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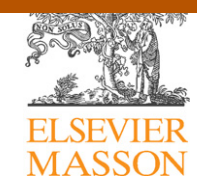

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REVIEW

Patterns of myocardial late enhancement: Typical and atypical features

Rehaussement tardif en IRM cardiaque : aspects typiques et atypiques

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KEYWORDS
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Summary Myocardial late enhancement, an imaging technique acquired after gadolinium administration, has become an integral part of cardiovascular magnetic resonance imaging over the past decade. Initially principally utilized for imaging myocardial infarction, more recently it has also become an invaluable tool for identifying myocardial scarring in other cardiomyopathic processes. Our experience using this technique has led us to identify several manifestations of late gadolinium enhancement imaging that can confound interpretation of pathology and potentially lead to misinterpretation and subsequently misdiagnosis for the patient. The purpose of this article is to review and illustrate typical and atypical myocardial late enhancement in the most common myocardial diseases seen in routine clinical practice.

MOTS CLÉS
Imagerie cardiaque par résonance magnétique ;
Rehaussement tardif

Résumé Les séquences de rehaussement tardifs, acquis après injection de produit de contraste sont devenues une part clé de l'IRM cardiaque. Initialement utilisée dans l'imagerie de l'infarctus du myocarde, ces séquences sont devenues un outil indispensable d'identification de cicatrice myocardique dans un grand nombre de cardiomyopathies. Notre expérience dans ce domaine nous a conduit à identifier des rehaussements tardifs typiques mais également

Abbreviations: CMR, cardiovascular magnetic resonance; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; LGE, late gadolinium enhancement; MI, myocardial infarction; MVO, microvascular obstruction.

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atypiques pouvant potentiellement conduire à des diagnostics erronés. Le but de cette revue est d'identifier et d'illustrer des rehaussements tardifs typique et atypiques dans quatre principales cardiomypathies (infarctus aigu du myocarde, myocardite, cardiopathie dilatée et cardiopathie hypertrophique).

Technical aspects of late gadolinium enhancement

A myocardial LGE study is performed 10 to 20 minutes after injection of an extracellular contrast agent that distributes in extracellular water but cannot cross the intact myocyte cell membrane. LGE imaging utilizes inversion-recovery gradient echo sequences with the inversion time set to null viable myocardium.

The technique for LGE imaging has been shown to be effective in identifying the presence and extent of myocardial scarring. Compared with normal myocardium, the wash out of gadolinium in a myocardial scar is delayed. In the acute stage (i.e. necrosis), leaky cell membranes allow gadolinium to enter the cells, thereby increasing the volume of distribution of gadolinium, resulting in a bright signal intensity of suitable inversion-recovery ('LGE') images [1]. In the chronic setting, fibrous tissue replaces necrotic tissue and is associated with a significant expansion of the interstitial space and a subsequent increase in the volume of distribution of gadolinium [1]. The enhancement is not disease specific and can be caused by ischaemic necrosis, inflammatory or infectious pathology, ischaemic and non-ischaemic scars and tumorous lesions.

Different patterns of LGE have been described, primarily segmented into ischaemic and non-ischaemic patterns. Ischaemic necrosis expands from the subendocardium to the epicardium with increasing coronary occlusion time [2]. Infarct-related areas of bright signal in LGE images are typically subendocardial with an increasing degree of transmural extension, depending on the extent of the infarct. The circumferential extent is related to the size of the perfusion bed and the location of the coronary artery occlusion. Conversely, in the non-ischaemic pattern, the subendocardium typically is spared, with the high-signal areas localized in the midwall and subepicardium, appearing patchy or more diffuse.

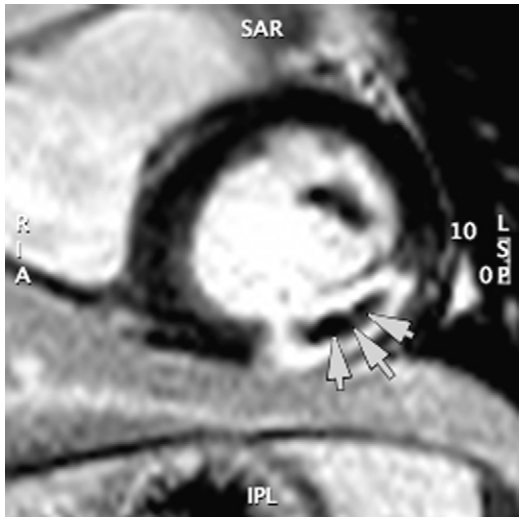

Figure 1. Typical transmural infarct with microvascular obstruction: a 37-year-old male with an acute reperfused inferolateral myocardial infarction. Mid short-axis image shows a dark zone embedded within a transmural region of late enhancement presenting microvascular obstruction in an infarct (arrows).

