CASE REPORT

Surgery for Isolated Non-Inflammatory Chronic Total Occlusion of the Left Main Coronary Artery: A Case Report and Literature Review

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SUMMARY
Chronic total occlusion (CTO) of the left main coronary artery (LMCA) is a rare manifestation of coronary artery disease (CAD) and defined by a total absence of antegrade blood flow to the left anterior descending (LAD) and circumflex (Cx) system. Patients are at high risk of myocardial ischaemia as a sizeable area of myocardium is at risk and thus require urgent intervention. Surgery is the treatment of choice especially with CTO lesions as percutaneous coronary intervention has limited success with a high restenosis rate. We report a rare case of a young Chinese male who presented acutely with a myocardial infarction and discuss the aetiology and peri-operative technical considerations for successful surgery with this condition.

KEY WORDS:
Chronic total occlusion (CTO), Coronary artery disease (CAD)

INTRODUCTION
Chronic total occlusion (CTO) of the left main coronary artery (LMCA) is very rare in patients with coronary artery disease (CAD) with a reported prevalence of 0.025% - 0.4% and is characterized by complete absence of antegrade blood flow to the left anterior descending (LAD) and left circumflex (Cx) arteries. We report a rare case of a young Chinese male who presented acutely with a myocardial infarction and discuss the aetiology and peri-operative technical considerations for successful surgery with this condition.

CASE SUMMARY
A 36-year-old Chinese male presented with an infero-lateral non-ST segment elevation myocardial infarction (NSTEMI) (CK: 2206) two months previously. His only cardiovascular risk factors were hyperlipidaemia and a family history of ischaemic heart disease. The patient was recovering from a tibial fracture (being treated conservatively) sustained from an unrelated earlier trauma which delayed his initial referral to our service. Coronary angiography revealed a CTO of the LMCA (Figure 1) with retrograde perfusion via a normal dominant right coronary artery (RCA) (Figure 2). Pre-operative carotid doppler scan was normal and his inflammatory markers (CRP 3, ESR 16) were not elevated. The patient had a slightly adverse lipid profile with a cholesterol and triglyceride of 5.2 mmol/L and 5.8 mmol/L respectively. The patient proceeded to urgent in-patient surgical revascularization (CABG) with a pedicled left internal mammary artery (LIMA) graft bypassing the LAD vessel and reversed autologous saphenous vein grafts bypassing the first diagonal (D1) and distal Cx arteries. The patient’s chest was re-explored on the evening of surgery for excessive mediastinal bleeding (sternal wire site) but otherwise he had an uneventful recovery and a week later was discharged back to the referring hospital for convalescence and definitive management of his tibial fracture. Pre-discharge post-operative echocardiogram showed preserved left ventricular function (ejection fraction 49%) with some antero-septal hypokinesia. Histology of aortic wall biopsy revealed degenerative changes with no evidence of vasculitis. The patient remains well and symptom-free on early follow-up at three months.

DISCUSSION
Isolated total occlusion of the LMCA is rare in patients with CAD and defined by a total absence of antegrade blood flow to the LAD and Cx coronary system. Total occlusion can be acute which usually presents with an MI, pulmonary edema, cardiogenic shock, sudden death or an abrupt change in angina severity. CTO manifests more insidiously usually over a period of more than three months and patients are often asymptomatic. LMCA stenosis usually occurs in association with concurrent distal CAD which may prove life saving as the patient will often be symptomatic thereby prompting earlier diagnosis and intervention before total occlusion of the LMCA occurs.

Isolated LMCA stenosis however is rare with an observed prevalence of 6-9% in patients undergoing coronary angiography. This case is unusual for several reasons. Firstly, our patient reported no previous angina and total occlusion of the LMCA was diagnosed at his index presentation NSTEMI. This would suggest an acute process although the presence adequate collateralization via the RCA system suggests a more chronic process. Indeed the lack of time for adequate collateralization would have put a large area of his myocardium in jeopardy.

Secondly, isolated LMCA disease is rare and usually has a vasculitic aetiology. This is an unlikely cause with our patient given the negative aortic wall biopsy, low inflammatory markers and absence of extra cardiac arterial disease. Wikelma et al has reported a genetic predisposition

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(genotype apolipoprotein E) in as many as 70% of the population with severe LMCA ostial stenosis. This may predispose to a pathologically increased response of proliferative repair mechanisms after arterial injury.

It is important that other differential aetiologies including Takayasu’s arteritis are considered and excluded.

Surgery is the treatment of choice especially with CTO lesions because crossing the occlusion with a wire has a limited success rate of 40%-80% and a high restenosis rate. Percutaneous coronary intervention (PCI) is generally considered for acute lesions or patients deemed surgically unfit for CABG and remains a potential therapeutic option if the patient has good collaterals and incomplete occlusion. Patients undergoing PCI however are more likely to require future repeat revascularization. Bare metal stenting for LMCA occlusion has a 1-year mortality of 3-28% and restenosis rate of 20%, the latter decreasing to 10% with use of a drug-eluting stent. Patients with a CTO of the LMCA are not ideal candidates for surgical angioplasty or reconstruction of the LMCA ostium as the lesions is usually progressive and often heavily calcified.

Our patient was young with good distal target vessels and hence offered CABG for prognostic benefit. We contemplated prophylactic use of a peri-operative intra-aortic balloon pump (IABP) to augment native coronary perfusion given the total occlusion of the left coronary system and a normal fully patent RCA however as the patient remained haemodynamically stable pre-cardiopulmonary bypass, an IABP was not inserted in this patient. Adequate collateral flow is however vital in preserving left ventricular function though it may be insufficient to prevent angina. Our intra-operative myocardial protection strategy involved antegrade cold blood cardioplegia with resulting prompt electromechanical diastolic arrest however additional retrograde delivery of cardioplegia would not be an unreasonable option given the patient’s anatomical disease. We however agree with the strategy of initial revascularization of a totally occluded vessel prior to bypassing a vessel supplying collaterals to it as has been previously reported.

Patients with a total occlusion of the LMCA especially acute lesions, due to a lack of sufficient collateralization, are at high risk of significant myocardial ischaemia as a large are of myocardium is at risk. Such patients should be operated on as soon as is feasible.

Isolated LMCA disease is a rare manifestation of CAD and may have a vasculitic aetiology which should be excluded. Prophylactic use of an IABP, a thoughtful coronary grafting sequence and route of cardioplegia delivery, are important technical surgical considerations for a successful outcome.

REFERENCES