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Left Main Coronary Artery Thrombus: A Case Series with Different Outcomes

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Abstract. **Left main coronary artery (LMCA) thrombosis with acute myocardial infarction is an uncommon condition with an extremely high mortality. The small number of reported cases prevents the development of an evidence-based approach. Hence there are no clear-cut guidelines describing the best management approach for this condition. We describe our experience with six patients who presented with LMCA thrombosis and discuss the epidemiology, etiology and management options available for this high-risk subgroup.**

Key Words. **left main coronary artery thrombosis, acute myocardial infarction, anticoagulation**

Introduction

Left main coronary artery (LMCA) disease with acute myocardial infarction (AMI) has a reported mortality of 40–80% and represents a particularly high-risk subset of patients with AMI [1–5]. Although it might be more common than reported, most of our current knowledge regarding this entity comes from case-reports or small retrospective studies. The small number of reported patients prevents the development of an evidence-based treatment strategy. Hence therapy for LMCA thrombosis or LMCA with AMI has been inferred from experience with LMCA stenosis, in a non-AMI situation or non-LMCA AMI, both of which are different from LMCA thrombosis or LMCA occlusion with AMI. The present case series describes our experience with six patients presenting with AMI and LMCA thrombus with varied outcomes.

Case Presentations

Case 1

A 79 year-old male with type 2 diabetes, hypertension and hyperlipidemia presented with 12 hours of left-sided chest pain. On initial examination the patient was diaphoretic with cold clammy extremities and a systolic blood pressure (SBP) of 80 mmHg. Electrocardiogram (ECG) showed sinus rhythm with 3–4 mm ST segment elevation in leads V1–V5. Aspirin (325 mg), weight based intravenous heparin, and dopamine infusion were initiated. Transthoracic echocardiography (TTE) showed a dilated left ventricle with an ejection fraction (EF) of 25% and global hypokinesis, moderate mitral and severe tricuspid regurgitation. Initial troponin I was 203 ng/ml (normal < 1 ng/ml). Emergent cardiac catheterization within 1 hour of presentation showed mild luminal irregularities in the LMCA with a large thrombus in the distal LMCA occluding the left anterior descending coronary artery (LAD) and extending into the left circumflex artery (LCX) (Fig. 1). The right coronary artery (RCA) was normal. An intra-aortic balloon pump (IABP) was inserted and temporary transvenous pacing was initiated (80 beats per min [bpm]) for symptomatic bradycardia (30 bpm) that developed during cardiac catheterization. The patient was referred for immediate coronary artery bypass grafting (CABG). While being prepped on the operating table the patient developed cardiac arrest and despite cardiopulmonary resuscitation died due to resistant ventricular fibrillation followed by asystole.

Case 2

A previously healthy 48 year-old female presented with a 2 hour history of chest pain and shortness of breath. Initial SBP was 100 mm Hg and ECG showed sinus tachycardia (120 bpm) with 1–2 mm ST segment depression in leads V1–V6. She developed worsening respiratory distress over the next hour requiring intubation for acute pulmonary edema. The patient received intravenous furosemide and weight-based unfractionated heparin. Troponin I was 10 ng/ml after 10 hours. Cardiac catheterization performed the next day showed a thrombus in the distal LMCA (Fig. 2A) with luminal irregularities at the ostia of LAD and RCA. Due to the absence of on-site surgical back-up at the outside hospital and the critical condition preventing immediate transfer, eptifibatide infusion was added and continued for 3 days. The patient was weaned off the ventilator

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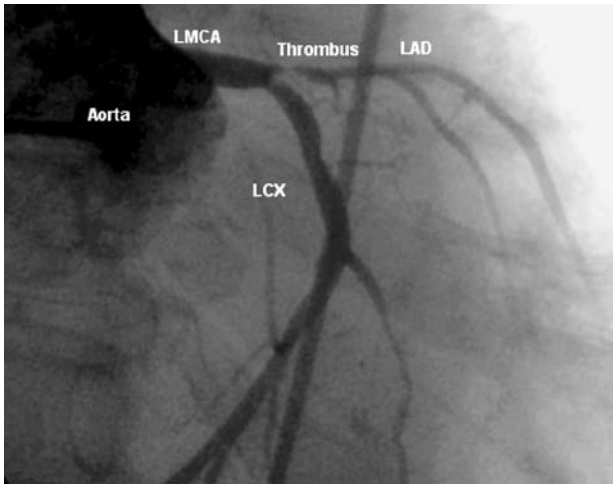

