Atrial fibrillation

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Abstract

Atrial fibrillation (AF) is the most common cardiac arrhythmia, with a high prevalence in elderly individuals. It is associated with increased mortality and morbidity, as a result of stroke, systemic embolism and heart failure. Stroke prevention is central in management of AF. AF management can be distilled into a simple integrated care approach – the ABC (Atrial fibrillation Better Care) pathway: (1) Avoid stroke, with anticoagulation. The risk of stroke in AF is related to increasing age and coexistent co-morbidities. All patients with AF should be risk stratified for stroke and bleeding, with the CHA_2DS_2-VASc and HAS-BLED scores, respectively. Until recently, vitamin K antagonists (VKAs) were the mainstay of antithrombotic therapy, but non-VKA oral anticoagulants are now increasingly preferred. (2) Better symptom management. The subsequent approach to management of AF is largely patient-centred and symptom-driven. It can be broadly described as 'rhythm control' and 'rate control'. Rate control is usually with β-blockers or non-dihydropyridine calcium channel blockers. Rhythm control may require antiarrhythmic drugs and/or electrophysiological procedures. (3) Cardiovascular and other risk factor management. Associated co-morbidities such as hypertension, diabetes mellitus, heart failure, cardiac ischaemia, and sleep apnoea should be addressed, and lifestyle changes (obesity, alcohol excess) discussed.

Keywords Anticoagulation; atrial fibrillation; MRCP; non-vitamin K anticoagulant oral anticoagulant; rate control; rhythm control; stroke; vitamin K antagonist

Key points

- Atrial fibrillation (AF) is a major cause of cardiovascular morbidity and mortality.
- Early diagnosis and timely treatment are crucial for management of AF, but remain challenging.
- AF requires integrated management, which can be provided with a simple 'ABC' strategy, ie. Avoid stroke, Better symptom management, and Cardiovascular and comorbidity risk reduction.

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice. In both developed and emerging economies, the incidence of AF is increasing as a result of improved longevity and expansion of the elderly population. It is projected that, by 2050, 2% of the general population will have AF. The prevalence of AF is age-dependent, affecting <0.5% of subjects aged 40–50 years, but 18% in individuals aged ≥85 years. Despite progress in the management of AF, this arrhythmia is still a major cause of cardiovascular morbidity and mortality by increasing the incidence of stroke, heart failure and sudden death. AF confers a 5-fold increase risk of stroke compared with normal sinus rhythm. Stroke in association with AF generally results in higher mortality, greater disability and longer inpatient stays than stroke in the absence of this arrhythmia. Furthermore, dementia and impaired quality of life associated with AF are well recognized. The treatment of AF and AF-related complications represents a costly public health burden, which will continue to increase unless AF is prevented and treated in a timely holistic manner.[1]

Definition and classifications of AF

AF is classified into paroxysmal, persistent, long-standing persistent and permanent based on the temporal pattern of arrhythmia. Decision-making on anticoagulation therapy for stroke prevention is driven by risk factors rather than pattern of AF or apparent success of rhythm control.

- Paroxysmal AF – recurrent, self-terminating episodes lasting <7 days (usually <24 hours).
- Persistent AF – episodes lasting >7 days or requiring termination by either pharmacological or electrical cardioversion.
- Long-persistent AF – AF lasting >1 year when decision is made for rhythm control strategy.
- Permanent AF – AF lasting for >1 year with a decision made by both patient and physician not to pursue rhythm control therapy.

Risk factors for development of AF

In population-based prospective studies, advancing age, heart failure, valvular heart disease, myocardial infarction, diabetes mellitus and hypertension were significantly associated with increased risk of developing AF. In addition, thyroid dysfunction, chronic obstructive pulmonary disease, sleep apnoea syndrome, chronic kidney disease, obesity, smoking and alcohol consumption are known risk factors (Table 1).

Brief overview of AF pathophysiology

The pathophysiology relating to the development of AF is complex, involving structural changes to the atrium and electrophysiological alterations. Intrinsic structural changes involve proliferation and differentiation of fibroblasts into myofibroblasts, resulting in atrial fibrosis. This leads to electrical dissociation between muscle bundles and local conduction, with

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Risk factors for the development of atrial fibrillation