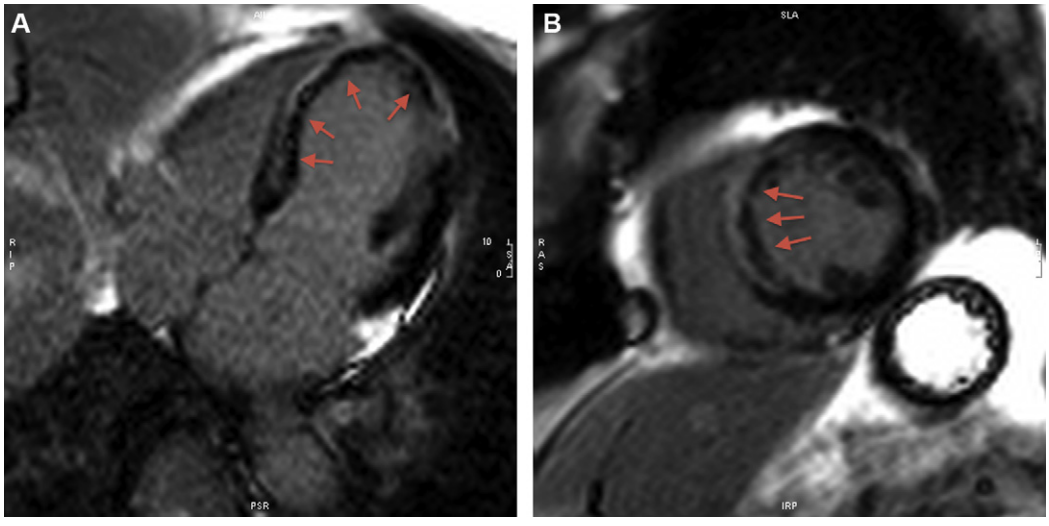

Figure 2. Atypical microvascular obstruction: an 83-year-old female with an acute anterior myocardial infarction with mid left anterior descending artery occlusion. Four-chamber long-axis (A) and mid short-axis (B) images show a laminated subendocardial dark area of microvascular obstruction overlying a near transmural anterior myocardial infarction (arrows).

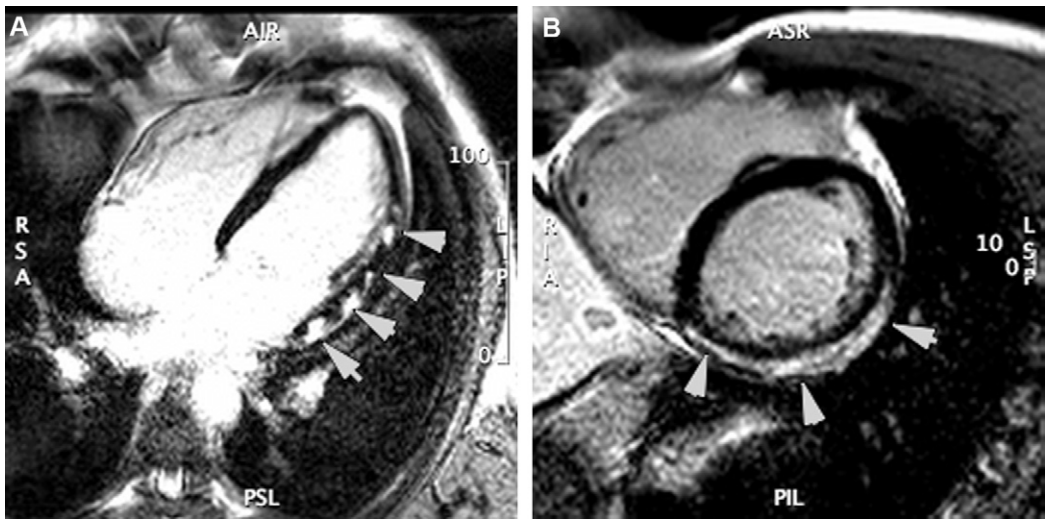

Figure 3. Typical late enhancement: 23-year-old male with myocarditis (viral prodrome, chest pain and elevated troponin). (A) Four-chamber long-axis image shows patchy midwall and subepicardial late enhancement involving the lateral wall of the left ventricle (arrows). (B) Basal short-axis image shows marked midwall to subepicardial enhancement in inferior, inferolateral and lateral walls of the left ventricle (arrows).

For a number of ischaemic and non-ischaemic disease processes, LGE has a well-defined typical pattern. However, LGE can appear atypical and can be challenging for the reader. The purpose of this article is to review and describe typical and atypical myocardial LGE in common myocardial diseases.

Clinical setting of acute myocardial infarction

LGE imaging facilitates establishing the diagnosis and quantitative assessment of MI. Hyperintense areas of LGE accurately depict the location and extent of irreversibly injured myocardium. Typically, LGE in acute MI almost always involves the subendocardial layer, with increasing degrees of transmurality depending on the total occlusion time, while the subendocardial extent of infarct depends on the localization of the occlusion (proximal versus distal) and the size of the perfusion bed dependent on the occluded coronary artery. An acute reperfused MI can be associated with MVO [3]. The presence and absolute amount of MVO are associated with adverse left ventricular remodelling and prognosis [4]. In LGE and other postcontrast images, MVO typically appears as a low-signal area within the bright infarcted zone, lacking significant uptake of gadolinium unlike the enhanced surrounding infarct tissue (Fig. 1). Atypically, MVO may involve the subendocardium predominantly, which can be challenging for the clinician, mimicking a 'normal myocardium' and suggesting a non-ischaemic pat-

