To the Editor:

Fatty metaplasia (FM), described for the first time in 1997 by Baroldi et al 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 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 Magneton, 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

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), 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. The 2 anatomopathological studies
published show a high prevalence of FM in chronic
infarctions, up to 84% in the most recent study. Its
presence is associated with extensive infarctions,
the age of the patient and previous coronary artery
bypass surgery. 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. Also, the presence of
mature adipocytes over the chronic infarctions is an
argument in favour of myocardial regeneration. 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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REFERENCES