

Evaluation of Cardiac Masses by CMR

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Introduction

The prevalence of primary tumors of the heart is less than 0.3%, making them a relatively rare cardiac disease compared to other cardiovascular disease entities [1].

Secondary cardiac tumors are approximately 20 times more common according to post mortem studies [2]. Secondary tumors are the result of local invasion of cancer or metastatic involvement of the myocardium and/or pericardium.

Although cardiac tumors are rare, the clinical relevance is high as primary tumors can be malignant, and therefore lethal if left untreated, but curable if excised in time.

Ventricular thrombi are an important differential diagnosis of cardiac masses. Thrombi are in most cases related to prior myocardial infarction with extensive left ventricular aneurysm formation, and carry a high risk of embolic complications. Apart from thrombi, there is a wide variety of diseases that can be confused with primary cardiac tumors such as:

- vegetations,
- atypical focal variants of hypertrophic cardiomyopathies,
- abscesses,
- anatomic variants,
- pericardial cysts, or
- juxtacardiac findings such as diaphragmatic hernia.

Cardiac tumors can be classified by histopathology (malignant, benign), location (pericardial, intracavitary, paracardial, intramyocardial), morphology (sessile, pedunculated, or by the effect on blood flow (obstructive, non-obstructive).

In pre-MRI times diagnosis of cardiac tumors was,

due to insufficient imaging techniques, mainly confined to autopsy, leaving the majority of tumors undiagnosed during life time.

With the rapid development of CMR, cardiac tumors are diagnosed more frequently.

Benign cardiac tumors

75% of all primary tumors of the heart are benign, including myxomas, rhabdomyomas, fibromas, papillary fibroelastomas, lipomas, hemangiomas and teratomas.

Myxomas account for 50% of all cardiac tumors. They are typically located within the left atrium, although left ventricular, or right atrial involvement occurs. The morphology is rather polypoid than round-shaped, and they are often pedunculated-prolapsing into the mitral valve apparatus or even the left ventricle during diastole [3].

On CMR, myxomas are diagnosed by the typical location close to the interatrial septum, high signal intensity on T2-weighted spin-echo sequences, and heterogeneous enhancement following contrast administration [4]. Myxomas can grow rather large, and may be complicated by mitral valve obstruction, endocarditis or thrombotic embolism.