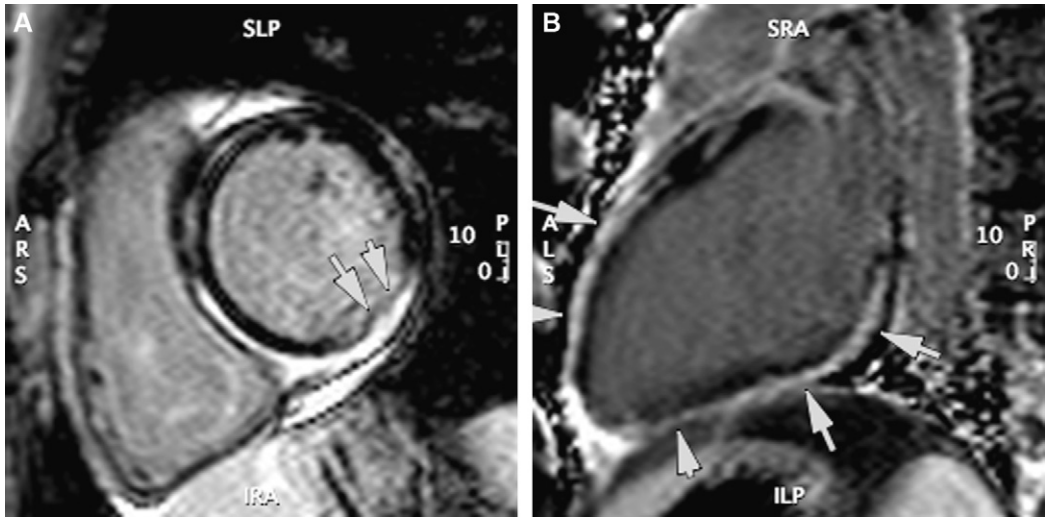

Figure 4. Atypical late enhancement: 36-year—old male with myocarditis. Mid short-axis image shows diffuse and heterogeneous myocardial involvement: subepicardial late enhancement on the right ventricular side, anteroseptal and anterior walls; focally transmural in the lateral wall (arrows).

tern or a left ventricular thrombus. Careful examination of short-axis and long-axis views enables differentiation between thrombus, normal myocardium and MVO (Fig. 2).

In the clinical setting of acute MI, CMR imaging is indicated for accurate assessment of right and left ventricular function, identifying the presence of thrombus, assessing the amount of salvaged myocardium and guiding prognosis by the presence of MVO.

Clinical setting of acute myocarditis

Acute myocarditis is an inflammatory disease of the myocardium associated with oedema, cellular infiltration and myocyte necrosis [5]. The clinical manifestations of myocarditis vary and are frequently insidious and non-specific. CMR is the non-invasive diagnostic tool of choice for assessing myocarditis and is recommended in symptomatic patients with suspected myocarditis [6]. Recently, standard diagnostic CMR criteria for myocarditis have been proposed based on oedema, hyperaemia/capillary leakage and irreversible injury (necrosis/fibrosis) detected in T2-weighted and T1-weighted images before and after contrast administration [6]. If two or more of the three criteria are positive, the diagnostic accuracy is 78%, while LGE imaging alone has a diagnostic accuracy of 68% with limited sensitivity.

In acute myocarditis, areas of LGE represent irreversibly injured myocardial tissue related to increased volume of distribution as a result of myocardial necrosis [7]. Typically, LGE is located in the lateral, inferolateral or inferior wall with a midwall to subepicardial distribution; in young male patients a 'pearl necklace' is observed frequently (Fig. 3). These regional distribution patterns are distinct from the more uniform patterns found in MI, which consistently includes the subendocardium. Importantly, however, atypical cases of myocarditis may present with transmural (Fig. 4) or diffuse enhancement (Fig. 5).

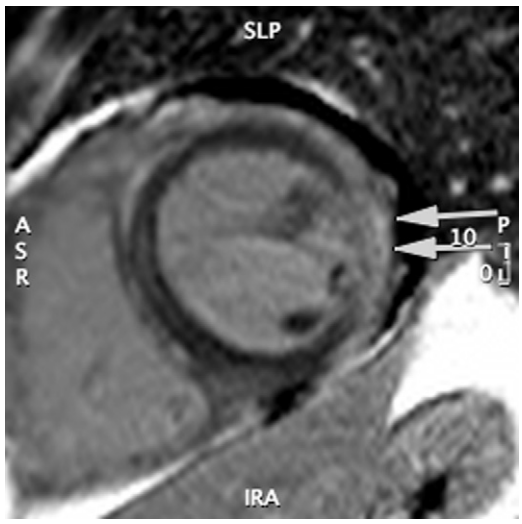

Figure 5. (A) Mid short-axis image shows extensive subepicardial to midwall late enhancement in the inferoseptal, anteroseptal and inferolateral walls of the left ventricle, with a thin subendocardial black line that represents the spared subendocardium (arrows). (B) Two-chamber long-axis image shows diffuse subepicardial late enhancement in the anterior and inferior walls of the left ventricle (arrows).

