European Journal of Radiology 81 (2012) 3900–3904

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

Left ventricular thrombus formation after acute myocardial infarction as assessed by cardiovascular magnetic resonance imaging

Ronak Delewi[a,b], Robin Nijveldt[c], Alexander Hirsch[a], Constantin B. Marcu[c], Lourens Robbers[c], Marriela E.C.J. Hassell[a], Rianne H.A. de Bruin[a], Jim Vleugels[a], Anja M. van der Laan[a], Berto J. Bouma[a], René A. Tio[d], Jan G.P. Tijssen[a], Albert C. van Rossum[c], Felix Zijlstra[e], Jan J. Piek[a,*]

[a] Department of Cardiology, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands
[b] Interuniversity Cardiology Institute of the Netherlands, The Netherlands
[c] Department of Cardiology, VU University Medical Center, Amsterdam, The Netherlands
[d] Thorax Center, University Medical Center Groningen, Groningen, The Netherlands
[e] Thorax Center, Department of Cardiology, Erasmus University Medical Center, Rotterdam, The Netherlands

ARTICLE INFO

Article history:
Received 17 April 2012
Received in revised form 21 June 2012
Accepted 22 June 2012

Keywords:
LV thrombus
Myocardial infarction
Cardiovascular magnetic resonance imaging

ABSTRACT

Introduction: Left ventricular (LV) thrombus formation is a feared complication of myocardial infarction (MI). We assessed the prevalence of LV thrombus in ST-segment elevated MI patients treated with percutaneous coronary intervention (PCI) and compared the diagnostic accuracy of transthoracic echocardiography (TTE) to cardiovascular magnetic resonance imaging (CMR). Also, we evaluated the course of LV thrombi in the modern era of primary PCI.

Methods: 200 patients with primary PCI underwent TTE and CMR, at baseline and at 4 months follow-up. Studies were analyzed by two blinded examiners. Patients were seen at 1, 4, 12, and 24 months for assessment of clinical status and adverse events.

Results: On CMR at baseline, a thrombus was found in 17 of 194 (8.8%) patients. LV thrombus resolution occurred in 15 patients. Two patients had persistence of LV thrombus on follow-up CMR. On CMR at four months, a thrombus was found in an additional 12 patients. In multivariate analysis, thrombus formation on baseline CMR was independently associated with, baseline infarct size (g) ($B = 0.02$, SE = 0.02, $p < 0.001$). Routine TTE had a sensitivity of 21–24% and a specificity of 95–98% compared to CMR for the detection of LV thrombi. Intra- and interobserver variation for detection of LV thrombus were lower for CMR ($\kappa = 0.91$ and $\kappa = 0.96$) compared to TTE ($\kappa = 0.74$ and $\kappa = 0.53$).

Conclusion: LV thrombus still occurs in a substantial amount of patients after PCI-treated MI, especially in larger infarct sizes. Routine TTE had a low sensitivity for the detection of LV thrombi and the interobserver variation of TTE was large.

1. Introduction

Mortality due to acute myocardial infarction (MI) has decreased since the introduction of primary percutaneous coronary intervention (PCI) [1]. Nevertheless, post infarct complications are still a major cause of morbidity and mortality. One of the most feared complications is the occurrence of thrombo-embolic events in patients with left ventricular (LV) thrombus [2,6]. To date, no randomized trials have evaluated the efficacy of anticoagulation therapy in preventing embolization in this group of patients. However, observational studies conducted in the pre-thrombolytic and thrombolytic era, provide support for long term anticoagulation with vitamin K antagonist [3–5]. This is reflected in the ESC and ACC/AHA guidelines recommending vitamin K antagonist therapy in patients with LV thrombus after MI for at least 3–6 months and even indefinitely in patients without increased risk of bleeding [7,8].

Nowadays, ST-segment elevated myocardial infarction (STEMI) patients undergo primary PCI and receive long term dual anti-platelet therapy (including aspirin and a P2Y12 inhibitor). Consequently, patients with LV thrombus or at increased risk of LV thrombus formation after an MI are frequently treated with vitamin K antagonist in addition to dual anti-platelet therapy and therefore subjected to an increased bleeding risk [9]. Adequate diagnosis and follow-up of LV thrombus formation is therefore essential.

