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Embolic Cerebral Infarction in Atrial Fibrillation: A Simple Review Article

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This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Stroke is known worldwide as the leading cause of premature death and disability. Developing countries are most exposed to the dual burden of infectious and non-communicable diseases. Poor people are increasingly affected by stroke due to changing risk factors in the population and, most tragically, not being able to afford the high costs of stroke treatment. Despite significant improvements in primary prevention and acute care over the last few decades, stroke remains a catastrophic illness. Unchangeable risk factors for stroke are some established and changeable risk factors for age, gender, race, ethnicity, heredity, and ischemic stroke.

Keywords: Stroke; embolism; cerebral infarction; atrial fibrillation.

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1. INTRODUCTION

Cerebrovascular accident is the leading cause of disability and the second leading cause of death worldwide [1,2]. Much is driven by demographic changes and underpinned by the growing prevalence of major modifiable risk factors.

Low income increases the likelihood of stroke, both because of population exposure to variable risk factors and, more tragically, an inability to pay the high costs of stroke care. ' The majority of stroke survivors continue to live with a disability and require long-term care from family members, which leaves their families poorer [3,4].

There are Different underlying mechanisms can cause stroke. Identifying the underlying etiology of stroke is essential to guide evidence-based treatment for secondary prevention, such as anticoagulation for atrial fibrillation and revascularization in cervical carotid stenosis. There are 3 main mechanisms that can cause ischemic stroke; thrombosis, hypoperfusion, or embolism [5].

An embolic stroke occurs when blood clots travel from the source to block the more distant cerebral arteries, causing brain tissue perfusion to be stopped and ischemic. The source of embolism may be the heart, aorta, or artery, originating in the veins of the pelvis or lower extremities in the presence of a cardiac shunt causing paradoxical embolism, or of unknown origin. Therefore, careful evaluation and workup for an embolic source with trans thoracic or trans esophageal echocardiogram if needed in addition to Holter monitoring and vascular imaging is mandatory to guide appropriate evidence-based treatment and prevent stroke recurrence. Cardio embolic stroke accounts for 1430% of all cerebral infarctions. In most cases, recurrence of cardio embolism can be prevented by oral anticoagulants. Therefore, early confirmation of the diagnosis of cardiac embolic infarction is very important for patients with cerebral infarction in order to initiate appropriate anticoagulant therapy for secondary prevention. Cardiac embolic stroke can occur in at least 12 specific heart diseases, including atrial fibrillation, left ventricular thrombosis, heart tumors, valve vegetation, and paradoxical embolism. Most heart embolic strokes can be prevented [6].

33 million people have atrial fibrillation (AF), cardiac rhythm disorders. Over the past few decades, this arrhythmia has resulted from interactions between genetic predisposition, ectopic electrical activity, and abnormal atrial tissue substrate, which are then fed back to reshape and degrade the tissue matrix, thereby I learned that it spreads in itself. Although the importance of atrial fibrillation is partly due to its strong association with ischemic stroke, the mechanism behind atrial fibrillation stroke is not well understood [7]. Undocumented atrial fibrillation (AF) is believed to be the leading cause of stroke in patients with unexplained embolic stroke (ESUS), but its prevalence is poorly known1. The most serious consequence of atrial fibrillation (AF) is stroke. AF is one of the most important causes of stroke in the elderly. The risk of stroke is increased at least 6-fold in patients with atrial fibrillation. Stroke in AF patients is generally severe, has a high risk of death, and is prone to early and long-term recurrence. The cause of the heart of a stroke can be strongly suspected by medical history, laboratory tests, and imaging findings. Paroxysmal atrial fibrillation is a major cause of cerebral embolism and is often difficult to document. Risk factors for stroke in AF include previous embolism (including previous transient ischemic attack (TIA) or ischemic stroke), age 65 and older, structural heart disease, rheumatic or other serious heart Valve disease, artificial valve prosthesis, high blood pressure, heart. Heart failure, and severe left ventricular dysfunction diabetes and coronary artery disease. All AF patients with TIA or stroke have formal indications for long-term anticoagulant therapy [8].

Atrial fibrillation (AF) is one of the most common causes of ischemic stroke, but coronary artery embolism (CE) due to AF is rare, and 2.9% of all myocardial infarctions are due to CE. Is triggered. We present a case of an 87-year-old female patient who developed ischemic stroke and myocardial infarction at the same time and underwent intracranial and coronary thrombosis. Pathological examination showed that the thrombi were similar and both infarcts were considered cardiac embolism [9].

