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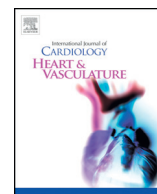
Incidence and predictors of left ventricular thrombus formation following acute ST-segment elevation myocardial infarction: A serial cardiac MRI study ☆

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ABSTRACT

Aims: Left ventricular (LV) thrombus is a complication of acute ST-segment elevation myocardial infarction (STEMI). We determined the incidence and predictors of LV thrombus formation using serial cardiac magnetic resonance (CMR) and two-dimensional echocardiography studies.
Methods and results: Two hundred and ten patients underwent CMR (median 4 days [IQR 3-7]) and transthoracic echocardiography (median 4 days [IQR 3-7]) early after STEMI presentation with serial follow-up CMR (median 55 days [IQR 46-64]) and echocardiography studies (median 54 days [IQR 45-64]) performed subsequently. The incidence of LV thrombus was 12.3% (26/210) by CMR and 6.2% (13/210) by two-dimensional echocardiography. Echocardiography had 50% sensitivity and 100% specificity for LV thrombus detection compared to CMR. LV thrombus was found in 23.6% of patients with anterior STEMI (22/93). Ischaemic stroke occurred in 1.4% of patients (3/210). Patients with LV thrombus had lower baseline LV ejection fraction (LVEF) (34.9% vs 47.4%, $p < 0.001$). Microvascular obstruction was more common in patients with LV thrombus (77% vs 39%, $p < 0.001$). Patients with LV thrombus had increased LV dimensions with larger LV end-diastolic (19 ml [IQR 9-44] vs 6 ml [IQR -4-18], $p < 0.001$) and end-systolic volumes (10 ml [IQR 0–22] vs -4 ml [IQR -12-4], p < 0.001).
Conclusion: CMR increases the detection of LV thrombi which standard echocardiography may underestimate. Serial studies post-STEMI may improve detection of LV thrombus, which is more prevalent in patients with anterior infarction, moderate LV dysfunction and adverse LV remodelling. This subgroup of patients may represent a high-risk group for targeted serial screening with CMR.

1. Introduction

Thrombus formation in the left ventricle following ST-segment myocardial infarction (STEMI) is a serious complication which may result in ischaemic stroke and systemic thromboembolism [1]. In the pre-thrombolytic and thrombolytic eras, the reported incidence of left ventricular (LV) thrombus varied from 7 to 46% [2–4], with significant variability in the time of performance of imaging as well as the modality used. Patients with STEMI have historically had imaging of LV function either with contrast left ventriculography or echocardiography (including with contrast). However, cardiac magnetic resonance (CMR) imaging has emerged as a powerful multi-parametric imaging tool [5]. The incidence of LV thrombus reported in more recent studies is lower at 3.5–8%, as detected by CMR, in patients treated with primary percutaneous coronary intervention (PCI) [6,7]. These studies may have underestimated the incidence of LV thrombus as they examined LV thrombus at a single early time point, post STEMI [8].

Our primary aim was to determine the incidence and predictors of LV thrombus after STEMI in the modern era of treatment by primary PCI or early angiography after thrombolytic therapy. We additionally

determined rates of stroke and systemic thromboembolism, as well as examined the utility of paired CMR examinations, performed acutely after STEMI and after 8 weeks. We also compared the sensitivity of two-dimensional transthoracic echocardiography for LV thrombus detection with CMR.

2. Methods

2.1. Study population

We prospectively recruited consecutive STEMI patients treated at our tertiary referral centre; STEMI patients presenting directly to Liverpool Hospital were treated with primary PCI, while patients presenting to other referring hospitals received thrombolytic therapy with either rescue PCI or PCI following successful reperfusion at Liverpool Hospital. This cohort comprised of patients who were enrolled in a STEMI study, results of which have been previously reported [9,10]. The diagnosis of STEMI required ischaemic symptoms lasting longer than 20 min, with ST-segment elevation in 2 or more contiguous leads on standard 12 lead ECG and a characteristic high sensitivity troponin T kinetic profile [11]. Our study consisted primarily of patients with first presentation STEMI ($n = 191$), with <10% who had a prior history of myocardial infarction (n = 19) as has been reported previously [9,10]. Patients underwent paired CMR studies to examine scar size but were additionally evaluated for the presence of LV thrombus. The study was approved by the Human Research Ethics Committee at Concord Hospital, Sydney, Australia (HREC/11/CRGH/224; approval CH62/6/2011-151) and all patients provided written informed consent.

