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Atrial Cardiopathy and Cryptogenic Stroke: A Cross-sectional Pilot Study

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Abstract

Background—There is increasing evidence that left atrial dysfunction or cardiopathy is associated with ischemic stroke risk independently of atrial fibrillation. We aimed to determine the prevalence of atrial cardiopathy biomarkers in patients with cryptogenic stroke.

Methods—We included consecutive patients with ischemic stroke enrolled in the New York Columbia Collaborative Specialized Program of Translational Research in Acute Stroke registry between December 1st, 2008 and April 30th, 2012. Medical records were reviewed and patients with a diagnosis of cryptogenic stroke were identified. Atrial cardiopathy was defined as at least one of the following: serum N- terminal pro brain natriuretic peptide (NT pro-BNP)>250 pg/mL, P-wave terminal force velocity in lead V1 on electrocardiogram (PTFV1) >5000 μV*ms, or severe left atrial enlargement (LAE) on echocardiogram. We compared clinical, echocardiographic, and radiological characteristics between patients with and without atrial cardiopathy.

Results—Among 40 patients with cryptogenic stroke, 63% had at least one of the biomarkers of atrial cardiopathy; 49% had elevated NT-proBNP, 20% had evidence of increased PTFV1 on

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ECG, and 5% had severe LAE. Patients with atrial cardiopathy were more likely to be older (76 vs. 62 years; p=0.012); have hypertension (96% vs. 33%, p<0.001), hyperlipidemia (60% vs. 27%, p=0.05), or coronary heart disease (28% vs. 0%, p=0.033), and less likely to have a patent foramen ovale(4% vs. 40%, p=0.007).

Conclusion—There is a high prevalence of biomarkers indicative of atrial cardiopathy in patients with cryptogenic stroke. Clinical trials are needed to determine whether these patients may benefit from anticoagulation to prevent stroke.

Keywords

Stroke; Atrial Cardiopathy; Cryptogenic; Anticoagulant; Prevention

Background

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and is a global healthcare problem, particularly due to its rising worldwide incidence and prevalence.[1, 2] To date, AF is the only marker of left atrial dysfunction for which anticoagulation has been shown to provide a greater reduction of stroke risk than antiplatelet therapy.[3] Other less established markers of left atrial dysfunction or “atrial cardiopathy” that have been associated with ischemic stroke risk in prospective studies include paroxysmal supraventricular tachycardia, elevated serum N-terminal pro-brain natriuretic peptide (NT-proBNP), increased P-wave terminal force velocity in lead V1 (PTFV1) on electrocardiogram (ECG), and moderate to severe left atrial enlargement (LAE) on echocardiogram. [4, 5] Several of these biomarkers are also associated with detection of AF in prospective studies. [6, 7]

Left atrial size, PTFV1, and NT pro-BNP all have been linked to detection of paroxysmal AF in patients with cryptogenic stroke [5–8–10], but the relationship and concordance between these three biomarkers is unclear. Understanding the relationship between these biomarkers and their association with cardiovascular risk factors in patients with cryptogenic stroke may shed light on the relationship between atrial cardiopathy and stroke, thereby helping to improve stroke prevention strategies. Our aim was to determine the prevalence of biomarkers of atrial cardiopathy in patients with cryptogenic stroke and their association with cardiovascular risk factors.

Methods

Population

We included consecutive cryptogenic stroke patients enrolled in the hospital-based New York Columbia Collaborative Specialized Program of Translational Research in Acute Stroke (NYCC SPOTRIAS) registry between December 1, 2008 and April 30, 2012. These patients were enrolled in NYCC SPOTRIAS within 12 hours from symptom onset, admitted to the hospital, and had a standard stroke diagnostic evaluation based on current clinical practice guidelines. Cryptogenic stroke was defined based on the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification.[11] Clinical information included baseline demographics (age, sex, race-ethnicity), risk factors (hypertension, hyperlipidemia, diabetes,

coronary heart disease, congestive heart failure, renal disease, smoking), imaging data (presence of prior embolic infarcts), ECG parameters, and echocardiographic results (ejection fraction, left ventricular hypertrophy, patent foramen ovale, and left atrial diameter).

