



Short Communication

Ostial left main coronary artery chronic total occlusion presenting as chronic stable angina

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ABSTRACT

Significant left main coronary artery (LMCA) disease is found in 5–6% of all patients undergoing coronary angiography. It usually presents as acute coronary syndrome and is commonly associated with multi-vessel coronary artery disease (CAD). Complete occlusion of LMCA is a much rarer finding, since these patients usually present as unstable angina, myocardial infarction and cardiogenic shock. We report a case of a young female, who presented with chronic stable angina and had an isolated chronic total occlusion (CTO) of LMCA with no lesions in the other coronary arteries. Aortogram failed to demonstrate the stump of occluded LMCA and demonstrated the filling of the left coronary system from the right coronary artery. Apart from dyslipidemia, she had no other risk factors for CAD. She was extensively evaluated for non-atherosclerotic causes of LMCA CTO including vasculitis. She underwent coronary artery bypass graft successfully without any peri-procedural complications.

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1. Introduction

Left main coronary artery (LMCA) disease is usually associated with multi-vessel disease and presents as an acute coronary syndrome (ACS).¹ Isolated stenosis of LMCA is quite rare. There are few cases of LMCA chronic total occlusion (CTO) presenting as stable ischemic heart disease. Several non-atherosclerotic causes, including vasculitis are associated with ostial stenosis of coronary arteries.² We present a case of a young female with few atherosclerotic risk factors who presented with stable angina and had LMCA CTO. Although percutaneous coronary intervention (PCI) is an upcoming treatment modality, coronary artery bypass grafting (CABG) remains the preferred treatment option in these patients.³

2. Case presentation

A 43-year-old premenopausal female presented to us with NYHA class II dyspnea and angina for 5 years. Her past history was

significant for abnormal uterine bleeding. She did not smoke, did not have any history of diabetes mellitus or hypertension and was not on oral contraceptive pills. She did not have a family history of premature CAD. She had no history of myocardial infarction (MI) or rest angina in the past. She had no history of fever, joint pains, surgery or radiation therapy in the past.

On presentation she had a pulse rate of 84/min and a blood pressure of 110/70 mmHg. All peripheral pulses were palpable and equal. Her respiratory rate was 18/min. Jugular venous pressure (JVP) was not raised and respiratory and cardiovascular system examination were within normal limits.

She was found to have high triglycerides (196 mg/dl), low high-density lipoprotein (HDL) cholesterol (45 mg/dl) and high very low-density lipoprotein (VLDL) cholesterol levels suggestive of dyslipidemia. Her renal and liver function tests were within normal limits.

Her ECG showed ST segment elevation in lead aVR, ST segment depression in V2–V6, II, III, aVF and T wave inversion in V1 (Fig. 1). Two-dimensional (2D) echocardiography revealed a normal left ventricular (LV) systolic function with an ejection fraction of 55–60% and grade I diastolic dysfunction. However, there was mild hypokinesia of the mid anterolateral and inferolateral segments corresponding to the Left circumflex (LCx) artery. There were no associated valvular lesions. Hence, we decided to proceed with coronary angiography and coronary revascularization, if indicated.