Novalvular (non-rheumatic) atrial fibrillation (NVAF) is the most common heart disease associated with suspected embolic stroke and accounts for about half of all cardiogenic embolic infarctions. Among the stroke patients at Tokyo Metropolitan Hospital for the Elderly, cerebral infarction was 75%, intracranial hemorrhage was 19%, and cerebral hemorrhage and infarction

were 6%. Twenty-eight percent of strokes were embolic infarcts of cardiac origin, of which 56% were caused by NVAF [10].

2. EPIDEMIOLOGY AND RISK FACTORS OF STROKE

Stroke is the third leading cause of death in the United States and the leading cause of chronic disability. Over the last 30-40 years, case series, case-control studies, and prospective cohort studies have included strokes such as age, gender, race, ethnicity, genetics, and some established modifiable risk factors. We have succeeded in identifying invariant risk factors. Ischemic stroke. Despite significant improvements in primary prevention and acute care in recent decades, stroke remains a catastrophic illness. At the turn of the century, age-standardized strokes in Europe ranged from 95 to 290 / 100,000 per year, with a monthly mortality rate of 13 to 35%. About 1.1 million people in Europe suffer from stroke each year, with ischemic stroke accounting for about 80% of cases.

Although stroke rates are declining globally, rates are increasing in young people, suggesting that strategies are needed to improve prevention. In addition, due to the aging population, the absolute number of strokes is predicted to increase significantly in the coming years: by 2025, 1.5 million Europeans will have a stroke each year. In addition to the life-threatening prognosis, stroke patients also had an increased risk of poor prognosis within the first year of the event, including re-hospitalization (33%), recurrent events (7-13%). dementia (7-23%) mild cognitive impairment (35 to 47%), depression (30 to 50%) and fatigue (35% to 92%), all of which contribute to poor quality health-related quality of life. In view of these reviews, there is an urgent need to develop acute care delivery, as well as resources for post-stroke treatment strategies [11].

Hypertension, atrial fibrillation, other heart disease, hyperlipidemia, diabetes, smoking, inactivity, carotid stenosis, and transient ischemic attack (TIA) are all potentially manageable conditions treatment leads to stroke. Research on other putative stroke risk factors, including anti phospholipid antibodies, high homocysteine, alcohol, inflammation, and infection, is ongoing. Controlled trials have shown that stroke risk can be reduced with blood pressure control, lipid-lowering drugs, surgery for carotid stenosis, warfarin for atrial fibrillation, and anti platelet agents. It is hoped that a better understanding of stroke risk factors will reduce the burden of stroke in the future [12].

Cardiovascular strokes seem to occur more frequently with age. Studies have estimated that they account for 14.6% of cases under the age of 65, but this rate rises to 36% for patients 85 years of age and older. [13] About 20% of strokes are considered cardiovascular. The risk of these strokes increases with age.

3. EPIDEMIOLOGY AND RISK FACTORS OF ATRIAL FIBRILLATION

Over the last two decades, atrial fibrillation (AF) has become one of the most important public health problems and a major cause of rising health care costs in Western countries. The prevalence of atrial fibrillation is increasing due to increased ability to treat chronic and non-heart disease and to suspect and diagnose atrial fibrillation. The prevalence of AF (2%) is now double that of the last decade. The prevalence of atrial fibrillation depends on age and gender. Atrial fibrillation is 0.12-0.16% of people under 49 years old, 3.7% -4.2% of people aged 60-70 years, and 10% -17% of people over 80 years old. In addition, it is more common in men, with a male-female ratio of 1.2: 1. The incidence of AF is between 0.21 and 0.41 per 1,000 people / year. Persistent atrial fibrillation occurs in about 50% of patients, and paroxysmal and persistent atrial fibrillation occurs in 25 Ch. Atrial fibrillation is often associated with heart disease and comorbidity. The most common comorbidities are coronary artery disease, valvular heart disease, and cardiomyopathy. The most common comorbidities are hypertension, diabetes, heart failure, chronic obstructive pulmonary disease, renal failure, stroke, and cognitive impairment. Paroxysmal atrial fibrillation occurs in younger patients and is less exposed to heart disease and comorbidities. In general, AF has a long history, suffers from frequent recurrences, and is associated with symptoms (two-thirds of patients). Patients with AF are 5 to 2 times more likely to have a stroke or death. The number of patients with atrial fibrillation in Europe is estimated to be 14 to 17 million in 2030, and the annual number of new cases of atrial fibrillation is estimated to be 120,000 to 215,000. Given that atrial fibrillation is associated with significant morbidity and mortality, this increased number of people with atrial fibrillation has significant public health implications [14]. Several established