Blood samples were drawn at enrollment and 24–48 hours after admission and stored in the Center for Advanced Laboratory Measurement (CALM) at Columbia University. We followed all patients prospectively for 30 days after their stroke as part of the registry. Two patients died during the hospitalization period and all the rest completed 30 day follow up. Follow up after 30 days was retrospective and based on availability of medical records.

The study was approved by the Columbia University Medical Center Institutional Review Board and all patients provided informed consent.

Atrial cardiopathy biomarkers

PTFV1 was determined, as in prior studies[12], as the absolute value of the depth (μV) of the downward deflection (terminal portion) of the P-wave in ECG lead V1 multiplied by its duration (ms). Measurements were made on the admission paper ECG using digital calipers in mm, and were then converted to μV and ms using the ECG calibration of 10 mm/mV and 25 mm/s. Elevated PTFV1 was defined as PTFV1 > 5000 μV*ms, a threshold that was associated with doubling of stroke risk in prior studies (Kamel H. Personal communication. 2015) and is the midpoint of thresholds used in prior studies.[13, 14]

NT-proBNP was measured in a clinical laboratory using the Cobas e601 analyzer (Roche Diagnostics, Indianapolis, IN). The coefficient of variation for the NT-proBNP assay was 2% to 5% during the testing period, and its analytic measurement range was 5 to 35,000 pg/mL, where 1 pg/mL equals 0.118 pmol/L. A threshold of 250 pg/mL was used as this threshold was also associated with a 2-fold increased risk of recurrent stroke compared to patients with a normal NT-proBNP level in the Warfarin Aspirin Recurrent Stroke Study.[15]

Left atrial diameter was obtained from the echocardiogram performed for clinical purposes and was divided by the echocardiographer into 4 groups: normal and mild, moderate and severe LAE. We defined atrial cardiopathy as the presence of severe left atrial enlargement based on prior work on the relationship between left atrial size and recurrent stroke.[16]

Statistical Analysis

Patients were divided into two groups based on whether or not they had atrial cardiopathy defined as having at least one of the biomarkers above. The baseline demographic characteristics (age, sex, and race-ethnicity), vascular risk factors (hypertension, hyperlipidemia, diabetes, coronary artery disease, congestive heart failure, smoking, renal disease), echocardiographic parameters (ejection fraction, patent foramen ovale, and left ventricular hypertrophy), and neuroimaging evidence of superficial cortical infarct were abstracted from the medical record and compared between the two groups using non-parametric tests for continuous variables and Fisher’s exact tests for categorical variables. $P < 0.05$ was considered statistically significant.

Results

The NYCC SPOTRIAS registry included 3596 patients. The mean age was 65.5 ± 16.6 years, 48.3% were males, the race-ethnic distribution was 30.3% non-Hispanic White, 18.2% non-Hispanic Black, and 42.4% Hispanic, and the median NIHSS was 3 (0 – 42).

There were 159 consecutive patients with ischemic stroke enrolled in the NYCC SPOTRIAS registry between December 1, 2008 and April 30, 2012 with blood samples available for analysis of NT-proBNP.

Of these, 40 patients had cryptogenic stroke. The median age of cryptogenic stroke patients was 68.5 years (range 24 – 88 years), 35% were males, and the race-ethnic distribution was 28% non-Hispanic White, 10% non-Hispanic Black, and 60% Hispanic (Table 1).

Biomarkers of left atrial cardiopathy

Among the 40 eligible patients with cryptogenic stroke, 25 patients (63%) had at least one of the biomarkers of left atrial cardiopathy. Half (49%) had NT-proBNP>250 pg/ml, 20% had PTFV1 > 5000 μV*ms on ECG, and 5% had severe LAE. All two patients with severe LAE had evidence of at least one other atrial cardiopathy biomarker. Only 25% of patients with evidence of abnormally increased PTFV1 met our criteria for elevated NT-proBNP level.

