Université de Bourgogne
UFR des Sciences de Santé
Circonscription Médecine

ANNEE 2020

N°

HIGH RATE OF CARDIAC THROMBUS DIAGNOSED BY ADDING CARDIAC IMAGING IN ACUTE STROKE CT PROTOCOL

DETECTION DE THROMBUS INTRA-AURICULAIRE GAUCHE PAR SCANNER CARDIAQUE LORS DU BILAN INITIAL D'ACCIDENT VASCULAIRE CEREBRAL ISCHEMIQUE

THESE
Présentée

à l'UFR des Sciences de Santé de Dijon
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et soutenue publiquement le 4 septembre 2020

pour obtenir le grade de Docteur en Médecine

Par Mme Angélique BERNARD

Née le 17 novembre 1990

À Lyon 4e

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AVERTISSEMENT

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SERMENT D'HIPPOCRATE

"Au moment d'être admise à exercer la médecine, je promets et je jure d'être fidèle aux lois de l'honneur et de la probité.

Mon premier souci sera de rétablir, de préserver ou de promouvoir la santé dans tous ses éléments, physiques et mentaux, individuels et sociaux.

Je respecterai toutes les personnes, leur autonomie et leur volonté, sans aucune discrimination selon leur état ou leurs convictions.

J'interviendrai pour les protéger si elles sont affaiblies, vulnérables ou menacées dans leur intégrité ou leur dignité.

Même sous la contrainte, je ne ferai pas usage de mes connaissances contre les lois de l'humanité.

J'informerai les patients des décisions envisagées, de leurs raisons et de leurs conséquences.

Je ne tromperai jamais leur confiance et n'exploiterai pas le pouvoir hérité des circonstances pour forcer les consciences.

Je donnerai mes soins à l'indigent et à quiconque me les demandera.

Je ne me laisserai pas influencer par la soif du gain ou la recherche de la gloire.

Admise dans l'intimité des personnes, je tairai les secrets qui me seront confiés. Reçue à l'intérieur des maisons, je respecterai les secrets des foyers et ma conduite ne servira pas à corrompre les mœurs.

Je ferai tout pour soulager les souffrances. Je ne prolongerai pas abusivement les agonies. Je ne provoquerai jamais la mort délibérément.

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ABBREVIATIONS AND ACRONYMS

AF	atrial fibrillation
AiCE	Advanced intelligent Clear IQ Engine
ASPECTS	Alberta Stroke Program Early CT Score
CI	confidence interval
CCTA	cardiac CT angiography
CT	computed tomography
DOA	direct oral anticoagulant
HR	hazard ratio
HU	Hounsfield units
INR	international normalized ratio
LA	left atrial
LAA	left atrial appendage
LAAt	left atrial appendage thrombus
LVEF	left ventricular ejection fraction
MRI	magnetic resonance imaging
mRS	modified Rankin scale
NASCET	North American Symptomatic Carotid Endarterectomy Trial
NIHSS	National Institutes of Health Stroke Scale
OR	odd ratio
ROI	region of interest
SEC	spontaneous echo contrast
TEE	trans-esophageal echocardiography
TIA	transient ischemic attack
TOAST	Trial of Org 10 172 in Acute Stroke Treatment
TTE	trans-thoracic echocardiography
VKA	vitamin K antagonist

INTRODUCTION

Diagnostiquer un accident vasculaire cérébral (AVC) ischémique est un défi majeur afin d'identifier rapidement l'étiologie de l'ischémie et prévenir le risque de récidive secondaire. La fibrillation atriale est la cause la plus fréquente d'AVC ischémique dans les pays occidentaux, représentant 20 à 30% des cas. La dilatation de l'oreillette gauche, provoquant une fibrose myocardique et des remodelages électriques, est le substrat de la survenue de la fibrillation atriale. L'auricule gauche, lieu privilégié de la formation des thrombus dans la fibrillation atriale non valvulaire, est associé à un risque accru de récidive d'AVC ischémique. Il est souvent considéré comme l'appendice humain le plus mortel. La détection de thrombus intra-auriculaire gauche est primordiale pour la prise en charge des patients en terme d'anticoagulation ainsi qu'au cours du suivi notamment pour le dépistage intensif de la fibrillation atriale.

L'imagerie joue un rôle central chez les patients suspects d'AVC ischémique. Le protocole d'exploration scanographique, utilisé dans de nombreux centres, comprend un scanner cérébral en contraste spontané, un scanner de perfusion cérébrale, un angioscanner des troncs supra-aortiques et des vaisseaux intracrâniens. Bien que l'échocardiographie trans-œsophagienne soit considérée comme la référence pour l'analyse de l'auricule gauche, elle n'est pas systématiquement effectuée pendant l'hospitalisation. En effet, il s'agit d'une technique semi-invasive, longue et limitée chez des patients à la phase initiale d'un AVC. Lorsqu'elle est réalisée, elle est souvent retardée de plusieurs jours, ce qui diminue le potentiel de détection de thrombus intra-auriculaire gauche. Le scanner cardiaque, avec une sensibilité de 0,99 et une spécificité de 0,94 est une technique alternative rapide et robuste pour identifier un thrombus intra-auriculaire gauche par rapport à l'échocardiographie trans-œsophagienne. Ainsi, un scanner cardiaque a été ajouté au protocole scanographique du bilan initial d'AVC ischémique, dans notre centre, en 2018.

Le but de cette étude est d'évaluer la prévalence et les facteurs associés au thrombus intra-auriculaire gauche chez les patients présentant un AVC ischémique aigu en ajoutant un scanner cardiaque au protocole scanographique initial et ainsi d'évaluer la prise en charge précoce des patients.

INTRODUCTION

The diagnostic work-up for ischemic stroke is a major challenge. It is critical to rapidly identify the underlying cause and tailor secondary prevention accordingly in the aim of reducing stroke recurrence [1–3]. Atrial fibrillation (AF) is currently the most frequent cause of ischemic stroke in western countries, accounting for 20 to 30% of overall cases [4,5]. Left atrial (LA) dilation, which causes myocardial fibrosis and electrical remodeling, is the substrate for AF occurrence [6]. The left atrial appendage (LAA) is a frequent location of thrombus formation in non-valvular AF, which is associated with an increased risk of recurrent stroke [7–10]. The LAA is often considered to be our most lethal human attachment [11]. Detection of LAA thrombus (LAAt) is of a major importance given the potential impact for patient management regarding anticoagulation and follow-up, including intensive screening for AF [12].

