

Myocardial interstitial remodelling in a patient with **cardiac amyloidosis** and **myocardial infarction**

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Male, 74 years old

Diabetes

Hypertension

Previous **anterior MI** (6 years before, PCI in LAD)

Previous **admissions for HF** (↑ troponin, ↑ BNP)

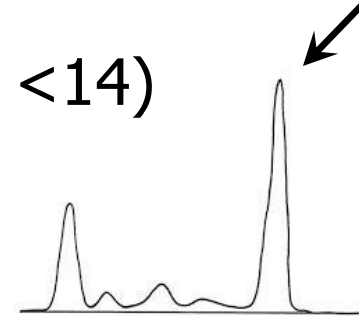
Admission for heart failure

At admission...

Creatinine **1.39** mg/dl (r.v. <1.2)

HS Troponin **304** ng/ L (r.v. <14)

Monoclonal gammopathy



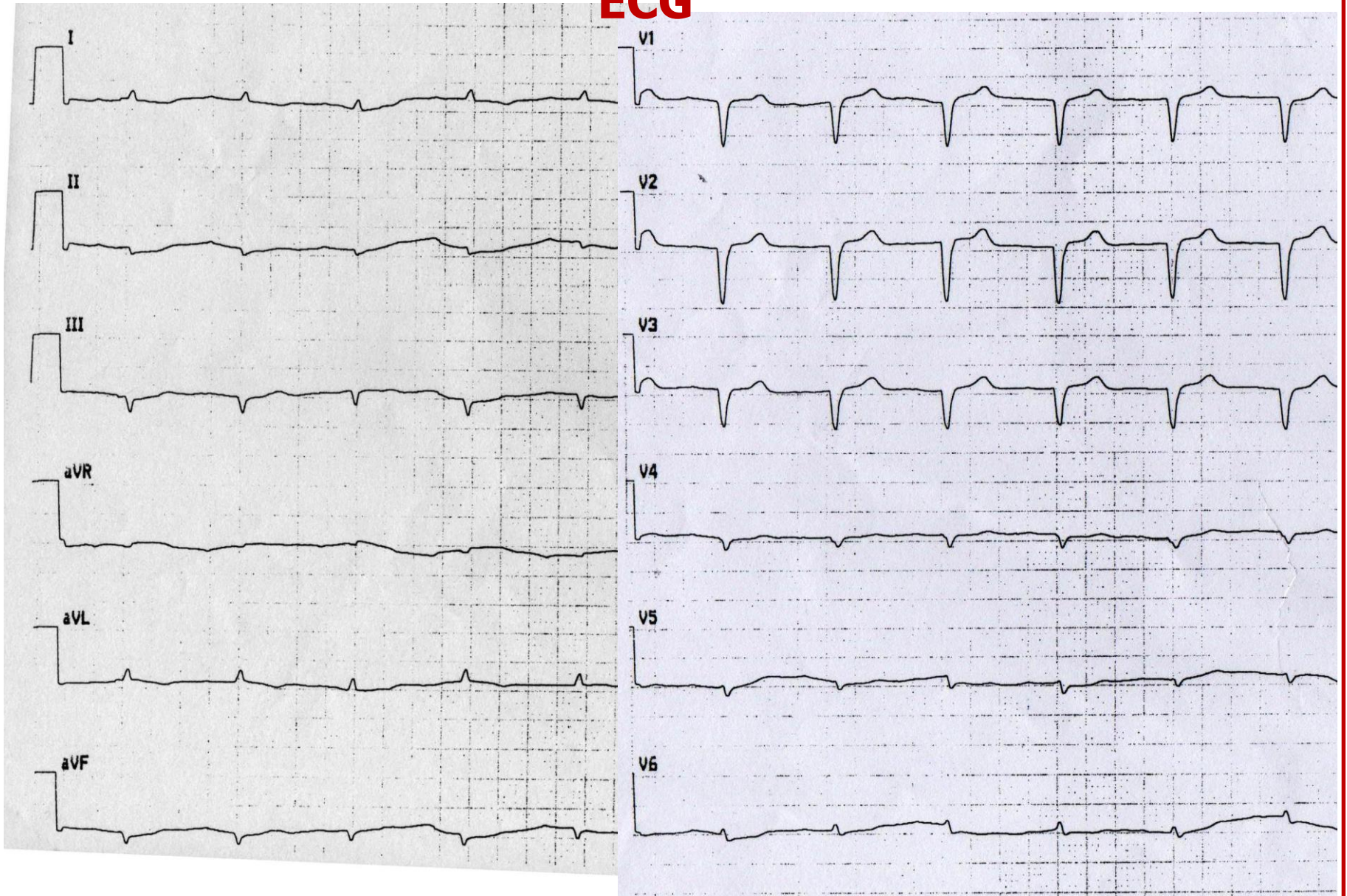
Echocardiography

LV hypertrophy (EDD 48, IVS 15, PW 13)

LV dysfunction (EF 35%; E/E' 30)

Mitral regurgitation (moderate-to-severe)

ECG



CMR

LV hypertrophy (LVMI 150 g/m², IVS 18, PW 17 mm)

LV dysfunction (EDV 93 ml/m², EF 35%)

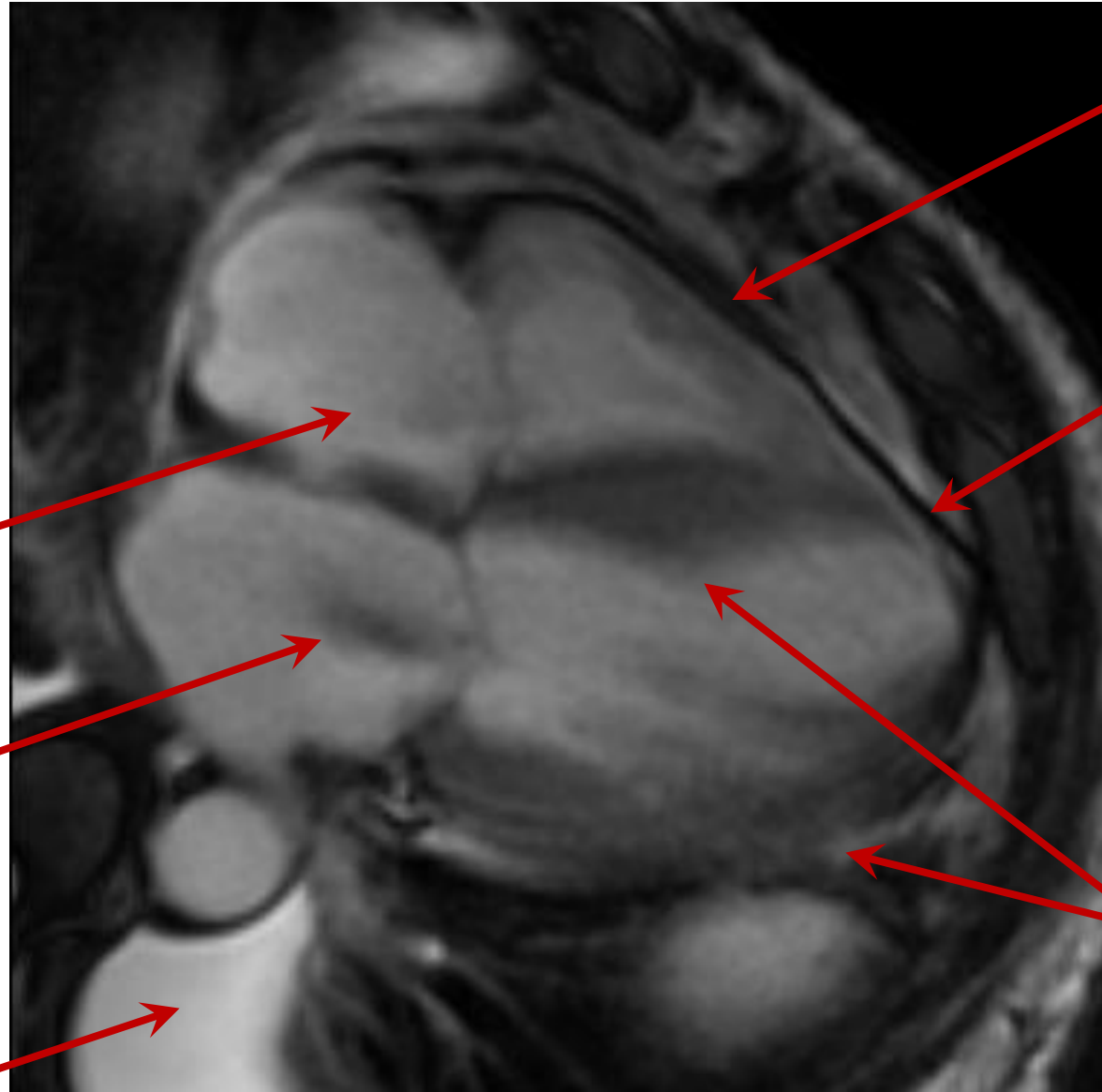
RV dysfunction (EDV 55 ml/m², EF 50%)