We performed a study to address the following issues: (1) assess the prevalence of LV thrombus in patients after STEMI treated with primary PCI and dual anti-platelet therapy, (2) compare the

* Corresponding author at: Department of Cardiology, Academic Medical Center, University of Amsterdam, Meibergdreef 9, 1105AZ Amsterdam, The Netherlands. Tel.: +31 20 5669111; fax: +31 20 6962609.
E-mail address: j.j.piek@amc.uva.nl (J.J. Piek).

0720-048X
http://dx.doi.org/10.1016/j.ejrad.2012.06.029

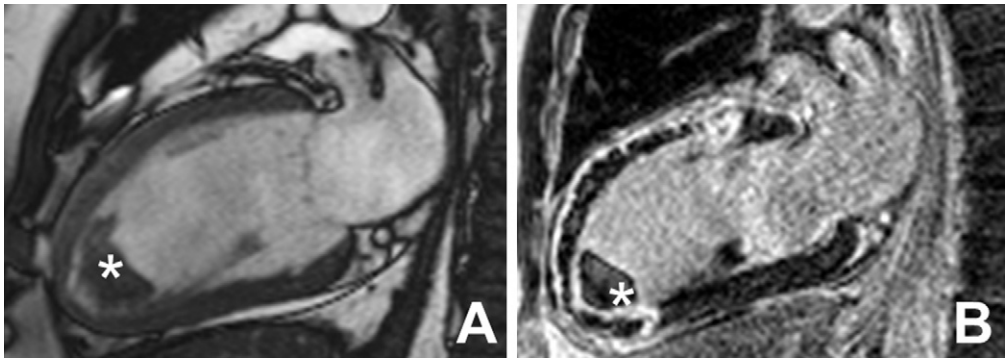

Fig. 1. Left ventricular thrombus formation on late gadolinium enhancement cardiac magnetic resonance imaging. (A) Cine cardiovascular magnetic resonance of a thrombus (asterisk) in the apex of the left ventricle. (B) Late gadolinium enhancement imaging neatly confirms the avascular non-enhancing thrombus (asterisk) close to the transmural infarcted myocardium with areas of microvascular obstruction. LV, left ventricular.

diagnostic accuracy of 2D-echocardiograpy vs. cardiovascular magnetic resonance (CMR), and (3) evaluate the course of LV thrombi in the modern era of primary PCI.

2. Methods

The present study is a substudy of the HEBE-trial, which evaluated the effects of injection of intracoronary bone-marrow cells [10]. A total of 200 patients presenting with a first STEMI was enrolled in this multicenter study involving 8 high volume primary PCI centers located in the Netherlands. All patients had undergone primary PCI with stent implantation within 12 h of symptom onset. Exclusion criteria were: unsuccessful PCI, hemodynamic instability, elevation of creatine kinase (CK) or CK-myocardial band <10 times the local laboratory upper limit of normal, and contraindications for CMR. The regional committees for medical research ethics approved the study protocol and all patients gave informed consent. All patients underwent transthoracic echocardiography (TTE) and CMR at baseline and at 4 months follow-up.

2.1. Cardiovascular magnetic resonance

CMR examinations were performed between 2 and 7 days and repeated at 4 months after primary PCI. The studies were performed on a clinical 1.5 T scanner as previously described [10]. Whole LV coverage was achieved on cine CMR with contiguous short axis slices using a segmented steady-state free-precession pulse sequence. Typical in-plane resolution was 1.6 mm × 1.9 mm, with slice thickness 5.0–6.0 mm (repetition time/echo time 3.2/1.6 ms, flip angle 60°, matrix 256 × 156, temporal resolution 35–60 ms). Late gadolinium enhancement (LGE) images were obtained to examine infarct size, 10–15 min after administration of a gadolinium-based contrast agent (Dotarem, Guerbet; 0.2 mmol/kg) using a 2D segmented inversion recovery gradient-echo pulse sequence, with slice position identical to the cine images. Typical in-plane resolution was 1.4 mm × 1.7 mm, with slice thickness 5.0–6.0 mm (repetition time/echo time = 9.6/4.4 ms, flip angle 25°, triggering to every other heartbeat). The CMR data was analyzed using a dedicated software package (Mass 2008beta, Leiden). Observers were blinded to echocardiographic results and patient identity. After endocardial and epicardial borders were outlined manually on short axis end-diastolic and end-systolic cine images, left ventricular end-systolic volume (LVESV), and left ventricular end-diastolic volume (LVEDV) were determined, after which left ventricular ejection fraction (LVEF) and myocardial mass were calculated. Microvascular obstruction (MVO) was defined as any region of hypoenhancement within the hyperenhanced infarcted area and was included in the calculation of total MI size.

