Hyperacute Incidental Late Myocardial Enhancement in Ischemic Stroke using Chest Spectral CT: Relationship with Etiology

Gastón A Rodriguez-Granillo (grodriguezgranillo@gmail.com)
Instituto Medico ENERI, Clinica La Sagrada Familia https://orcid.org/0000-0003-0820-2611
Juan J. Cirio
Clínica Sagrada Família: Clinica Sagrada Familia
Celina Ciardi
Clínica Sagrada Família: Clinica Sagrada Familia
Maria L. Caballero
Clínica Sagrada Família: Clinica Sagrada Familia
Lucia A. Fontana
Clínica Sagrada Família: Clinica Sagrada Familia
Mariano D. Buezas
Clínica Sagrada Família: Clinica Sagrada Familia
Pablo Diluca
Clínica Sagrada Família: Clinica Sagrada Familia
Pedro Lylyk
Clínica Sagrada Família: Clinica Sagrada Familia

Research Article

Keywords: Embolic stroke, Cardiac disease, Cardiovascular computed tomography, Myocardial delayed-enhancement, Dual energy computed

Posted Date: July 7th, 2021

DOI: https://doi.org/10.21203/rs.3.rs-672739/v1

Version of Record: A version of this preprint was published at Reviews in Cardiovascular Medicine on March 9th, 2022. See the published version at https://doi.org/10.31083/j.rcm2303093.

Abstract

Purpose

Hyperacute cardiac imaging of patients with acute ischemic stroke (AIS), though desirable, is impractical. Using delayed-enhancement, low-dose, non-gated, chest spectral computed tomography scans (DESCT), we explored the prevalence and patterns of incidental myocardial late iodine enhancement (LIE) and embolic sources, and the relationship with stroke etiology.

Methods

Since July 2020, DESCT was performed after cerebrovascular CT angiography (CTA) among patients with suspected AIS undergoing CT using a dual-layer spectral scanner, without additional contrast administration. Images were analyzed using monoenergetic reconstructions and iodine density maps, and the myocardial extracellular volume fraction (ECV, %) was calculated.

Results

Eighty consecutive patients with AIS were included. DESCT identified a cardiac thrombi in 6 patients (7.5%), and a complex aortic plaque in 4 (5%) cases; reclassifying 5 embolic strokes of uncertain source (28% of ESUS) to cardioembolic (CE, n=3) and non-CE (n=2) etiologies. LIE, most commonly ischemic (82%), was identified in 38 (48%) patients. We did not identify significant relationships between AIS etiology and the presence, pattern, and extension of LIE ($p>0.05$); ECV ($p=0.56$), severe aortic ($p=0.25$) or valvular ($p=0.26$) disease, or the extent of coronary calcification ($p=0.39$). Patients with evidence of major cardiovascular DESCT findings had higher rate rates of all-cause death at 90 days (42% vs. 19%, $p=0.037$).

Conclusion

In this study, hyperacute cardiac imaging of AIS with DESCT identified a high prevalence of incidental cardiac disease predominantly involving LIE of ischemic etiology and mostly not related to the stroke etiology.

Introduction

Recently, we reported in a preliminary investigation the potential usefulness of delayed-enhancement, low-dose, non-gated, chest spectral computed tomography scans (DESCT) for the early triage of cardioembolic sources (CES) in acute ischemic stroke (AIS) [1]. Such unsophisticated approach might emerge as particularly useful in the COVID-19 pandemic context, given the increasingly limited healthcare personnel and resources, and thus an intrinsic call for optimization. However, in comprehensive stroke centers involving large volume of patients admitted with AIS among whom approximately 40% require advance cardiac imaging to rule out CES, the usefulness of such a simple tool might transcend the pandemic. [1, 2]

In parallel, aside from the need to establish the presence of CES and determining stroke etiology as early as possible in order to implement appropriate treatment including antithrombotic therapy, the identification of myocardial disease, particularly of myocardial infarcts (MI), is highly relevant given the close relationship between AIS and myocardial injury both as a consequence or cause of stroke. [3-6]

Hyperacute cardiac imaging of patients admitted with AIS, though desirable, is impractical. We therefore sought to explore, by means of DESCT immediately after cerebrovascular CT angiography (CTA) among patients with AIS, the prevalence and patterns of incidental myocardial late iodine enhancement (LIE) and of CES, and the relationship with stroke etiology.

Methods

Since the COVID-19 pandemic onset, patients admitted in our emergency department underwent low-dose chest CT and since July 2020, among patients with suspected AIS, the same scan was performed after CTA with the main attempt to simultaneously rule out cardiovascular thrombotic complications using the same scan. All DESCT scans were performed using a dual-layer spectral CT (IQon Spectral CT, Philips Medical Systems Nederland B.V.). Details regarding DESCT scan acquisition protocol and analysis have been previously reported.[1] Despite some population overlap must be acknowledged with such previous smaller study, there are significant differences including the objectives (with the former aimed at evaluating CES and without clinical follow-up) and analyses (with the current study including detailed analysis of LIE, extracellular volume, clinical follow-up, and discrimination of embolic stroke of undetermined etiology; ESUS).

