

Phasic Compression of Left Circumflex Coronary Artery during Atrial Systole

Edward Samuel Roberto, MD
Ajay Agarwal, MBBS, FACC

Phasic coronary artery compression is typically associated with spasm or myocardial bridging. Compression caused by acquired anatomic changes to the surrounding heart chambers has been reported only infrequently. We present a possibly unique case of phasic compression of the proximal left circumflex coronary artery during atrial contraction in association with a dilated left atrium.

A 55-year-old man with multiple cardiac risk factors presented with worsening exertional dyspnea. An electrocardiogram and echocardiogram revealed marked left atrial dilation and a left ventricular ejection fraction of 0.15 to 0.20 with elevated filling pressures. Angiograms showed compression of the proximal segment of the left circumflex coronary artery during late ventricular diastole: the compression occurred in phase with atrial systole, whereas good flow without compression was present during atrial diastole. We attributed this phenomenon to ballooning of the lateral region of the atrial wall toward the atrioventricular groove during atrial systole. The patient complied with antihypertensive therapy, and his status improved after one year.

To identify coronary artery compression in the presence of abnormal chamber geometry and to guide the treatment of the contributing medical conditions, we recommend careful analysis of angiographic results. (*Tex Heart Inst J* 2017;44(2):131-4)

Key words: Constriction, pathologic/etiology/physiopathology; coronary angiography; coronary vessel anomalies/complications/diagnosis/therapy; coronary vessels/physiopathology; heart atria/diagnostic imaging; myocardial contraction; systole; treatment outcome

From: Department of Internal Medicine (Drs. Agarwal and Roberto), Boonshoft School of Medicine, Wright State University, Dayton, Ohio 45409; and Department of Cardiology (Dr. Agarwal), Dayton VA Medical Center, Dayton, Ohio 45428

Address for reprints:
E. Samuel Roberto, MD,
1400 Abingdon Rd.,
Dayton, OH 45409

E-mail:
samroberto@mac.com

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Coronary artery phasic compression can be caused by spasm or myocardial bridging, or by acquired anatomic changes to the surrounding heart chambers that exert mechanical pressure in conjunction with contractility. Treatment varies in accordance with cause; therefore, recognizing and understanding this phenomenon is important. We report here the case of a symptomatic patient in whom we found phasic coronary artery compression, and we discuss the few published, relevant reports.

Case Report

In September 2013, a 55-year-old obese black man (body mass index, 43.5 kg/m²) presented with exertional dyspnea that had worsened over several months. He had multiple cardiac risk factors, including uncontrolled hypertension and a distant history of alcohol abuse. He reported orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema, but no fever, chills, chest pain, nausea, vomiting, or recent illness. His blood pressure upon presentation was 145/85 mmHg. A chest radiograph showed cardiomegaly and bilateral pulmonary congestion. He had a mildly elevated pro-brain natriuretic peptide level, and test results for cardiac troponins were negative.

An electrocardiogram (ECG) revealed an ectopic atrial rhythm that indicated left atrial enlargement (Fig. 1). A transthoracic echocardiogram showed an enlarged left ventricle (LV) with severe global hypokinesis, and marked left atrial dilation (Fig. 2). The patient's LV ejection fraction was low (0.15–0.20). Grade III diastolic dysfunction and elevated left atrial filling pressure were also seen.

A coronary angiogram showed a left-dominant system with nonobstructive coronary artery disease. The LV end-diastolic pressure was elevated (33 mmHg) and decreased only minimally when intraventricular nitroglycerin was administered, further correlating with chronically elevated left atrial pressure.

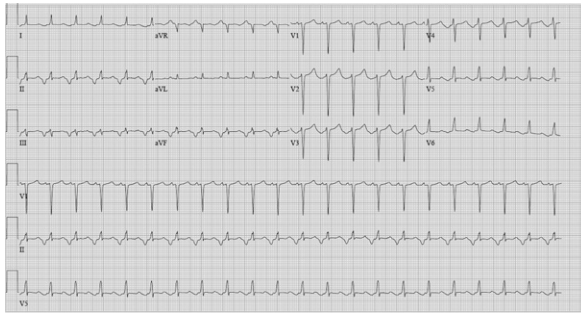


Fig. 1 Electrocardiogram shows an ectopic atrial rhythm, indicating left atrial enlargement.

