ESC European Society of Cardiology
European Heart Journal - Case Reports
doi:10.1093/ehjcr/ytaa472

CASE REPORT
Cardiac imaging

Incidental detection of myocardial clefts in a patient with acute inferior ST-segment elevation myocardial infarction: a very unusual and potentially ominous association—a case-report

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Received 2 April 2020; first decision 19 May 2020; accepted 12 November 2020

Background The crescent availability of high-resolution cardiac imaging allows detection of myocardial structural variations. Differentiate these entities from others with different clinical significance can be challenging. Clinicians should be familiar with myocardial clefts to avoid erroneous diagnosis.

Case summary A 63-year-old smoker man alerted the emergency medical system for sudden chest pain. The electrocardiogram showed Pardee wave in inferior leads. Coronary angiography evidenced a 100% occlusion of right coronary artery that was treated by angioplasty and drug-eluting stent implantation with optimal angiographic result. At ventriculography, two fissure-like protrusion were observed in the inferior wall. Urgent transthoracic echocardiogram (TTE) demonstrated two deep fissures on the mid-inferior wall, contained by a thin sub-epicardial layer, with sub-total obliteration during systole. A diagnosis of myocardial clefts was suspected and after Heart Team discussion, a conservative strategy was proposed. Early cardiac magnetic resonance (CMR) confirmed two myocardial crypts on the mid-inferior wall. Stability of myocardial fissures and absence of left ventricular remodelling was confirmed by TTE, in a 2 years of follow-up period.

Discussion Myocardial cleft should always be considered in the differential diagnosis of myocardial wall defects. In a patient presenting with an acute myocardial infarction, the main differential diagnosis is pseudoaneurysm. In this setting modified TTE views and meticulous analysis of CMR sequences are recommended to confirm the diagnosis and estimate the risk of myocardial rupture.

Keywords Myocardial cleft • Pseudoaneurysm • Acute myocardial infarction • Echocardiography • Cardiac magnetic resonance • Case report

Learning points

- Myocardial clefts should be considered in the differential diagnosis of myocardial wall defects and modified transthoracic echocardiogram views are often needed to their optimal delineation.
- When a myocardial wall defect is demonstrated in the context of acute myocardial infarction, the risk of ventricular rupture should always be estimated by meticulous analysis of cardiac magnetic resonance sequences.

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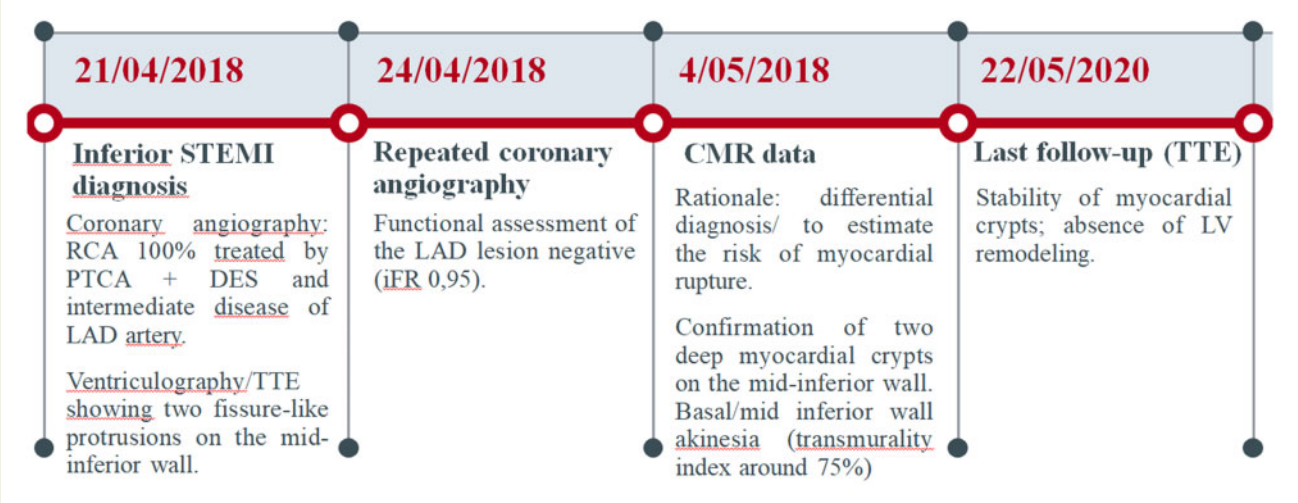
Handling Editor: Andre Dias
Peer-reviewers: Marcelo Haertel Miglioranza and Luca Arcari
Compliance Editor: Linh Ngo
Supplementary Material Editor: Fabienne Vervaat

