Determination of the prevalence and predictors of ventricular thrombus with assessment of the risk of systemic embolization to the CNS in patients after acute myocardial infarction using magnetic resonance imaging, echocardiography and cardiac markers – a prospective, unicentric, observational study

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Background. Left ventricular thrombus (LVT) formation is one of the well-known and serious complications of acute myocardial infarction (AMI) due to the risk of systemic arterial embolization (SE). To diagnose LVT, echocardiography (TTE) is used. Late gadolinium-enhanced cardiovascular magnetic resonance (DE-CMR) is the gold standard for diagnosing LVT.
Objectives. The aim of this observational study was to determine the role of transthoracic echocardiography and cardiac markers in predicting the occurrence of LVT compared with a reference cardiac imaging (DE-CMR) and to determine the risk of systemic embolization to the CNS using brain MRA.
Methods. Seventy patients after MI managed by percutaneous coronary intervention (localization: 92.9% anterior wall, 7% other; median age 58.7 years) were initially examined by transthoracic echocardiography (TTE, n=69) with a focus on LVT detection. Patients were then referred for DE-CMR (n=55). Laboratory determination of cardiac markers (Troponin T and NTproBNP) was carried out in all. Brain MRA was performed 1 year apart (n=51).
Results. The prevalence of LVT detected by echocardiography: (n=11/69, i.e. 15.9%); by DE-CMR: (n=9/55, i.e. 16.7%). Statistically significant parameters to predict the occurrence of LVT after AMI (cut off value): (a) detected by echocardiography: anamnestic data – delay (≥ 5 hours), echocardiographic parameters – left atrial volume index (LAVI≥ 32 mL/m^2), LV EF Simpson biplane and estimated (≤ 42%), tissue Doppler determination of septal A wave velocity (≤ 7.5cm/s); (b) detected by DE-CMR: anamnestic data – delay (≥ 13 hours), DE-CMR parameters – left ventricular end-diastolic diameter (≥ 54mm). The value of cardiac markers (Troponin T and NTproBNP in ng/L) in LVT detected by echocardiography did not reach statistical significance. In LVT detected by DE-CMR, NTproBNP was statistically significantly increased at 1 month after AMI onset (no optimal cut-off value could be determined). There was no statistically significant association between the LVT detection (both modalities) and the occurrence of clinically manifest and silent cardioembolic events.
Conclusion. Our study confirmed a relatively high prevalence of LVT in the high-risk group of patients with anterior wall STEMI. Due to the low prevalence of thromboembolic complications, no significant association between the LVT detection and the occurrence of a cardioembolic event was demonstrated.

Key words: ventricular thrombus, ST-elevation myocardial infarction, predictors of ventricular thrombus, stroke

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INTRODUCTION

Formation of a thrombus in the left ventricle (LVT) is one of the well-known and serious complications of acute myocardial infarction (AMI) due to the risk of systemic arterial embolization (SE) with variable clinical presentation and potentially severe consequences depending on the degree of target organ damage. The presence of LVT influences significantly mortality and morbidity[1,2]. LVT is one of the main causes of ischaemic cardioembolic stroke after MI, and finding this source of cardioembolization is crucial to initiate adequate anticoagulation therapy as

a secondary prevention. The pathogenesis of ventricular thrombus after myocardial infarction involves factors of the well-known Virchow triad – vascular stasis, endothelial damage and prothrombotic state[3].

The available studies show that LVT occurs most frequently in STEMI arising in the perfusion territory of the ramus interventricularis anterior (RIA) (ref.[4-6]). In the vast majority of cases, it occurs in the anterior wall MI (ref.[7]).

In the modern era of interventional AMI therapy, the incidence of LVT in patients after anterior AMI is reported to be in the wide range of 4–15% (ref.[5,8]). In a systematic review by Massussi et al. that included only studies with patients after anterior wall STEMI using DE-CMR to detect LVT, the mean prevalence of ventricular thrombi was 8% (with a wide range of 7–26%) (ref.[9]).

