

Left Ventricular Thrombosis: New Perspectives on an Old Problem

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Abstract

Left ventricular thrombosis (LVT) is a major risk factor for systemic thromboembolism and might complicate both the acute and the chronic phase of ischemic heart disease after myocardial infarction and, less frequently, non-ischemic cardiomyopathies.

The pathophysiology of thrombus formation is complex and involves the three aspects of Virchow's triad: blood stasis, prothrombotic state and tissue injury. Advances in technology have improved the detection rate of intracardiac thrombi, but several uncertainties still remain regarding the optimal treatment strategy within daily clinical practice. Of note, anticoagulation therapy with heparin and vitamin K antagonists decrease the risk of embolic stroke though exposing patients to an undeniable risk of bleeding complications. Although limited data on the off-label use of direct oral anticoagulants have reported safety and efficacy for LVT resolution, yet more evidence is needed to justify their use in clinical practice.

Key words: left ventricular thrombosis; acute myocardial infarction; anticoagulation therapy

Abbreviation list:

AMI: acute myocardial infarction

DAPT: dual antiplatelet therapy

DOACs: direct oral anticoagulants

LVT: left ventricular thrombosis

OAC: oral anticoagulation

VKA: vitamin K antagonists

Introduction

Intraventricular thrombus represents a potential life-threatening condition due to the significant risk of stroke and systemic thromboembolism. In older series, intracardiac thrombus has been reported in **more than 30%** of patients after anterior myocardial infarction.¹

The occurrence of left ventricular thrombosis (LVT) has substantially decreased over the last decades due to the greater use of primary percutaneous coronary intervention (PCI) and dual antiplatelet therapy (DAPT). However, a prevalence ranging between 3% and 9% after transmural myocardial infarction (MI) is still reported.² This condition poses several clinical challenges mainly related to the use of anticoagulation therapy, especially since many such patients already need DAPT for concomitant PCI. An increased prevalence of LVT has also been recorded among populations affected by dilated cardiomyopathy (DCM).³ While numerous data are available regarding the prevalence of LVT among patients with ischemic DCM, only few data have been published regarding non-ischemic etiology.⁴

The pathophysiological mechanisms underlying the formation of LVT in these different clinical subsets are multiple, but above all we can recognize left ventricular dysfunction and dilation and prothrombotic state associated with congestive heart failure (CHF). Oral anticoagulation (OAC) with vitamin K antagonists is the only therapeutic strategy approved for the prevention of thromboembolic events after MI complicated by LVT. Limited data on the off-label use of direct oral anticoagulants (DOACs) have reported safety and efficacy for LVT resolution, but more evidence is needed to justify their use in clinical practice.

The aim of this review article is to provide an overview of the current prevalence of LVT in its different settings, to summarize the main pathophysiological mechanisms and to evaluate all the available therapeutic options.

Prevalence in ischemic and non-ischemic cardiomyopathies

Although LVT can occur both in ischemic and non-ischemic cardiomyopathies, in clinical practice the vast majority of left ventricular thrombi are diagnosed after MI.

Post-Myocardial Infarction

During pre-PCI and pre-thrombolytic era intra-cardiac thrombosis occurred in almost 30% of patients after acute anterior transmural MI.⁵⁻⁷ Studies published between 1980 and 1990 demonstrated that the formation of LVT is mostly enhanced by location and size of the ischemic injury, *eg.* patients with the highest risk were those with anterior-apical MI associated with severe wall motion abnormality.⁵ Published studies agreed on reporting an overall prevalence of 17-24% of LVT after acute MI (AMI) being higher in those patients with anterior AMI, *eg.* 17% vs. 34%, 17% vs. 28% in the studies by Asinger⁷ and Johannessen,⁵ respectively; the peak value was found by Lamas who examined a cohort of 77 patients who experienced anterior AMI and observed a 46% prevalence of LVT, **Table 1.**^{1,5-9}

In the current era of PCI, there still remains a wide distribution of the prevalence, ranging from the lowest figure (0,4%) reported by Ram¹⁰ who screened with the use of echocardiography 157.891 patients after anterior ST-elevation MI (STEMI) to the highest prevalence found by Meurin¹¹ (26%) who examined 100 anterior-STEMI patients with a moderate left ventricular ejection fraction (LVEF) reduction (mean LVEF 33±6%). The differences that characterize the examined data are mostly explained by different patient selection criteria, selected diagnostic techniques and the antithrombotic regimen applied to STEMI patients¹² **Tables 2-3.**¹⁰⁻³⁰

In studies where cardiac magnetic resonance (CMR) imaging was used, an average prevalence of LVT of 8% after STEMI was reported, with a wider range between 7% and 26% if only anterior STEMI were taken into account. Considering that CMR has been regarded as the gold-standard technique for the diagnosis of LVT,²⁷ we can easily justify the higher prevalence above reported if compared to that recorded by the studies performed using ultrasounds imaging.

