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EPIDEMIOLOGICAL STUDIES OF ATRIAL FIBRILLATION WITH OTHER COMORBIDITIES AND TREATMENT

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ABSTRACT
Atrial fibrillation (AF) is an increasingly prevalent arrhythmia; its pathophysiology and progression are well studied. Stroke and bleeding risk models have been created and validated. Decision tools for stroke prophylaxis are evolving, with better options at hand. Utilization of various diagnostic tools offer insight into AF burden and thromboembolic risk. Rate control, rhythm control, and stroke prophylaxis are the cornerstones of AF therapy. Although antiarrhythmic drugs are useful, AF ablation has become a primary therapeutic strategy. Pulmonary vein isolation is the cornerstone of AF ablation, and methods to improve ablation safety and efficacy continue to progress. Ablation of non-pulmonary vein sites is increasingly being recognized as an important strategy for treating non-paroxysmal AF. Several new ablation techniques and technologies and stroke prophylaxis are being explored. This is a contemporary review on the prevalence, pathophysiology, risk prediction, prophylaxis, treatment options, new insights for optimizing treatment outcomes, and emerging, concepts of AF.

KEY WORDS: Atrial fibrillation; rhythm control; rate control; AF ablation.

INTRODUCTION
Atrial fibrillation the most common type of cardiac arrhythmia. It is due to abnormal electrical activity within the atria of the heart, causing them to fibrillate. It is characterized as a tachyarrhythmia, which means that the heart rate is often fast. This arrhythmia may be paroxysmal (less than seven days) or persistent (more than seven days). Due to its rhythm irregularity, blood flow through the heart becomes turbulent and has a high chance of forming a thrombus (blood clot), which can ultimately dislodge and cause a stroke. Atrial fibrillation is the leading cardiac cause of stroke. Risk factors for atrial fibrillation include advanced age, high blood pressure, underlying heart and lung disease, congenital heart disease, and increased alcohol consumption. Symptoms vary from asymptomatic to symptoms such as chest pain, palpitations, fast heart rate, shortness of breath, nausea, dizziness, diaphoresis (severe sweating), and generalized fatigue. Although atrial fibrillation may be a permanent disease, various treatments, and risk modifying strategies have been developed to help reduce the risk of stroke in patients that remain in atrial fibrillation. Treatments include anticoagulation, rate control medication, rhythm control medication, cardioversion, ablation, and other interventional cardiac procedures.[1][2][3]

CLASSIFICATION
- Paroxysmal atrial fibrillation is atrial fibrillation that lasts < 1 week having converted spontaneously or by an intervention to normal sinus rhythm. Episodes may recur.[4]
- Persistent atrial fibrillation is continuous atrial fibrillation that lasts > 1 week.[4]
- Long-standing persistent atrial fibrillation lasts > 1 year, but there is still the possibility of restoring sinus rhythm.[4]
- Permanent atrial fibrillation cannot be converted to sinus rhythm (the term also includes patients for whom a decision has been made not to attempt conversion to sinus rhythm.[4] The longer atrial fibrillation is present, the less likely is spontaneous conversion and the more difficult is cardioversion because of atrial remodeling (rapid atrial rate-induced changes in atrial electrophysiology, including a decrease in atrial refractoriness, spatial dispersion of atrial refractoriness, slowed atrial conduction velocity, or combinations of these).[5]

HISTORY

History and physical exam are crucial for diagnosing and risk stratifying patients with atrial fibrillation. The presentation of AF can range from asymptomatic to devastating complications such as cardiogenic shock and ischemic stroke. A complete history should focus on symptoms such as palpitations, chest pain, shortness of breath, increased lower extremity swelling, dyspnea on exertion, and dizziness. In addition, history is imperative in identifying risk factors such as hypertension, history of valvular, structural, or ischemic heart disease, obstructive sleep apnea, obesity hypoventilation syndrome, smoking, alcohol intake, illicit drug use, history of rheumatic fever/heart disease, history of pericarditis, and hyperlipidemia. Initial evaluation of any patient presenting with features of AF should include the assessment for hemodynamic instability. Assessment of patients with existing AF includes questions regarding:

- History of triggers
- Duration and frequency of symptoms
- Previously successful modes of termination
- The use of anti-arrhythmic drugs
- Antecedent cardiac diseases

A physical exam should always begin with the assessment of airway breathing and circulation as it is going to affect the decision-making regarding management. On general physical examination, patients may be tachycardic with an irregularly irregular pulse. The heart rate usually ranges from 110/min to 140/min. Extremities should be evaluated for edema, peripheral pulses in both upper and lower extremities, and integumentary signs of peripheral vascular disease (PVD), such as hair loss and skin breakdown.

PATHOPHYSIOLOGY

The normal electrical conduction system of the heart allows electrical impulses generated by the heart's own pacemaker (the sinoatrial node) to spread to and stimulate the muscular layer of the heart (myocardium) in both the atria and the ventricles. When the myocardium is stimulated it contracts, and if this occurs in an orderly manner allows blood to be pumped to the body. In AF, the normal regular electrical impulses generated by the sinoatrial node are overwhelmed by disorganized electrical waves, usually originating from the roots of the pulmonary veins. These disorganized waves conduct intermittently through the atrioventricular node, leading to irregular activation of the ventricles that generate the heartbeat.