Introduction

Myocardial clefts are asymptomatic unusual findings during diagnostic imaging techniques.[1] Up to now, there is no definite evidence that their presence is associated with pathological causes/process. Clinicians should be aware of this rare entity in order to avoid erroneous diagnosis.

Timeline



Case presentation

A 63-year-old Caucasian smoker man alerted the emergency medical system for sudden chest pain. He neither had a relevant past medical history nor other cardiac risk factors. Physical examination at presentation was normal. The electrocardiogram showed Pardee wave in inferior leads. Immediate coronary angiography evidenced a 100% occlusion of the right coronary artery (RCA), complete absence of visible retrograde collateral flow and intermediate disease of the left anterior descending (LAD) artery. The culprit lesion was treated by angioplasty and drug-eluting stent implantation with optimal angiographic result. Symptom onset-to-balloon time was 120 minutes. At ventriculography, two finger-shaped images with contrast penetration were observed in the inferior wall (*Figure 1* and Supplementary material online, *Video S1*). Urgent transthoracic echocardiogram (TTE) was performed to assess myocardial and valve function and further characterize this finding. It showed a normal left ventricular (LV) cavity with mild concentric hypertrophy, an LV ejection fraction (LVEF) of 50% with severe hypokinesia of inferior wall. Two fissure-like protrusions penetrating more than 50% of the wall thickness, contained by a thin sub epicardial layer and with sub-total obliteration during systole, were confirmed on the mid-inferior wall (*Figure 2* and Supplementary material online, *Videos S2* and *S3*). A diagnosis of incidental myocardial clefts was suspected. After Heart Team discussion, a conservative approach was applied. Three days later, the patient underwent repeated coronary angiography: functional assessment of the LAD lesion was negative with instantaneous fractional reserve of 0.95. Early after discharge, cardiac magnetic resonance (CMR) was performed with demonstration of two deep myocardial crypts on the mid-inferior wall (*Figure 3A* and Supplementary material online, *Video S4*). Stability of myocardial fissures and absence of LV remodelling was confirmed by regular TTE follow-ups in a 2 years window.

Discussion

Clefts (also called 'crypts', or 'fissures') are defined as invaginations penetrating more than 50% of the adjoining compact myocardium thickness, perpendicular to the long axis of the LV, tending to narrow or occlude in systole and without local hypokinesia or dyskinesia.[2,3] They are more commonly found in the interventricular septum and LV inferior wall.[2] Congenital LV clefts are usually asymptomatic and nowadays more often incidentally discovered during cross-sectional diagnostic imaging procedures.[4] Their aetiopathogenesis remains a source of debate but it is generally admitted that they represent a failure to resorb the trabeculated part of ventricular wall during normal embryological development. Myocardial clefts were initially described in early postmortem studies of patients with hypertrophic cardiomyopathy (HCM).[5] Donald Teare, with his historical publication in 1958, did the first pathological description.[6] Their systolic narrowing or obliteration explains why these entities remained largely under-recognized by pathologists: postmortem heart specimens are always in a contracted state and features are closer to end-systolic cardiac images than end-diastolic ones. In addition, they may be easily overlooked by TTE due to their frequent location in non-standard views.[7] These findings were later described in patients with genetic mutations related to HCM suggesting a potential role as preclinical

