Myocardial crypts, recesses, and outpouchings: it is time to clarify

To the editor Crypts, recesses, and outpouchings are terms often indiscriminately used for identifying a broad spectrum of distinctive congenital or acquired entities, with different natural history, prognostic outcomes, and therapeutic options, knowledge of which is still limited in clinical practice. In the same way, the increased diagnostic accuracy of imaging techniques, such as cardiac magnetic resonance, led to a progressive improvement in appreciating subtle structural abnormalities in the cardiac wall, thus representing a useful tool which may help physicians to provide the most appropriate clinical approach and therapeutic strategy.[1]

The term "myocardial crypts" was first coined by Germans et al,[2] to describe discrete V- or U-shaped contractile invaginations, mainly located at the inferior basal wall and the interventricular septum, and penetrating more than 50% of adjacent compacted myocardial thickness, with total or near total systolic obliteration. Albeit firstly identified as early markers of gene carrier mutations in hypertrophic cardiomyopathy, data from the literature show that myocardial crypts are present even in patients with hypertensive cardiomyopathy, as well as in healthy volunteers.[2] For these reasons, their clinical significance is still uncertain, particularly in asymptomatic subjects, raising the suspicion of myocardial structural bystanders, incidentally detected by cardiac imaging tools in this subset population.

As opposed to myocardial crypts, the terms "recesses" or "partial crypts" have been proposed to identify invaginations involving less than 50% of myocardial thickness, defined as just irregularities of the ventricular wall arising from the endocardial border, without any clinical significance.[3] Both crypts and recesses share a common etiopathogenesis, related to a failure in reabsorbing ventricular myocardial trabeculations during the embryologic period. In this context, Singh et al[4] have hypothesized a common developmental anomaly with isolated ventricular noncompaction, which is distinguished by the presence of a bilayered myocardium with multiple prominent trabeculations, communicating intertrabecular spaces, and an end-systolic ratio between noncompacted and compacted myocardium greater than 2:1.[4,5]

On the other hand, the term "outpouching" classically refers to the protrusion of a structure outside the anatomic boundaries of an organ, as a consequence of pathologic processes or physiologic anatomic variants.[1] Cardiac outpouchings encounter the following distinctive structural entities: 1) aneurysms, 2) pseudoaneurysms, 3) diverticula, and 4) herniations.

Left ventricular (LV) aneurysms are outpouchings involving the endocardium, the epicardium, and a dyskinetic scarred myocardium, with paradoxical bulging expansion during systole. Distinctive morphological features generally include: a laminar blood flow through a wide neck, a smooth transition from a normal myocardium to a thinned aneurysmal wall, and a ratio of the maximum neck diameter to the maximum aneurysm diameter greater than 0.5.[6] Acquired LV aneurysms most frequently develop after transmural myocardial infarctions (mainly involving the apical or anterior wall as a consequence of the occlusion of the descending anterior coronary artery), while other potential etiologies include chest trauma, cardiac surgery, hypertrophic cardiomyopathy, or infectious disease.[7] Less frequently, true aneurysms are detected in childhood, and may be caused by stenosis or intimal proliferation, coronary hypoplasia, or intrauterine viral infections, which occurred during the prenatal period.[8] Unlike true aneurysms, LV pseudoaneurysms (or "false aneurysms") develop when a cardiac rupture is contained by the adherent overlying pericardium and thrombotic material, thus preventing self-evident cardiac tamponade and death. More frequently, they are consequent to ischemic heart disease (particularly after transmural myocardial infarctions involving the circumflex coronary artery), and less commonly, they occur after cardiac surgery, chest trauma, or infections.[6] Morphologically, the presence of a narrow neck connecting a globular echo-free space to the cardiac chamber, an abrupt transition from a normal myocardium to an altered ventricular wall, a turbulent flow through the neck of the cavity, and a maximum orifice to chamber diameter ratio less than 0.5 are the main distinctive features of LV pseudoaneurysms.[9] Due to a lacking