PATHOLOGY

The primary pathologic change seen in atrial fibrillation is the progressive fibrosis of the atria. This fibrosis is due primarily to atrial dilation; however, genetic causes and inflammation may be factors in some individuals. Dilation of the atria can be due to almost any structural abnormality of the heart that can cause a rise in the pressure within the heart. This includes valvular heart disease (such as mitral stenosis, mitral regurgitation, and tricuspid regurgitation), hypertension, and congestive heart failure. Any inflammatory state that affects the heart can cause fibrosis of the atria.

Once dilation of the atria has occurred, this begins a chain of events that leads to the activation of the renin–angiotensin–aldosterone system (RAAS) and subsequent increase in the matrix metalloproteinases and disintegrin, which leads to atrial remodeling and fibrosis, with loss of atrial muscle mass. This process occurs gradually, and experimental studies have revealed patchy atrial fibrosis may precede the occurrence of atrial fibrillation and may progress with prolonged durations of atrial fibrillation.

Fibrosis is not limited to the muscle mass of the atria and may occur in the sinus node (SA node) and atrioventricular node (AV node), correlating with sick sinus syndrome. Prolonged episodes of atrial fibrillation have been shown to correlate with prolongation of the sinus node recovery time[6] this suggests that dysfunction of the SA node is progressive with prolonged episodes of atrial fibrillation.

Along with fibrosis, alterations in the atria that predispose to atrial fibrillation affect their electrical properties, as well as their responsiveness to the autonomic nervous system. The atrial remodeling that includes the pathologic changes described above has been referred to as atrial myopathy.[7]

ETIOLOGY

There are many causes of atrial fibrillation (AF), but it shares a strong association with other cardiovascular diseases. The commonly encountered causes include:

- Advanced age
- Congenital heart disease
- Underlying heart disease - valvular disease, coronary artery disease, structural heart disease, atrial ischemia
- Increased alcohol consumption
- Hypertension - systemic or pulmonary
- Endocrine disorders - diabetes, pheochromocytoma, and hyperthyroidism
- Genetic factors
- Neurologic disorders - subarachnoid hemorrhage or stroke
- Hemodynamic stress - mitral or tricuspid valve disease, left ventricular dysfunction, pulmonary embolism
- Obstructive sleep apnea
- Inflammation - myocarditis and pericarditis

Any condition that leads to inflammation, stress, damage, or ischemia affecting the anatomy of the heart can result in the development of atrial fibrillation. In some cases, the cause is iatrogenic.[8]

SIGNS AND SYMPTOMS

Some people have symptoms, and some don't. It depends on how fast your ventricles are beating. If they're beating

at a normal or slightly elevated pace, you probably won't feel anything. But if your ventricles beat faster, you'll start to notice symptoms. These can include:

- Extreme fatigue.
- An irregular heartbeat.
- Heart palpitations.
- A feeling of butterflies or a fish flopping in your chest.
- Dizziness or lightheadedness.
- Fainting (syncope)
- Shortness of breath (dyspnea).
- Chest pain (angina).

RISK FACTORS

Some people who are living healthy lives and have no other medical problems do develop Atrial Fibrillation (AFib). The most common risk factors include:

- Older than 60 years of age
- Diabetes
- High blood pressure
- Coronary artery disease
- Cardiomyopathy
- Pericardial inflammation
- Prior heart attacks
- Congestive heart failure
- Structural heart disease (valve problems or congenital defects)
- Prior open-heart surgery
- Untreated atrial flutter (another type of abnormal heart rhythm)
- Thyroid disease
- Chronic lung disease
- Sleep apnea
- Excessive alcohol or stimulant use
- Serious illness or infection separator.

DIAGNOSIS

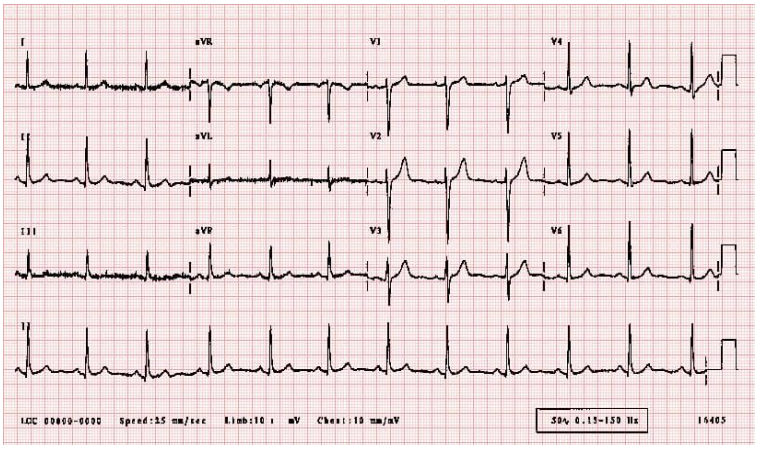
Atrial fibrillation is diagnosed on an electrocardiogram (ECG/EKG).

