MRS Detectable Lipid Signal in Low- and High-Grade Gliomas, Recurrent Gliomas, Metastases, Lymphomas, and Abscesses

University Hospital Frankfurt, Frankfurt, Germany

Purpose
MR spectroscopy (MRS) has been shown to be an important tool for characterization of brain lesions. The main focus of most studies laid on the quantitative analysis of changes of the main signals obtained from normal brain tissue (e.g. NAA, Choline, Creatine) but the signal at 1.3 ppm, which is sometimes visible in brain lesions has been used also for differential diagnosis. This signal is originating from lipids and can be discriminated from lactate since it is not inverted at TE of 136 ms. Based on in vitro studies with cell culture, the MRS detectable lipid signal has been attributed to the association of mobile neutral lipids with proteins intercalated within the membrane bilayer (Mountford and Wright, 1988), but it has also been correlated with the accumulation of lipid droplets in the cytoplasm (Callies et al., 1993); ( Remy et al., 1997). It was shown in vitro, that cell culture conditions as well as the exposure to several kind of stress will modulate this signal, indicating that the neutral lipid signal may provide important information on cell proliferation and could be a marker for unfavorable environmental conditions (e.g., hypoxia). Consequently, it has been associated with necrotic areas (Gotsis et al., 1996); ( Kuesel et al., 1996); ( Sijens et al., 1996), inflammation and lesions with ongoing lysis. In this study we examined a group of 96 consecutive cases of brain lesions with regard to the histopathology and the occurrence and intensity of the mobile lipid signal at 1.3 ppm.

Materials & Methods
Ninety-five cases of brain lesions were examined: 38 gliomas WHO IV, 6 astrocytomas WHO I/II, 10 astrocytomas WHO grade III, 2 oligoastrocytomas WHO grade II, 1 oligoastrocytomas WHO grade III, 3 oligodendroglioma WHO grade II, 5 oligodendrogliomas WHO grade III, 13 metastases, 4 lymphomas, 4 abscesses, 3 meningoma, 2 ependymoma, 1 PNET, 1 neurocytom, 1 inflammation, 1 colloidcyst. Prior to stereotactic biopsy or surgical removement, patients with suspected primary brain tumors were subjected to a single voxel 1H MRS exam of the tumor. Within 5 days after the 1H MRS exam patients underwent surgery. Postsurgically histopathologic diagnoses were established in accordance with the current WHO classification system. 1HMRS studies were performed with a clinical 1.5 T MR scanner. After acquisition of axial T2- and postcontrast T1-weighted axial and coronal MR tomograms, single voxel 1H MRS was conducted. Based on MR criteria, the volume of interest (VOI) was placed within viable tumor, excluding necrotic or cystic areas and avoiding the inclusion of tumor-adjacent edematous brain. Radiologic criteria for necrosis was a noncontrast enhancing irregularly bordered area within the tumor showing high intensity in T2-weighted tomograms and low intensity in T1-weighted tomograms. Cysts were identified as well circumscribed, rounded lesions with high signal intensity, usually brighter than necrotic areas, in T2- and low signal in
T1-weighted images while no contrast enhancement was observed (21). Usually, the VOI was placed in the tumor area which was later removed by surgery or targeted during stereotactic biopsy. After selection of the VOIs one or two water-suppressed metabolite spectra were acquired using a double spin-echo localization technique (PRESS) and frequency selective water suppression (CHESS). Reference data were obtained with the VOI located in the mirror-image region of the contralateral hemisphere. Depending on the size of selected VOIs, 128 to 512 acquisitions with an echo time of 135 ms and repetition time of 1500 ms were added. Spectroscopic raw data were transferred to a Unix workstation and analyzed with the MRUI tool. The resulting metabolite signal intensities from the tumor tissue were quantified (normalized) by calculating their ratio to the signal intensity of tCr measured in the reference spectrum and expressed as ratio of the reference tCr.

Results
Lipids were detected in 11 of 13 metastases and 14 out of 38 gliomas WHO IV. These gliomas also showed necrotic areas in the MR exam which could not always be completely excluded from the VOI. Only 4 out of the 28 astrocytomas of lower WHO grade (I-III) showed a significant lipid signal. All of these cases were recurrent tumor, three received radiation therapy. High mobile lipid signals were obtained from almost all metastases (11/13). In contrast to gliomas WHO IV, lipids were also detected in metastasis without any MR visible necrosis or where necrotic tissue could be excluded from the VOI. From the other entities 1 out of the 4 lymphomas, all abscesses, and the inflammation showed a lipid signal as well.

Conclusion
Our data confirm that the existence of a lipid signal in neuroepithelial brain tumors is a strong indicator for necrotic processes and therefore, is present only in gliomas grade WHO IV. Lipid signals obtained in lower grade glial tumors were observed solely in recurrent tumors and are possibly a consequence of treatment (Kinoshita et al., 1997). Cell debris present in abscesses and inflammation may account for the lipid signal in this entities. In contrast, metastases frequently show an intense narrow lipid signals even in the absence of MR detectable necrosis. The question remains to be solved whether these lipids indicate necrotic or prenecrotic transformation or just reflect the histogenetical origin of the cells.

References