Non-ischemic cardiopathy

Limited data are available on the epidemiology of LVT in patients affected by non-ischemic heart diseases. Within this subgroup, DCM represents the most common underlying cardiomyopathy. In these patients, LVT may develop as a complication due to blood stasis in a severely dilated LV. Notably, in pediatric populations affected by cardiomyopathies, the presence of DCM exposes young patients to a risk of intracardiac thrombus, with LVT prevalence ranging from 6% to 53%.³¹ The course of hypertrophic cardiomyopathy, especially when associated with deteriorated LV systolic function and apical aneurysms, can be complicated by LVT. Thus, the presence of mid-ventricular obstruction has been proposed as leading factor in the progression of LV apical myocardial damage resulting in aneurysm formation.^{32,33}

LVT has also been occasionally described in the following diseases: Takotsubo cardiomyopathy, amyloidosis, hypereosinophilic syndrome, and Chagas' disease.

Thrombus formation can involve the acute phase of Takotsubo syndrome: small observational studies have shown a wide range incidence with the highest figure recorded by Santoro and colleagues (14% rate of LVT) in the presence of apical ballooning and elevated admission levels of Troponin I.³⁴ The largest observational study on 1.676 patients from the International Takotsubo Registry recorded a 3.3% prevalence of intraventricular thrombus and embolism in the acute phase of the disease³⁵. While the apical ballooning is a predisposing condition to thrombi development, atypical Takotsubo syndromes (midventricular and the basal types) are rarely complicated by LVT.

Intracardiac thrombosis occurs frequently in cardiac amyloid patients. The largest series published by DaLi Feng and colleagues (n=159 cardiac amyloid patients) reported a 27% (n=42) prevalence of intracardiac thrombosis.³⁶ Although almost all the thrombi (88%) were located in the atrial appendages, LVT can complicate this prothrombotic disease. Indeed, left ventricular diastolic dysfunction was an independent predictor of intracardiac thrombosis.

In the context of hypereosinophilic syndrome the cardiac involvement (Löffler's endocarditis) is associated with thrombus forming on the surface of the denuded myocardium of both ventricles,

particularly in the ventricular apices.³⁷ Among patients with rheumatic conditions, those affected by Behçet's disease may exhibit a rare cardiac manifestation of ventricular thrombosis, in 1.5% of patients³⁸.

Clinical predictors and risk factors

There is an extensive body of evidence pointing out that the principal risk factors closely related with LVT after AMI are both the presence of an anterior-apical scar and the reduction of systolic function.^{19,20} LV dysfunction is the strongest independent predictor of LVT formation and this finding is supported by the LVEF, which is significantly lower in LVT patients subgroup; furthermore the apex of the left ventricle is largely depicted as the region where more likely thrombi occur.

The components of Virchow's triad are present as main characters of post-infarction DCM: abnormal blood flow, hypercoagulability and wall tissue injury, **(Central Illustration)**.³⁹ The presence of LVT was strongly associated with apical LV dysfunction, thus emphasizing the role of blood stasis due to regional LV dysfunction as an important determinant of cardiac thrombus pathophysiology.²⁷ In addition to the main clinical predictors of LVT, various studies have discussed the impact of mitral regurgitation (MR) on the formation of thrombi within the left heart chambers proposing a protective effect of MR in LVT formation as a consequence of the decreased blood stasis.³ Different clinical data are evident: while Kalaria³ and Blondheim⁴⁰ independently showed that severe MR was not present in patients with LVT formation (protective role), Ascione and coll. reported that the presence of MR was associated with an higher incidence of thromboembolic events and LVT occurrence.⁴¹ This finding which appears to be in conflict with the rest of literature can be explained by the great extent of wall motion abnormality being related to both LVT and MR and acting as confounding factor; indeed at multivariate analysis the inclusion of MR (at any time after AMI) did not change the results.