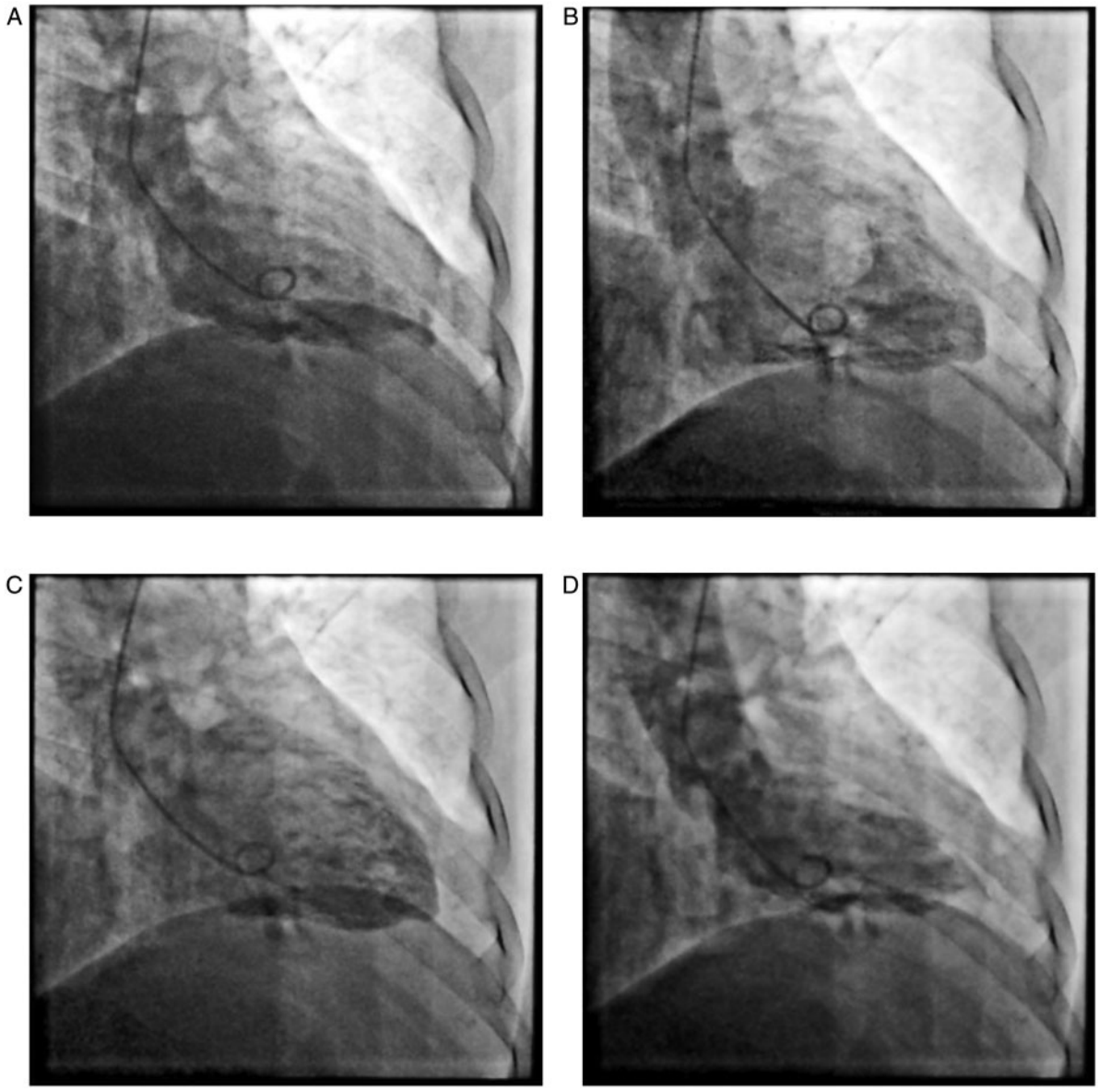


Figure 1 Ventriculography showing two finger-shaped images with contrast penetration in the inferior wall during different phases of cardiac cycle (*A*, proto-diastole; *B*, tele-diastole; *C*, proto-systole; and *D*, tele-systole).