TABLE 1 Characteristic features of myocardial structural abnormalities

Abnormality	Diagnostic feature	Main LV location	Neck	Contractility	CMR appearance	Histology
Recess	<50% wall thickness	Variable	Narrow	Synchronous	Absence of delayed enhancement	Endocardium, myocardium, epicardium
Crypt	>50% wall thickness but not reaching the epicardium	Inferior, septal	Narrow	Synchronous	Absence of delayed enhancement	Endocardium, myocardium, epicardium
Diverticulum	Beyond the cardiac wall	Apical, subaortic	Variable	Synchronous	Absence of delayed enhancement	Endocardium, myocardium, epicardium
Aneurysm	Beyond the cardiac wall	Apical	Wide	Dyskinesia	Delayed enhancement of the myocardial wall	Fibrous tissue interposed to the spared myocardium
Pseudoaneurysm	Beyond the cardiac wall	Posterior	Narrow	Akinesia	Delayed enhancement of the overlying pericardium	Pericardium with mural thrombus
Herniation	Beyond the cardiac wall	Variable	Variable	Synchronous	Absence of delayed enhancement and discontinuity of the overlying pericardium	Endocardium, myocardium

Abbreviations: LV, left ventricle; CMR, cardiac magnetic resonance

structural support of myocardial layer, LV pseudoaneurysms carry a higher early risk of rupture, therefore, urgent surgical treatment is often required, compared with LV aneurysms, which are less prone to rupture and are often treated medically, reserving surgical repair only for patients with associated complications, including systemic embolism, recurrent heart failure, or refractory arrhythmias.[1]

Unlike ventricular aneurysms and pseudoaneurysms, LV diverticula are defined as congenital outpouchings containing all 3 layers of the ventricular wall (endocardium, myocardium, and pericardium), with synchronous myocardial contractility and systolic obliteration, in the absence of structural wall abnormalities. Their pathogenic process has been postulated to be the result of failure in fusion of the cardiac loop to the yolk sack during the fourth embryonic week, which may be triggered by favoring conditions, such as intrauterine viral infections, muscular or connective tissue defects, or excessive primordial cell stimulation.[10] LV diverticula are often located at the cardiac apex, and are mostly involved as part of Cantrell syndrome, characterized by midline thoracoabdominal defects (including omphalocele, epigastria hernia, or sternal abnormalities), congenital heart disease (mainly atrial or ventricular septal defects, tricuspid atresia, or tetralogy of Fallot), and pericardial agenesis.[11] Nonapical diverticula are generally described as isolated cardiac defects of multiple shapes and sizes usually arising from the anterior wall or the subaortic region.[10] Both apical and nonapical LV diverticula are often asymptomatic findings incidentally detected by imaging tools, and in few cases, spontaneous regression has been noticed. For these reasons, a close follow-up is often pursued, reserving further management only for symptomatic patients or those with associated congenital defects.[1]

Finally, cardiac herniations are infrequent, life-threatening outpouchings, defined as myocardial protrusions through a pericardial tear. They occur as a consequence of congenital pericardial defects, blunt or penetrating chest trauma, or after surgical procedures (most frequently after lobectomy, pneumonectomy, or consequent to cardiac surgery).[12] Clinical presentation is widely variable, ranging from asymptomatic patients to detrimental consequences, such as the twisting of great vessels and decreased cardiac output, or myocardial strangulation, leading to subsequent ischemic injury or potential lethal arrhythmias. Therefore, in such clinical contexts, emergency surgical repair is mandatory.[13] Cardiac magnetic resonance plays a pivotal role in differential diagnosis of ventricular structural abnormalities due to its excellent tissue characterization.[1] Crypts, recesses, and diverticula are characterized by a preserved contractility and normal myocardial signal intensity, as opposed to true ventricular aneurysms (marked by a dyskinetic, delayed enhanced wall without pericardial enhancement, referring to scarred myocardium), and pseudoaneurysms (which are often identified by the obliteration of the myocardial-pericardial interface, and sharp, delayed enhancement of the overlying pericardium).[14] Furthermore, cardiac magnetic resonance may also detect cardiac herniations through pericardial tears, although their identifications are not always straightforward due to their intermittent appearance and variations with the patient's positional changes.[15] The main distinctive features of the aforementioned myocardial structural abnormalities are summarized in TABLE 1.