The link between CHF and prothrombotic state, in terms of abnormalities in hemostatic markers and platelet function, has been widely explored since platelets and proteins of the coagulation cascade are

the major promoters of thrombogenesis.⁴² A reduced platelet survival time, increased mean platelet volume and greater platelet activation and reactivity have been confirmed within the context of CHF. Moreover, high levels of circulating fibrinogen, D-dimer and von Willebrand factor (vWF) in patients affected by HF with both reduced and preserved LVEF strengthens the association between heart failure and hypercoagulability.⁴³ Of note, patients with apical/anterior ventricular aneurysms have higher plasma fibrinogen and vWF levels when compared with healthy controls of similar age in sinus rhythm, and with patients affected by coronary artery disease in the absence of LV aneurysm.⁴⁴ Other works highlighted the importance of pro-inflammatory state and hypercoagulability to such an extent as to propose serum inflammatory markers at admission (C-reactive protein and fibrinogen) and neutrophil lymphocyte ratio as independent predictors of early LVT occurrence after AMI.⁴⁵ Indeed, Chin et al showed that pro-inflammatory cytokine IL-6 with its intrinsic capacity to induce increased levels of vWF and fibrinogen was a potential contributor of thrombotic complications of CHF.⁴⁶ Lastly, Zabczyk et al. reported a cohort of 58 patients with previous LVT unrelated to recent MI or severe HF,⁴ demonstrating how LVT of unknown origin occurrence and recurrences are strongly associated with a prothrombotic state, inclusive of fibrin clot resistant to lysis and lower plasma levels of alpha-2 antiplasmin.

Diagnostic tools

Transthoracic echocardiography (TTE) is the primary screening test for LVT since it is widely used to assess post-MI LV function and structure, as well as to exclude early post-infarction mechanical complications. LVT appears as an echo-dense mass with well-defined edges distinct from the endocardium, often contiguous to an abnormally contracting LV segment, akinetic or hypokinetic, or an aneurysmal myocardium (**Figure 1, panel 1 and 2**).²⁷ Thrombi can be classified in relation to their appearance: for example, protuberant thrombi have borders which mainly stick out into LV cavity while mural thrombi are flat masses. Protuberant thrombi may be pedunculated or sessile and, since they are mobile masses, they are associated with an increased risk of ischemic stroke.

The major obstacles to LVT detection by TTE are the visualization of the true apex, the presence of small mural thrombi and a poor TTE window. According to the previous literature, TTE has a reported overall diagnostic accuracy greater than 90% if compared with CMR (gold standard technique).²⁵ However, the diagnostic performance of TTE strongly depends on image quality *e.g.* endocardial definition and absence of artifacts.²⁷

Indeed, transesophageal echocardiography (TEE) is rarely appropriate for LVT detection because the left ventricular apex is foreshortened and in the far field. Both TEE and TTE detection rate of LVT correlates with thrombus size and the reported overall accuracy may vary according to the clinical indication which led to its execution.

The use of contrast echocardiography improves the endocardial border definition, particularly if the echographic window is suboptimal. Testing with contrast echocardiography allows to overcome the burden of a blurred visualization of the LV apex yielding improved image quality and sensitivity (64% vs. 35% in contrast vs. non-contrast echo, respectively) for LVT in patient affected by anterior AMI. Despite the improvements reported, still one third of LVT detected on CMR are missed. For this reason the use of echocardiography should be regarded as stratification tool to select those patients who require CMR assessment.²⁷

CMR can determine the presence of LVT based on avascular tissue characteristics and it is considered the gold standard imaging technique in assessing the presence, size and location of thrombi. LVT can be identified on delayed-enhanced CMR as low-signal intensity intraventricular filling defects, generally adherent to areas of hyper-enhanced LV myocardial scarring (**Figure 1 panels 3A-B**), while on cine-CMR LVT commonly are adjacent to thinned and dysfunctional LV walls.⁴⁷

LVT may also be discovered as a cavity filling-defect on multi-detector computed tomography, usually reported as incidental finding (**Figure 1 panel 4**).⁴⁸ Comparison between arterial and delayed phase does not provide an optimal tissue characterization and the use of spectral computed tomography dual-substance separation (iodine vs. blood based) technology has been proposed to overcome this limitation.⁴⁹

To the best of our knowledge it is not possible to determine thrombus age with the currently available imaging techniques.

Risk of embolization

It is generally acknowledged that LVT is associated with an increased embolic risk, particularly during the first 1 to 2 weeks after AMI.⁵⁰ A recent matched cohort study, which aimed at ascertaining the incidence of systemic embolism, proved that patients affected by LVT observed during a median follow up of 3 years had a 3.7% annualized rate of a composite of stroke, transitory ischemic attack and extracranial systemic arterial embolism, four times higher when compared with matched non-LVT patients.⁵¹ Older studies documented substantial embolic risk, ranging between 13% and 22%, proving that thrombus mobility and thrombus protrusion were the main risk factors for embolization with a reported positive predictive value in predicting embolism of 83% and 57%, respectively.⁵²

The embolic rates of the studies that detected LVT with the use of CMR are shown in the bubble chart, **Figure 2**. While studies that included few patients with a relatively short-term follow up (<12 months) recorded almost no embolic events, those with a larger population and longer follow-up showed an obviously increased embolic rate.

