

Dynamic Contrast MR of Hepatic Hemangiomas: Evaluation of Temporal Signal Intensity Changes

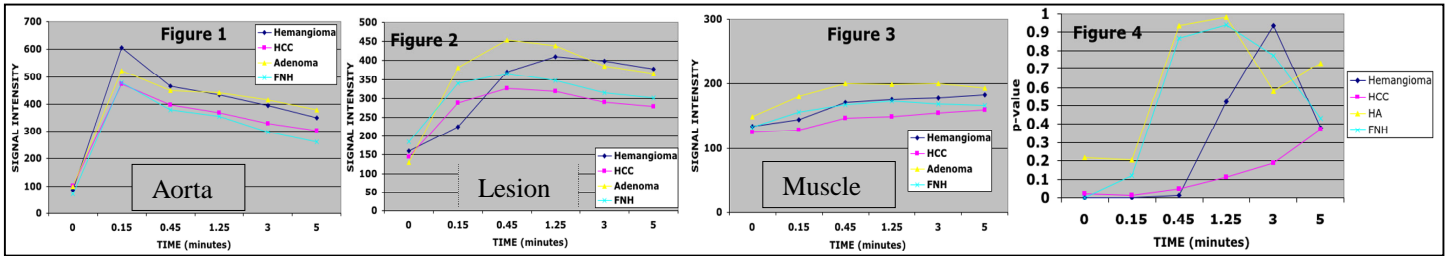
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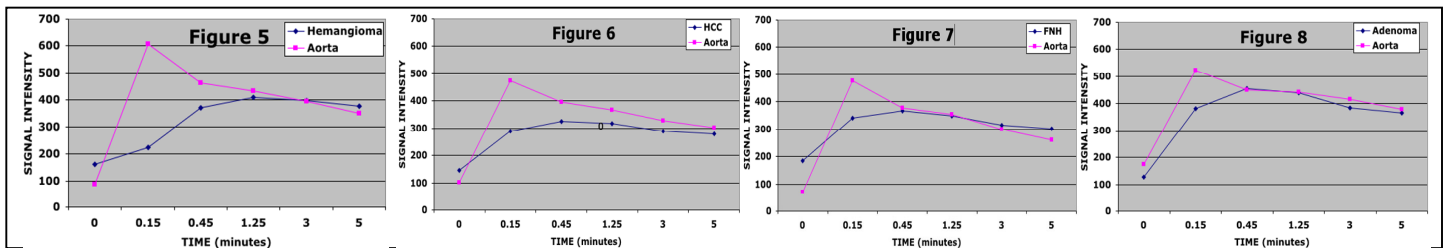
Introduction: Hepatic hemangiomas (HH) are common benign liver lesions found in approximately 7% of the population. HH are distinguished from other mass lesions by analysis of MR signal characteristics and more recently the dynamic enhancement pattern. With regards to dynamic enhancement pattern, a small percentage of HH demonstrate rapid diffuse enhancement, precluding analysis of the established characteristic pattern of peripheral nodular enhancement. In addition a small percentage of hepatocellular carcinomas (HCC) have peripheral nodular enhancement (1). To improve lesion specificity, densitometry as measured on multiphase CT, has shown that HH remains isodense relative to the aorta in the arterial phase of contrast enhancement and isodense with IVC in the blood pool phase (1). This approach, however, has not been extensively evaluated with MR. On MR contrast enhancement is non-linear relative to concentration, may vary from scanner to scanner, position from a surface coil and may further be altered in the vasculature due to time-of-flight effects and turbulence. The goal of this study was to evaluate the time related signal intensity changes of HH compared to the abdominal aorta (AA) to improve differentiation from other mass lesions of the liver in cases where conventional qualitative analysis techniques are not feasible or equivocal.