In the clinical setting of acute myocarditis, CMR represents the non-invasive tool of choice; it allows for the assessment of the activity of inflammatory changes, including myocardial oedema, hyperaemia and degree of irreversible injury. CMR is able to differentiate acute inflammation from chronic forms. The prognostic significance of CMR criteria for myocarditis is currently not known.

Clinical setting of dilated cardiomyopathy

DCM is characterized by enlargement of the left ventricle or both ventricles, associated with globally impaired systolic

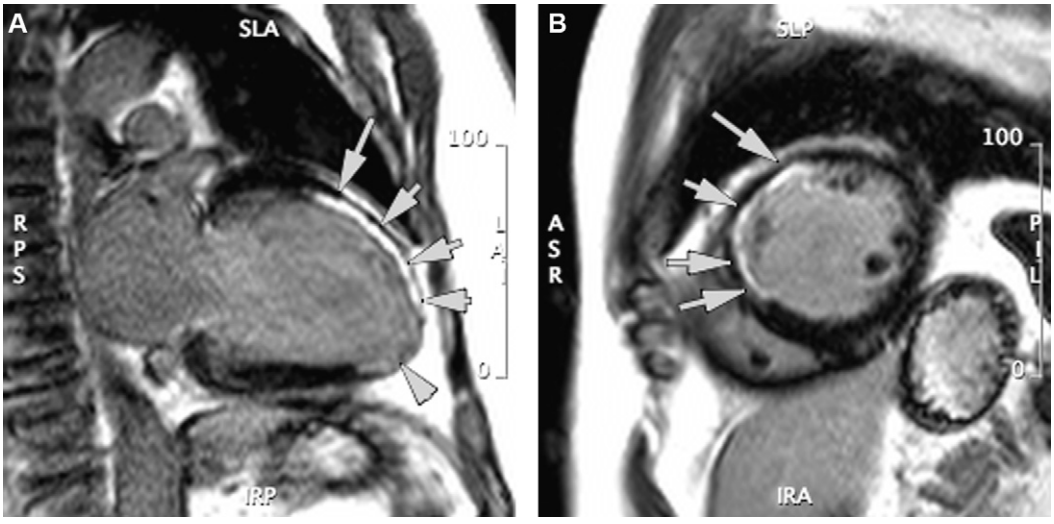

Figure 6. Late enhancement in a 58-year-old man with severe ischaemic dilated cardiomyopathy. Two-chamber long-axis (A) and mid short-axis (B) images show near transmural late enhancement in the mid-to-apical anterior and anteroseptal segments (arrows), indicating irreversible damage in the left anterior descending territory.

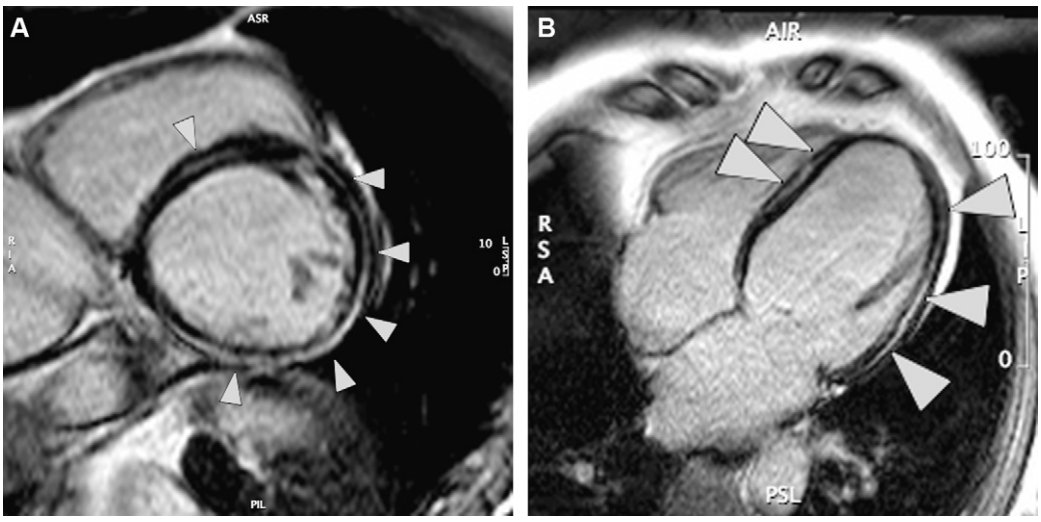

Figure 7. Diffuse late enhancement in a 46-year-old man with dilated cardiomyopathy. Mid short-axis (A) and four-chamber long-axis (B) images show diffuse midwall late enhancement (arrows) within the left ventricle, consistent with non-ischaemic cardiomyopathy.

function. DCM may be caused by ischaemic or non-ischaemic aetiologies, including myocarditis, infiltrative disease, cytotoxicity, metabolic disease or idiopathic disease.

