Myocardial Cleft in a Patient with Acute Coronary Syndrome Assessed by Multimodal Imaging

Natalia Lorenzo, Ana Mª Rodríguez, Sonia Bartolomé, Rosa González
Department of Cardiology, Hospital Universitario Infanta Cristina, Madrid, Spain

ABSTRACT

Myocardial clefts are rare incidental findings without clinical relevance up to now. The recognition of this imaging entity is of crucial importance to avoid misdiagnosis and even more in the context of coronary artery disease.

Key words: Coronary artery disease, multimodal imaging, myocardial cleft

INTRODUCTION

Myocardial clefts are asymptomatic unusual findings during diagnostic imaging techniques. Up to now, there is no evidence that the identification of myocardial clefts is associated with pathology. Differential diagnosis of this condition is important to avoid a chain of complimentary explorations and aggressive procedures.

CASE REPORT

A 53-year-old man was admitted for unstable angina. He had previous percutaneous revascularization of the left main coronary artery, left circumflex, and right coronary artery. He had medical history of hypertension.

The electrocardiogram on admission did not show signs of ischemia and cardiac troponins were normal. Vital signs on presentation were normal. His physical findings was not remarkable.

A coronary angiogram showed progression of coronary disease, with two new severe stenoses in the right coronary artery, which were treated with two new drug-eluting stents.

In the ventriculography, a finger-shaped image was observed in the inferior basal segment with contrast penetration [Figures 1a and 2a]. Transthoracic echocardiogram was performed to further characterize this finding. It showed normal LV cavity with mild concentric hypertrophy.

Global LV ejection fraction was normal, and no segmental wall motion abnormalities were identified. A myocardial protrusion was identified in the mid-inferior wall [Figure 1b], corresponding with the ventriculography findings. It was better characterized after Sonovue® echocardiographic contrast [Figures 1c and 2b], and it was consistent with myocardial cleft. Cardiac magnetic resonance imaging (CMRI) confirmed the diagnosis [Figures 1d and 2c], showing that the cleft was contained by a normally contracting well-perfused myocardium.

DISCUSSION

Clefts (also called “crypts,” “crevices,” or “fissures”) are defined as invaginations penetrating >50% of the thickness of adjoining compact myocardium, perpendicular to the long axis of the LV, tending to narrow [Figure 2] or occlude in systole and without local hypokinesia or dyskinesia.¹ ²

Congenital LV clefts are usually asymptomatic and discovered incidentally during cross-sectional diagnostic imaging procedures. Alterations in myocardial structure...
frequently overlooked in the past can nowadays become apparent with the increasing use of multislice computed tomography and CMRI.3,4

The etiopathogenesis of myocardial clefts remains a matter of debate. It is a common belief that they represent a failure to resorb the trabeculated part of ventricular wall during normal embryological development.

Myocardial clefts were initially described in patients with genetic mutations related to hypertrophic cardiomyopathy;5 however, larger and more recent series with CMRI have found myocardial clefts in 6% of healthy individuals, commonly seen in the interventricular septum and inferior wall of the LV and rarely in apical segments of the septum and ventricular apex.6,7 Until now, there is no evidence that the identification of myocardial clefts in healthy population is associated with myocardial disease.

Crypts need to be differentiated from other myocardial wall defects with different pathological profile and clinical significance. It is important to differentiate myocardial clefts from non-compacted myocardium. A non-compacted myocardium is characterized by hypertrabeculated endocardium and a ratio of maximal thickness of the noncompacted to compacted layers of >2 (measured at end-systole in a parasternal short-axis view).8 It is also necessary to differentiate them from LV diverticulum which is defined as an outpouching structure that extends beyond the anatomic boundaries of LV, and contains endocardium, myocardium, and pericardium.7

In our case, in the setting of coronary artery disease, it should be differentiated from aneurysms and pseudoaneurysms. LV pseudoaneurysms form when cardiac rupture is contained by adherent pericardium or scar tissue. Most common causes are
myocardial infarction (MI) and surgery. LV aneurysm has a thin, scarred or fibrotic wall, devoid of muscle, or containing necrotic muscle that is a result of a transmural MI.\[8,9\]

While clefts are diagnosed with CMRI relatively easily, echocardiographic visualization can be challenging. Although it is not a frequent finding, it is important to recognize it to avoid misdiagnosis that could lead to aggressive procedures.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES
1. Johansson B, Maceira AM, Babu-Narayan SV, Moon JC, Pennell DJ, Kilner PJ. Clefts can be seen in the basal inferior wall of the left ventricle and the interventricular septum in healthy volunteers as well as patients by cardiovascular magnetic resonance. J Am Coll Cardiol 2007;50:1294-5.
2. Erol C, Koplay M, Olcay A, Kivrak AS, Ozbek S, Seker M, et al. Congenital left ventricular wall abnormalities in adults detected by gated cardiac multidetector computed tomography: Clefts, aneurysms, diverticula and terminology problems. Eur J Radiol 2012;81:3276-81.
3. Srichai MB, Hecht EM, Kim DC, Jacobs JE. Ventricular diverticula on cardiac CT: More common than previously thought. AJR Am J Roentgenol 2007;189:204-8.
4. Wein M, Wolf-Puetz A, Niehues R, Klein T, Kilner PJ, Klein RM. Multiple left ventricular interseptal clefts. Multimodality imaging appearance and differential diagnosis. Herz 2011;36:438-43.
5. Germans T, Wilde AA, Dijkmans PA, Chai W, Kamp O, Pinto YM, et al. Structural abnormalities of the interseptal left ventricular wall detected by cardiac magnetic resonance imaging in carriers of hypertrophic cardiomyopathy mutations. J Am Coll Cardiol 2006;48:2518-23.
6. Weiford BC, Subbarao VD, Mulhern KM. Noncompaction of the ventricular myocardium. Circulation 2004;109:2965-71.
7. Aquaro GD, Di Bella G, Strata E, Deiana M, De Marchi D, Pingitore A, et al. Cardiac magnetic resonance findings in isolated congenital left ventricular diverticuli. Int J Cardiovasc Imaging 2007;23:43-7.
8. Ohlow MA. Congenital left ventricular aneurysms and diverticula: Definition, pathophysiology, clinical relevance and treatment. Cardiology 2006;106:63-72.
9. Bisoyi S, Dash AK, Nayak D, Sahoo S, Mohapatra R. Left ventricular pseudoaneurysm versus aneurysm a diagnosis dilemma. Ann Card Anaesth 2016;19:169-72.