Fig. 1. *Right anterior oblique view showing thrombus in distal LMCA extending to the LAD and LCx artery.*

and transferred to our hospital on the 4th day. Repeat coronary angiography showed complete resolution of the LMCA thrombus (Fig. 2B) with luminal irregularities in the LMCA and LAD. TTE prior to discharge showed normal left ventricular size and EF (60%), mild hypokinesis of the mid-distal anterior and anteroseptal segments, and mild mitral regurgitation. The LDL cholesterol was found to be elevated (188 mg/dl). She was discharged on aspirin, clopidogrel, a beta-blocker, angiotensin converting enzyme inhibitor, and statin. She has been asymptomatic for ten months after discharge.

Case 3

A 63 year-old male smoker with hypertension and chronic alcohol abuse, presenting with shortness of breath and chest discomfort, had a cardiac arrest in the emergency room from which he was successfully resuscitated. Post-resuscitation SBP was 100 mmHg. ECG showed sinus tachycardia and 1–2 mm ST depression in V1–V6. Chest computed tomography showed small pulmonary emboli in the left upper pulmonary artery branches. The patient was given aspirin, weight-based intravenous unfractionated heparin infusion and admitted to the intensive care unit. Troponin I was 6.9 ng/ml after 6 hours and peaked (26.7 ng/ml) after 14 hours. Coronary angiography the following day showed a 40% stenosis in the distal LMCA with a thrombus extending into the LCX and a complete occlusion of the distal RCA. IABP was placed and an urgent 3-vessel CABG performed 3 hours later. The patient has been symptom-free at 2 months follow-up. Subsequent TTE 3 months later demonstrated a normal left ventricular size, EF of 50%, without regional wall motion abnormalities, mild tricuspid regurgitation and absence of patent foramen ovale.