marker of the disease.[8] However, larger and more recent CMR series have found myocardial clefts in 6% of healthy individuals, toning down the initial enthusiasm; noteworthy, on follow-up, there was no significant difference in major adverse cardiac events or stroke incidence compared to the control group.[9] Crypts need to be differentiated from other myocardial wall defects with different pathological profile and clinical significance. Especially in presence of multiples clefts, they can mimic trabeculations characteristic of non-compacted myocardium (NCM). There is considerable debate about the NCM diagnostic criteria. 'Jenni criteria' are the most used in the literature and include: a bilayered myocardium, a non-compacted to compacted ratio >2:1, communication with the intertrabecular space demonstrated by colour Doppler, absence of coexisting cardiac abnormalities, and presence of multiple prominent trabeculations in end-systole.[10,11] It is also mandatory to differentiate them from LV diverticulum, aneurysm and pseudoaneurysm (*Tables 1* and *2*). Diverticulum is an out-pouching that contains endocardium, myocardium and pericardium, and displays synchronous contractility.[12–14] LV aneurysm represents a potential complication of a transmural acute myocardial infarction (AMI) and it is characterized by a thin, scarred or fibrotic wall, devoid of muscle or containing necrotic muscle; the involved wall segment is either akinetic (without movement) or dyskinetic (with paradoxical ballooning) during systole.[13,15] Pseudoaneurysm forms when cardiac rupture is contained by

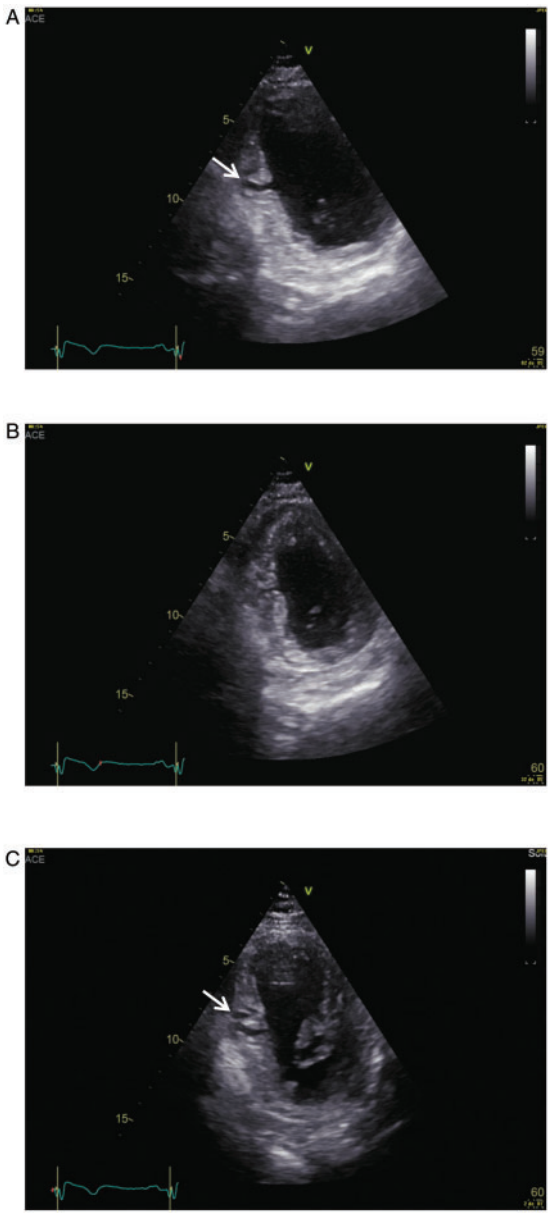


Figure 2 Echocardiographic features of myocardial crypts. (*A*) Transthoracic echocardiogram two-chamber view showing diastolic features of a myocardial crypt: penetration perpendicular to myocardial wall and contention by thin sub epicardial layer (white arrow); (*B*) systolic sub-total obliteration on transthoracic echocardiogram two-chamber view; and (*C*) modified two-chamber view demonstrates the presence of second, more apical crypt parallel of the previous one (white arrow).

