JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue

JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)

An International Scholarly Open Access, Peer-reviewed, Refereed Journal

RISK OF ISCHEMIC STROKE IN PATIENT WITH NON VALVULAR ATRIAL FIBRILLATION.

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ABSTRACT

Atrial fibrillation is an irregular rapid heart rate that commonly causes poor blood flow .it is the most common cardiac arrhythmia .Non valvular atrial fibrillation is not caused by a problem with the heart valve. It's caused by other things such as high blood pressure or an overactive thyroid gland. An atrial fibrillation without valves is associated with a considerable risk of thyroid cancer. The haemorrhage occurs in ischemic strokes. Treatment methods should be considered as pharmacological rate control, cardioversion and antiarrhythmic therapy. Restore and maintain sinus rhythm and prophylactic anticoagulation or Antiplatelet therapy to reduce the risk of stroke. The aim is to assess the Risk of ischemic stroke and prevention of the risk of stroke in non-valvular atrial Fibrillation. A process called WATCHMAN LAA was used to close the loop A device developed to prevent embolization of thrombi, which can form LAA Such patients include those with atrial fibrillation.

KEY WORDS

Atrial fibrillation oral anticoagulation Risk assessment stroke

INTRODUCTION

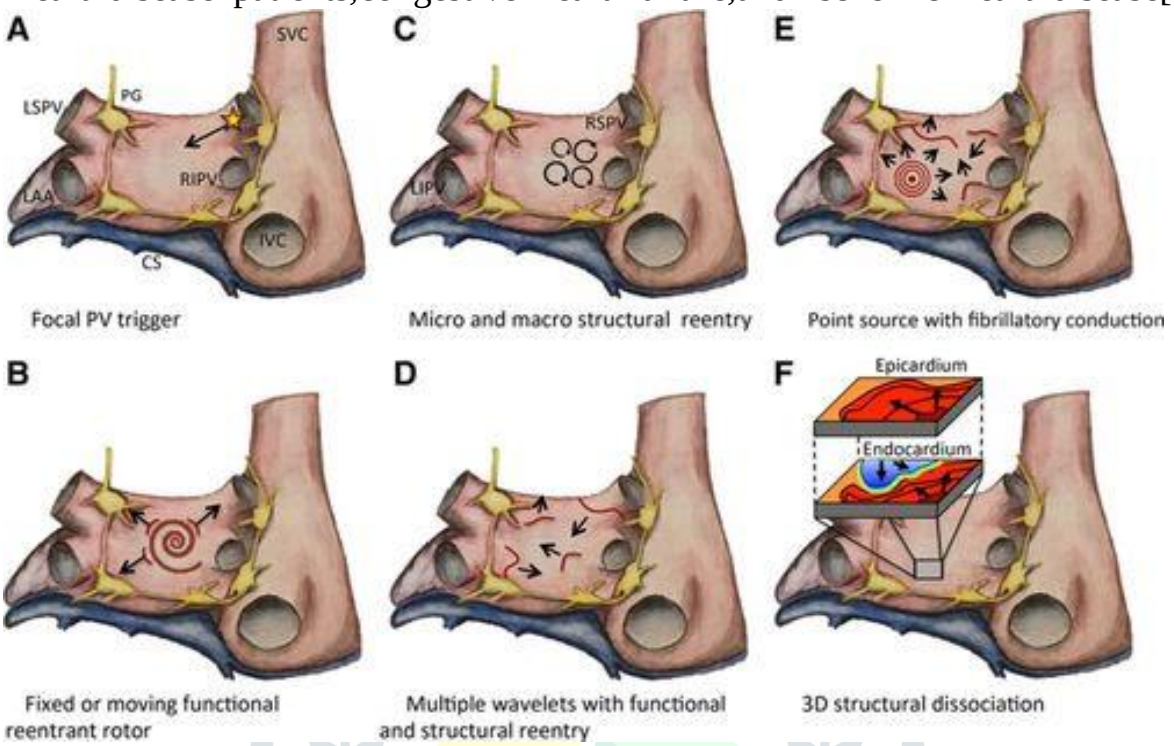
In the United States, more than 12 million people are projected to have atrial fibrillation (AF) by 2030[1,2,3]. The most serious complication of AF is stroke, and its prevention therefore represents the cornerstone of AF management[1,4]. There are many stroke prevention strategies, such as anticoagulation centres of excellence, direct oral anticoagulants, and percutaneous left atrial appendage occlusion devices (LAAO)[1,5]. These advancements have not been reflected in stroke risk prediction models. Currently, societal guidelines use a single clinical risk score(CHA2DS2-VASc) to determine an individual's eligibility for pharmacological ormechanical stroke prevention[6,7,8]. In real-world cohorts, however, despite its utility and ease of calculation, the CHA2DS2-VASc score has demonstrated modest performance in predicting strokes[9]. A number of key anatomical, physiological, and other factors that may influence the risk of stroke in patients with AF are also notincluded in the score. The purpose of this paper is to review current and emerging methodologies for determining ischemic stroke risk in patients with nonvalvular AF (NVAF), and to suggest frameworks for further investigation to achieve optimal stroke prevention for these patients. The data in this article were gathered via areview focused on ischemic stroke risk prediction in patients with atrial fibrillation (non-systematic review)