Treatment options

Current international guidelines

International guidelines agree on recommending anticoagulation therapy for patients affected by definite LVT. The pharmacodynamic and pharmacokinetic aspects of the anticoagulant drugs involved in LVT treatment are collected in **Supplementary Table 1**.

The efficacy of parenteral therapy with heparin has been proved both for the prevention of LVT formation after STEMI and for the treatment of LVT with high embolic risk.^{53,54} The recommendations taking into account the management of LVT derive from the exhaustive 2012

American College of Chest Physicians (ACCP) guidelines for primary and secondary prevention of Cardiovascular Disease and from the most recent 2014 guidelines by the American Heart Association (AHA/ASA) and 2017 guidelines by the European society of Cardiology (ESC), which suggested OAC with vitamin K antagonists (VKA) as first-line therapy, **Table 4.**^{50,55–58} The body of evidence of European and American guidelines are largely based on studies published in the pre-PCI era, thus stressing the efficacy of parenteral therapy with heparin.⁵⁹ A meta-analysis comprehensive of 11 observational studies proved that anticoagulation with VKA effectively decreased the risk of LVT formation and embolization.⁶⁰ Given the fact that oral VKA therapy in appropriate patients is likely to be as effective and more practical than anticoagulation with heparin, prolonged parenteral therapy it is seldom recommended, **Table 4.** Of note, there are no randomized trials that have prospectively examined the efficacy of VKA anticoagulation compared to parenteral therapy. Also, no prospective studies have evaluated the optimal timing of parenteral anticoagulation in patients being started on VKA. Consensus is that OAC therapy with VKA should start as soon as LVT is identified and parenteral anticoagulation should be discontinued when effective therapeutic range with warfarin has been achieved (INR of 2 to 3). In order to assess thrombus resolution with the use of VKA all the guidelines agree in recommending an echocardiography at 3 months, whereas a more tight follow-up of 1 to 2 weeks is suggested for those patients who have contraindications to anticoagulation.⁵⁰

OAC should be discontinued as soon as thrombus resolution has been established: a 3-month therapy is usually needed.^{59,61,62} In case of thrombus permanency, prolonged anticoagulation and repeated imaging assessment are recommended, **Table 4.**

It is well known that triple antithrombotic therapy with VKA is associated with an increased bleeding risk as evident from randomized controlled trials which investigated patients affected by atrial fibrillation (AF)^{63–66} and by ISAR-TRIPLE and WOEST trials, which included patients with indications for OAC including AF, venous thromboembolism, mechanical valves, etc.^{67,68} Despite the lack of power to detect significant differences in ischemic endpoints, all these trials reported an increased rate of bleeding events with dose-adjusted VKA plus DAPT. However, no risk scoring

system has been robustly validated for bleeding risk stratification with triple antithrombotic therapy. The 2017 ESC focus update on DAPT and the 2019 ESC guidelines on diagnosis and management of chronic coronary syndromes state that triple antithrombotic therapy duration should be minimized with respect to both bleeding and ischemic risks, and triple therapy for ≥ 1 month should be considered only in those patients whose risk of stent thrombosis outweighs the bleeding risk.^{69,70} In patients with an indication for VKA in combination with antiplatelet therapy, the dose of VKA should be regulated so that the INR is in the lower part of the recommended target range with a time in therapeutic range $> 70\%$.^{69,70}

Direct Oral Anticoagulants

Limited data are available on the use of DOACs to replace warfarin in patients with LVT. The role of DOACs has been investigated by two reviews of published case reports, which respectively collected a total of 36 and 41 patients with LVT treated with different molecules.^{71,72} There are, thus far, only a few retrospective studies on the topic with the first one published as the result of a small monocentric retrospective registry from St Luke's Mid America Heart Institute collecting 52 cases of confirmed LVT treated with DOACs.⁷³ All these studies reported thrombus resolution at follow-up in more than 80% of patients. These evidences are undermined by the strong limitations of publication bias, particularly for the collections of case reports. The latter data on antithrombotic therapy for patients with LVT come from 2 retrospective analyses. The ACTION study group (Pitié-Salpêtrière Hospital, Paris, France) collected 159 patients with LVT and, albeit not being designed and powered for a direct comparison between DOACs and VKA, suggested a similar rate of LVT regression with both anticoagulant regimens⁶². On the other hand a multicenter retrospective cohort study evidenced that DOACs treatment was associated with a higher risk of stroke and systemic embolism than VKA therapy.⁷⁴ Those results may be considered exploratory rather than definitive, emphasizing the need for randomized clinical trials to provide more robust evidences.

