Catheterization and Cardiovascular Diagnosis 5:269–275 (1979)

Survival With Total Occlusion of the Left Main Coronary Artery. Significance of the Collateral Circulation

M. Valle, MD, K. Virtanen, MD, P. Hekali, MD and M.H. Frick, MD

Four patients with total occlusion of the left main coronary artery are described. Angina pectoris was severe (NYHA class 3–4) and had lasted 20 months to seven years. Three patients had experienced a myocardial infarction. All displayed large collaterals arising from a nearly normal right coronary artery and feeding both the left anterior descending and the left circumflex arteries. The left ventricular ejection fractions ranged from 20% to 65%, and all patients had varying degrees of left ventricular asynergy. Coronary artery bypass surgery resulted in a marked improvement in three patients; one patient who underwent an aneurysmectomy died two months after the operation.

The data show that total occlusion of the left main coronary artery is compatible with survival if adequate collateral supply develops from the right coronary artery. In this rare angiographic subset collateral circulation is clearly functionally significant.

Key words: coronary angiography, coronary atherosclerosis

INTRODUCTION

Data on the significance of coronary collaterals in preventing ischemia, influencing left ventricular function, and modifying prognosis in stable ischemic heart disease are at variance [1–5]. A recent study focused on coronary collateral circulation in left main stem (LM) disease and found no evidence that collateral vessels have a protective effect [6].

We describe four patients with total occlusion of the LM with rich collateral networks to demonstrate the significance of collateral supply in this angiographic subset.

From the Department of Diagnostic Radiology and the Cardiovascular Laboratory, First Department of Medicine, University Central Hospital, Helsinki, Finland.

Received January 30, 1979; revision accepted June 5, 1979.

Address reprint requests to M.H. Frick, MD, Cardiovascular Laboratory, First Department of Medicine, University Central Hospital, Haartmaninkatu 4, SF-00290 Helsinki 29, Finland.

0098-6569/79/0503-0269$01.70

METHODS AND PATIENTS

Selective coronary angiography was performed in multiple projections using the Judkins technique as described previously from this laboratory [7,8]. Coronary angiography was preceded by left ventricular cineangiography in the right anterior oblique projection. The left ventricular ejection fraction was calculated [9], and regional contraction abnormalities [10] were assessed. Maximal exercise testing on a bicycle ergometer was performed as described elsewhere [11]. Exercise tolerance is given as the maximum tolerated load in kpm/min.

Out of 948 coronary angiographies, four cases (0.42%) were found with total occlusion of the LM. All the patients were studied as potential candidates for coronary artery bypass surgery.

CASE REPORTS

Data from the noninvasive investigations are shown in Table I and those from the invasive studies in Table II.

Case 1

A 45-year-old foreman had progressively worsening angina pectoris for 20 months but no episodes indicative of myocardial infarction. Blood pressure (BP) was 110/70 and a paradoxical pulsation was palpated parasternally, with both atrial and ventricular gallops on auscultation. Two 10% stenoses were visualized in the right coronary artery (RCA). Faultless left anterior descending (LAD) and left circumflex (LCX) coronary arteries were excellently filled via large collaterals from the distal RCA running along the septum, and from the atrioventricular branch to the LCX. In addition to the large collaterals, a dense network of smaller collateral vessels arose from the distal RCA towards the LAD and LCX. The conus branch was not visualized.

TABLE I. Some Characteristics of the Series

Patient number	Functional class (NYHA)	Heart volume (ml/m^2 BSA)	Resting ECG	Maximum ST depression in exercise (mm)	Maximal load (kpm/min)
1	3	550	LAHB, OS V_{1-3}	2	400
2	4	480	Patol Q AVL, QS V_{1-2}, negat T I, AVL, V_{4-6}	2	100
3	3	440	LAHB, Patol Q I, AVL, V_{2-6}	2	600
4	4	370	pRBBB	3	400

Abbreviations: NYHA = New York Heart Association; BSA = body surface area, normal heart volumes in males = < 500 ml/m^2 BSA, LAHB = left anterior hemiblock, pRBBB = partial right bundle branch block, kpm = kilopond-meters.

Case 2

A 46-year-old blacksmith had a seven-year history of angina pectoris with worsening during the last year. He had experienced a myocardial infarction six years previously and was treated in a municipal hospital. The electrocardiogram showed negative T waves in inferior leads, accompanied by elevations in the appropriate enzyme values. He had had an anterolateral infarction six months before admission, followed by severe angina pectoris. BP was 140/90. The LAD was supplied both proximal and distal to a 25% stenosis by large collaterals arising from the distal RCA along the septum and from the right ventricular branch directly. All the collaterals to the LCX went distal to a 25% stenosis in the proximal part of the vessel (Fig. 1). The sinus node branch arising from the RCA sent collaterals to the LCX.