Acute imaging is crucial in patients suspected of ischemic stroke. Computed tomography (CT) is one of the tools for stroke investigation, and the protocol used in many centers includes non-enhanced brain CT, brain perfusion CT, carotid and intracranial CT [13,14]. Although trans-esophageal echocardiography (TEE) is considered the gold standard to analyze LAA and detect LAAt [15], it is not routinely performed during hospitalization because it is time-consuming and semi-invasive, thus restricting its use in clinical practice [16]. Moreover, when performed, it is often delayed by several days, thus potentially decreasing the chance of detecting LAAt. Cardiac CT, which has a sensitivity of 0.99 and a specificity of 0.94, is considered a rapid and safe alternative imaging modality for identification of LAAt when compared to TEE [15]. Based on these data, cardiac CT angiography (CCTA) was added to the stroke CT protocol used in our institution in 2018.

The aim of the present study was to evaluate the prevalence and factors associated with LAA thrombus (LAAt) in patients with acute ischemic stroke by adding cardiac CT to the initial imaging protocol, and to evaluate the early management and outcome of the patients managed with this enhanced imaging protocol.

MATERIALS AND METHODS

Patients

This retrospective analysis included 875 consecutive patients with suspected acute stroke or transient ischemic attack (TIA) who were referred to the Dijon University Hospital (France) between November 2018 and October 2019. Standard exclusion criteria for contrast-enhanced CT were applied, including allergic reaction to iodinated contrast, renal disease, age less than 18 years, and pregnancy.

Stroke and cardiac CT examination

CT was performed in routine conditions according to the local procedure for acute stroke management, using the Aquilion ONE GENESIS (Canon Medical Systems, Otawara, Japan). Patients were scanned in an emergency setting, so the arms were placed alongside the body. The scan range was determined from the scanogram to include the heart with a maximum length of 16 cm (**Figure 1**). Our stroke protocol consisted of the following 5 steps: non-enhanced brain CT, perfusion brain CT if necessary, cranial to caudal supra-aortic and aortic arch CT angiography, cardiac CT and, finally, post contrast brain CT.

If a brain perfusion was required, a bolus of 40 ml of nonionic contrast agent (Iomeron 350, Bracco Imaging, Italy) was injected at a rate of 5 ml/s followed by 40 ml of saline at the same rate via an 18G needle (right or left arm). For the carotid and cardiac CT, a three-phase protocol was used: 50 ml of nonionic contrast agent at a rate of 5ml/s followed by 60 ml of a contrast/saline mix (50% contrast and 50% saline) at a flow rate of 3.5 ml/s followed by 50 ml saline at a rate of 3.5 ml/s. Bolus tracking software (^{SURE}Start, Canon Medical) was used to initiate the carotid scan followed by the cardiac scan. A region of interest (ROI) was placed in the aorta arch and the scan was triggered when the Hounsfield Units (HU) value in the ROI reached 150-170 HU. A prospective ECG gated volume scan was performed during a single heartbeat. The target scan phase was calculated automatically by the CT scanner based on the patient's heart rate (^{SURE}Cardio, Canon Medical). Images were reconstructed at the most motion-free cardiac phase as determinate by the CT scanner (PhaseXact, Canon Medical) in either the end diastolic or systolic phase. Beta blockers were not administered to save time, and because the objective of the CCTA was to investigate the LAA rather than coronary arteries. A single breath hold command was given for the carotid CT and cardiac CT if the patient was able to understand. Immediately following the carotid scan, the cardiac CT was performed in

the same contrast injection. The mean time of cardiac CT after injection was 36 ± 4 s. The scan parameters for the cardiac CT were as follows (**Table 1**): tube voltage 120 kV, automatic tube current modulation (^{SURE}Exposure 3D) 40-650 mA, collimation 0.5 mm, field of view 230 mm, rotation time 275 ms.

Effective dose was calculated by multiplying the dose-length product with the International Commission on Radiological Protection conversion factor for cardiac CT imaging (0.014 mSv/mGy.cm) [17,18].

Image reconstruction

Images were reconstructed with a slice thickness of 0.5 mm and an increment of 0.25 mm. A deep learning reconstruction, Advanced Intelligent Clear IQ Engine (AiCE, Canon Medical, Otawara, Japan) was used to analyze images. AiCE is also fully integrated into the mA modulation system [19]. The system automatically adjusts each individual patient's mA profile based on the associated benefits and dose reduction abilities of AiCE reconstruction.

Image analysis

All images were analyzed by a radiologist (AB) using a workstation (Centricity Universal Viewer, GE Healthcare, General Electric Company, United States, version 6.0). All cardiac CTs were reviewed by a cardiac imaging expert blinded to clinical data (TL). Disagreement was resolved by consensus. The image quality was acceptable for heart cavities. Each reader was blinded to the results of clinical status and trans-thoracic echocardiography (TTE) or TEE information.

For cardiac CT, low density in the LAA was classified as a thrombus or a blood stasis. A thrombus was defined as round, oval or spike low density. A blood stasis was defined as a level or a triangular filing defect [20,21]. **(Figure 2)**.

LA size and LAA morphology were obtained thanks to a 3-dimensional and multiplanar reconstruction. LA area was manually traced at the level of the right inferior pulmonary vein ostium excluding the left ventricular outflow tract and the LAA [22].

For TTE and TEE, the experienced team of the cardiology department recorded the measurement of LA and cardiac functional and morphological parameters, including left ventricular ejection fraction (LVEF) using bi-plane Simpson's method and left atrial volume (LAV) using the bi-plane area-length method on apical 4-chamber and 2-chamber views. The measurements were indexed to body surface area [23].

Acute stroke was defined by imaging as a low density brain CT corresponding to a vascular territory, or an increase of the time to peak on the perfusion CT. In case of a normal brain CT and persistence of clinical symptoms compatible with a cerebrovascular event, magnetic resonance imaging (MRI) was performed to detect an intense high signal on diffusion-weighted imaging. The Alberta Stroke Program Early CT Score (ASPECTS) was used to determine middle cerebral arterial stroke severity using available computed tomography data [24]. Significant carotid stenosis was defined a narrowing > 50% of vessel diameter according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) [25].

The ischemic stroke subtype was diagnosed retrospectively by the neurologists responsible for patient management in the Stroke Unit using the original Trial of Org 10 172 in Acute Stroke Treatment (TOAST) criteria [26].

Clinical data

Clinical data including demographics, vascular risk factors, and prestroke treatments were retrospectively collected from computerized medical records. Stroke clinical severity was quantified by the National Institutes Health Stroke Scale (NIHSS) [27]. The modified Rankin scale (mRs) was used to measure the degree of disability of stroke patients at discharge [28].