Mitral regurgitation

Pleural effusion

Diffuse LGE

Cine bSSFP



RV hypertrophy

Antero-septal thinning and akinesia

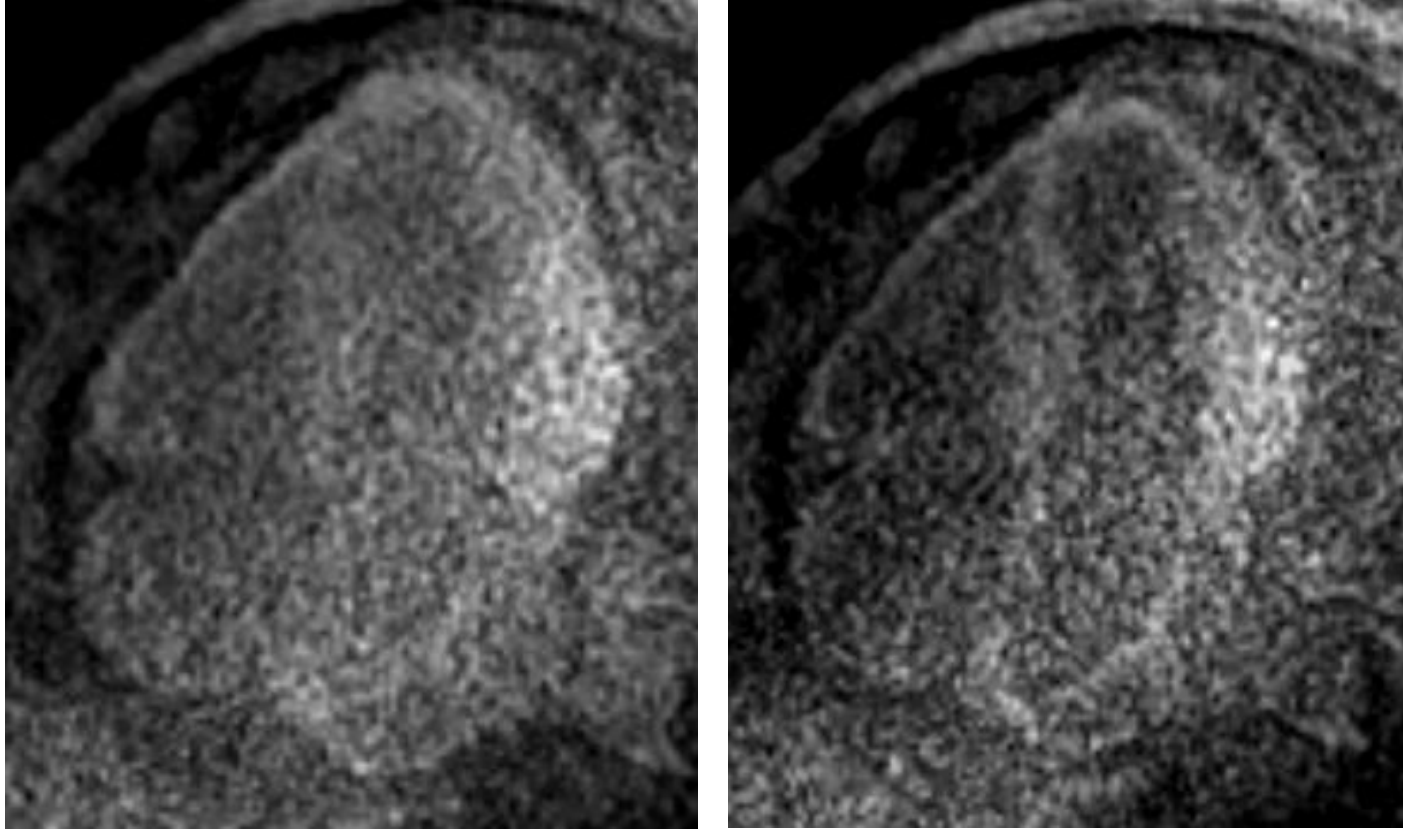
LV hypertrophy

Tricuspid regurgitation

Mitral regurgitation

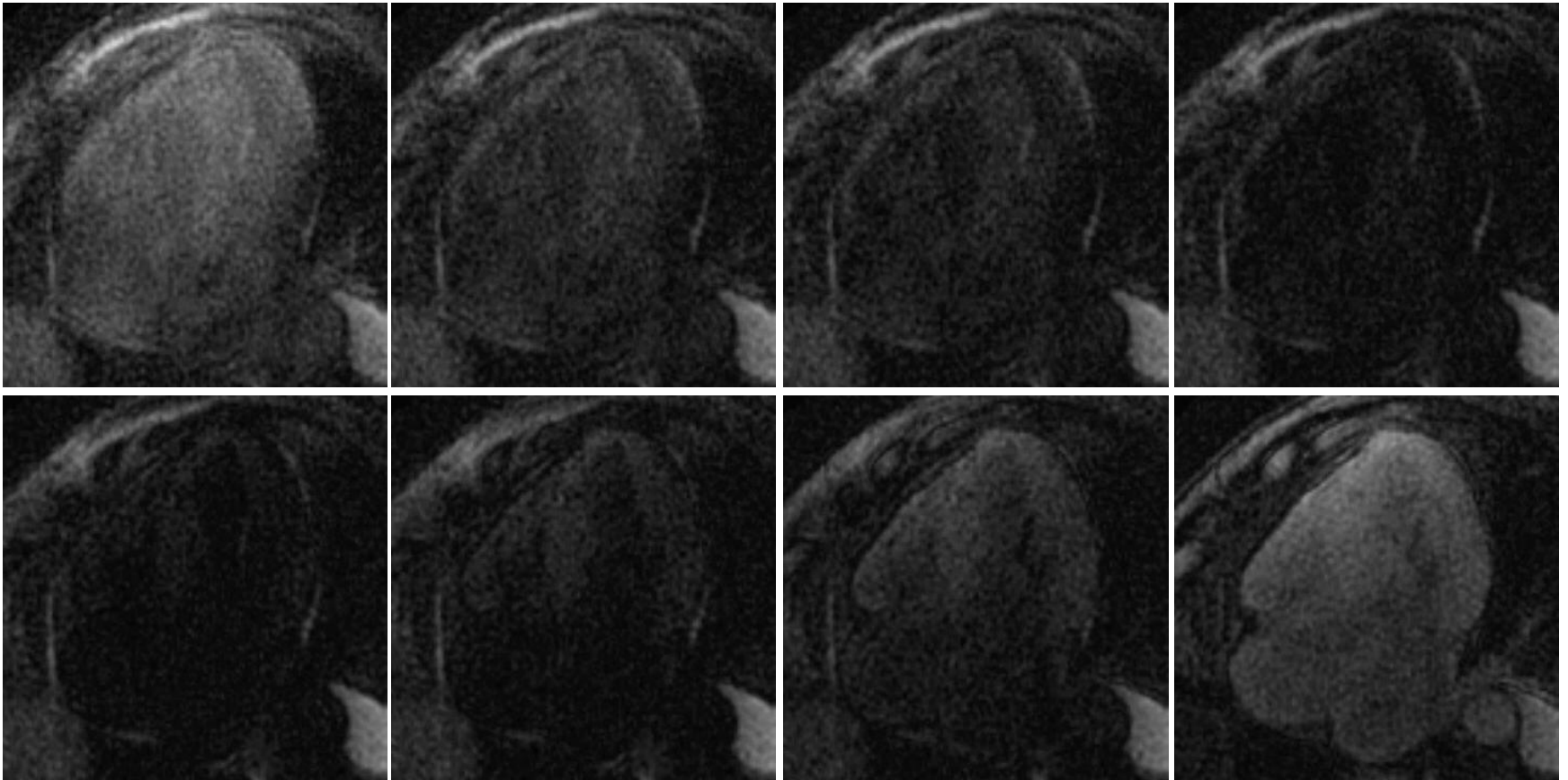
Pleural effusion

LGE



Diffuse LGE, with **difficult myocardial nulling** (variable appearance according to TI; early Gd washout...)