Case 3

A 44-year-old farmer had a two-year history of angina pectoris and an anterior wall infarction 11 months before admission. BP was 120/70. A ventricular gallop was associated with an apical paradoxical pulsation. There were 25% stenoses in the RCA proximally and in the middle section. Both the LAD and LCX were totally occluded in the proximal part. The middle and distal parts of the LAD received several large collaterals arising both from the distal RCA via the interventricular septum and from the right ventricular branch directly. The middle and distal LCX were supplied by collaterals from the sinus node branch and the atrioventricular branch of the RCA.

Case 4

A 53-year-old mechanic had a three-year history of worsening angina leading to a consumption of 30–35 sublingual nitroglycerin tablets daily in addition to therapy with propranolol. There were no episodes indicative of myocardial infarction. BP was 145/85 and an atrial gallop was audible. There was a 50% stenosis in the distal

TABLE II. Data From Coronary and Left Ventricular Angiography

Patient number	Additional coronary obstructions (%) LAD	LCX	RCA	LVEDP (mmHg)	LV EF (%)	Regional asynergy
1	0	0	10[a,b]	30	20	Anterior apical akinesis and dyskinesis
2	25[b]	25[a]	10[a]	22	40	Anterior apical and apical akinesis
3	100[a]	100[a]	25[a,b]	16	31	Anterior apical and apical akinesis
4	0	0	50[c]	12	65	Apical and apical inferior hypokinesis

[a] Site of obstruction = proximal segment.
[b] Site of obstruction = middle segment.
[c] Site of obstruction = distal segment.
Abbreviations: LAD= left anterior descending; LCX = left circumflex; RCA = right coronary artery; LVEDP= left ventricular end-diastolic pressure; LV EF = left ventricular ejection fraction.