Exclusion criteria included significant chronic kidney disease (eGFR < 30 ml/min/1.73 m^2 or renal replacement therapy), previous cardiac surgery, known cardiomyopathy, previous atrial fibrillation, significant psychiatric illness, age <18 years or > 85 years and contra-indications to CMR (claustrophobia, gadolinium allergy and ferrous metallic implants).

2.2. Demographic and clinical data

All patients had detailed demographic data recorded including age, sex, cardiovascular risk factors and discharge medications. Serial high sensitivity troponin T (Roche Diagnostics, Mannheim, Germany) levels were sampled at admission and at 24-hour intervals until 72 h as previously described [9,10]. All patients were treated with dual antiplatelet therapy.

2.3. CMR acquisition and analysis

Details of our CMR protocols have previously been described [10]. Briefly, patients underwent paired baseline and follow-up CMR studies on a commercially available 1.5T MRI scanner (Siemens Symphony, Germany). A standard multi-sequence protocol was used, with sequences done during breath-hold. A 6-channel body array and spine coil were used. Retrospective vector ECG gating was used for cardiac synchronisation. Cine images, using a steady-state free precession sequence, were obtained in standard views. Late gadolinium enhancement (8–10 min) sequences were obtained after a bolus injection of 0.10 mmol/kg gadoteric acid (Dotarem, Guerbet, France).

2.4. LV thrombus detection

LV thrombus was detected on CMR examinations as a mass within the LV cavity, with avascular tissue properties on post-contrast imaging as previously published [6,12]. Avascular tissues were selectively nulled in order to distinguish thrombus from surrounding high density tissues, such as blood and LV myocardium [6]. LV thrombus was differentiated

from microvascular obstruction based on an intra-cavitary location, and lack of contrast fill-in on delayed enhancement.

2.5. Echocardiography

Paired two-dimensional echocardiographic studies using standard views were performed post-myocardial infarction, at similar time points as the CMR. Left ventricular thrombus assessment was not routinely specified on echocardiography requests and hence, echocardiographic contrast agents were not routinely used. All echocardiograms were reviewed independently by a clinician who was blinded to the CMR results.

LV thrombus was detected on echocardiographic studies as a discrete mass in the left ventricle, distinct from the LV endocardium, in an area with corresponding LV regional or global wall motion abnormality [13].

2.6. Late clinical outcomes

Stroke and systemic thromboembolism outcomes were based upon review of hospitalisation records and imaging studies at our institution. Patients were also followed up by phone at three and six months with a standardised questionnaire, including questions about symptoms and hospitalisations for stroke or systemic embolism.

2.7. Statistical analysis

Statistical analyses were performed using SPSS version 24.0 (IBM SPSS, IBM Corporation, Armonk, NY). Continuous data with normal distribution are presented as mean and standard deviation. Continuous data with non-normal distribution are presented as median and inter-quartile range. Categorical data are presented as frequencies and percentages. Statistical comparison between groups was performed using Student's t-test (normal distribution), Mann-Whitney's U test (non-normally distributed data) and Chi-squared tests (categorical data). Logistic regression using a stepwise model was used to determine predictors of LV thrombus. Variables were selected based on significance on univariate analysis. Receiver-operator characteristic curves were used to assess the performance of test parameters in predicting LV thrombus. All tests were 2-tailed, and p-values $\ll 0.05$ were considered statistically significant.

3. Results

During the study period, 409 consecutive STEMI patients were screened from May 2012 to June 2014, of which 265 were enrolled in our study (72 declined participation, 28 could not consent due to language barriers, 15 died prior to consent, 12 had chronic renal failure, 5 had prior cardiothoracic surgery and 12 were excluded for various reasons). A further 29 patients could not undergo CMR, 12 withdrew consent, 8 did not have paired CMR data and 6 were lost to follow-up (see Appendix 1); resulting in 210 patients being included in this paired CMR study. The clinical characteristics are shown in Table 1.

