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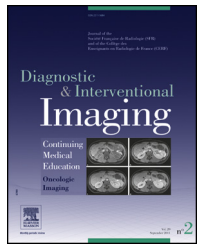

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Low-grade gliomas: The challenges of imaging

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Abstract WHO grade II gliomas are a major challenge for magnetic resonance imaging (MRI) due to their delayed anaplastic transformation. Today it is possible to individually characterize tumor progression from diagnosis to anaplastic transformation based on the many parameters identified in studies in the literature and the possibility of integrating these data into mathematical models. Early identification of negative morphological and metabolic factors, as well as treatment follow-up, help identify predictive factors of tumor progression, as well as determine treatment response to adapt management of this disease.

At present MRI is the first line imaging technique for the non-invasive exploration of intracranial tumor progression. Nevertheless, the sensitivity and specificity of existing MRI protocols are limited [1,2]. According to the WHO classification, grade II gliomas, also called low-grade gliomas (LGG), express atypical nuclei and inevitably progress at a rate that varies from one case to another. In the last decade, the pre-therapeutic characterization of these tumors has improved significantly thanks to advances in imaging techniques. Thus, MR spectroscopy (which is essentially protonic) and dynamic perfusion imaging have helped improve the specificity and sensitivity of MRI for the diagnosis and follow-up of gliomas (Fig. 1). Tumor volume, relative cerebral blood volume and MR spectroscopy results have also provided predictive factors of disease progression or response to treatment, while other MRI parameters are correlated to molecular profiles.

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Improving the diagnostic accuracy: how aggressive is a WHO grade II (OK?) tumor?

From diagnosis to prognosis. Diffuse low-grade gliomas (WHO grade II gliomas, LGG) are heterogenous on MRI. These infiltrating tumors have ill-defined margins on T2-weighted (hyperintense) as well as T1-weighted (hypointense) images and usually have no contrast enhancement following gadolinium injection. When enhancement occurs, in 10 to 50% of the cases depending on the series, it is moderate and only in a small portion of the tumor. The prognostic value of this event is still a subject of debate [3,4]. Only clearly nodular-shaped tumors and time-progressive tumor enhancement have been shown to have a negative prognostic value [5]. Moreover, there is significant intraobserver variability as well as an inter-institutional heterogeneity of parameters depending on the magnetic field and the types of sequences obtained. T1-weighted spin echo sequences have been shown to be better than gradient-echo sequences [6,7]. Recent studies have shown that diffusion imaging with ADC variations is better at identifying anaplastic transformation in progressive LGG [8]. The 1p/19q co-deletion can be confirmed by quantifying texture [9]. Several correlations have been established between proton spectroscopy and pathology, immunohistology and molecular biology, and some of these criteria are considered to be predictive of anaplastic transformation of LGG. Thus, there is a correlation with the Cho/Cr index (all levels). More recently, specific spectral patterns for a range of Ki-67 values have been established for LGG [10]. Thus, the presence of increased lactate resonance, indicative of underlying anaerobiosis, is predictive of a Ki-67 index of 4 to 8% (Fig. 2). Later, membrane alterations result in resonance of mobile lipids, which is correlated to atypical cells and predictive of a Ki-67 index of above 8% (Figs. 1 and 2). Thus, the results of ''spectroscopic'' analysis suggest that there is a critical Ki-67 threshold value. Similarly, indirect identification of the IDH mutation based on 2 hydroxyglutarate resonance on MR spectroscopy using separation spectral editing, a tool that is now available in some centers, has diagnostic and predictive value [11]. Multinuclear MR spectroscopy, and in particular ^{31}P phosphorus spectroscopy, can also be used to investigate