LV thrombi on CMR were defined as filling defects within the LV cavity, typically adherent to regions of abnormal wall motion (hypokinesis, akinesis, or dyskinesis) on cine sequences and had to be confirmed by late gadolinium enhancement (see Fig. 1). Thrombi were carefully differentiated from regions of MVO which appear on LGE as dark areas within the core, and thus completely surrounded, by the hyper intense acute myocardial infarct. The presence or absence of LV thrombus on CMR was scored by two blinded, independent, experienced examiners, blinded to patient identity and TTE results. A consensus was reached in case of disagreement.

2.2. Transthoracic echocardiography

TTE was performed within one day of the CMR study. Parasternal, apical, and subcostal views were obtained, with special focus on the apical views, since thrombi are most frequent, although not exclusively, located at the apex. On TTE, thrombus was defined as a discrete echo dense structure in the LV cavity with well-defined margins, distinct from the endocardial border, visible in both systole and diastole in at least two views and located adjacent to a hypokinetic or akinetic area of the LV wall. TTE studies were independently analyzed from CMR results. The presence or absence of LV thrombus on echocardiography was scored by two blinded, independent, experienced examiners, blinded to patient identity, and CMR results. A consensus was reached in case of disagreement.

2.3. Follow-up

All patients were treated with a loading dose of clopidogrel 300 or 600 mg and thereafter 75 mg/day in addition to an initial loading dose of aspirin followed by 80–100 mg/day. Patients were treated with vitamin K antagonist at the discretion of the treating physician with a target international normalized ratio of 2.0–3.0 for ≥3–6 months. Patients were seen at the outpatient clinic at 1, 4, 12, and 24 months after primary PCI, for assessment of clinical status and adverse events. Stroke was defined as the new onset of focal or global neurological deficit caused by ischemia or hemorrhage assessed by appropriate imaging, e.g., computed tomography or magnetic resonance imaging, within or around the brain and lasting for more than 24 h or leading to death. Bleeding was defined according to the criteria of the thrombolysis in myocardial infarction trial. Major bleeding was defined as a decrease in the hemoglobin level of >5 g/dl, intracranial hemorrhage or a >15% absolute decrease in hematocrit, or cardiac tamponade. Minor bleeding was defined as a decrease in the hemoglobin level of 3 g/dl or >10% decrease in hematocrit from an identified site or: >4 g/dl decrease in the hemoglobin concentration or >12% decrease in hematocrit when

Table 1
Baseline demographics and characteristics stratified according to the early, late, or absence of left ventricular thrombus at cardiovascular magnetic resonance imaging (CMR) in patients after acute myocardial infarction.

Characteristic	Early LV thrombus			Late LV thrombus[c]		
	Presence ($n=17$)	Absence ($n=177$)	p-Value	Presence ($n=14$)	Absence ($n=176$)	p-Value
Age (years)	57 ± 8	56 ± 10	0.46	57 ± 10	56 ± 9	0.64
Male gender	14 (82%)	151 (85%)	0.75	13 (93%)	148 (84%)	0.70
Risk factors						
Diabetes mellitus	0 (0%)	12 (7%)	0.60	2 (14%)	10 (6%)	0.22
Known hypertension	4 (24%)	50 (28%)	0.78	4 (29%)	49 (28%)	1.00
Hypercholesterolemia	1 (6%)	37 (21%)	0.20	2 (14%)	36 (20%)	0.74
Current cigarette smoking	6 (35%)	95 (54%)	0.63	6 (43%)	94 (53%)	0.74
Angiography and infarct treatment						
Time from symptom onset to PCI (h)	2.8 (2.1–5.1)	3.4 (2.3–4.6)	0.88	2.9 (2.0–5.3)	3.3 (2.4–4.5)	0.83
Anterior infarction	17 (100%)	106 (60%)	<0.001	14 (100%)	107 (61%)	<0.001
Medication at discharge						
Aspirin	17 (100%)	170 (96%)	1.00	13 (93%)	170 (97%)	0.42
Clopidogrel	17 (100%)	177 (100%)	1.00	14 (100%)	175 (99%)	1.00
Coumarin derivate	8 (47%)	23 (13%)	0.001	1 (7%)	31 (18%)	0.47
Medication at 4 months follow-up						
Aspirin	17 (100%)	162 (92%)	0.63	13 (93%)	158 (90%)	1.00
Clopidogrel	17 (100%)	150 (85%)	0.48	14 (100%)	150 (85%)	0.22
Coumarin derivate	8 (47%)	27 (15%)	<0.001	2 (14%)	34 (19%)	1.00
CMR (day 2–7)						
Presence of microvascular obstruction[a]	15 (88%)	96 (56%)	0.01	12 (92%)	99 (57%)	0.02
LVEF (%)[a]	37 ± 10	43 ± 9	0.10	42 ± 6	43 ± 9	0.68
Infarct size (g)[b]	28 ± 12	22 ± 12	0.045	32 ± 8	22 ± 13	<0.001
CMR (4 months)						
LVEF (%)[a]	43 ± 9	47 ± 9	0.08	42 ± 9	47 ± 9	0.04
Infarct size (g)[b]	17 ± 9	14 ± 8	0.20	19 ± 7	14 ± 8	0.02

