AF to allow for adequate ventricular filling time to improve cardiac output. These drugs act primarily by increasing the AV Nodal refractoriness[16]. Rate control medication include beta-blockers (Atenolol, Bisoprolol, Metoprolol), Non-dihydropyridines calcium channel antagonists (Diltiazem, Verapamil), Digitalis glycosides (Digoxin, Digitoxin) and Other drugs eg Amiodarone. In our study only 1 (2.8%) patient was on thebeta-blocker Atenolol and 3 (8.3%) were on calcium channel blockers. This is a very low use of rate control medication even when compared to the rest of Africa which is reported to have a generally low rate of beta blocker use at 21.7% (1). While beta-blockers or rate-limiting calcium channel blockers are preferred for initial monotherapy, in the elderly or sedentary patients digoxin monotherapy may suffice. Digoxin is indicated in patients with heart failure and systolic dysfunction but may suffice as a rate control agent in AF, thus may be used in acute conditions although it is said to be rather ineffective in active patients. Amiodarone may also be used as a rate control agent especially in patients who are intolerant of diltiazem or metoprolol[5]. Lenient rate control (< 100 bpm) is advised as strict rate control (<80 bpm) is said to be not as beneficial and less convenient[17]. However, if the patient remains symptomatic, a stricter HR control (<80 bpm) may be beneficial[18].

Rhythm control, once considered as the primary goal of therapy in AF, has been shown to be no better than rate control[15,16,18]. Antiarrhythmic drugs are used to attain and maintain sinus rhythm. These include multi-channel blockers (Amiodarone, Dronedarone), Potassium channel blockers (Dofetilide), and Sodium channel blockers (Disopyramide, Flecainide, Propafenone, [d,l-]sotalol). In this study, this line of drugs was also underutilized with only 1 (2.8%) patient being on Amiodarone. A rhythm control strategy is highly recommended for symptomatic patients who are younger[13] as was the case with some of our patients. This is also recommended for patients who present for the first time with lone AF, and those with AF secondary to a treated/corrected precipitant[13,16,18]. Those patients in whom a rhythm control strategy remains desired specialist alternative therapies such as cardioversion, catheter ablation, surgery, and left atrial appendage closure/occlusion may be considered[3,5,13,14]. Indeed there are patients in whom early rhythm control is preferred[3,5].

Rate control should be attempted first, as studies have failed to show a survival benefit for rhythm-control over rate-control therapy[14,16]. Amiodarone has been shown to be an optimal agent to use for this purpose. However, ideally an ECG is mandatory in all cases of AF for treatment to exclude pre-excitation syndromes as blocking the AV node in these patients may cause an exclusive impulse transmission through the accessory pathway that may precipitate ventricular fibrillation. This would necessitate the use of a defibrillator which is hard to find in the UTH. Calcium channel blockers and digoxin are therefore contraindicated in these patients. Amiodarone and flecainide would be preferred instead[19].

Cardioversion

An attempt to revert atrial fibrillation rhythm to sinus rhythm is advised and may be urgent in new onset AF (especially within 7 days of onset)[19,21], heart failure[20], angina pectoris[20] and/or hypotension[20]. Cardioversion may be either pharmacologic or electrical (DC shocks using a defibrillator). Pharmacological cardioversion was the only type attempted in the UTH. While this was preferred in UTH it has a potential risk of initiating ventricular tachycardia and other serious arrhythmias[22]. Defibrillation is the more preferred mode for achieving sinus rhythm in symptomatic patients of AF. This may be done electively or as an emergency[19,21]. The elective mode

is preferred as it gives time to initiate anticoagulation prior to the procedure thus reducing the risk of thromboembolism. The procedure requires the use of sedation and/or anaesthesia.

Overall, in patients with chronic, symptomatic AF, initial therapy may be anticoagulation and temporary rate control, while the long-term goal will be to restore sinus rhythm by cardioversion. The decision should be symptoms directed and patient centred.