Regarding previous AF, the CHA^2DS^2-VASc scale was used to estimate the premorbid risk of stroke with non-valvular AF [29]. Anticoagulation (vitamin K antagonist (VKA) therapy or a direct oral anticoagulant (DOA)) was considered as being indicated when CHA^2DS^2-VASc scale >1. In patients on VKA before stroke, effective anticoagulation was defined as international normalized ratio (INR) >2. Non-optimal anticoagulation was defined as INR under 2 with VKA therapy or non-disturbed coagulation test with DOA therapy.

Statistical analysis

The statistical results of the continuous variables are presented as means ± standard deviation for Gaussian distribution, medians (first quartile - third quartile) for non-Gaussian distribution after the Shapiro-Wilk test, and the results of the dichotomous variables as n (%). For categorical data, a chi-square or Fischer exact test was used, while a Student's t-test was used for the comparison of continuous data with normal distribution variables or a Mann-Whitney test for non-parametric variables. The threshold of significance was set at 5%.

For multivariate model, factors associated with LAAt were assessed using a stepwise backward multivariate regression analysis with inclusion and exclusion threshold values of 5%. Before the multivariate model was constructed, collinearity between the variables was excluded. The correlation matrix of the multivariate model shows that the correlation between variables (r-coefficient) did not exceed a positive or negative value of 0.25. Chi-square improvements and maximum log likelihood stepwise methods, including the Hosmer-Lemeshow goodness-of-fit test, were used to evaluate the regression model. All multivariate models were tested for multicollinearity and were stable: tolerance ranged from 0.66 to 0.96. The variation inflation factor ranged from 1.04 to 1.51. To evaluate the best LA volume value for LAA thrombus prediction, we constructed a ROC curve. The best sensitivity and specificity thresholds were determined using the Youden method. All analyses were performed using SPSS 26.0 0 software (IBM Corp., Armonk, N.Y., USA).

Ethics

According to institutional policy, approval from our Institutional Review Board was not required.

RESULTS

Patients' characteristics

Among 875 patients who were included, 325 were finally diagnosed with a stroke. Among excluded patients, 121 had a TIA, 429 had a stroke-like symptoms but no stroke, and 1 had a ventricular support system (Impella®) generating imaging artefacts. Finally, 324 stroke patients were analyzed, among whom 35 (11%) had a LAAt. (**Figure 3**).

Baseline characteristics of patients according to LAAt are shown in **Table 2**. Compared to patients without LAAt, those with LAAt were significantly older (mean age of 82 ±12 years versus 74 ± 14 years, p=0.002), more frequently women (71% versus 45%, p=0.004), and were more likely to have previous AF (63% versus 16%, p<0.001) and previous stroke (32% versus 14%, p=0.005). There was no difference between the two groups in terms of the distribution of other risk factors or stroke severity at admission. Regarding premorbid treatments, anticoagulant therapy was more frequently prescribed in patients with than in those without LAAt (43% versus 15%, p<0.001).

Brain, vessels and cardiac imaging (Table 3)

No significant difference was observed between patients with and without LAAt with regard to arterial territory of the ischemic stroke (p=0.162) or carotid and aortic arch atherosclerosis (p=0.065 and p=0.074, respectively). The M1 segment of the middle cerebral artery was the most common occlusion site in the LAAt group (n=11, 50%) compared with the no-LAAt group (n=33, 25%, p=0.016). Atherosclerotic stenosis of carotid and vertebral arteries was more frequently observed in the no-LAAt group (21% versus 3%, p=0.006). Regarding TOAST classification, cardioembolism was the most common cause of stroke in the LAAt group (80% versus 32% in no-LAAt group, p<0.001). (**Table 4 and Figure 4**).

On cardiac CT data, there was no significant difference in the reconstruction phase of the heart cycle in the end diastolic phase (75% of the RR) or systolic phase (40% of the RR) (p= 0.494). Mean LA volume was 129 ml (±48) and 70 ml/m² (±25) in the LAAt group, compared to 89 ml (±35) and 48 ml/m² (±19) in the no-LAAt group (p<0.001). After ROC curve analysis, the LA volume value that was best able to predict LAAt was 86 ml, with a sensitivity (Se) of 86 % and a specificity (Sp) of 56% (area under the ROC curve (AUC) 0.765; 95% CI: 0.691-0.840; p<0.001). (**Figure 5**).

All patients with LAAt had coronary calcifications, especially left artery descending (n=35, 100% compared to n=234, 81% in the no-LAAt group, p=0.002). No significant difference in LAA morphology (p=0.719) or LVEF (p=0.969) was observed between the two groups.

Factors associated with left atrial appendage thrombus

After multivariable backward stepwise logistic regression, female sex (hazard ratio (HR) 2.51; 95% CI: 1.09-5.77, p=0.031), previous AF (HR 4.87; 95% CI: 2.11-11.22, p<0.001) and

LA volume >86ml (HR 5.33; 95% CI: 1.70-16.69, p=0.004) were independently associated with LAAt. **(Table 5)**.

Stroke management and early outcome (Table 6)

Anticoagulation therapy (heparin) was administered more often in LAAt patients than in no-LAAt patients during hospitalization (37% versus 13%, p<0.001). There was no significant difference concerning treatment with mechanical thrombectomy, intra-venous (IV) thrombolysis, or endarterectomy (p>0.05 between the 2 groups). Patients with LAAt were more frequently treated with anticoagulants at discharge. (**Table 7**). No significant difference was observed in the diagnosis of new onset AF during hospital stay (n= 7, 20% in LAAt group versus n=44, 15% in no-LAAt group, p=0.464).

LAAt patients had markedly higher mortality rates during hospitalization than no-LAAt patients (n=13, 37% versus n=36, 13%, p<0.001), but there was no difference in the frequency of hemorrhagic complications (p=0.209). Among the 19 LAAt patients who did not receive heparin, excluding 3 patients in whom hemorrhagic infarction was identified on brain CT, 12 died during the hospital stay (63%).

Previous atrial fibrillation and left atrial appendage thrombus

In patients with previous AF (n=68), 22 had LAAt (32%). All of them had a CHA^2DS^2-VASc >0 before stroke. In the LAAt group, prestroke anticoagulation was effective for only 6 patients (27%), indicated but not prescribed for 5 patients (23%), and non-optimal for 8 patients (36%). (**Table 8**).

DISCUSSION

Our study demonstrated that LAAt was frequently diagnosed on initial imaging in patients with acute ischemic stroke, accounting for almost 11% of cases, and was independently associated with female sex, previous AF, and LA enlargement. The detection of LAAt made it possible to prescribe anticoagulation therapy, and it was associated with excess mortality.