Data are number (%), mean ± SD or median (25th–75th percentile). LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention.
[a] The analysis included 189 patients.
[b] The analysis included 167 patients.
[c] 4 follow-up CMR were excluded from this analysis due to poor quality.

bleeding site is not identified or spontaneous gross hematuria, hematemesis, or hemoptysis. A clinical event committee independently adjudicated all potential clinical events.

2.4. Statistical analysis

Values are reported as mean ± SD or median (25th–75th percentile) for continuous variables and as frequency with percentage for categorical variables. Unpaired Student's t-test and a Fisher's exact test were used to compare differences between groups of continuous and categorical variables, respectively. To assess intraobserver variation, a kappa statistic was calculated for both CMR and TTE for diagnosing LV thrombus. For inter-observer variation, a kappa statistic was calculated from the results from the first examination. Covariates of interest as predictors of LV thrombus formation were investigated using multivariable logistic regression. Baseline variables that were significant at $p \leq 0.10$ on univariate analysis were entered into a multivariate model. Clinical events were presented for descriptive purposes only.

All statistical tests were 2-tailed, and a value of $p < 0.05$ was considered statistically significant. Calculations were generated by SPSS software (version 17.0 for Windows, SPSS, Inc., Chicago, Illinois).

3. Results

A total of 6 patients were excluded from this sub-analysis due to a non-evaluable CMR. In the remaining 194 patients, mean age was 56 ± 9 years, 85% of the patients were men, mean baseline LV ejection fraction was 42 ± 9% and 90% had TIMI flow grade 3 after primary PCI. CMR was performed 4 ± 2 days and TTE 3 ± 2 days after primary PCI. During the 24-month of clinical observation, 10 patients were lost to follow-up (all without LV thrombus on baseline CMR).

3.1. Incidence of LV thrombus at baseline and at follow-up

On CMR at baseline, a thrombus was found in 17 of 194 (8.8%) patients. LV thrombus resolution occurred in 15 patients. Two patients had persistence of LV thrombus on follow-up CMR. On CMR at four months, a thrombus was found in an additional 12 patients, making a total of 14 of 190 (7.4%) patients with LV thrombus (see Fig. 2). Clinical, angiographic characteristics and CMR results stratified according to the absence or presence of LV thrombus at baseline and at 4 months are described in Table 1. All LV thrombi were seen in patients with anterior infarctions. Patients with both early and late LV thrombus had more often microvascular obstruction on baseline CMR and larger infarct sizes than patients without LV thrombus formation (see Table 1) In multivariate analysis, thrombus formation on baseline LGE was independently associated with, baseline infarct size (g) ($B = 0.02$, SE = 0.02, $p < 0.001$) and anterior infarction ($B = 19.47$, SE 4900, $p < 0.001$). At 4 months, LV thrombus formation was also independently associated with, baseline infarct size (g) ($B = 0.06$, SE = 0.02, $p < 0.001$).

3.2. CMR vs. TTE for the detection of LV thrombus

Of the 17 thrombi visualized on CMR, 4 were visible also on TTE. In 9 patients, TTE suggested an apical thrombus, which could not be confirmed on CMR. Sensitivity and specificity of baseline TTE compared to baseline CMR was respectively 21–24% and 95–98% (see Table 2). CMR intra- and interobserver variation for the detection of LV thrombus was κ 0.91 and κ 0.96 respectively. TTE intra- and interobserver variation for the detection of LV thrombus was κ 0.74 and κ 0.53 respectively.

