LEFT VENTRICULAR ANEURYSM AND THROMBUS FORMATION AFTER ACUTE MYOCARDIAL INFARCTION: TREATMENT OPTIONS

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ABSTRACT
Left ventricular aneurysm and thrombus are clinically significant complications of acute myocardial infarction. However, clear guidelines for selecting a treatment approach are lacking. A literature review was conducted to discuss the pathophysiology, aetiology, and associated risks of these complications, and to summarise treatment strategies and the process of selecting a suitable one. Conclusions: Left ventricular aneurysm with a mobile thrombus is a rare complication; therefore, therapeutic recommendations are based mainly on retrospective case series, and there are no clear treatment guidelines. Depending on the size of the aneurysm and the clinical significance of symptoms, either surgical treatment or conservative management with anticoagulation is chosen. More commonly, initial pharmacological therapy is selected, with therapeutic efficacy assessed over time and the need for surgical intervention reconsidered accordingly. Treatment is planned individually for each patient, taking into account the risks associated with surgery.

Keywords: *left ventricle aneurysm; left ventricle thrombus; acute myocardial infarction; left ventricular reconstruction.*

1. INTRODUCTION
In recent decades, outcomes of diagnosis and treatment of acute myocardial infarction (AMI) have improved significantly. According to 2019 data, 30-day mortality after diagnosis in European Union countries was 9% (the lowest in the Netherlands (3,2%); the highest, in Latvia (17%); and 13.4% in Lithuania) [1]. With improving survival rates after acute myocardial infarction, increasing attention should be directed toward complications that occur in the post-infarction period. These may be ischemic, mechanical, arrhythmic, embolic, or inflammatory in origin [2]. One of the most common complications of transmural AMI is left ventricular aneurysm. Epidemiological data reported in the literature indicate that it develops in 10–35% of all acute transmural myocardial infarction cases [3, 4].
Left ventricular aneurysm (LVA) is defined as a portion of the ventricular wall that has undergone fibrotic remodelling and paradoxically bulges outward during systole, resulting in reduced left ventricular ejection fraction (LVEF). Akinesia or dyskinesia of the affected myocardial segment is typically observed [5]. In 40–60% of cases, left ventricular thrombus (LVT) is also diagnosed concomitantly with post-AMI aneurysm formation, potentially leading to thromboembolic events [2, 3, 6]. Thrombus protrusion from the ventricular wall and its mobility are the principal determinants of embolic risk [7]. Several key factors predispose LV thrombus formation: large anterior wall or apical ST-segment elevation myocardial infarction (STEMI), especially when treatment is initiated more than 12 hours after symptom onset; reduced LV ejection fraction; and impaired contractility of the infarcted myocardial segment [8]. With increasing availability of percutaneous coronary intervention (PCI), advanced cardiac imaging, and early pharmacological therapy, the incidence of LV thrombus in AMI patients has decreased to 3–10% [9, 10]. A 2022

meta-analysis reported that LV thrombus formation after PCI occurs in 2–4% of cases [11].

This literature review aims to discuss the pathophysiology, aetiology, and clinical risks of left ventricular aneurysm and thrombus formation following acute myocardial infarction, and to analyse available treatment strategies for this patient population.

The aforementioned AMI complications and their associated symptoms directly affect patient quality of life, a major priority in contemporary medicine. Therefore, it is clinically relevant to review current strategies and the optimal timing for restoring LV structure in the presence of these complications. According to the literature, surgical aneurysm repair with thrombectomy is indicated in cases of refractory heart failure, thromboembolism, or life-threatening tachyarrhythmias [5]. In these cases, one of the most frequently performed procedures is surgical ventricular restoration (the Dor procedure), aimed at aneurysm resection and LV shape restoration [12].

2. MATERIALS AND METHODS

A search of scientific publications was conducted in the MEDLINE database using the PubMed search engine. The study includes an analysis of literature reviews, systematic reviews, meta-analyses, as well as retrospective clinical studies, observational studies, and case reports.