Association between atrial cardiopathy and vascular risk factors and neuroimaging parameters

Table 1 shows the distribution of vascular risk factors and neuroimaging findings in patients with and without atrial cardiopathy. Patients with atrial cardiopathy were older (median age 76 years vs. 62 years; p=0.012) and more likely to have hypertension (96.0% vs. 33.3%, p<0.001), hyperlipidemia (60.0% vs. 26.7%, p = 0.055), and history of coronary heart disease (28% vs. 0%, p = 0.033). They were less likely to have a patent foramen ovale on echocardiogram (4% vs. 40%, p = 0.007). Other characteristics were not significantly different between the two groups (Table 1).

Of the 40 patients, 4 (10%) developed paroxysmal atrial fibrillation after their stroke. Of those, 2 patients had baseline evidence of atrial cardiopathy.

Discussion

While recent evidence suggests that the prevalence of minor risk echocardiographic abnormalities was similar between cryptogenic stroke and other non-embolic subytpes[17], we found a relatively high prevalence of biomarkers of atrial cardiopathy in patients with cryptogenic stroke. In particular, we found that patients with atrial cardiopathy and cryptogenic stroke had a higher proportion of vascular risk factors, such as hypertension and coronary heart disease, which are associated with left atrial dysfunction, and had a lower proportion of alternative sources of unexplained stroke, such as patent foramen ovale. The specific association of atrial cardiopathy with risk factors for heart disease versus other mechanisms such as PFO provides indirect evidence of a possible causal association between atrial cardiopathy and stroke risk.

Our findings are consistent with recent data calling into question the current paradigm by which AF is presumed to cause stroke.[4] According to this traditional paradigm, stasis present in the fibrillating atrium leads to thrombus formation, and thromboembolism does not occur unless there is electrocardiographic AF or flutter. Several recent lines of evidence suggest, however, that the electrocardiographic signature of AF need not be present for thromboembolism to occur. First, in populations of patients undergoing continuous cardiac monitoring for long periods of time there is a lack of temporal relationship between episodes of paroxysmal AF and ischemic stroke; i.e., stroke may occur months before or after episodes of AF.[18] Second, there is evidence of atrial electromechanical dissociation, as patients with cardiac amyloidosis may be in electrocardiographic sinus rhythm even while the atria are fibrillating.[19] Third, there are data to suggest that the majority of cardiac thrombi form in the left atrial appendage rather than in the fibrillating atrium itself, highlighting the importance of left atrial dysfunction in embolic risk even in the absence of fibrillation.[20] Fourth, results of left atrial appendage closure studies show a reduction in stroke risk with successful left atrial appendage closure despite continuing AF.[21] Fifth, genetic studies have indicated that patients with mutations that predispose to AF are at increased risk even before AF develops.[22] Finally, several studies have shown that cardiac biomarkers indicative of atrial dysfunction, like those we tested, are associated with risk of stroke and recurrent stroke, even in the absence of a diagnosis of AF.[4] Taken together, these results imply that electrocardiographic AF may be recategorized as a biomarker of stroke risk, but one that is itself related to underlying atrial structural and functional changes, including atrial fibrosis, endothelial dysfunction, and inflammation, that are in themselves the underlying cause of embolism.

In our study, there was some, but not complete, overlap among the different biomarkers tested. It is thus possible that each biomarker reflects a different mechanistic marker of left atrial dysfunction, indicating the potential importance of measuring several different biomarkers when evaluating patients with cryptogenic stroke. We were limited in this pilot study by the availability of routine clinical tests and the potential to use stored blood specimens to measure NT-proBNP. The optimal spectrum and combination of biomarkers has not been determined, however, and could include magnetic resonance imaging measures, genetic tests, and others.[4]

Left atrial enlargement carries a thrombogenic potential likely by promoting stasis, endothelial injury, and thrombus formation. Data from transesophageal studies showed a relationship between severe left atrial enlargement, spontaneous echocardiographic contrast, and left atrial appendage thrombi.[23] In addition, recent evidence supports the association between left atrial enlargement and recurrent embolic stroke subtype.[16]

Abnormally increased PTFV1 is another biomarker of left atrial dysfunction that is associated with pathophysiological changes such as hypertrophy and elevated filling pressures.[24] Evidence from observational cohorts suggests that elevated PTFV1 is associated with the risk of ischemic stroke[25], particularly those related to embolism (cryptogenic or cardioembolic).