In brief, the diagnostic algorithm of patients with suspected AIS undergoing CT comprised a non-contrast brain CT (ruling out contraindications for intravenous tPA, if indicated), cerebrovascular CTA with or without brain perfusion at discretion of the attending physician and to the time since symptoms onset, and a low dose chest CT (64 x 0.625 mm; voltage 120 kV; current 70-140 mA; gantry speed 270 ms; pitch 1.23; slice thickness 2.0 mm) aimed at 5 minutes after contrast injection (DESCT) provided that endovascular therapy was not delayed if indicated. All images were analyzed using dedicated software (IntelliSpace Portal version 11.1; Philips Medical Systems Nederland B.V.) by an observer with experience in dual energy cardiac CT blinded to the clinical history, demographical characteristics, and stroke etiology. Images were specifically analyzed offline for this study, although online information regarding the presence of embolic sources was provided if requested, at discretion of the Stroke unit treating physician. Images were evaluated using low (40-50 keV) monoenergetic imaging and iodine-based results. Average multiplanar reconstructions (initially thin-slab and gradually increasing up to 8 mm if necessary) were used adjusting width and level at discretion to each specific energy level. Myocardium late iodine enhancement (LIE) was defined as areas with focal increase in signal attenuation compared to the normal myocardium or areas with clearly interrupted non-enhanced myocardium (**Figure 1**). [7] Thrombus was defined as an abrupt filling defect with none or non-significant contrast enhancement, clearly discriminated from surrounding structures involving high or intermediate contrast uptake such as blood and myocardial or vessel walls (**Figure 2**).[8, 9] Regions of interest (ROI) were manually traced at

the septal wall in a short axis view, within an area showing the greatest myocardial thickness and avoiding the myocardium periphery, areas with significant artifacts, or poor endocardial definition. Blood pool iodine content was calculated by placing a circular ROI at the left ventricular cavity within the same image, avoiding papillary muscles (Figure 1). Myocardial extracellular volume (a surrogate marker of diffuse myocardial fibrosis) was calculated at the septal wall using the same-day haematocrit, using the following formula: (1-Ht)*($Iodine_{myocardium}$ / $Iodine_{blood}$). Myocardial iodine ratio was calculated as $Iodine_{myocardium}$ / $Iodine_{blood}$ **(Figure 3)**.[10-14] Using 5-point Likert scales, the confidence degree for excluding CES and LIE was also evaluated being assigned a score of 1 if non-assessable, and a score of 5 if deemed of excellent quality. Left atrial area was manually traced using axial images at the image showing the maximal left atrial area, excluding the pulmonary veins and the left atrial appendage. Left atrial dilatation was defined as an area larger than 26.8 cm2, as previously reported.[15] We evaluated the presence of major cardiovascular DESCT findings defined as the presence of any of the following: CES (cardiac thrombi or >4 mm non-calcified plaque at the ascending aorta or aortic arch), extensive (>2 segments) LIE, severe aortic disease (>4 mm non-calcified plaque or severe concentric calcification), severe valvular calcification, ventricular dilatation (diastolic diameter > 55 mm), left atrium dilatation (area >26.8 cm2), or extensive (>5 segments) coronary artery calcification. Using electronic health records, patients were grouped according to the stroke etiology as follows: non-cardioembolic (non-CE), cardioembolic (CE), and embolic stroke of undetermined etiology (ESUS). Patients in whom DESCT identified a cardiac embolic source were reclassified as CE whereas those with a complex (ascending or arch) aortic plaque were reclassified as non-CE. All patients or tutors involved provided a written informed consent (habeas data) and the institutional review board approved the protocol of this observational registry. The data that support the findings of this study are available upon reasonable request.

Statistical analysis

Continuous data were reported as means ± standard deviation, or as median (interquartile range, IQR) in case of non-uniform distribution, and categorical variables were reported as frequency and percentages. Differences between groups were assessed using one-way analysis of variance for continuous variables, and chi-square tests for categorical variables. Differences between groups with non-parametric distribution were evaluated using Kruskal-Wallis tests. A two-sided p value of less than 0.05 indicated statistical significance. Statistical analyses were performed using SPSS software, version 22.0 (IBM SPSS Statistics for Windows, Armonk, NY).

Results

Study population

Between July 2020 and January 2021, 80 consecutive patients (**Table 1**) with AIS who underwent DESCT after cerebrovascular CTA were included, with a baseline median NIHSS of 10 (2-18). Fifty-nine (74%) patients also underwent brain CT perfusion. The mean age was 70.9±15.2 years, with no significant differences between etiological groups (non-CE 68.9±16.0 years, CE 73.9±13.3 years, ESUS 65.5±16.2

years, p=0.18). Eleven (14%) patients had a previous history of MI and 17 (22%) atrial fibrillation. Five patients (6%) had increased cardiac enzyme (troponin T>40 U/L) levels upon admission. Thirty-seven (46%) patients underwent acute treatment of the AIS.

DESCT cardiovascular findings

DESCT involved a mean radiation dose-length product of 200.0±67.6 mGy*cm and scans were performed at a mean heart rate of 82.0±12.7 bpm. All cases were deemed assessable (Likert≥2) for ruling out cardiac thrombi, whereas for the identification of LIE DESCT was considered non assessable in 5 cases (6%) and of regular quality in 23 (20%) cases. The mean confidence degree for the identification of cardiac thrombi was significantly higher than for LIE (Likert 4.6±0.7 vs. 3.2±1.3, p<0.0001).

