Extensive Fibrosis Surrounding Myocardial Bridging: an Intravascular Ultrasound Study

Harel Gilutz MD, Carlos Cafri MD and Reuben Ilia MD

Department of Cardiology, Soroka University Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer Sheva, Israel

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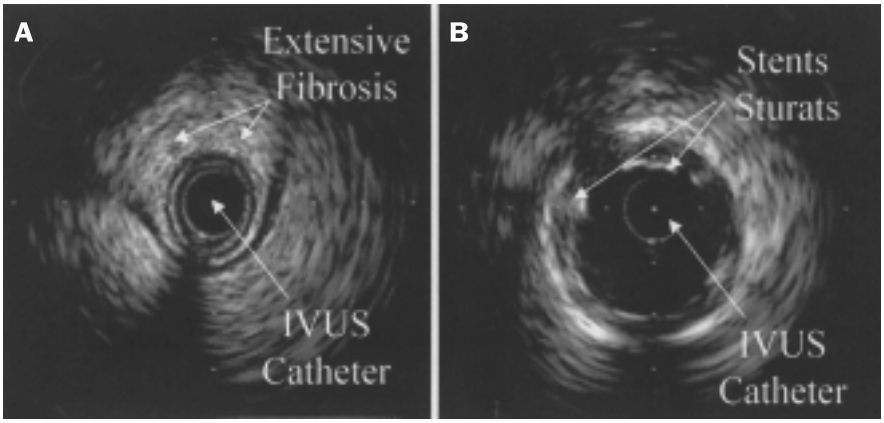
A 60 year old hypertensive, non-smoking, caucasian male had a history of long-standing precordial pain at effort with no evidence of significant ischemia on provocative radionuclear test. The patient was hospitalized due to an increase in the intensity of his pain and dynamic T-wave inversion. Angiography detected a mid-left anterior descending coronary myocardial bridging with typical signs of bridging. However, no signs of atherosclerosis were detected in the coronary tree. The patient responded well to medical treatment (aspirin, atenolol, mononitrates and enalapril for high blood pressure). Global systolic function assessed by echocardiography was normal.

Ten days after angiography, the patient was readmitted with recurrent precordial pain. Another angiography was performed, this time with intravascular ultrasound study. After diagnostic angiography, the left coronary artery was cannulated with a 7-French Extrabackup guiding catheter (Cordis, USA) and the LAD negotiated with a coronary wire (0.014", Choice Intermediate, Boston Scientific, Sunnyvale, CA, USA). A 3.2-French, 30 MHz IVUS catheter (Boston Scientific) was advanced into the LAD distal to the site of CMB.

IVUS images showed moderate systolic eccentric compression of the coronary lumen, from a diastolic area of 3.9 mm^2 to a systolic lumen area of 2.1 mm^2. However, at the most stenotic area at the bridge site, a minimal lumen area of 1.7 mm^2 was found, with no systolic to diastolic lumen difference. At this site, a highly dense ecogenic tissue equivalent to extensive dense fibrosis surrounding the vessel, more extensive at the epicardial side of the vessel, was seen [Figure A]. The proximal reference lumen area was 6.5 mm^2 and the distal reference lumen area 5.6 mm^2. No signs of atherosclerosis were detected along the LAD.

Subsequently, coronary angioplasty was performed with direct stenting (Duett stent, 3 mm width, 23 mm length, Guidant, USA). Deployment was performed at 10 atmospheres, yielding good angiographic results. However, a second IVUS run revealed an underexpanded stent with a lumen area of 4.9 mm^2. More aggressive inflation (16 atmospheres) using a 3.5 mm balloon was performed, increasing lumen cross-section area to 5.7 mm^2 [Figure B]. The procedure was stopped and the patient was discharged the next morning without complications.

Both the clinical significance of and the mode of therapy for CMB are controversial. CMB is diagnosed in approximately 0.5–16% of patients examined by coronary angiography [1] although the prevalence is 5–86% at autopsy [2]. Normally, the main coronary arteries and the proximal segments of their major branches lie free on the epicardial surface of the heart. In some cases, the coronary vessels may penetrate into the muscle surrounding the myocardium with overlying muscle, which is called



[A] Pre-intervention IVUS images of the myocardial bridge at the maximal stenotic area. A lumen area of 1.7 mm^2 with no signs of plaque formation but with an extensive peri-vessel fibrosis is imaged. **[B]** Final post-intervention IVUS images of the stent in the myocardial bridge. Stented lumen cross-sectional area is 5.7 mm^2.

LAD = left anterior descending artery
IVUS = intravascular ultrasound

CMB = coronary myocardial bridging

a "bridge." At coronary angiography, a systolic compression, usually of LAD coronary artery, is observed in only 1–3% of patients. This phenomenon is considered benign, although it is debated whether CMB may contribute to myocardial ischemia [3]. Some reports suggest an association with myocardial ischemia, infarction, vasospasm, cardiac arrhythmia and sudden death [4], which implies that documenting the presence of CMB is of clinical importance. The condition of symptomatic patients usually improves with medical treatment. Stenting of such lesions was recently described [5].

Intravascular ultrasound, Doppler signs, and characteristics of myocardial bridging have been studied thoroughly: myocardial bridging is characterized by a specific, echolucent, half-moon phenomenon over the bridge segment, which exists throughout the cardiac cycle, and systolic compression of the bridge segment of the coronary artery.

This report adds the presence of focal stenosis secondary to epicardial fibrosis of the artery wall to the known IVUS imaging of CMB, and is the first *in vivo* documentation of the fibrotic patches of the left anterior descending CMB previously described at autopsy [4]. More than anecdotally, this finding could contribute to our understanding regarding symptomatic patients with CMB when they are refractory to medical treatment, and supports the need for IVUS imaging when these cases are catheterized.

The cause of this phenomenon is unclear. We hypothesize that the long-standing "wear and tear" may support the arterial wall and thus deter total compression of the coronary lumen. Such a finding can produce rigid coronary stenosis and refractory symptoms and could be treated by mechanical revascularization with stent.

References

1. Ishimori T, Raizner AF, Chabine RA, Awdeh M, Luchi R. Myocardial bridges in man: clinical correlations and angiographic accentuation with nitroglycerin. *Cathet Cardiovasc Diagn* 1977;3:59–65.
2. Polachek P. Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions *Am Heart J* 1961;61:44–52.
3. de Winter RJ, Kok WE, Piek JJ. Coronary atherosclerosis within a myocardial bridge, not a benign condition. *Heart* 1998; 80:91–3.
4. Desseigne P, Tabib A, Loire R. Myocardial bridging on the left anterior descending coronary artery and sudden death. Apropos of 19 cases with autopsy. *Arch Mal Coeur Vaiss* 1991;84(4):511–16.
5. Ge J, Jeremias A, Rupp A, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. *Eur Heart J* 1999;20(23):1707–16.

Correspondence: Dr. H. Gilutz, Dept. of Cardiology, Soroka University Medical Center, P.O. Box 151, Beer Sheva 84101, Israel.
Phone: (972-8) 640-3469
Fax: (972-8) 623-9269
email: gilutz@bgumail.bgu.ac.il