A Fatal Giant Left Ventricular Thrombus Protruding To the Left Atrium after Anterior Wall Myocardial Infarction

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Introduction

The incidence of post-infarction LV thrombus is greatly reduced by routine primary percutaneous intervention (PCI) and intensive anticoagulant therapy [1,2]. The risk of LV thrombus formation is higher within the first few weeks after acute myocardial infarction (MI), on the other hand spontaneous resolution also may occur (1). The risk of thrombus formation is higher in anterior MI than other infarct locations [3]. Two-dimensional echocardiography has high sensitivity (95%) and specificity (85-90%) in the diagnosis of LV thrombus [4,5]. LV thrombus is often located within the apical, rarely septal (11%) and inferoposterior wall (3%) [5]. We report a rare case of acute MI with LV thrombus is often located within the apical, rarely septal (11%) and inferoposterior wall (3%) [5]. We report a rare case of acute MI with unsuccessful revascularization and subsequent massive LV thrombus formation obliterating the LV and protruding to the LA causing a refractory cardiogenic shock and death.

Case

A 54 years old male was presented to the emergency room complaining of sudden chest pain and shortness of breath for the past 19 hours till first medical contact. His past medical history was significant for diabetes mellitus type 2, hyperlipidemia and tobacco use. Physical examination revealed heart rate of 103 beat per minute, blood pressure of 90/59 mmHg, respiratory rate of 24 per minute, O2 saturation of 88% at room air, normal heart sounds without murmurs and bibasilar lung crackles. Electrocardiogram (ECG) showed a sinus tachycardia, acute extensive anterior myocardial infarction and right bundle branch block (RBBB) (Figure 1). Chest radiography showed an increased interstitial markings and pulmonary vascular congestion. Initial laboratory work up showed an elevated Troponin I of 303.43 ng/ml and brain natriuretic peptide (bnp) of 350pg/ml. Patient was emergently transferred to the cardiac catheterization lab. Coronary angiogram revealed an acute total occlusion of the proximal left anterior descending coronary artery (LAD) with a thrombus (Figure 2) and attempted PCI with export thrombectomy, balloon angioplasty and drug-eluting stenting Promus Premier 2.5 × 20 mm × 1 of proximal to mid LAD was unsuccessful due to large thrombus burden with spontaneous and procedural distal embolization effectively occluding the microvasculature causing additional large irreversible infarcted myocardium (Figure 3). Patient had a sudden cardiac arrest due to ventricular fibrillation during his PCI attempt with successful cardiopulmonary resuscitation (CPR) for 3 minutes, was intubated on ventilator support. Complete heart block was noted and a temporary pacer wire was inserted and he eventually developed acute profound refractory cardiogenic shock to vasopressors and intra-aortic balloon pump requiring an escalation of mechanical support to impella and ultimately VA ECMO. Patient was not considered to be an ideal candidate for a salvage coronary artery bypass grafting (CABG) by cardio-thoracic surgery due to extensive irreversible myocardium infarction. Patient was loaded with 180 mg of Ticagrelor then 90 mg twice daily, loaded with 325 mg of Aspirin then 81 mg daily, 80 mg of Atorvastatin, Tirofiban (GPIIb/IIIa inhibitors) bolus of 0.4 mcg/kg/min and then 12 mcg/kg over 30 minutes and unfractionated Heparin infusion bolus of 60 units/kg then 12 units/kg infusion with cardiac intensive care unit admission. Initial transthoracic echocardiogram (TTE) revealed severely reduced left ventricle ejection fraction (LVEF) <15% and left ventricular regional wall motion abnormalities in the proximal LAD territories without evidence of LV thrombus (Figures 4, 5, 6, 7, 8, 9). Patient developed bleeding-coagulopathy diathesis with severe anemia requiring blood transfusion led to a temporary holding of the Heparin infusion in addition to further deteriorating cardiogenic shock despite VA ECMO support, therefore, a repeated TTE was performed showing worsening of LV function and massive thrombus obliterating the LV cavity causing inadequate cardiac filling and intractable cardiogenic

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shock (Figure 10). Patient was urgently taken to the OR for sternotomy and LA drainage with a 22-French venous cannula connected to the venous cannula for ECMO with perioperative transesophageal echocardiogram (TEE) showing the massive LV thrombus protruding to the LA (Figures 11, 12). He was considered a poor candidate for thrombectomy by cardiac thoracic surgery. This finding precluded him from further advanced heart failure therapy consideration. The patient experienced a low cardiac output syndrome deteriorated to multiple organ failure and death.

