

excellent tissue characterisation that it provides, is capable of identifying the different components of the necrotic scar such as fibrosis, fat or calcium. We present a 69-year-old patient with a history of a previous infarction in 1986 and an inferior infarction in 1996, sent for a cardiac magnetic resonance imaging using a 1.5 Tesla magnet system (Sonata Magnetom, Siemens; Erlangen, Germany), because of an echocardiographic persistence of severe left ventricular systolic dysfunction after complete surgical revascularisation with a triple aorto-coronary bypass. The functional studies of cine-MR confirmed the severe systolic dysfunction of the left ventricle showing a marked thinning of the anterior and inferior segments with the appearance of various hyper-intensive intramyocardial lines (Figure 1A, diastole, and B, systole, arrows) compatible with FM areas. To confirm these findings, anatomic sequences were carried out using the T1-weighted turbo spin echo images in the short axis (Figure 2A) and 2-chamber-views (Figure 3A), that show the same lines with a similar signal intensity to that found in fat and that are suppressed

Lipomatous Metaplasia. Two Chronic Infarcts in the Same Patient Detected by Cardiac Magnetic Resonance

To the Editor:

Fatty metaplasia (FM), described for the first time in 1997 by Baroldi et al¹ in explanted hearts, is a frequent histological finding in old myocardial infarctions, which, however, have hardly been mentioned in literature. The term described mature adipose tissue that infiltrates myocardium that has experienced infarction, with a substitution of collagen scar tissue by adipocytes. The cardiac magnetic resonance imaging, due to the

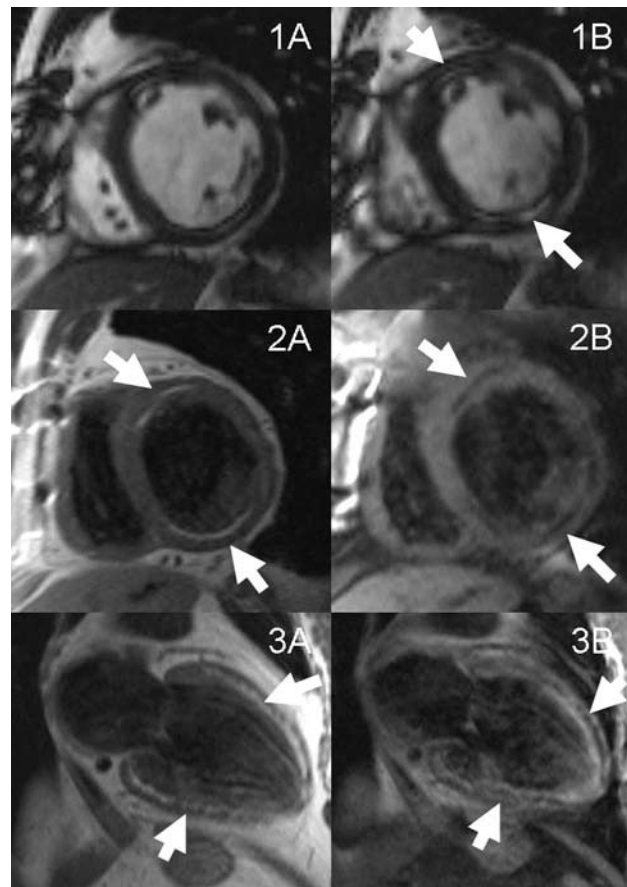


Figure 1. Film MR image in the short mid-ventricular cut (1A: diastole; 1B: systole).

Figure 2. Anatomic turbo-spin-echo image of the mid-ventricular axis cut (2A: T1-weighted T1; 2B: after fat suppression).

Figure 3. Anatomic turbo-spin-echo image of 2 chambers (3A: potentiated in T1; 3B: after fat suppression).

The arrows indicate areas of fatty metaplasia.

after the fat-saturation pulse (Figures 2B y 3B). In the gadolinium late-enhancement sequences, these zones correspond with a transmural scar in these segments, without being able to differentiate, after the contrast, the zone corresponding to the fat transformation of the area of fibrosis, as both present the same signal intensity.

These findings confirm the diagnosis of FM in both infarctions. Although FM is a frequent histological finding (68%, 24%, and 37% of the areas of myocardial scar tissue in explanted hearts that had suffered ischemic, dilated, and valve cardiomyopathies, respectively),¹ its identification *in vivo* has been limited to a few clinical cases and images that demonstrate the usefulness of the computerised tomography and cardiac magnetic resonance imaging to diagnose it.²⁻⁸ The 2 anatomopathological studies published show a high prevalence of FM in chronic infarctions, up to 84% in the most recent study.^{1,9} Its presence is associated with extensive infarctions, the age of the patient and previous coronary artery bypass surgery.⁹ The aetiology of the FM is unknown and it may be influenced by improved treatment of ischemic cardiopathies, which would explain that it was not described until 1997.⁹ Also, the presence of mature adipocytes over the chronic infarctions is an argument in favour of myocardial regeneration.¹⁰ To demonstrate the presence of FM in infarctions, beyond the mere detection of the necrotic scar using the gadolinium late-enhancement, that does not allow for the differentiation of fibrosis from fat transformation, may be important for better stratification after infarction and the development of new treatments for myocardial regeneration.

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