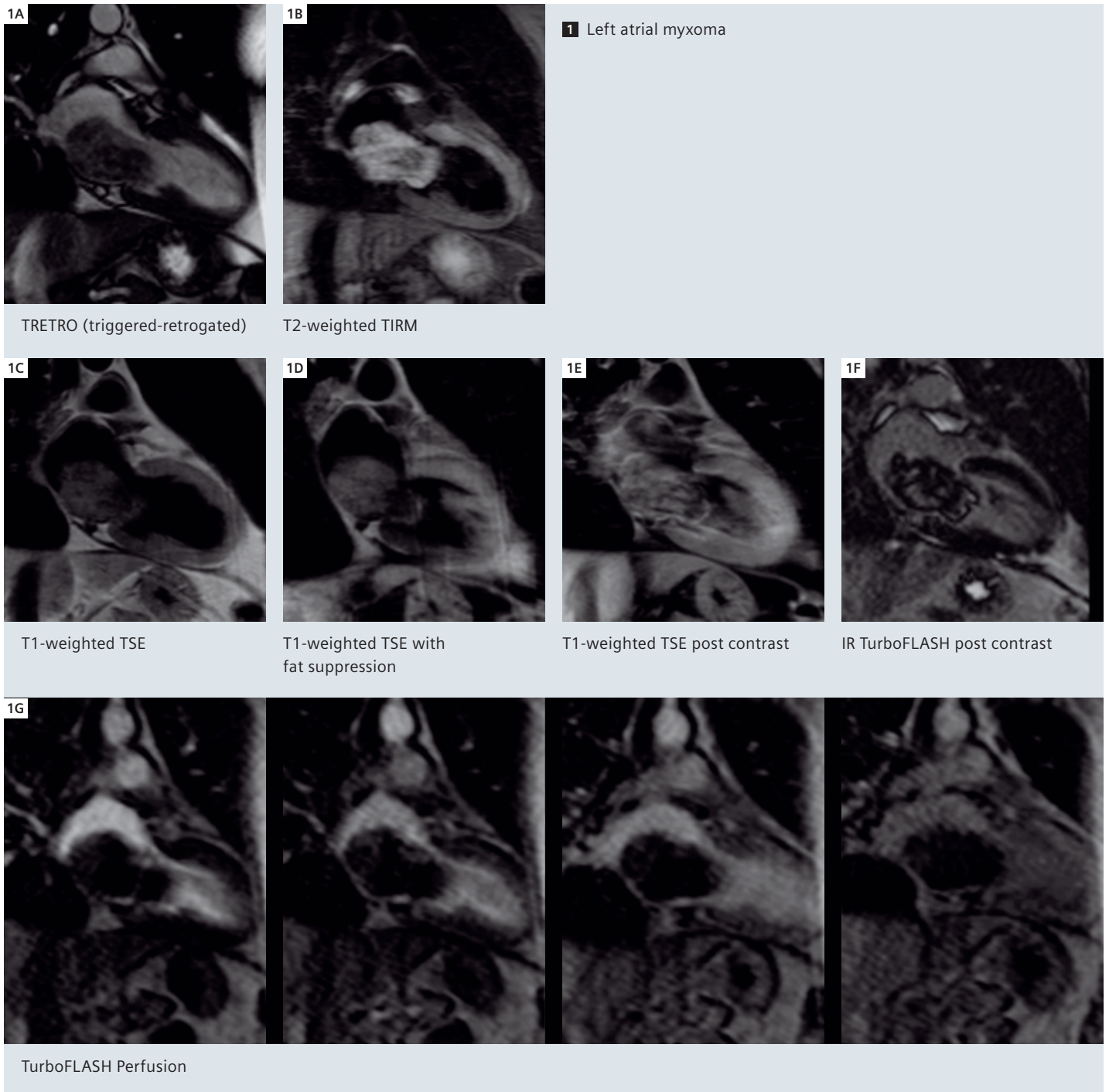
Rhabdomyomas present as several small tumors in the interventricular septum myocardium. Rhabdomyomas are most frequently found in young children.

Fibroelastomas are very small, mobile tumors that are fixed to the aortic or mitral valve leaflets.

Malignant cardiac tumors

25% of all primary cardiac tumors exhibit local tissue invasion and metastasize, and are therefore classified as malignant.