Cardiac	Hypertension
	Ischaemic heart disease
	Congestive heart failure
	Rheumatic valvular heart disease
	Sick sinus syndrome
	Pre-excitation syndromes (Wolff–Parkinson–White syndrome)
	Atrial septal defect
	Atrial myxoma
	Pericarditis and pericardial effusion
	Cardiomyopathies
	Postoperative AF (POAF)
Non-cardiac	Acute infections
	Electrolyte imbalance
	Chronic obstructive pulmonary disease
	Pulmonary embolism
	Lung carcinoma
	Pleural effusion
	Thyrotoxicosis
	Chronic kidney disease
	Sleep apnoea syndrome
Miscellaneous	Obesity, smoking, alcohol, excess caffeine, illicit drugs (e.g. cocaine, amphetamine, MDMA), emotional and physical stress

Table 1

persistence and stabilization of small re-entrant circuits. At the same time, intracellular changes involving inward Ca^+ and K^+ currents occur, causing shortening of atrial refractory periods. The overall effect is to cause electrophysiological changes in the orientations of myocyte fibres in the pulmonary vein, which perpetuates or initiates further AF. The combination of cellular, structural and electrical remodelling provides the substrate promoting the initiation and self-perpetuation of this arrhythmia, causing AF to beget more AF.

Thrombogenesis in AF

The presence of AF results in a 5-fold increase in risk of stroke. This can be explained by components of Virchow's triad – abnormal changes in the vessel wall, blood flow and blood constituents – leading to a prothrombotic state.[2] Abnormal vessel wall' is recognized as gradual left atrial dilatation, endocardial denudation and microscopic fibro-elastic changes in the extracellular matrix, which promote thrombogenesis. 'Abnormal blood flow' resulting from loss of atrial systole manifests as stasis within the left atrium, spontaneous echocardiographic contrasts in the left atrium on trans-oesophageal echocardiography and reduced left atrial appendage velocities; these facilitate thrombus formation. 'Abnormal blood constituents' involving platelet activation and abnormal coagulation indices have also been shown to contribute to thrombogenesis.

Clinical features, detection and confirmation

The most common symptoms in patients with AF are palpitations, shortness of breath, chest pain, dizziness and fatigability. However, a significant proportion of patients with AF (around 12–15%) are asymptomatic, with the arrhythmia discovered by chance. It is not common to diagnose incidental AF in patients with so-called cryptogenic stroke, especially with more prolonged monitoring.

Detection of an irregular pulse should trigger suspicion of AF and allow for confirmation by electrocardiogram (ECG). A standard 12-lead ECG is the gold standard tool for the diagnosis of AF; this demonstrates loss of distinct P waves, oscillating baseline f (fibrillation) waves and irregular R–R intervals. Given the paroxysmal, asymptomatic or self-terminating nature of AF, long-term ECG monitoring such as ambulatory Holter ECG monitoring (24 hours–7 days) and implantable loop recording can be useful to detect undiagnosed AF.

Cardiac implantable electronic devices such as pacemakers and defibrillators can detect atrial high-rate episodes (AHREs), which are associated with documented AF on ECG, and increased risk of stroke. However, there is still debate over whether AHREs confer the same therapeutic requirements as overt clinical AF, hence their alternative terminology of 'subclinical atrial tachyarrhythmias'.

General management strategy in AF patients

AF management can be distilled into a simple integrated care approach: the ABC (Atrial fibrillation Better Care) pathway, as follows: (1) Avoid stroke, with anticoagulation; (2) Better symptom management, with patient-centred decisions on 'rhythm control' or 'rate control'; and (3) Cardiovascular and other risk factor management.

The cornerstone of management in AF is stroke prevention.[3] Subsequently, the choice between rate control and rhythm control strategies depends on the pattern of AF as well as the patient's symptoms. Rate control is generally the first-line treatment, while rhythm control is used for the management of symptomatic, paroxysmal/persistent AF on optimal rate control therapy (Figure 1).

Avoid stroke

The risk of stroke with AF is not homogeneous and depends on several common stroke risk factors. Regardless of the temporal pattern of AF, the risk of ischaemic stroke and thromboembolism persists. The current approach is initially to identify truly 'low-risk' patients with AF who will not benefit from antithrombotic therapy.

Stroke and bleeding risk assessment

Various stroke risk stratification schemes have been proposed, but the CHA_2DS_2-VASc score is now widely used. This denotes Congestive heart failure, Hypertension, Age ≥75 (doubled), Diabetes mellitus, Stroke (doubled) – Vascular disease, Age 65–74 and Sex category (female) (Table 2). Low risk is defined as a CHA_2DS_2-VASc score of 0 in a male or 1 in a female patient. Patients with a CHA_2DS_2-VASc score of ≥1 (unless female and age <65) will have an estimated yearly stroke risk of >1.75%, warranting consideration for oral anticoagulation.

Any decision made for long-term prophylaxis against ischaemic stroke using oral anticoagulants should be balanced against risk of major haemorrhage, especially intracranial haemorrhage,

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