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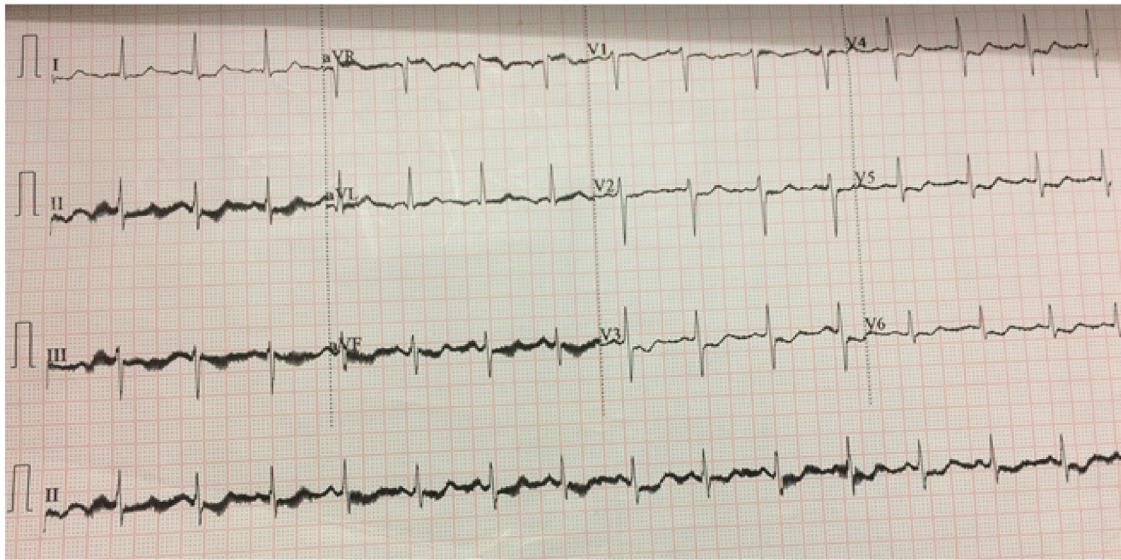


Fig. 1. 12 lead ECG.



Fig. 2. Non-selective injection in the left sinus does not locate the LMCA.

Coronary angiography was done, after informed patient consent, via the trans-femoral route. 2500 units unfractionated heparin was given at the beginning of the procedure. The LMCA could not be engaged with 6 Fr. Judkins left (JL) catheter (Medtronic Co, USA). Non-selective injection in the left sinus did not reveal the LMCA (Fig. 2). The right coronary artery (RCA) was engaged with the Judkins right (JR) 4 catheter (Medtronic Co, USA). RCA was normal in its course. Posterior descending and posterolateral ventricular branches were also normal. RCA angiogram showed filling of the LMCA up to the ostium, left anterior descending (LAD) and the LCx branches by Rentrop grade 3 collaterals (Fig. 3a & b). The distal left coronary arteries (LCA) appeared normal on RCA angiogram. Aortic root angiogram with a 6 Fr. pigtail catheter (Medtronic Co, USA) failed to demonstrate the stump of occluded LMCA (Fig. 4), which was further confirmed on the CT angiogram.

The coronary angiography was suggestive of CTO of LMCA and a normal RCA. To evaluate the cause, other additional investigations were done. ESR and CRP were elevated, likely due to underlying CAD. Homocysteine and Lp(a) levels were normal. Apo A1 levels

Table 1
Additional Laboratory Investigations.

Investigation	Value
ESR(mm/h)	27 (0–20)
CRP (mg/dl)	40.70 (0–6)
ANA (Units)	12.01 (<20.00)
RF(IU/ml)	<10 (<14)
Homocysteine (umol/L)	9.07 (4.44–13.56)
Lipoprotein A (mg/dl)	25.7 (<30)
Apo A1 (mg/dl)	85 (105–205)
Apo B (mg/dl)	94 (55–130)
ApoB/Apo A1	1.11 (0.35–0.98)
IgG (U/l)	1.91 (<10)
IgM (U/L)	0.60 (<10)

Values in brackets represent the normal laboratory reference values.

ESR- Erythrocyte Sedimentation rate; CRP-C-Reactive Protein; ANA- Anti Nuclear Antibody; RF- Rheumatoid Factor; Apo A1- Apolipoprotein A1; Apo B- Apolipoprotein B; IgG- Immunoglobulin G; IgM- Immunoglobulin M.

were low. Autoimmune markers were negative (Table 1). CT coronary angiography and CT aortogram of thoracic and abdominal aorta was done in view of ostial LMCA involvement on 128 dual source CT scanner. The RCA was normal and LMCA was not visualised. LAD and LCx were normal but reduced in caliber (Fig. 5a–c). The thoracic and abdominal aorta were normal with no evidence of vasculitis (Fig. 5d, e).

She underwent on pump CABG with left internal mammary artery graft (LIMA) to LAD and saphenous vein grafts (SVG) to obtuse marginal (OM) and diagonal successfully without any procedural complications. She is asymptomatic after surgery and under regular follow up.