cardiovascular risk factors are known to be independent predictors of AF development. Long-term follow-up data from the original Flamingam Heart Study Cohort over 20 years ago identified aging, hypertension, congestive heart failure, coronary artery disease, valvular heart disease, and diabetes mellitus as independent risk factors [15]. The latest meta-analysis of 12 cohort and case-control studies showed an independent relationship between these risk factors and the development of A [16]. These included additional risk factors such as male gender, left ventricular hypertrophy, obesity, and excessive alcohol consumption. Inter-venting risk factors before the onset of AF can be beneficial. For example, in obese patients without atrial fibrillation, obesity surgery, which reduces body weight by about 20%, has been shown to reduce the long-term risk of atrial fibrillation [17]. In another study of overweight and obese people with diabetes, intensive lifestyle interventions with reduced caloric intake and increased physical activity through counseling sessions (weekly for the first 6 months and less often thereafter) Could not reduce the risk of atrial fibrillation [18]. This may be due to moderate weight loss (6%) after nearly 10 years of follow-up compared to what was achieved with obesity surgery. More work is needed to investigate the role of various interventions in primary AF prevention. More recently, data on atherosclerosis risk (ARIC) in community studies indicate that suboptimal control of risk factors may account for 56.5% of cases of atrial fibrillation observed in the middle-aged cohort. Shown [19]. Due to the high prevalence of hypertension, the proportion of the population with atrial fibrillation was the highest among the risk factors studied, including increased body weight index (13%) and diabetes mellitus (3.1%) (22%). It's worth noting.) And smoking (9.8%) 46 In addition, in patients with metabolic syndrome, increased component numbers are associated with a gradual increase in the risk of developing atrial fibrillation [20].

4. EMBOLIC STROKE

Embolic stroke is one of the most common causes of mortality and morbidity all over the world. To avoid the high morbidity and mortality associated with this condition, it must be promptly diagnosed and treated both in the acute stage and for secondary prevention. This activity reviews the clinical evaluation, use of neuroimaging, and other tools of investigations to guide evidence-based treatment, and highlights the role of the inter-professional team in evaluating and treating patients with this condition [21].

Embolic strokes origenates when the blood clots migrate from the source to block blood viscle causing cessation of brain tissue perfusion and ischemia. Therefore, careful evaluation and workup for an embolic source with transthoracic or trans esophageal echocardiogram if needed.

Ischemic strokes of cardio embolic source are generally the most severe ischemic stroke subtype. Although cardiac emboli can be variable in size, the emboli arising from blood stasis within the left cardiac chambers are usually large in size and may cause large vessel occlusions such as Middle Cerebral Artery (MCA) and basilar artery occlusions, and hence severe strokes with higher rates of morbidity and mortality. They also carry a higher risk of stroke recurrence [22,23].

Emboli can happen due to different mechanisms including blood stasis in an abnormal, structurally enlarged left cardiac chamber such as left ventricular aneurysm with subsequent thrombus formation, material detachment from structurally abnormal calcific degenerative valves, or embolus passage from the venous to the arterial circulation (paradoxical embolism) because of the presence of right to left cardiac shunt such as Patent Foramen Ovale (PFO) [24].

5. POSSIBLE STROKE MECHANISMS IN ATRIAL FIBRILLATION

Atrial fibrillation and stroke have been linked in rigorous studies, suggesting a real link rather than a false finding. Epidemiological logic provides three explanations that 1) atrial fibrillation causes stroke, 2) atrial fibrillation causes stroke, and / or 3) atrial fibrillation is associated with other factors that cause stroke. Suggests [25].

AF is the most common persistent arrhythmia and is the cause of atrial embolic stroke. It is associated with decreased cardiac output, congestion, and elevated levels of thrombus-promoting fibrinogen, dimer, and von Willebrand factors, causing thrombus formation in the cardio-ear and embolic. Increases the risk of stroke. The prevalence of atrial fibrillation increases with age, peaking at 5% in this age group, especially in people over the age of 65.

Hypertensive heart disease continues to be the leading cause of atrial fibrillation in developed countries. Other related causes of atrial fibrillation are high alcohol intake, especially valvular heart disease in developing countries, and thyrotoxicosis [26].

The risk of embolism varies. The most common potential high-risk heart diseases that can cause embolic ischemic stroke are atrial fibrillation (AF), recent myocardial infarction, mechanical valve prosthesis, diastolic cardiomyopathy, and rheumatism, respectively. Includes mitral valve stenosis. However, other identifiable high-risk sources of embolism include infective endocarditis, nonbacterial endocarditis, and, rarely, the most common tumors of heart origin includes one left atrial myxoma.