3.3. Clinical course of LV thrombi

In 8 of the 17 patients with LV thrombus at baseline (Fig. 2), the treating physician started vitamin K antagonist on top of aspirin

Table 2
Sensitivity, specificity, and predictive value of transthoracic echocardiography (TTE) compared to cardiovascular magnetic resonance (CMR).

TTE (baseline)	CMR (baseline)	
	Presence	Absence
Presence	4	5
Absence	13	172
Sensitivity = 24%		
Specificity = 95%		
TTE (4 months)	CMR (4 months)	
	Presence	Absence
Presence	3	4
Absence	11	172
Sensitivity = 21%		
Specificity = 98%		

CMR, cardiovascular magnetic resonance imaging; TTE, transthoracic echocardiography.

and clopidogrel. The other 9 patients were treated with dual anti-platelet therapy. In 15 of these 17 patients (vitamin K antagonist therapy: $n = 7$, no vitamin K antagonist therapy: $n = 8$), no thrombus was present at 4 months follow-up (see Fig. 2). Of the two patients with persisting LV thrombus, one was treated with vitamin K antagonist therapy. In an additional 12 patients (6.2%) LV thrombus was absent at baseline CMR but occurred at 4 months follow-up. Four of these patients were on triple-antithrombotic therapy for other indications; eight patients were on dual anti-platelet therapy.

3.4. Clinical events

One of the 194 patients had clinical evidence of ischemic stroke, however this patient did not have evidence of LV thrombus formation on either CMR or TTE or any other known risk factors for systemic embolism. In three other patients, gastro-intestinal bleedings were documented; all of them received triple anti-thrombotic therapy.

4. Discussion

Our study demonstrates an LV thrombus prevalence of 8.8% as assessed by CMR among a population of patients with a first large STEMI treated by primary PCI (mean LVEF 42 ± 9%). Routine TTE had a sensitivity of 21–24% and a specificity of 95–98% compared to CMR for the detection of LV thrombi and the interobserver variation of TTE was large ($\kappa = 0.53$). Finally, LV thrombi documented in the modern era of primary PCI and its concomitant dual anti-platelet therapy, appear to have a relatively benign course, irrespective of anti-thrombotic therapy.

4.1. Left ventricular thrombus and incidence

Early data from the pre- and thrombolytic era suggest that in the setting of acute MI, LV thrombus may be present in 7–46% of patients, most frequently in acute anterior or apical myocardial infarction [11–13]. In the era of primary PCI and dual anti-platelet therapy, there is limited data concerning the frequency of LV thrombus in AMI. In our study, the incidence of LV thrombus was 8.8% in a selected group of first STEMI patients treated by primary PCI as detected on LGE-CMR. Solheim et al. [14], reported a higher incidence of 15% in the first 3 months, Zielinska et al. [15], found LV thrombi formation in 68 of 1251 patients (5.4%) with acute anterior wall myocardial infarctions and only in 0.3% in those with non-anterior wall MI. Patients with LV thrombus had larger infarct sizes and were more often anterior MI, confirming earlier observations [16,17].

4.2. Left ventricular thrombus and diagnosis

Among the 17 patients with LV thrombus identified by CMR in the current study, 4 were visible also on TTE. Sensitivity and specificity of baseline TTE compared to baseline CMR was