Patients underwent CMR studies at a median of 4 days (IQR 3-7) and 55 days (IQR 46-64) post-STEMI. Baseline and follow up echocardiograms were performed at a median of 4 days (IQR 3-7) and 54 days (IQR 46-64). The incidence of LV thrombus by CMR was 12.3% (26/210). The incidence of LV thrombus by two-dimensional echocardiography was only 6.2% (13/210). Two-dimensional echocardiography detected LV thrombus with 50% sensitivity and 100% specificity when compared with CMR; all patients who had thrombus detected on echocardiography had a thrombus visualised on CMR. In 22 of 26 (85%) patients, LV thrombus was identified on the baseline CMR, whereas in 4

Table 1
Baseline characteristics.

	Overall (n = 210)	LV Thrombus + (n = 26)	LV Thrombus - (n = 184)	p
Male sex, n (%)	179 (85)	22 (85)	157 (84)	0.92
Hypertension, n (%)	99 (47)	15 (58)	84 (46)	0.25
Hypercholesterolaemia, n (%)	96 (46)	13 (50)	83 (45)	0.64
Diabetes mellitus, n (%)	41 (20)	5 (19)	36 (20)	0.97
Smoker, n (%)	121 (58)	14 (54)	107 (58)	0.66
Family history of CAD, n (%)	52 (25)	5 (19)	47 (26)	0.49
First MI, n (%)	191 (91)	21 (81)	170 (92)	0.053
Beta-blocker, n (%)	198 (94)	26 (100)	172 (93)	0.2
ACE inhibitor/angiotensin receptor blocker, n (%)	174 (83)	19 (73)	155 (84)	0.16
Statin, n (%)	205 (98)	24 (92)	181 (98)	0.058
Loop diuretic, n (%)	15 (7)	6 (23)	9 (5)	0.001
Mineralocorticoid antagonist, n (%)	11 (5)	6 (23)	5 (3)	<0.001
DAPT, n (%)	210 (100%)	26 (100%)	184 (100%)	ns
Anterior STEMI, n (%)	115 (55)	22 (85)	93 (51)	0.001
Primary PCI, n (%)	168 (80)	23 (88)	145 (79)	ns
Successful Thrombolysis, n (%)	27 (13)	2 (8)	25 (14)	ns
Rescue PCI, n (%)	15 (7)	1 (4)	14 (8)	ns

CAD = coronary artery disease.
DAPT = dual antiplatelet therapy.
STEMI = ST-segment myocardial infarction.
ACE = angiotensin-converting enzyme.
PCI = percutaneous coronary intervention.
ns = not significant.

(15%), LV thrombus was only detected on their follow-up CMR. The typical CMR appearance of LV thrombus is shown in Fig. 1.

In our cohort with LV thrombus 22/26 (85%) of patients had anterior STEMI. LV thrombus was detected in 23.6% of patients with anterior STEMI (22/93). In the remaining 4 patients, 1 patient presented with an inferior territory STEMI, but had prior anterior territory myocardial infarction and had a new apically located thrombus. The other 3 patients had inferior territory STEMI with infero-apical wall motion abnormalities with apically located LV thrombi. Patients with and without LV thrombus did not differ with respect to gender, cardiovascular risk factors or treatment modality.

All patients were treated with dual antiplatelet therapy. The rate of beta-blocker, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and statins did not differ between the groups. Patients with LV thrombus were more likely to be treated with loop diuretics (23% vs 5%, $p = 0.001$) and mineralocorticoid antagonists (23% vs 3%, $p < 0.001$). The frequency of LV thrombus in patients treated with primary PCI was 13.7% (23/168) in the primary PCI group and 7.1% (3/42) in those receiving thrombolytic therapy, $p = 0.3$. There was no difference in symptom onset to reperfusion time in patients with LV thrombus (Table 2).

The infarct and imaging characteristics by CMR are shown in Table 2. Patients with LV thrombus had larger infarcts as assessed by 72-hour high sensitivity troponin T levels (3691 ng/l [IQR 2634-5694] compared to those without LV thrombus, 2159 ng/l [IQR 1111–3134]; $p = 0.006$). The CMR parameters of patients with LV thrombus were similar for both baseline and follow-up CMR studies. Patients with LV thrombus tended to have higher LV end-diastolic and end-systolic dimensions, as well as lower LV ejection fraction and stroke volume (Table 2). LV thrombus patients also tended to have larger infarct sizes as measured by the delayed gadolinium enhancement LV percentage. Microvascular obstruction (MVO) was more common in patients with LV thrombus (77% vs 39%, $p < 0.001$). The extent of microvascular obstruction was not statistically different between patients with and without LV thrombus (0.33% vs 0.81%, $p = 0.2$).