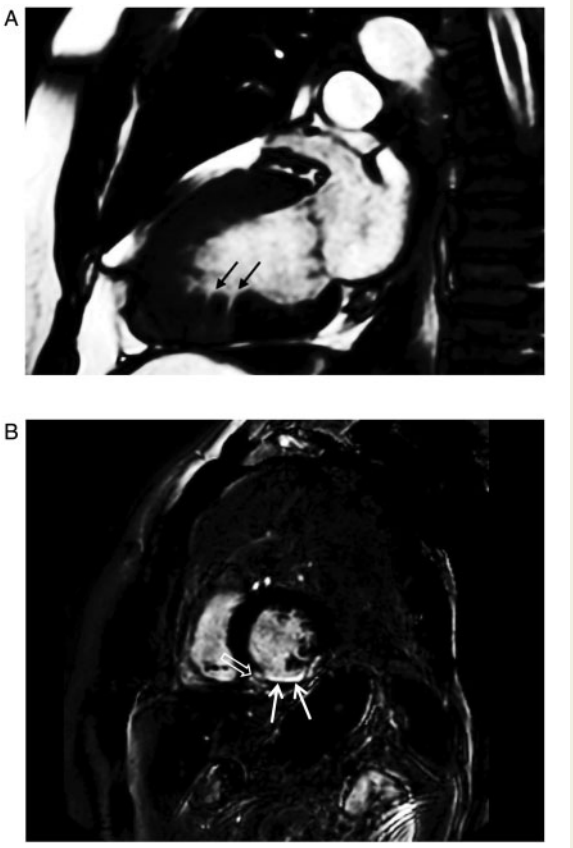


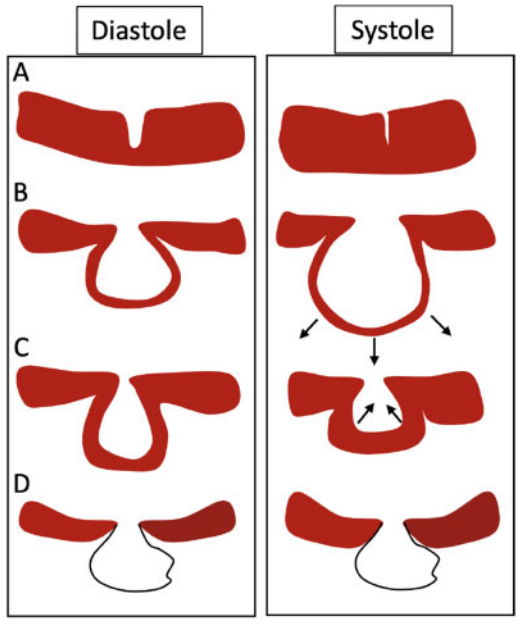
Figure 3 (*A*) Optimal delineation of the two crypts in cardiac magnetic resonance two-chamber view sequence (black arrows) and (*B*) obvious hyperenhancement in the inferior wall (white arrows) with adjacent myocardial crypt (open arrow) on the cardiac magnetic resonance-late gadolinium enhancement short-axis sequence.

adherent pericardium or scar tissue. The most common causes are AMI and surgery.[15,16] In our case, in the setting of inferior AMI, the main differential diagnosis was with LV pseudoaneurysm. Analysing CMR sequences we observed an LVEF of 48% with basal and mid inferior wall akinesia, without septal, apical, or lateral extension (2 of 16 segments affected). These two segments were moderately but not severely thinned; all segments supplied by RCA were affected but interestingly, the only frankly transmural [late gadolinium enhancement (LGE) transmurality around 75%] was the mid-inferior one (*Figure 3B*). The more clarifying sequence to detect myocardial clefts was the two-chamber cine view, in which two deep myocardial crypts were evident on mid-inferior wall, surrounded by moderately thinned myocardium and with sub-total obliteration during systole (Supplementary material online, *Video S4*). The differential diagnosis was challenging but several features argued for a fortuitous finding: relatively brief symptom onset-to-balloon time with low probability of acute mechanical complications; moderate but not severe thinned wall associated with myocardial invaginations; absence of dyskinesia; absence of scar discontinuity in LGE sequences. We empirically assess a low risk of myocardial rupture and a conservative approach was considered the best management in this setting. Stability of myocardial crypts was confirmed by regular TTE follow-ups in a 2 years window. To our knowledge, this is the first reported case of clefts located in a myocardial region affected by AMI.