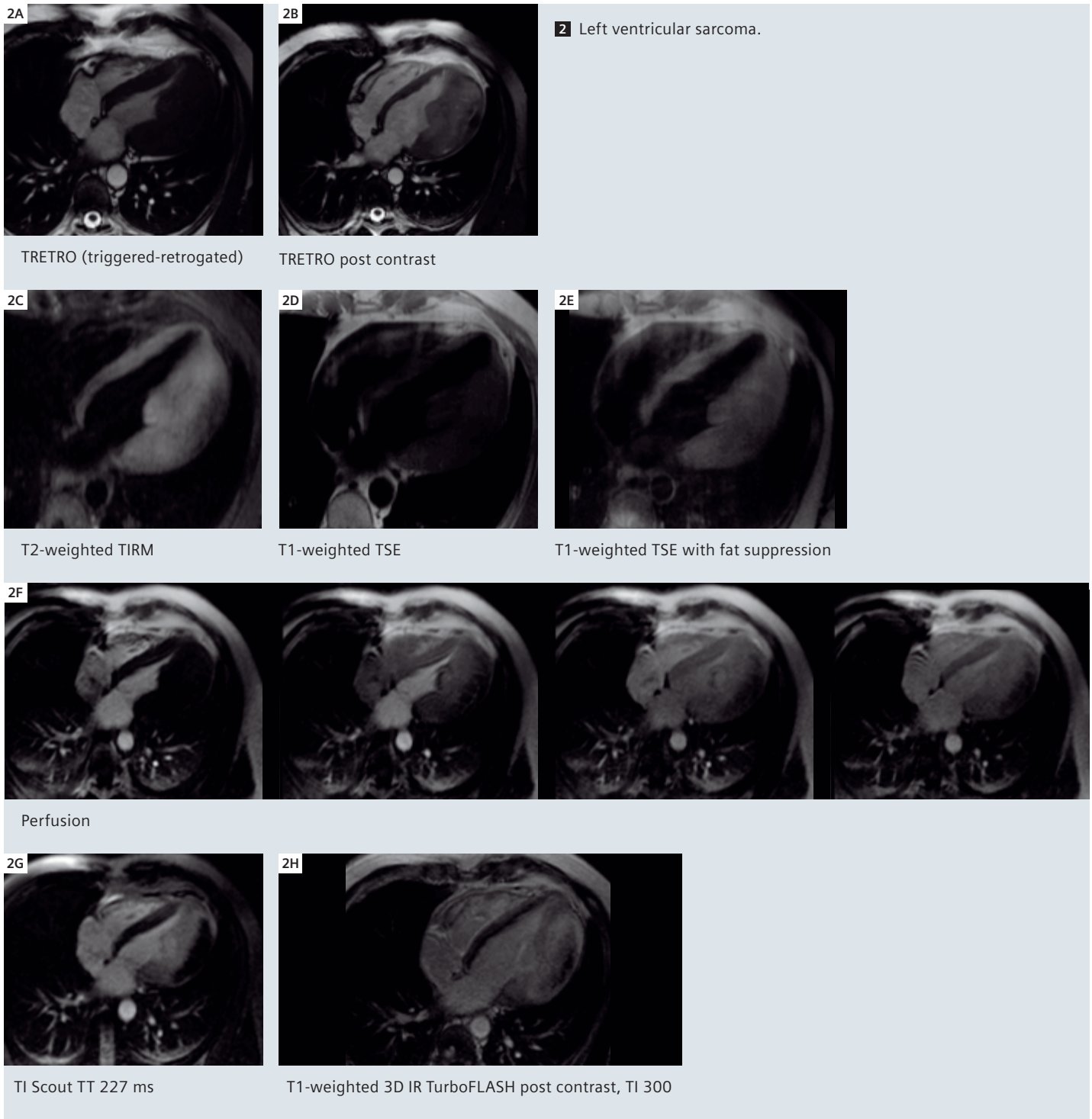
With the rapid development of CMR cardiac tumors are diagnosed more frequently. Secondary cardiac tumors are twenty times more common than primary tumors.



Sarcomas constitute the majority of malignant primary cardiac tumors (95%) including – in order of descending frequency – angiosarcomas (50%), leiomyosarcomas, rhabdomyosarcomas, and liposarcomas [5].

On CMR, malignant tumors are characterized by heterogeneous tissue characterization on T1-

