Quantitative analysis of the diffusion-weighted steady-state free-precession signal in vertebral bone-marrow lesions

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Introduction: Diffusion-weighted steady-state free-precession (DW-SSFP) sequences are extremely valuable for the differential diagnosis of benign osteoporotic and malignant neoplastic vertebral compression fractures, which appear in DW-SSFP MRI hypo- to isointense or hyperintense, respectively [1,2]. This differentiation, which can be difficult on conventional MR images, is highly relevant in MRI of the spine for an accurate diagnosis, appropriate treatment, and prognosis. In contrast to other diffusion-weighting sequences, the DW-SSFP signal depends not only on the apparent diffusion coefficient (ADC), but also on the tissue relaxation times and several sequence parameters. However, a theoretical analysis of the measured DW-SSFP signal in vertebral bone marrow (VBM) has not been performed yet and a physical understanding of the observed signal contrast (hypo-/isointense vs. hyperintense signal) is still lacking. The purpose of the present study was, therefore, to provide a detailed analysis of the DW-SSFP signal in VBM and in benign as well as in malignant vertebral lesions (VLs) in order to understand the observed signal alterations and their dependence on tissue and sequence parameters.

Theory: The description of the NMR SSFP signal formation in the presence of an additional diffusion-sensitizing gradient was based on the results by Wu, Buxton, and Deoni et al. [3–5]. The established signal description was extended with additional terms describing the T_2^* relaxation and the effects of the combination of the fat and water signal in VBM, yielding the signal equation

$$S_{\mathrm{VBM}} = f_{\mathrm{water}} S^-_{\perp,\mathrm{water}} \exp(-T_{\mathrm{E}}/T^*_{2,\mathrm{water}}) + f_{\mathrm{fat}} S^-_{\perp,\mathrm{fat}} \exp(-T_{\mathrm{E}}/T^*_{2,\mathrm{fat}}) \exp(-i\Delta\omega_{\mathrm{fw}} T_{\mathrm{E}})$$

depending on the relative water and fat fractions f_{water}, f_{fat}, the corresponding T_2^* relaxation times $T^*_{2,\mathrm{water}}$, $T^*_{2,\mathrm{fat}}$, the frequency difference between water and fat $\Delta\omega_{\mathrm{fw}}$, the echo time T_{E}, and the original DW-SSFP signals $S^-_{\perp,\mathrm{water}}$, $S^-_{\perp,\mathrm{fat}}$ according to [5], which depend on the relaxation times, T_1 and T_2, and the ADCs of both signal components as well as on the sequence parameters (repetition time T_{R}, diffusion gradient shape, and the flip angle).

Methods: MRI was performed on a 1.5-T whole-body scanner in 40 patients with benign (n=20) or malignant (n=20) VLs to determine the fat fraction and tissue parameters (ADC, T_1, T_2, T_2^*) for both the water and fat signal component (using single-shot turbo-spin-echo (ssTSE) sequences for ADC, T_1, and T_2 quantification, and gradient-echo sequences for fat-fraction and T_2^* quantification). With these values, the DW-SSFP signal was simulated and compared with the measured signals (PSIF diffusion sequence: T_{E} = 7.17 ms, T_{R} = 25 ms, flip angle 40°; diffusion gradient 23 mT/m, duration δ = 0.5,1.5,3.0,5.0,7.4 ms) for different diffusion gradients. Simulations and measurement results were compared by determining the signal ratios R_{VL} between the SSFP signals of the lesions and of normal-appearing VBM for both malignant and benign vertebral lesions.

Results: The simulated DW-SSFP contrast agreed well with the measured contrast (cf. Fig. 1) and provided a very good differentiation between benign osteoporotic and malignant VLs with a sensitivity of up to 95% and a specificity of 100%. ADCs were significantly different in both lesion types; however, the greatest contributions to the observed total image contrast resulted from the differences (between both patient groups) of f_{fat} in the lesions as well as in normal-appearing VBM, of $T^*_{2,\mathrm{fat}}$ in normal-appearing VBM, and of $T^*_{2,\mathrm{water}}$ in the lesions (cf. Fig. 2). Smaller effects are caused by the differences of $T_{1,\mathrm{fat}}$ and $T_{2,\mathrm{fat}}$ in normal-appearing VBM and (with opposite sign) of $T_{2,\mathrm{water}}$ in the lesions. The other parameter differences do not contribute substantially to the observed contrast.