Left atrial appendage thrombus prevalence

The prevalence of LAAt in our stroke population was similar to previous studies. Hur et al. found LAAt in 8/101 patients (8%) with ischemic stroke on TEE performed within 2 weeks of stroke onset, [20] and a study by Wang et al also identified LAAt in 8% of patients with ischemic stroke [30]. In our study, cardiac CT added to the acute ischemic stroke is an alternative to the TEE to detect LAAt as the same time as stroke symptoms.

Causes of stroke

Cardioembolism was the most common cause of stroke in patients with LAAt, which is not surprising considering that the LAA has an undisputed role in thrombosis and acute stroke [7]. In AF, blood stays in the LA longer, causing blood stagnation which can be visualized on echocardiography by spontaneous echo contrast (SEC). In previous studies, SEC was found in 50% of patients with AF and in 90% of patients with LAAt [21,31]. In addition, previous episodes of AF were shown to cause LA thrombus in patients without apparent risk factors [32]. Identifying ischemic stroke etiology is crucial for tailoring secondary prevention [14]. Here, according to TOAST classification, the two common causes of ischemic stroke in no-LAAt patients were large-atherosclerosis and other undetermined etiology. There was no difference in the localization of brain ischemia. However, in case of arterial intracranial thrombus, M1 segment occlusion was frequent in the LAAt group, suggesting that the size of thrombi could differ according to their origin.

Risk factors for left atrial appendage thrombus

ROC curve analysis demonstrated that an LA volume of 86 ml, which demonstrated high sensitivity and acceptable specificity, was a suitable cut-off value for the diagnosis of LAAt. This value could be used by clinicians to diagnose LAAt in case of artefacts or when

there is uncertainty between LAAt or blood stasis. Moreover, previous studies showed that LA dilation was associated with an increase in thromboembolic risk [10,21] and AF status [33]. A multicenter prospective study including 1611 patients showed that LA enlargement was associated with an increased risk of stroke recurrence in non-valvular AF patients (HR 1.60; 95% CI: 1.30-1.98) [34]. LA enlargement and CHA^2DS^2-VASc ≥2 were independent predictors of LA thrombus in another previous AF population [35]. This suggested cut-off value could also be used to help predict unknown AF and to intensively screen for AF during hospitalization or post-discharge diagnostic workup of stroke. Moreover, in our study, 6 LAAt patients had no previous AF or new onset AF. However, they had a LA dilation which was found to be an independent risk factor for LAAt in a study of 778 TEE [36]. Cardiac abnormalities (mitral stenosis, left ventricular dysfunction and paroxysmal AF) associated with LA stasis led to LAAt even in sinus rhythm patients [32]. Previous reviews introduced the concept of atrial cardiomyopathy as a risk factor of stroke independently of atrial rhythm [37,38], thus suggesting that the atrial substrate (fibrosis, inflammatory processes) itself could lead to cardiac thrombus formation, even in sinus rhythm patients[39].

In our study, female sex was an independent risk factor of LAAt formation. This result is consistent with previous work and with the fact that the CHA^2DS^2-VASc score includes female gender as a clinical risk for predicting stroke and embolism in AF [29].

Role of cardiac CT in acute stroke patients

In our center, following the implementation of cardiac CT for acute stroke, all patients underwent cardiac imaging including LAA contrast filling. In order to reduce blood stasis in LAA, we adapted our protocol using delayed acquisition and two injections in case of brain CT perfusion [15,40–42]. Moreover, cardiac CT was included in initial brain imaging without delaying stroke diagnosis and treatment [43]. A meta-analysis of 22 studies demonstrated a sensitivity of 0.99 (CI: 0.93-1.00) and a specificity of 0.94 (CI: 0.90-0.97) when compared to TEE [15]. Martinez et al. [44] showed that cardiac CT had a predictive negative value and a sensitivity of 100% for excluding LAA thrombus. Unfortunately, we were not able to reliably evaluate these values in our study because only 193 (60%) patients had a TTE and 10 (3%) a TEE (only in no-LAAt group) during hospitalization.

In addition to intracardiac thrombi, cardiac CT can be used to detect coronary calcifications, especially on the descending left artery, which was frequently the case in patients with LAAt. With the cardiac CT, we were also able to analyze LAA morphology for each

patient. Windsock LAA morphology has been described as more common in stroke patients, whereas chicken wing LAA is the most usual morphology in the general population [10], and Di Biase et al. [10] concluded that individuals with chicken wing LAA morphology were less likely to suffer a stroke or TIA (OR 0.18; 95% CI: 0.04 to 0.77, p=0.021). However, another large study found than extensive LAA trabeculations and a small LAA orifice diameter were associated with stroke [45]. We did not find that LAA morphology was associated with LAAt.

In terms of safety, the mean radiation dose of cardiac CT in our study was 1.52 mSv (±0.66), which is considerably lower than previous studies at 6-10 mSv [41] or 3.98 mSv [40]. Here, the dose was lower because we used deep learning acquisition [19].

Management

There is a paucity of data regarding the optimum timing for the initiation of anticoagulation in stroke patients with AF or LAAt, [12] and clinicians have to carefully weigh the risks of recurrent stroke and bleeding complications during decision making. In our population, 13 LAAt patients (37%) received an early prescription for heparin after the absence of bleeding was confirmed on brain CT. Among these patients, only one died from age-related cardiopulmonary complications (96 years old) during hospitalization and none had a hemorrhagic complication.

At admission, 22 patients in the LAAt population had previous AF, and all of them had a theoretical indication for anticoagulant therapy (CHA^2DS^2-VASc>0). However, only 6 (27%) had been prescribed effective anticoagulation, while 13 (59%) had indicated anticoagulation but either it was not prescribed or non-optimal. The underuse of effective therapy may have contributed to the high rate of LAAt in our study. In a previous study, the prevalence of LAAt detected by TEE was 9.9% in AF patients [46]. Despite the effectiveness of oral anticoagulants in stroke reduction in AF patients [47], other studies found that 1.6% of patients treated with anticoagulation for 1 month had a LAA thrombus on TEE [46] and that the risk of thrombus could be 5% despite continuous anticoagulation [48]. Moreover, LA dilation in AF patients was associated with an increase in stroke recurrence even when they were treated with anticoagulants (HR 1.59; 95% CI: 1.27-2.00), or when death was regarded as a competing risk [34]. However, data from the literature indicate that between 30 and 50% AF patients do not receive anticoagulation because of either contraindications or patient and/or physician reluctance [9,49]. In our study, all but one patients with LAAt had anticoagulation therapy (mostly DOA) at hospital discharge.