The sensitivity and specificity of DE-CMR for the diagnosis of LVT are high (88% and 99%, respectively), whereas these values are significantly lower for routinely used transthoracic echocardiography (TTE) (35% and 60%, respectively), but reach higher values (up to 60%) in well-defined clinical situations in patients after anterior STEMI. Contrast echocardiography is a very useful diagnostic method (88% and 96%, respectively) (ref.[10,11]). The use of other diagnostic methods (cardiac CT, oesophageal echocardiography) in this isolated indication has no more justification (similar sensitivity to TTE).

It is well known that the presence of LVT is associated with an increased risk of SE. In a study looking at the risk of SE (3-year follow), the annual rate was 3.7%, four times higher than in patients without evidence of LVT (ref.[12]). LVT formation was found to be associated with an even higher risk of SE than AF alone (HR 5.2 vs. 3.2) (ref.[13]).

The risk of SE is higher for mobile thrombi or thrombi protruding into the LV cavity – the positive predictive value is 83% and 57%, respectively[14].

Early initiation of anticoagulation therapy is essential to reduce this risk, but there is insufficient evidence to support the use of new oral anticoagulants. According to AHA and ESC guidelines, warfarin only is indicated for oral use, despite the low level of and relatively outdated evidence[15,16].

The aim of this observational study was to determine the value of TTE and cardiac markers in predicting the occurrence of LVT in comparison with the reference cardiac imaging method – DE-CMR. Another aim was to determine the risk of clinically manifest SE and the concomitant risk of clinically silent cerebral embolizations to the CNS demonstrated by magnetic resonance imaging (MRI) of the brain during a 12-month period of adequately guided pharmacotherapy after AMI (dual antiplatelet therapy in the absence of thrombus finding and combined anticoagulation/antiplatelet therapy in the presence of thrombus according to current ESC recommendations) (ref.[17]).

MATERIALS AND METHODS

Patient recruitment for this study took place from 12/2018 to 6/2021. A total of 70 consecutive patients who were diagnosed with AMI and referred for additional examination after signing informed consent were included in the study (n=69, 1 death); for the nature of the patient cohort, see Tables 1 and 2. The diagnosis of AMI was made according to the criteria of the Fourth Universal Definition of MI from the ESC guidelines[18]. All patients underwent coronary angiography, laboratory investigations, TTE, DE-CMR, and brain MRA.

Patients with haemodynamically significant cerebral artery stenosis, evidence of arrhythmias with cardioembolic potential (atrial fibrillation/flutter) and patients with a previous history of anticoagulation therapy were excluded from the study.

The aim of the study was to determine:

1. The prevalence of ventricular thrombi in patients after AMI detected by (a) TTE, (b) DE-CMR.

2. Statistically significant independent parameters (P<0.05) to predict the occurrence of LVT obtained from two imaging modalities (TTE, DE-CMR) and cardiac markers (Troponin T, NTproBNP). For statistically significant parameters/predictors, the optimal cut-off value was defined (ROC analysis). All these significant parameters from univariate analysis, including the parameters categorized by the cut-off values, were subjected to multiple (multivariate) logistic regression (STEPWISE FORWARD model).

3. The relationship between the presence of LVT detected by TTE and DE-CMR and the occurrence of a clinically manifest (acute stroke) or silent (postischaemic changes, microembolization) cardioembolization event to the CNS (MRA of the brain) during the 12-month follow-up period on dual antiplatelet therapy in the absence of LVT or on combined antithrombotic therapy (anticoagulation/antiplatelet) in the presence of LVT according to current ESC recommendations[17].

Echocardiography – TTE (n=69)

Echocardiographic examination was performed using a Vivid 7 device (GE Healthcare Technologies, Waukesha, Wisconsin, USA) equipped with an M3S probe (2–5 MHz) early after the diagnosis of AMI (within 24 h of AMI diagnosis), with a recommended optimization of the device (Table 3). The examination was aimed at quantifying ventricular morphology and function and at detecting LVT.

