

Scary Acute Left Main Coronary Artery Thrombus as an Initial Presentation of a Hereditary Thrombophilia: When to Go Out of Routine?

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ABSTRACT

Patients with either hereditary or acquired thrombophilia can present with arterial and venous thrombotic complications. However, it is unclear to whom the thrombophilia panel should be assessed, particularly in patients presenting with a common cardiovascular risk factor and acute coronary thrombus. Herein, we presented the management of an active smoker female patient who presented to our emergency room with inferior acute ST-segment elevation myocardial infarction, and hereditary thrombophilia has been diagnosed due to the presence of substantial left main coronary artery thrombus.

Keywords: acute coronary syndrome, coronary thrombus, thrombophilia

INTRODUCTION

Acute left main coronary artery (LMCA) occlusion is a life-threatening condition that can cause ventricular arrhythmias, cardiogenic shock, and sudden cardiac death. The LMCA as an infarct-related artery in acute ST-segment elevation myocardial infarction (STEMI) is rare [1]. Acute myocardial infarction may occur in patients with no traditional risk factors and who also have normal coronary arteries. No consensus exists in clinical practice as to which patients with acute coronary syndromes and coronary artery disease should be tested for thrombophilia. Mutations related to the coagulation pathway can be investigated in patients who are unexpectedly having coronary artery disease. Herein, we presented the management of an active smoker female patient who presented to our emergency room with acute inferior STEMI, and hereditary thrombophilia has been diagnosed due to the presence of a substantial LMCA thrombus.

CASE

A 64-year-old female patient with a diagnosis of anxiety disorder applied to the emergency room with the first episode of resting angina pectoris lasting more than 30 minutes. Her past medical history was unremarkable except for a two package year of smoking. She had not been taking any medication, including oral contraceptives. Family history was also unremarkable for premature coronary artery disease. Physical examination on

admission revealed no abnormal findings with stable vital signs. Initial laboratory test results were within normal reference limits, including cardiac biomarkers. The chest X-ray was also normal. 12-lead electrocardiography on admission indicated infero-postero-lateral STEMI (ST-segment elevation at II, III, aVF, V₄₋₆, V₇₋₉ leads) (Figure 1). Bedside emergent echocardiography revealed a motion abnormality at the posterior and inferior walls. Emergency room medications included unfractionated heparin, ticagrelor, aspirin, and statin administration. She has been immediately transferred to the catheter angiography room. Coronary angiography showed a non-dominant right coronary artery (RCA) and a huge thrombus at the left main coronary artery, which has moved along the left circumflex coronary artery during contrast injection (Figure 2, Video 1). However, none of the coronary segments revealed a complete occlusion. There were also atherosclerotic lesions at the ostial and mid segments of the left anterior descending (LAD) artery. The thrombus aspiration catheter was non-available in our catheter laboratory at the time of coronary angiography. Following intracoronary bolus tirofiban administration, no change in thrombus size was observed. Continuous infusion of unfractionated heparin and tirofiban was given for 24 hours. Lipoprotein (a) level was 49.5 mg/dl (normal reference range: 5.6-33.8 mg/dl), low-density lipoprotein (LDL)-cholesterol level was 133.4 mg/dl, high-density lipoprotein (HDL)-cholesterol level was 56.7 mg/dl, triglycerides

How to cite: Kilic S, Canpolat U, Ates AH, Aytemir K (2023) Scary Acute Left Main Coronary Artery Thrombus as an Initial Presentation of a Hereditary Thrombophilia: When to Go Out of Routine?. *Eur J Ther.* 29(2):247-249. <https://doi.org/10.58600/eurjther.20232902-446.y>

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Received: 06.04.2023 • **Accepted:** 26.04.2023 • **Published Online:** 06.05.2023

level was 99 mg/dl. After intensive antiplatelet and anticoagulant therapy, a control coronary angiography revealed no thrombus in any coronary segment except the LAD artery distal segment (Video 2). A 3.0x20 mm drug-eluting stent (DES) and a 4.5x12 mm bare-metal stent (BMS) were implanted in the LAD artery mid and ostial 70% lesions. Percutaneous coronary angioplasty was also performed with a 2.0x12 mm balloon in the distal LAD artery segment, which resulted in a TIMI 1 flow. Her detailed physical examination and systemic symptom assessment were negative for any rheumatologic disease.