RISK FACTORS

- **Advanced age**: The number of adults developing A Fib increases markedly with older age. Atrial fibrillation in children is rare, but it can and does happen.
- **High blood pressure**: Longstanding, uncontrolled high blood pressure can increase your risk for A Fib.
- **Underlying heart disease**: Anyone with heart disease, including valve problems, hypertrophic cardiomyopathy, acute coronary syndrome, Wolff-Parkinson-White (WPW) syndrome and history of heart attack. Additionally, atrial fibrillation is the most common complication after heart surgery.
- **Drinking alcohol**: Binge drinking (having five drinks in two hours for men, or four drinks for women) may put you at higher risk for AFib.
- **Family history**: Having a family member with AFib increases your chances of being diagnosed.
- **Sleep apnea**: Although sleep apnea isn’t proven to cause A Fib, studies show a strong link between obstructive sleep apnea and A Fib. Often, treating the apnea can improve AFib.
- **Athletes**: AFib is common in athletes and can be triggered by a rapid heart rate called a supraventricular tachycardia (SVT).
- **Other chronic conditions**: Others at risk are people with thyroid problems (specifically hyperthyroidism), diabetes, asthma and other chronic medical problems.

PATHOPHYSIOLOGY

Atrial fibrillation needs a trigger to begin.the trigger is usually in the form of tachycardia or multiple wavelets extending to left atrium. The substrate for the maintainace of arrhythmia iscommonly heterogenous tissue [10].Hypothesis of focal automaticity in atrial tissue result in chaotic activation of atria. The focal automaticity most commonly occur in pulmonary veins which generate microreenterant circuits that extend to adjustant left atrial tissues[10,11].the stretching of atria is considered as a trigger of recurrent atrial fibrillation especially in valvular heart disease patients,congestive heart failure,and ischemic heart disease[12 ,13 ,14,15].



ECTOPIC FIRING

The hypothesis of AF propagate the reentrant waves in a vulnerable atrial substrate. The initiating trigger may decrease because of the AF substrate progresses and AF becomes more stabilized. Haïssaguerre was first to identify focal ectopic firing arising from myocyte sleeves within the pulmonary veins (PVs) in patients with paroxysmal AF. Removal of these ectopic foci reduced AF burden. (Figure A). It is now known that the PVs have specific electric properties and complex fiber structure that promote reentry and ectopic activity . The PV triggers has been attributed to abnormal calcium Ca^{2+}. A diastolic influx of Ca^{2+} from the sarcoplasmic reticulum activates an inward of Na^{+} current. The $Na^{+}Ca^{2+}$ exchanger resulting in spontaneous myocyte depolarization . Hyperphosphorylation of regulatory proteins and enzymes like protein kinase A, calmodulin kinase II, phospholamban, and the ryanodine receptor type 2, is essential to contribute to Ca^{2+} overload and diastolic membrane instability. A reentrant mechanism for PV triggers also has been described that a Gradual decrease in the conduction and repolarization of heterogeneity within the PV enable localized reentry and cause AF.