As part of the protocol, patients were examined in the left lateral recumbent position at quiet respiration, and the sequences recorded for subsequent evaluation were obtained in quiet expiration to minimize translational cardiac motion. Regional and global left and right ventricular function were analyzed using projections according to recent recommendations[19,20]. Detailed evaluation of the findings was performed off-line in the environment of the archiving program EchoPac 7 Option (BT version 10.0.0).

A description of all the parameters investigated is not part of this text due to its extension.

Laboratory examinations (n=69)

All laboratory analyses were performed in certified laboratories of the University Hospital in Olomouc on the day of admission and one month after the diagnosis of AMI. Serum levels of aminoterminal fragment of the

brain natriuretic peptide (NT-proBNP) were determined using an electrochemiluminescence method (Elecsys proBNP Gen 2, Cobas 8000 e602, Roche Diagnostics, Mannheim, Germany). The lower limit of detection was 5 ng/L, intra-assay coefficient of variation (CV) was <1.8%, and inter-assay CV was <3.1%. Cardiac troponin T (hs-cTnT) was detected using electrochemiluminescence immunoassay (Elecsys Tropo T, Cobas 8000 e602, Roche Diagnostics, Mannheim, Germany). The lower limit of detection was 5 ng/L, CV at 13 ng/L was 10%, intra-assay CV was <4.8%, and inter-assay CV was <5.2%. The upper reference limit was set at 14 ng/L.

Evaluation of other laboratory parameters was not the aim of this work.

Magnetic resonance imaging of the heart (n=55)

DE-CMR was performed between Month 1 and Month 3 after AMI.

The standardized protocol included the examination for morphology, function, viability, and LVT presence by TrueFisp2D-CINE sequence acquisition with determination of the extent of myocardial delayed enhancement (DE) using CE-IR-MRI sequences. To visualize the DE phenomenon, gadolinium contrast agent (1M Gadovist, Bayer Schering Pharma, Berlin, Germany) was administered i.v. at a dose of 0.15 mL/kg. The sequence acquisition was performed in breath hold 15 min after the contrast agent application.

A description of all the parameters investigated is not part of this text due to its extension.

Magnetic resonance imaging of the brain and cerebral arteries (n=51)

Brain MRA was performed 12 months after the diagnosis of AMI according to a specific embolization protocol (Table 4). Examinations were performed on a Siemens

Table 1. Cohort characteristics (n=70).

Cohort characteristics		Number	Percentage
Gender	Male	51	73.9%
	Female	18	26.1%
AMI location	Lateral	1	1.4%
	Anterior	65	92.9%
	Inferior	4	5.7%
Infarct related artery	ACD	1	1.4%
	RCX	3	4.3%
	RIA	65	92.9%
	RMS	1	1.4%
STEMI	No	3	4.3%
	Yes	67	95.7%
First episode of CAD	No	7	10.0%
	Yes	63	90.0%
Previous antiplatelet therapy	No	59	84.3%
	Yes	11	15.7%
Smoker	No	29	41.4%
	Yes	41	58.6%
Diabetes mellitus	No	58	82.9%
	Yes	12	17.1%
Atrial fibrillation	No	66	94.3%
	Yes	4	5.7%
Claustrophobia	No	61	87.1%
	Yes	9	12.9%
Obesity (BMI > 30)	No	47	66.7%
	Yes	23	33.3%
Death	No	69	98.6%
	Yes	1	1.4%

Table 2. Age and anthropometric parameters.

Age and anthropometric parameters	Mean	SD	Median	Minimum	Maximum
Age at the time of AMI	58.7	13.1	59.0	34.0	90.0
Weight (kg)	87.5	18.3	85.0	46.0	125.0
BMI	28.9	5.0	28.7	20.1	46.3
BSA (m^2)	2.0	0.2	2.0	1.4	2.5
Delay (hrs.)	6.6	8.4	3.0	0.5	48.0

Magnetom Avanto 1.5 T, Q engine (33 mT/m), Tim 76x18 (Siemens AG, Erlangen, Germany) equipped with Syngo 2004A software.