Patients with LV thrombus, compared to those without LV thrombus, were more likely to have adverse remodelling characteristics with larger increases in left ventricular end-diastolic as assessed by paired CMR, of 19 ml [IQR 9-44] compared to 6 ml [IQR -4-18], $p < 0.001$, and on paired echocardiography of 10 ml [IQR 1-23] compared to 3 ml [IQR -4-13], $p = 0.013$); similar changes occurred in end-systolic volumes (on CMR 10 ml [IQR 0-22] and -4 ml [IQR -12-4], $p < 0.001$) and on echocardiography 6 ml [−8–4] compared to -2 ml [−4–6], $p < 0.001$. Left ventricular ejection fraction changes in patients with, and without, LV thrombus on CMR were 2.7% (IQR -1.4–6.7%) compared to 5.0% (IQR 1.4–7.8%); $p < 0.02$ and on echocardiography were 0.7% (IQR -2.9-3.2%) compared to 4.5% (IQR 1.2–9.0%); $p = 0.006$.
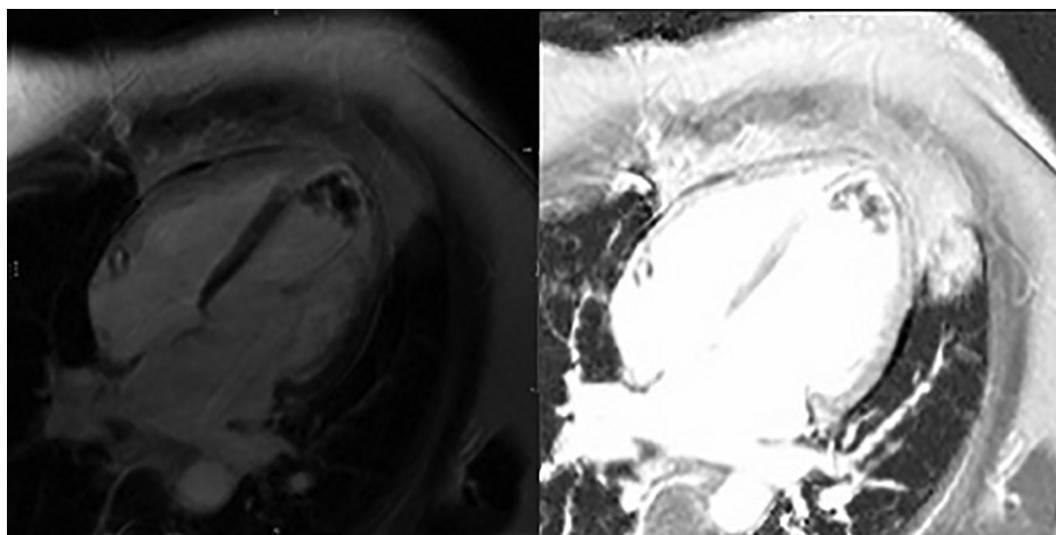
Fig. 1. Typical CMR appearance of LV thrombus in a patient presenting with an anterior territory STEMI, on delayed gadolinium enhancement (left); and early gadolinium enhancement (right).

Table 2
Clinical and imaging infarct characteristics.

