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Left ventricular thrombosis can still complicate acute myocardial infarction

A 62-year-old man with hypertension, type 2 diabetes mellitus, and hypercholesterolemia presented to the emergency department with substernal chest pain that started about 15 hours earlier while he was at rest watching television.

On examination, his pulse was 92 beats per minute and regular, his blood pressure was 160/88 mm Hg, and he had no evidence of jugular venous distention or pedal edema. Lung examination was positive for bibasilar crackles.

Electrocardiography revealed Q waves with ST elevation in leads I, aVL, V_4, V_5, and V_6 with reciprocal ST depression in leads II, III, and aVF.

His troponin T level on presentation was markedly elevated.

He underwent heart catheterization and was found to have 100% occlusion of the proximal left anterior descending artery. He underwent successful percutaneous coronary intervention with placement of a drug-eluting stent, and afterward had grade 3 flow on the Thrombolysis in Myocardial Infarction (TIMI) scale.

Echocardiography the next day revealed a mobile echo-dense mass in the left ventricular apex (**Figure 1**) and a left ventricular ejection fraction of 35%.

THE INCIDENCE OF LEFT VENTRICULAR THROMBOSIS IN ACUTE MI

1 What is the incidence of left ventricular thrombosis after acute myocardial infarction (MI), now that primary percutaneous coronary intervention is common?

- ☐ 0.1%
- ☐ 2%
- ☐ 20%
- ☐ 40%

Left ventricular thrombosis is a serious complication of acute MI that can cause systemic thromboembolism, including stroke.[1] Before thrombolytic therapy was available, this complication occurred in 20% to 60% of patients with acute MI.[2,3] But early reperfusion strategies, anticoagulation for the first 48 hours,

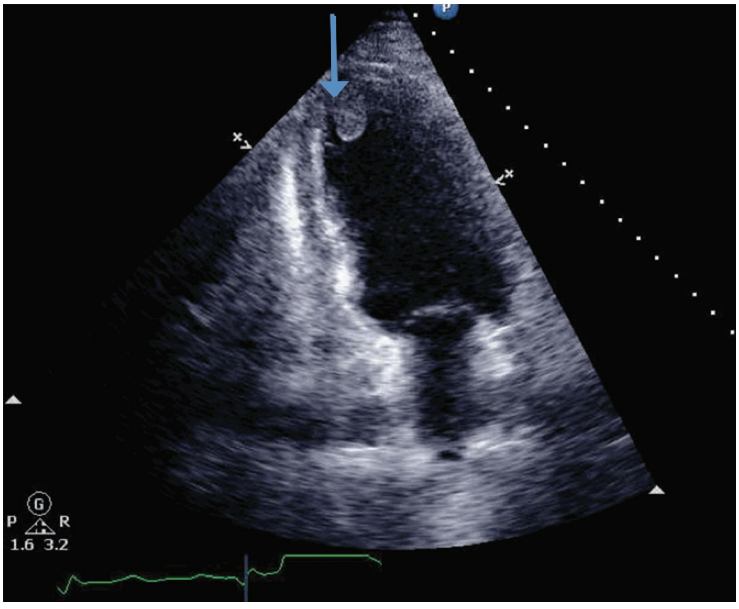

FIGURE 1. Transthoracic echocardiography, apical four-chamber view, shows thrombus in the left ventricular apical cavity. The blue arrow points to the well-demarcated thrombus adhering to the endocardium.

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TABLE 1

Incidence of left ventricular thrombosis after ST-segment elevation myocardial infarction

Authors	No. of patients[a]	Incidence of left ventricular thrombosis	Predictors of left ventricular thrombosis
Kalra and Jang,[4] 2000	71	4%	Anterior infarction
Nayak et al,[5] 2004	200	11%	Anterior infarction
Rehan et al,[6] 2006	92	4%	Anterior infarction
Zielinska et al,[7] 2008	2,911	2.5%	Anterior infarction Left ventricular ejection fraction < 40% Hypertension
Osherov et al,[8] 2009	642	6%	Severe mitral valve regurgitation Low left ventricular ejection fraction
Solheim et al,[9] 2010	100	15%	Higher peak creatine kinase level Larger infarcts Lower left ventricular ejection fraction
Shacham et al,[10] 2013	207	5%	Higher C-reactive protein levels Higher fibrinogen levels
Gianstefani et al,[11] 2014	1,059	4%	Low left ventricular ejection fraction Anterior infarction Use of glycoprotein IIb/IIIa inhibitors

[a] Most patients underwent percutaneous coronary intervention.