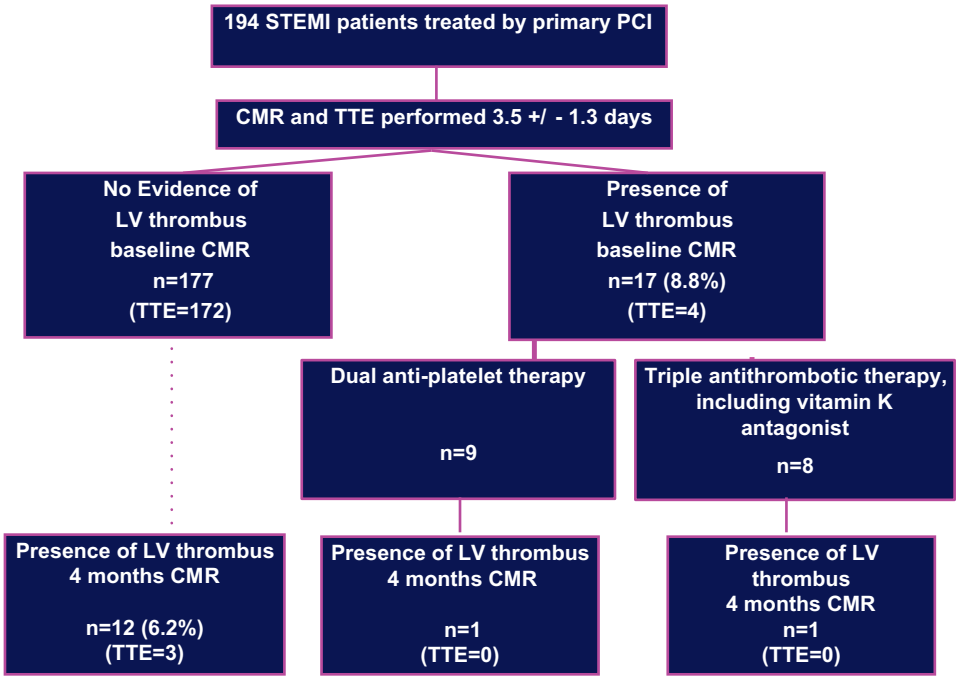

Fig. 2. The course of left ventricular thrombus formation. CMR, cardiovascular magnetic resonance imaging; LV, left ventricular; PCI, percutaneous coronary intervention; TTE, transthoracic echocardiography; STEMI, ST-segment elevated myocardial infarction.

respectively 21–24% and 97–98%. No independent gold-standard (e.g., histopathology or surgery) was reachable to determine the better diagnostics accuracy. However, this discrepancy has been reported in two prior studies. One study compared CMR and LGE with echocardiography in a cohort of patients undergoing LV reconstruction surgery in whom surgical and/or pathology verification of thrombus was uniformly performed [20]. This study reported that the sensitivity of TTE was 40%, compared with 88% for CMR, showing the superiority of CMR. Another study reported an echo sensitivity and specificity of 33% and 91% in a heterogeneous population of patients with LV systolic dysfunction [16]. Intravenous echo contrast during TTE may improve the diagnostic assessment of LV thrombus [18,19]. However, only one compound (SonoVue, Bracco, Milan, Italy) is approved in the Netherlands and its use is contraindicated by the European Medicines Agency in cardiac patients with acute coronary syndrome, recent percutaneous coronary intervention, acute or chronic severe heart failure or severe cardiac arrhythmias.

4.3. Left ventricular thrombus and management

Previous studies performed in the pre-thrombolytic and thrombolytic era reported varying data concerning the risk of future embolic events [3–6]. A 1992 meta-analysis included 11 studies of 856 patients who had an anterior MI; the odds ratio for an embolic event was 5.5 (95% CI 3.0–9.8) [3]. In clinically stable patients with chronic heart failure, the risk of thromboembolism seems low (1–3% per year), even in those with very impaired LV function and echocardiographic evidence of intracardiac thrombi [21–23]. Our analysis also indicates that documented LV thrombus (by CMR or TTE) is accompanied by a relative benign course since none of these patients, irrespective of type of anti-thrombotic regimen, experienced a major thrombo-embolic event up to 2 years after myocardial infarction.

Whether management of LV thrombus requires vitamin K antagonist therapy on top of dual anti-platelet therapy cannot be concluded from this observational study and requires the conduction of a randomized trial in acute MI patients with LV thrombus.

Disclosures

None of the authors have any financial interest to disclose with respect to this study.

Fundings

This work was supported by a grant by the Dutch Heart Foundation and National Health Insurance Board/ZON MW, the Netherlands to RD.