Ablation Therapy

Electrophysiologists have taken over the management of AF, appreciating the electrophysiological basis of the generation and conduction pathways of the flutter waves[23,24,25]. Thus ablation therapy[24,25,26,27] has now become the ultimate in the treatment of AF. While surgical ablation, as in the Cox-Maze III procedure, was the forerunner[25,27], catheter ablation, as in the Saline Irrigated Cooled Tip Ablation (SICTRA) is now preferred[24].In this procedure complete lines of ablation or suture lines are said to offer fixed anatomical obstacles with which fibrillatory wavelets collide thus limiting the critical mass required to sustain AF. Individuals with focal origins of the AF also benefit from catheter ablation[27]. About 80% success in reduction of frequency and duration of AF in patients who do not tolerate AF has been documented resulting from simple electric isolation of the origins of the pulmonary veins[24]. However, ablation therapy is still "a work in progress". While SICTRA has been effective in restoring sinus rhythm in patients undergoing Mitral Valve Repair and persistent AF, only a reduction of AF in persistent AF following the Cox-Maze III has been shown[26]. However both procedures have not demonstrated any reduction in survival[26].

Anticoagulation Therapy

Anticoagulation therapy has been shown to reduce AF-related morbidity and mortality[1]. However, of the patients who were included in our study, only 3(6.9%) patients had everbeen on OAC (warfarin) and only in 2 did we find more than 2 INR readings which were all outside the therapeutic range (2.5-3.0). Anti-platelet therapy was better utilized with 41.7% of patients being on low dose Aspirin.

The main constraint to the use of warfarin has been said to be the availability of laboratory capacity to determine the International Normalized Ratio (INR). In the absence of INR monitoring it would be considered more risky to start anticoagulation. However, antiplatelet therapy is known to be inferior to anticoagulation in preventing stroke.

A rather low threshold is advised for the use of oral anti-coagulants (OAC). It is generally advised that OAC should be considered for patients with AF with at least one stroke risk factor provided there are no contraindications.Anticoagulation should be based upon the absolute risks of stroke and the bleeding risk–benefit ratio for thepatient. A titrated dose of OAC, such as warfarin, is recommended to achieve the target intensity international normalized ratio (INR) of2.0 –3.0. INR should be monitored at least weekly during initiation of therapy and monthly when anticoagulation is stable. While the ESC[3] guidelines acknowledge that patients with just one (non-major) risk factor, aspirin 75–325 mg/d is acceptable, an OAC is still preferred[1,35].

The newer oral anticoagulants are promising to change the approach to anticoagulation though the cost may prove to be a challenge to their use in primary care settings in the low middle income countries like Zambia. The oral direct thrombin inhibitor dabigatran etexilatehas been said to be a good alternative to warfarin[37,38].Another drug is Rivaroxaban[39], a highly selective direct Factor Xa inhibitor. It has demonstrated equivalent efficacy to warfarin in patients with nonvalvular AF[39]. Another new drug is the Factor Xa inhibitor, Apixaban which has been shown to be superior to warfarin in preventing stroke or systemic embolism, causing less bleeding, and lower mortality[41,42,43]though caution is being advised in its use in patients with a prosthetic heart valve or AF caused by a heart valve problem. However, an individual's risk of bleeding can be determined using the HAS-BLED score which provides a practical tool to assess the individual bleeding risk and can aid clinical decision making regarding antithrombotic therapy in patients with AF[44].

What about treatment guidelines and clinical practice protocols?

The findings of this study demonstrate a need for local treatment guidelines to mitigate the effects on AF on morbidity and mortality. Several treatment guidelines do exist facilitated by different regional bodies such as the European Society for Cardiology(ESC), the American College of Cardiology(ACC), the American Heart

Association (AHA), and the Canadian Cardiovascular Society. These guidelines show notable differences reflecting different approaches being taken by the bodies responsible in response to their local environments. This calls for an adaptation of these guidelines to suit the Zambian environment and to address the perculiar challenges being evidenced. There is therefore reason to develop Zambian specific, evidence-based clinical guidelines and practice protocols for the management of AF in primary care settings. This is a matter that calls for urgent action.

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