The following keywords were used during the search: “left ventricular aneurysm” OR “left ventricular thrombus” OR “left ventricular mobile thrombus” AND “treatment” OR “surgery” OR “surgical treatment” OR “left ventricular reconstruction” OR “Dor procedure” AND “myocardial infarction” OR “postinfarct*”.

Articles published in English or Lithuanian were selected for analysis. Initially, information was collected on the characteristics of left ventricular thrombus and aneurysm formation following acute myocardial infarction, including their features and their relevance to choosing an appropriate treatment strategy.

Subsequently, the search was narrowed by applying additional restrictions: articles published within the past 20 years; studies conducted in human subjects; patients older than 18 years; and cases in which left ventricular thrombus and aneurysm developed as a consequence of a newly occurring acute myocardial infarction. Particular attention was given to identifying the treatment strategies chosen in cases of mobile left ventricular thrombus.

3. RESULTS AND DISCUSSION

3.1. Pathophysiology of Left Ventricular Thrombus (LVT) Formation

The pathophysiological mechanism of left ventricular thrombus (LVT) formation can be explained by Virchow’s triad (blood stasis, myocardial injury, and hypercoagulability or inflammation) [10]. All components of this triad are present in the setting of acute myocardial infarction (AMI). A hypokinetic or akinetic ventricular segment promotes blood stasis. Studies demonstrate that AMI patients with reduced left ventricular ejection fraction (LVEF) at hospitalisation have a higher risk of developing LVT [14–16]. Decreased LVEF is considered a principal risk factor for thrombus formation. Furthermore, increased left ventricular dimension (>60 mm) is associated with a higher likelihood of thrombus formation [16].

The extent of local myocardial injury influences the risk of LVT. Compared with patients with non-ST-segment elevation myocardial infarction (NSTEMI), those with ST-segment elevation myocardial infarction (STEMI) are more likely to develop LVT. The final component of Virchow’s triad— inflammation and

hypercoagulability—also contributes to thrombus formation. Monocytes and macrophages participate in myocardial healing; local inflammation promotes platelet aggregation and fibrin network formation through interaction with pro-inflammatory cytokines. Elevated platelet count and increased C-reactive protein levels are associated with a higher incidence of LVT following AMI [6,8,16].

LVT most commonly develops within the first two weeks after myocardial infarction [17]. Diagnosis may be challenging, as many thrombi form during the second week, by which time patients have already been discharged from the hospital. Therefore, the 2023 European Society of Cardiology guidelines emphasise the importance of the appropriate timing of imaging (transthoracic echocardiography or cardiac MRI). The imaging tests are recommended at least two weeks after AMI [18].

LVT is morphologically classified according to its configuration: a flat thrombus parallel to the endocardial surface is defined as mural, whereas a thrombus protruding into the left ventricular cavity is classified as protruding. If any part of the thrombus exhibits independent motion relative to the myocardium, it is defined as mobile [19].

3.2. Risk of Embolisation in Left Ventricular Thrombus

The presence of LVT is associated with a substantial risk of embolisation. According to data from the American Heart Association, the risk of thromboembolism may reach up to 22%, depending on thrombus morphology and additional risk factors [8]. Most embolic events occur within the first three months after MI, with the highest risk during the initial weeks.

The most important predictor of embolic risk is thrombus mobility and protrusion into the ventricular cavity [20]. Thrombus size appears less significant in predicting embolisation [8]. Interestingly, mobile thrombi have been reported to respond more favourably to pharmacological therapy than mural thrombi and may resolve more rapidly (within approximately 1 month) [21].