P2Y₁₂ receptor inhibitors

The introduction of potent platelet P2Y₁₂ receptor inhibitors in clinical practice may have contributed to a reduction of LVT incidence after AMI, as suggested by a retrospective analysis opposing ticagrelor to clopidogrel.⁷⁵ Despite being biologically plausible, there is no trial in literature confirming that DAPT plays a protective role against the formation and embolization of LVT, thus antiplatelet therapy cannot be considered as a substitute for anticoagulation to prevent embolization of LV thrombus.

Surgical treatment for LVT

Although not being mentioned by the aforesaid international guidelines, surgical removal of left ventricular thrombus is an option for patients with high-embolic risk profile, for example those undergoing extra-corporeal mechanical oxygenation. The standard operative technique involves left ventriculotomy, which eventually poses the added concern of closing a friable myocardium with increased risk of bleeding as well as added insult to left ventricular function. The use of either cardio-endoscopic techniques or robotic surgery seems to be a very good option for these patients since they provide the advantage of preventing complications such as ventricular dysfunction, arrhythmias and left ventricular aneurysm formation and allow rapid recovery of myocardium from inflammatory process. Data on operative treatment of LVT are scarce and mostly confined to case reports or case series.⁷⁶ While surgical removal might reduce the incidence of thromboembolism more effectively than anticoagulation therapy, the risks do not necessarily outweigh the benefits and it is not recommended to perform open-heart surgery when the only indication is represented by LVT.

Diagnostic and Treatment Algorithm

We propose an evidence-based algorithm for a combined diagnosis and treatment approach of LVT, **Figure 3**. For diagnosis, an accurate TTE is the imaging modality of choice to screen high-risk patients. In case of indeterminate results or persisting high suspicion of LVT, second level tests such as contrast-TTE, CMR or computed tomography should be performed, even if their applicability may be limited by local availability and resources. A serial imaging strategy is preferred in case of

inconclusive results and significant bleeding risk. When dealing with therapeutic regimen and duration, the physician should best tailor antithrombotic therapy to the patient being guided by an individualized risk stratification based on baseline features and LVT evolution. In the presence of defined LVT and in the absence of clear contraindications, OAC should be started immediately, and concurrent parenteral therapy should be administered until two days of therapeutic INR (target 2.5, range 2-3) are obtained. In intensive care unit patients with large and mobile LVT for whom surgical therapy is being evaluated, unfractionated heparin with target aPTT ~ 60s (50-70s) may be a reasonable alternative. In case of intolerance to VKA, prolonged LMWH therapy or DOACs may be considered. The duration of antithrombotic therapy should be guided by imaging assessment and should be continued until resolution of LVT.

Conclusions

Even if cardiovascular therapy made great strides in the past 10 years, intraventricular thrombosis, principally as a consequence of myocardial infarction, still remains a clinical challenge. Early detection of LVT and an optimal antithrombotic therapy are of utmost importance in reducing potentially life-threatening complications. Off-label use of DOACs suggests a potential role of these drugs for LVT treatment. Available data does not yet allow to consider them valid alternatives to warfarin but highlights the need for larger randomized trials.

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Figure legends

Central Illustration. The pathophysiology of left ventricular thrombosis involving Virchow's triad.

Figure 1. Left ventricular thrombosis (LVT) diagnostic tools. LVT appears as an echo-dense mass on transthoracic echocardiography: **panel 1** shows a small size apical thrombus, (white arrows), **panel 2** shows a large apical pedunculated thrombus (yellow arrows). Cardiac magnetic resonance imaging can detect LVT on delayed-enhancement phase-sensitive inversion recovery image, vertical long-axis view (the black asterisk shows a non-enhancing mass in the apex, consistent with thrombus, **panel 3A**). The same view on turbo inversion recovery magnitude sequence shows high signal in the mid and apical anterior segments of the left ventricle (red arrows) consistent with myocardial edema and apical thrombus (white asterisk, **panel 3B**). Multi-slice computed tomography shows low-density shadow of filling defects indicating LVT on a long-axis and a short-axis view (white asterisk, **panel 4A-B**).

Figure 2. Embolic events rate after Left Ventricular Thrombosis (LVT) diagnosed with late gadolinium enhancement cardiac magnetic resonance. The size of the dots is proportional to the LVT patients' sample which is reported next to first-author's name. The meta-regression curve (dashed line), its corresponding coefficient of determination (R^2) and equation denote a positive linear relationship between embolic events rate and follow up time.

Figure 3. Evidence-based algorithm for a combined diagnosis and treatment approach of LVT.

