# Perfusion quantification in hepatocellular carcinoma using dynamic contrast-enhanced MRI.

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**Introduction:** The incidence of hepatocellular carcinoma (HCC) has doubled over the past 2 decades in the US, currently estimated between 8,500 to 11,500 a year (1), and is predicted to increase over the next years. HCC is a typical angiogenic tumor, with increased arterial supply. We have already demonstrated the role of DCE-MRI for the diagnosis of advanced liver fibrosis (2). There is limited data on the use of dynamic contrast-enhanced (DCE) CT or MRI to describe vascular characteristics of HCC (3). The purpose of our study was to quantify perfusion metrics of HCC with DCE-MRI in patients with cirrhosis and HCC.

**Methods:** DCE-MRI of the liver was prospectively performed on 24 patients, including 22 with cirrhosis (in whom 16 had HCC) and 2 with normal liver. Coronal 3D interpolated spoiled GRE sequence was performed at 1.5 T after injection of 10 mL of Gd-DTPA using TR/TE 1.7-3.2/0.8, flip angle 9°, 128 x 256, in-plane pixel size of 3.1x 1.8 mm, 18 x 40 cm FOV, slice thickness 2-4 mm, parallel imaging factor 2-3, temporal resolution 2.5-5 sec for approximately 4 min. total acquisition time. ROIs were drawn by an observer on the portal vein, abdominal aorta (used as a surrogate for the hepatic artery), liver parenchyma and HCC lesions. Time concentration curves were analyzed using a dual-input single-compartmental model (2). The following parameters were obtained: Fa (absolute arterial flow, in ml/min/100g), Fp (portal venous flow, in ml/min/100g), arterial fraction (ART %), portal venous fraction (PV %), distribution volume (DV %) of Gd-DTPA and mean transit time (MTT, in sec) of Gd-DTPA. Perfusion parameters were compared between HCC and liver parenchyma, and between treated [post transarterial chemoembolization (TACE)] and untreated HCCs.

| | Fa | Fp | ART (%) | PV (%) | MTT | DV (%) |
|---|---|---|---|---|---|---|
| **All HCCs (n=19)** | 47.2±38.2 | 27.5±41.9 | 65.0±32.7 | 34.9±32.7 | 21.4±18.6 | 17.8±10.5 |
| **HCCs post TACE (n=3)** | 8.2±9.0 | 19.4±5.2 | 23.0±18.3 | 76.9±18.3 | 20.3±21.9 | 8.3±8.5 |
| **Untreated HCCs (n=16)** | 58.6±38.5 | 24.9±40.1 | 75.3±27.4 | 24.6±27.4 | 21.9±18.7 | 19.5±11.0 |
| **Liver (n=24)** | 10.6±5.3 | 37.0±20.2 | 23.9±9.5 | 76.0±9.5 | 24.7±11.5 | 16.2±3.8 |
| **p all HCCs vs. liver parenchyma** | **0.0003** | **0.0035** | **0.0001** | **0.0001** | 0.117 | 0.741 |
| **p untreated HCC vs. HCC post TACE** | **0.03** | 0.24 | **0.03** | **0.03** | 0.86 | 0.13 |

**Results:** 19 HCCs in 16 patients (of which 3 were necrotic post TACE) were studied. HCC was confirmed at histology in 10 patients. There were significant differences between HCC and liver parenchyma for Fa, Fp, ART and PV, but not for MTT and DV (Table). In addition, there were differences in Fa, ART and PV between HCC necrotic and untreated HCCs (Table). An example of perfusion images, with time concentration curves are shown (Fig. 1, 2).

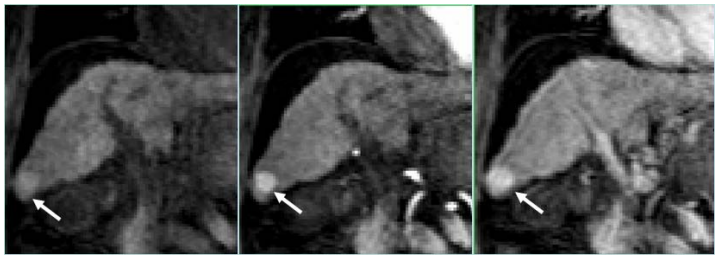

**Fig. 1:** Representative images from liver DCE-MRI acquisition are shown, pre-contrast (left), aorta (center), portal venous phase (right), in a cirrhotic patient with HCC located in segment 6 (arrow).

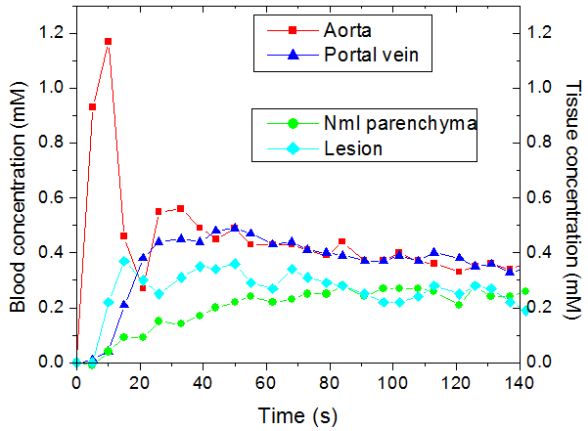


**Fig. 2:** Liver perfusion time concentration curve in the same patient.

**Discussion**: Our findings confirm the increased arterial supply of HCC lesions, higher that than of the cirrhotic liver, which is already elevated, confirming our prior results. We were also able to demonstrate decreased arterial flow in necrotic HCC post TACE. Based on this preliminary data, we believe that DCE-MRI can be used as a non-invasive marker of HCC angiogenesis, and could be used for predicting and monitoring response to targeted anti-VEGF drugs currently investigated in HCC (4) as well as to evaluate response to TACE.

**References:**

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