3.3. Aneurysm Formation and Associated Symptoms

Myocardial changes leading to aneurysm formation begin within the first hours after AMI. The infarcted myocardium becomes thinned; during the initial days, inflammatory cells migrate into the affected region, and necrotic myocytes undergo lysis. The ventricle dilates, and akinetic and dyskinetic fibrotic tissue develops, leading to ventricular remodelling. Left ventricular aneurysm typically begins to form within 2–10 days after AMI [2].

An aneurysm develops when, following transmural myocardial infarction, a damaged myocardial segment loses its contractile function and bulges outward during systole due to increased intracavitary pressure, resulting in reduced left ventricular stroke volume [2].

Over time, LV dilation progresses, wall stiffness increases, and LV end-diastolic pressure rises. Laplace's law ($\sigma = Pr/2\ h$) explains these processes [22]. At constant ventricular pressure (P), increased radius (r) and reduced wall thickness (h) increase wall stress (σ), promoting further deformation and ischemia. Functionally, this leads to ventricular dilation and increased end-diastolic volume, contributing to myocardial hypertrophy, increased oxygen demand, and symptoms such as angina or arrhythmias [5].

Fibrosis, wall thinning, and dilation are interrelated processes. LV dilation promotes aneurysm formation, and aneurysm formation further contributes to progressive myocardial hypertrophy and clinical deterioration. Some reports suggest that mural LV thrombus may exert a mechanical stabilising effect at the infarct site, potentially

reducing the risk of ventricular rupture. By partially restoring wall thickness, mural thrombus may reduce wall stress, as per Laplace's law, and thereby improve contractile efficiency [23].
LV aneurysm diameter typically ranges from 1 to 8 cm. In 90% of cases, aneurysms develop in the apical or anterior wall, and in 10% in the inferoposterior wall [5].
The clinical presentation of LV aneurysm is variable. Some patients are asymptomatic, and the aneurysm is detected incidentally on TTE or cardiac MRI. The most common symptom is angina [5]. Dyspnea, arrhythmias, and heart failure may also occur. In severe cases, pulmonary oedema, thromboembolism, or ventricular rupture may develop [3].

3.4. Myocardial Remodelling and Scar Formation After MI: Implications for Treatment Strategy

Myocardial remodelling is a dynamic process comprising three phases: inflammatory-necrotic, proliferative-fibrotic, and long-term remodelling.
Following AMI, fibroblasts are activated and differentiate into myofibroblasts, which are the primary source of extracellular matrix during myocardial healing. A temporary fibrin-based matrix forms in the inflammatory environment [24]. The final phase, long-term remodelling, lasts several months. During this phase, collagen content stabilises, the provisional fibrin matrix is replaced by mature collagen, and the scar gains structural stability through collagen cross-linking [25].
Aneurysmectomy is recommended during the third remodelling phase, after stable scar formation, as it reduces the risk of bleeding and improves surgical outcomes [19]. Delayed intervention also facilitates differentiation between viable and non-viable myocardium intraoperatively.
Recently, a modified surgical approach for LVT removal has been introduced, aiming to preserve LV wall integrity through trans-mitral thrombectomy. This approach avoids direct ventricular incision in fragile post-infarction myocardium but is applicable only in cases of LVT without concomitant aneurysm [26,27].

3.5. Treatment Recommendations

The European Society of Cardiology and the European Association for Cardio-Thoracic Surgery guidelines on myocardial revascularisation recommend considering LV aneurysmectomy combined with coronary artery bypass grafting (CABG) in patients with NYHA class III–IV heart failure, large LV aneurysm, large thrombus, or aneurysm-related arrhythmias (Class IIa, Level C recommendation). LV reconstruction during CABG is performed in selected patients at specialised centres (Class IIa, Level B recommendation) [22,28]. No specific timing for surgery is defined in the guidelines.
Isolated LVT without aneurysm is not considered an indication for open-heart surgery [9]. The 2023 ESC guidelines recommend vitamin K antagonists or direct oral anticoagulants for 3–6 months, with repeat imaging (TTE or cardiac MRI), bleeding risk assessment, and consideration of concomitant antiplatelet therapy. The guidelines acknowledge the lack of prospective randomised trials to determine optimal anticoagulation regimens and duration [18]. Increasingly, patients with both LV aneurysm and thrombus are managed conservatively with outpatient anticoagulation therapy [9].
Regarding mobile and protruding LVT, available literature consists primarily of case reports and case series describing heterogeneous strategies, including urgent thrombectomy and conservative management [13]. Neither European nor American