PERPTUATION: REENTRY

A vulnerable atrial substrate is important for triggering AF. Abnormalities in atria promote the perpetuation of AF by stabilizing reentry. The mechanism of reentry in AFis associated with 2 hypothesis , including reentrant rotors or multiple independent wavelets. There is a third hypothesis, called the double layer hypothesis estimated that electric dissociation of epicardial and endocardial layers also lead to reentry .For a functional reentry, the wavefront must complete 1 circus movement in a time period long enough for atrial tissue within that circuit to recover excitability. This lead to slow conduction velocity and a short ERP produce reentry. Atrial substrates that produce reentry have characterized by abnormalities of the atrial cardiomyocyte, fibrotic changes, and changes in the interstitial matrix with non collagen deposits.These changes histological changes impair normal anisotropic conduction and may shorten atrial ERP. . Thus, the development of the vulnerable atrial substrate is creating the predisposing AF

FACTORS THAT INCREASES THE RISK OF NON VALVULAR ATRIAL FIBRILLATION

- Drinking a lot of alcohol
- Regular smooch even in the past
- Obesity
- Too little exercise performance

GROUP AT RISK

- More risk at male when compared to female
- People older than 65 years
- Risk increase with age

Having history of any of the following condition

- Heart disease
- Lung disease
- Diabetes
- Sleep disorders
- Metabolic syndrome
- Pericarditis
- Hyperthyroidism
- Heart surgery

SYMPTOMS AND COMPLICATION

The most symptoms of atrail fibrillation as well as non valvular atrail fibrillation is quivering,fluttering or rapid heart beat,irregular pulse,thumping feeling in the chest.palpitation etc.

