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# ASSOCIATION OF ISCHEMIC STROKE IN CASES WITH ATRIAL FIBRILLATION. REVIEW

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**Abstract:**

*This review describes present evidence relating to the association in between known stroke danger elements, underlying problems and consequences and the progression and development of AF. We searched PubMed, Medline and web of science databases for recent for studies concerning Association of ischemic stroke in cases with atrial fibrillation published up to September, 2019. Medical Mesh terms were used in our search as following; "ischemic heart disease" "atrial fibrillation". On its own, AF is generally not a lethal condition, however it can cause serious clinical complications. AF can add to coronary infarction and various other significant heart disease. Perhaps one of the most significant issue of AF is stroke. A stroke occurs when the flow of blood to part of the brain is substantially reduced or blocked. Usually, this causes the fatality of mind cells, which can bring about irreversible damage and even death. One of the most common root cause of stroke is an embolism. AFib puts patients at a raised threat for stroke because blood may not be appropriately pumped out of the heart, which might cause it to swimming pool and create an embolism. This embolism can then travel to the brain and obstruct the flow of blood to part of the brain which can lead to a stroke.*

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## INTRODUCTION:

Atrial fibrillation (AF) is one of the most prevalent scientifically substantial arrhythmias, with a total frequency of around 1 % in the general populace [1]. One of the most clinically crucial problem from AF hinges on the risk for cardiac thrombus development and systemic embolism. It is an essential risk factor for ischemic stroke given that it associates with a 5-fold greater danger of stroke compared with the basic populace [2]. AF accounts for 15% of all ischemic strokes and 33% of strokes in the elderly [2]. Stroke occasions arising from AF are much more serious, related to high dangers of morbidity and mortality than those of non-AF etiologies [3]. Therefore, stroke prevention is a vital part of management for patients with AF. Independent threat factors for stroke in patients with AF consist of age ≥ 65 years, female sex, congestive heart failure, prior stroke or short-term ischemic attack, high blood pressure, diabetes mellitus and vascular disease. Although AF can be entirely asymptomatic, regarding two-thirds of patients experience a minimum of intermittent signs and symptoms, which can be disabling and markedly impair health-related quality of life [3].

In patients with non-valvular AF, anticoagulation therapy (OAC) with warfarin reduces stroke by 64% compared to no antithrombotic agents. OAC is considerably much more reliable in decreasing stroke threat in AF than antiplatelet agents [2]. American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) standards advise OAC treatment for patients with AF who are at modest or high danger of stroke (i.e., have a CHA2DS2-VASc score of ≥ 1) [4]. Nevertheless, OAC treatment is underused in AF: only 50% (USA), 67% (Europe), 75% (Japan), and 2.5% (China) of AF patients with CHADS2 or CHA2DS2-VASc scores of ≥ 2 get OAC treatment. This reflects its tight healing array and/or its association with issues [5]. Identifying AF patients in danger for stroke has crucial healing and prognostic effects. Thromboprophylaxis with anticoagulants and anti-platelet representatives can decrease the danger of stroke in properly decided on patients with AF but carries a raised danger of bleeding and may call for way of living modifications such as dietary adjustments, and regular surveillance if warfarin is utilized [4].

Atrial fibrillation (AF) is one of the most typical scientific arrhythmias and is related to increased morbidity and death. There is expanding evidence that many cardiovascular diseases and danger factors relate with the event of AF and that alone AF is rare. This review describes present evidence relating to the association in between known stroke danger elements, underlying problems and consequences and the progression and development of AF.

## METHODOLOGY:

We searched PubMed, Medline and web of science databases for recent for studies concerning Association of ischemic stroke in cases with atrial fibrillation published up to September, 2019. Medical Mesh terms were used in our search as following; “ischemic heart disease” “atrial fibrillation”. We applied restriction to our search for only English language articles with human subjects.

