Transient Marked Myocardial Thickening after Reperfused Myocardial Infarction Causing Refractory Heart Failure

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INTRODUCTION

Transient and mild myocardial thickening is a relatively common and insignificant phenomenon that occurs after reperfusion therapy in acute myocardial infarction (MI),[1] but reports of marked thickening with adverse effects are rare. The wall thickening of the infarcted area is thought to be caused by myocardial interstitial edema and/or hemorrhage.[1,2] Cardiac magnetic resonance imaging has great potential for identifying the etiological diagnosis of the wall thickening of the reperfused myocardium.[2] In general, mild left ventricular (LV) wall thickening of an infarcted area after reperfusion is associated with an increase in both LV end-systolic and end-diastolic volume in patients with acute MI.[3] According to Starling's law, this LV dilatation is useful for maintaining stroke volume (SV). It is unclear whether marked LV wall thickening after reperfusion treatment of acute MI causes severe hemodynamic deterioration.

CASE PRESENTATION

A 77-year-old man was admitted to the emergency department of our hospital because of syncope. Electrocardiography showed ST-segment elevation in inferior leads II, III, and aVF. Initial echocardiography before emergent percutaneous coronary intervention (PCI) showed mildly reduced LV ejection fraction (LVEF; 41%), mild LV dilatation (LV end-diastolic volume [LVEDV] index 49 mL/m^2) and mildly reduced SV (SV index 22 mL/beat/m^2) without LV hypertrophy (inferoposterior wall thickness 10.8 mm; Figure 1A, Video 1). We performed emergent coronary angiography. Because coronary angiography revealed total thrombotic occlusion of the left circumflex coronary artery (LCX; Figure 2A, Video 2), we performed PCI. After balloon dilatation, Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow was detected in the LCX. Unfortunately, no-reflow phenomenon developed immediately after stenting (TIMI grade 0). Because the patient's blood pressure decreased to 70 mm Hg, a bolus of noradrenaline was administered, and an intra-aortic balloon pump was inserted. An intracoronary bolus of 150 μg sodium nitroprusside was administered, and we crushed the thrombus in the implanted stent repeatedly using a larger balloon; filter protection prevented further distal embolization. The no-reflow phenomenon resolved gradually. However, final angiography showed a patent LCX, but with only TIMI grade 2 flow (Video 3).

Follow-up echocardiography immediately after PCI showed no change in the LVEF of 43%; however, there was a severe reduction in LV volume (LVEDV index 38 mL/m^2) leading to a severely reduced SV index of 18 mL/beat/m^2 and remarkable inferoposterior wall thickening of 25.5 mm. These showed the acute adverse changes of LV diastolic function after PCI (Figure 1B, Video 4). On the following day, the peak creatine kinase level was 14,450 IU/L. Associated congestive heart failure was refractory and required intra-aortic balloon pump support, continuous hemodiafiltration, and administration of catecholamine. The patient's heart failure gradually improved over a few weeks. Echocardiography 19 days after angioplasty revealed an improved LVEF of 52%, a restored LVEDV index of 56 mL/m^2, an improved SV index of 29 mL/beat/m^2, and reduction of abnormal inferoposterior wall thickening to 18.4 mm (Figure 1C, Video 5). Finally, we withdrew the intra-aortic balloon pump support, continuous hemodiafiltration, and catecholamine administration. Follow-up coronary angiography before discharge revealed TIMI grade 3 flow in the LCX (Figure 2B, Video 6). After discharge, this patient maintained good condition for 1 year, and echocardiography 12 months after acute MI showed disappearance of the inferoposterior wall thickening (12 mm).

To evaluate the mechanism of reduced LVEDV after reperfusion in this patient, we performed measurements of LV myocardial volume and pericardial total volume using two-dimensional echocardiography (modified Simpson's method; Figure 3), left atrial (LA) volume, right atrial area, and right ventricular area[4] in addition to the usual measurements of LVEDV, end-systolic volume, LVEF, and SV. Immediately after emergent PCI, LVEDV, LV end-systolic volume, and SV decreased, while LA volume and LV myocardial volume increased. Total pericardial volume and right ventricular and right atrial sizes remained constant (Figures 1A and 1B).

DISCUSSION

In general, modest LV wall thickening in the infarcted area after reperfusion is associated with an increase in LVEDV.[3] However, even if the LVEF is preserved, the development of unusual and advanced LV wall thickening may decrease LV end-diastolic volume

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VIDEO HIGHLIGHTS

Video 1: Short-axis view on transthoracic echocardiography showing hypokinesis of the inferoposterior wall with no wall thickening at admission.