For the purpose of our study, 3 groups of changes on MRA of the brain were defined according to radiological description – acute ischaemic lesions, postischaemic lesions, nonspecific lesions – microembolization (Table 5).

All patients underwent MR angiography of the intracranial arteries to exclude haemodynamically significant stenosis of extra- and intracranial sections of the cerebral arteries.

Clinical evaluation of systemic embolizations and new ischaemic stroke

All patients with suspected new clinically manifest SE and stroke were examined by a board-certified cardiologist and a neurologist experienced in cerebrovascular issues.

Table 3. Optimization of the ultrasound device during the examination.

Nyquist limit setting in the range of 50 to 70 cm/s
Adjustment of depth, gain, mechanical index, compression or focus
CWD, PWD, TDI modalities – acquisition of 3-cycle sequences; 5 cycles in atrial fibrillation with a speed of 50–100 mm/s
B-mode 2D imaging – evaluation of 3-cycle sequences; 5 cycles in atrial fibrillation with a frame rate of 40–80/s
Doppler examinations – optimization of the sector width for the region of interest

Table 4. Embolization protocol – individual sequences.

Localizer T2 weighted turbo spin echo (TSE)
Fluid attenuated inversion recovery (FLAIR)
Diffusion-weighted imaging (DWI)
3D time of flight magnetic resonance angiography (TOF MRA)

Table 5. Brain MRA – percentage of radiologically described changes.

Changes on MRI of the brain	Number	Percentage
Acute stroke	1	2.0%
Postischaemic changes	5	9.8%
Nonspecific foci/ microembolisation	26	51.0%

The National Institutes of Health Stroke Scale (NIHSS) was used to assess the severity of new ischaemic events[21,22].

Statistical methods

IBM SPSS Statistics version 23 statistical software (Armonk, NY: IBM Corp.) was used for the data analysis. The Fischer's exact probability test was used to process the data.

A total of 70 patients who signed informed consent were included, 69 of whom underwent echocardiography and laboratory examination (1 death); 55 patients underwent DE-CMR and 51 underwent brain MRA.

RESULTS

In our cohort, there was a high prevalence of STEMI with localization to the anterior wall of the LV with culprit lesion in the RIA territory (95.7% and 92.9%, respectively). All LVTs were detected (by TTE and DE-CMR) while the apicoanterior kinetic impairment was present (100%). The assessment of statistical significance in terms of predicting the development of LVT was not possible for the localization of AMI and culprit lesion due to the low representation of other localizations (territories) of AMI.

In our cohort after the first AMI, the prevalence of LVT according to TTE (without the contrast agent) was 15.9% (n=11). However, in the case of DE-CMR which was not performed or could not be performed in all patients (claustrophobia, ICD, etc.), 16.7% of LVT was detected (n=9).

None of the anamnestic data (except for the delay) – gender, age, smoking, diabetes mellitus, obesity – reached statistical significance to predict the formation of LVT detected by both imaging modalities.

Among the parameters obtained by TTE, the following were statistically significant: left atrial size (LAVI mL/m^2), LV EF in % measured by the Simpson biplane method and also by estimation (upper limit), tissue Doppler determination of septal A wave velocity (cm/s). Optimal cut-off values were determined for the given parameters (Table 6).

Among the parameters obtained by DE-CMR, the left ventricular end-diastolic diameter (mm) reached statistical signifikance; LV EF (%) almost reached statistical significance (P=0.056) (Table 7). All parameters are listed in Table 8.

The values of cardiac markers (baseline and 1 month after AMI diagnosis) were not statistically significant

Table 6. Quantitative significant parameters for predicting LVT formation detected by TTE.