CMR has emerged as an important tool for the evaluation of DCM, providing accurate information about cardiac morphology, function and tissue characterization [8,9]. LGE images may show hyperintense areas that reflect fibrosis, which is associated with impaired prognosis [10]. The pattern of LGE can offer important information regarding the underlying aetiology of DCM. In ischaemic DCM, LGE is typically subendocardial, fitting a coronary distribution (Fig. 6). Conversely, in patients with non-ischaemic cardiomyopathy, the typical LGE pattern shows a midmyocardial, layered ('midwall sign') or subepicardial appearance, not matching a coronary perfusion bed.

LGE may even be diffuse within the myocardium (Fig. 7) or localized, predominantly in the inferoseptal wall (Fig. 8). In this latter case, LGE must be distinguished from mimickers of late enhancement, such as a prominent septal perforator (Fig. 9) or right ventricular trabeculation (Fig. 10); these may mimic septal LGE in long- and short-axis images and may lead to false positive results.

In the clinical setting of DCM, CMR is indicated for accurate assessment of ventricular function and dilatation and for guiding the aetiology of DCM according to the pattern of LGE (ischaemic or non-ischaemic).

Clinical setting of hypertrophic cardiomyopathy

HCM is a genetic cardiac disorder characterized by hypertrophy of the left ventricle in the absence of any hypertrophic stimuli, with a non-dilated left ventricle.

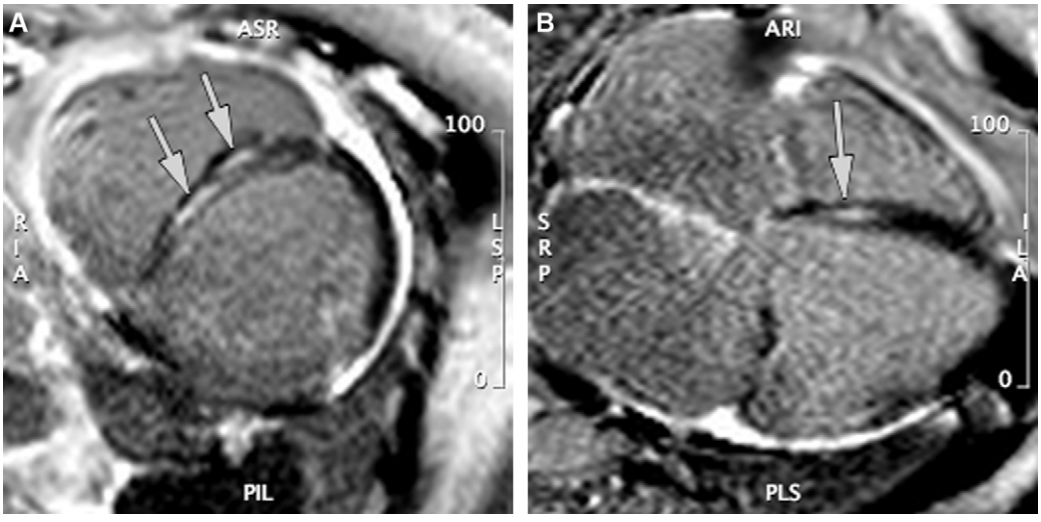

Figure 8. Late enhancement mid septum in a 54-year-old woman with dilated cardiomyopathy. Basal short-axis (A) and four-chamber long-axis (B) images show linear non-ischaemic midwall late enhancement located in the basal mid septal wall (arrows). Signal intensity of the late enhancement is brighter than the blood pool indicating that it represents true late enhancement.