Discussion

Patients with large anterior MI associated with an LVEF <40% and anteroapical wall-motion abnormalities are at increased risk for developing LV mural thrombus because of stasis of blood in the ventricular cavity and endocardial injury with associated inflammation [5]. Before the advent of acute reperfusion interventions and aggressive antiplatelet and antithrombotic therapy in the peri-infarct period, LV mural thrombus was documented in 20% to 50% of patients with acute MI [9-12]. More recent studies indicate that the incidence of mural thrombus is ≈15% in patients with anterior MI and 27% in those with anterior STEMI and an LV ejection fraction <40% [9-12]. In the absence of systemic anticoagulation, the risk of embolization within 3 months among patients with MI complicated by mural thrombus is 10% to 20% [13-15]. RCTs to assess the value of antithrombotic therapy for prevention of mural thrombus and stroke in patients with STEMI have not been conducted [12-16].

The potential benefits of systemic anticoagulation for prevention of LV mural thrombus formation and stroke/arterial embolization must be balanced against the risks of major bleeding complications. Most patients with anterior STEMI will receive dual antiplatelet therapy (DAPT). Whether the addition of warfarin to DAPT provides incremental benefit in preventing stroke in high-risk patients is unknown. Among patients with documented LV thrombus, warfarin added to DAPT would prevent 44 nonfatal strokes at the same cost of 15 nonfatal extra cranial bleeds [17]. In addition, it was estimated that compared with DAPT, triple therapy would be associated with 11 fewer MIs per 1000 treated patients [17].

The duration of risk of thrombus formation and embolization after a large MI is uncertain, but the risk appears to be highest during the first 1 to 2 weeks, with a subsequent decline over a period of up to 3 months [17].

After 3 months, the risk of embolization diminishes as residual thrombus becomes organized, fibrotic, and adherent to the LV wall. However, patients with persistent mobile or protruding thrombus visualized by echocardiography or another imaging modality may remain at increased risk for stroke and other embolic events beyond 3 months [8].

To date, no studies have examined the efficacy and safety of newer antithrombotic agents, including dabigatran, rivaroxaban, apixaban, or fondaparinux, for prevention of LV thrombus or stroke in patients with acute MI. Therefore, if long-term anticoagulation is planned, Vitamin K antagonists (VKA) therapy remains the agent of choice for this indication [8]. Current ACC/AHA guidelines for the treatment of acute STEMI provide a Class IIa recommendation (Level of Evidence C) for VKA therapy in patients with STEMI and asymptomatic LV thrombus [8]. Treatment with VKA therapy (target INR, 2.5; range, 2.0–3.0) for 3 months is recommended in most patients with ischemic stroke or TIA in the setting of acute MI complicated by LV mural thrombus formation identified by echocardiography or another imaging modality (Class I; Level of Evidence C). Additional antiplatelet therapy for cardiac protection may be guided by recommendations such as those from the ACCP. (Revised recommendation). Treatment with VKA therapy (target INR, 2.5; range, 2.0–3.0) for 3 months may be considered in patients with ischemic stroke or TIA in the setting of acute anterior STEMI without demonstrable LV mural thrombus formation but with anterior apical akinesis or dyskinesis identified by echocardiography or other imaging modality (Class IIb; Level of Evidence C). (New recommendation)

Thrombectomy, thrombolysis and anticoagulant therapy are options [6]. Treating with fibrinolytic agents is not preferred because of the high risk of embolization. Surgical thrombectomy is preferred and appears to be clinically effective in cases with large, mobile pedunculated thrombi and it should be considered if anticoagulant therapy fails [18-20].

We describe a rare case of rapid giant LV thrombus formation protruding to the LA in the setting of late presentation of acute massive anterior ST elevation myocardial infarction caused a significant hemodynamic compromise despite therapeutic anticoagulation and dual antithrombotic therapy. Notably, the LV contractility was severely reduced. It remains unclear whether the impella device has contributed to LV thrombus formation. Whether more intensive anticoagulation therapy with higher activated clotting time (ACT) goals can prevent thrombus formation will have to be elucidated; it may be worth consideration in this case. Thrombolytic therapy was not appropriate in our case due to recent severe bleeding-coagulopathy diathesis. On the other hand, salvage CABG and surgical thrombectomy were not considered given his overall poor prognosis. In addition, a frequent echocardiographic surveillance may be helpful in guiding therapy decision making in the aftermath of acute large anterior wall MI with severely reduced LVEF and intractable cardiogenic shock.

Conclusion

LV thrombus is a rare complication of myocardial infarction but it can be fatal. Embolization of thrombus and thrombus size burden can cause severe hemodynamic sequelae with unfavorable morbidity and mortality outcome. Therefore early diagnosis and proper management are important.
Reference