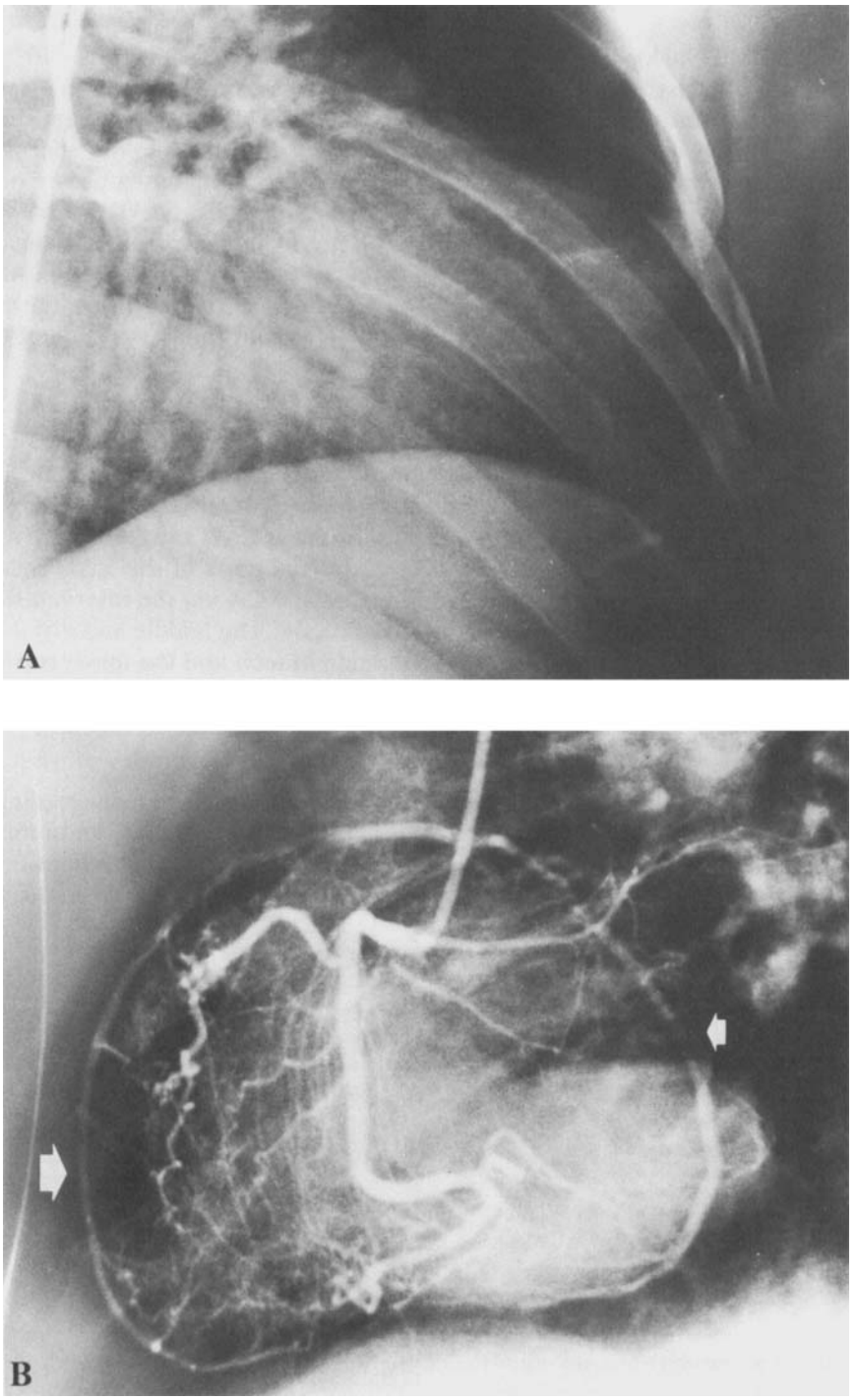

Fig. 1. A. Selective injection into totally occluded left main coronary artery. B. Selective injection into the right coronary artery sending large collaterals to left circumflex (smaller arrow) and left anterior descending (large arrow) coronary arteries. Detailed description of the collaterals in text.

RCA. A normal LAD was excellently visualized via collaterals from the right ventricular branch and conus branch to the middle section and via collaterals along the septum to the entire LAD. The septal collaterals were beyond the 50% stenosis in the RCA. The LCX was supplied by collaterals from the sinus node branch and the atrioventricular branch of the RCA.

All the patients underwent coronary artery bypass surgery. Venous grafts were used to bypass the LCX, and the left internal mammary artery was used to bypass the LAD. In Case 1 a paradoxically bulging area of the anterior myocardial wall was excised as well. This patient died two months after the operation due to a wound infection leading to a pseudoaneurysm rising from the left ventricular suture line. The other patients had their grafts patent in control angiographies, have no angina pectoris, and have resumed work.

DISCUSSION

In series specifically describing different characteristics of LM disease [12–17] total occlusion of this artery has been found only sporadically [18,19]. In a series of 2,200 coronary angiographies, six cases with total occlusion of LM were encountered [20]. At the Cleveland Clinic, with extensive experience covering 63,500 coronary angiographies, 14 patients with completely occluded LM were found [21]. It can be speculated that this low prevalence is attributable to a high preangiographic mortality associated with massive myocardial infarctions, but not a single case with total occlusion was found in a necropsy series specifically analyzing LM disease [22]. The low prevalence of total occlusion of the LM in both angiographic and necropsy series presumably reflects the high mortality associated with subtotal occlusions of the LM [23,24].

The history of angina pectoris in our patients ranged from 20 months to seven years, with a clear worsening prior to the investigations in three patients. Within this time range there was probably a gradual narrowing of the LM, coupled with collateral development. None of the patients had had a massive infarction and one patient (Case 4) had no evidence of a clear-cut prior infarction. The inferior infarction in Case 2 was subendocardial, leaving no subsequent inferior contraction abnormalities. The reasonably well preserved ejection fractions and relatively minor contraction abnormalities in three patients (Table II) suggest a protective role of the collateral circulation originating from the RCA, which in every case showed only minor stenoses. This line of reasoning is supported by a finding of relatively minor contraction abnormalities in three patients with total LM occlusion and extensive collaterals from the RCA in contrast to more severe left ventricular asynergy in three similar patients with sparse collaterals [20]. Indirect supporting evidence for the protective role of collateral circulation comes from the observation that patients with LM disease and collaterals have a lower surgical mortality than similar patients without collaterals [18]. Data on the state of RCA in patients with total occlusion of LM are sparse and not amenable to detailed analysis. However, larger series of LM disease have shown that the RCA is significantly involved in the majority of cases [12–19,22,24]. This was also true in the study of Iskandrian and associates [6], which did not show a protective role of collateral circulation in LM disease. In their series of 50 patients with LM disease the RCA was involved in 34 cases, with total occlusion in 20 patients. In this setting the

impact of collateral circulation may be difficult to demonstrate without detailed analysis on the topography of the collaterals in relation to RCA obstructions.

All our patients had severe angina pectoris (Table I), suggesting that the amount of blood supplied via the collateral channels was insufficient to meet the metabolic demands of the jeopardized myocardial segments under stress. This is in accord with our previous data on the limited transport capacity of the collaterals under provoked ischemia [25,26].

The data demonstrate that total occlusion of the LM is not necessarily associated with massive myocardial infarctions with a fatal outcome. Well-developed collateral circulation from the uncompromized right coronary artery seems to be sufficient to prevent this catastrophe and preserve reasonable left ventricular function, but it is insufficient to prevent ischemia when myocardial oxygen demand is increased.