APT = antiplatelet therapy; aPTT = activated partial thromboplastin time; CICU = coronary intensive care unit; CMR = cardiac magnetic resonance; CT = computed tomography; DCM = dilated cardiomyopathy; DOACs = direct oral anticoagulants; HCM = hypertrophic cardiomyopathy; INR = international normalized ratio; LMWH = low-molecular-weight heparin; LVEF = left ventricular ejection fraction; LVT = left ventricular thrombosis; MI = myocardial infarction; TIA = transitory ischemic attack; TTE = transthoracic echocardiography; UFH = unfractionated heparin; VKA = vitamin K antagonist.

Table 1. Observational studies examining left ventricular thrombus prevalence after acute myocardial infarction during the pre-reperfusion era

Study	AMI patients	Overall LVT Prevalence	LVT prevalence in anterior AMI
Asinger 1981⁷	70 STEMI (35 Anterior)	17% (12)	34% (12)
Johannessen 1984⁵	90 STEMI (53 anterior)	17% (15)	28% (15)
Lamas 1988⁶	77 anterior STEMI	-	46% (35)
Kupper 1989⁸	92 anterior STEMI	-	33% (32)
Nihoyannopoulos 1989¹	87 STEMI (53 anterior)	24% (21)	40% (21)
Motro 1991⁹	144 STEMI (76 anterior)	6,25% (9)	9,2% (7)

AMI = acute myocardial infarction; LVT = Left ventricular thrombosis; STEMI = ST-elevation myocardial infarction.

Table 2. Incidence of LVT after acute myocardial infarction in CMR studies

Study	AMI patients	Overall LVT Prevalence	LVT prevalence in anterior AMI
Weir 2009²⁴	100 AMI (55 anterior)	15% (15)	27% (15)
Weinsaft 2011²⁵	243 STEMI, LVEF < 50%	10% (24)	-
Delewi 2012²⁶	194 STEMI (123 anterior)	9% (17)	14% (17)
Weinsaft 2015²⁷	201 STEMI (108 anterior)	8% (17)	15% (16)
Meurin 2015¹¹	100 anterior STEMI (LVEF <45%)	-	26% (26)
Poss 2015²⁸	738 STEMI (339 anterior)	3,5% (26)	7% (24)
Bièrre 2016²⁹	329 STEMI (183 anterior)	9% (30)	16% (30)
Gellen 2017³⁰	265 anterior STEMI	-	13% (34)
Cambronero -Cortinas 2017¹³	392 STEMI (207 anterior)	7% (27)	12% (25)

AMI = acute myocardial infarction; CMR = cardiac magnetic resonance; LVEF = left ventricular ejection fraction; LVT = Left ventricular thrombosis; STEMI = ST-elevation myocardial infarction.

