Journal of Cardiology 65 (2015) 514–518

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Manifestation of latent left ventricular outflow tract obstruction caused by acute myocardial infarction: An important complication of acute myocardial infarction

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ARTICLE INFO

Article history:
Received 25 December 2013
Received in revised form 28 July 2014
Accepted 3 August 2014
Available online 2 September 2014

Keywords:
Left ventricular outflow tract obstruction
Acute myocardial infarction
One-vessel disease
Major septal branch
Sigmoid-shaped septum

ABSTRACT

Background: Although transient left ventricular outflow tract (LVOT) obstruction is reported as a complication with acute myocardial infarction (AMI), the mechanisms and features of LVOT obstruction in AMI are unclear.

Methods and results: Herein, we present two cases of transient LVOT obstruction with anteroseptal AMI. The features of these two cases were one-vessel disease (1-VD) of the left anterior descending artery (LAD) and maintenance of blood flow to the major septal branch (SB). Moreover, LVOT obstruction was revealed after dobutamine infusion in the chronic phase and the aorto-septal angle was low in these two cases, meaning that latent LVOT obstruction was due to sigmoid-shaped septum.

Conclusions: Latent LVOT obstruction would be manifested in the acute phase of AMI. 1-VD of LAD and the maintenance of major SB blood flow are important factors with respect to the manifestation of latent LVOT obstruction.

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Introduction

Dynamic left ventricular outflow tract (LVOT) obstruction is a rare complication in acute myocardial infarction (AMI), and about 20 cases have been reported to date [1–10]. While LVOT obstruction is a potentially reversible complication of AMI, some patients with transient LVOT obstruction during AMI have experienced myocardial rupture and died [3,6]. The mechanisms and features of the appearance of LVOT obstruction in AMI are unclear. We report two cases of transient LVOT obstruction in AMI that reoccurred with dobutamine (DOB) provocation in the chronic phase. We consider the mechanisms and features of LVOT obstruction in AMI with these two cases in addition to another case which we have previously reported [9].

Case 1

A 53-year-old man with hypertension (HT) was admitted to our hospital for severe chest pain. His blood pressure (BP) was 180/110 mmHg, and heart rate (HR) was 95 beats/min. A grade 2/6 ejection systolic murmur was detected at the apex. Electrocardiogram (ECG) revealed sinus rhythm (80 beats/min) and ST elevation in the precordial leads (Fig. 1A). Transthoracic echocardiogram (TTE) showed anterior–apical hypokinesis, compensatory hyperkinesis of other segments including basal intraventricular septum and a sigmoid-shaped septum with a diminished aorto-septal angle of 89° (Fig. 1B1). This examination also demonstrated a peak Doppler velocity of 3.9 m/s corresponding to a peak pressure gradient (PG) of 61 mmHg across the LVOT (Fig. 1B2). Systolic anterior movement of mitral valve (SAM) and mild level of mitral regurgitation (MR) were noted. There were no findings of left ventricular hypertrophy (LVH). Emergent coronary angiography (CAG) was immediately performed and revealed one-vessel coronary heart disease (1-VD) with total occlusion of the mid-left anterior descending artery (LAD) (Fig. 1C1). The blood flow of the major septal branch (SB) was maintained because the LAD was occluded distal from the branching point of the major SB. Direct percutaneous coronary intervention (PCI) was performed for the occluded LAD. A metal stent was implanted into the occluded lesion, and recanalization was achieved (Fig. 1C2). Left ventriculography (LVG) demonstrated anteroseptal–apical akinesia and hypercontractility of other regions (Fig. 1C3). His peak creatine

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http://dx.doi.org/10.1016/j.jjcc.2014.08.004
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phosphokinase (CK) level was 1022 IU/L. On the next day, his ejection systolic murmur was diminished, and TTE revealed complete resolution of the LVOT obstruction. After completion of cardiac rehabilitation without any complications, exercise stress TTE (ExTTE) using the Bruce protocol was performed to examine the LVOT obstruction. The PG across the LVOT was 5 mmHg at rest, and a PG of 66 mmHg was evoked by Bruce test with 8 min 00 s of exercise. Therefore, he was treated with atenolol (25 mg/day), and follow-up ExTTE revealed a PG 14 mmHg PG after provocation using the Bruce test with 9 min 00 s of exercise. He was discharged a few days later.