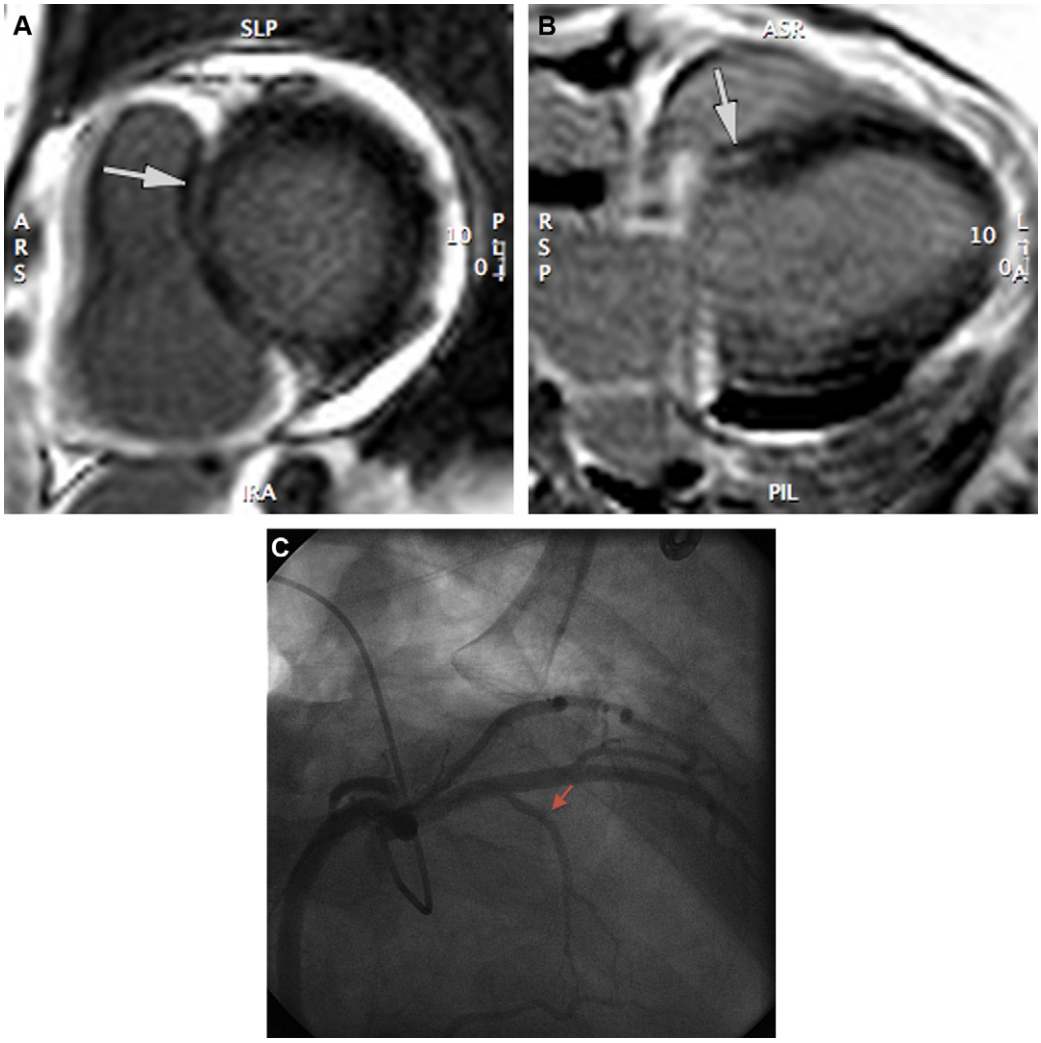

Figure 9. False positive late enhancement: septal perforator in a 64-year-old woman. Basal short-axis (A) and two-chamber long-axis (B) images show linear region of increased midwall signal intensity. This has a tapered appearance with a similar signal intensity to that of the blood pool. Overhanging muscle bundles are not identified, suggesting that this likely represents a septal perforator mimicking a midwall late enhancement in the basal septal wall. (C) Coronary angiogram reveals a dominant septal perforator (arrows).

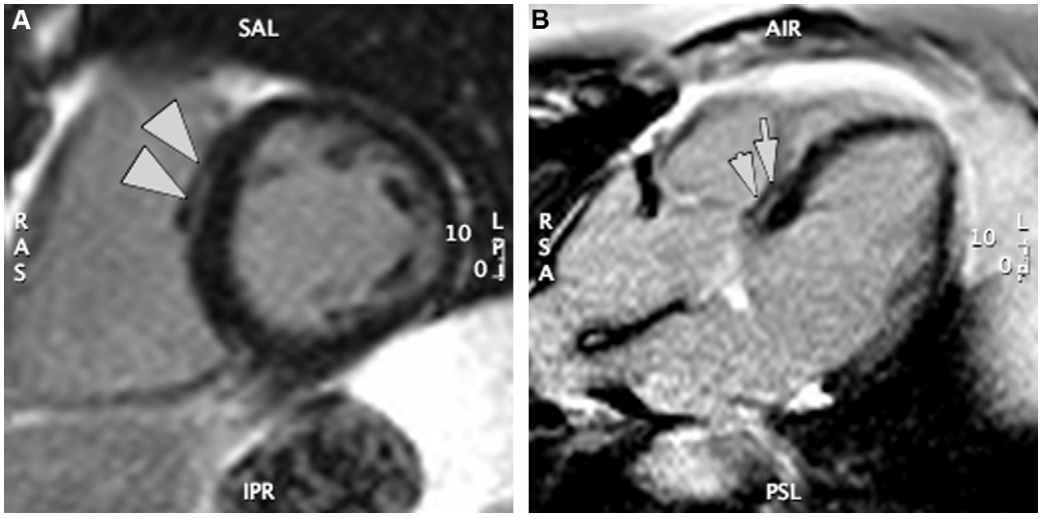

Figure 10. False positive late enhancement: right ventricular trabeculation in a 64-year-old man. Mid short-axis (A) and four-chamber long-axis (B) images show a linear region of increased signal intensity in the basal septal wall (arrows). There is myocardial discontinuity along the inferior extent of the mid septum, which confirms that the ridge of myocardium infarct represents a right ventricular muscle bundle, with contrast extending underneath the muscle rather than a true midwall focus of late enhancement.

