Quantitative perfusion of hepatocellular carcinoma before and after Y-90 radioembolization using a MR angiographic technique with multi-echo and radial k-space sampling

N. Chatterjee[1,2], R. J. Lewandowski[1], E. Semaan[1], R. Salem[1], R. Ryu[1], K. Sato[1], F. Miller[1], J. C. Carr[1], T. J. Carroll[1,2], and J. D. Collins[1]

[1]Department of Radiology, Northwestern University Feinberg School of Medicine, Chicago, IL, United States, [2]Department of Biomedical Engineering, Northwestern University, Chicago, IL, United States

Introduction: Yittrium-90 (Y90) radioembolization (RAE) therapy is a therapeutic option for patients with hepatocellular carcinoma (HCC) that can downstage patients to enable liver transplantation or improve progression free survival in those with locoregional liver disease [1]. Y-90 RAE is performed by selectively infusing radioactive microspheres to the arterial branches supplying the tumor from a microcatheter positioned in a branch of the hepatic artery. While the Y90 therapy has become a well-recognized therapeutic option, it can sometimes be difficult to assess treatment response early (1-3 months) following embolization on conventional MR and CT imaging due to the confounding effects of liver radiation injury with hyperemia. The purpose of this study is to evaluate target lesion perfusion as a biometric for HCC treatment response using a highly accelerated CE-MRA employing echo-sharing and radial k-space sampling. We hypothesize that that target lesion perfusion will be minimally changed immediately post and decrease at 1-month following Y-90 RAE using glass microspheres.

Methods: Semi-quantitative MR perfusion was performed on 11 HCC lesions in 9 patients (5 male, avg age = 65 yrs) before, immediately following, and 1-month post Y90 RAE. As part of an IRB approved prospective study, subjects received a contrast enhanced MR angiogram. 28 slices were acquired at slice thickness of 3mm, 192x192 matrix, 360x360mm FOV, for a voxel size of 3x1.875x1.875mm^3. A sliding window reconstruction was used enabling us to decrease our effective TR by taking advantage of the radial k-space acquisition. In this approach, intermediate frames are reconstructed by using radial lines acquired from adjacent acquisitions. While the scan was acquired with a TR of 2900ms, the sliding window allowed for a 64-fold reduction in effective TR, resulting in an effective TR of 45ms (22 frames/second) [2].
Perfusion images were generated offline from unmasked angiographic data using custom scripts in MATLAB [3]. This allowed us to acquire perfusion data without the need for an additional scan. The arterial input function (AIF) was sampled in the abdominal aorta, and perfusion images were calculated using singular value decomposition (Figure 1). Lesional, embolized, and remote non-embolized parenchyma perfusion was measured and scaled to normal renal cortex to generate units of mL/100g/min. Lesion response to therapy was evaluated using mRECIST criteria on routine MR imaging by a blinded observer, and responders were defined as stable disease, partial response, or complete response. T-tests were used to compare perfusion between baseline, immediate post, and 1-month post treatment.

Results: Imaging follow-up was available for an average of 4 (1-8) months. 10 lesions were responders while 1 lesion progressed. Target lesions averaged 2.9 (1.3-6.8) cm. Baseline lesion perfusion was similar between responders and non-responders (138 vs 133.8 mL/100g/min). Lesion perfusion increased significantly from a baseline of 134.5 (96.1 – 138) to 179.6 (100.4 - 312.2) immediately post therapy and 236.3 (39.2-432.4) mL/100mg/min 1 month after therapy. Normalizing to remote liver, the target lesion perfusion had indices of 2.48, 1.86, and 1.35 at each time point respectively. Correcting for regional hyperemia resulted in indices of 0.994 and 0.629 immediately after and 1 month after treatment.

Discussion: Perfusion measured using data from a CE-MRA acquisition showed significant increases in absolute perfusion (mL/100mg/min) after treatment due to regional hyperemia. However when corrected by normalizing to remote liver parenchyma, lesion perfusion significantly decreased after treatment. Our results demonstrate that the absolute target lesion perfusion increase seen following Y-90 RAE is due to regional hyperemia. Normalizing to remote, non-embolized liver parenchyma and the adjacent hyperemic, embolized liver parenchyma is necessary to demonstrate target lesion treatment effects.

Figure 1: Un-subtracted angiographic images with tumor pre (left) and 1-month post (right) Y-90 radioembolization. The core of the tumor is darker after treatment but there is adjacent hyperemia.
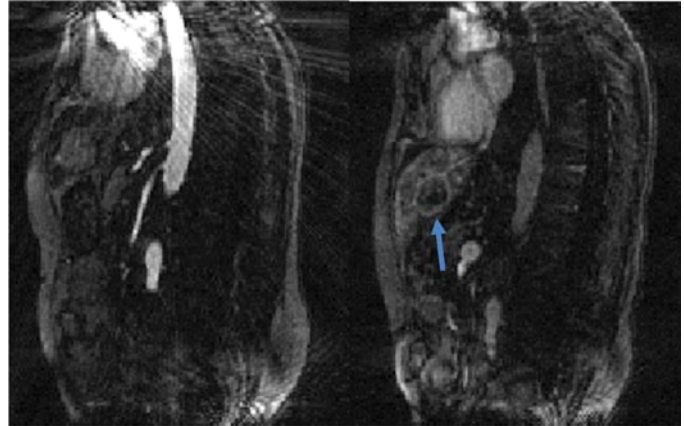

Figure 2: Perfusion maps of a patient with HCC pre (left) and 1-month post (right) Y-90 radioembolization demonstrating a significant decrease in the tumor perfusion post embolization (arrows). Note the increased perfusion following Y-90 radioembolization in the adjacent, embolized hepatic parenchyma.
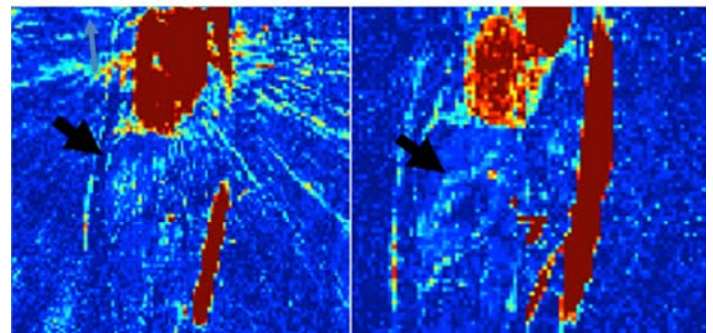

Figure 3: Post contrast VIBE images for a patient with partial response by RECIST and EASL criteria. Images are pre, 1 month post, and 3 months post RAE

Conclusion: Arterial input function calculated perfusion at sliding window CE-MRA is feasible for evaluating HCC response to Y-90 RAE. Tumor perfusion increases post RAE due to regional hyperemia, but normalizing to adjacent and remove liver tissue perfusion appears to be a promising biometric to evaluate response to RAE.

References: [1] Kulik LM et al. Hepatology 2008; 47:71-81 [2] Cashen, et al. Magn Reson Med 2007; 58:962-972 [3] Vakil, et al. Magn Reson Imaging 2012; 30(6):878-85

Proc. Intl. Soc. Mag. Reson. Med. 22 (2014)

2168.