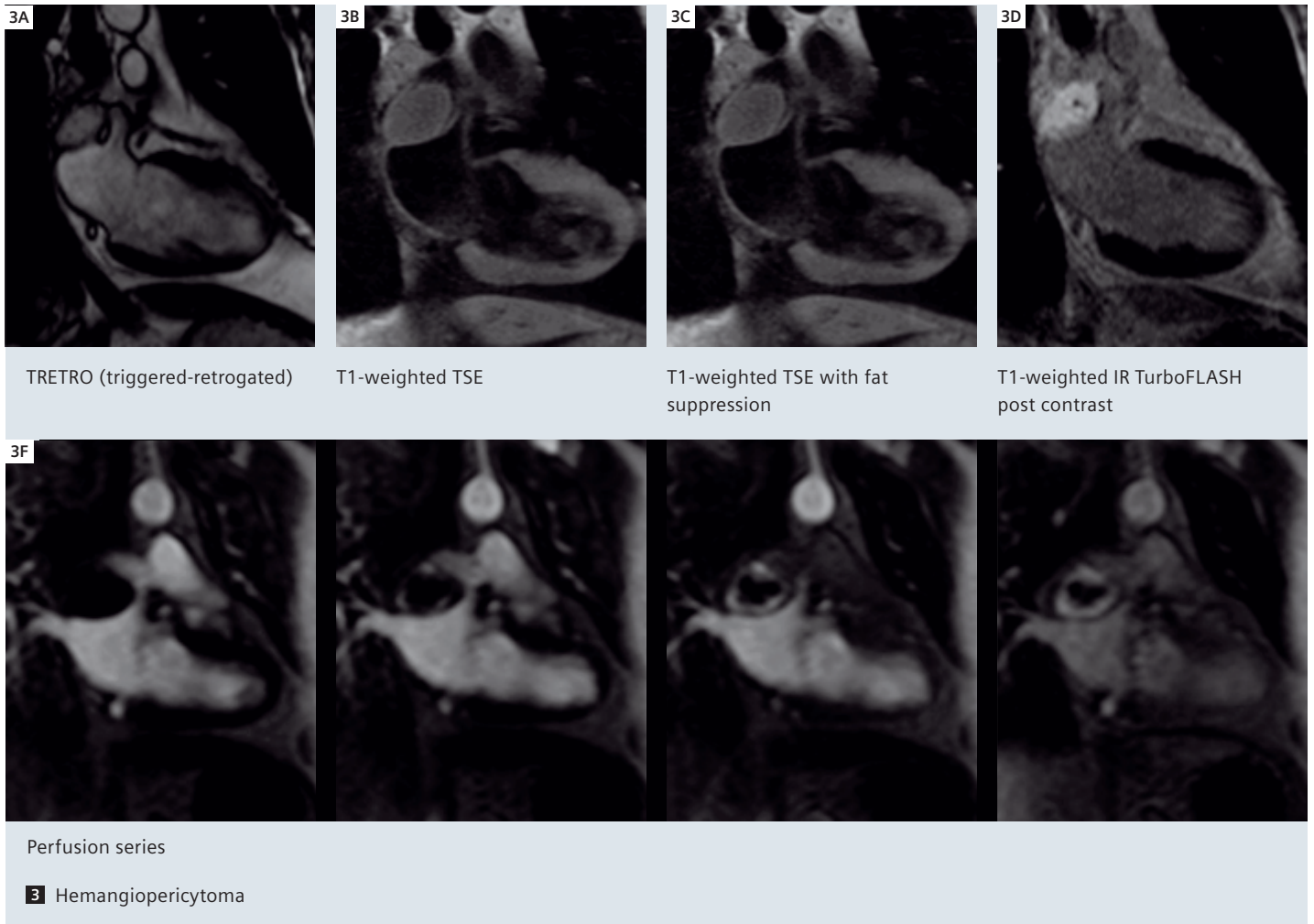
weighted, T2-weighted, and contrast-enhanced pulse sequences, a sessile morphology with broad invasive attachment to the myocardium, large size, central necrosis, and invasion into more than one cardiac chamber [6]. They are often, especially with angiosarcomas, complicated by cardiac tamponade and tend to metastasize into the spine.



CMR protocol for cardiac tumors

A typical protocol consists of anatomic images in sagittal, coronal and axial orientation (e.g. fast low-angle shot gradient echo, FLASH), cine-imaging for function (steady-state free precession, TrueFISP), tissue characterization with various

T1-weighted and T2-weighted spin-echo sequences with and without fat suppression, and finally contrast-enhanced T1-weighted imaging for vascularisation. Apart from those general recommendations CMR protocols for cardiac tumors should be adapted to the specific needs of the individual patient.