societies provide specific recommendations for these thrombi. A 2022 American Heart Association scientific statement concluded that current evidence is insufficient to routinely recommend surgical intervention in the absence of other operative indications. Surgical management may be considered in exceptional circumstances, such as intolerance to anticoagulation or high embolic risk [8], although clear criteria for defining embolic risk are not provided.

3.6. Left Ventricular Restoration Surgery

Although surgical treatment of LV aneurysms has been performed for more than 50 years, opinions regarding its benefit in advanced ischemic heart disease remain divided. The RESTORE group reported in 2004 that LV restoration surgery significantly improved outcomes in ischemic cardiomyopathy, with a 5-year survival of 68.6 ± 2.8% and a significant improvement in NYHA functional class [29].

However, the 2009 randomised controlled STICH (Surgical Treatment of Ischemic Heart Failure) trial demonstrated no significant difference in outcomes between CABG alone and CABG with LV volume reduction [30]. LV volume reduction did not significantly improve symptoms, exercise tolerance, or mortality. The study was later criticised for including patients with relatively small aneurysms. Subsequent analyses suggest that LV volume restoration is beneficial in patients with large symptomatic aneurysms and a significantly reduced LV volume, particularly when postoperative end-systolic volume index is ≤ 70 mL/m² [31]. Patients with extensive LV scarring, who may benefit most, were excluded from the STICH trial [32].

A 2018 systematic review including 27 studies concluded that LV restoration surgery may improve long-term outcomes in selected patients [33].

3.7. Review of Treatment Strategies for Mobile and Protruding Thrombi

Cases of mobile LVT with concomitant LV aneurysm following recent AMI are rare. A review of the past 20 years identified 9 case reports, 1 retrospective descriptive study, and 1 retrospective cohort study involving patients with LV aneurysm and thrombus after recent AMI (Table 1). Reported management strategies included urgent surgery, initial medical therapy followed by surgery, and exclusive pharmacological management. Most reports describe favourable outcomes without major complications, highlighting the perceived success of the selected treatment strategy.

The case report by F. He and colleagues described an adverse outcome in a patient with a hypermobile left ventricular thrombus: during conservative management with low-molecular-weight heparin, the patient developed an ischemic stroke two days after AMI [34]. The patient was transferred to the neurology department, and surgical treatment was performed one month later. It is important to note that this patient had multiple high-risk factors, including a myocardial bridge of the right coronary artery, alcohol abuse, and active smoking.

Bennett and colleagues reported a case in which vitamin K antagonists were contraindicated for the patient; therefore, treatment with apixaban was initiated. A favourable outcome was observed, with thrombus resolution within six days and no complications.

S. Pasli and colleagues suggest that surgery should be performed immediately. Although urgent surgical intervention may reduce the risk of thromboembolism, it is rarely chosen [13,42], yet reported cases describe favourable outcomes. H. Minami and colleagues reported surgery performed three days after AMI using the David–Komeda technique, in which a patch is sutured to the non-infarcted endocardial

surface, thereby reducing bleeding risk. However, they suggested that optimal timing for surgery may be approximately two weeks after AMI.

In a retrospective study, Joo Myung Lee and colleagues reported that no thromboembolic complications occurred after surgical treatment. In contrast, thromboembolic events were observed in 17% of patients treated with warfarin, despite maintaining a therapeutic INR (2.75 ± 0.43). However, it should be emphasised that left ventricular restoration surgery carries inherent risks, including postoperative ventricular dysfunction, bleeding, and arrhythmias [43].