TI-scout



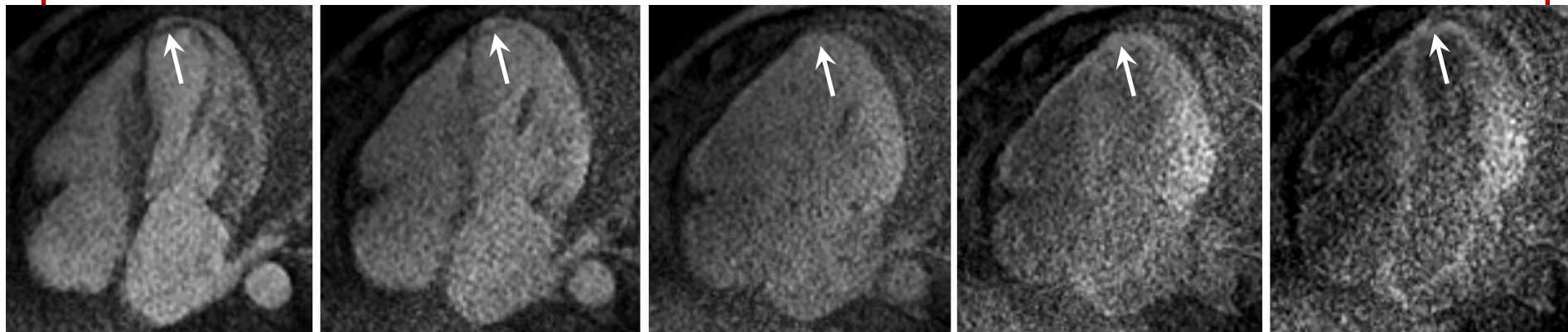
The myocardium nulls before the bloodpool, indicating diffuse interstitial expansion typical of amyloidosis (*Maceira et al, Circ 2005*)

WHAT'S INSIDE LGE?

Gadolinium kinetics from LGE

LGE at 1,2,3,4... min after contrast injection (0.2 mmol/Kg Gd)

Aquaro et al, Int J Cardiovasc Imaging 2014



1 min

3 min

5 min

7 min

10 min

Early and diffuse myocardial enhancement → cardiac amyloid

Early bloodpool darkening (rapid Gd wash-out) → systemic amyloid

Apical late enhancement (arrow) → myocardial infarction

WHAT'S INSIDE LGE?

Gadolinium kinetics from T1 mapping

-**before** contrast injection

-**1, 3, 5, 10 min after** contrast injection (0.2 mmol/Kg Gd)