In conclusion, crypts, recesses, and outpouchings encompass a broad spectrum of distinctive

and heterogeneous entities, with a different impact on the patient's natural history and prognostic outcome. Therefore, their knowledge and timely recognition is of paramount importance to provide the most appropriate diagnostic and therapeutic decisions.

ARTICLE INFORMATION

AUTHOR NAMES AND AFFILIATIONS Riccardo Scagliola, Sara Seitun, Gian M. Rosa (RS: Division of Cardiology, Department of Emergency, Cardinal Massaia Hospital, Asti, Italy; SS: Department of Radiology, IRCCS Ospedale Policlinico San Martino, Genoa, Italy; GMR: Cardiovascular Disease Unit, IRCCS Ospedale Policlinico San Martino, Genoa, Italy)

CORRESPONDENCE TO Riccardo Scagliola, MD, Division of Cardiology, Department of Emergency, Cardinal Massaia Hospital, Asti, Italy, C.so Dante n. 202, 14 100 Asti, Italy, phone: +39 3407326833, email: risca88@live.it

CONFLICT OF INTEREST None declared.

HOW TO CITE Scagliola R, Seitun S, Rosa GM. Myocardial crypts, recesses, and outpouchings: it is time to clarify. Pol Arch Intern Med. 2021; 131: 769-771. doi:10.20452/pamw.16063

REFERENCES

1 Sharma A, Kumar S. Overview of left ventricular outpouchings on cardiac magnetic resonance imaging. Cardiovasc Diagn Ther. 2015; 5: 464-470.

2 Germans T, Wilde AA, Dijkmans PA, et al. Structural abnormalities of the inferoseptal left ventricular wall detected by cardiac magnetic resonance imaging in carriers of hypertrophic cardiomyopathy mutations. J Am Coll Cardiol. 2006; 48: 2518-2523.

3 Petryka J, Baksi AJ, Prasad SK, et al. Prevalence of inferobasal myocardial crypts among patients referred for cardiovascular magnetic resonance. Circ Cardiovasc Imaging. 2014; 7: 259-264.

4 Singh Y, Singh B, Bhairappa S, et al. A rare association of left ventricular diverticulum and noncompaction: continuum of common developmental abnormality? Echocardiography. 2013; 30: E171-E174.

5 Bennett CE, Freudenberger R. The current approach to diagnosis and management of left ventricular noncompaction cardiomyopathy: review of the literature. Cardiol Res Pract. 2016; 2016: 5172308.

6 Gatewood RP Jr, Nanda NC. Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. Am J Cardiol. 1980; 46: 869-878.

7 Zoffoli G, Mangino D, Venturini A, et al. Diagnosing left ventricular aneurysm from pseudo-aneurysm: a case report and a review in literature. J Cardiothorac Surg. 2009; 4: 11.

8 Ohlow MA, Secknus MA, Geller JC, et al. Prevalence and outcome of congenital left ventricular aneurysms and diverticula in an adult population. Cardiology. 2009; 112: 287-293.

9 Gologorsky E, Gologorsky A, Barron ME, et al. Echo rounds: transesophageal echocardiography images of an inferior wall pseudoaneurysm: a difficult differential diagnosis. Anesth Analg. 2010; 111: 613-615.

10 Halpern L, Garabedian C, Worrall NK. congenital ventricular diverticulum or aneurysm: a difficult diagnosis to make. Case Rep Cardiol. 2018; 2018: 5839432.

11 Manieri S, Adurno G, Iorio F, et al. Cantrell's Syndrome with left ventricular diverticulum: a case report. Minerva Pediatr. 2013; 65: 93-96.

12 Kawamukai K, Antonacci F, Di Saverio S, Boaron M. Acute postoperative cardiac herniation. Interact Cardiovasc Thorac Surg. 2011; 12: 73-74.

13 Wielenberg AJ, Demos TC, Luchette FA, Bova D. Cardiac herniation due to blunt trauma: early diagnosis facilitated by CT. AJR Am J Roentgenol. 2006; 187: W239-W240.

14 Konen E, Merchant N, Gutierrez C, et al. True versus false left ventricular aneurysm: differentiation with MR imaging-initial experience. Radiology. 2005; 236: 65-70.

15 Bogaert J, Francone M. Pericardial disease: value of CT and MR imaging. Radiology. 2013; 267: 340-356.