3. Discussion

LMCA disease is found in 5–6% of all patients undergoing coronary angiography and is usually associated with multi-vessel CAD.⁴ LMCA occlusion usually presents as ACS. Since LMCA supplies 75% of the LV in right dominant circulation and 100% in left dominant circulation, acute LMCA occlusion usually presents as massive infarction and cardiogenic shock and is associated with a high mortality rate.⁵

CTO of LMCA is a very rare condition that exists only with a dominant RCA and excellent collateral supply.⁶ One of the reviews

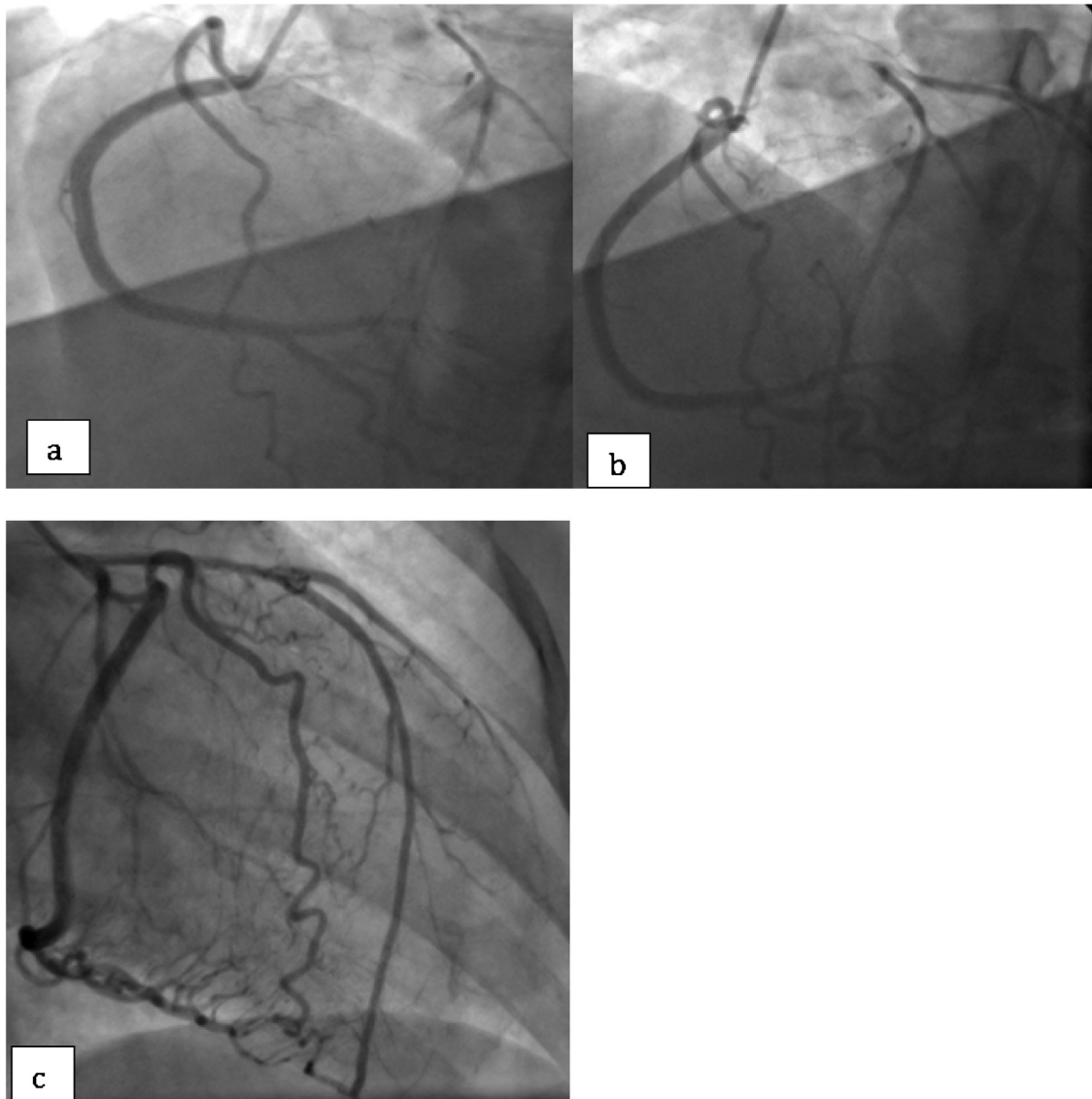


Fig. 3. RCA angiogram in the LAO Cranial view (a and b) and RAO view (3c) demonstrates a normal RCA and retrograde filling of LMCA, LAD and LCX by grade 3 collaterals.

estimated the prevalence of LMCA occlusion to be 0.04%. In these patients, visualization of RCA is easy; however, visualization of distal LCA is difficult due to the dependence on collateral circulation. Patients with normal or near normal LV function generally tend to have normal distal left vessels and normal RCA. Approximately half of the patients with LMCA CTO have disease in RCA. Several studies have highlighted the role of collaterals in preservation of systolic function. However collaterals are not enough to prevent the development of angina.⁷

The most frequent site of LMCA stenosis is the distal bifurcation followed by ostium and the mid-shaft. Several studies have shown that ostial stenosis of LMCA and RCA was more common in females.⁸ Sasaguri et al. reported that patients with ostial lesions were usually younger and presented with fewer risk factors for atherosclerosis.⁸