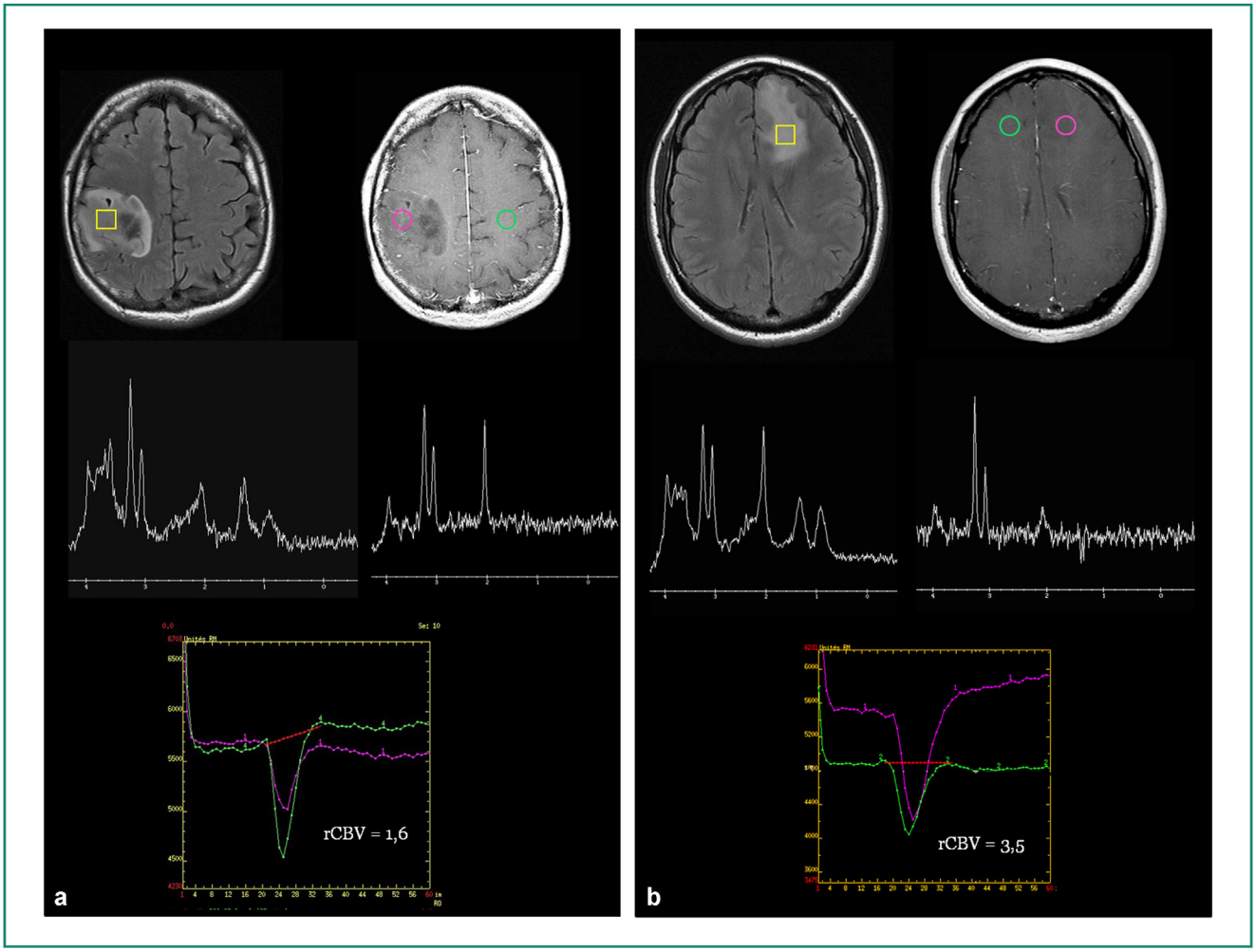

Figure 1. Is this a low-grade glioma? (a) Heterogeneous gadolinium enchanced right parietal rolandic tumor. On MR spectroscopy, CNI is moderately increased with the presence of lactate resonance. The rCBV is 1.6. WHO grade II glioma with Ki-67 index = 4%. (b) Homogenous frontal lobe tumor on all sequences, no gadolinium enchancement. The CNI is much higher here with mobile lipids and lactate resonance and the rCBV is 3.5. WHO grade II glioma, Ki-67 index 12%.