Rheumatologic markers, including ANA, ENA, RF, C3, C4, lupus anticoagulant, protein C activity, anti-dsDNA, anticardiolipin antibody IgG & IgM were negative. Only the beta-2 glycoprotein IgM level was 46.8 RU/ml, above the reference limit (normal reference level of <20 RU/ml). Fibrinogen level was 444.93 mg/dl (normal reference range: 180-350 mg/dl), and homocysteine level was 15.4 µmol/L (normal reference level of <15 µmol/L). Genetic thrombophilia panel including factor V Leiden, prothrombin, MTHFR (677), MTHFR (1298), PAI mutation analysis showed the MTHFR (677) and MTHFR (1298) heterozygote mutations and PAI 4G/4G homozygote mutation. Antiplatelet therapy on discharge was ordered as acetylsalicylic acid 1x100 mg, ticagrelor 2x90 mg, and warfarin 5 mg for 6 months. Maintenance antiplatelet therapy was planned as ticagrelor 2x90 mg and warfarin 5 mg after 6 months.

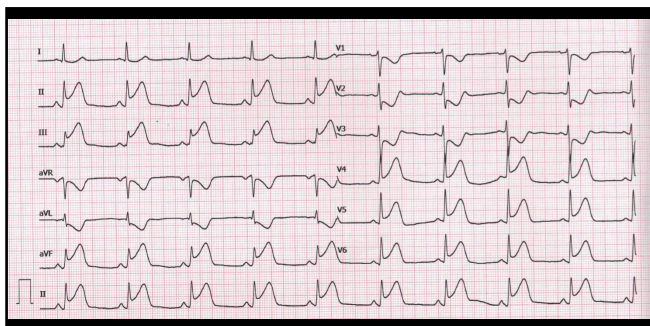


Figure 1. 12-lead electrocardiography of the patient on admission to the emergency room was consistent with an inferior ST-segment elevation myocardial infarction.

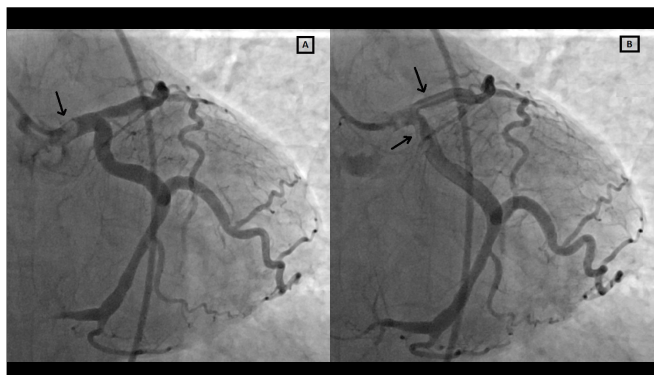
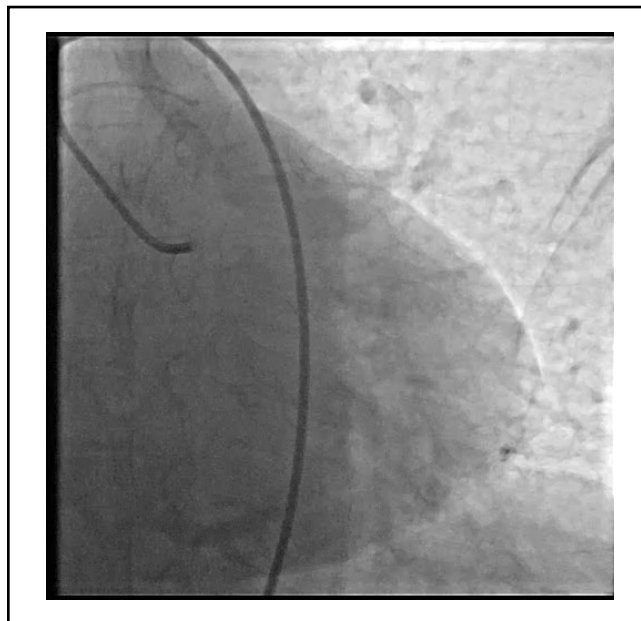
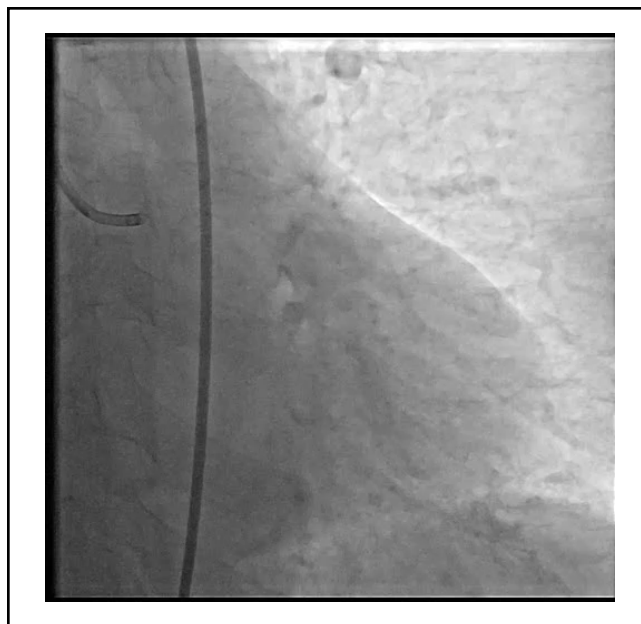