The etiology in most of the cases is atherosclerotic. Non-atherosclerotic causes include radiation therapy, rheumatoid arthritis, syphilis, Takayasu's arteritis, aortic valve disease, aortic valve replacement, Kawasaki's disease, left coronary ostial isolation due to aortic valve anomalies, injury following percutaneous coronary interventions and severe pulmonary artery hypertension

(PAH) leading to enlargement of pulmonary arteries and dynamic LM compression.⁹ Most of these patients present with typical symptoms of angina, have a history of prior MI and may present with symptoms of heart failure. However, some patients may be asymptomatic.

CABG is considered the treatment of choice for LMCA CTO (3). However, several reports of successful PCI have been published, particularly in patients with protected LCA by previous CABG and patients with good collaterals.¹⁰ Guidewire selection and crossing of the occlusion are the most crucial steps in CTO PCI. Recent advances in guidewire technology and crossing techniques yield a success rate of 80–90% in CTO PCI.¹¹ PCI can be done either by antegrade or retrograde approach.

Apart from dyslipidemia, our patient had no risk factors for CAD. She presented with typical symptoms as described previously. She had normal LV function due to normal RCA, excellent collateral vessels and normal distal left system. She was extensively investigated for the cause of ostial LMCA involvement. Although her inflammatory markers were raised, the autoimmune markers were negative. CT angiogram of thoracic and abdominal aorta was normal. Coronary artery involvement is rare in Takayasu arteritis and is usually associated with involvement of arch vessels.