Elevated NT-proBNP, in addition, is a measure of cardiac dysfunction and stretch, volume overload, and a predictor of incident atrial fibrillation.[26] Studies have shown an association between elevated NT-proBNP and ischemic stroke, particularly of the embolic subtype.[6] In fact, a post-hoc analysis of the WARSS trial showed a reduction in the risk of stroke or death among those assigned to warfarin rather than aspirin among the 5% of patients with highest levels of NT-proBNP.[15]

Other possible sources of embolism in patients with cryptogenic stroke include the aortic arch and paradoxical embolism through a patent foramen ovale. Recent evidence suggests that the Risk Of Paradoxical Embolism (ROPE) score provides insight into the degree of causality between PFO and stroke in patients with cryptogenic stroke found to have a PFO.[27] In our study, patients with atrial cardiopathy were less likely to have a PFO. This may imply that in patients with vascular risk factors, who typically have low ROPE scores, left atrial cardiopathy may be the mechanism of the stroke whereas in patients without vascular risk factors, the PFO may be a more likely culprit.

Our study has several limitations including its small sample size. In addition, mobile continuous outpatient telemetry and implantable loop recorders, which have been shown to increase the detection rates of paroxysmal atrial fibrillation[28, 29], were not routinely performed in our patient cohort. However, the proportion of patients with left atrial cardiopathy in our cohort (~60%) is twice as large as the reported detection rates of paroxysmal atrial fibrillation (15–30% depending on duration of monitoring)[28, 29] in patients with cryptogenic stroke, highlighting the potential importance of using these biomarkers to detect atrial thromboembolic risk. The absence of data on prevalence of biomarkers of atrial cardiopathy in other stroke subtypes is another limitation of this study. We would like to emphasize, however, the pilot nature of this study. Funding did not permit measurement of serum NT-proBNP levels on all patients. In future studies we plan to measure these markers in patients with other stroke subtypes as well. The strength of this study is that it included all three biomarkers of atrial cardiopathy and that it included not only clinical, but also echocardiographic and neuroimaging parameters, which provides a more comprehensive evaluation of patients with and without atrial cardiopathy.

Conclusion

There is a high prevalence of biomarkers of atrial cardiopathy, primarily affecting the left atrium, in patients with cryptogenic stroke, and these biomarkers are partially independent of each other, implying that each may reflect different mechanisms promote thromboembolism. These data support the concept of an underlying atrial cardiopathy as a stroke mechanism independent of electrocardiographic AF which is another biomarker of left atrial cardiopathy. Clinical trials are needed in patients with left atrial cardiopathy to determine whether anticoagulation is more effective than aspirin to reduce risk of recurrent, and potentially incident, stroke.