	Overall (n = 210)	Thrombus + (n = 26)	Thrombus - (n = 184)	p
72-hour hsTnT (ng/l)	2306 [1192–3456]	3691 [2634–5694]	2159 [1111–3134]	0.006
Symptom onset to reperfusion time (min)	219 [142–367]	236 [151–505]	217 [136–359]	0.41
CMR 1 LVEDV (ml)	158 [132–179]	177 [157–205]	155 [129–179]	0.001
CMR 1 LVESV (ml)	86 [66–104]	116 [102–140]	82 [63–98]	<0.001
CMR 1 LVEF (%)	45.8 (9.7)	34.9 (7.5)	47.4 (8.9)	<0.001
CMR 1 SV (ml)	68 [58–84]	60 [53–68]	70 [60–85]	0.002
CMR 1 LV mass (g)	130 [116–155]	129 [118–160]	130 [116–154]	0.68
CMR 1 LV percentage DGE (%)	19.3 [12.2–27.8]	34 [26.6–44.3]	18 [11.9–25.7]	<0.001
CMR 1 Microvascular obstruction present, n (%)	92 (44)	20 (77)	72 (39)	<0.001
CMR 2 LVEDV (ml)	167 [135–190]	203 [175–234]	161 [133–184]	<0.001
CMR 2 LVESV (ml)	77 [57–106]	125 [115–160]	73 [56–94]	<0.001
CMR 2 LVEF (%)	50 (11)	37.0 (9.5)	52.0 (10.0)	<0.001
CMR 2 SV (ml)	79 [68–93]	70 [59–86]	80 [70–93]	0.03
CMR 2 LV mass (g)	123 [108–143]	122 [104–153]	125 [109–141]	0.97
CMR 2 LV percentage DGE (%)	16.1 [10.1–24.4]	29.2 [24.7–37.3]	14.5 [9.6–20.7]	<0.001
LVEF change (%)	4.6 [1.2–7.6]	2.7 [−1.4–6.7]	5.0 [1.4–7.8]	<0.02
LVEDV change (ml)	7.8 [−2.6–20.0]	19 [9–44]	6 [−4–18]	<0.001
LVESV change (ml)	−2.4 [−11.6–6.5]	10 [0–22]	−4 [−12–4]	<0.001

hsTnT = high sensitivity troponin T.
LVEDV = left ventricular end-diastolic volume.
LVESV = left ventricular end-systolic volume.
LVEF = left ventricular ejection fraction.
SV = stroke volume.
DGE = delayed gadolinium enhancement.
CMR 1 = baseline study.
CMR 2 = follow up study.

All patients with LV thrombus were treated with oral anticoagulation with warfarin. Four of 22 patients with LV thrombus detected on their baseline CMR had persistent LV thrombus on follow-up. The rate of ischaemic stroke was 1.4% (3/210) in the total cohort, and 2 of the 3 patients with ischaemic stroke had documented LV thrombus. In the patient with ischaemic stroke and no LV thrombus, no other cardioembolic source was identified. One patient with stroke had concomitant left atrial and left ventricular thrombus.
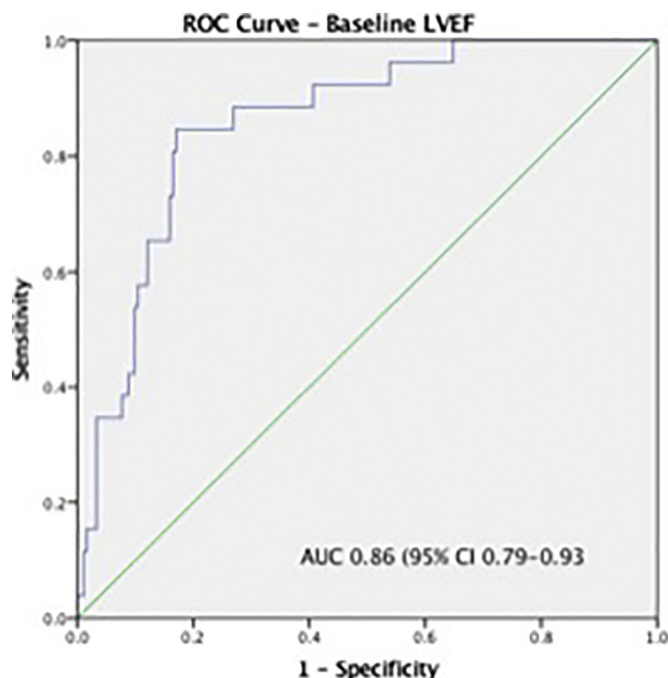
Fig. 2. Receiver-operator curve for baseline LVEF as a predictor of LV thrombus.

One patient suffered a haemorrhagic stroke requiring surgical decompression.