DESCT identified a cardiac thrombi in 6 patients (7.5%), located at the left atrial appendage (LAA) in 3 cases and at the left ventricle in 3 cases (**Table 2** and **Figure 2**). Three of these were confirmed with transesophageal echocardiogram (TEE)/cardiac CT and in 3 other cases the Stroke unit decided not to undergo further advanced imaging given the clearness of the DESCT images (**Figure 2, panels A-C**). Two LAA thrombi not detected by DECST comprised patients who developed atrial fibrillation later during the same hospitalization and underwent TEE 3 and 4 weeks after DSCT. The presence of a complex (>4mm) non-calcified plaque at the ascending aorta/aortic arch was identified in 4 (5%) patients. One patient with ESUS had marked dilatation of right chambers motivating the search and identification of an atrial septal defect (**Figure 1**). Based on DESCT findings, 5 (28%) patients with ESUS were reclassified to CE (n=3) and non-CE (n=2) etiologies. Overall, patients with CE stroke had a larger prevalence of major DESCT findings (non-CE 42%, CE 80%, ESUS 29%, p=0.0001).

Myocardial late iodine enhancement patterns

Myocardial LIE (**Figure 1**) was identified in 38 (48%) patients, involving a significant (more than 2 ventricular segments) territory in 19 (24%) cases. The most common (82%) pattern of LIE was of ischemic (subendocardial or transmural) etiology. We did not identify a relationship between AIS etiology and the presence, pattern, and extension of LIE. The mean myocardial ECV was 33.5±6.7%, with no significant differences between etiologies (non-CE 32.7±6.0%, CE 33.9±8.2%, ESUS 34.9±4.5%, p=0.56).

Additional cardiac findings

Twenty-nine (36%) patients had extensive coronary artery calcification (more than 5 coronary segments). Left atrial dilatation was identified in 24 (30%) cases using DESCT, and was significantly more prevalent in CE stroke (non-CE 17%, CE 57%, ESUS 7%, p<0.0001). Patients with left atrial dilatation did not show evidence of incremented myocardial fibrosis compared to those without left atrial dilatation (iodine ratio 0.58±0.2 vs. 0.54±0.1, p=0.13; ECV 34.6±9.4% vs. 33.1±5.3%, p=0.39).

We did not identify significant differences between stroke etiologies regarding the presence of severe aortic (p=0.10) or valvular (p=0.23) disease, or with respect to the presence and extension of coronary calcification (**Table 2**). Sixty-six (83%) patients underwent transthoracic echocardiogram (TTE), with a

mean left ventricular ejection fraction of 58.7±7.7% and the identification of wall motion abnormalities in only 9 (11%) patients.

Clinical outcome

Clinical follow-up data was available in 77 (96%) cases. Among these, 24 (31%) patients died and 40 (52%) had functional independence (modified Rankin scale ≤2) after 90 days of follow-up. Age and NIHSS scale were the only clinical variables explored associated with functional dependence and death, whereas the stroke etiology was not related to such adverse outcome (**Table 3**). Patients with evidence of major cardiovascular DESCT findings had higher rate rates of all-cause death at 90 days compared to patients without major findings (42% vs. 19%, p=0.037), and left atrial area evaluated with DESCT was significantly larger among patients with death (26.4±10.3 cm2 vs. 21.6±6.4 cm2, p=0.04).

Discussion

The main findings of the present observational study can be summarized as follows. Firstly, DESCT identified a high prevalence of incidental cardiac disease among patients with AIS, mostly involving LIE of ischemic etiology. Secondly, DESCT findings reclassified 28% of patients with ESUS. And thirdly, most of these findings were not related to the stroke etiology, being the presence of CES and left atrial dilatation the only more prevalent in CE strokes. In a pilot investigation, we recently reported a good performance of DESCT for hyperacute ruling out of CES in patients with AIS undergoing CTA, without the need of additional contrast administration.[1] This tool, provided that a dual-layer spectral CT scanner is available, comprises a low-dose, non-gated chest CT scan that does not require any modification of the acquisition protocol. Besides, since non-contrast brain CT is performed before contrast injection, the decision to administrate intravenous fibrinolysis, of indicated, is not delayed.

One of the most interesting findings was the documentation of LIE in approximately half of the patients, unrelated to the stroke etiology and most of ischemic pattern, including 14% of ESUS with extensive LIE. Whether these patients should be reclassified to CE, as well as those with left atrium dilatation (7%), remains uncertain. Despite the fact, related to the observational nature of this study, that only 13 patients underwent cardiac CT to validate such findings, the identification of ischemic LIE particularly when involving more than 2 myocardial segments was feasible and well defined. The ability of such unsophisticated tool for the simultaneous assessment of both CES and myocardial LIE, aside from the improved tissue characterization enabled by spectral imaging, is also partly related to a temporal resolution of DESCT (135 ms) that exceeds in most cases the threshold at which significant cardiac motion artifacts occur.[16]

MI has been unequivocally recognized as a major cause of AIS, related not only to left ventricular thrombus, but also to other diverse mechanisms usually not identified by TTE [3, 6]. In this regard, two very large registries have reported an enduring incremented risk of stroke among patients with previous MI.[4, 6] In addition, patients with AIS have an increased risk of MI.[5, 17] In keeping with this, 39% of the patients included in our study had evidence of ischemic LIE, being extensive in 24%. Although this might

seem a high figure, previous studies have shown that silent infarcts are significantly more common than expected.[18] As a counterpart, only 11% of TTE showed wall motion abnormalities. In keeping with this, though exploratory, at 90 days of follow-up, left atrial area evaluated with DESCT but not with TTE was related to mortality, as it was the evidence of major cardiovascular DESCT findings.