A 2019 systematic review including 3,220 patients reported 30-day mortality of 7.1% and 5-year mortality of 29% following left ventricular restoration surgery [33]. Factors associated with worse outcomes included higher NYHA functional class, LVEF < 20%, age > 70 years, urgent surgery, concomitant mitral valve replacement, prolonged operative time, and higher EuroSCORE index [43,44].

Across all reviewed case reports, a recurring limitation was the lack of randomised controlled trials to guide management decisions in patients with mobile left ventricular thrombus and left ventricular aneurysm following AMI. Furthermore, comparative studies evaluating direct oral anticoagulants versus vitamin K antagonists in this clinical setting remain limited.

Table 1. Studies and their characteristics

Study	Study design	Patient Age (years)	Management Strategy	Thrombus' Characteristics	LVEF (%)	Clinical Outcomes / Complications
He et al (2023) [34]	Case report	33	Pharmacological/surgical	Hypermobile	63	Stroke before surgical treatment
Pasli et al (2022) [13]	Case report	74	Urgent surgery	Mobile	10-15	No complications reported
Shi et al (2022) [35]	Retrospective cohort study	-	21 patients pharmacological and 7 surgical	Mobile	-	Not mentioned separately
Demirci et al (2020) [36]	Case report	35	Surgical	Hypermobile	35	No complications reported
Bennett et al.	Case report	42	Pharmacological	Mobile	29	Thrombus dissolved in 6 days

(2018) [37]						
Kolekar et al (2015) [38]	Case report	61	Pharmacological	Mobile		No complications reported, thrombus dissolved
Oyedeji et al (2013) [39]	Case report	50	Pharmacological	Protrudent, mobile	30-35	No complications reported, thrombus dissolved
Lee et al (2013) [40]	Retrospective descriptive	-	Pharmacological/surgical	Mobile/ several thrombi	-	Thromboembolism in 5 of 9 patients
Cimadevilla et al (2010) [41]	Case report	37	Pharmacological	Mobile		Size reduction, loss of mobility (at 2 months)
Kikuchi et al (2009) [42]	Case report	59	Urgent surgery	2 mobile thrombi	38	No complications reported
Minami et al (2008) [19]	Case report	61	Pharmacological and after 3 days of surgery	Mobile	34	No complications reported

4. CONCLUSIONS

Based on the literature review, it can be concluded that current decision-making regarding the management of left ventricular aneurysm and mobile thrombus is largely based on institutional experience, case reports, and individualised clinical assessment of the patient. European and American treatment recommendations lack specificity, and no dedicated guidelines exist for the management of mobile left ventricular thrombus. The timing of surgery is determined on an individual basis, taking into account the patient's bleeding risk and the risk of thromboembolism; conservative management may be beneficial prior to surgical intervention [16].

Left ventricular volume restoration surgery is effective in cases of large, symptomatic aneurysms and markedly reduced left ventricular function (LV ejection fraction < 30%) [31]. Small aneurysms and associated thrombi are managed conservatively, as

aneurysmectomy in such cases does not provide significant hemodynamic benefit and may increase perioperative risk.

Pharmacological treatment with anticoagulants is more commonly selected as primary therapy, with therapeutic efficacy dynamically assessed and the need for surgery reconsidered accordingly, as LV reconstruction is safer after the formation of stable scar tissue within the weakened, fragile myocardial wall. Immediate surgery is associated with increased bleeding risk at suture lines and higher intraoperative risk, as it is difficult to differentiate viable from non-viable myocardium. However, in cases of high embolic risk when the LV thrombus is mobile and protruding from the ventricular wall, urgent thrombectomy is considered. Nevertheless, the literature lacks consensus and detailed guidelines regarding the optimal therapeutic strategy in such cases. Further randomised controlled trials are needed to guide evidence-based management and improve patient outcomes.

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