Risk factors for thrombosis: large infarct, low ejection fraction, anterior MI, hypertension, delayed therapy

and dual antiplatelet therapy have reduced the incidence of this complication significantly.

In the thrombolytic era, the incidence of left ventricular thrombosis was 5.1% in the *Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico* (GISSI) 3 study, which had 8,326 patients. A subset of patients who had an anterior MI had almost double the incidence (11.5%).[3]

The incidence has further declined with the advent of primary percutaneous coronary intervention, likely thanks to enhanced myocardial salvage, and now ranges from 2.5% to 15% (**Table 1**).[4–11] The largest observational study, with 2,911 patients undergoing percutaneous coronary intervention, reported an incidence of 2.5% within 3 to 5 days of the MI.[7] At our center, the incidence was found to be even lower, 1.8% in 1,700 patients presenting with ST-elevation MI undergoing primary percutaneous coronary intervention. Hence, of the answers to the question above, 2% would be closest.

Large infarct size with a low left ventricular ejection fraction (< 40%), anterior wall MI, hypertension, and delay in time from symptom onset to intervention were independent predictors of left ventricular thrombus formation in most studies.[7,12] The risk is highest during the first 2 weeks after MI, and thrombosis almost never occurs more than 3 months after the index event.[5,13–16]

WHAT IS THE PATHOGENESIS OF LEFT VENTRICULAR THROMBOSIS?

A large transmural infarct results in loss of contractile function, which causes stagnation

and pooling of blood adjacent to the infarcted ventricular segment. In addition, endocardial injury exposes tissue factor, which then initiates the coagulation cascade. To make matters worse, MI results in a hypercoagulable state through unclear mechanisms, which completes the Virchow triad for thrombus formation. Elevations of D-dimer, fibrinogen, anticardiolipin antibodies (IgM and IgG), and tissue factor have also been reported after acute MI.[17]

Thrombus formation begins with platelet aggregation at the site of endocardial damage, forming a platelet plug, followed by activation of clotting factors. These thrombi are referred to as "mural," as they adhere to the chamber wall (endocardium). They are composed of fibrin and entrapped red and white blood cells (**Figure 2**).

The natural course of thrombus evolution is established but variable. A left ventricular thrombus may dislodge and embolize, resulting in stroke or other thromboembolic complications. Alternately, it can dissolve over time, aided by intrinsic fibrinolytic mechanisms. On other occasions, the thrombus may organize, a process characterized by ingrowth of smooth muscle cells, fibroblasts, and endothelium.