High sensitivity troponin T levels, baseline left ventricular ejection fraction and MVO were found to be significant univariate predictors for the development of LV thrombus after STEMI ($p < 0.05$). Logistic regression was performed using these variables. The model was statistically significant, $\chi^2(3) = 9.7$, $p = 0.02$ and explained 21% of variance in LV thrombus (Nagelkerke R^2) and correctly classified 87% of cases. The receiver-operator curve for baseline LVEF is displayed in Fig. 2. The area under the curve (AUC for baseline LVEF was found to be 0.86 (0.79–0.93). A baseline LVEF cut-off of 40% identified LV thrombus with 85% sensitivity and 79% specificity.

4. Discussion

In this cohort study, the incidence of LV thrombus predominantly detected by CMR was 12%, among patients with STEMI of whom 80% were treated by primary PCI. Echocardiography detected LV thrombus commonly. Approximately 1 in 4 patients with anterior STEMI had LV thrombus detected. The incidence of LV thrombus in the present study is slightly higher than recently reported rates of 3.5–8% by CMR [6,7], but is significantly lower than historical studies from the pre-thrombolysis era [4]. The slight difference between studies may be due to relatively small sample size, as the clinical characteristics of our patient cohorts (e.g. proportion of anterior STEMI) are similar [6,7]. One large retrospective study of 2071 patients found an incidence of left ventricular thrombus of 1.5% following STEMI, as detected by paired echocardiography within the first week [14]. The significant lower rate of LV thrombus in this study may be explained by the under-detection of left ventricular thrombi by routine echocardiography, serial imaging was performed within one week of the infarct, and that high-risk patients in this cohort (patients with LVEF < 35% or with apical akinesis) were empirically treated with therapeutic anticoagulation. This practice is not currently routinely recommended and requires further study.

The addition of serial CMR imaging at 1–2 months after presentation with STEMI slightly increased the number of LV thrombi detected (2% of

patients in our cohort). Nevertheless, given the potentially devastating outcome of stroke or systemic thromboembolism, certain subsets of high-risk patients may benefit from further examination. Our study found that anterior territory STEMI, significant LV dysfunction and the presence of microvascular obstruction to be the salient risk factors for development of LV thrombus. Thus, patients with a baseline LVEF ≤ 40% and anterior infarct location, could undergo both a baseline and follow up CMR study to detect later LV thrombus formation. Indeed, based on the high frequency (roughly one quarter) of detectable thrombi in anterior MI, it could be argued that CMR should be routine in patients with anterior STEMI. Furthermore, adverse LV remodelling as defined by larger increases in LVEDV and LVESV, and smaller increases in LVEF appear to more common in patients with LV thrombus, demonstrated by both CMR and echocardiographic measurements in our study. This has been previously described in other small studies [15,16], and may suggest a 'gatekeeper role' for echocardiography in stratifying patients who may benefit from serial CMR imaging.

Two-dimensional echocardiography underestimates the incidence of LV thrombus when compared with CMR. One study compared the detection rate for LV thrombus between two-dimensional echocardiography and CMR in 243 patients with LV systolic dysfunction and found that non-contrast echocardiography had a sensitivity of 33% and specificity of 91%, as compared to CMR [17]. Our study did not examine contrast echocardiography for the detection of LV thrombus; however, a previous report showed that contrast echocardiography improves LV thrombus detection compared to non-contrast echocardiography, but that the detection rates remains below CMR [18]. Overall, CMR may offer a more comprehensive multi-parametric assessment post-MI, including identification of other post-MI complications.

The overall rate of ischaemic stroke and systemic embolism in our cohort was 1.4% (3/210). The rate of clinical ischaemic stroke or systemic embolism among patients with documented LV thrombus was 7.7% (2/26), which is relatively low. A 1993 meta-analysis of 11 studies and 856 patients found that the risk of thromboembolism in patients with LV thrombus was comparable at 11% [19]. This suggests that thromboembolism consequent to LV thrombus continues to be a problem in the current era including the use of dual antiplatelet therapy in STEMI patients. A recent study of 142 primary PCI-treated STEMI patients found an overall LV thrombus rate of 8.5%, as detected by CMR. In this study, all patients were treated with dual antiplatelet therapy, with warfarin being added at the treating physician's discretion. There were no clinical thromboembolic events occurring over a 2-year follow-up period in both warfarin treated and untreated patients [15].