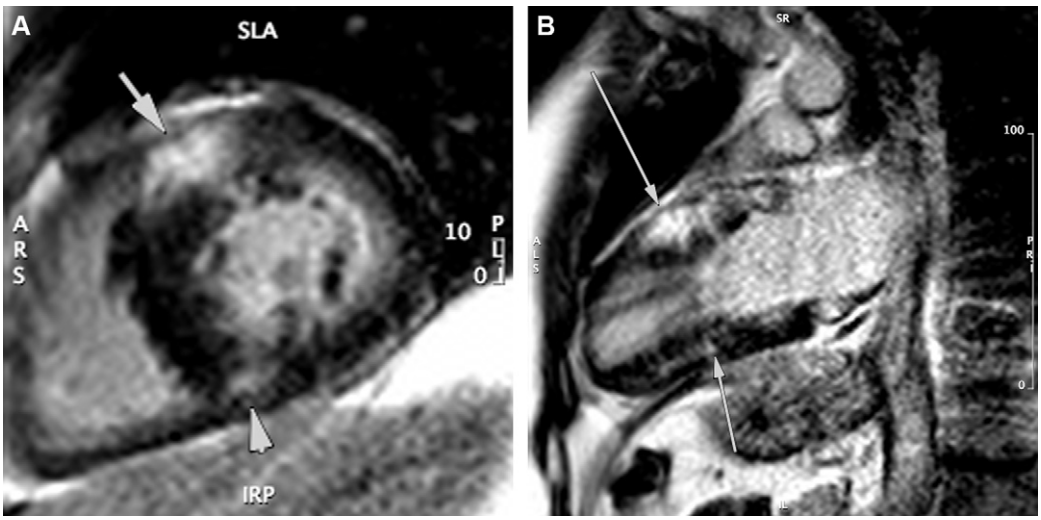

Figure 11. Typical focal late enhancement: a 52-year-old male with asymmetrical hypertrophic cardiomyopathy (basal to mid inferoseptal). (A) Mid short-axis image shows focal patchy late enhancement mainly confined to the superior and inferior right ventricular insertion points (arrows). Subepicardium and midwall myocardium are maximally involved at the superior insertion point. (B) Two-chamber long-axis image shows subepicardial to midwall late enhancement in the basal to mid anterior and inferior walls (arrows).

Transthoracic echocardiography is the first-line imaging modality for diagnosing HCM. However, CMR imaging has evolved into a highly useful technique, providing a complete description of abnormalities in HCM, including morphology, mass, function, haemodynamic obstruction and tissue characterization [11,12]. LGE imaging provides evidence for myocardial fibrosis in HCM being an independent predictor of cardiovascular death and arrhythmia [13].

The typical pattern of LGE includes focal areas of bright signal in hypertrophied regions or small patches close to the superior and inferior right ventricular insertion points (Fig. 11). Atypically, LGE can extend into the right ventricle (Fig. 12) or can also involve non-hypertrophied segments (Fig. 13). Myocardial fibrosis may be diffuse and more extensive, making distinction from infiltrative cardiomyopathy more difficult (Fig. 14). In the setting of apical HCM, the apical wall of the left ventricle may develop chronic ischaemia with wall thinning in the absence of epicardial coronary artery disease. In this setting, LGE can be transmural, mimicking ischaemic injury as in MI (Fig. 15).

In the clinical setting of HCM, CMR is indicated for accurate assessment of left ventricular mass and location of left ventricular hypertrophy as well as for guiding prognosis by the degree of myocardial fibrosis. CMR is probably the examination of choice for the detection of the apical forms of HCM and for evaluating the involvement of the right ventricle. Furthermore, CMR can be used for guiding biopsy and for follow-up after septal ablation.

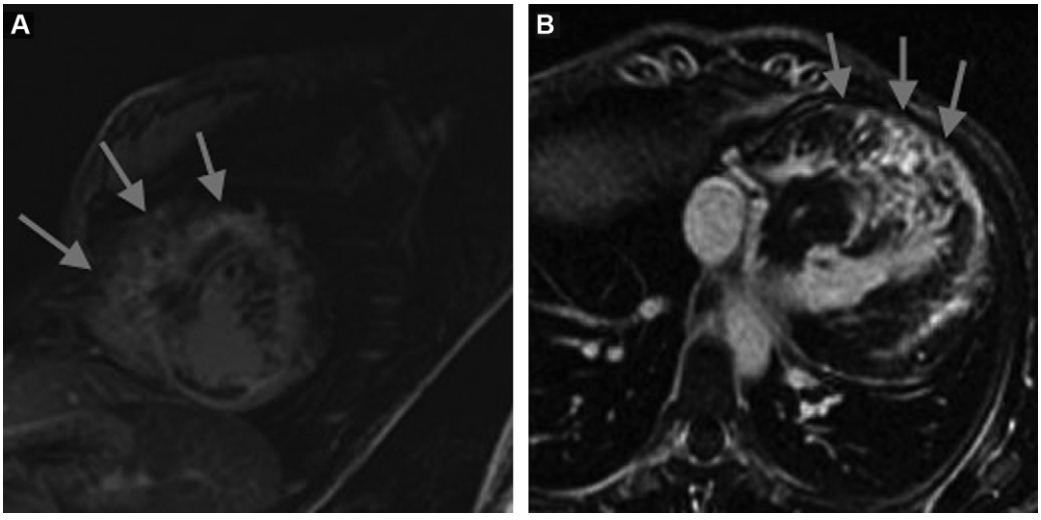

Figure 12. Typical diffuse late enhancement: a 29-year-old woman with known hypertrophic cardiomyopathy. Mid short-axis (A) and four-chamber long-axis (B) images show very extensive mid wall late enhancement in the left ventricle extended to the entire right ventricle (arrows).