Table 3. Incidence of LVT after acute myocardial infarction by TTE imaging

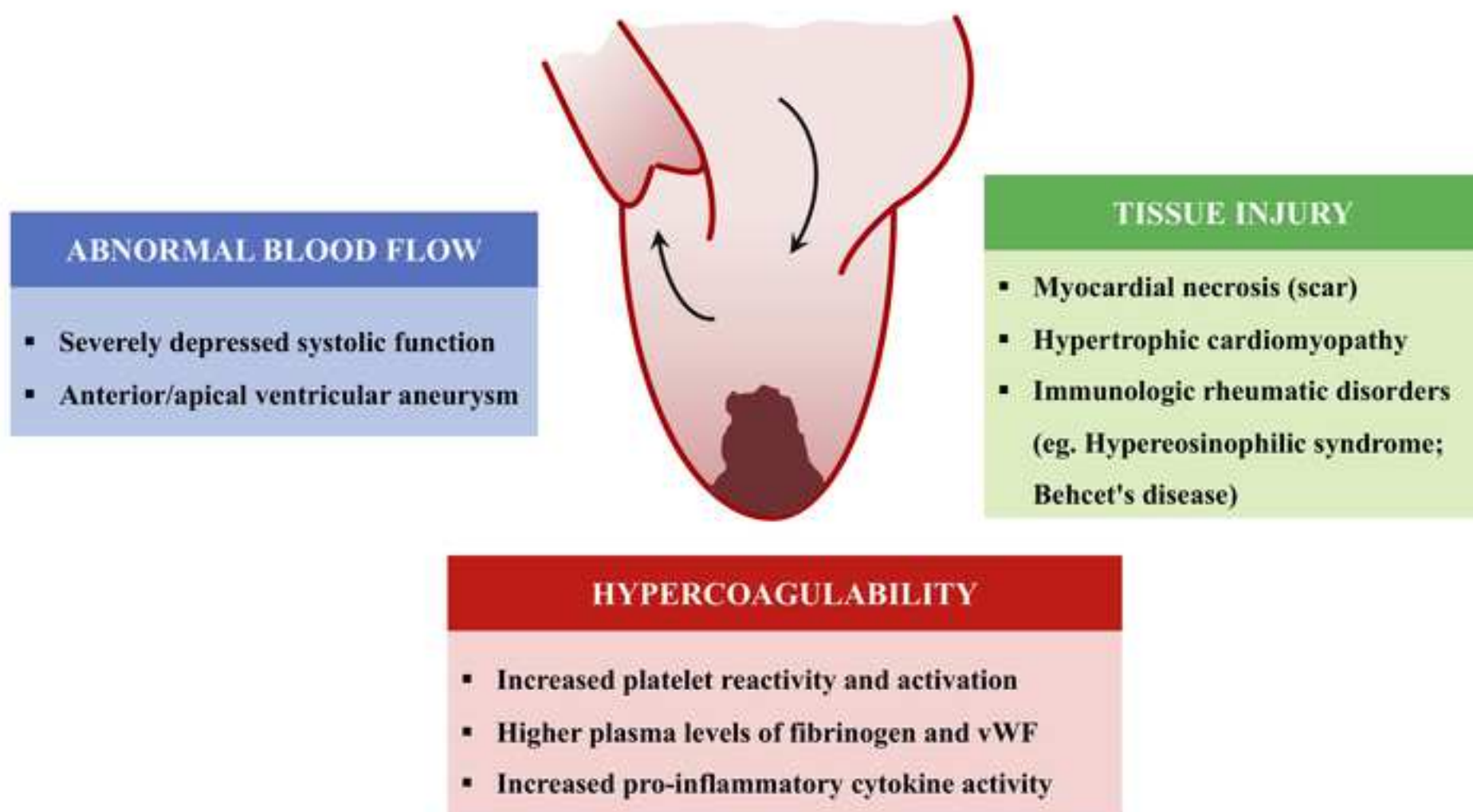
Study	AMI patients	Overall LVT Prevalence	LVT prevalence in anterior AMI
Greaves 1997¹⁴	309 anterior STEMI	-	6% (19)
Kalra 2000¹⁵	71 STEMI (30 anterior)	4% (3)	10% (3)
Zielisnka 2007¹⁶	2911 AMI (1251 anterior)	2,5% (73)	5,5% (69)
Osharov 2009¹⁷	642 anterior AMI	-	6% (40)
Solheim 2010¹⁸	100 anterior AMI	-	15% (15)
Shacham 2013¹⁹	429 anterior STEMI	-	4% (18)
Gianstefani 2013²⁰	1059 STEMI (454 anterior)	4% (42)	8% (37)
Driesman 2015²¹	310 anterior STEMI	-	15% (47) probable, 3% (9) definite
Khoury 2017¹²	2071 STEMI	1,5% (31)	-
Zhang 2018²²	1488 anterior STEMI	-	7% (106)
You 2018²³	267 LVEF < 45%	9% (25)	-
Ram 2018¹⁰	157891 anterior STEMI	-	0,4% (649)

AMI = acute myocardial infarction; LVEF = left ventricular ejection fraction; LVT = Left ventricular thrombosis; NSTEMI = non ST-elevation myocardial infarction; STEMI = ST-elevation myocardial infarction; TTE = transthoracic echocardiography.

Table 4. Guidelines on treatment of left ventricular thrombosis

Guidelines	Recommendation
ACCP 2012⁵⁰	Patients with anterior MI and definite LVT or at high risk for LVT (LVEF < 40%, anterior-apical wall motion abnormality) who undergo DES placement should be treated with VKA anticoagulation plus DAPT (low-dose aspirin and clopidogrel) for 3 – 6 months (Grade 2C).
ESC STEMI 2012⁵⁸	In case of definite thrombus, VKA anticoagulation for a minimum of 3 months (class IIa, LOE B). In patients with large anterior wall motion abnormalities at low risk of bleeding consider anticoagulation.
ACCF/AHA STEMI 2013⁵⁷	In patients with asymptomatic mural thrombi VKA anticoagulation (class IIa, LOE C). In patients with anterior-apical akinesis consider anticoagulation (class IIb, LOE C)
AHA/ASA STROKE 2014⁵⁵	Patients with ischemic stroke or TIA in the setting of acute MI complicated by LVT should be treated with VKA anticoagulation therapy for 3 months (class I, LOE C). If intolerant to VKA therapy because of non-hemorrhagic adverse events, anticoagulation with an LMWH, dabigatran, rivaroxaban, or apixaban for 3 months is an alternative to VKA therapy (class IIb, LOE C).
ESC STEMI 2017⁵⁶	In case of definite thrombus, anticoagulation for up to 6 months guided by repeated imaging (class IIa, LOE C).