Although clinical severity (NIHSS score) was similar between the 2 groups, functional outcome was poorer in patients with with LAAt (mRS score at discharge 2 versus 0). In addition, thirteen patients (37%) with LAAt died during hospitalization compared to 36 (13%) without LAAt. Among them, 12 patients (63%) were not prescribed prompt heparin therapy. It should be noted that, in patients who received heparin therapy, no death was linked to hemorrhagic complications. A recent meta-analysis was interested in the role of heparin in acute ischemic stroke [50]. Unfortunately, there were no recommendations concerning LAAt, but they highlight that heparin should be administered acutely in the left ventricular thrombus after ischemic stroke. Moreover, in a previous study, stroke mortality was associated with age, diabetes, hypertension, smoking, hyperlipidemia and lifestyle factors [51]. Here, age was the only factor that could explain the differing mortality rate between LAAt and no-LAAt patients.

Limitations

In addition to the small number of patients who had TTE or TEE, other limitations must be acknowledged. First is the single-center design of the study and the retrospective collection of clinical data. However, the results of standardized questionnaires are systematically collected in a single computerized medical file, thus limiting the amount of missing data. Another limitation pertains to early anticoagulation therapy, which was administered by stroke physicians in a non-standardized manner with various initiation times. In addition, cardiac imaging was acquired either at the end systolic or diastolic phase, which could be a bias for LA volume quantification. However, there were no significant differences in reconstruction phase between the LAAt and no-LAAt group. Finally, stroke recurrence was not analyzed.

Conclusion

To conclude, this study highlights the contribution of cardiac CT for LAA thrombus evaluation in addition to usual brain CT in routine acute stroke imaging protocol. Our strategy could be helpful for decision-making with regard to the initiation of early anticoagulation. Further studies are needed to assess the impact on patient outcomes.

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THESE SOUTENUE PAR Mme Angélique BERNARD

CONCLUSIONS

Diagnostiquer un accident vasculaire cérébral (AVC) ischémique est un défi majeur pour identifier rapidement l'étiologie de l'ischémie et prévenir le risque de récidive secondaire. Les causes cardio-emboliques, telles que la fibrillation atriale, sont responsables de 20 à 30% des AVC ischémiques. L'auricule gauche, site privilégié de formation des thrombus intracardiaques, mérite une analyse particulière par imagerie. Le scanner cardiaque s'est révélé être une alternative de choix à l'échographie trans-œsophagienne notamment pour la détection des thrombus intra-auriculaires gauches. Ainsi, un scanner cardiaque a été ajouté au protocole scanographique du bilan initial d'un AVC ischémique.

Parmi 324 AVC ischémiques confirmés au cours d'une année, 35 patients (11%) présentaient un thrombus intra-auriculaire gauche. Les patients avec un thrombus intra-auriculaire gauche étaient plus souvent âgés, de sexe féminin, avec des antécédents de fibrillation auriculaire ou d'AVC ischémique. Le volume de l'oreillette gauche était associé à la présence d'un thrombus intra-auriculaire gauche. Le taux de mortalité était plus important chez les patients présentant un thrombus intra-auriculaire gauche (37% contre 13% dans le groupe sans thrombus intra-auriculaire gauche). Seuls 6% des patients avec antécédent de fibrillation atriale et découverte d'un thrombus intra-auriculaire gauche avaient une décoagulation efficace avant l'hospitalisation. Plus d'un tiers des patients avec thrombus intra-auriculaire gauche (37%) ont pu bénéficier d'une prescription d'héparine plus rapidement au cours de l'hospitalisation sans mortalité liée à un saignement.

Le scanner cardiaque, ajouté au protocole initial d'AVC ischémique, a permis de détecter des thrombus intracardiaques permettant ainsi une prise en charge adaptée des patients. D'autres études seraient intéressantes pour créer un consensus concernant l'initiation des traitements anticoagulants chez les patients présentant un thrombus intracardiaque.

Le Président du jury,

Pr. F. RICOLFI

Vu et permis d'imprimer
Dijon, le 22 Mai 2020
Le Doyen

Pr. M. MAYNADIÉ

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APPENDICES

Table 1. Acquisition parameters of cardiac CT.

	Acquisition
Tube voltage (kVp)	120
Tube current (mAs)	Automatic tube current modulation (^{SURE}Exposure 3D) 40-650 ± 40
Collimation (mm)	320 x 0.5
Rotation time (s)	0.275
Field Of View (mm)	230
Exposure window (ms)	300
Slice thickness (mm)	0.5
Interval (mm)	0.25
Pitch	Not applicable

± standard deviation

Table 2. Baseline characteristics of stroke patients according to LAA thrombus. n (%), mean (±SD) or median (IQR).

	LAA thrombus n= 35	No-LAA thrombus n= 289	P
Demographics and medical history			
Age, years	82 (±12)	74 (±14)	0.002
Female gender	25 (71%)	131 (45%)	0.004
Hypertension	23 (66%)	199 (69%)	0.794
Diabetes	4 (11%)	66 (23%)	0.187
BMI, kg/m^2	25 (±6)	26 (±5)	0.331
Current smoking	3 (9%)	36 (13%)	0.527
Past smoking	2 (6%)	47 (16%)	0.104
Hypercholesterolemia	10 (29%)	96 (33%)	0.608
Obstructive sleep apnea syndrome	2 (6%)	17 (6%)	0.981
Atrial fibrillation	22 (63%)	46 (16%)	<0.001
Previous ischemic stroke	11 (32%)	39 (14%)	0.005
Previous hemorrhagic stroke	0 (0%)	2 (1%)	0.625
Previous transient ischemic attack	1 (3%)	23 (8%)	0.285
Cardiac valve surgery	3 (9%)	13 (5%)	0.294
Peripheral artery disease	4 (11%)	19 (7%)	0.284
Coronary artery disease	6 (17%)	43 (15%)	0.709
Non-ischemic cardiomyopathy	5 (14%)	22 (8%)	0.167
Clinical data at admission			
NIHSS score	12 (3-20)	5 (2-11)	0.121
Heart rate (bpm)	79 (±18)	79 (±22)	0.941
Premorbid treatments			
Antiplatelet therapy	8 (23%)	96 (33%)	0.192
Anticoagulant therapy prescribed	15 (43%)	42 (15%)	<0.001
VKA	9 (26%)	18 (6%)	<0.001
DOA	6 (17%)	24 (9%)	0.103

BMI: body mass index (kg/m^2); DOA: direct oral anticoagulant; LAA: left atrial appendage; NIHSS: national institutes of health stroke scale; VKA: vitamin K antagonist.

Table 3. Imaging data for stroke patients according to LAA thrombus. n (%), mean (±SD) or median (IQR).

