

# Early and Delayed Contrast Enhancement MRI of the knee

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## INTRODUCTION

The contrast distribution in articular cartilage is not only depended on fixed charge density, but also related to transport of contrast agent (*Magn Reson Med* 2010; 64:1267-1273). Articular cartilage is known to be avascular, but neo-angiogenesis has been reported in osteoarthritis (OA) [*Osteoarthritis Cartilage* 2008;16(3):279-286]. The transport of contrast agent into articular cartilage has been assumed to include both direct blood supply from the bone and permeation from synovium (synovial fluid) [*Radiology* 1997;205(2):551-558]. Dynamic Contrast Enhancement (DCE) MRI has been shown to differentiate diseased from healthy cartilage based on the level of enhancement [*Osteoarthritis and Cartilage* 2009;17:1350-1355]. We hypothesized that DCE MRI may be useful in evaluating the early enhancement in cartilage and its relationship to the blood supply from the bone. In this preliminary study, we wanted to observe any correlation between the blood flow in the bone and early contrast enhancement in the articular cartilage. Further, we wanted to evaluate any correlation between the early and delayed enhancement in articular cartilage.

## MATERIALS AND METHODS

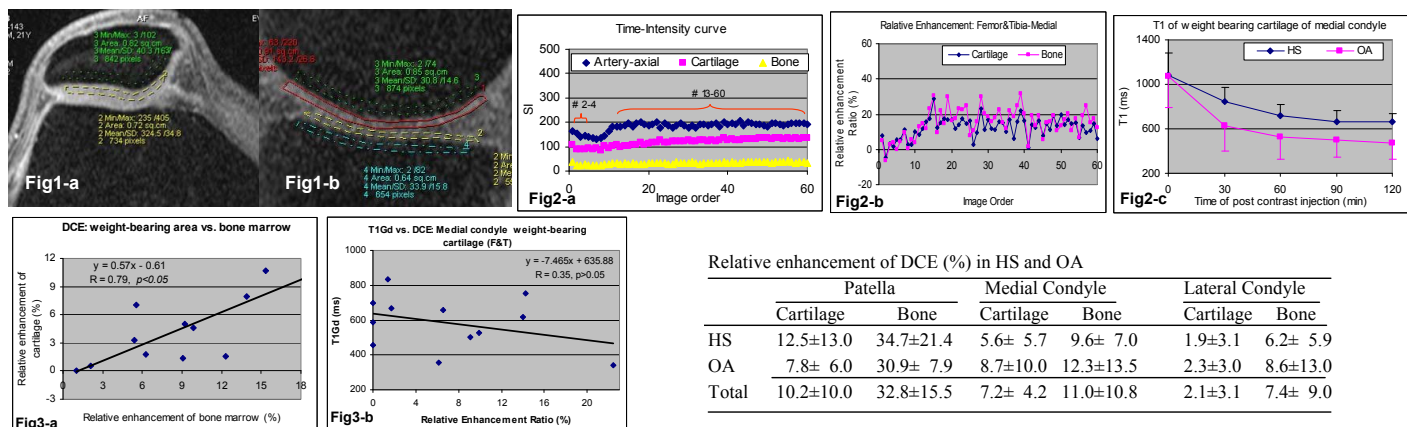
**Subjects:** Six asymptomatic subjects (HS, M=5, F=1; Age:  $29.8 \pm 10.3$ ) and 6 osteoarthritic patients with K-L scale of 2 (OA, M=3, F=3; Age:  $55.8 \pm 6.4$ ) were included in this study.

**Imaging:** Knee images were obtained on a 3.0 T MR scanner (Siemens Healthcare, Verio) using 8-channel receiver knee coil. DCE imaging was performed with a 2D flash sequence (TR/TE = 33/2.4 ms, FL=10°, FOV = 16 cm, matrix =  $256^2$ ), with temporal resolution of 5 sec over 5 min (60 phases). Three slices were obtained for each phase to show patellar cartilage on the axial slice, and weight-bearing articular cartilage on the sagittal slices through medial and lateral condyles. Double dose GdDTPA<sup>2-</sup> was injected at the same time as the start of the DCE MRI acquisition, with a rate of 2ml per sec. In addition, T1 mapping was performed at 30, 60, 90 and 120 min after contrast administration using a 3D Variable Flip Angle (VFA) method (flip angles of 4 and 25 degree). B1 correction was performed immediately before the VFA acquisition (*Magn Reson Med* 2010; *in press*).

**ROI Positioning and Data analysis:** In this preliminary study we did not perform pharmacokinetic model analysis. We analyzed the relative enhancement in cartilage and bone. For DCE images, ROIs were placed on articular cartilage and sub-chondral bone marrow in the patella, and medial and lateral condyles (Fig 1, a-b). The average signal intensity of image #2-4 (5-20 sec post injection) was used as pre-contrast signal intensity (SI-pre), and the average signal intensity of image #13-60 (from start of 2<sup>nd</sup> min to the end of 5<sup>th</sup> min) was used as post-contrast signal intensity (SI-post) (Fig 2-a). Relative Enhancement ratio (RER) for each tissue was calculated as (SI-post - SI-pre) / SI-pre (Fig 2-b). For delayed enhancement, automatic inline T1 mapping of cartilage in the medial condyle was performed on the scanner. ROIs at weight-bearing area were segmented as deep layer, superficial layer or full thickness.

## RESULTS

1. In 3 HS and 4 OA, RERs of articular cartilage (the average of cartilage in patella, medial and lateral condyles) were >5%. RERs of patellar cartilage and medial condyle were higher compared to lateral condyle (see table below) ( $p < 0.05$ ); but no significant difference in RER was observed between cartilages in medial condyle compared to patellar.
2. No statistical difference between OA and HS was observed in RER of cartilage ( $p = 0.5$ ).
3. T1 of cartilage at weight-bearing regions of medial condyle steadily decreased after contrast injection with lowest T1 values observed at 120min (Fig 2-c).
4. A significant correlation was observed in RER between articular cartilage and sub-chondral bone ( $p < 0.05$ ) (Fig 3-a), whereas the correlation between RER and T1Gd of cartilage was much weaker (Fig 3-b) ( $p > 0.05$ ).



## DISCUSSION AND CONCLUSIONS

- While early enhancement was observed in the articular cartilage, it was not unique to subjects with OA. The significant correlation between RER in the cartilage with that of bone suggests that the enhancement seen during the early phase is probably related to transport of contrast from the bone interface.
- Absence of any significant correlation between the early and delayed enhancement may suggest that during the delayed phase most of the contrast uptake may be from the synovial fluid interface rather than from the bone. This is consistent with a recent report based on layered analysis of delayed enhancement [*Proc. Intl. Soc. Mag. Reson. Med.* 17 (2009) 3963].
- Contrary to a previous report suggesting that early enhancement is observed primarily in subjects with OA, we have observed enhancement both in healthy and OA while at the same time we observed very little enhancement in some subjects who were healthy or with OA. While it is not yet clear what the underlying cause may be, we believe the enhancement pattern may be more reflective of the presence of active inflammation. Since the subjects with OA could be taking medications such as NSAIDs, their level of inflammation may be reduced at the time of the study. Similarly, healthy (asymptomatic) subjects could have inflammation following strenuous work such as being on their knees for prolonged durations, like in construction workers.

Further studies are warranted to better evaluate early enhancement in cartilage of healthy subjects and those with OA. Better characterization of the health status of the cartilage and instructions to avoid strenuous exercise or work and NSAIDs for at least a day prior to the study may be necessary.