Methods: A retrospective analysis of 46 patients with contrast enhancing hepatic lesions was conducted. All studies were performed using a 1.5 Tesla magnet (GE, Milwaukee, WI) with 23-40 mT/m gradient amplifiers and using the body coil for optimal signal uniformity. A contrast exam was obtained using a series of six consecutive axial 8mm skip 1.5mm T1-weighted breath-hold FMSPGR (TR=150ms, TE=1.9ms, Matrix=256x160, FA=80 degrees, NEX=1) and spectral fat pre-saturation. Following a baseline non-contrast FMSPGR sequence, Gadodiamide (Nycomed-Amersham, Bucks, UK) was injected intravenously with a Medrad power injector (Medrad, Indianola, PA) at 2 cc's per second for a total dose of 0.1 mmol per Kg of body weight. Five post-contrast FMSPGR sequences were obtained with the first three sequences obtained in rapid succession at approximately 15, 45 and 75 seconds, followed by acquisitions at 3 and 5 minutes. HH lesions were defined by discontinuous nodular peripheral enhancement. All remaining lesions were correlated with biopsy and laboratory results. Utilizing functional analysis software (FUNCTOOLS) on an Advantage Workstation (GE) regions of interest (ROI) were placed in the AA, lesion and paraspinal muscle to determine signal intensity units over time. In each case, a single optimal axial slice was determined for the lesion that minimized misregistration between temporal phases and allowed for the optimal selection of ROI. To compare the signal intensity of AA to lesion for the same subject, a student t-test was performed using the two-tailed distribution.

Results: The temporal signal intensity of AA, lesion and muscle for each pathologic category is shown in figures (1-3) respectively. A total of 24 patients had 26 lesions meeting the criteria for HH and ranged in age from 32 to 86 years (Ave.=54). A total of 12 patients in age from 45 to 65 years old (Ave.=57) with cirrhosis and end-stage liver disease were diagnosed with hepatocellular carcinoma (HCC), 10 with pathologic confirmation and 2 patients with presumed HCC due to markedly elevated alpha-fetoprotein levels. A total of 5 patients ages from 21 to 76 (Ave.=47) were diagnosed with hepatic adenoma (HA) by pathologic confirmation for a total of 5 lesions. A total of 5 patients ages from 35-42 years (Ave.=38) were diagnosed with focal nodular hyperplasia (FNH) by pathologic confirmation for a total of 6 lesions. Calculated p-values for comparing signal intensity of the AA to lesion for each phase of contrast enhancement is shown in figure 4.



Discussion: Our results show that the arterial phase coincided for all groups at 15 seconds (Fig.1). HCC, HA and FNH all demonstrated rapid enhancement with HA demonstrating the most rapid wash-in and wash-out and largest peak enhancement (Fig.2). Muscle in all groups reached signal stability (Fig.3) at approximately 45 seconds indicating the beginning of the blood pool phase. Although multiphase CT studies demonstrated that HH are most similar to AA at the arterial phase (1) our results showed that signal intensity of HH and the AA are most similar at 3 minutes ($p=0.934$) which is well into the blood pool phase of enhancement (Fig.5). This is in contradistinction to HCC (fig.6), which remained significantly less enhancing than the AA throughout the entire five minutes of imaging ($p=0.021-0.373$). FNH was similar to AA signal intensity at 75 seconds (fig.7) ($p=0.939$) and HA was most similar to AA at 45 ($p=0.935$) and near identical at 75 seconds ($p=0.984$) (fig.8), indicating both lesions tracked the AA closely within the early blood pool phase.



Conclusion: Dynamic contrast MR can provide additional qualitative information about hepatic masses through the analysis temporal signal intensity referenced to the AA. HCC demonstrated the least similarity of signal intensity to AA compared to any of the lesions tested. HA and FNH were most similar to aorta in the early blood pool phase whereas HH was most similar at mid blood pool phase. The differences between these lesions were significant and indicate qualitative analysis of temporal signal intensity may be helpful in distinguishing HH from HCC, FNH and HA when other analysis techniques are problematic or equivocal.

1. Kim T, Federle MP, et al. Discrimination of Small Hepatic Hemangiomas from Hypervascular Malignant Tumors Smaller than 3cm with Three-Phase Helical CT. Radiology 2001;219:699-706.