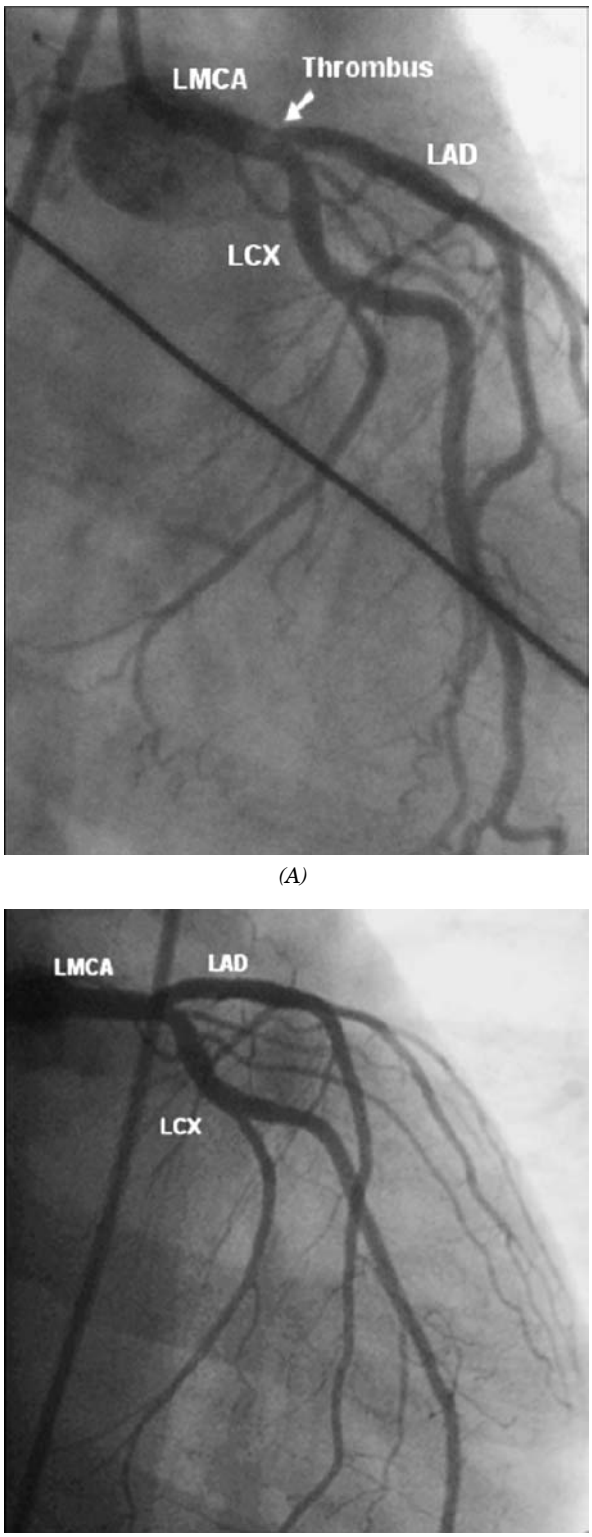

Fig. 2. *(A) Antero-posterior view showing thrombus in distal LMCA extending to LAD. (B) Antero-posterior view in the same patient showing resolution of thrombus after aspirin, eptifibatide and heparin therapy.*

Case 4

A 61 year-old male smoker with type 2 diabetes, hypertension, hyperlipidemia, and peripheral vascular disease presented with chest pain for 2 hours. SBP was 110 mmHg. ECG showed sinus rhythm and 1–2 mm ST segment depression in leads V1–V5. Serum creatine kinase-MB at presentation was normal but increased to 22.1 ng/ml (normal 0–5) 12 hours after presentation. He was given 325 mg aspirin and weight-based intravenous heparin and monitored in the coronary care unit. Coronary angiogram performed the next day showed luminal irregularities with a thrombus in the distal LMCA, 70% proximal LAD stenosis, 70% mid-left circumflex stenosis and total occlusion of the mid-RCA. TTE showed normal left ventricular size, EF of 50%, and mild mitral regurgitation. He underwent a 3-vessel CABG the same day. The patient has been symptom-free at follow-up and TTE 3 months later showed EF of 55% with basal inferior and posterior segment hypokinesis and mild mitral regurgitation.

Case 5

A 51 year-old male with type 2 diabetes, hyperlipidemia and hypertension developed syncope at home. Emergency Medical Services (EMS) personnel found patient to be in cardiac arrest and brought him to the emergency room with cardiopulmonary resuscitation in progress. After 45 minutes the patient regained a SBP of 100 mmHg on intravenous dopamine infusion. ECG at the time showed sinus rhythm and 2–3 mm ST segment elevation in V1–V3 with ST segment depression in leads II, III, aVF and V3–V6. Coronary angiography performed within 1 hour showed a 40% stenosis in the distal LMCA with a thrombus extending into the LAD and total occlusion of the RCA. Aspirin, IABP support, weight-based unfractionated heparin and eptifibatide bolus (180 mcg/kg) followed by infusion (2 mcg/kg/min) were started. Troponin I, initially normal, peaked at 50.1 ng/ml after 14 hours. Emergent CABG was planned but deferred as the patient was deeply comatose and the prospects of functional neurological recovery were considered poor. The following day the patient developed resistant ventricular fibrillation and died despite resuscitative measures.

Case 6

A 64 year-old previously healthy smoker presented with chest pain and dyspnea for 2 hours. Vital signs included a blood pressure of 118/99, an irregularly irregular heart rate of 145 bpm and a respiratory rate of 22/min. There were clinical signs of heart failure. ECG showed atrial fibrillation with ST segment depression in II, III and aVF and V1–V6. He received intravenous metoprolol and furosemide with decrease in heart rate, resolution of chest pain and improvement of dyspnea. Troponin I was 11 ng/ml initially and peaked at 102 ng/ml. TTE showed global hypokinesis (EF 10%) and mild tricuspid regurgitation. Intravenous heparin and furosemide, oral aspirin, metoprolol and enalapril were begun with improvement of symptoms. Coronary angiography was performed 2 days after admission. There was a large intraluminal filling defect in the distal segment of the LMCA with total occlusion and a 50% stenosis in the mid-segment of the RCA with collaterals to LAD and LCX. An IABP was inserted. A 3-vessel CABG with plication of a left ventricular apical-lateral aneurysm was performed the following day.