Video 2: Angiographic view of the total occlusion of the LCX.

Video 3: Angiographic view of TIMI grade 2 flow of the LCX immediately after PCI.

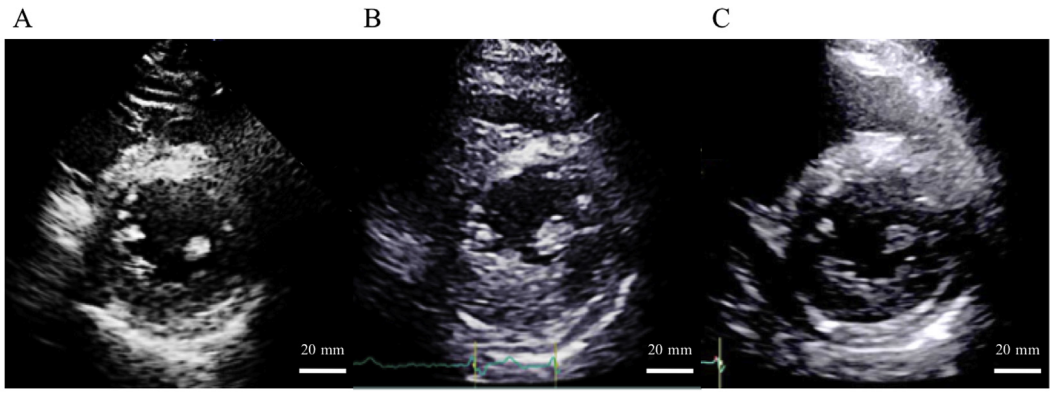
Video 4: Short-axis view on transthoracic echocardiography showing marked inferoposterior wall thickening immediately after PCI.

Video 5: Short-axis view on transthoracic echocardiography showing hypokinesis of the inferoposterior wall with no wall thickening after 19 days.

Video 6: Angiographic view of TIMI grade 3 flow of the LCX just before discharge.

View the video content online at www.cvcasejournal.com.

and SV. Immediately after emergent PCI in this patient, LVEDV, LV end-systolic volume, and SV decreased, while total pericardial volume and right ventricular and right atrial sizes remained constant. These results suggested that SV was reduced as a result of decreased LVEDV. In the fixed total pericardial volume during the acute phase, LA volume and LV myocardial volume increased. Because increasing LA volume may not be the primary phenomenon, reduced LVEDV is presumed to be caused by increased LV myocardial volume with marked inferoposterior wall thickening (Figures 1A and 1B). In contrast to this, total pericardial volume, LVEDV, and SV increased, while LV myocardial volume decreased during the subsequent 19 days compared with the post-PCI acute phase (Figure 1C). It is possible that the adaptive increase in total pericardial volume and improvement in abnormal inferoposterior wall thickening may have increased LVEDV and SV. Although little is known about the dynamics of myocardial interstitial edema after acute MI, this case may help in understanding part of that mechanism. If heart failure associated with myocardial thickening in the acute phase is severe, surgical pericardiotomy may have the potential to increase LVEDV and SV.



	A	B	C
LVEF (%)	41.2	43.3	51.5
LVEDVI (ml/m^2)	49.1	37.5	55.8
LVESVI (ml/m^2)	28.8	21.2	27.3
LVMI (g/m^2)	99.5	129.0	105.4
SV (ml/beat)	48.1	37.7	62.0
SVI (ml/beat/m^2)	22.3	17.7	29.4
Pericardial total volume (ml)	494	491	694
LA volume (ml)	59	72	72
RA area (mm^2)	18.0	17.7	19.2
RV area (mm^2)	19.8	18.5	20.1

Figure 1 Parasternal short-axis view of the end-diastolic left ventricle in echocardiography that revealed remarkable inferoposterior LV wall thickening after PCI. No wall thickening (10.8 mm) was observed at admission **(A)**. The development of remarkable wall thickening (25.5 mm) at the inferoposterior wall and contracted LV cavity can be observed on day 1 and was associated with considerable reduction in SV **(B)**. The inferoposterior wall thickening (18.4 mm) was improved on day 19 **(C)**. *LA*, Left atrial; *LVEDVI*, LVEDV index; *LVESVI*, LV end-systolic volume index; *LVMI*, LV mass index; *RA*, right atrial; *RV*, right ventricular; *SVI*, SV index.