## DISCUSSION:

### Stroke and systemic thromboembolism in patients with atrial fibrillation:

Atrial fibrillation is a major threat aspect for stroke, boosting the risk of ischemic stroke around fivefold and accounting for about 45% of all embolic strokes in the United States (or roughly 100,000 strokes each year) [6], [7]. Not just is age an independent and powerful predictor of stroke, however other risk elements for stroke in patients with atrial fibrillation additionally enhance with age, consisting of diabetic issues, congestive heart failure, high blood pressure, left ventricular disorder, and vascular disease [3]. Of note, in patients less than 60 years of ages without recognized threat aspects the danger of stroke is extremely low, suggesting that it is not simply the arrhythmia but the "company it maintains" that is responsible for systemic thromboembolism [7].

The pathophysiological devices underlying stroke in patients with atrial fibrillation are multifactorial. It is well approved that stasis in the left atrium and left atrial appendage bring about thrombus development is likely a primary root cause of systemic thromboembolism (Figure 1). Comorbidities in the senior, including diastolic disorder, diabetic issues, high blood pressure, and aortic atherosclerosis, are associated with an increase in left atrial volume and perhaps an increased predisposition for left atrial stasis or thrombus formation [8], [9]. Left atrial quantity overload and dilatation may by itself result in a prothrombotic state using stretch-induced systems and to endothelial disorder. On top of that, the onset of atrial fibrillation might result in activation of hemostatic elements [10]. A number of research studies have actually recommended that atrial fibrillation generates a hypercoagulable state, and this has actually been kept in mind in both paroxysmal and persistent atrial fibrillation [10], [11]. Moreover, enhanced plasma concentrations of markers of platelet activation (beta thromboglobulin, platelet variable IV,

and soluble P-selection), enhanced plasma markers of thrombogenesis (thrombin- antithromben complexes), and evidence of endothelial dysfunction and damage (von Willebrand factor) have actually been shown as independent correlates of thromboembolism [12].

In an undefined percentage of patients, atrial fibrillation may be a surrogate or manifestation of intensified aortic tightness, diffuse atherosclerosis, and vascular disease. In these medical parts the suppositional root cause of stroke and systemic blood clot might be aortic atherosclerosis, existing side-by-side cerebrovascular illnesses, and perhaps an inflammatory "milieu". The demographic tide, resulting in aged cultures in both the high and low-middle earnings nations, is inexorable and emphasizes the growing value of stroke avoidance in atrial fibrillation.

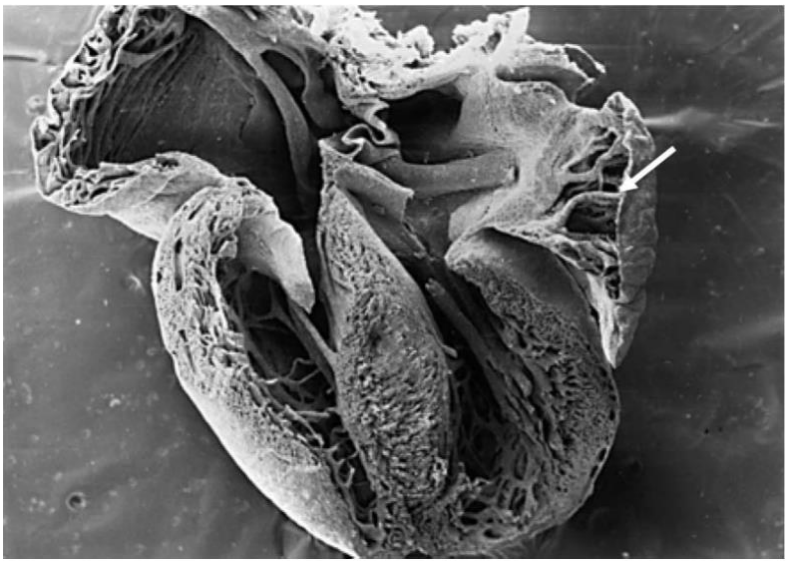

**Figure 1.** A coronal section of the heart showing a trabeculated left atrial appendage (arrow) in contrast to the left atrial body with smooth walls [13].