Conclusions: In this study, we could confirm previous results that the DW-SSFP sequence provides an excellent differentiation between benign osteoporotic and malignant VLs. We could also show that the main reasons for the different lesion contrasts (hypo-/isointense vs. hyperintense signal) are an opposed-phase readout in combination with the differing fat fractions and T_2^*-values in the lesions as well as in normal-appearing VBM of both entities. The observed signal contrast is therefore rather fat- and T_2^*-weighted than diffusion-weighted. The intermediate diffusion weighting of the applied SSFP sequence, however, helps to shift the different contrasts into a signal range that is easily visually accessible.

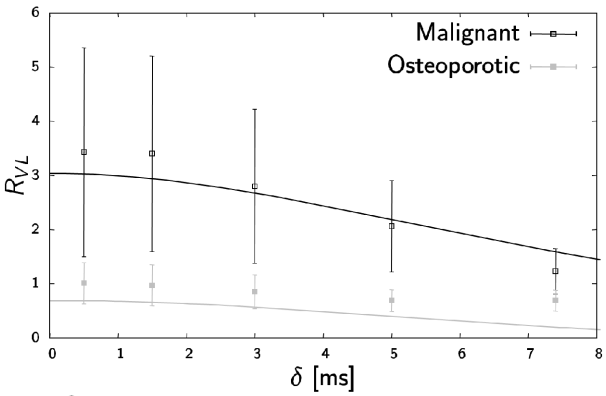

Fig. 1: Simulations and measurements of the signal ratio, R_{VL}, between the DW-SSFP signal of a VL and of normal-appearing VBM as a function of the diffusion gradient duration, δ; R_{VL} >1 corresponds to hyperintensity, R_{VL} <1 to hypointensity. The measured mean values of R_{VL} (squares with errorbars) are compared with the simulated values (solid lines). The length of the errorbars corresponds to the twice the standard deviation.

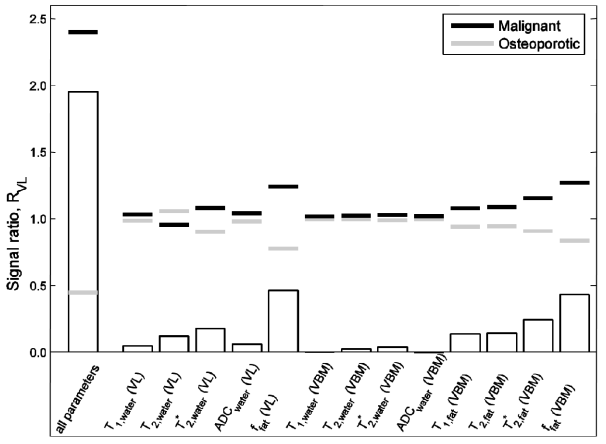

Fig. 2: Influence of the different tissue parameters on the simulated signal ratio, R_{VL}. All parameters except the one given at the horizontal axis were set to the mean value of both patient groups. The solid horizontal lines indicate the mean values of the signal ratios, R_{VL}, averaged over all evaluated diffusion weightings (gradient duration 0 ≤ δ ≤ 9ms) for malignant or osteoporotic lesions. The bars correspond to the average contrast difference between both patient groups.

References: **[1]** Baur A, et al. Radiology 1998; 207: 349–356 **[2]** Karchevsky M, et al. Skeletal Radiol 2008; 37: 791–795. **[3]** Wu EX, Buxton RB. J Magn Reson 1990; 90: 243–253. **[4]** Buxton RB. Magn Reson Med 1993; 29: 235–243. **[5]** Deoni SCL, et al. Magn Reson Med 2004; 51: 428–433.