Discussion

Epidemiology of LMCA thrombosis

LMCA disease was first described by James Herrick in 1912 [6]. His patient was a 55 year-old male who died of cardiogenic shock 52 hours after symptom onset. Autopsy revealed extensive necrosis of the left ventricle with total occlusion of the LMCA by a thrombus overlying an area of atherosclerotic narrowing. In a report of 2,800 AMI patients treated with primary PTCA, 24 (0.8%) had acute subtotal or total LMCA occlusion [1]. A higher incidence was found in a post-mortem study from Portugal in 167 patients dying from AMI without thrombolysis [7]. Coronary thrombosis was found in 74% patients of which 5% had LMCA thrombus [7]. The true incidence of LMCA thrombosis thus remains unclear as many patients may die from sudden cardiac death and not reach the hospital. On the other hand, autopsy series suffer from the selection bias of studying only AMI patients who died. The true incidence likely falls somewhere between <1–5%.

Etiology of LMCA thrombosis

LMCA thrombus formation may arise from spontaneous plaque rupture [8,9], or secondary to coronary manipulation during angiography [10], balloon angioplasty [11], stent implantation [12], or intracoronary vascular ultrasound [13,14]. Uncommon causes include coronary vasospasm [15], coronary artery embolism from thrombus or vegetation arising from the aorta [16] or native/mechanical valves [17,18] and paradoxical embolism [19,20]. Prothrombotic conditions including factor V Leiden [21], essential thrombocythemia [22], and post-partum state [23] may be associated with LMCA thrombosis. Other rare causes include cocaine use [24], blunt chest trauma [25], and Kawasaki's disease [26]. In the present series coronary angiography showed luminal irregularities or milder degree of stenosis in the LMCA suggesting that the likely cause of the thrombus was rupture of an atherosclerotic plaque in the LMCA.

Clinical presentation of LMCA thrombosis

Most reported cases of LMCA thrombosis have presented with acute pulmonary edema requiring ventilatory support, AMI (ST/ non-ST segment elevation), cardiac arrest, cardiogenic shock or sudden cardiac death [1–5,27]. In one study, 63% of the 24 patients with LMCA occlusion presented with cardiogenic shock, 42% were intubated and all required IABP [1]. Age > 61 years, male sex, and a smoking history had a high prevalence in this patient cohort. However, no specific predictors of LMCA thrombosis have been identified so far. Electrocardiography has variably demonstrated ST segment elevation in most patients or diffuse ST segment depression in the anterior precordial leads, and cardiac enzymes are typically elevated in most cases [1–5].

There is no uniformity in the angiographic definition of intracoronary thrombus. We used the most conservative definition of intraluminal globular filling defects in multiple angiographic projections, haziness, or a convex margin in presence of total occlusion to diagnose a thrombus. Detection of luminal irregularities or milder degree of stenosis in LMCA is important and suggests the presence of an atherosclerotic plaque potentially responsible for thrombus formation. Ostial atherosclerotic plaque of the LAD or LCX may predispose to thrombosis that may extend retrogradely into the LMCA [28]. LMCA occlusion with AMI may be associated with triple vessel coronary artery disease in 50% of patients and RCA stenosis (defined as > 75% diameter stenosis) in 17% [1].

Management of LMCA thrombosis

The reported treatment options for LMCA thrombosis include emergent CABG [2,29,30], emergent LMCA stenting [1,3–5,9,10,31–39], anticoagulation with heparin or glycoprotein IIb/IIIa inhibitors [21,22] and thrombolysis [40–42]. Since most patients have hemodynamic compromise, support with IABP is usually initiated [1]. The choice of management of LMCA thrombus has been largely dependent on the acuity or severity of presentation and presence of immediate on-site surgical back-up.

CABG

Patients with LMCA thrombus are generally referred for emergent or urgent CABG although mortality is high [27]. Emergent CABG in patients with LMCA occlusion and AMI has a high mortality ranging from 46–50% [2,29,30]. In a series of 13 patients with AMI and LMCA disease, there was 46.2% perioperative mortality (53% for patients in cardiogenic shock) [2]. In a series of 13 patients with LMCA occlusion and AMI, (7 with angioplasty followed by CABG, 6 with CABG alone), in-hospital mortality was 46% [29]. There is one report of surgical thrombus removal from LMCA [19].