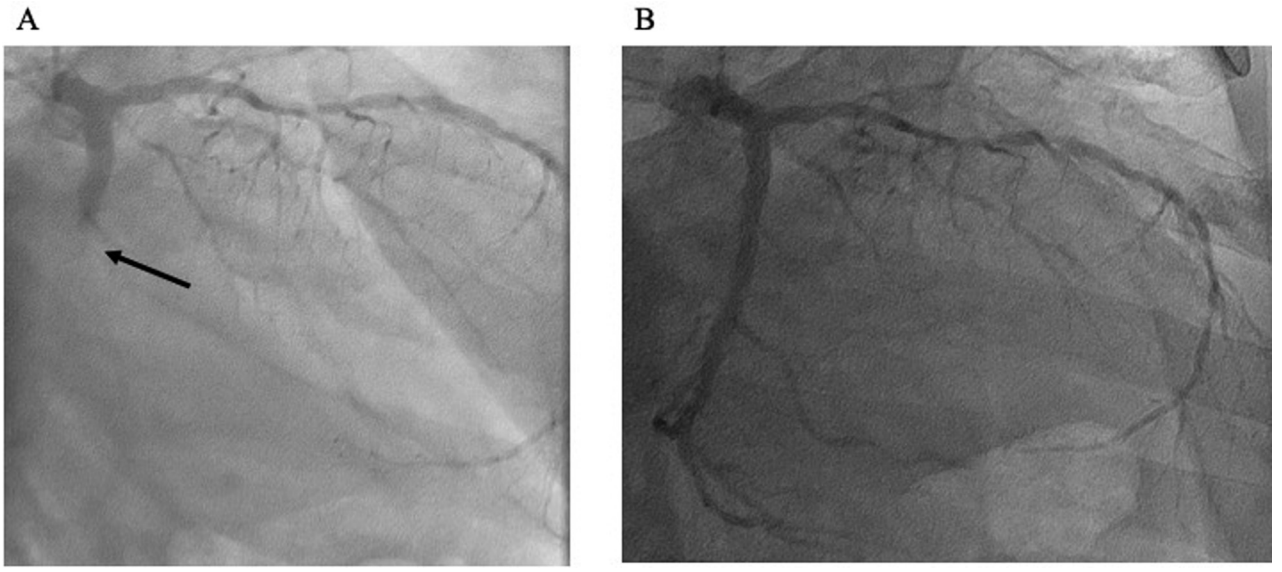


Figure 2 Coronary angiography revealed total occlusion of the proximal LCX (**A**, *arrow*). The LCX showed TIMI grade 3 flow just before discharge **(B)**.

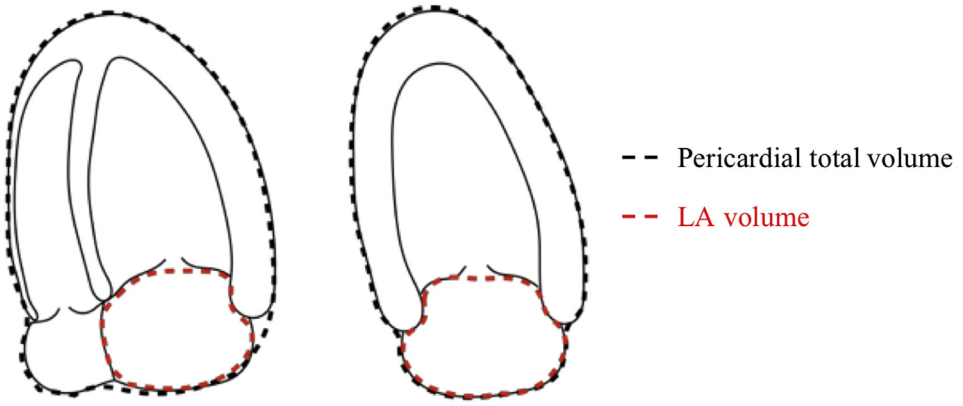


Figure 3 Schemas of apical four- and two-chamber views on echocardiography. We measured the total pericardial volume (*black dotted line* enclosing the outside of the epicardium) and left atrial (LA) volume (*red dotted line* enclosing the lumen of the left atrium) using the modified Simpson's method.

CONCLUSION

We report a case of markedly transient myocardial thickening, following a no-reflow phenomenon after emergent PCI for LCX acute MI, which caused severe refractory heart failure for a few weeks due to LV diastolic dysfunction; however, the patient recovered.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2019.09.007.

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