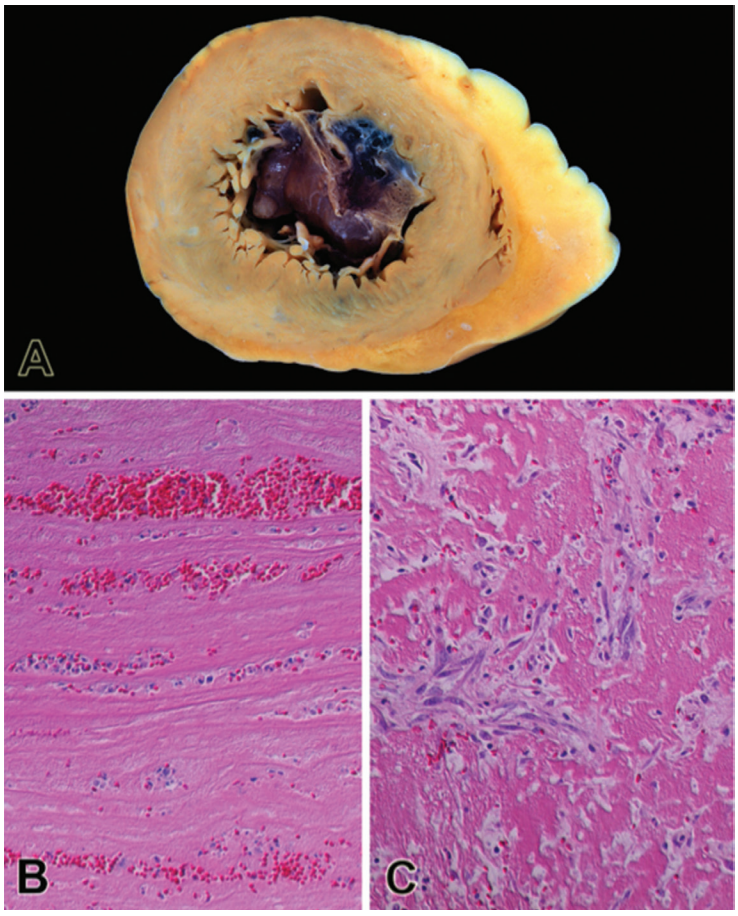

FIGURE 2. (A) A cross section of the apical segment of the left ventricle shows a mildly dilated cavity filled with mural thrombus. (B) Photomicrograph of an acute thrombus shows alternating layers of fibrin and platelet with red and white blood cells (hematoxylin and eosin, original magnification × 200). (C) Organization of a thrombus is characterized by infiltration of fibroblasts and newly formed capillaries (hematoxylin and eosin, original magnification × 200).

HOW IS LEFT VENTRICULAR THROMBOSIS DIAGNOSED?

2 What is the best imaging test for detecting a thrombus?

- ☐ Transesophageal echocardiography
- ☐ Transthoracic echocardiography
- ☐ Cardiac magnetic resonance imaging (MRI) without gadolinium contrast
- ☐ Cardiac MRI with gadolinium contrast

Evaluation of left ventricular function after acute MI carries a class I indication (ie, it should be performed).[18]

Echocardiography is commonly used, and it has a 60% sensitivity to detect a thrombus.[19] In patients with poorer transthoracic echocardiographic windows, contrast can be used to better delineate the left ventricular cavity and show the thrombus. Transesophageal echocardiography is seldom useful, as the left ventricular apex is foreshortened and in the far field.

A left ventricular thrombus is confirmed if an echo-dense mass with well-demarcated margins distinct from the endocardium is seen throughout the cardiac cycle. It should be evident in at least two different views (apical and short-axis) and should be adjacent to a hypokinetic or akinetic left ventricular wall. False-positive findings can occur due to misidentified false tendons, papillary muscles, and trabeculae.

Cardiac MRI with late gadolinium enhancement is now the gold standard for diagnostic imaging, as it accurately characterizes the shape, size, and location of the thrombus (**Figure 3**). Gadolinium contrast increases the

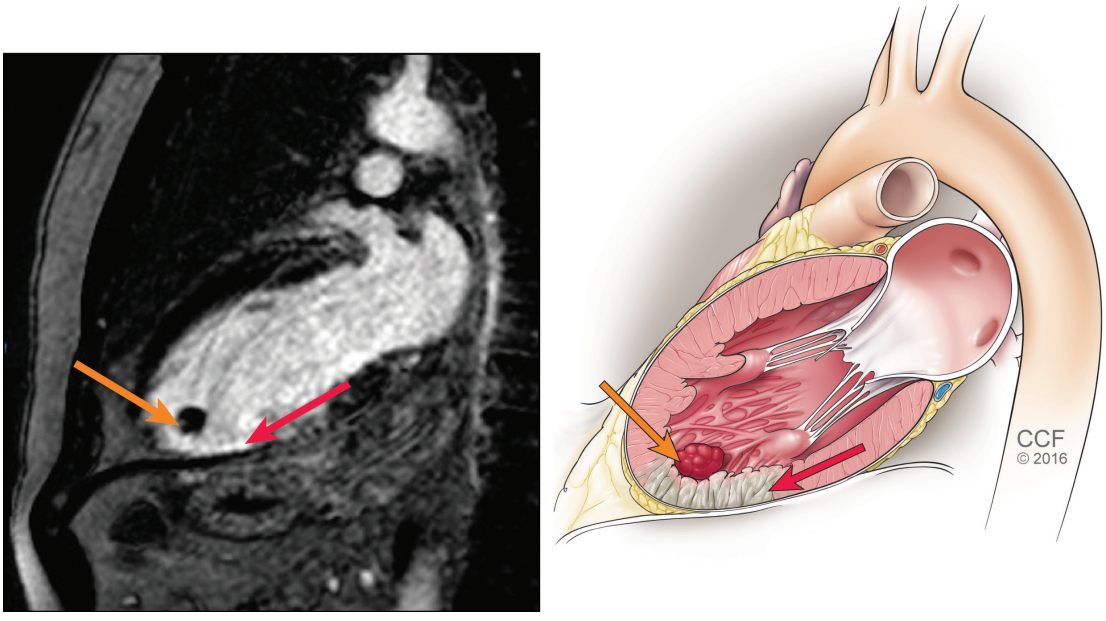

FIGURE 3. Cardiac magnetic resonance imaging with a delayed-enhancement phase-sensitive inversion recovery image, vertical long-axis view. The red arrow points to dense subendocardial delayed enhancement in the apex extending into the mid-inferior wall, consistent with scar in the distal left anterior descending artery territory. The orange arrow shows a nonenhancing mass in the apex, consistent with thrombus.