Prediction of LVT formation detected by TTE	P	OR	95% CI for OR	
			Lower	Upper
Delay ≥ 5 hours	0.037	4.221	1.092	16.32
LVEF 2D Simpson biplane ≤ 42%	0.006	10.00	1.959	51.04
LVEF estimated upper limit ≤ 42%	0.008	7.000	1.648	29.74
TDI SEPT. A ≤ 7.5 cm/s	0.007	6.900	1.694	28.10
LAVI ≥ 31.5 mL/m^2	0.041	4.444	1.060	18.63

Table 7. Quantitative significant parameters for predicting LVT formation detected by DE-CMR.

Prediction of LVT formation detected by DE-CMR	*P*	OR	95% CI for OR	
			Lower	Upper
Delay ≥ 13 hours	0.037	1.126	1.007	1.258
End-diastolic diameter of the LV ≥ 54 mm	0.047	5.962	1.023	34.76

Table 8A. Parameters evaluated to assess predictors of LVT formation detected by TTE.

Prediction of LVT formation detected by TTE	*P*	OR	95% CI for OR	
			Lower	Upper
Gender male	0.519	0.583	0.114	2.998
Age at the time of AMI	0.604	1.013	0.964	1.064
BMI (kg/m^2)	0.899	1.008	0.887	1.146
BSA (m^2)	0.204	6.233	0.370	105.0
Anterior AMI location	0.999	–	–	–
Infarct related artery RIA	0.999	–	–	–
Delay (h)	0.011	1.106	1.023	1.195
Smoker	0.720	0.789	0.215	2.888
Diabetes mellitus	0.940	1.067	0.199	5.705
Obesity (BMI > 30)	0.643	0.713	0.170	2.987
LVEF 2D Simpson biplane (%)	0.024	0.927	0.867	0.990
LVEF estimated upper limit (%)	0.039	0.934	0.875	0.997
Septum (mm)	0.408	1.204	0.776	1.867
Posterior wall (mm)	0.432	1.183	0.778	1.797
LVEDd (mm)	0.461	1.033	0.948	1.125
E/Em	0.347	1.080	0.920	1.266
LAVI (mL/m^2)	0.025	1.134	1.016	1.265
LA1 (mm)	0.933	1.005	0.897	1.126
E (cm/s)	0.898	1.002	0.975	1.030
A (cm/s)	0.542	0.989	0.955	1.024
E dec. time (ms)	0.217	1.008	0.995	1.021
TDI LAT. (cm/s) E	0.187	0.827	0.624	1.096
TDI LAT. (cm/s) A	0.101	0.812	0.633	1.041
TDI SEPT. (cm/s) E	0.714	0.949	0.717	1.256
TDI SEPT. (cm/s) A	0.013	0.668	0.485	0.920
Mitral regurgitation	0.999	–	–	–
TAPSE (mm)	0.701	0.973	0.844	1.121
RV1 diameter (mm)	0.415	0.941	0.813	1.089
RV4 diameter (mm)	0.264	0.923	0.801	1.063
PASP estimation (mm/Hg)	0.067	1.056	0.996	1.120
TrV < 3.4 (m/s)	0.241	0.333	0.053	2.095
TrV > 3.4 (m/s)	0.241	3.000	0.477	18.86
3D TTE LVQ (%)	0.341	0.967	0.902	1.036
3D GLS (%)	0.482	0.929	0.756	1.141
2D GLS (%)	0.323	0.903	0.737	1.106

Results of logistic regression analysis – estimation of unadjusted values of odds ratios (OR).

for the prediction of LVT detected by TTE. The median baseline NTproBNP value is non-significantly higher (1.483 ng/L in case of LVT detection vs. 870 ng/L in the absence of LVT, =0.104). The median NTproBNP value is significantly higher for thrombi detected by DE-CMR at 1 month (median 1.167 vs. 290 ng/L, *P*=0.01). The baseline NTproBNP value almost reached statistical significance (^{P}=0.053). However, no reliable cut-off value can be determined for the NTproBNP value (AUC statistic = 0.630, < 0.75) (Table 9).

No statistically significant relationship was found between the detection of LVT (by either imaging method) and the finding of changes on the brain MRI (cardioembolization to CNS). There was one clinically manifest acute stroke that occurred in a patient with LVT proven by DE-CMR (16.7% vs. 0% in the group without LVT detected by DE-CMR, *P*=0.133).