**Atrial fibrillation as a risk factor for stroke:**

A variety of research studies have proven that patients with AF have elevated stroke danger. In a meta-analysis of 5 randomized regulated trials performed by the AF Investigators, the annual stroke rate was 4.5% in non-anticoagulated patients: nevertheless, the danger was not uniform and certain danger aspects carried a higher-relative danger compared to others [14]. Moreover, every one of the danger elements were enhanced with age delineating why stroke danger is so age-dependent. Interestingly, patients without any risk factors have a much-reduced stroke risk, estimated at<1% annually [14]. Therefore, although AF can cause left atrial appendage (LAA) thrombus and following stroke, the majority of the enhanced risk is most likely second to connected comorbidities and not purely a manifestation of the arrhythmia itself however 'the company it maintains'. This was shown in case - control research of 110 patients with AF found to have a LAA thrombus on transesophageal echocardiography. While patients with LAA thrombus had a greater CHADS2 score compared to controls (2.8 vs. 1.6), there was significant overlap suggesting that other essential elements existed [15]. Nevertheless, recognition of linked threat factors develop the basis for risk stratification scores focused on establishing which patients have adequate stroke risk to have more benefit than threat from anticoagulation.

There are two major postulates pertaining to AF and following stroke. The very first focuses on lessened LAA flow velocities throughout AF with secondary thrombus development and thromboembolism. In this situation, AF comes before stroke. Thus, it complies with that removal of AF or exemption of the LAA may avoid stroke. Nevertheless, the reality that the real stroke occasion does not always happen throughout (or immediately after) an AF episode in those with paroxysmal AF recommends that mechanisms go to play [16]. The 2nd propose is that AF is mostly a marker of vascular disorder concern which leads to boosted stroke threat. The linked atherosclerotic risk variables cause vascular swelling with linked diastolic and endothelial disorder. This causes an atrial myopathy with left atrial (LA) augmentation and secondary fibrosis leading to electrical heterogeneity from which AF might emerge. Coincident with this,

endothelial dysfunction from the underlying vascular swelling may cause a hypercoagulable state and enhanced thrombus formation. This is suggested by raised levels of coagulation factors such as d-dimer, fibrinogen, von Willebrand factor, and platelet factor-4 in individuals with AF. For example, in a research study of 591 patients, d-dimer levels were highest possible in those with AF, lowest in controls and intermediate in those treated with warfarin [17]. Moreover, modulation of inflammatory markers has been related to a reduction in cardiovascular occasions [18]. Associated vascular disease of the aorta might likewise add to stroke risk pertaining to aeroembolism from aortic plaque.

Patients with elevated CHADS2 and CHA2DS2-VASc score seem to have an elevated stroke threat also in the absence of AF. In an analysis of 916 non-anticoagulated patients with steady coronary cardiovascular disease and without AF, patients with a score of ≥ 5 had equivalent stroke rates to AF patients with moderate CHADS2 ratings [19]. While this may be additional to unnoticed ('silent') AF, it highlights that AF may be an indication of an overriding vascular disease as described over. Additional proof originates from the CHADS2 and CHA2DS2-VASc ratings being predictive of heart attack [20]. In a multicenter empirical research study of 3183 patients with acute coronary syndrome, CHA2DS2-VASc ≥ related to a greater danger of damaging events compared to CHA2DS2-VASc < 2 [21]. In one more research study of 565 patients post-AF ablation, those with CHA2DS2-VASc 0 - 1 had a 1.1% event rate compared to 7.1% in those with a score ≥ 2 [22].

**Table 1.** CHADS2 scoring system [19],[20] .

| | **Condition** | **Points** |
|---|---|---|
| **C** | Congestive heart failure | 1 |
| **H** | Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication) | 1 |
| **A** | Age ≥75 years | 1 |
| **D** | Diabetes mellitus | 1 |
| **$S_2$** | Prior Stroke or TIA or Thromboembolism | 2 |

The yearly threat of stroke increases as the threat rating is higher, offering advice for making use of oral anticoagulants (OACs) in patients with AF (Table 2) [23].