6. ATRIAL FIBRILLATION AS A CAUSE OF STROKE

In assessing whether one factor causes another, or whether the two simply correlate, epidemiologist Bradford Hill has proposed the following generally accepted criteria: 1) Strength of association, 2) consistency, 3) specificity, 4) transient, 5) biological gradient, 6) validity, 7) consistency, 8) agreement with experimental results, and 9) analogy. [27] The association between atrial fibrillation and stroke meets some of these criteria. Patients with atrial fibrillation have a significantly increased risk of stroke-about 3-5 times higher after adjusting for risk factors [28]. Atrial fibrillation was consistently associated with stroke in various cohorts. [29] And the causal relationship is biologically plausible. Intuitively, unregulated muscle cell activity explains atrial contractility in atrial fibrillation, and the resulting congestion should increase the risk of thromboembolism, according to the Virchow triad.

However, some other Hill criteria do not support a direct relationship between atrial fibrillation and stroke. Many studies have found a biological gradient between AF exposure and stroke [30].

This is not consistent in all studies [31]. In addition, a single short episode of asymptomatic atrial fibrillation in elderly patients with vascular risk factors is associated with a two-fold increased risk of stroke [32], but clinically apparent atrial fibrillation. Young and healthy patients with stroke [33]. These contradictory data are insufficient to demonstrate a clear biological gradient between exposure to atrial fibrillation and stroke risk.

The relationship between atrial fibrillation and stroke is also below Hill's specificity criteria. If atrial fibrillation causes thromboembolism, it should be particularly associated with embolic stroke. There appears to be a particularly strong link between atrial fibrillation and embolic stroke [34].

However, 10% of patients with lacunar attacks have AF17, and atherosclerosis of the aorta is twice as common as patients without AF [35]. The association between atrial fibrillation and non-cardiac embolic stroke suggests that the fact that atrial fibrillation directly causes stroke does not fully explain the risk of stroke in atrial fibrillation.

Third, the association between atrial fibrillation and stroke does not fully meet Hill's transient criteria. A crossover analysis of current cases showed an increased risk of stroke shortly after the onset of AF [36], while two other current studies showed about 3 of AF and stroke patients. It was found that one-third showed AF only after the stroke, despite the stroke. Continuous cardiac rhythm monitoring for several months before stroke [37,38]. These results suggest that while the arrhythmia itself can cause thromboembolism, other factors are also involved in the strong association between atrial fibrillation and stroke.

Fourth, the causal interpretation of the association between atrial fibrillation and stroke does not fit well with the experimental evidence available. If arrhythmia is the only cause of thromboembolism, maintaining a normal rhythm should eliminate the risk of stroke. However, in a meta-analysis of eight randomized clinical trials, rhythm control strategies did not affect the risk of stroke (odds ratio 0.99; 95% confidence interval 0.761.30) [39]. It is unlikely that this was simply because the sinus rhythm was not reliably maintained (odds ratio 4.39; 95% confidence interval 2,846.78), as the rhythm control strategy has shown great success in maintaining normal sinus rhythm. In addition, the structural remodeling observed in the experimental AF model occurs after at least one week of continuous rapid stimulation [37]. Human stroke [32]. Therefore, there is a lack of solid experimental evidence that atrial fibrillation is a necessary step in thrombus formation.

7. ATRIAL FIBRILLATION-ASSOCIATED FACTORS AS CAUSES OF STROKE

In addition to causing a stroke, atrial fibrillation may be associated with other factors that cause a stroke. Age, male gender, hypertension, diabetes mellitus, valvular heart disease, heart failure, coronary artery disease, chronic renal disease, inflammatory disease, sleep apnea, and tobacco use are risk factors for atrial fibrillation and stroke. AF-stroke-related confusion is indicated by its weakening, as more shared risk factors are considered [40]. Nevertheless, AF is independently associated with stroke, even after a thorough adjustment of common risk factors. AF is also most strongly associated with stroke, which has a neurological imaging pattern that resembles that of cardiac embolism, as well as general stroke [41].

AF stroke is caused by the left atrium, but other atrial factors in addition to AF can cause thromboembolism. Is AF not the only cause of atrial fibrillation, but may it be a marker of other atrial abnormalities that are themselves the root cause of stroke? AF often coexists with atrial abnormalities such as endothelial dysfunction, fibrosis, muscle cell dysfunction, ventricular dilatation, and mechanical dysfunction of the left atrial appendage [42].