References

1. Chugh SS, Havmoeller R, Narayanan K, Singh D, Rienstra M, Benjamin EJ, et al. Worldwide epidemiology of atrial fibrillation: A global burden of disease 2010 study. Circulation. 2014; 129:837–847. [PubMed: 24345399]
2. Lip GY, Brechin CM, Lane DA. The global burden of atrial fibrillation and stroke: A systematic review of the epidemiology of atrial fibrillation in regions outside north america and europe. Chest. 2012; 142:1489–1498. [PubMed: 22459778]
3. Hart RG, Pearce LA, Aguilar MI. Meta-analysis: Antithrombotic therapy to prevent stroke in patients who have nonvalvular atrial fibrillation. Annals of internal medicine. 2007; 146:857–867. [PubMed: 17577005]
4. Kamel H, Okin PM, Longstreth WT Jr, Elkind MS, Soliman EZ. Atrial cardiopathy: A broadened concept of left atrial thromboembolism beyond atrial fibrillation. Future Cardiol. 2015; 11:323–331. [PubMed: 26021638]
5. Kamel H, Elkind MS, Bhave PD, Navi BB, Okin PM, Iadecola C, et al. Paroxysmal supraventricular tachycardia and the risk of ischemic stroke. Stroke. 2013; 44:1550–1554. [PubMed: 23632982]
6. Folsom AR, Nambi V, Bell EJ, Oluleye OW, Gottesman RF, Lutsey PL, et al. Troponin t, n-terminal pro-b-type natriuretic peptide, and incidence of stroke: The atherosclerosis risk in communities study. Stroke; a journal of cerebral circulation. 2013; 44:961–967.
7. Rodriguez-Yanez M, Arias-Rivas S, Santamaria-Cadavid M, Sobrino T, Castillo J, Blanco M. High pro-bnp levels predict the occurrence of atrial fibrillation after cryptogenic stroke. Neurology. 2013; 81:444–447. [PubMed: 23803318]
8. Stahrenberg R, Edelmann F, Haase B, Lahno R, Seegers J, Weber-Kruger M, et al. Transthoracic echocardiography to rule out paroxysmal atrial fibrillation as a cause of stroke or transient ischemic attack. Stroke. 2011; 42:3643–3645. [PubMed: 21998056]
9. Soliman EZ, Prineas RJ, Case LD, Zhang ZM, Goff DC Jr. Ethnic distribution of ecg predictors of atrial fibrillation and its impact on understanding the ethnic distribution of ischemic stroke in the atherosclerosis risk in communities (aric) study. Stroke; a journal of cerebral circulation. 2009; 40:1204–1211.
10. Tamura H, Watanabe T, Nishiyama S, Sasaki S, Wanezaki M, Arimoto T, et al. Elevated plasma brain natriuretic peptide levels predict left atrial appendage dysfunction in patients with acute ischemic stroke. Journal of cardiology. 2012; 60:126–132. [PubMed: 22525966]
11. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. Toast. Trial of org 10172 in acute stroke treatment. Stroke. 1993; 24:35–41. [PubMed: 7678184]
12. Soliman EZ, Juma H, Nkosi N. A simple electrocardiogram marker for risk stratification of ischemic stroke in low-resources settings. Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association. 2010; 19:388–392. [PubMed: 20472463]
13. Morris JJ Jr, Estes EH Jr, Whalen RE, Thompson HK Jr, McIntosh HD. P-wave analysis in valvular heart disease. Circulation. 1964; 29:242–252. [PubMed: 14119389]
14. Miller DH, Eisenberg RR, Kligfield PD, Devereux RB, Casale PN, Phillips MC. Electrocardiographic recognition of left atrial enlargement. Journal of electrocardiology. 1983; 16:15–22. [PubMed: 6220099]
15. Longstreth WT Jr, Kronmal RA, Thompson JL, Christenson RH, Levine SR, Gross R, et al. Amino terminal pro-b-type natriuretic peptide, secondary stroke prevention, and choice of antithrombotic therapy. Stroke. 2013; 44:714–719. [PubMed: 23339958]
16. Yaghi S, Moon YP, Mora-McLaughlin C, Willey JZ, Cheung K, Di Tullio MR, et al. Left atrial enlargement and stroke recurrence: The northern manhattan stroke study. Stroke; a journal of cerebral circulation. 2015; 46:1488–1493.
17. Li L, Yiin GS, Geraghty OC, Schulz UG, Kuker W, Mehta Z, et al. Incidence, outcome, risk factors, and long-term prognosis of cryptogenic transient ischaemic attack and ischaemic stroke: A population-based study. Lancet Neurol. 2015