In LVT detected by TTE, there was a trend towards a higher proportion of microembolizations (66.7% vs. 47.6%, *P*=0.465) and postischaemic changes (22.2% vs.

Table 8B. Parameters evaluated to assess predictors of LVT formation detected by DE-CMR 41.

Prediction of LVT formation detected by DE-CMR	*P*	OR	95% CI for OR	
			Lower	Upper
Gender male	0.130	3.200	0.711	14.40
Age at the time of AMI	0.344	1.027	0.972	1.086
BMI (kg/m^2)	0.257	0.902	0.754	1.078
BSA (m^2)	0.082	0.042	0.001	1.494
Anterior AMI location	0.999			
Infarct related artery RIA	0.999			
Delay (h)	0.016	1.107	1.019	1.202
Smoker	0.709	0.759	0.179	3.223
Diabetes mellitus	0.999			
Obesity (BMI > 30)	0.380	0.471	0.087	2.533
LVEF (%)	0.056	0.952	0.905	1.001
DE location-lateral	0.999			
DE location-anterior	0.102	3.690	0.772	17.64
DE location-apical	0.142	5.115	0.580	45.13
DE location-septal	0.643	0.667	0.120	3.709
DE location-inferior	0.999			
DE location-diffuse	1.000			
Aneurysm	0.000	64.50	7.604	547.1
LVEDd (mm)	0.043	1.101	1.003	1.209
RV4 diameter (mm)	0.547	0.947	0.793	1.131
Septum (mm)	0.698	1.151	0.566	2.342
LVEDV (mL)	0.154	1.008	0.997	1.020
LVESV (mL)	0.099	1.010	0.998	1.022
SV (mL)	0.645	0.990	0.947	1.034

Table 9. Cardiac marker values in relation to the prediction of LVT using DE-CMR.

	Presence of LVT using DE-CMR						
	NO			YES			*P*
	Median	Minimum	Maximum	Median	Minimum	Maximum	
NTproBNP (ng/L) –baseline	793	32	6,500	1,483	814	6,229	0.053
Troponin T – peak (ng/L) – baseline	2,433	31	10,000	3.371	166	10,000	0.789
NTproBNP (ng/L) – after 1M	290	11	2,678	1,167	247	3,270	0.010
Troponin T (ng/L) – after 1M	12.0	3.0	87.0	24.0	4.0	468.0	0.173

7.1%, *P*=0.209) compared to the group of patients without LVT detetcted by TTE.

Paradoxically, LVT detected by DE-CMR had a lower proportion of postischaemic changes (0% vs. 10.3%, *P*=1.0) and micromebolisations (33.3% vs. 51.3%, *P*=0.665) compared to the group without LVT detected by DE-CMR.

DISCUSSION AND LIMITATIONS

The relatively higher prevalence of LVT in our cohort of patients compared with the previously published data (16% vs. 8%) may be due to the very high proportion of patients with anterior wall STEMI and may also be related to the trend described so far, where studies with smaller patient cohorts (n < 100) report a higher prevalence of LVT (ref.[7,9]). The prevalence of LVT in the present study is similar for both imaging modalities. The similar prevalence for TTE and DE-CMR may be related to the greater sensitivity of TTE when the clinical question is well defined (35% vs. 60%), i.e. the detection of LVT in patients with anterior wall STEMI who were the most represented in this study. It should be noted that the nature of LVT may also have differed between the groups (LVT detected by TTE/DE-CMR). Early LVTs that occur within 7 days of AMI diagnosis were detected by TTE while DE-CMR detected late LVTs (1–3 months after AMI diagnosis). DE-CMR was performed at least 1 month apart and, for the reasons mentioned above, a direct comparison of LVT prevalence between imaging methods is not possible in this study. Patients treated with dual antiplatelet (and, if LVT was detected, anticoagulant) therapy after AMI may have experienced thrombus resolution (by the time of DE-CMR examination) or, conversely, de-novo LVT formation (for LVT detected by DE-CMR). A direct comparison of the prevalence of LVT detected by TTE/DE-CMR would require a same day examination with both imaging modalities, which is not practically feasible

