

Serial apparent diffusion coefficient measurements in hepatocellular carcinoma before and after transarterial chemoembolization

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Introduction:

Transarterial chemoembolization (TACE) is an effective therapy of hepatocellular carcinoma (HCC) resulting in ischemic tumor necrosis. TACE is used as a palliative treatment in non resectable HCC or as a bridge to radical surgical treatment, and has been shown to improve survival (1). There are limited reports on the use of diffusion-weighted imaging (DWI) to evaluate HCC after TACE (2-4). These studies show the potential usefulness of apparent diffusion coefficient (ADC) as a marker of HCC necrosis. The purpose of this study is to assess serial changes in tumor ADC of HCC before and after TACE, using MRI subtraction and explant as the reference.

Methods:

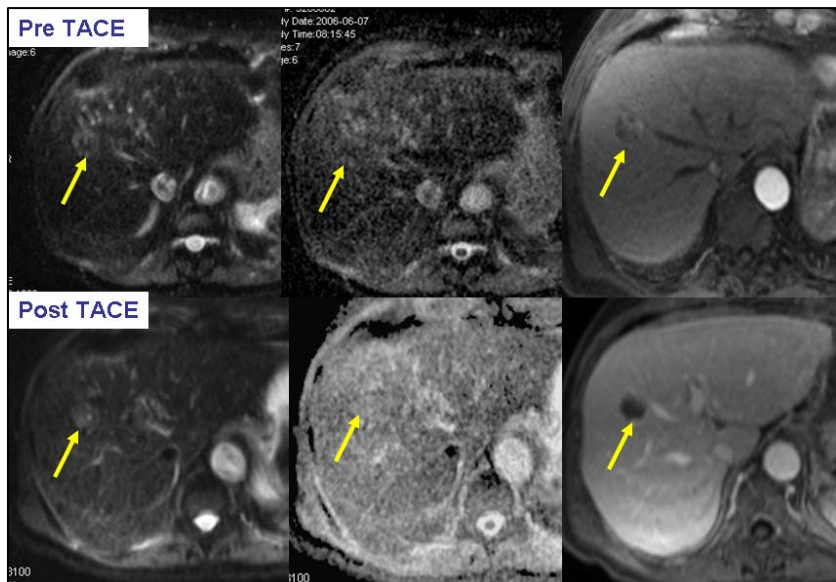
36 patients (31 men, 5 women, mean age 61 y) with liver cirrhosis and HCC treated with TACE were retrospectively evaluated. Pre- and post-TACE MRI included pre-contrast T1 and T2, post-contrast 3D GRE T1 (early-arterial, arterial, portal-venous and equilibrium phases), and breath hold DWI (using single-shot EPI) with b-values of 0-50-500 sec/mm². Patients were scanned within 90 days before and after TACE. Two observers measured ADC of HCCs and liver parenchyma before and after TACE, as well as the percentage of HCC necrosis on the 4 subtracted post-contrast phases. Explant correlation was available in 13 patients (14 HCCs). ADCs before and after TACE were compared using a paired t-test, and ADC post TACE values were correlated with % necrosis at image subtraction and explant.

Results:

47 HCCs (mean size pre-TACE 4.4 ± 3.2 cm, mean size post-TACE 4.2 ± 3 cm) were evaluated. Mean (±SD) whole lesion ADC before and after TACE was 1.53 ± 0.43 vs. 1.77 ± 0.54 × 10⁻³ mm²/s (p < 0.001, paired t-test) (Fig.). ADC of necrotic and viable portions of HCCs after TACE was 1.04 ± 0.51 vs. 1.34 ± 0.37 × 10⁻³ mm²/s (p < 0.0001). There was a significant correlation between post-TACE lesion ADC and % of necrosis measured at the portal venous phase (r = 0.63, p < 0.0001). In patients with explant, there was a significant correlation between ADC and % of necrosis at pathology (r = 0.73, p = 0.004), and between image subtraction and pathology (r = 0.79, p = 0.001). Post-TACE ADC was significantly different in ≥ 75% necrotic HCCs vs. < 75% necrotic HCCs (2.10 ± 0.53 vs. 1.48 ± 0.38 × 10⁻³ mm²/s, p < 0.01). There was no significant difference in liver ADC before and after TACE (1.42 ± 0.30 vs. 1.33 ± 0.36, p = 0.21).

Discussion:

Our findings show increased ADC after HCC ischemic necrosis, similar to prior studies (2-4). This is likely related to decreased tumor cellularity and increased extravascular extracellular space. Our data validate the use of ADC as a marker of HCC response to TACE.



HCC of the right lobe (arrow) before and after TACE, DWI (b=0, left, ADC map, middle) and post-contrast images (right). The lesion becomes necrotic on the post-TACE images, with an increase in ADC from 1.03 to 2.38 (x 10⁻³ mm²/sec).

References:

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2. Kamel IR, et al. AJR 2003;181:708-710.
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4. Chen CY, et al. Radiology 2006;239:448-456.