Triple antithrombotic therapy is associated with a bleeding risk of 4% to 16%

enhancement of the ventricular cavity, thus allowing easy detection of thrombus, which appears dark. Cardiac MRI with delayed enhancement has 88% to 91% sensitivity and 99% specificity to detect left ventricular thrombosis.[20,21] However, compared with echocardiography, routine cardiac MRI is time-intensive, costly, and not routinely available. As a result, it should be performed only in patients with poor acoustic windows and a high clinical suspicion of left ventricular thrombosis.

Delayed-contrast cardiac computed tomography can be used to identify left ventricular thrombosis, using absence of contrast uptake. The need to use contrast is a disadvantage, but computed tomography can be an alternative in patients with contraindications to cardiac MRI.

WHAT COMPLICATIONS ARISE FROM LEFT VENTRICULAR THROMBOSIS?

The most feared complication of left ventricular thrombosis is thromboembolism. Cardioembolic stroke is generally severe, prone to early and long-term recurrence, and associated with a higher death rate than noncardioembolic ischemic stroke.[22,23] Thrombi associated with thromboembolism are often acute and mobile rather than organized and immobile.[24] They may embolize to the brain, spleen, kidneys, and bowel.[25] In a meta-analysis of 11 studies, the pooled odds ratio for risk of embolization was 5.45 (95% confidence interval [CI] 3.02–9.83) with left ventricular thrombi vs without.[26] Before systemic thrombolysis and antiplatelet therapy became available, stroke rates ranged from 1.5% to 10%.[27–29]

In a meta-analysis of 22 studies from 1978 to 2004, the incidence of ischemic stroke after MI during hospitalization was around 11.1 per 1,000 MIs.[30] This study found that anterior MI was associated with a higher risk of stroke, but reported no difference in the incidence of stroke with percutaneous coronary intervention, systemic thrombolysis, or no reperfusion.

In a large prospective cohort study of 2,160 patients,[31] 259 (12%) had a stroke after MI. In multivariable analysis, age, diabetes, and previous stroke were predictors of stroke after MI. This study reported significantly fewer strokes in patients who underwent percutaneous coronary intervention than with other or no reperfusion therapies.[31]

TABLE 2

Bleeding risk after percutaneous coronary intervention: Triple thrombotic therapy vs dual antiplatelet therapy

Authors	No. of patients	Absolute difference in major bleeding with warfarin	Comments
Khurram et al,[49] 2006	107	6.6% higher	Bleeding defined as requiring > 2 units of packed red blood cells, or intraocular or disabling bleeding
DeEugenio et al,[52] 2007	194	11% higher	Hazard ratio 5.0 (95% confidence interval [CI] 1.4–17.8) with warfarin
Karjalainen et al,[40] 2007	478	5.6% higher	Odds ratio 3.4 (95% CI 1.2–9.3) with warfarin Increased stent thrombosis with warfarin-aspirin combination
Ruiz-Nodar et al,[53] 2008	426	5.9% higher	All patients had atrial fibrillation Mortality rate was higher without anticoagulation
Sarafoff et al,[54] 2008	515	1.7% lower (not statistically significant)	Both dual antiplatelet therapy and triple therapy had favorable efficacy and safety
Rossini et al,[55] 2008 [a]	204	0.9% higher (not statistically significant)	Bleeding rate was lower if the international normalized ratio was kept between 2 and 2.5: 4.9% vs 33% at 3 months

[a] This was a prospective study. The other studies in this table were retrospective.

ANTICOAGULATION TREATMENT

3 How would you treat a patient who has a drug-eluting stent in the left anterior descending artery and a new diagnosis of left ventricular thrombosis?

- ☐ Warfarin
- ☐ Aspirin and clopidogrel
- ☐ Aspirin, clopidogrel, and warfarin
- ☐ Aspirin and warfarin

The management of left ventricular thrombosis has been summarized in guidelines from the American College of Chest Physicians (ACCP) in 2012,[32] and from the American College of Cardiology/American Heart Association in 2013,[18] which recommend anticoagulation for at least 3 months, or indefinitely if bleeding risk is low, for all patients developing a left ventricular thrombus.