The issue of routine oral anticoagulation in those at high risk post-STEMI has been contentious, despite the ATLAS ACS 2-TIMI 51 trial [20] showing benefit of low dose rivaroxaban combined with DAPT, and this combination of agents has not been widely adopted. Our data suggests that specifically patients with anterior STEMI and LV dysfunction, should be evaluated in a prospective multicentre trial, though large numbers may need to be screened, given our low systemic embolism rate.

5. Study limitations

Whilst this was a single centre study and may not necessarily be representative of patient populations at other centres, its strength is the performance of paired early and convalescent CMR and echocardiography studies. Our study did not employ the routine use of echocardiographic contrast, which may have led to an improved LV thrombus detection rate by echocardiography. Furthermore, left ventricular thrombus assessment was not routinely specified especially for the second echo/CMR study as the clinical indication, which has previously been shown to improve sensitivity for thrombus detection [17]. CMR, however, has been shown to outperform both contrast and non-

contrast echocardiography for the detection of LV thrombus [18]. Inflammation, as defined by the C-reactive protein (CRP), has been shown in small studies to predict the development of left ventricular thrombus following STEMI [21]. Serum CRP levels were not collected during our study and may have helped further identify high risk patients.

The rate of stroke or systemic thromboembolism in our study may have been higher, as events may be undetected if patients presented to another hospital. Liverpool Hospital is the tertiary referral site for PCI, with patients otherwise followed up at their respective referral hospitals. Some high-risk patients with significant renal dysfunction (eGFR < 30 ml/min/1.73 m^2 or renal replacement therapy), gadolinium allergy and ferrous magnetic implants were excluded from our study, which is a limitation of all contrast CMR studies. This may have led to underestimation of the rate of LV thrombi among all-comers.

6. Conclusion

CMR examination after acute myocardial infarction increases the detection rate of left ventricular thrombus formation. Two-dimensional echocardiography without contrast underdiagnoses and significantly underestimates the incidence of LV thrombus compared to CMR. Performance of paired CMR permits the detection of late LV thrombus, albeit in a small percentage of patients. The risk of stroke and systemic thromboembolism in a contemporary cohort are largely similar to historical studies. Anterior territory STEMI, LV dysfunction, the presence of microvascular obstruction and adverse LV remodelling are more prevalent in patients with LV thrombi, and may represent a high-risk cohort for targeted screening with paired CMR.

Declaration of Competing Interest

Acknowledgements

Appendix 1. CONSORT diagram of study population.CKD = chronic kidney disease

Eligible STEMI patients screened
(n = 409)

Excluded (n = 144)
72 declined participation
28 non-English speaking
15 died prior to consent
12 CKD (eGFR < 30mL/min/1.73m^2)
5 prior cardiothoracic surgery
12 other causes

Enrolled patients (n = 265)

Excluded (n = 55)
29 no CMR
12 withdrew consent
8 no paired CMR data
6 lost to follow up

Study cohort (n =210)
Baseline and follow up CMR
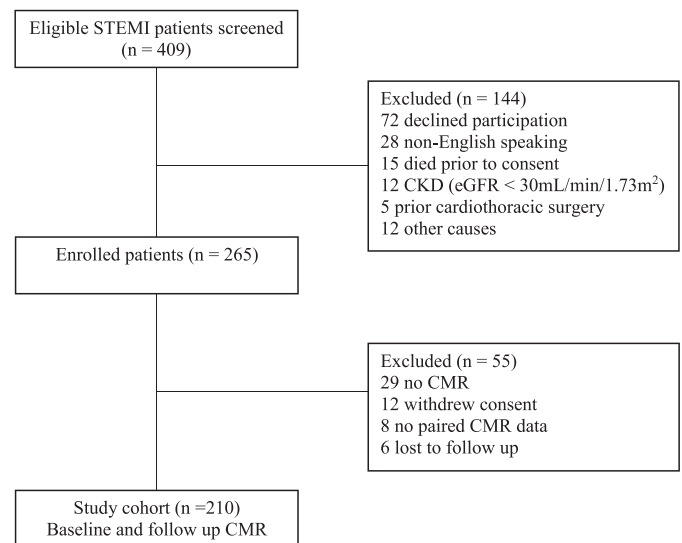
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