References

[1] Rogers WJ, Canto JG, Lambrew CT, et al. Temporal trends in the treatment of over 1.5 million patients with myocardial infarction in the US from 1990 through 1999: the National Registry of Myocardial Infarction 1, 2 and 3. Journal of the American College of Cardiology 2000;36:2056–63.
[2] Stratton JR, Resnick AD. Increased embolic risk in patients with left ventricular thrombi. Circulation 1987;75:1004–11.
[3] Cregler LL. Antithrombotic therapy in left ventricular thrombosis and systemic embolism. American Heart Journal 1992;123:1110–4.
[4] Weinreich DJ, Burke JF, Pauletto FJ. Left ventricular mural thrombi complicating acute myocardial infarction. Long-term follow-up with serial echocardiography. Annals of Internal Medicine 1984;100:789–94.
[5] Keating EC, Gross SA, Schlamowitz RA, et al. Mural thrombi in myocardial infarctions. Prospective evaluation by two-dimensional echocardiography. American Journal of Medicine 1983;74:989–95.
[6] Vaitkus PT, Barnathan ES. Embolic potential, prevention and management of mural thrombus complicating anterior myocardial infarction: a meta-analysis. Journal of the American College of Cardiology 1993;22:1004–9.
[7] Van de Werf F, Bax J, Betriu A, et al. Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. European Heart Journal 2008;29:2909–45.
[8] Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). Circulation 2004;110:588–636.
[9] Sorensen R, Hansen ML, Abildstrom SZ, et al. Risk of bleeding in patients with acute myocardial infarction treated with different combinations of aspirin, clopidogrel, and vitamin K antagonists in Denmark: a retrospective analysis of nationwide registry data. Lancet 2009;374:1967–74.
[10] Hirsch A, Nijveldt R, van der Vleuten P, et al. Intracoronary infusion of mononuclear cells from bone marrow or peripheral blood compared with standard therapy in patients after acute myocardial infarction treated by primary percutaneous coronary intervention: results of the randomized controlled HEBE trial. European Heart Journal 2010;32:1736–47.
[11] Nihoyannopoulos P, Smith GC, Maseri A, et al. The natural history of left ventricular thrombus in myocardial infarction: a rationale in support of masterly inactivity. Journal of the American College of Cardiology 1989;14:903–11.
[12] Jugdutt BI, Sivaram CA. Prospective two-dimensional echocardiographic evaluation of left ventricular thrombus and embolism after acute myocardial infarction. Journal of the American College of Cardiology 1989;13:554–64.
[13] Asinger RW, Mikell FL, Elsperger J, et al. Incidence of left-ventricular thrombosis after acute transmural myocardial infarction. Serial evaluation by two-dimensional echocardiography. New England Journal of Medicine 1981;305:297–302.
[14] Solheim S, Seljeflot I, Lunde K, et al. Frequency of left ventricular thrombus in patients with anterior wall acute myocardial infarction treated with percutaneous coronary intervention and dual antiplatelet therapy. American Journal of Cardiology 2010;106:1197–200.
[15] Zielinska M, Kaczmarek K, Tylkowski M. Predictors of left ventricular thrombus formation in acute myocardial infarction treated with successful primary angioplasty with stenting. American Journal of the Medical Sciences 2008;335:171–6.
[16] Mollet NR, Dymarkowski S, Volders W, et al. Visualization of ventricular thrombi with contrast-enhanced magnetic resonance imaging in patients with ischemic heart disease. Circulation 2002;106:2873–6.
[17] Weinsaft JW, Kim HW, Crowley AL, et al. LV thrombus detection by routine echocardiography: insights into performance characteristics using delayed enhancement CMR. Journal of the American College of Cardiology 2008;52:148–57.
[18] Srichai MB, Junor C, Rodriguez LL, et al. Clinical, imaging, and pathological characteristics of left ventricular thrombus: a comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathological validation. American Heart Journal 2006;152:75–84.
[19] Mansencal N, Nasr IA, Pilliere R, et al. Usefulness of contrast echocardiography for assessment of left ventricular thrombus after acute myocardial infarction. American Journal of Cardiology 2007;99:1667–70.
[20] Kurt M, Shaikh KA, Peterson L, et al. Impact of contrast echocardiography on evaluation of ventricular function and clinical management in a large prospective cohort. Journal of the American College of Cardiology 2009;53:802–10.
[21] Cioffi G, Pozzoli M, Forni G, et al. Systemic thromboembolism in chronic heart failure. A prospective study in 406 patients. European Heart Journal 1996;17:1381–9.
[22] Baker DW, Wright RF. Management of heart failure. IV: anticoagulation for patients with heart failure due to left ventricular systolic dysfunction. The Journal of the American Medical Association 1994;272:1614–8.
[23] Lip GYH, Gibbs CR. Antiplatelet agents versus control or antigoagulation for heart failure in sinus rhythm: a Cochrane systematic review. Quarterly Journal of Medicine 2002;95:461–8.