**Table 2.** $CHADS_2$ score and annual adjusted stroke rate [23].

| **$CHADS_2$ Score** | **Adjusted Stroke Rate (%/year) (95% CI)** |
|---|---|
| **0** | 1.9 (1.2–3.0) |
| **1** | 2.8 (2.0–3.8) |
| **2** | 4.0 (3.1–5.1) |
| **3** | 5.9 (4.6–7.3) |
| **4** | 8.5 (6.3–11.1) |
| **5** | 12.5 (8.2–17.5) |
| **6** | 18.2 (10.5–27.4) |

Abbreviation (CI)-confidence interval used in statistics

The annual modified stroke danger according to the CHA2DS2-VASc rating is summed up in Table 3. This danger stratification plan is a lot more complicated; nonetheless, a recent research suggests that the CHA2DS2-VASc -statistic is very comparable to that for CHADS2, however the CHA2DS2-VASc boosts danger forecast amongst patients at lower risk of stroke, i.e., with CHADS2 rating ≤ 1 [24] .

**Table 3**. Annual adjusted stroke rate according to the $CHA_2DS_2$-VASc score [24].

| $CHA_2DS_2$-VASc Score | Adjusted Stroke Rate (%/year) |
|---|---|
| 0 | 0 |
| 1 | 1.3% |
| 2 | 2.2% |
| 3 | 3.2% |
| 4 | 4.0% |
| 5 | 6.7% |
| 6 | 9.8% |
| 7 | 9.6% |
| 8 | 6.7% |
| 9 | 15.2% |

**Consequences of stroke:**

According to the American Heart Association, stroke accounts for more than 1 in every 15 deaths in the United States and rates number 3 amongst all causes of death, superseded just by cardiac death and deaths as a result of cancer. Strokes connected with AF are usually much more extreme and confer an excess risk of subsequent morbidity, mortality, and bad functional result independent of the underlying heart problem. Danger of frequent stroke is high, particularly within the very first year, as a result of hemostatic problems complying with the index occasion. In the Cardiovascular Health Study of a subset of 546 patients with initial ischemic stroke throughout 1989 - 2001, rates for reoccurring stroke were 105.4 per 1,000 within the very first year and 52 per 1,000 thereafter [25]. Cardioembolic strokes related to the highest possible death (185.4/ 1,000) and reoccurrence rates (86.6/ 1,000), whereas lacunar strokes had the lowest death (119.3/ 1,000) and reoccurrence rates (43.0/ 1,000).

The record from the Framingham Study has revealed that stroke associated with AF was nearly two times as most likely to be fatal [26]. Recurrence was much more constant, and useful shortages were more probable to be serious amongst survivors. Almost three-quarters of stroke sufferers with AF were severely dependent in activities of everyday living compared to about one-third of their equivalents with sinus rhythm. The presence of AF almost increased the regularity of being bedridden complying with first ischemic stroke (41.2% in patients with AF compared to 23.7% in patients without AF) and this raised extent was independent of advanced age and other stroke threat variables [27].

AF boosted the possibility of continuing to be handicapped by around 1.5-fold [28]. In the European Community Project involving 7 European countries (United Kingdom, France, Germany, Hungary, Italy, Portugal, and Spain), AF was a considerable forecaster of complication (probabilities proportion 1.41), coma (chances ratio 1.74), paralysis (odds proportion 1.66), aphasia (probabilities proportion 1.51), dysphagia (odds proportion 1.88), and urinary incontinence (probabilities ratio 1.64) in 4,534 patients with first stroke [28]. Overall former flow infarct happened regularly in patients with AF (33.8%) compared with 25.1% in patients with sinus rhythm; odds proportion 1.59.