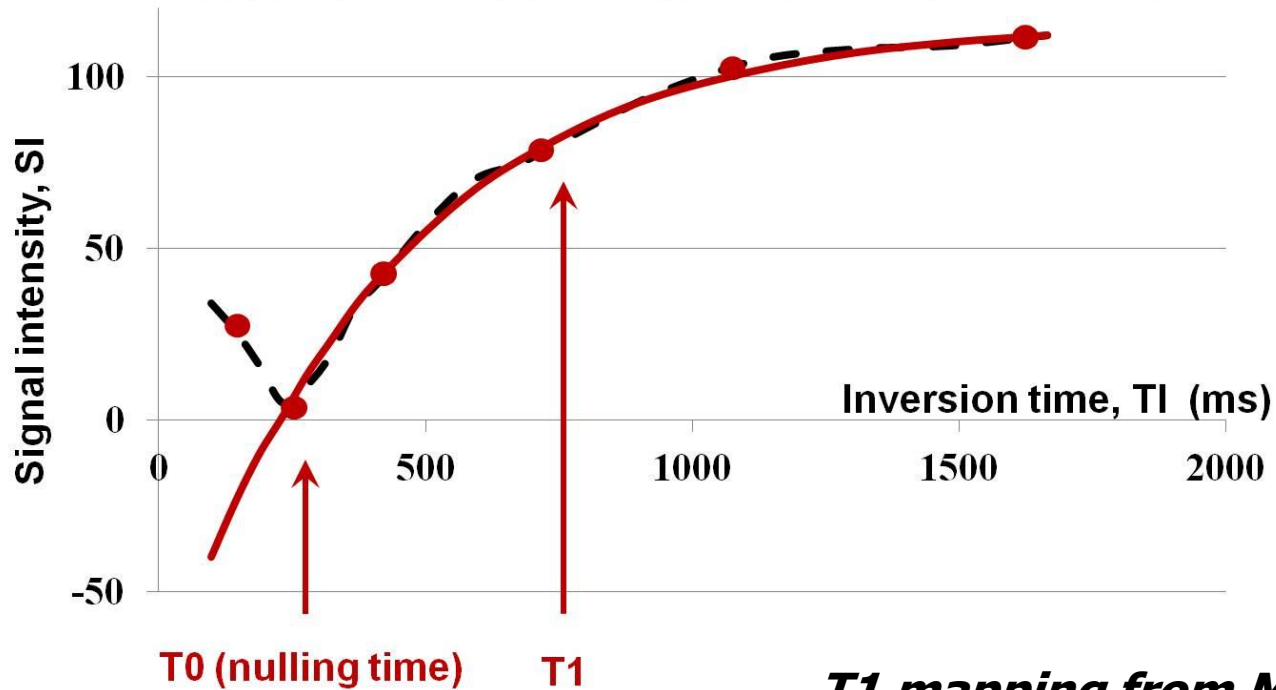
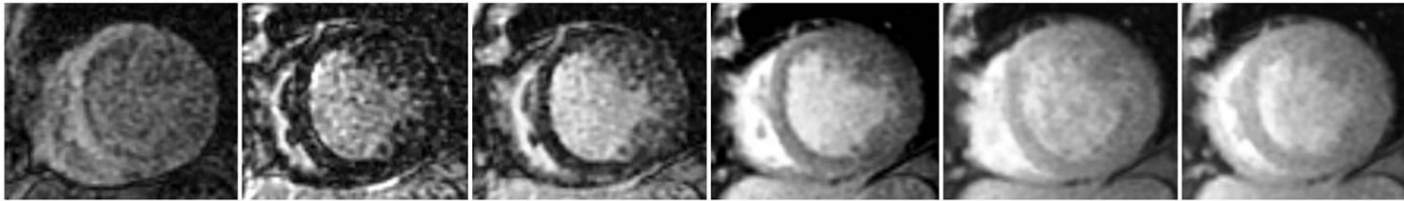
$$R1_{\text{post}} - R1_{\text{pre}} = r * [\mathbf{Gd}] \quad \text{where } R1 = 1/T1$$

$$[\mathbf{Gd}] = (R1_{\text{post}} - R1_{\text{pre}}) / r$$

If r is assumed $\approx 3.5 \text{ (mM*s)}^{-1}$ at 1.5T (*Weinmann et al, Acad Radiol 2002*) absolute Gd concentration can be calculated; otherwise, gadolinium concentration can be expressed in arbitrary units (as in this case).

WHAT'S INSIDE LGE?

