Total Left Main Coronary Artery Occlusion*
The Acute, the Chronic, and the Iatrogenic
On Topaz, M.D.

(Chest 1992; 101:843-46)

| CASS = coronary artery surgery study; LMCA = left main coronary artery; RCA = right coronary artery |

Complete obstruction of the left main coronary artery (LMCA) is an intriguing, rare angiographic and pathologic finding. Etiologic factors accountable for total LMCA occlusion include a variety of pathologic processes as well as iatrogenic mechanisms. Despite the crucial anatomic location of the occlusion and its severe interference with the left ventricular function, clinical presentation may vary considerably. The purpose of this review is fourfold: to delineate the etiology of total left main occlusion; to describe clinical manifestations; to report related angiographic findings; and to discuss therapeutic options for treatment of patients presenting with this unique condition.

CHRONIC LMCA OBSTRUCTION

Etiology

Severe coronary atherosclerotic disease is the most common cause of total LMCA occlusion. Patients having complete LMCA obstruction nearly always present with associated disease in two or three remaining major epicardial coronary vessels.1 Other diseases that cause gradual and progressive left main occlusion include syphilitic arteritis, rheumatic arteritis, polyarteritis nodosa, and arterial medial calcification. Tumors can cause external compression on the left main stem leading to tight LMCA narrowing.2 Mediastinal irradiation can promote intimal fibrous proliferation resulting in progressive severe left main narrowing.3 Several congenital heart diseases can be associated with critical occlusion of the left main ostium. Among them are supravalvular aortic stenosis,4 truncus arteriosus,5 and congenital fusion of the left coronary cusp to the aortic wall.6 Congenital ostial membrane of the LMCA can cause tight stenosis,7 and in congenital atresia of the LMCA, a fibromuscular cord replaces the normal vessel.8

Incidence

Patients who survive to present with either acute or chronic complete LMCA occlusion consist of a selective group, as a high mortality is already associated with subtotal narrowing of the vessel.9,10 Thus, it is not surprising that angiographic as well as necropsy studies11 continue to show a low prevalence of this condition. The reported angiographic incidence varies from 0.03 percent in 20,332 patients in the Medical College of Virginia experience12 to 0.06 percent in 20,197 patients in the coronary artery surgery study (CASS) study.13

Symptoms

Most patients with this condition receive medical attention and are referred for cardiac catheterization due to decreased exercise tolerance and severe angina. The duration of angina can vary from a very prolonged period of many years14 to a relatively short term of a few weeks.15 Notably, patients may only have stable class 1 angina16 or may lack chest pain altogether.

FIGURE 1. Total left main coronary artery occlusion at the origin. Delicate homocollaterals create a vascular cuff surrounding the occluded vessel (arrows).

*From the Cardiac Catheterization Laboratory, Section of Cardiology, St. Paul-Ramsey Medical Center, University of Minnesota School of Medicine, St. Paul. Reprint requests: Dr. Topaz, Section of Cardiology, 640 Jackson Street, St. Paul 55102

©1992 American College of Chest Physicians

Downloaded From: http://journal.publications.chestnet.org/pdffaccess.ashx?url=/data/journals/chest/21640/ on 06/25/2017
Others can present with symptoms and signs of congestive heart failure\textsuperscript{17,18} that are related to the associated impaired hemodynamics and abnormal left ventricular function.

**Angiographic Findings**

Two distinct angiographic features can be identified in these patients. The first is a lack of opacification of any part of the LMCA (Fig 1) as a result of complete obstruction at its origin or opacification of only a segment of the vessel up to a total occlusion (Fig 2). The other facet is the demonstration of intercoronary (Fig 3 and 4) and rare intracoronary collateral channels (Fig 1). Although intercoronary collateral vessels exist virtually in every case of long-term total occlusion of the left main, they can well be absent in cases of acute total closure.

Attesting to the richness of the collateral system in long-term total LMCA obstruction is the finding in an angiographic study\textsuperscript{12} that showed in each of six patients with total chronic left main occlusion there were two to six different intercoronary collateral pathways; 13 specific collateral channels were identified. The conus branch of the right coronary artery (RCA) and the anterior and posterior septal branches were the most common vessels participating in the creation of right coronary to left coronary communication. Nevertheless, several authors\textsuperscript{12,19,20} have shown that even in patients with extensive, well-developed collateral circulation providing blood flow from the right coronary system to the occluded left coronary system, these are insufficient collaterals for increased myocardial demand. Clinically, in most patients, the collaterals’ insufficiency is manifested by significant angina, decreased exercise tolerance, and congestive heart failure.