ACCP = American College of Chest Physicians; ACCF/AHA= American College of Cardiology Foundation/American Heart Association; AHA/ASA = American Heart Association/American Stroke Association; DAPT= dual anti-platelet therapy; DES = drug-eluting stent; ESC = European Society of Cardiology; LMWH = low-molecular-weight heparin; LOE = level of evidence; LVEF = left ventricular ejection fraction; LVT = left ventricular thrombosis; STEMI= ST-elevation myocardial infarction; TIA = transitory ischemic attack; VKA= vitamin K antagonist.



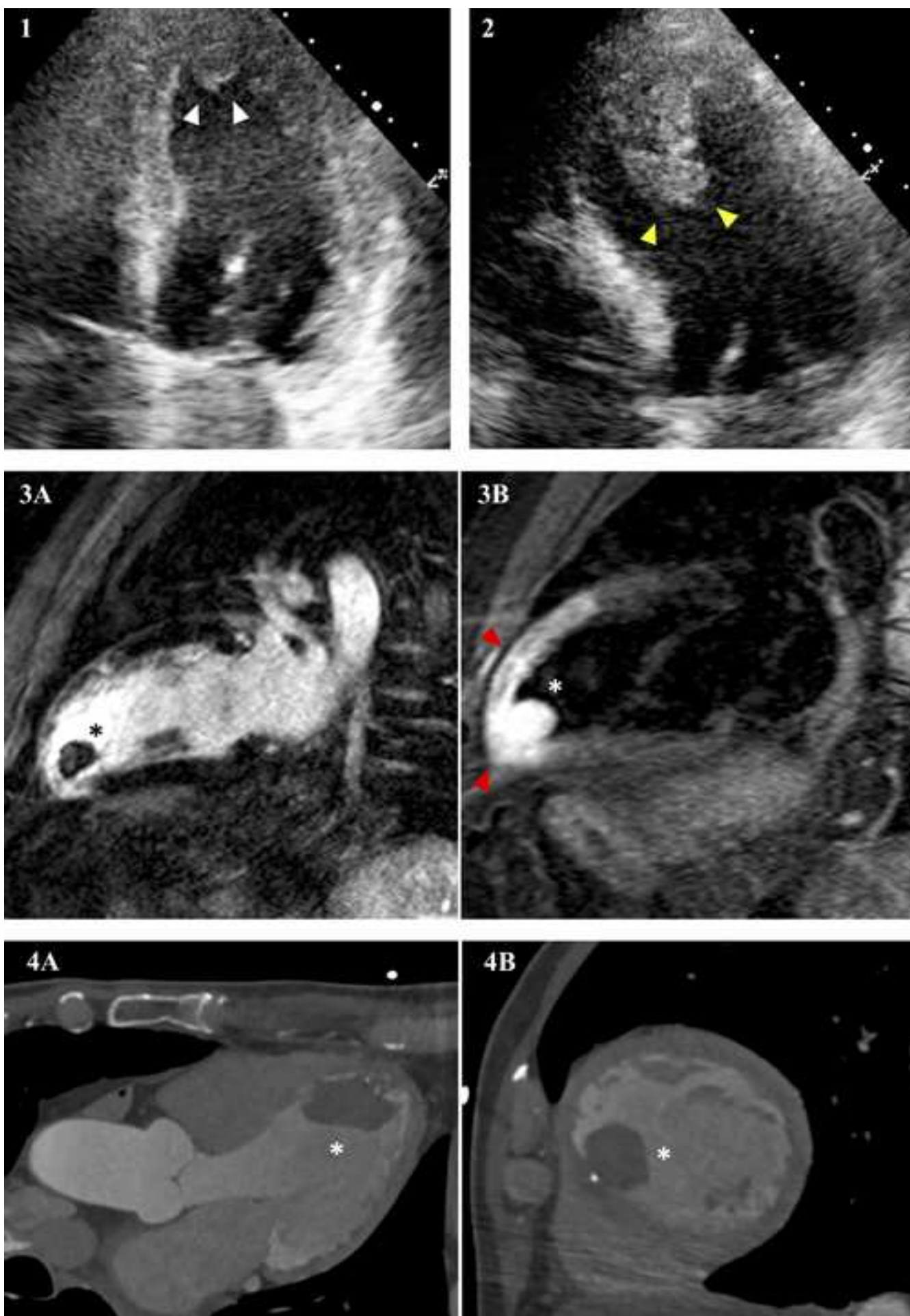


Figure 1

Figure 2

