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Three-compartment modeling of chronic myocardial infarction gadolinium kinetics

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Introduction

The mechanism behind late gadolinium-enhanced (LGE) chronic myocardial infarct (MI) imaging is widely thought to be an increased gadolinium (Gd) concentration due to fibrotic tissue. Several groups have employed T1 measurements to measure the partition coefficient (ratio of tissue-to-blood Gd-concentrations) using a two-compartment model. A three-compartment model yields not only information about flow of Gd from the capillaries to the intracellular space, but in the case of chronic MI to fibrotic tissue and in the case of acute MI in the myocytes themselves. With model inputs of the LV bloodpool and tissue Gd-concentrations, the model yields transfer constants (K) between the compartments, compartment fractional volumes (v) and Gd-concentrations curves for tissue blood plasma, the extravascular extracellular space (EES) and fibrotic tissue. A detailed model may not only be useful to detect and characterize MI, but non-ischemic cardiomyopathies with global or diffuse fibrosis.

Purpose

To investigate the suitability of a three-compartment pharmacokinetic model of late gadolinium-enhancement for chronic myocardial infarcts.

Methods

Twenty-five individuals underwent MR imaging at 1.5T. The infarct age ascertained from medical history was on average 11.6 ± 10.1 years. Single slice T1 measurements were performed before contrast administration and after injection of 0.2 mmol/kg of gadodiamide, approximately every two minutes using an inversion-recovery bSSFP technique. Gd-concentrations of blood, viable, and infarcted myocardium were calculated and interpolated to one minute intervals and averaged across all subjects. The blood concentration was modeled with a bi-exponential and tissue concentration with a three compartment model, including vascular (plasma), EES and fibrotic compartments (Fig 1A).

Results

Tissue gadolinium concentrations (FIG 1B) followed expected curves, with infarcted myocardium greater than viable myocardium and the infarcted myocardium curve crossing the LV-blood curve at 10 minutes. Calculated three-compartment model parameters for viable tissue were: K1 = 2.39; K2 = 5.76; ve = 0.42; K3 = 0.03; K4 = 1.24; vfibrosis = .02; R2 = 0.93 and for infarcted myocardium were: K1 = 0.93; K2 = 1.72; ve = 0.54; K3 = 0.06; K4 = 0.19; vfibrosis = 0.35; R2 = 0.92. Gd-concentration curves of the three compartments for infarcted and viable myocardium are plotted in FIG 1C and 1D. The three-compartment model yielded markedly increased fibrotic volumes and decreased transfer constants.

Conclusion

A three-compartment model is suitable for detailed modeling of chronic MI Gd-pharmacokinetics. This model provides further justification that fibrotic tissue retains the Gd-contrast agent while concentrations in the EES remain similar. Studies for individual subjects, evaluating the effects of fitting algorithms and sampling requirements are necessary.

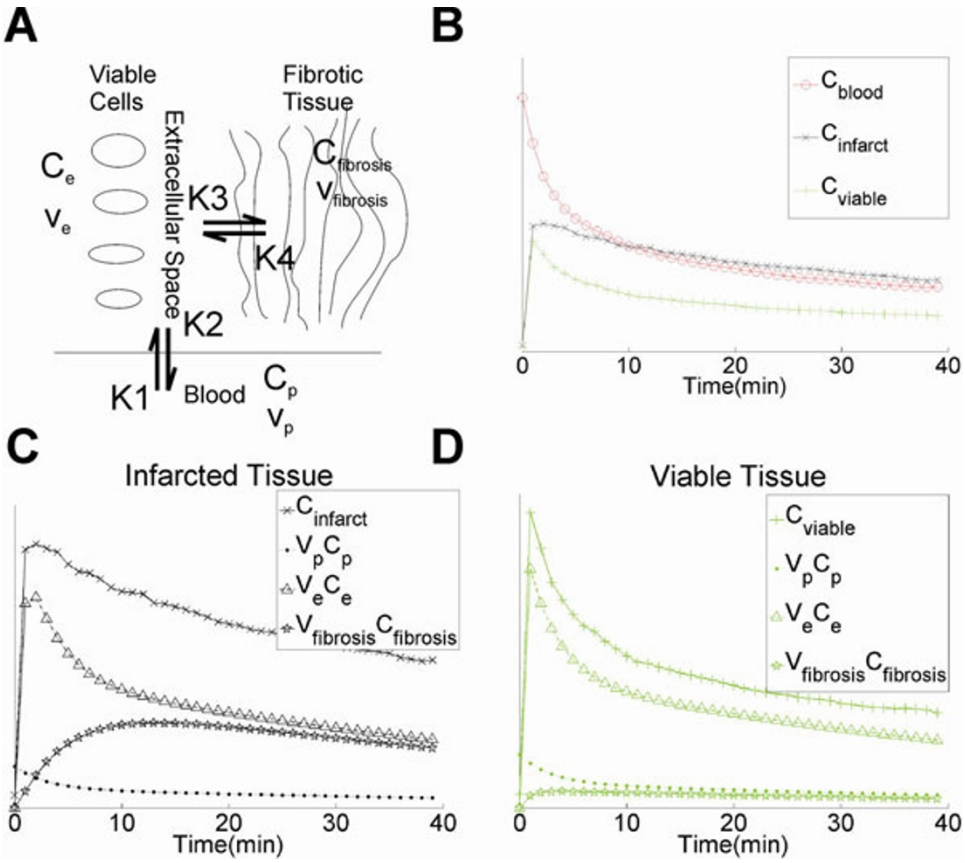

Figure 1
A) Three-compartment model; B) Measured blood and myocardial Gd-concentration; Model compartmental tissue Gd-concentrations for C) infarcted and D) viable myocardium (suript-p = tissue plasma, suript-e = extracellular space)