Figure 2A-B. Initial coronary angiography revealed a massive thrombus at the left main coronary artery moving along the left circumflex coronary artery.

Video 1. Initial coronary angiography showed a massive thrombus at the left main coronary artery moving along the left circumflex coronary artery.



Video 2. Control coronary angiography revealed no thrombus at the left main coronary artery.



DISCUSSION

Atherosclerotic plaque rupture is the main pathophysiological mechanism of acute myocardial infarction. Hypertension, diabetes, hyperlipidemia, and smoking are major modifiable risk factors, but rarely the underlying cause may be thrombophilia. Inherited hyper coagulopathies usually lead to the formation of venous thrombi; however, they uncommonly lead to arterial thrombosis such as in coronary arteries [2].

Acute LMCA thrombus is a very dangerous condition due to large myocardial tissue under threat and that requires immediate management and therapy. Emergency primary percutaneous coronary intervention, surgery, or thrombolysis are therapeutic options. Besides maximal antiplatelet and anticoagulant therapy, Gp IIb/IIIa inhibitors or thrombolytic agents can also be used. If the preference will be primary percutaneous coronary intervention, an adequate thrombus removal before stenting is an important factor that predicts procedural success, infarct size, long term clinical outcomes [3]. But the procedure may be complicated by distal embolization of thrombus with infarct extension [4]. In addition, thrombus aspiration can be an option especially if coronary diagnostic angiography reveals a massive thrombus like the patient described herein. However, ischemic cerebral events may occur due to embolization during the aspiration of such a thrombus in the LMCA body. In our case, after an intracoronary tirofiban bolus dose administration during the procedure, infusion of unfractionated heparin and tirofiban was continued for 24 hours. We did not administrate intracoronary thrombolytic because of normal distal coronary flow. Thrombus aspiration may be a treatment option in these patients. But the thrombus aspiration catheter was non-available in our catheter laboratory during the procedure. Furthermore, thrombus aspiration in our case might have an increased risk for cerebral or systemic thrombus embolization because of the ostial location of the thrombus.

In addition to conventional risk factors, patients with acquired or genetic thrombophilia can also present with acute coronary syndrome. It is little known in which patients mutations related to the coagulation pathway should be investigated, particularly in patients presenting with common cardiovascular risk factors and acute coronary thrombus. These mutations should be kept in mind in patients, especially without traditional risk factors or family history that predict premature coronary artery disease. When the diagnosis of thrombophilia is confirmed, adding anticoagulant agents to antiplatelet therapy may be beneficial in preventing recurrent events at long-term follow-up.

CONCLUSION

Besides well-known common cardiovascular risk factors like hypertension, diabetes, and smoking for coronary artery disease and acute coronary syndromes, acquired or genetic thrombophilia should be kept in mind and investigated, particularly in extreme cases like the presented case. The detection of abnormality in the coagulation pathway or process in such cases may change the management of patients in whom the administration of appropriate therapies can prevent further thrombotic events.

Funding: The authors declared that this study has received no financial support.

Peer-review: Externally peer-reviewed.

Competing interest for all authors: No financial or non financial benefits have been received or will be received from any party related directly or indirectly to the subject of this article. The authors declare that they have no relevant conflict of interest.

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