REFERENCES

1. Helfant RH, Vokonas PS, Gorlin R: Functional importance of the human coronary collateral circulation. N Engl J Med 284:1277–1281, 1971.
2. Miller RR, Zelis R, Mason DT, Amsterdam EA: Relation of coronary collateral vessels to ventricular function in patients with equal extent of coronary artery disease. Circulation 44(Suppl 2):202, 1972 (abstr).
3. Levin DC, Sos TA, Lee JG, Baltaxe HA; Coronary collateral circulation and distal coronary runoff: The key factors in preserving myocardial contractility in patients with coronary artery disease. Am J Roentgenol 119:474–483, 1973.
4. Lavine P, Filip Z, Najmi M, Kimbiris D, Segal BL, Linhart JW: Clinical and hemodynamic evaluation of coronary collateral vessels in coronary artery disease. Am Heart J 87:343–349, 1974.
5. Carroll RJ, Verani MS, Falsetti HL: The effect of collateral circulation on segmental left ventricular contraction. Circulation 50:709–713, 1974.
6. Iskandrian AS, Tendler S, Mintz GS, Bemis CE, Kimbiris D, Segal BL: Significance of collateral circulation in patients with left main coronary artery disease. Cathet Cardiovasc Diagn 4:135–141, 1978.
7. Valle M: Postoperative coronary angiography. Acta Radiol Suppl 333, 1973.
8. Frick MH, Valle M, Harjola P-T, Korhola O: Changes in native coronary arteries after coronary bypass surgery. Role of graft patency, serum lipids and hypertension. Am J Cardiol 36:744–750, 1975.
9. Lewis RP, Sandler H: Relationship between changes in left ventricular dimensions and the ejection fraction in man. Circulation 44:548–557, 1971.
10. Herman MV, Gorlin R: Implications of left ventricular asynergy. AM J Cardiol 23:538–547, 1969.
11. Frick MH, Harjola P-T, Valle M: Effect of aortocoronary grafts and native vessel patency on the occurrence of angina pectoris after coronary bypass surgery. Br Heart J 37:414–419, 1975.
12. Rösch J, DeMots H, Antonovic R, Rahimtoola SH, Judkins MP, Dotter CT: Coronary arteriography in left main coronary artery disease. Am J Roentgenol 121:583–590, 1974.
13. Lavine P, Kimbiris D, Segal BL, Linhart JW: Left main coronary artery disease. Clinical, arteriographic and hemodynamic appraisal. Am J Cardiol 30:791–796, 1972.
14. Cohen MV, Gorlin R: Main left coronary artery disease. Clinical experience from 1964–1974. Circulation 52:275–285, 1975.
15. Banim SE, Donaldson RM, Russell DC, Sturridge MF, Wright JEC, Honey M, Balcon R: Clinical experience with left main coronary artery stenosis. Br Heart J 38:71–76, 1976.
16. Bopp P, Haissly JC, Fournet PC: Les sténoses du tronc coronaire gauche. Arch Mal Coeur 69:467–473, 1976.
17. Zeft HJ, Manley JC, Huston JH, Tector AJ, Auer JE, Johnson WD: Left main coronary artery stenosis. Results of coronary bypass surgery. Circulation 49:68–76, 1974.
18. Talano JV, Scanlon PJ, Meadows WR, Kahn M, Pifarré R, Gunnar RM: Influence of surgery on survival in 145 patients with left main coronary artery disease. Circulation 51(Suppl 1):105–111, 1975.

19. Khaja F, Sharma SD, Earley RM, Jr., Heinle RA, Goldstein S: Left main coronary artery lesions. Risks of catheterization, exercise testing and surgery. Circulation 49(Suppl 2):136–140, 1974.
20. Golberg S, Grossman W, Markis JE, Cohen MV, Baltaxe HA, Levin DC: Total occlusion of the left main coronary artery. A clinical, hemodynamic and angiographic profile. Am J Med 64:3–8, 1978.
21. Sones FM Jr: Personal communication, 1978.
22. Bulkley 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 53:823–828, 1976.
23. Bruschke AVG, Proudfit WL, Sones FM Jr: Progress study of 590 consecutive nonsurgical cases of coronary disease followed 5–9 years. I. Arteriographic correlations. Circulation 47:1147–1153, 1973.
24. Lim JS, Proudfit WL, Sones FM Jr: Left main coronary arterial obstruction: Long-term follow-up of 141 nonsurgical cases. Am J Cardiol 36:131–135, 1975.
25. Frick MH, Valle M, Korhola O, Riihimäki E, Wiljasalo M: Analysis of coronary collaterals in ischaemic heart disease by angiography during pacing induced ischaemia. Br Heart J 38:186–196, 1976.
26. Frick MH, Korhola O, Valle M, Riihimäki E, Wiljasalo M: Radiologically detected collaterals and regional myocardial flow responses to ischaemia in ischaemic heart disease. Ann Clin Res 8:241–247, 1976.