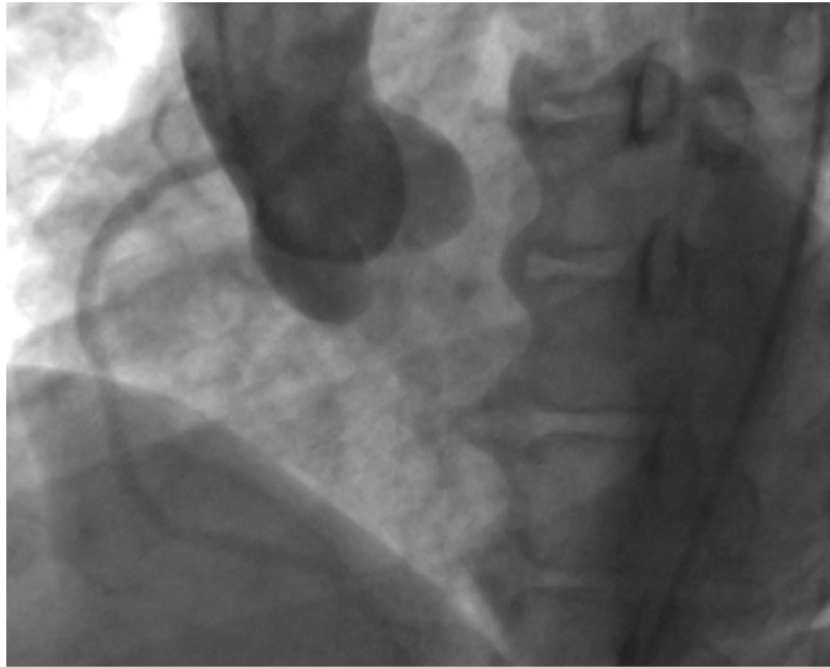


Fig. 4. Aortic root angiogram in LAO.

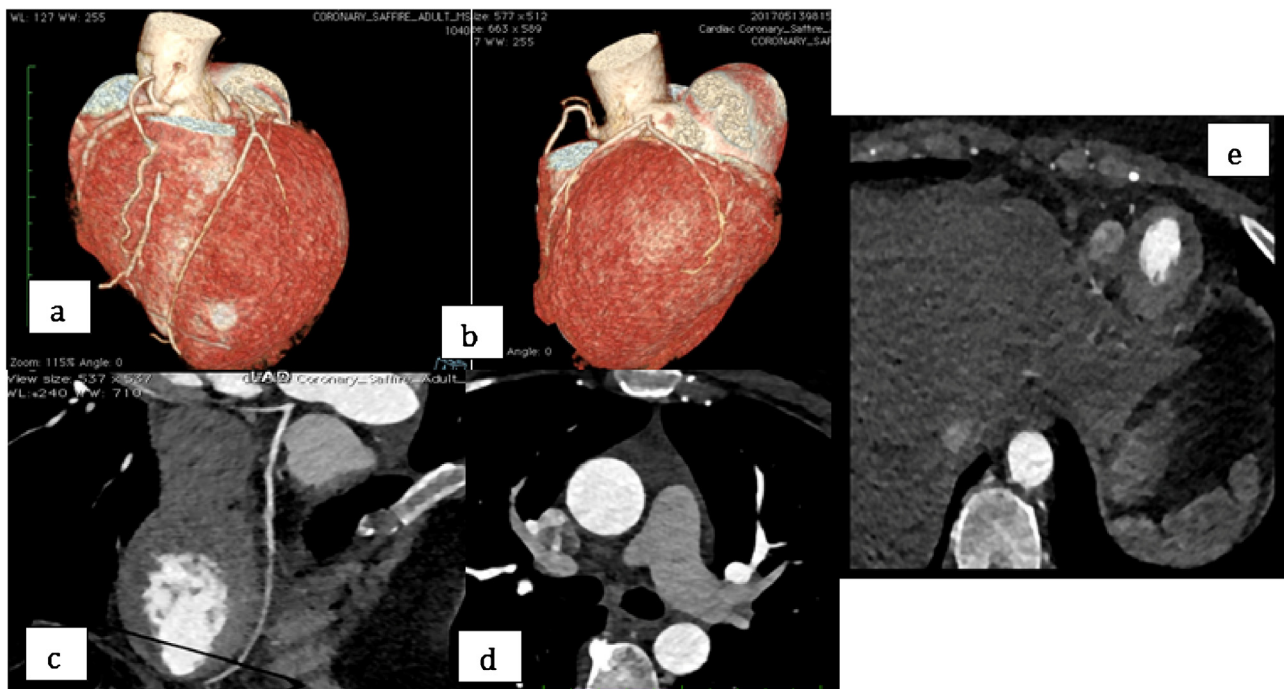


Fig. 5. CT Coronary Angiogram (a–c) demonstrates normal RCA, reduced filling of LAD and fails to demonstrate the LMCA. CT Angiogram of thoracic and abdominal aorta (d & e) was normal with no evidence of vasculitis.

Isolated coronary artery involvement is a rarer finding. Hence, we presumed the etiology of the disease to be atherosclerotic. The patient underwent CABG successfully without any complications. She is currently asymptomatic and doing well on follow-up.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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