A number of limitations should be acknowledged. Since DESCT images were analyzed by a specialist with experience in dual energy imaging within a comprehensive stroke center, extrapolation of our results should be cautious. Moreover, since DESCT was not compared to an established standard, findings must be interpreted as exploratory. For the same reason, the 3 cases among whom further testing was not performed given the assuring DESCT findings (Figure 2) cannot be conclusively confirmed. Besides, as an observational study where downstream testing was left at the discretion of treating physicians, the ability of DESCT to accurately reclassify stroke etiology was not specifically tested. In this regard, since the rate of further advanced cardiac imaging (34%) was average, incidental findings such as LIE did not seem to trigger additional testing. Future prospective studies powered for clinical outcomes and cost-effectiveness are warranted.

Conclusions

In this study, hyperacute cardiac imaging of AIS by means of DESCT identified a high prevalence of incidental predominantly ischemic cardiac disease, and most findings were not related to the stroke etiology. This simple tool might potentially aid reclassification of ESUS, although this should be demonstrated in prospective studies.

Declarations

Ethical standards

This retrospective study has been approved by the Institutional Review Board of Clinica La Sagrada Familia and all procedures have been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

All patients or tutors gave their informed consent prior to their inclusion in the study.

Funding: None

Conflicts of interest/Competing interests: Dr. Pedro Lylyk is Consultant of Philips Healthcare.

Availability of data and material: Datasets are available upon reasonable request

Code availability: Not applicable

Authors' contributions: Guarantor of integrity of the entire study: GRG, JJC. Study concepts and design: GRG, JJC. Literature research: GRG, JJC, CC. Clinical studies: MLC, MB, LAF, PD. Data analysis: GRG, PD.

Statistical analysis: GRG. Manuscript preparation: GRG, JJC. Manuscript editing: JJC, CC, MLC, MB, PD, PL.

Ethics approval: The protocol of this retrospective study was approved by the local Institutional Review Board

Consent to participate: Written informed consent (habeas data) was obtained from all patients or tutors.

Consent for publication: All the authors agree to publish the article

References

1. Rodriguez-Granillo GA, Cirio JJ, Ciardi C, Caballero ML, Ceron M, Bleise C, Diluca P, Lylyk P: **Early Triage of Cardioembolic Sources Using Chest Spectral Computed Tomography in Acute Ischemic Stroke**. *J Stroke Cerebrovasc Dis* 2021, **30**(6):105731.

2. Chung JW, Park SH, Kim N, Kim WJ, Park JH, Ko Y, Yang MH, Jang MS, Han MK, Jung C *et al*: **Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification and vascular territory of ischemic stroke lesions diagnosed by diffusion-weighted imaging**. *J Am Heart Assoc* 2014, **3**(4).

3. Witt BJ, Ballman KV, Brown RD, Jr., Meverden RA, Jacobsen SJ, Roger VL: **The incidence of stroke after myocardial infarction: a meta-analysis**. *Am J Med* 2006, **119**(4):354 e351-359.

4. Merkler AE, Diaz I, Wu X, Murthy SB, Gialdini G, Navi BB, Yaghi S, Weinsaft JW, Okin PM, Safford MM *et al*: **Duration of Heightened Ischemic Stroke Risk After Acute Myocardial Infarction**. *J Am Heart Assoc* 2018, **7**(22):e010782.

5. Touze E, Varenne O, Chatellier G, Peyrard S, Rothwell PM, Mas JL: **Risk of myocardial infarction and vascular death after transient ischemic attack and ischemic stroke: a systematic review and meta-analysis**. *Stroke* 2005, **36**(12):2748-2755.

6. Sundboll J, Horvath-Puho E, Schmidt M, Pedersen L, Henderson VW, Botker HE, Sorensen HT: **Long-Term Risk of Stroke in Myocardial Infarction Survivors: Thirty-Year Population-Based Cohort Study**. *Stroke* 2016, **47**(7):1727-1733.

7. Rodriguez-Granillo GA, Deviggiano A, Capunay C, De Zan M, Fernandez-Pereira C, Carrascosa P: **Role of Iterative Reconstruction Algorithm for the Assessment of Myocardial Infarction with Dual Energy Computed Tomography**. *Acad Radiol* 2019, **26**(9):e260-e266.

8. Hur J, Kim YJ, Lee HJ, Nam JE, Ha JW, Heo JH, Chang HJ, Kim HS, Hong YJ, Kim HY *et al*: **Dual-enhanced cardiac CT for detection of left atrial appendage thrombus in patients with stroke: a prospective comparison study with transesophageal echocardiography**. *Stroke* 2011, **42**(9):2471-2477.

9. Li W, Yu F, Zhu W, Zhang W, Jiang T: **Detection of left atrial appendage thrombi by third-generation dual-source dual-energy CT: Iodine concentration versus conventional enhancement measurements**. *Int J Cardiol* 2019, **292**:265-270.