Table 1 Differential diagnosis of myocardial wall defects

	Myocardial cleft	Diverticulum	Pseudoaneurysm	Aneurysm
Aetiology	Embriogenesis defect	Embriogenesis defect	Acquired (most related to AMI and cardiac surgery)	Congenital Acquired (in the context of transmural AMI)
Implantation base on ventricle	Narrow base	Narrow base	Variable (usually narrow base)	Wide base
Size	Small	Variable (usually small/moderate size)	Variable	Large
Common location	Interventricular septum and inferior wall	Most on the apex of left ventricle	Variable (particularly on inferior wall)	Variable (commonly on the apex and rarely in posterolateral wall)
Histology	Myocardial disarray of adjacent myocardium	The three layers are usually present (endocardium, myocardium, and pericardium)	The wall contains only a pericardial layer	The three layers are present: myocardial layer is tinned and composed of scar/fibrotic tissue
Kinesis	Normal	Normal	Akinesia/dyskinesia	Akinesia/dyskinesia
Contractility	Synchrony with heart's rhythm (tending to narrow/occlude in systole)	Synchrony with heart's rhythm	Absent contractility	Absent or paradoxical contractility
Complications	Usually not occurring	Usually not occurring	Rupture Thromboembolism	Arrhythmia, thromboembolism, heart failure, rupture
Prognosis	Good	Depending on the associated anomalies	Bad	Bad

Table 2 Schematic representation of myocardial cleft (A), ventricular aneurysm (B), ventricular diverticulum(C), and pseudoaneurysm (D) in diastole and systole



Conclusion

This case highlights the practical issues related to the differential diagnosis of myocardial wall defects, especially in the setting of acute coronary syndromes. Myocardial cleft should be considered in the diagnostic process to limit useless interventions; modified TTE views may help to its optimal delineation. In the setting of AMI, meticulous analysis of CMR sequences and high-resolution TTE images are imperative to estimate the risk of myocardial rupture.

Lead author biography

Dr Etienne Hoffer, corresponding author, is a cardiologist with tropism for echocardiography, including stress method. Since 2010, he's head of the cardiology department at the CHR Citadelle in Liège. He has conducted several multicentres studies in the fields of heart failure, dyslipidaemia, and anticoagulant therapy. He has published more than 40 papers in peer-reviewed journal and is currently reviewer for a couple of them. He's a collaborator of the Liège University and secretary of the local ethics committee.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

Funding: none declared.

References

1. Child N, Muhr T, Sammut E, Dabir D, Ucar EA, Bueser T *et al.* Prevalence of myocardial crypts in a large retrospective cohort study by cardiovascular magnetic resonance. *J Cardiovasc Magn Reson* 2014;**16**:66.
2. 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**:1294–1295.
3. 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**:3276–3281.
4. Cresti A, Cannarile P, Aldi E, Solari M, Sposato B, Franci L *et al.* Multimodality imaging and clinical significance of congenital ventricular outpouchings: recesses, diverticula, aneurysms, clefts, and crypts. *J Cardiovasc Echography* 2018;**28**:9–17.
5. Kuribayashi T, Roberts WC. Myocardial disarray at junction of ventricular septum and left and right ventricular free walls in hypertrophic cardiomyophaty. *Am J Cardiol* 1992;**70**:1333–1334.
6. Teare D. Asymmetrical hypertrophy of the heart in young adults. *Br Heart J* 1958;**20**:1–8.
7. Juan H, Waller AH, Sabo SS. Myocardial crypts: role of modified two-chamber view Q. *J Med* 2015;**108**:139–140.
8. Germans T, Wilde AA, Dijkmans PA, Chai W, Kamp O, Pinto YM *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.
9. Petryka J, Baksi AJ, Prasad SK, Pennell DJ, Kilner PJ. Prevalence of inferobasal myocardial cripts in patients referred for cardiovascular magnetic resonance. *Circ Cardiovasc Imaging* 2014;**7**:259–264.
10. Weiford BC, Subbarao VD, Mulhern KM. Noncompaction of the ventricular myocardium. *Circulation* 2004;**109**:2965–2971.
11. Bennett CE, Freudenberger R. The current approach to diagnosis and management of left ventricular noncompaction cardiomyopathy: review of the literature. *Cardiol Res Pract* 2016;**51**:72–308.
12. 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**:43–47.
13. Ohlow MA. Congenital left ventricular aneurysms and diverticula: Definition, pathophysiology, clinical relevance and treatment. *Cardiology* 2006;**106**:63–67.
14. Tullu MS, Vaideeswar P, Deshmukh CT. Congenital left ventricular diverticula. *Int J Cardiol* 2000;**73**:293–295.
15. Bisoyi S, Dash AK, Nayak D, Sahoo S, Mohapatra R. Left ventricular pseudoaneurysm versus aneurysm a diagnosis dilemma. *Ann Card Anaesth* 2016;**19**:169–172.
16. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol* 1998;**32**:557–561.