For patients with acute MI and left ventricular thrombosis, the ACCP guidelines recommend warfarin with a target international normalized ratio of 2.0 to 3.0 plus dual antiplatelet therapy (eg, aspirin plus clopidogrel) for 3 months, after which warfarin is discontinued but dual antiplatelet therapy is continued for up to 12 months.[32]

The European Society of Cardiology guidelines[33] recommend 6 months of anticoagulation. However, if the patient is receiving dual antiplatelet therapy, they recommend repeated imaging of the left ventricle after 3 months of anticoagulation, which may allow for earlier discontinuation of anticoagulation if the thrombus has resolved and apical wall motion has recovered. Therefore, most experts recommend 3 months of anticoagulation when used in combination with dual antiplatelet therapy and repeating echocardiography at 3 months to safely discontinue anticoagulation. The best answer to the question posed here is aspirin, clopidogrel, and warfarin.

Decisions about antithrombotic therapy may also depend on stent type and the patient's bleeding risk. With bare-metal stents, dual antiplatelet therapy along with anticoagulation should be used for 1 month, after which anticoagulation should be used with a single antiplatelet agent for another 2 months; after this, the anticoagulant can be discontinued and dual antiplatelet therapy can be resumed for a total of 12 months. Newer anticoagulants such as rivaroxaban, dabigatran, edoxaban, and apixaban may also have a role, but they have not yet been studied for this indication.

Surgical thrombectomy is rarely considered now, given the known efficacy of anticoagulants in dissolving the thrombus. It was done in the past for large, mobile, or protruding left ventricular thrombi, which have a higher potential for embolization.[34] Currently, it can be done under very special circumstances, such as before placement of a left ventricular assist device or if the thrombus is large, to prevent embolism.[35,36]

BLEEDING COMPLICATIONS WITH TRIPLE ANTITHROMBOTIC THERAPY

After stent placement, almost all patients need to be on dual antiplatelet therapy for a specified duration depending on the type and generation of stent used. Such patients end up on "triple" antithrombotic therapy (two antiplatelet drugs plus an anticoagulant), which poses a high risk of bleeding.[37] Consideration needs to be given to the risks of stroke, stent thrombosis, and major bleeding when selecting the antithrombotic regimen.[38] Triple antithrombotic therapy has been associated with a risk of fatal and nonfatal bleeding of 4% to 16% when used for indications such as atrial fibrillation.[39–41]

Risks of triple antithrombotic therapy (aspirin 80–100 mg, clopidogrel 75 mg, and warfarin) were compared with those of clopidogrel plus warfarin in the What Is the Optimal Antiplatelet and Anticoagulant therapy in Patients With Oral Anticoagulation and Coronary Stenting Trial,[37] which reported a significantly lower risk of major and minor bleeding with clopidogrel-plus-warfarin therapy than with triple antithrombotic therapy, 14.3% vs 31.7% (hazard ratio 0.40, 95% CI 0.28–0.58, $P < .0001$).

Additionally, the increased risk of major and minor bleeding associated with triple antithrombotic therapy has been confirmed in many observational studies; other studies found a trend toward lower risk with triple therapy, but this was not statistically significant (**Table 2**).[38,40,42–55] A large multicenter European trial is being conducted to compare dual antiplatelet therapy vs triple antithrombotic therapy in patients with left ventricular thrombosis.

CASE FOLLOW-UP

Our patient was started on warfarin, clopidogrel 75 mg, and aspirin 75 mg at the time of discharge. He was continued on warfarin for 3 months, at which time a follow-up echocardiogram showed no thrombus in the left ventricle. Warfarin was discontinued, and he had no thromboembolic complications.

TAKE-HOME POINTS

Left ventricular thrombosis after an acute MI is very important to detect, as it can lead to serious complications through arterial embolism.

The incidence of left ventricular thrombosis has declined significantly with the use of percutaneous coronary intervention. However, it may still occur in a small number of patients with larger infarcts owing to delay in revascularization or proximal (left main or left anterior descending) occlusions with larger infarct size.

Echocardiography, which is routinely performed after acute MI to assess myocardial function, uncovers most left ventricular thrombi. In high-risk cases, MRI with late gadolinium enhancement can increase the diagnostic yield.

Anticoagulation with warfarin is recommended for at least 3 months. Post-MI patients undergoing stent implantation may need triple antithrombotic therapy, which, however, increases the bleeding risk significantly. Large randomized trials are needed to guide physicians in risk stratification of such patients.

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