The RCA herein plays a crucial role as the donor artery to collaterals. It is clear that development of obstructions of the RCA can result in further significant deterioration of the global function of both ventricles. It should be noted that in patients with associated RCA disease, several collateral channels initially (prior to complete left main occlusion) provide blood supply from the left coronary system to the RCA. Once a total LMCA obstruction develops, reversal of blood flow across the collaterals occurs. The reversal of blood flow is essential in maintaining certain myocardial viability. This phenomenon also occurs in patients after coronary artery bypass grafting\textsuperscript{21} and patients with angina whose diseased collaterals’ donor artery sustains complete obstruction.\textsuperscript{22}

Unique homocollaterals can be demonstrated angiographically in patients with chronic total LMCA. They are represented as a network of very delicate vessels...
extending from the aorta and forming a vascular cuff surrounding the obstruction. Topaz et al.14 and DePace et al.15 each reported the case of one patient presenting with these homocollaterals, and according to James,24 these vessels represent enlarged vasa vasorum or adventitial arteries.

**Hemodynamics**

Cardiac catheterization often reveals elevated mean wedge and left ventricular end-diastolic pressures. The left ventricular ejection fraction is significantly reduced in all these patients, ranging from a moderately depressed to severely depressed ejection fraction. Global and regional wall abnormalities are common. Consequently, cardiac output is very low and most patients, especially those with acute closure, need intra-aortic balloon counterpulsation for hemodynamic support.

**Acute LMCA Closure**

Acute noniatrogenic total LMCA occlusion is a clinical catastrophe caused by several mechanisms. It has been ascribed to spontaneous spasm of the left main trunk26 or to spasm on an atherosclerotic plaque. Dissection of an atherosclerotic plaque can result in subintimal hemorrhage and thrombosis occluding the LMCA. Another mechanism is the formation of a thrombus on a critical atherosclerotic lesion. An embolus occluding the left main coronary artery was described in two men, causing sudden death in each. In both, a fibrin-platelet embolus was found at the left main artery, while the major coronary arteries were free of atherosclerotic plaque.11 In one of these patients, the source of acute closure was a paradoxic embolus. Another report36 described a myocardial embolus acutely occluding the left main artery. The source of this embolus was a fragment of the mitral papillary muscle during mitral valve replacement.

Most patients with acute total occlusion of the LMCA suffer extensive transmural myocardial infarction.27 Acute LMCA obstructions resulting in minimal myocardial damage17,28 or in a non-Q-wave infarction29 have rarely been reported. In a vast majority of the patients, a significant decrease of global and regional left ventricular function is immediately encountered, followed by cardiogenic shock and severe ventricular arrhythmias. The management is based on urgent reestablishment of coronary flow. Emergency utilization of intracoronary thrombolysis, left main balloon angioplasty, and insertion of an intra-aortic counterpulsation balloon pump30 are among the recommended urgent means of revascularization. Most of these patients are then referred for emergency bypass grafting.31 Although a review of the literature can initially give the impression that attempts at emergent mechanical revascularization are commonly associated with survival,32-34 and at times with only minimal myocardial damage,35 one should be aware that there is a tendency to report only successful outcomes of acute interventions. Even with successful emergency balloon angioplasty, some patients do not survive the postoperative period as a result of extensive myocardial damage and subsequent multisystem failure.12 Survival with noninterventional, conservative medical management is extremely rare.27 We suggest that a natural selection process explains rare reports of patients who survived acute LMCA occlusion without the institution of any interventional therapy.

**Iatrogenic LMCA Closure**

Intracoronary maneuvers with diagnostic catheters35 or manipulations with equipment for coronary angioplasty can lead to the development of either acute36 or chronic27 total occlusion of the left main artery. Keltai et al.38 reported a case in which a snap of a guidewire caused immediate coronary thrombosis resulting in acute total LMCA occlusion. Left main obstruction is also recognized23,39 as a late complication of coronary perfusion or cardioplegia in cardiothoracic surgery. Trimble and coworkers39 suggest that in these cases, the left main intimal insult and damage begin with intraoperative cannulation and follow with a progressive, usually slow, critical obliteration of the left main lumen.

**Conclusions**

Total occlusion of the left main coronary artery represents a unique clinical condition with specific angiographic characteristics. Its development is attributed to certain acute and chronic pathologic processes or to iatrogenic factors relating to mechanical manipulations in the left main artery such as coronary arteriography and angioplasty. Symptoms correspond to the presence and qualities of collateral vessels, associated myocardial damage, and impaired hemodynamics.

The ability of collaterals to maintain myocardial demand is affected by the extent of atherosclerotic disease in the collaterals' donor artery. In acute cases, the management includes immediate restoration of left main blood flow and hemodynamic support by mechanical interventional means such as balloon angioplasty and intra-aortic balloon counterpulsation, commonly followed by surgical revascularization. In chronically symptomatic patients, the recommended treatment is coronary artery bypassgrafting.

**ACKNOWLEDGMENT**: The author is indebted to Laurie Topaz, Keith Dixon, and Claudia DuBord for their invaluable assistance in the preparation of this manuscript.

**REFERENCES**

1. Bulkeley BH, Roberts WC. Atherosclerotic narrowing of the left main coronary artery: a necropsy analysis of 152 patients with
fatal coronary heart disease and varying degrees of left main narrowing. Circulation 1976; 53:823-28
3 Brostius FC, Waller BF, Roberts WC. Radiation heart disease: analysis of 16 young (aged 15 to 33 years) necropsy patients who received over 3,500 rads to the heart. Am J Med 1981; 70:519-30