It is also not clear whether patients with successful LMCA PCI should undergo CABG. In a 6 patient series with left main occlusion and AMI undergoing PCI, three patients died [30]. The survivors underwent CABG and were alive at 2–8 years follow-up.

Emergent PCI

PCI of LMCA during AMI with [9,10,14,24,31–39] or without [1,3–5,40] LMCA thrombus have been reported. In 24 patients, in-hospital mortality was 43% with stents versus 80% with balloon angioplasty alone (p = ns), 58% overall and 80% with cardiogenic shock [1]. In a prospective multicenter international registry of 277 patients who underwent LMCA PCI, 40 patients had emergency reperfusion for AMI (23 PTCA, 17 stent) [2]. All but 3 patients were in cardiogenic shock. Angiographic success was 88%, and in-hospital mortality 70% with angioplasty alone versus 35% with stent (p = ns); One-year survival was 35% with angioplasty versus 53% with stent (p = ns); In-hospital CABG was performed in 18% (6 in PTCA, 2 in stent group). In another study among 6 patients with AMI and LMCA PCI, 5 died, 3 in the catheterization laboratory after successful LMCA dilatation and 2 in the hospital [5]. In the Rapamycin Eluting Stent Evaluated At Rotterdam Cardiology Hospital (RESEARCH) registry of 31 patients with LMCA disease, 5 had AMI [4]. Three patients (all with cardiogenic shock) died in-hospital; in the remaining 2, there was no restenosis at 5 months. Another group with anterolateral AMI and LMCA PCI (n = 38, 28 with cardiogenic shock) reported 76% successful reperfusion and 55% in-hospital mortality (71% in patients with cardiogenic shock) [40]. There are no reports in the literature regarding mechanical rheolytic thrombectomy for LMCA thrombosis.

PCI versus CABG

CABG has generally been regarded as the primary therapeutic choice for LMCA occlusion and AMI. This choice is an extrapolation from large studies (Veterans Administration Cooperative Study [randomized trial], Coronary Artery Surgery Study [registry]) of CABG versus medical therapy for CAD, that showed a survival advantage with surgical revascularization in LMCA disease [43,44]. Patients with AMI and LMCA thrombosis represent a significantly higher risk subgroup than patients with chest pain and LMCA stenosis described in these older clinical trials. In-hospital mortality with emergent CABG seems to be comparable to emergent PCI in AMI with LMCA occlusion [1–5]. However, no head-to-head comparison between PCI versus CABG has been performed in this setting. As such, treatment should be individualized based on specific circumstances. In critical situations without on-site surgical back-up, or when the patient is too unstable to be taken for

emergent CABG, unprotected LM stenting may represent a therapeutic alternative.

Anticoagulation / antiplatelet therapy

Intravenous or intracoronary thrombolysis or glycoprotein IIb/IIIa inhibitors have been reported to be effective in individual cases [12,21,22,41,44–47]. Pulse spray thrombolysis, a method that uses the synergistic mechanical maceration of thrombi and spray of thrombolytic solution into the thrombus has been utilized in one patient with alteplase 24 million units/100 ml of saline (starting dose of 0.7 ml bolus, 5 times per minute, 3 ml/sec compulsive force). TIMI 3 flow was achieved after 25 minutes [41]. The technique, however, may induce distal embolization. Besides, waiting 25 minutes before achievement of normal flow may not be realistic for most patients. Tissue plasminogen activator (tPA) and streptokinase have been used effectively in individual cases [46,47]. Prolonged abciximab infusion with aspirin and heparin in one case and aspirin and ticlopidine in another case was associated with angiographic LMCA thrombus resolution [21,22]. As adjunctive therapy to PCI for LMCA occlusion and AMI, abciximab was used in 5 patients in the ULTIMA registry [3].

Case Series Analysis

In the present series (Table 1), all patients had evidence of LMCA atherosclerosis suggesting plaque rupture leading to the acute presentation.