10. Lee HJ, Im DJ, Youn JC, Chang S, Suh YJ, Hong YJ, Kim YJ, Hur J, Choi BW: **Myocardial Extracellular Volume Fraction with Dual-Energy Equilibrium Contrast-enhanced Cardiac CT in Nonischemic Cardiomyopathy: A Prospective Comparison with Cardiac MR Imaging**. *Radiology* 2016, **280**(1):49-57.

11. Treibel TA, Bandula S, Fontana M, White SK, Gilbertson JA, Herrey AS, Gillmore JD, Punwani S, Hawkins PN, Taylor SA *et al*: **Extracellular volume quantification by dynamic equilibrium cardiac computed tomography in cardiac amyloidosis**. *J Cardiovasc Comput Tomogr* 2015, **9**(6):585-592.

12. Chevance V, Damy T, Tacher V, Legou F, Ridouani F, Luciani A, Kobeiter H, Rahmouni A, Deux JF: **Myocardial iodine concentration measurement using dual-energy computed tomography for the diagnosis of cardiac amyloidosis: a pilot study**. *Eur Radiol* 2018, **28**(2):816-823.

13. Wang R, Liu X, Schoepf UJ, van Assen M, Alimohamed I, Griffith LP, Luo T, Sun Z, Fan Z, Xu L: **Extracellular volume quantitation using dual-energy CT in patients with heart failure: Comparison with 3T cardiac MR**. *Int J Cardiol* 2018, **268**:236-240.

14. Abadia AF, van Assen M, Martin SS, Vingiani V, Griffith LP, Giovagnoli DA, Bauer MJ, Schoepf UJ: **Myocardial extracellular volume fraction to differentiate healthy from cardiomyopathic myocardium using dual-source dual-energy CT**. *J Cardiovasc Comput Tomogr* 2020, **14**(2):162-167.

15. Currie BJ, Johns C, Chin M, Charalampopolous T, Elliot CA, Garg P, Rajaram S, Hill C, Wild JW, Condliffe RA *et al*: **CT derived left atrial size identifies left heart disease in suspected pulmonary hypertension: Derivation and validation of predictive thresholds**. *Int J Cardiol* 2018, **260**:172-177.

16. Otton JM, Phan J, Feneley M, Yu CY, Sammel N, McCrohon J: **Defining the mid-diastolic imaging period for cardiac CT - lessons from tissue Doppler echocardiography**. *BMC Med Imaging* 2013, **13**:5.

17. Adams RJ, Chimowitz MI, Alpert JS, Awad IA, Cerqueria MD, Fayad P, Taubert KA, Stroke C, the Council on Clinical Cardiology of the American Heart A, American Stroke A: **Coronary risk evaluation in patients with transient ischemic attack and ischemic stroke: a scientific statement for healthcare professionals from the Stroke Council and the Council on Clinical Cardiology of the American Heart Association/American Stroke Association**. *Circulation* 2003, **108**(10):1278-1290.

18. Arenja N, Mueller C, Ehl NF, Brinkert M, Roost K, Reichlin T, Sou SM, Hochgruber T, Osswald S, Zellweger MJ: **Prevalence, extent, and independent predictors of silent myocardial infarction**. *Am J Med* 2013, **126**(6):515-522.

Tables

Table 1. Demographics

	Total population
Age (years)	70.2±15.3
Male sex (%)	50 (63%)
Systolic blood pressure (mmHg)	160.4±31.7
Diastolic blood pressure (mmHg)	89.2±16.8
Creatinine levels (mg/dl)	1.05±0.4
Glucose levels (mg/dl)	131.4±65.2
NIHSS	10.0 (2.0; 18.0)
NIHSS-24hours	9.0 (1.0; 19.3)
Diabetes (n, %)	15 (19%)
Hypertension (n, %)	66 (84%)
Hypercholesterolemia (n, %)	25 (32%)
Smoking (n, %)	16 (13%)
Obesity (n, %)	12 (15%)
Atrial fibrillation (n, %)	17 (22%)
Previous myocardial infarction (n, %)	11 (14%)
Previous stroke (n, %)	14 (18%)

NIHSS: National Institutes of Health Stroke Scale

Table 2. Hyperacute identification and characterization of comprehensive cardiovascular findings identified by delayed-enhancement, low-dose, non-gated, chest spectral CT scans (DESCT); discriminated

by stroke etiology.