	LAA thrombus n= 35	No-LAA thrombus n= 289	P
Brain and vessels imaging			
Stroke diagnosed by CT	32 (91%)	217 (75%)	0.031
Carotid atherosclerosis	31 (89%)	203 (70%)	0.065
Carotid atherosclerotic stenosis	1 (3%)	62 (21%)	0.006
Aortic arch atherosclerosis	26 (74%)	212 (73%)	0.074
Intracranial vessel occlusion	22 (63%)	132 (46%)	0.055
M1	11 (50%)	33 (25%)	0.016
M2	5 (23%)	43 (33%)	0.356
T carotid	2 (9%)	18 (14%)	0.740
Tandem occlusion	2 (9%)	10 (8%)	0.682
Basilar artery	2 (9%)	25 (19%)	0.370
ACA	0 (0%)	3 (2%)	1
Stroke localization			0.162
Carotid territory	29 (83%)	206 (71%)	
Vertebrobasilar territory	3 (9%)	63 (22%)	
Multiple territories	3 (9%)	21 (7%)	
ASPECT score	8 (5-9)	8 (7-9)	0.049
Cardiac CT			
Reconstruction based on heart cycle			0.494
40% of RR	17 (49%)	158 (55%)	
75% of RR	18 (52%)	131 (45%)	
LA volume, ml	129 (±48)	89 (±35)	<0.001
LA volume, ml/m²	70 (±25)	48 (±19)	<0.001
LA area, mm²	29 (±8)	23 (±7)	<0.001
Coronary calcifications	35 (100%)	234 (81%)	0.002
LM	26 (74%)	176 (75%)	0.905
LAD	35 (100%)	216 (92%)	0.142

Cx	22 (63%)	137 (59%)	0.629
RCA	24 (69%)	146 (62%)	0.480
Coronary stent	5 (14%)	46 (16%)	0.803
LAA morphology			0.719
Windsock	24 (69%)	181 (63%)	
Chicken	5 (14%)	57 (20%)	
Cactus and cauliflower	6 (17%)	51 (18%)	
Transthoracic echocardiography	n= 18	n= 175	
LVEF, %	60 (39-61)	60 (54-60)	0.969
LA volume, ml	72 (40-98)	51 (38-73)	0.182
LA volume, ml/m²	47 (32-61)	27 (21-36)	0.002
LA area, mm²	19 (13-26)	18 (14-24)	0.600
Carotid and vertebral arteries Doppler ultrasound	n= 25	n= 226	
Atherosclerotic stenosis	1 (4%)	57 (25%)	0.044

ACA: anterior cerebral artery; ASPECT score: Alberta Stroke Program Early CT score; Cx: circumflex; LA: left atrial; LAA: left atrial appendage; LAD: left artery descending; LM: left main artery; LVEF: left ventricular ejection fraction; RCA: right coronary artery.

Table 4. TOAST classification of ischemic stroke according to LAA thrombus. n (%).

	LAA thrombus n= 35	No-LAA thrombus n= 289	P<0.001
Large-artery atherosclerosis	2 (5%)	76 (26%)	0.007
Cardioembolism	28 (80%)	91 (32%)	<0.001
Small-vessel occlusion	1 (3%)	12 (4%)	0.670
Stroke of other determined etiology	2 (6%)	34 (12%)	0.400
Stroke of other undetermined etiology	2 (6%)	76 (26%)	0.006

LAA: left atrial appendage; TOAST: trial of org 10 172 in acute stroke treatment

Table 5. Univariate and multivariate logistic regression analysis to evaluate factors associated with LAA thrombus in ischemic stroke patients (n=324).

	Univariate			Multivariate		
Variable	HR	95% CI	P	HR	95% CI	P
Age > 80 years	3.62	1.68-7.82	0.001			
Female sex	3.02	1.40-6.51	0.005	2.51	1.09-5.77	0.031
Previous AF	9.49	4.39-20.51	<0.001	4.87	2.11-11.22	<0.001
Previous stroke	3.03	1.36-6.67	0.006			
LA volume > 86ml	7.65	2.89-20.29	<0.001	5.33	1.70-16.69	0.004

AF: atrial fibrillation; CI: confidence interval; HR: hazard ratio; LA: left atrial; LAA: left atrial appendage

Table 6. In-hospital management and early outcome of patients according to LAA thrombus. n (%).

	LAA thrombus n= 35	No-LAA thrombus n= 289	P
Stroke treatment			
Thrombectomy	10 (29%)	55 (19%)	0.183
Thrombolysis	5 (14%)	59 (20%)	0.390
Heparin	13 (37%)	37 (13%)	<0.001
Endarterectomy	0 (0%)	13 (5%)	0.201
New onset AF	7 (20%)	44 (15%)	0.464
In-hospital death	13 (37%)	36 (13%)	<0.001
Hemorrhagic complication	3 (9%)	17 (6%)	0.209

AF: atrial fibrillation; LAA: left atrial appendage.

Table 7. Characteristics of patients at discharge according to LAA thrombus. n (%) or median (IQR).

	LAA thrombus n= 22	No-LAA thrombus n= 278	P
Final NIHSS score	2 (0-14)	1 (0-5)	0.208
mRS score	2 (1-5)	0 (0-2)	0.010
Treatments			
Aspirin	1 (4%)	163 (59%)	<0.001
Other antiplatelet therapy	0 (0%)	31 (11%)	0.077
Anticoagulation therapy	21 (96%)	84 (30%)	<0.001
VKA	5 (24%)	16 (19%)	0.446
DOA	16 (76%)	68 (81%)	0.639

DOA: direct oral anticoagulant; LAA: left atrial appendage; mRS: modified Rankin score; NIHSS: national institutes of health stroke scale; VKA: vitamin K antagonist.

Table 8. Characteristics of patients with previous atrial fibrillation according to LAA thrombus. n (%).

	LAA thrombus n= 22	No-LAA thrombus n= 46	P
CHA^2DS^2-VASc >0	22 (100%)	46 (100%)	1
Effective anticoagulation	6 (27%)	22 (48%)	0.107
Indicated anticoagulation but not prescribed	5 (23%)	13 (28%)	0.764
Non-optimal anticoagulation	8 (36%)	8 (17%)	0.084
Contraindicated anticoagulation	3 (14%)	3 (7%)	0.380

LAA: left atrial appendage.

Figure1. Scan Plan for the carotid CT (blue box) and cardiac CT acquisition (yellow box).