18. Brambatti M, Connolly SJ, Gold MR, Morillo CA, Capucci A, Muto C, et al. Temporal relationship between subclinical atrial fibrillation and embolic events. Circulation. 2014; 129:2094–2099. [PubMed: 24633881]
19. Dubrey S, Pollak A, Skinner M, Falk RH. Atrial thrombi occurring during sinus rhythm in cardiac amyloidosis: Evidence for atrial electromechanical dissociation. British heart journal. 1995; 74:541–544. [PubMed: 8562243]
20. Stoddard MF, Dawkins PR, Prince CR, Ammash NM. Left atrial appendage thrombus is not uncommon in patients with acute atrial fibrillation and a recent embolic event: A transesophageal echocardiographic study. J Am Coll Cardiol. 1995; 25:452–459. [PubMed: 7829800]
21. Reddy VY, Doshi SK, Sievert H, Buchbinder M, Neuzil P, Huber K, et al. Percutaneous left atrial appendage closure for stroke prophylaxis in patients with atrial fibrillation: 2.3-year follow-up of the protect af (watchman left atrial appendage system for embolic protection in patients with atrial fibrillation) trial. Circulation. 2013; 127:720–729. [PubMed: 23325525]
22. Disertori M, Quintarelli S, Grasso M, Pilotto A, Narula N, Favalli V, et al. Autosomal recessive atrial dilated cardiomyopathy with standstill evolution associated with mutation of natriuretic peptide precursor a. Circulation Cardiovascular genetics. 2013; 6:27–36. [PubMed: 23275345]
23. Benjamin EJ, D'Agostino RB, Belanger AJ, Wolf PA, Levy D. Left atrial size and the risk of stroke and death. The framingham heart study. Circulation. 1995; 92:835–841. [PubMed: 7641364]
24. Hancock EW, Deal BJ, Mirvis DM, Okin P, Kligfield P, Gettes LS, et al. Aha/accf/hrs recommendations for the standardization and interpretation of the electrocardiogram: Part v: Electrocardiogram changes associated with cardiac chamber hypertrophy: A scientific statement from the american heart association electrocardiography and arrhythmias committee, council on clinical cardiology; the american college of cardiology foundation; and the heart rhythm society: Endorsed by the international society for computerized electrocardiology. Circulation. 2009; 119:e251–261. [PubMed: 19228820]
25. Kamel H, Soliman EZ, Heckbert SR, Kronmal RA, Longstreth WT Jr, Nazarian S, et al. P-wave morphology and the risk of incident ischemic stroke in the multi-ethnic study of atherosclerosis. Stroke. 2014; 45:2786–2788. [PubMed: 25052322]
26. Patton KK, Ellinor PT, Heckbert SR, Christenson RH, DeFilippi C, Gottdiener JS, et al. N-terminal pro-b-type natriuretic peptide is a major predictor of the development of atrial fibrillation: The cardiovascular health study. Circulation. 2009; 120:1768–1774. [PubMed: 19841297]
27. Kent DM, Ruthazer R, Weimar C, Mas JL, Serena J, Homma S, et al. An index to identify stroke-related vs incidental patent foramen ovale in cryptogenic stroke. Neurology. 2013; 81:619–625. [PubMed: 23864310]
28. Gladstone DJ, Spring M, Dorian P, Panzov V, Thorpe KE, Hall J, et al. Atrial fibrillation in patients with cryptogenic stroke. N Engl J Med. 2014; 370:2467–2477. [PubMed: 24963566]
29. Sanna T, Diener HC, Passman RS, Di Lazzaro V, Bernstein RA, Morillo CA, et al. Cryptogenic stroke and underlying atrial fibrillation. The New England journal of medicine. 2014; 370:2478–2486. [PubMed: 24963567]

Table 1

Demographic, clinical, and radiological characteristics of patients with and without atrial cardiopathy

	Atrial Cardiopathy (N = 25)	No Atrial Cardiopathy (N = 15)	p-value
Age	74 (71, 81)	64 (52, 71)	0.061
Gender (% male)	8 (32.0%)	6 (40.0%)	0.736
Race-ethnicity			0.762
White	6 (20.0%)	5 (33.3%)	
Black	3 (20.0%)	1 (4.2%)	
Hispanic	15 (60.0%)	9 (62.5%)	
Hypertension	24 (96%)	5 (33%)	<0.001
Diabetes	9 (36.0%)	2 (13.3%)	0.158
Hyperlipidemia	15 (40.0%)	4 (26.7%)	0.055
Coronary heart disease	7 (28.0%)	0 (0.0%)	0.033
Congestive heart failure	3 (12.0%)	1 (6.7%)	1.000
Smoking	5 (6.7%)	2 (24.0%)	0.691
Renal disease	5 (20.0%)	0 (0.0%)	0.137
Echocardiogram findings			
PFO	1 (4.0%)	6 (40.0%)	0.007
Low ejection fraction	8 (13.3%)	2 (32.0%)	0.269
LVH	13 (40.0%)	6 (52.0%)	0.767
Prior embolic infarct on imaging	7 (28.0%)	5 (33.3%)	0.736