	Overall (n=80)	Non-CE (n=36)	CE (n=30)	ESUS (n=14)	p value
DESCT findings					
Embolic sources (n, %)					
LAA thrombi	3 (4%)	0	3 (10%)	0	0.048
Ventricular thrombi	3 (4%)	0	3 (10%)	0	0.048
Complex aortic (ascending/arch) plaque	4 (5%)	3 (8%)	1 (3%)	0	0.31
Myocardium					
Late iodine enhancement (n, %)	38 (48%)	17 (47%)	15 (50%)	6 (43%)	0.91
Ischemic	31 (82%)	15 (88%)	12 (80%)	4 (67%)	0.49
Non-ischemic/mixed	7 (17%)	2 (12%)	3 (20%)	2 (33%)	
Myocardial iodine ratio	0.55±0.1	0.55±0.1	0.55±0.1	0.59±0.1	0.47
Extracellular volume (%)	33.5±6.7	32.7±6.0	33.9±8.2	34.9±4.5	0.56
Left atrium					
Area (cm2)	23.1±8.0	21.0±6.9	27.1±8.8	19.8±5.4	0.001
Dilatation (n, %)*	24 (30%)	6 (17%)	17 (57%)	1 (7%)	<0.0001
Coronary artery calcification (CAC)					
CAC (n, %)	67 (84%)	30 (83%)	27 (90%)	10 (71%)	0.30
CAC (n segments, %)	4.3±3.6	3.9±3.6	5.0±3.5	3.7±3.7	0.39
CAC >5 segments	29 (36%)	10 (28%)	13 (43%)	6 (43%)	0.36
Severe aortic disease (n, %)	6 (8%)	4 (11%)	2 (7%)	0	0.25
Severe valve calcification (n, %)	6 (8%)	1 (3%)	4 (13%)	1 (7%)	0.26
Transthoracic echocardiography					
Left ventricular ejection fraction (%)	58.7±7.7	58.6±8.0	58.2±9.1	60.2±2.3	0.77
Wall motion abnormalities (n, %)	9 (14%)	4 (13%)	5 (21%)	0	0.11
Left atrial area (cm2)	19.8±5.4	19.7±4.6	21.9±6.2	16.3±4.1	0.013
CAC >5 segments	29 (36%)	10 (28%)	13 (43%)	6 (43%)	0.36
Severe aortic disease (n, %)	6 (8%)	4 (11%)	2 (7%)	0	0.25
Severe valve calcification (n, %)	6 (8%)	1 (3%)	4 (13%)	1 (7%)	0.26
Transthoracic echocardiography					
Left ventricular ejection fraction (%)	58.7±7.7	58.6±8.0	58.2±9.1	60.2±2.3	0.77
Wall motion abnormalities (n, %)	9 (14%)	4 (13%)	5 (21%)	0	0.11
Left atrial area (cm2)	19.8±5.4	19.7±4.6	21.9±6.2	16.3±4.1	0.013

LAA: left atrial appendage; CE: cardioembolic, ESUS: embolic stroke of uncertain source. *LA dilatation (>26.8 cm2)

Table 3. Cardiovascular findings identified by delayed-enhancement, low-dose, non-gated, chest spectral CT scans (DESCT); discriminated by the occurrence of death or functional dependence, among patients with 90-day clinical follow-up.

	Death		p value	Functional dependence		p value
	No (n=53)	Yes (n=24)		No (n=40)	Yes (n=37)	
Age (years)	66.6±15.9	78.4±11.1	0.002	65.7±15.8	75.2±13.8	0.007
NIHSS admission	7.5±8.0	17.7±9.4	<0.0001	5.3±6.6	16.5±8.9	<0.0001
NIHSS-24h	6.5±8.2	23.4±12.2	<0.0001	3.4±5.0	20.6±11.7	<0.0001
Etiology			0.21			0.45
Non-CE	28 (79%)	8 (22%)		21 (58%)	15 (42%)	
CE	16 (57%)	12 (43%)		14 (50%)	14 (50%)	
ESUS	9 (69%)	4 (31%)		5 (39%)	8 (62%)	
DESCT major finding*	7 (19%)	17 (42%)	0.037	18 (62%)	11 (37%)	0.17
LIE n segments	1.6±2.0	1.1±1.6	0.31	1.6±2.2	1.2±1.6	0.38
CAC (n segments)	4.2±3.6	4.8±3.6	0.45	4.2±3.8	4.6±3.5	0.64
Left atrial area$_{DESCT}$ (cm2)	21.6±6.4	26.4±10.3	0.04	21.9±5.5	24.5±10.1	0.16
Extracellular volume (%)	34.2±0.1	32.6±0.1	0.35	34.0±0.1	33.3±0.1	0.63
LVEF$_{echo}$ (%)	58.5±7.5	58.5±9.4	0.94	58.4±8.4	58.6±6.9	0.91
Left atrial area$_{echo}$ (cm2)	20.1±4.9	20.9±6.2	0.65	20.5±5.2	19.9±4.9	0.64

CE: cardioembolic, ESUS: embolic stroke of uncertain source; LIE: late iodine enhancement; CAC: coronary artery calcification; LVEF: left ventricular ejection fraction; NIHSS: National Institutes of Health Stroke Scale. * CES (cardiac thrombi or >4 mm non-calcified plaque at the ascending aorta or aortic arch), extensive (>2 segments) LIE, severe aortic disease (>4 mm non-calcified plaque or severe concentric calcification), severe valvular calcification, ventricular dilatation (diastolic diameter > 55 mm), left atrium dilatation (area >26.8 cm2), or extensive CAC (>5 segments).