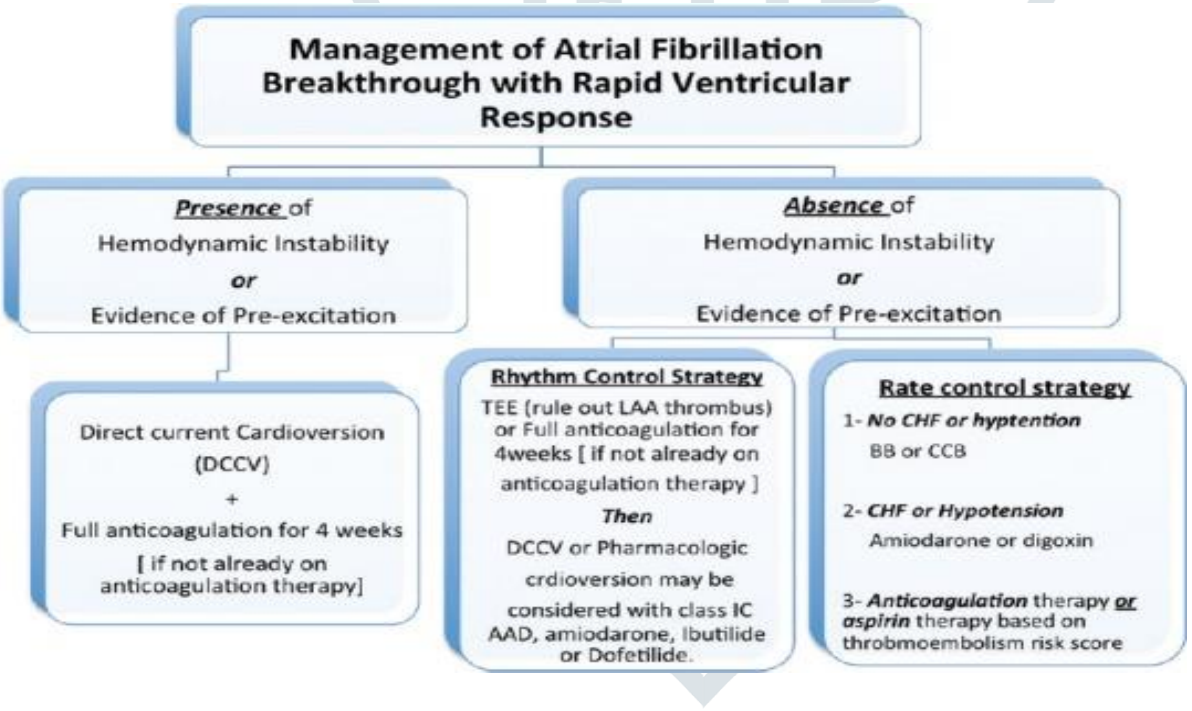
Other symptoms includes

- Shortness of breath

- Feeling week
- Tiredness
- Dizziness
- Fainting
- Chest pain or pressure

MANAGEMENT

Vit k antagonist are most effective for the treatment of thromboembolic events The vitk antagonist drugs have limitations in food and drug interactions .it have narrow therapeutic events it requires frequent monitoring with the particular concern for the greatest risk of intracranial events [16]. Alternative of vit k
antagonist are Direct Oral Anticoagulants(DOACS) which inhibit coagulation by directly and specifically binding to factorXa.Due to the large therapeutic index DOACS can be in fixed dose without routine coagulation monitoring and it have less food and drug interactions[17].Abixaban for reduction in stroke and other thromboembolic events in atrial fibrillation is termed as ARISTOTLE and compared apixaban with warfarin in patients having non valvular atrial fibrillation .



VITAMIN K ANTAGONIST THERAPY FOR ATRIAL FIBRILLATION

Vit k antagonist drug warfarin should functionally inhibiting the production of clotting factor (factor II, VII, IX, X Protein C). warfarin has slow onset of action so it requires bridging with intravenous heparin or subcutaneous low molecular heparin. The risk reduction of stroke by warfarin is 62%.[24] .The target INR should be between 2.0 and 3.0.[25]

NOVEL ORAL ANTICOAGULANTS THERAPY IN ATRIAL FIBRILLATION

It is used in patient with non valvular atrial fibrillation for the prevention of embolic stroke .NOACS includes direct thrombin inhibitor(Dabigatran) or factor Xa inhibitor(Rivaroxaban,Apixaban,Edoxaban,).The novel oral anticoagulants have rapid onset of action and more predictable dosing when compared to Warfarin.

DABIGATRAN

The dose of DABIGATRAN is 150mg twice daily for patients creatine clearance greater than30ml/min.patien twith creatine clearance less than 30 that effect Dabigatran can last greater than 4.Hemodialysis can rapidly reduce thedabigatran blood concentration and anticoagulant effect for few hours .

RIVAROXABAN

The dose of RIVAROXABAN is 20mg daily for patient with creatine clearance greater than 50Ml/min and 15 mg daily for patient with creatine clearance between 15 and 50mL/min.Rivaroxaban is non inferior to warfarin in patient with non valvular atrial fibrillation and history of stroke or CHADS2 score of 2

APIXABAN

The dose of APIXABAN is 5 mg twice daily for patient with non valvular atrial fibrillation and preserved renal function .APIXABAN 2.5 mg twice daily for patient with non valvular atrial fibrillation and two of the following characteristics

1) Age >80 years
2) Body weight <60kg
3) Serum creatinine >1.5 mg/dl

CHA2DS2-VASc score

CHADS2's shortcomings were addressed in Lip et al[18]. Added an additional risk factor (vascular disease [coronary, peripheral arterial, and venous], and female sex) as well as age categories (<60 years, 60 to 74 years, and ≥75 years) to create a new risk scorin +zg system: CHA2DS2-VASc. In their study, the investigators found that the CHA2DS2-VASc score outperformed the CHADS2 score in identifying stroke-at-risk NVAF patients. A study by Lip et al. Documents an incremental benefit of CHA2DS2-VASc. The study's modest discriminatory power and limited follow-up (1 year) were initially questioned as well as its incomplete surveillance (31% of patients had no follow-up data)[19]. Furthermore, several validation studies have shown that CHA2DS2-VASc accurately quantifies stroke risk better than CHADS2, especially in its ability to identify low-risk patients not in need of stroke prevention[20,21,22,23, 24, 25]. In 2012, the European Society of Cardiology updated its guidelines, as did the American College of Cardiology/American Heart Association in 2014, recommending the use ofCHA2DS2-VASc instead of the CHADS2 score to guide OAC in NVAF patients.

CONCLUSION

Worldwide, AF is associated with an increasing number of morbidities. Despite the advances in ischemic stroke prevention in these patients, current stroke risk prediction tools remain basic. There are various elements to the current risk factor–based stroke prediction schemes to optimize stroke prevention.The comprehensive study provides a holistic approach to risk assessment in this high-risk population.

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