The association of AF with inadequate stroke result might be due a considerable reduction in local cerebral blood circulation triggered by consistent AF. On the other hand, collateral circulation in the mind might be less created in patients with an unexpected disruption of blood circulation brought on by an embolus than in those who experience a stroke as the result of an underlying chronic arterial atherosclerotic disease. These 2 variables might better compromise cerebral circulation, increase the infarction dimension, and delay function healing. AF patients with large strokes are extra susceptible to additional hemorrhagic change.

**Bleeding Risk:**

Bleeding is the solitary essential element that restricts prevalent indication for OAC therapy. Although recent reports suggest low rates of OAC relevant intracerebral hemorrhage (between 0.1 and 0.6%), major bleeding can take place in up to 4% annually making bleeding danger evaluation critical prior to initiating OAC therapy [29]. To avoid blood loss,

careful dosage titration and ample high blood pressure control are the cornerstone for the prevention of bleeding.

Stroke danger elements are likewise related to and a higher threat for hemorrhage, i.e., greater bleeding risk relates to enhancing CHADS2 rating. A variety of scoring systems have been developed to determine clinical threat factors connected with an incremental danger for hemorrhage. The HAS-BLED score offers helpful anticipating capability for bleeding. One point is assigned to every of the adhering to markers: hypertension, irregular renal/liver function, stroke, hemorrhaging background or tendency, labile INR, aged and concomitant use of drugs/alcohol (Table 4).

**Table 4.**HAS-BLED score.

| Letter | Characteristics | Points |
|---|---|---|
| H | Hypertension | 1 |
| A | Abnormal renal and liver function (1 point each) | 1 or 2 |
| S | Stroke | 1 |
| B | Bleeding | 1 |
| L | Labile INR | 1 |
| E | Elderly (≥65 years old) | 1 |
| D | Drugs or alcohol 1 point each) | 1 or 2 |

High blood pressure is specified as systolic high blood pressure > 160 mmHg, unusual kidney function is defined as the presence of chronic dialysis or kidney transplant or serum creatinine ≥ 200 µmol/ L and unusual liver function is specified as chronic hepatic illness (e.g., cirrhosis) or biochemical proof of significant hepatic derangement (bilirubin > 2 × upper limit of typical in association with AST/ALT > 3 × upper limit typical). Drugs users refer to concomitant use of medications such as antiplatelet representatives, non-steroidal anti-inflammatory medicines, etc. A rating ≥ 3 suggests "high threat", needing care and routine testimonial complying with the initiation of antithrombotic treatment [29].

## CONCLUSION:

On its own, AF is generally not a lethal condition, however it can cause serious clinical complications. AF can add to coronary infarction and various other significant heart disease. Perhaps one of the most significant issue of AF is stroke. A stroke occurs when the flow of blood to part of the brain is substantially reduced or blocked. Usually, this causes the fatality of mind cells, which can bring about irreversible damage and even death. One of the most common root cause of stroke is an embolism. AFib puts patients at a raised threat for stroke because blood may not be appropriately pumped out of the heart, which might cause it to swimming pool and create an embolism. This embolism can then travel to the brain and obstruct the flow of blood to part of the brain which can lead to a stroke.

When it concerns dealing with AF there are 2 primary therapy objectives: 1) Reducing the threat of blood clots and stroke and 2) Managing unusual heart rate or rhythm. Stroke avoidance in patients with AF is the foundation of management for this usual arrhythmia. Present efforts will still focus on the following: (i) Improving the precision of recognizing patients at reduced risk for thromboembolism; (ii) Exploring more effective and safer methods to prevent stroke, based on a far better understanding of the device of stroke events in AF; and (iii) Increasing OAC usage and patients' adherence, specifically in several regions of the world, such as Asia, Africa and Middle East. Stroke avoidance is simply one component of an integrated handling technique to AF, and interest to signs and symptoms and cardiovascular or comorbidity management ought to likewise be emphasized.

Uncommon heart beats, another problem in treating AF, may be treated with drugs that impact the heart's rate (pacing) or rhythm (coordination), or a surgical procedure such as electric cardioversion, catheter ablation or medical intervention.