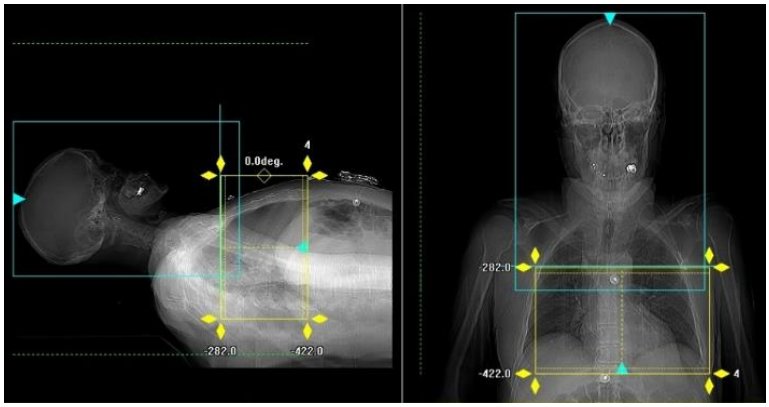
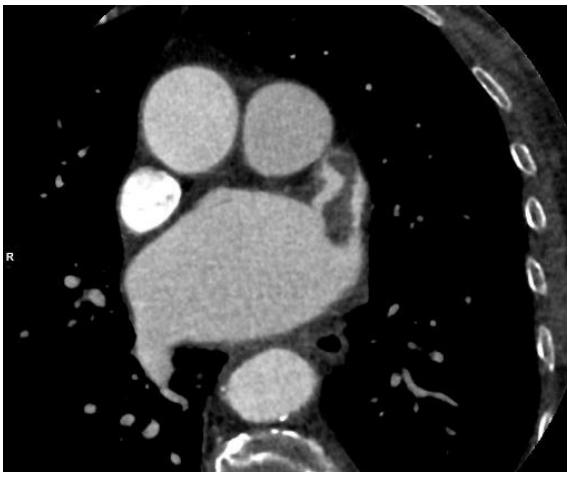
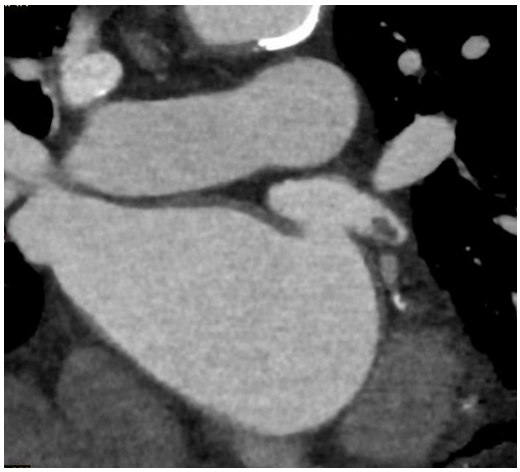


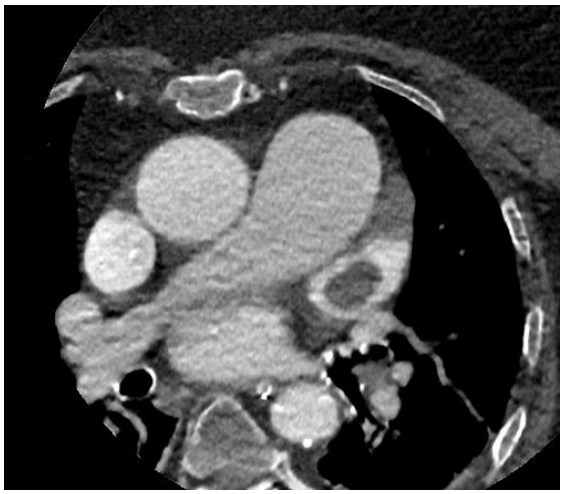
Figure 2. Examples of LAA thrombi on cardiac CT during acute stroke protocol (A and B) and LAA thrombus with blood stasis in the bottom LAA (C).