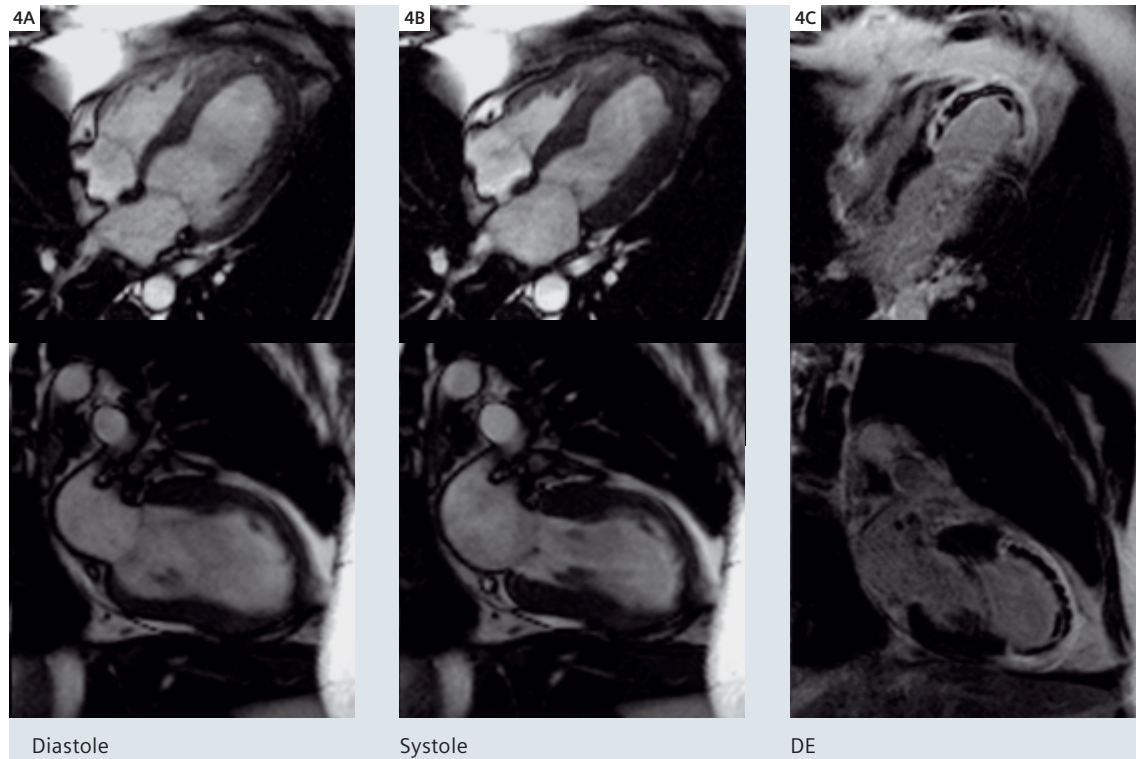


Cardiac thrombi

Left ventricular thrombus formation is a frequent complication following myocardial infarction in approximately 15% of patients [7]. Patients with left ventricular thrombi are treated with oral anti-coagulation to reduce the risk of embolization. In clinical practice LV thrombi are diagnosed by transthoracic echocardiography. The diagnosis of thrombi by echocardiography, however, is challenging due to insufficient acoustic windows, problematic apical segment imaging, and reduced endocardial border delineation. With contrast-enhanced CMR, LV function and LV tissue characterization are combined in one examination with a single imaging modality. Steady-state cine imaging further improves delineation of endocardial borders compared to ultrasound, allowing for a superior differentiation of thrombus and ventricular myocardium. In most cases thrombi are located at

the side of myocardial infarction, and are closely related to the extent of myocardial damage and impairment of segmental and global LV function. With the rare exception of large, old, and extensively vascularized thrombi, cardiac thrombi do not show enhancement following contrast application. On contrast-enhanced CMR using segmented gradient-echo sequences with an inversion-recovery prepulse (e.g. IR TurboFLASH) thrombi are visualized as dark areas between enhanced left ventricular cavity and infarct delayed enhancement. A 3D gradient echo sequence with a short inversion time early after contrast administration further improves cavity-to-thrombus contrast [8]. The "optimal TI" of a thrombus is different than the one for optimal myocardial suppression in delayed enhancement. Therefore, a brighter appearance of a thrombus when visualizing the myocardium for delayed enhancement imaging should not be

Contrast-enhanced CMR detects more thrombi than echocardiography in chronic myocardial infarction.



4 CMR in a patient with acute myocardial infarction. Note the black (nulled) subendocardial zone surrounded by delayed enhancement. This corresponds to a no-reflow zone. Additionally, a small apical thrombus can be seen in DE imaging.

In a pulmonary embolism protocol, MRA of the pulmonary arteries can be combined with cine and DE imaging of the right ventricle to detect RV thrombi.

mixed with a vascularisation of the thrombus. Not surprisingly, contrast-enhanced CMR detects more thrombi than echocardiography in chronic myocardial infarction [9]. In the setting of acute myocardial infarction, however, it can be difficult to differentiate thrombus and no-reflow zones, since microvascular obstruction (no-reflow) is predominantly located in the subendocardial layer of the left ventricular myocardium with no, or a very small rim of signal enhancement separating cavity from infarct no-reflow.

In pulmonary embolism right ventricular thrombi are frequently missed by echocardiography. In this setting MR angiography of the pulmonary arteries can be easily combined with delayed enhancement and cine imaging of the right ventricle [10].

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