## REFERENCES:

1. Kannel WB, Wolf PA, Benjamin EJ, Levy D. Prevalence, incidence, prognosis, and predisposing conditions for atrial fibrillation: population-based estimates. Am J Cardiol 1998; 82:2N–9N.
2. Heart disease - Atrial fibrillation - Heart and Stroke Foundation of Canada

http://www.heartandstroke.ca/heart/conditions/atrial-fibrillation.

3. Henninger N, Goddeau RP, Jr., Karmarkar A, Helenius J, McManus DD. Atrial Fibrillation Is Associated With a Worse 90-Day Outcome Than Other Cardioembolic Stroke Subtypes. Stroke. 2016;47:1486-92.
4. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC Jr, et al. AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol. 2014;64:e1–76.
5. Rahman F, Kwan GF, Benjamin EJ. Global epidemiology of atrial fibrillation. Nat Rev Cardiol 2014; 11: 639–654.
6. Estes NA, Halperin JL, Calkins H, Ezekowitz MD, Gitman P, Go AS, et al, American College of Cardiology; American Heart Association Task Force on Performance Measures; Physician Consortium for Performance Improvement..
7. ACC/AHA/Physician Consortium 2008 Clinical Performance Measures for Adults with Nonvalvular Atrial Fibrillation or Atrial Flutter: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures and the Physician Consortium for Performance Improvement (Writing Committee to Develop Clinical Performance Measures for Atrial Fibrillation) Developed in Collaboration with the Heart Rhythm Society.J Am Coll Cardiol. , 51 (2008), pp. 865-884.
8. Wolf PA, Abbott RD, Kannel WB.Atrial fibrillation as an independent risk factor for stroke: the Framingham Study.Stroke. , 22 (1991), pp. 983-988.
9. Tsang TS, Barnes ME, Gersh BJ, Takemoto Y, Rosales AG, Bailey KR, et al..Prediction of risk for first age-related cardiovascular events in an elderly population: the incremental value of echocardiography.J Am Coll Cardiol. , 42 (2003), pp. 1199-1205.
10. Tsang TS, Gersh BJ, Appleton CP, Tajik AJ, Barnes ME, Bailey KR, et al.Left ventricular diastolic dysfunction as a predictor of the first diagnosed nonvalvular atrial fibrillation in 840 elderly men and women.J Am Coll Cardiol. , 40 (2002), pp. 1636-1644
11. Watson T, Shantsila E, Lip GY.Mechanisms of thrombogenesis in atrial fibrillation: Virchow's triad revisited.Lancet. , 373 (2009), pp. 155-166.http://dx.doi.org/10.1016/S0140-6736(09)60040-4
12. Heppell RM, Berkin KE, McLenachan JM, Davies JA.Haemostatic and haemodynamic abnormalities associated with left atrial thrombosis in non-rheumatic atrial fibrillation.Heart. , 77 (1997), pp. 407-411.
13. Al-Saady NM, Obel OA, Camm AJ. Left atrial appendage: structure, function, and role in thromboembolism. Heart. 1999;82:547–54.
14. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation. Analysis of pooled data from five randomized controlled trials. Arch Intern Med 1994;154:1449–1457.
15. Wysokinski WE, Ammash N, Sobande F, Kalsi H, Hodge D, McBane RD. Predicting left atrial thrombi in atrial fibrillation. Am Heart J 2010;159:665–671.
16. Brambatti M, Connolly SJ, Gold MR, Morillo CA, Capucci A, Muto C, Lau CP, Van Gelder IC, Hohnloser SH, Carlson M, Fain E, Nakamya J, Mairesse GH, Halytska M, Deng WQ, Israel CW, Healey JS; ASSERT Investigators. Temporal relationship between subclinical atrial fibrillation and embolic events. Circulation 2014;129:2094–2099.
17. Ohara K, Inoue H, Nozawa T, Hirai T, Iwasa A, Okumura K, Lee JD, Shimizu A, Hayano M, Yano K. Accumulation of risk factors enhances the prothrombotic state in atrial fibrillation. Int J Cardiol 2008;126:316–321.
18. Ridker PM, Everett BM, Thuren T, MacFadyen JG, Chang WH, Ballantyne C, Fonseca F, Nicolau J, Koenig W, Anker SD, Kastelein JJP, Cornel JH, Pais P, Pella D, Genest J, Cifkova R, Lorenzatti A, Forster T, Kobalava Z, Vida-Simiti L, Flather M, Shimokawa H, Ogawa H, Dellborg M, Rossi PRF, Troquay RPT, Libby P, Glynn RJ; CANTOS Trial Group. Antiinflammatory therapy with canakinumab for atherosclerotic disease. N Engl J Med 2017;377:1119–1131.
19. Welles CC, Whooley MA, Na B, Ganz P, Schiller NB, Turakhia MP. The CHADS2 score predicts ischemic stroke in the absence of atrial fibrillation among subjects with coronary heart disease: data from the Heart and Soul Study. Am Heart J 2011;162:555–561.
20. Pang H, Han B, Fu Q, Zong Z. Predictive value of CHADS2 and CHA2DS2-VASc scores for acute myocardial infarction in patients with atrial fibrillation. Sci Rep 2017;7:4730.
21. Chua SK, Lo HM, Chiu CZ, Shyu KG. Use of CHADS(2) and CHA(2)DS(2)- VASc scores to predict subsequent myocardial infarction, stroke,