Figures

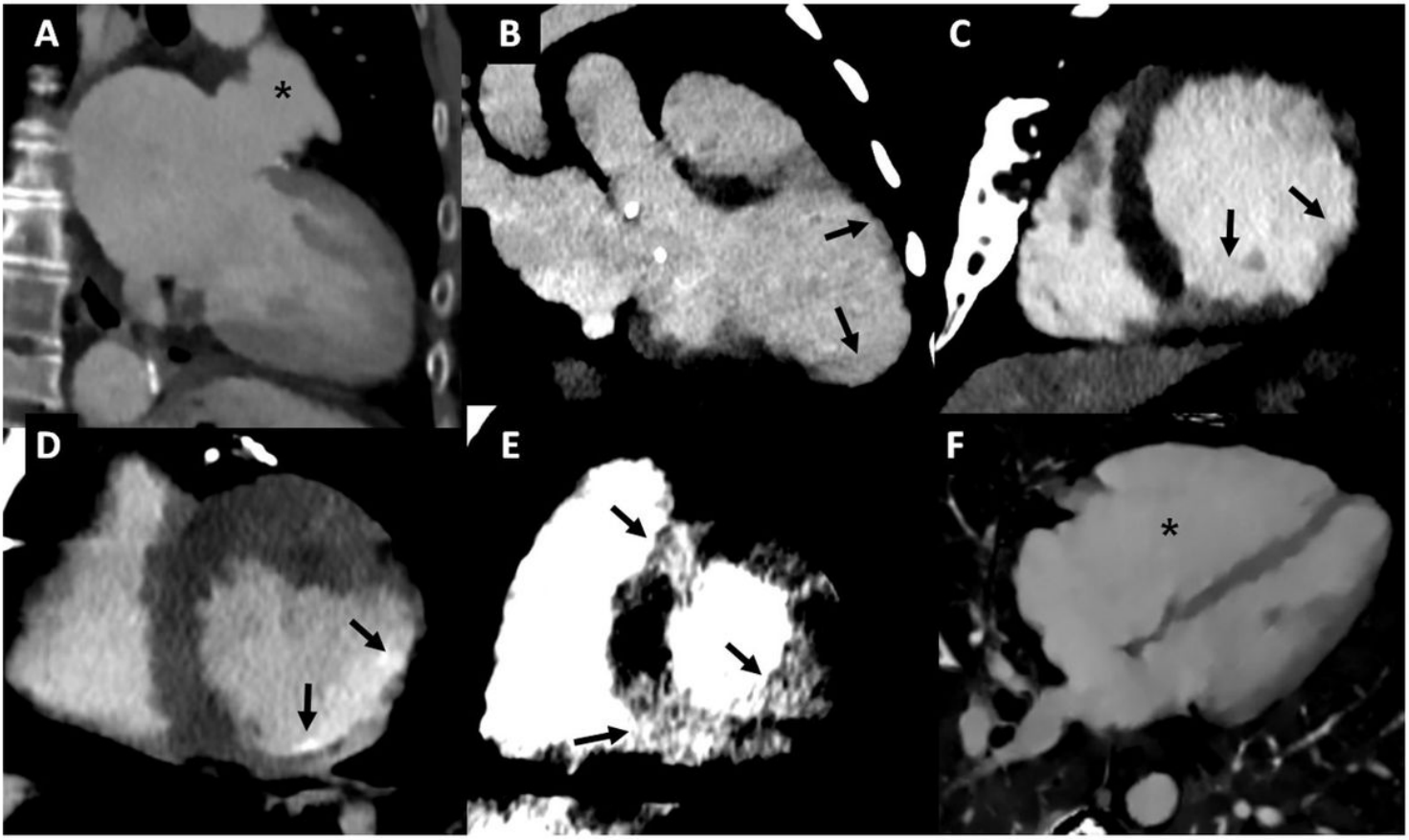

Figure 1

A. Left atrial dilatation without left atrial appendage thrombus (*). B-D. Myocardial infarcts (arrows). E. Non-ischemic late iodine enhancement (arrows). F. Marked dilatation of the right cardiac chambers (*), leading to diagnosis of atrial septal defect.