After 1 year, he returned to our hospital for follow-up cardiac catheterization with no symptoms. Restenosis of the LAD lesion was not apparent by CAG. There was no PG between the left ventricular apex and the ascending aorta following interruption of atenolol administration, and intravenous infusion of DOB (15 μg/kg/min) increased the PG to 110 mmHg (Fig. 1D). BP and HR before and after DOB infusion were 170/96 mmHg and 60 beats/min, 168/82 mmHg

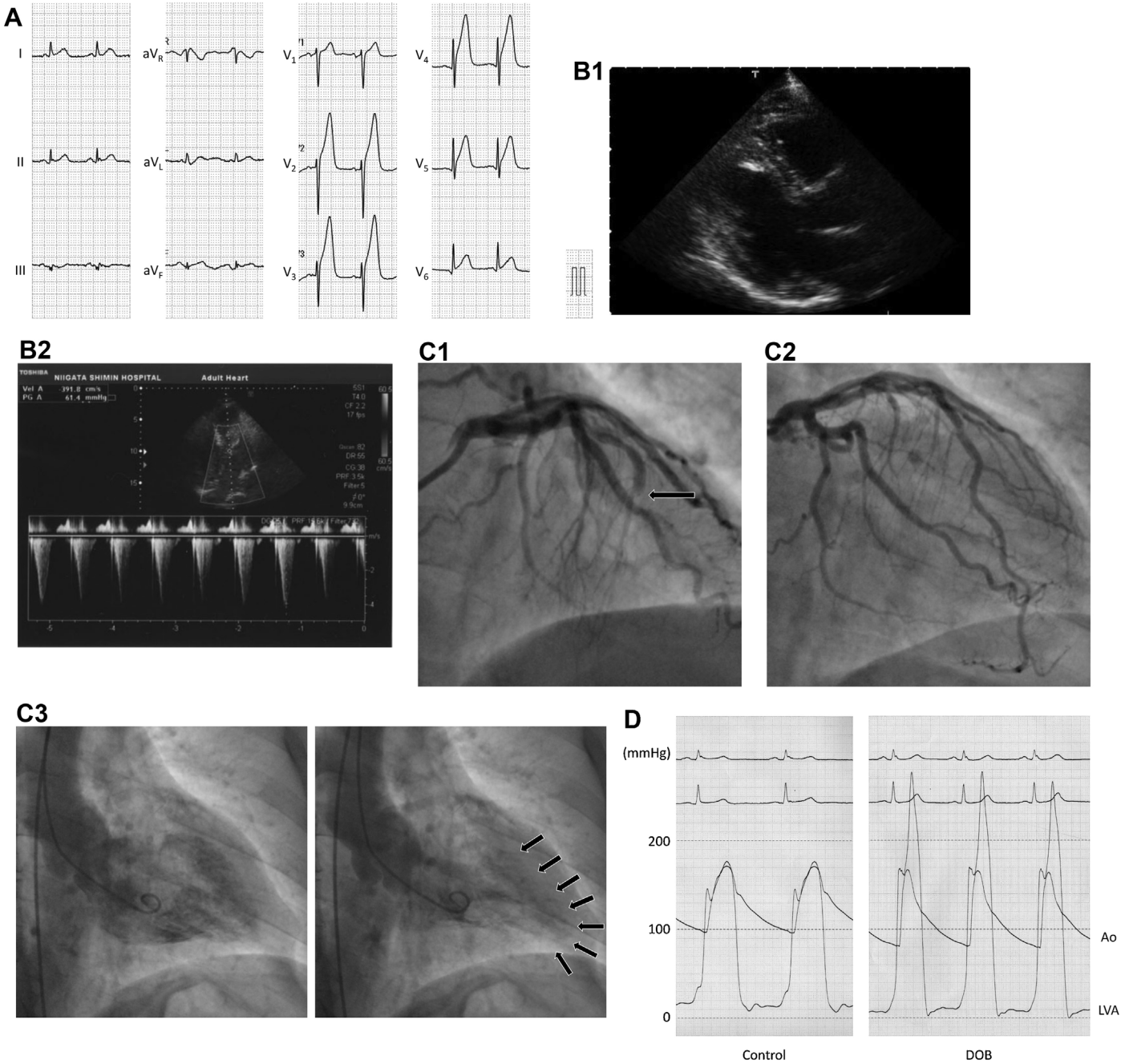

Fig. 1. (A) Electrocardiogram upon admission in Case 1. ST elevation was shown in the precordal leads. (B1) Transesophageal echocardiography findings upon admission in Case 1. Parasternal long-axis view of two-dimensional echocardiography showed a sigmoid-shaped septum with diminished aorto-septal angle of 89°. There were no indications of left ventricular hypertrophy. (B2) Spectral imaging of continuous wave Doppler of the left ventricular outflow tract from the apical position revealed the peak velocity value of 3.92 m/s indicating a peak gradient of 61 mmHg. (C) The findings of coronary angiography (CAG) in the right anterior oblique (RAO) cranial view (1), RAO view (2), and left ventriculography (LVG) in the RAO view in Case 1. Emergent CAG revealed one-vessel coronary heart disease with total occlusion of the mid left anterior descending artery (LAD) (arrow) and blood flow maintained in the major septal branch (1). A metal stent was implanted into the occluded lesion and recanalization of the LAD was achieved (2). LVG findings, in diastole (3, left) and systole (3, right), revealed anteroseptal–apical akinesia (arrows in 3, right) and hypercontractility of other regions. (D) The findings of the left ventricular apex (LVA) and the ascending aorta (AAo) pressure before (left) and after (right) dobutamine (DOB) infusion in Case 1. DOB infusion provoked a 110 mmHg pressure gradient between the LVA and the AAo.

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