and death in patients with acute coronary syndrome: data from Taiwan acute coronary syndrome full spectrum registry. PLoS One 2014;9:e111167.

22. Chao TF, Lin YJ, Tsao HM, Tsai CF, Lin WS, Chang SL, Lo LW, Hu YF, Tuan TC, Suenari K, Li CH, Hartono B, Chang HY, Ambrose K, Wu TJ, Chen SA. CHADS(2) and CHA(2)DS(2)-VASc scores in the prediction of clinical outcomes in patients with atrial fibrillation after catheter ablation. J Am Coll Cardiol 2011;58:2380–2385.
23. Gage B.F., Waterman A.D., Shannon W., Boechler M., Rich M.W., Radford M.J. Validation of clinical classification schemes for predicting stroke: Results from the National Registry of Atrial Fibrillation. JAMA. 2001;285:2864–2870.
24. Lip G.Y., Nieuwlaat R., Pisters R., Lane D.A., Crijns H.J. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: The Euro heart survey on atrial fibrillation. Chest. 2010;137:263–272.
25. Kaplan RC, Tirschwell DL, Longstreth WT Jr, Manolio TA, Heckbert SR, Lefkowitz D, et al. Vascular events, mortality, and preventive therapy following ischemic stroke in the elderly. Neurology. 2005;65:835–42.
26. Lin HJ, Wolf PA, Kelly-Hayes M, Beiser AS, Kase CS, Benjamin EJ, et al. Stroke severity in atrial fibrillation. The Framingham Study. Stroke. 1996;27:1760–4.
27. Gage BF, van Walraven C, Pearce L, Hart RG, Koudstaal PJ, Boode BS, et al. Selecting patients with atrial fibrillation for anticoagulation: stroke risk stratification in patients taking aspirin. Circulation. 2004;110:2287–92.
28. Dulli DA, Stanko H, Levine RL. Atrial fibrillation is associated with severe acute ischemic stroke. Neuroepidemiology. 2003;22:118–23.
29. Lip G.Y., Frison L., Halperin J.L., Lane D.A. Comparative validation of a novel risk score for predicting bleeding risk in anticoagulated patients with atrial fibrillation: The HAS-BLED (Hypertension, Abnormal Renal/Liver Function, Stroke, Bleeding History or Predisposition, Labile INR, Elderly, Drugs/Alcohol Concomitantly) score. J. Am. Coll. Cardiol. 2011;57:173–180.