Nonsymptomatic myocardial bridge causing systolic total narrowing of circumflex artery

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Myocardial bridge is defined as the narrowing of any coronary artery segment in systole but a normal diameter in diastole. It is most frequently seen on left anterior descending (LAD) artery. Left circumflex artery (LCx) is very rare. A 62 year-old male patient presented with severe, squeezing chest pain. The electrocardiogram showed T wave inversion in V1–V4 and ST depression in DII, DIII, aVF. Coronary angiography showed complicated lesion on after S2 branches of LAD and myocardial bridge causing 100% systolic narrowing of fourth obtus marginal branch of LCx. Bare metal stent was placed to LAD lesions with no residual occlusion. The patient was discharged with beta-blocker therapy. He had no recurrent chest pain during six months of follow-up.

Keywords: Myocardial bridging, Coronary circulation

Introduction

Myocardial bridge is defined as the narrowing of any coronary artery segment in systole but a normal diameter in diastole. Myocardial bridging generally occurs in the left anterior descending artery (LAD); left circumflex artery (LCx) involvement is very rare [1]. Although it is a benign condition, myocardial bridging can manifest symptoms such as acute coronary syndrome, arrhythmias, and sudden cardiac death [2].

We report an incidentally determined nonsymptomatic myocardial bridge that caused 100% systolic narrowing of the LCx in a patient with acute coronary syndrome.

Case report

A 62-year-old male patient presented with severe, squeezing chest pain. Aortic 2/6 systolic murmur and 1/4 diastolic murmur were observed; the result of his physical examination was otherwise normal. Blood pressure and cardiac rate were in normal ranges. The electrocardiogram (ECG) showed T wave inversion and R wave progression

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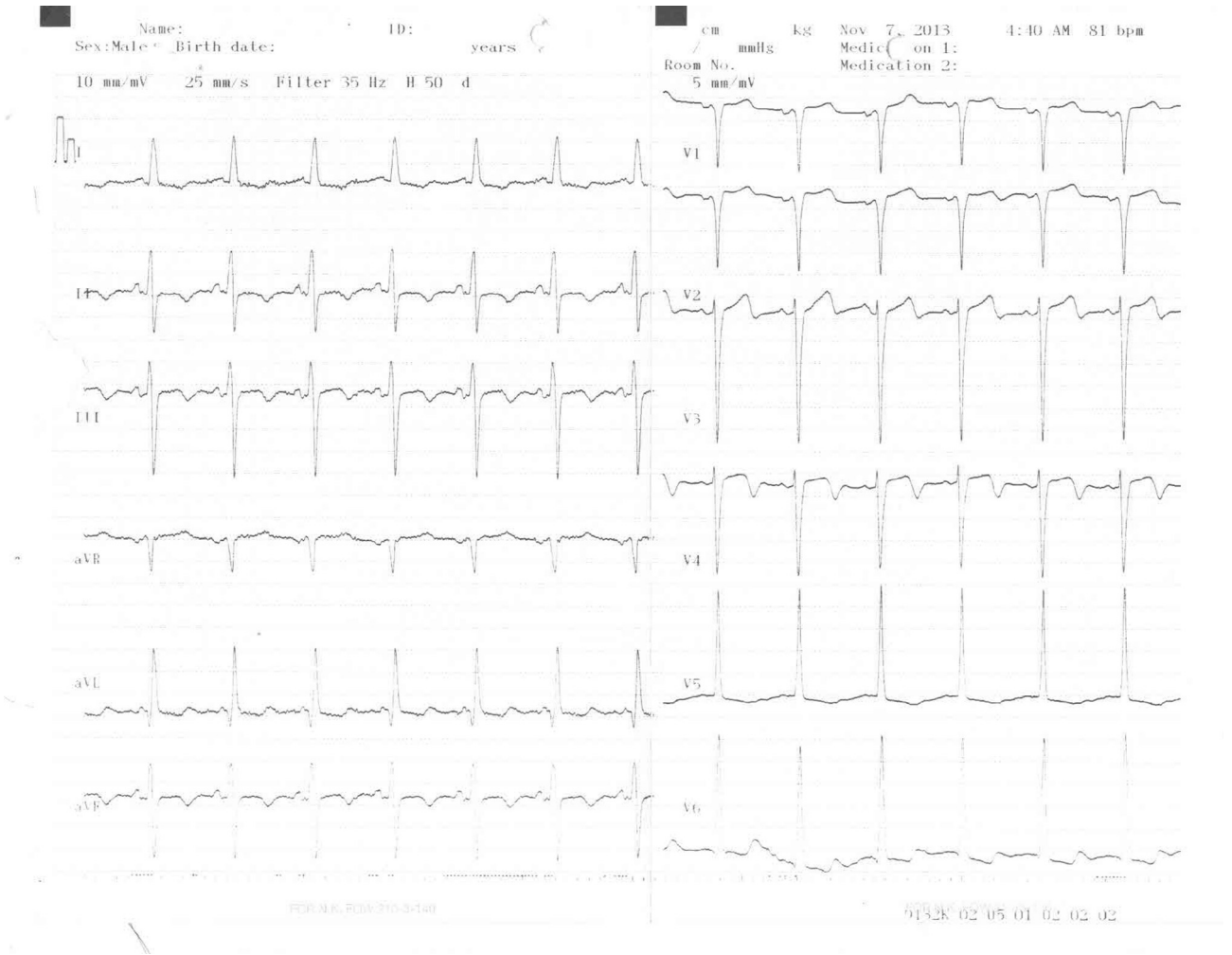

Figure 1. Electrocardiogram (ECG) readings obtained at admission of the patient.

loss in V1–V4, ST depression and T wave inversion in DII, DIII, aVF, and left ventricular hypertrophy, left axis (Fig. 1). Age, obesity, and smoking were present as risk factors for coronary heart disease. Cardiac enzymes levels were elevated. Creatine kinase (CK), myocardial band (MB) fraction of CK (CK-MB), and troponin T levels were high with values of 181 IU/L (normal range, 0–17 1U/L), 21 U/L (normal range, <24 U/L), and 0.365 ng/mL (normal range, 0–0.014 ng/mL), respectively. Other hematological and biochemical tests revealed the following: prothrombin time of 11.3 seconds (normal range, 9.4–12.5 seconds), activated partial thromboplastin time of 36 seconds (normal range, 25.4–38.4 seconds), total cholesterol 200 mg/dL (normal range, 110–200 mg/dL), triglyceride 91 mg/dL (normal range, 0–200 mg/dL), and low density lipoprotein-cholesterol 141 mg/dL (normal range, 0–130 mg/dL).

Transthoracic echocardiography revealed a left ventricular ejection fraction of 40%, and hypokinesia in the anterior wall of the left ventricle and apex. Mild aortic stenosis and aortic regurgitation were identified. The patient was admitted to the coronary intensive care unit with a diagnosis of acute coronary syndrome. Cardiac catheterization was performed. Coronary angiography revealed a complicated lesion after S2 branches of LAD, coronary artery ectasia (CAE) involving the left main artery, LCx, and myocardial bridge causing 100% systolic narrowing of LCx (Figs. 2 and 3). LCx flow was normal in systole (Fig. 4). The right common artery was normal (Fig. 5). A bare metal stent was placed in the LAD lesions with no residual occlusion. Flow defined as grade 3 according to thrombolysis in myocardial infarction scale was achieved in the areas of intervention (Fig. 6). Medications administered in the catheterization laboratory and coronary care unit included aspirin,

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