A



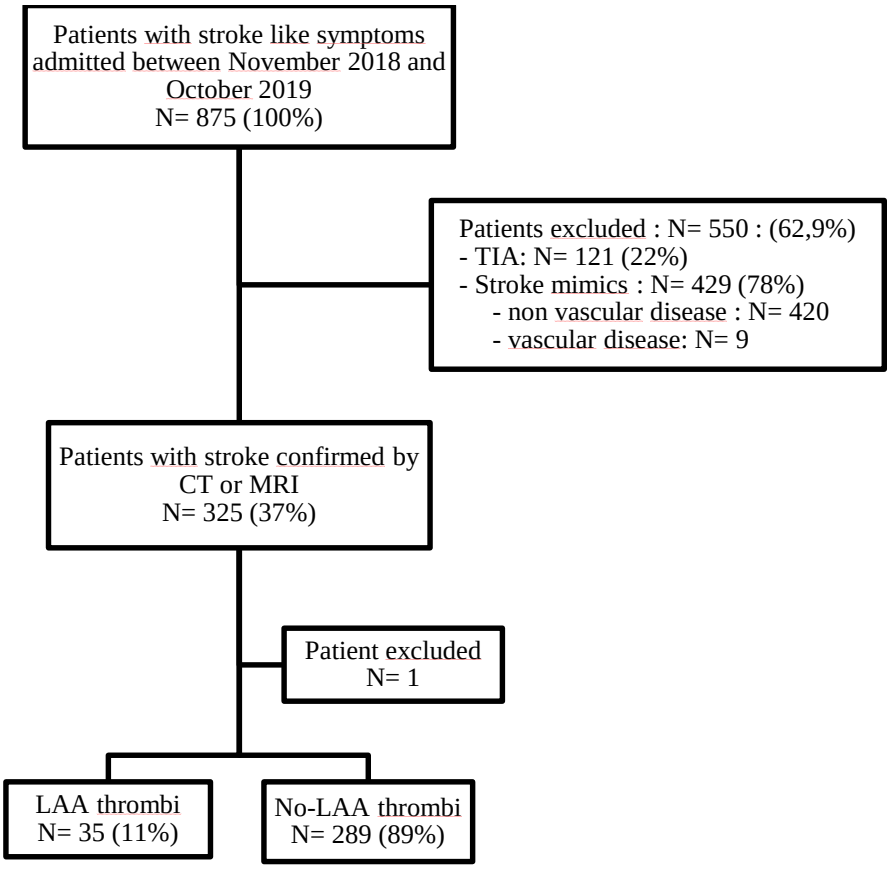
B



C

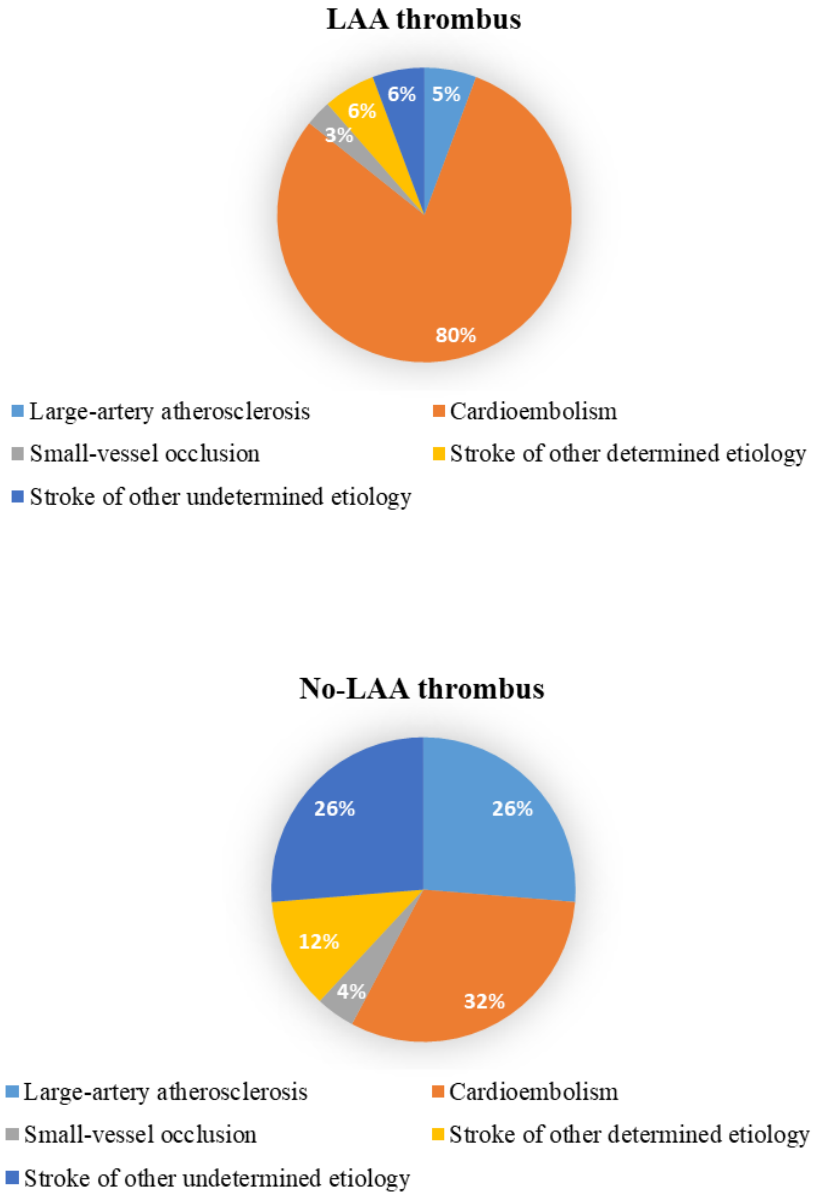
LAA: left atrial appendage.

Figure 3. Flow chart.



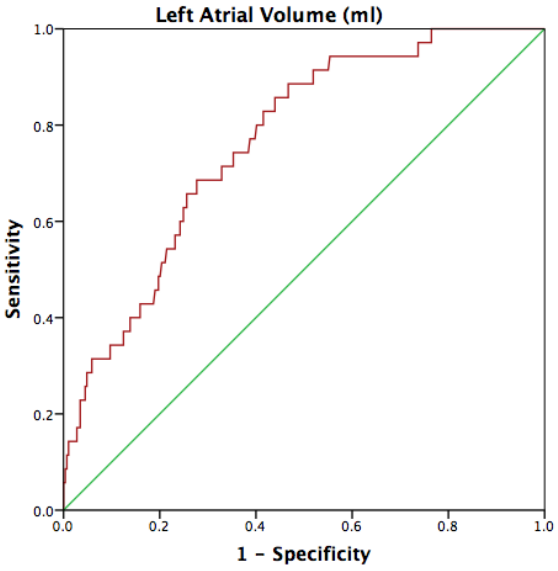
CT: computed tomography; LAA: left atrial appendage; MRI: magnetic resonance imaging; TIA: transient ischemic attack.

Figure 4. Distribution of TOAST classification in stroke patients with or without LAA thrombus (p<0.001 between the two groups).



LAA: left atrial appendage; TOAST: trial of org 10 172 in acute stroke treatment.

Figure 5. ROC curve of left atrial volume values for left atrial appendage thrombus prediction using CT scan (ml).



Université de Bourgogne
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TITRE DE LA THESE : DETECTION DE THROMBUS INTRA-AURICULAIRE GAUCHE PAR SCANNER CARDIAQUE LORS DU BILAN INITIAL D'ACCIDENT VASCULAIRE CEREBRAL ISCHEMIQUE

AUTEUR : MME ANGELIQUE BERNARD

RESUME :

INTRODUCTION : Les facteurs cardio-emboliques sont responsables de 20 à 30% des accidents vasculaires cérébraux (AVC) ischémiques, en particulier la fibrillation atriale (FA) par la formation de thrombus intra-auriculaire gauche (LAAt). Le but de cette étude était d'évaluer la prévalence et les facteurs associés au LAAt chez les patients présentant un AVC ischémique en ajoutant un scanner cardiaque au protocole scanographique initial.

MATERIEL ET METHODES : De novembre 2018 à octobre 2019, 875 patients consécutifs, admis pour des symptômes d'AVC, ont été inclus. Tous les patients ont bénéficié d'un protocole scanographique d'AVC. Le scanner cardiaque a été réalisé à l'aide d'une acquisition volumique synchronisée de manière prospective à l'échocardiogramme.

RESULTATS : Parmi 324 AVC confirmés, 35 patients LAAt (11%) et 289 non LAAt ont été analysés. Comparativement au groupe sans LAAt, les patients LAAt étaient significativement plus âgés (82 ± 12 contre 74 ± 14 ans, p = 0,002), à prédominance féminine (71% contre 45%, p = 0,004) et avaient plus souvent des antécédents de FA (63 % contre 15%, p <0,001) et d'AVC ischémique (32% contre 14%, p = 0,005). Après analyse de la courbe ROC, le volume de l'oreillette gauche était fortement associé au LAAt (ASC = 0,765; IC à 95% : 0,691-0,840; p <0,001) avec une valeur seuil de 86 ml (sensibilité 86% ; spécificité 56%). Malgré une gravité comparable de l'AVC à l'admission, le taux de mortalité était plus élevé dans le groupe LAAt (37% contre 13%, p <0,001).

CONCLUSION : Cette étude met en évidence la contribution du scanner cardiaque dans le protocole d'imagerie de l'AVC ischémique aigu en routine clinique, pour l'évaluation de thrombus intra-auriculaire gauche.

MOTS-CLES : AVC ischémique - Thrombus intra-auriculaire gauche - Scanner cardiaque