The evaluation of atrial fibrillation involves a determination of the cause of the arrhythmia, and classification of the arrhythmia. Diagnostic investigation of AF typically includes a complete history and physical examination, ECG, transthoracic echocardiogram, complete blood count, serum thyroid stimulating hormone level[9] and may include a functionality of some smartwatches.[10]

Screening

Numerous guidelines recommend opportunistic screening for atrial fibrillation in those 65 years and older. These organizations include the: European Society of Cardiology,[11] National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand,[12] European Heart Rhythm Society,[13][14] AF-SCREEN International Collaboration,[15] Royal College of Physicians of Edinburgh[16] European Primary Care Cardiovascular Society,[17] and Irish Health Information and Quality Authority.[18]

Electrocardiogram



ECG recordings of normal sinus rhythm (top) with atrial fibrillation and absence of P waves (bottom)Atrial fibrillation is diagnosed on an electrocardiogram (ECG), an investigation performed routinely whenever an irregular heartbeat is suspected. Characteristic findings are the absence of P waves, with disorganized electrical activity in their place, and irregular R–R intervals due to irregular conduction of impulses to the ventricles.[19] At very fast heart rates, atrial fibrillation may look more regular, which may make it more difficult to separate from other supraventricular tachycardias or ventricular tachycardia.[20]

QRS complexes should be narrow, signifying that they are initiated by normal conduction of atrial electrical activity through the intraventricular conduction system. Wide QRS complexes are worrisome for ventricular tachycardia, although, in cases where there is a disease of the conduction system, wide complexes may be present in A-fib with a rapid ventricular response.

If paroxysmal AF is suspected, but an ECG during an office visit shows only a regular rhythm, AF episodes may be detected and documented with the use of ambulatory Holter monitoring (e.g., for a day). If the episodes are too infrequent to be detected by Holter

monitoring with reasonable probability, then the person can be monitored for longer periods (e.g., a month) with an ambulatory event monitor.[19]

Echocardiography

In general, a non-invasive transthoracic echocardiogram (TTE) is performed in newly diagnosed AF, as well as if there is a major change in the person's clinical state. This ultrasound-based scan of the heart may help identify valvular heart disease (which may greatly increase the risk of stroke and alter recommendations for the appropriate type of anticoagulation), left and right atrial size (which predicts the likelihood that AF may become permanent), left ventricular size and function, peak right ventricular pressure (pulmonary hypertension), presence of left atrial thrombus (low sensitivity), presence of left ventricular hypertrophy and pericardial disease.[19]

Chest X-ray

In general, a chest X-ray is performed only if a pulmonary cause of atrial fibrillation is suggested, or if other cardiac conditions are suspected (in particular congestive heart failure). This may reveal an underlying problem in the lungs or the blood vessels in the chest.[19]

Transesophageal echocardiogram

A regular echocardiogram (transthoracic echocardiogram; TTE) has a low sensitivity for identifying blood clots in the heart. If this is suspected (e.g. when planning urgent electrical cardioversion), a transesophageal echocardiogram (TEE or TOE where British spelling is used) is preferred.[19]

The TEE has much better visualization of the left atrial appendage than transthoracic echocardiography.[21] This structure, located in the left atrium, is the place where a blood clot forms in more than 90% of cases in non-valvular (or non-rheumatic) atrial fibrillation.[22][23] TEE has a high sensitivity for locating thrombi in this area and can also detect sluggish blood flow in this area that is suggestive of blood clot formation.[21] If a blood clot is seen on TEE, then cardioversion is contraindicated due to the risk of stroke, and anticoagulation is recommended.

TREATMENT

You have been diagnosed with atrial fibrillation; you will have regular check-ups to make sure your symptoms are under control.

You may be given medicine to:

- control the rate and rhythm of your heart, such as beta blockers
- lower the risk of blood clots or stroke (anticoagulants)

Other possible treatments for atrial fibrillation include:

- surgery to burn or freeze a section of the heart (ablation)
- using electricity to reset your heart rhythm (electrical cardioversion)
- having a pacemaker or implantable cardioverter defibrillator (ICD) fitted
- Sometimes atrial fibrillation can be caused by another health condition or medicine. In these cases, treating the condition or stopping the medicine can improve the symptoms.

PREVENTION

Not all cases of atrial fibrillation can be prevented. By taking steps to avoid coronary artery disease or high blood pressure, however, you can help avoid developing A-fib due to these causes. The basics include not smoking, following a heart-healthy Mediterranean-style diet (high in plant-based foods, fruits and vegetables, and low in saturated fats), being physically active and keeping to a normal weight.

CONCLUSION

The aging of the population, improved survival after myocardial infraction and congestive heart failure, and the increase in the number of cardiac procedures will all contribute to the inevitable increase in the burden of AF. With roughly 2.2 million people currently suffering from AF, it is imperative that we focus on earlier detection, better individualized patient management, and, most importantly, reduction of the morbidity and mortality of patients suffering from AF with evidence-based therapies, as well as good clinical judgment.

ABBREVATIONS:

Atrial Fibrillation – AFIB (or) AF.

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