Can these associations be communicated via AF? Abnormalities in the left atrium cause atrial fibrillation, which is paroxysmal and difficult to detect, and may be due to the atrial atrial matrix that causes stroke. However, clinically apparent indications for atrial fibrillation do not change the association between left atrial abnormalities and stroke [42]. Unexpected discoveries when atrial fibrillation mediates those relationships. Another interpretation of these associations is that asymptomatic atrial fibrillation causes atrial matrix, which causes stroke. In this interpretation, AF is needed again for downstream changes to occur, leading to thrombus formation. However, structural remodeling appears to require weeks of atrial fibrillation 23, as well as 6 minutes sufficient to show an increased risk of stroke [43].

Such discrepancies undermine the notion of atrial fibrillation as the only cause of stroke-related atrial abnormalities. These associations and their lack of attenuation after adaptation to atrial fibrillation suggest that atrial disease causes thrombus formation by other pathways than atrial fibrillation. As a rule, homozygous mutations in the natriuretic peptide precursor A gene provide proof. Even in the absence of atrial fibrillation, the disease causes atrial dilatation, progressive loss of atrial activity, and possible atrial arrest and thromboembolism [44].

8. PATHOPHYSIOLOGY

As with all other thrombi, the underlying pathophysiology belongs to the Virchow triad. Blood stasis, as occurs with ventricular dyskinesia or aneurysms, predisposes to thrombus formation. Likewise, atrial incompatibility in atrial fibrillation leads to an increased predisposition to clot formation, especially in the left atrial appendage. These thrombus may be inactive and then undergo tissue or embolism until a systemic stroke is a potential consequence. With atrial fibrillation, this risk is greatest when converting patients to sinus rhythm [45].

9. HISTORY AND PHYSICAL FINDINGS

The classic clinical situation is that of a neurological deficit of sudden onset and maximal intensity within minutes and then gradual improvement. As with all strokes, clinical features depend on the extent and location of neurovascular involvement. However, impaired consciousness is generally a more favorable factor for cardiovascular disease than thrombotic stroke [46].

In addition, obtaining a history of a stroke-inducing Valsalvalike maneuver also supports cardiovascular etiology [47]. Cardiovascular strokes cause more episodes of distal ischemia.

10. CLASSIC FEATURES OF CARDIO EMBOLIC STROKE INCLUDE THE FOLLOWING

Abrupt decline in mental status, change in the level of consciousness, presence of neurological deficits,

11. CARDIAC FINDINGS MAY INCLUDE

atrial fibrillation**,** presence of a cardiac murmur**,** signs of congestive heart failure**,** recent myocardial infarction**,** recent diagnosis of infective endocarditis [45].

12. TREATMENT / MANAGEMENT

The fundamental principle of cardiovascular stroke management involves the use of

anticoagulants for secondary prevention. Current guidelines suggest an arbitrary delay of anticoagulation for two weeks after the event, based on extrapolation from trials based on the use of heparin [47]. The foramen ovale patent closure is also increasingly recognized as an essential tool in the prevention of stroke recurrence. It shows a clear superiority over antiplatelet therapy and is not inferior to anticoagulants. [46] Any patient who has had a cardiovascular stroke younger than 60 years of age should be evaluated for the existence of foramen ovale. For patients with left ventricular thrombosis and myocardial infarction, we generally recommend anticoagulation for at least three months because of the risk of embolism.

13. DIFFERENTIAL DIAGNOSIS

Atherothrombotic strokes**,** hemorrhagic stroke**,** transient ischemic attack**,** stroke mimic syndromes [45].

14. PROGNOSIS

If not well managed, cardio embolic strokes have a higher probability than atherothrombotic strokes to present both early and late recurrences [45].

15. COMPLICATIONS

Haemorrhagic transformations, both spontaneous and post-anticoagulation therapy, are potentially grave consequences of this condition. In addition, long-term disability, bed-rest related complications such as pressure sores, may all occur but vary depending on the severity and extent of neuro deficit. [45]

16. CONCLUSION

The most powerful model of AF and thromboembolism is probably incomplete. A direct association between AF and stroke does not conclusively demonstrate temporality or biological gradients. A model in which thromboembolism is caused by both systemic abnormalities and by AF and atrial tissue stroma is better suited to the available data. Such a model has several important implications for stroke prevention plans to identify and treat patients at risk for thromboembolism. More research is needed to improve stroke care and reduce the burden of this disabling disease.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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