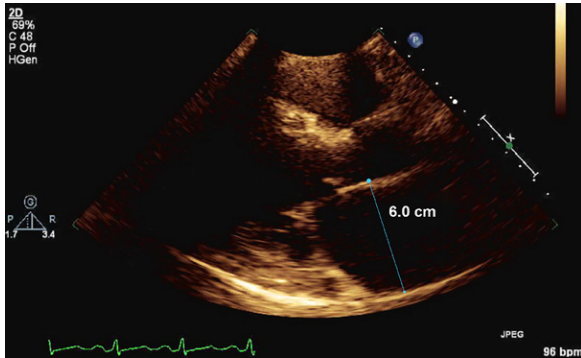


Fig. 2 Transthoracic echocardiogram (parasternal long-axis view) shows the markedly dilated left atrium.

Of note, we detected phasic compression of the proximal segment of the left circumflex coronary artery (LCx) in late ventricular diastole (Fig. 3). The rhythmic compression, with asymmetric narrowing of the vessel wall, appeared to originate from underneath the artery—just after the P wave on ECG, and in phase with atrial systole (Fig. 4A). Conversely, we observed good flow with no compression during atrial diastole (Fig. 4B).

The patient was prescribed guideline-directed anti-hypertensive therapy (diltiazem, lisinopril, and metoprolol) and enrolled in a cardiac rehabilitation program. He complied with his therapy and continued to abstain from alcohol. One year later, an echocardiogram revealed an LV ejection fraction of 0.40 to 0.45. In follow-up examinations, his clinical status remained improved.

Discussion

To our knowledge, we are the first to describe phasic compression of a proximal segment of the LCx in the atrioventricular groove during atrial systole in association with a dilated left atrium.

Beyond associations with myocardial bridging or spasm, the phasic compression of coronary arteries has been described very infrequently. The reported causes were pericardial adhesions,¹ hypertrophic cardiomy-

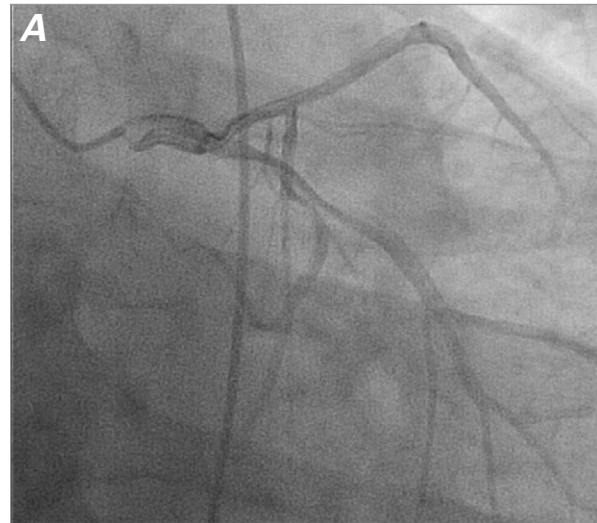


Fig. 3 Angiograms in **A**) right and **B**) left anterior oblique views (cranial angulation) show phasic compression of the proximal left circumflex coronary artery during late ventricular diastole and atrial systole.

Supplemental motion images are available for Figures 3A and 3B.

opathy,¹ constrictive pericarditis,² surgical sequelae,^{3,4} LV pseudoaneurysm,^{1,4} and anomalous right coronary artery.⁵ The clinical presentations, which correlated with the territory and severity of myocardial ischemia, included angina, ECG changes, myocardial infarction, and cardiomyopathy. Phasic arterial compression can lead to diminished coronary blood flow.⁵ Lysis of the pericardial adhesions through thoracotomies resolved the compression and improved the affected patients' clinical status.^{1,2} In the cases of LV pseudoaneurysm, coronary compression and impaired contractility resolved after bovine patch repair.^{1,4} Diastolic compression resulting from fibrous adhesions after cardiac transplantation was successfully treated with stent placement,³ as