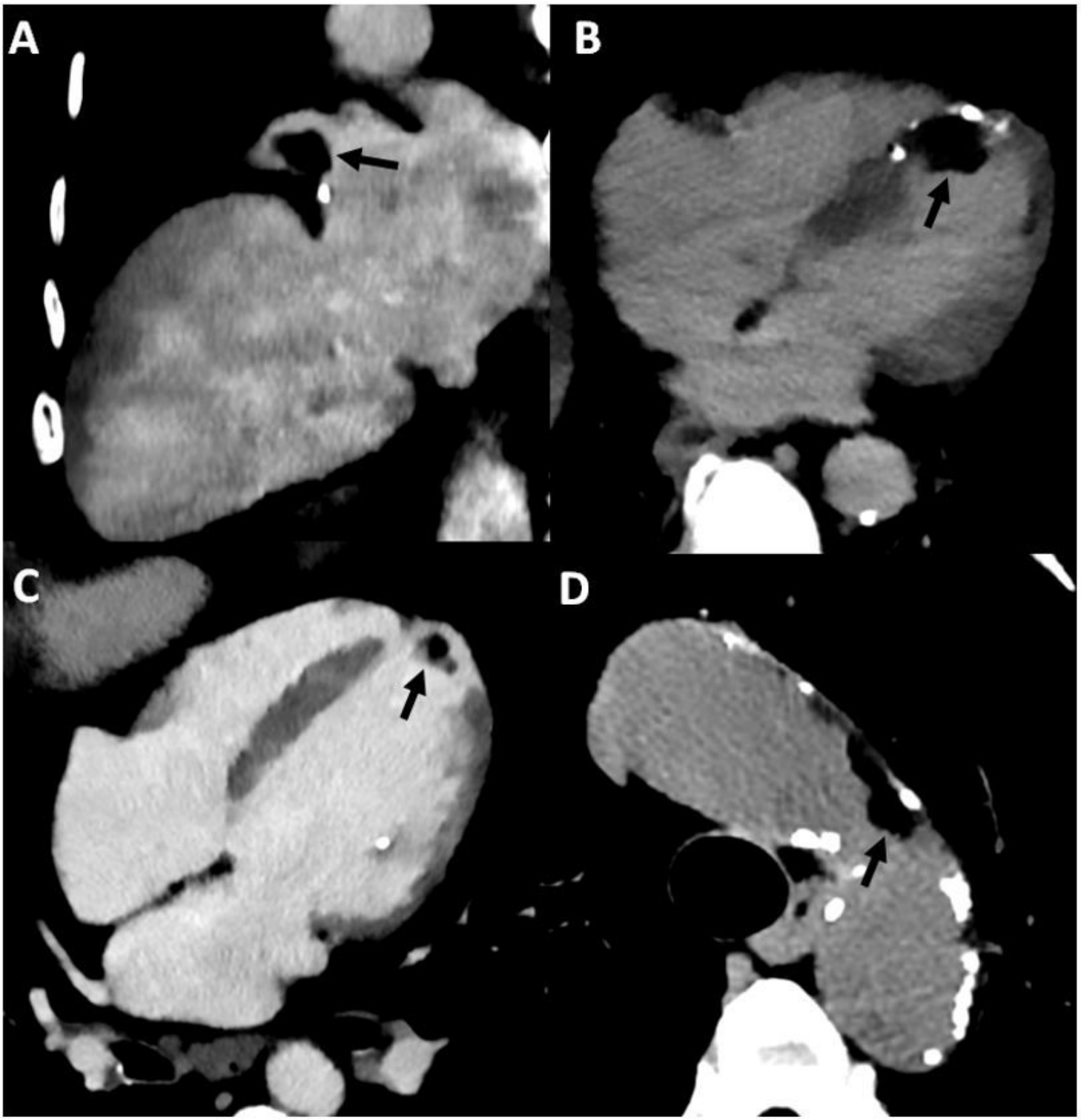

Figure 2

A. Large left atrial appendage thrombus (arrow). B and C. Left ventricular thrombus (arrows). D. Complex aortic plaque (arrow).

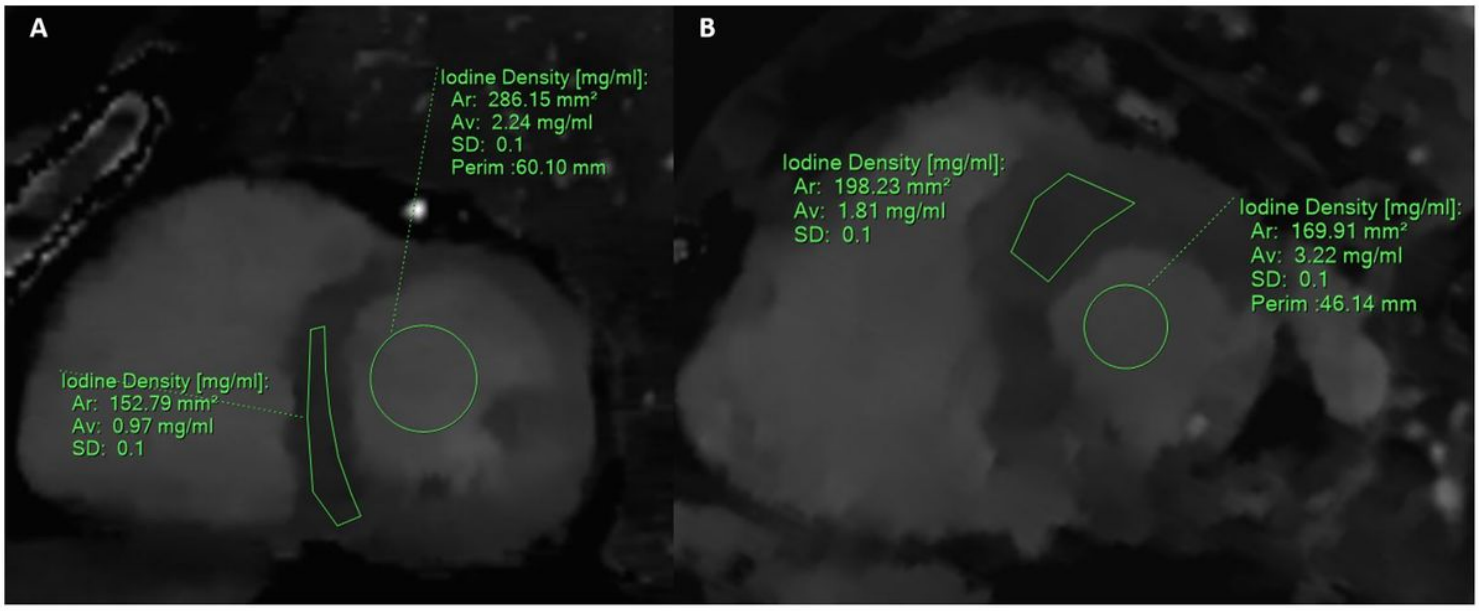

Figure 3

Regions of interest traced at the septal wall and at the left ventricular cavity in order to measure iodine content (mg/ml). A. Patient with a myocardial extracellular volume of 24 % (based on a haematocrit of 45.5%). B. Patient with myocardial disease and incremented extracellular volume of 35% (based on a haematocrit of 38.6%).