Four of 6 patients survived in this case series; 2 (cases # 3 + 4) underwent urgent CABG after diagnosis and survived; the other 2 (cases # 2 + 6) received prolonged anticoagulation, 1 eventually had CABG and the other demonstrated angiographic thrombus resolution. Only 1 patient (case # 6) had complete occlusion of LMCA but had well developed collaterals from RCA to LAD and LCx suggesting gradual LMCA stenosis. These collaterals probably allowed for his survival. No patient had LMCA PCI.

Two of our cases were given eptifibatide with 1 patient surviving. This patient (case #2) received prolonged intravenous heparin and eptifibatide with angiographic thrombus resolution. This case highlights the clinical dilemma as to the best treatment strategy for LMCA thrombosis in the patient without hemodynamic compromise. These are the first 2 reported cases of LMCA thrombosis in the literature using this glycoprotein IIB/IIIa inhibitor.

Long-term prognosis and anticoagulation regimen in survivors is unknown. One of the survivors had resolution of the thrombus with anticoagulation was

Table 1. *Patient characteristics and outcomes in LMCA thrombosis*

No.	Age/ sex	Risk factors	Presentation	EKG	Trop (ng/ml)	LMCA Stenosis (%)	Treatment	Outcome	EF (%)
1	79/M	DM, HTN HLP	CP, cardiogenic shock	3–4 mm ST↑ in V1–V5	203	20	Aspirin, heparin, dopamine, IABP*	Death	25****
2	48/F	HLP	Pulmonary edema, CP	1–2 mm ST↓ in V1–V6	10	10	Aspirin, clopidogrel, heparin, eptifibatide	Survival	60 (1 week post)
3	63/M	HTN, S	Cardiac arrest	1–2 mm ST↓ in V1–V6	26.7	40	Aspirin, heparin, IABP, emergent CABG	Survival	50 (3 mo post)
4	61/M	DM, HTN, HLP, PVD, S	CP	1–2 mm ST↓ in V1–V5	**	10	Aspirin, heparin, emergent CABG	Survival	55 (3 mo post)
5	51/M	DM, HTN HLP	Cardiac arrest	2–3 mm ST↑ in V1–V3	50	40	Aspirin, heparin, IABP, eptifibatide	Death	–
6	64/M	S	CP, SOB, atrial fibrillation	2 mm ST↓ in II, III, aVF, V1–V6	102	***	Aspirin, heparin, IABP, CABG	Survival	10****

CABG = coronary artery bypass surgery; CP = chest pain; DM = diabetes mellitus; EF = ejection fraction; F = female; HLP = hyperlipidemia; HTN = hypertension; IABP = intra-aortic balloon pump; M = male; PVD = peripheral vascular disease; S = smoking; SOB = shortness of breath; trop = troponin.

*Patient died before CABG.

**Creatine phosphokinase (CK-MB) myocardial band in this patient was 22 ng/ml (normal < 5 ng/ml).

***Patient had 100% occlusion with thrombus precluding estimation of LMCA stenosis.

****EF on the day of presentation.

started on long-term clopidogrel with aspirin. The other three with CABG were continued on aspirin only. In our case series, 3 out of 4 survivors were asymptomatic at follow-up with normal left ventricular EF (range 50–60%). The fourth survivor has been recently discharged.

Prognosis

Cardiogenic shock is a grave prognostic marker after AMI in general [48] and in the subgroup with LMCA thrombus (up to 94% mortality) [48]. Some authors have suggested that collateral circulation from the RCA is the major determinant for survival [50,51]. The combined coexistence of dominant RCA, intercoronary collaterals and incompletely occluded LMCA appear to be important deterministic factors for survival [52].

Conclusion

Patients with LMCA thrombus represent a high-risk subgroup of AMI patients that characteristically present with acute pulmonary edema, cardiac arrest, cardiogenic shock and sudden cardiac death. Clinically there are no predictors of LMCA thrombosis. No prospective study has described a preferred treatment modality. Almost half the reported patients with LMCA thrombosis die irrespective of therapy instituted and the mortality increases if associated with cardiogenic shock. Whether therapies including PCI with stenting, particularly using drug eluting stents or mechanical rheolytic thrombectomy with aggressive antiplatelet/anticoagulation therapy will have an impact on the disease remains to be seen.

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