in clinical practice given the acuteness of the condition and the availability of DE-CMR . Therefore, the slight difference in parameters predicting the development of LVT in TTE and DE-CMR is likely related to the different pathogenesis of early and late LVTs. In the case of LVT detection by DE-CMR, parameters indicative of irreversible LV remodeling (dilated LV) with persistent (apicoanterior) kinetic impairment seem to be of greater importance, which is consistent with the significance of the NTproBNP parameter after a longer time interval – at 1 month from AMI diagnosis, as well as a larger endiastolic diameter of LV and also a longer delay (13 h vs. 5 h) compared to early LVTs detected by TTE. However, the possibility of false-positive findings with TTE cannot be excluded, e.g., due to near-field artifact when contrast agent is not used to opacify the LV. In TTE significant parameters, a possible association with diastolic LV dysfunction and rather acute deterioration of LV systolic function can be identified. The tissue Doppler velocity of the septal A wave in predicting the development of LVT is a rather surprising, previously unpublished finding for predicting LVT. The equivocal results with 3D echocardiography and in the assessment of global longitudinal strain (2D and 3D GLS) are probably due to limitations in the investigability of the patients, in particular the worse imaging of the entire musculature in the anterior wall region, as a good assessment of kinetics/strain in this region is very important for the prediction of LVT.

The small number of patients is an obvious limitation of this study. This is due to the time of recruitment of patients (COVID pandemic with the change in the structure of our department), the recruitment of patients in one cardiac centre, but also to the high rate of non-compliance – especially in the completion of magnetic resonance imaging (however, compliance parameters were not assessed in the protocol of our study). Other limitations of MRI included the relatively high incidence of claustrophobia (n=9) and poor evaluability of findings due to the presence of artifacts after defibrillator/pacemaker implantation (n=7). Subsequently, because of the newly identified claustrophobia in DE-CMR, even brain MRA could not be performed. In our cohort, one acute stroke only occurred, in a patient with LVT detected by DE-CMR (n=1/6). Furthermore, a relatively higher proportion of nonspecific (clinically asymptomatic) findings on the brain MRA can be observed in patients with LVT detected by TTE. It is questionable whether these findings are related to embolization due to the presence of LVT or are rather a manifestation of preexisting atherothrombotic changes in the CNS that merely reflect a generalized atherosclerotic involvement in a cardiovascularly high-risk group of patients after AMI. Because of the low prevalence of thromboembolic complications, it was not possible to demonstrate a statistically significant association between the detection of LVT and the occurrence of embolizations in the CNS. The low incidence of clinically manifest cardioembolization events may also be due to the duration of the patient follow-up (1 year), whereas the incidence of CNS embolization appears to increase positively and linearly with the duration of follow-up[9].

CONCLUSION

LVT formation is still a major complication of AMI even in the era of PCI therapy. Our results confirm the high prevalence of LVT in patients with anterior wall STEMI with apicoanterior kinetic impairment (RIA perfusion territory). TTE is an essential investigation for early detection of LVT with bedside examination (in the acute setting), and the results assist in stratification of patients who are most at risk of LVT formation. Identification of patients at increased risk of developing LVT should result in more frequent imaging examinations with higher sensitivity and specificity, i.e. contrast TTE or, possibly, DE-CMR, the gold standard for the diagnosis of LVT. All this is aimed at reducing the risk of SE (even clinically asymptomatic) by optimizing the antithrombotic therapy after AMI with the initiation of anticoagulation therapy.

There was no statistically significant association between LVT detection and the occurrence of SE detected on the brain MRA . There is a need for further research with a larger number of patients aimed at optimizing the management of antithrombotic (anticoagulation) therapy in the case of LVT detection, clarifying the role of NOACs in this indication, and establishing a screening algorithm for patients at increased risk of LVT formation.

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