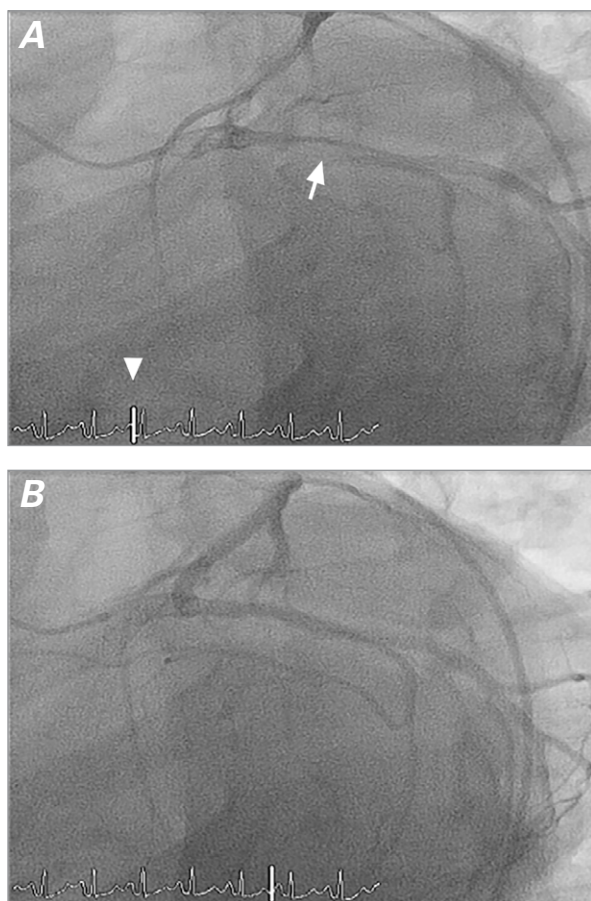


Fig. 4 Angiograms of the proximal left circumflex coronary artery show **A**) asymmetric compression (arrow) during atrial systole (arrowhead), and **B**) no compression and good flow during atrial diastole.

was systolic-phase arterial compression in the case of anomalous right coronary artery.⁵

In our patient, we determined that the rhythmic compression of the proximal LCx segment was unrelated to myocardial bridging for 2 reasons: compression occurred in atrial systole rather than in ventricular systole, and it appeared to originate from underneath the artery. The compression during atrial systole suggests that atrial contraction was the cause, and the asymmetric narrowing of the LCx wall segment indicates that the compressive force was generated from underneath the artery, not from above it. In contrast, bridging involves overlying myocardial bands that compress an artery from above.

We surmise that our patient's LCx was compressed because of its nearness to the left atrium and its course along the atrioventricular groove (Fig. 5).⁶ During atrial systole, the lateral atrial wall ballooned toward the atrioventricular groove, in the presence of atrial dilation and severely elevated LV filling pressure. In addition, dyskinetic myocardium might have increased the stretching forces on the vessel beyond the usual coronary perfusion

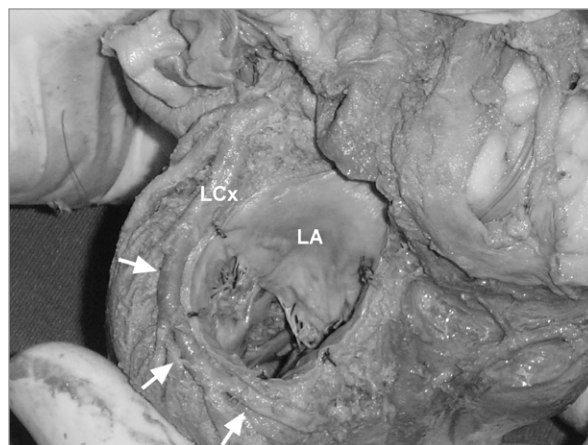


Fig. 5 Representative photograph shows the anatomic relationship between the left circumflex coronary artery (LCx) and the mitral annulus as the artery courses along the left atrium (LA) (arrows) in the atrioventricular groove.

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pressures, further contributing to the filling defect. Accordingly, we attribute the phasic compression of the LCx segment to the abnormal geometry of the dilated left atrium and the restrictive filling pattern consequent to elevated LV end-diastolic pressure.

With appropriate antihypertensive therapy, the patient's left atrial filling pressures were reduced, and his clinical and functional status subsequently improved. Careful analysis of angiographic results enabled us to identify phasic coronary artery compression in our patient.

Acknowledgments

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