Gadolinium kinetics from T1 mapping

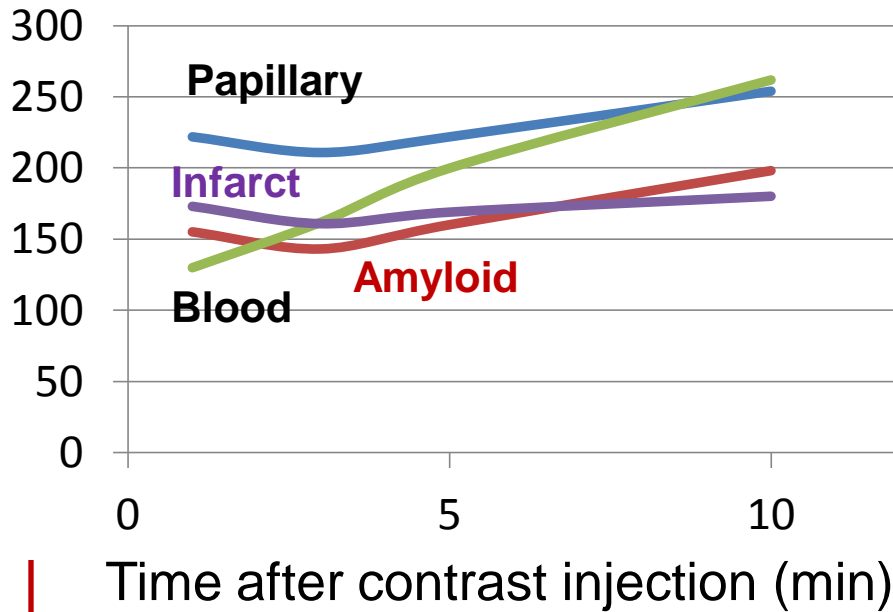


***T1 mapping from MCine-IR
(Milanesi et al, JMRI 2013)***

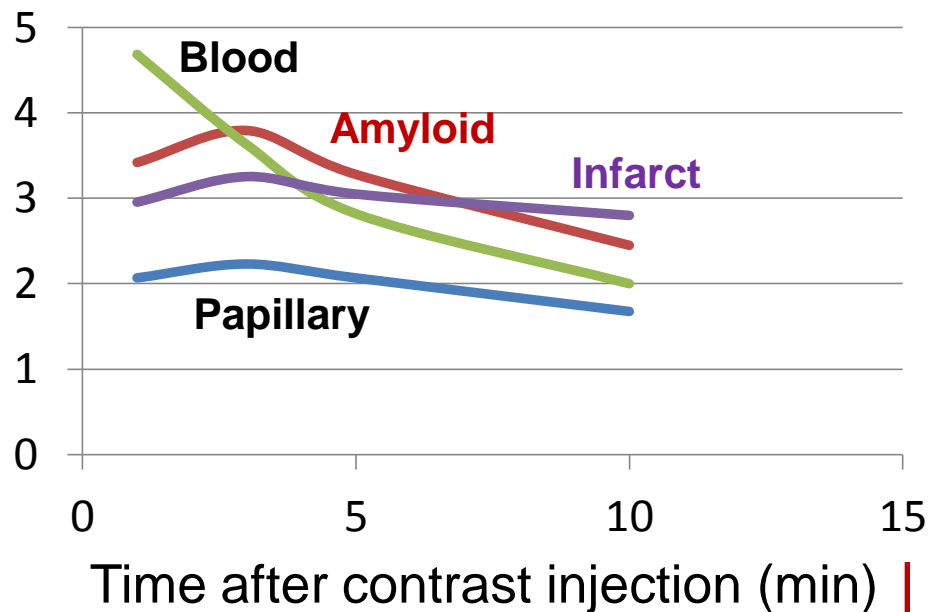
WHAT'S INSIDE LGE?

Gadolinium kinetics from T1 mapping

A. Tissue nulling times (T0) (ms)



B. Tissue Gd concentration (a.u.)



The papillary muscle displayed normal nulling times, possibly because it was spared by both ischaemic necrosis and amyloidosis (normal myocardium?)

CONCLUSIONS

Early post-contrast imaging

- First pass perfusion (ischaemia, MVO...)
- Early enhancement (thrombus, MVO...)

Intermediate post-contrast imaging (at 1,2,3... 10 min)

- Progressive enhancement
- T1 mapping

Gd kinetics

(fibrosis vs. amyloid ...)

Late post-contrast imaging

- Late gadolinium enhancement (replacement fibrosis)
- T1 mapping (interstitial fibrosis, equil/pseudoequil ECV)