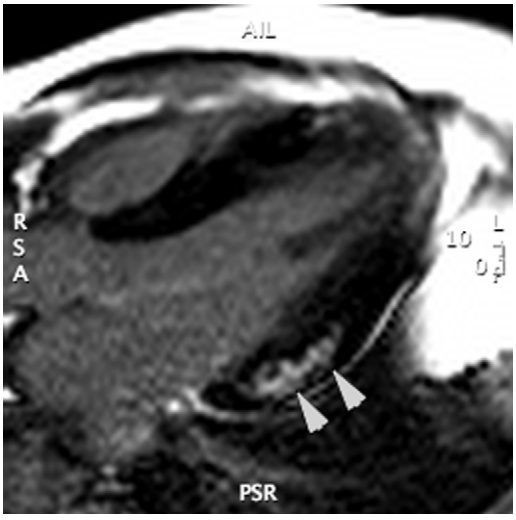

Figure 13. Typical and atypical LE in the same patient: a 20-year-old male with apical hypertrophic cardiomyopathy. Three-chamber long-axis image shows typical apical late enhancement but an atypical basal midwall late enhancement in the inferolateral wall (arrows), corresponding to a non-hypertrophied area.

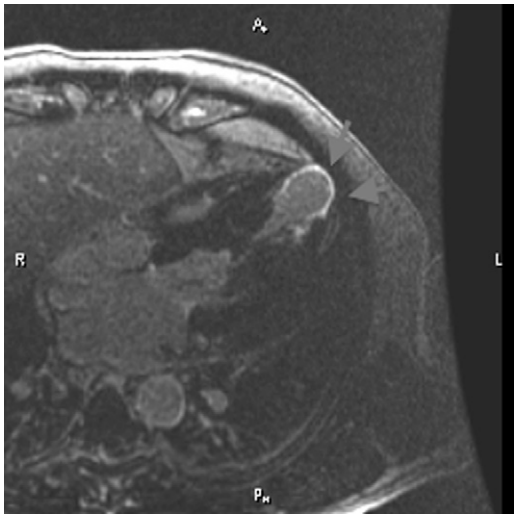

Figure 15. A 54-year-old male with known apical hypertrophic cardiomyopathy. Two-chamber long-axis image shows transmural late enhancement at the apex of the left ventricle with a similar appearance to that of an apical myocardial infarct but with normal epicardial coronary arteries. There is near obliteration of the mid cavity by myocardial hypertrophy and hypertrophic papillary muscle.

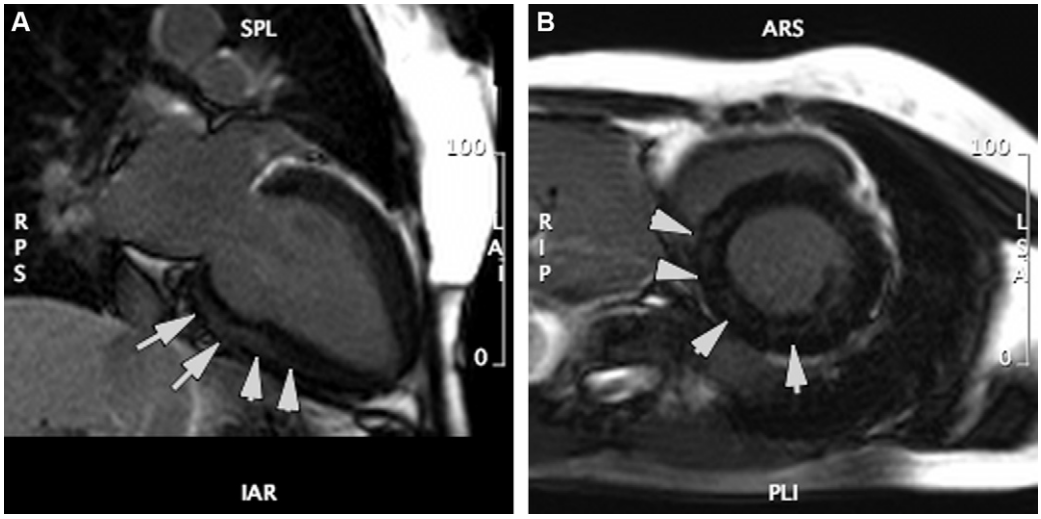

Figure 14. Atypical late enhancement: a 47-year-old female sent for myocardial biopsy for suspected infiltrative cardiomyopathy based on cardiovascular magnetic resonance imaging. Biopsy confirmed diagnosis of hypertrophic cardiomyopathy. Two-chamber long-axis (A) and mid short-axis (B) images show patchy diffuse midwall late enhancement in the inferolateral, inferior and inferoseptal walls (arrows).

Disclosure of interest

The authors have not supplied their declaration of conflict of interest.

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