IDH-mutant glioma magnetic resonance imaging

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IDH-mutant gliomas may appear as a well-defined, low-grade mass with irregular borders and a heterogeneous appearance. These tumors may show a “bubbly” or “swiss cheese” appearance on imaging, with small cystic areas within the tumor. They may also show a characteristic “ring-enhancement” pattern, where the outer edge of the tumor appears brighter on contrast-enhanced imaging.

In addition to MRI, other imaging modalities may be used to diagnose and monitor IDH-mutant gliomas, including computed tomography (CT) scans and positron emission tomography (PET) scans.

It's important to note that imaging alone cannot definitively diagnose IDH-mutant gliomas, and a biopsy or surgical resection may be necessary to confirm the diagnosis.

In recent years, magnetic resonance imaging (MRI), whose main functions has been to detect a tumor, to provide spatial information for neurosurgical and radiotherapy planning, and to monitor treatment response, has shown potential in assessing molecular features of gliomas from image-based biomarkers. As an outstanding example, numerous studies have proven that the T2/FLAIR mismatch sign can identify IDH-mutant, 1p/19q non-codeleted astrocytomas with a specificity of up to 100%. For other purposes, multiparametric MRI, often coupled with machine learning methods, seems to achieve the highest accuracy in predicting molecular markers. Relevant future applications might be anticipating changes in the molecular composition of gliomas and providing useful information

about the cellular and genetic heterogeneity of gliomas, especially in the non-resected tumor parts [1].

Certain MRI morphologic features and visual detection of diffusion restriction on DWI and quantitative ADC parameters consisting of ADCmean, ADCmin, and ADCr can be considered non-invasive, significant independent imaging predictors in the discrimination and can obviate invasive procedures for histopathological diagnosis [2]

Adding DSC-PWI to conventional MRI can improve molecular subtype prediction in patients with diffuse gliomas [3].

Computer-aided diagnosis (CADx) systems using machine learning have been noted as innovative diagnostic methods. However, it is difficult to promote the clinical application of machine learning systems at each institute because the support of various specialists is essential. Nishikawa et al. established an easy-to-use computer-aided diagnosis system using Microsoft Azure Machine Learning Studio (MAMLS) to predict these statuses. They constructed an analysis model using 258 adult-type diffuse glioma cases from The Cancer Genome Atlas (TCGA) cohort. Using MRI T2-weighted images, the overall accuracy, sensitivity, and specificity for the prediction of IDH mutation and 1p/19q codeletion were 86.9%, 80.9%, and 92.0%, and 94.7%, 94.1%, and 95.1%, respectively. We also constructed an reliable analysis model for the prediction of IDH mutation and 1p/19q codeletion using an independent Nagoya cohort including 202 cases. These analysis models were established within 30 min. This easy-to-use CADx system might be useful for the clinical application of CADx in various institutes [4]

Yano et al. retrospectively analyzed astrocytic tumors, including 18 grade II astrocytomas (isocitrate dehydrogenase (IDH)-mutant: IDH-wildtype = 8:10) and 56 grade III anaplastic astrocytomas (37:19). They recorded the maximum methionine (MET) uptake ratios (tumor-to-normal: T/N) on positron emission tomography (PET) and three MRS peak ratios: choline (Cho)/creatine (Cr), N-Acetylaspartic acid (NAA)/Cr, and Cho/NAA, between June 2015 and June 2020. They then evaluated the cut-off values to differentiate between grades II and III. They compared the grading results between contrast enhancement effects on MR and combinational diagnostic methods (CDM) on a scatter chart using the cutoff values of the T/N ratio and MRS parameters.

The IDH-mutant group showed significant differences in the Cho/NAA ratio between grades II and III using univariate analysis; however, multiple regression analysis results negated this. The IDH-wildtype group showed no significant differences between the groups. Contrast enhancement effects also showed no significant differences in IDH status. Accordingly, regardless of the IDH status, no statistically independent factors differentiated between grades II and III. However, CDMs showed higher sensitivity and negative predictive value in distinguishing them than MRI contrast examinations for both IDH statuses. We demonstrated a significantly higher diagnostic rate of grade III than of grade II with CDM, which was more striking in the IDH-mutant group than in the wild-type group.

CDM could be valuable in differentiating between grade II and III astrocytic tumors [5)]

Branzoli and Marjańska highlighted the utility of MRS in the noninvasive glioma diagnosis with mutations in isocitrate dehydrogenase (IDH) genes, by providing an overview of the neurochemical alterations observed in different glioma subtypes, as well as during treatment and progression, both in vivo and ex vivo.

D-2-hydroxyglutarate (2HG) decrease during anticancer treatments was recently shown to be associated with altered levels of other metabolites, including lactate, glutamate and glutathione, suggesting that tumour treatment leads to a metabolic reprogramming beyond 2HG depletion. In combination with 2HG quantification, cystathionine and glycine seem to be the most promising candidates for higher specific identification of glioma subtypes and follow-up of disease progression and response to treatment.

The implementation of advanced MRS methods in the routine clinical practice will allow the quantification of metabolites that are not detectable with conventional methods and may enable immediate, accurate diagnosis of gliomas, which is crucial for planning optimal therapeutic strategies and follow-up examinations. The role of different metabolites as predictors of patient outcome still needs to be elucidated [6)]

Forty-eight isocitrate dehydrogenase-mutant and 28 isocitrate dehydrogenase-wild-type low-grade gliomas were studied. Isocitrate dehydrogenase mutation was related to more frequency of cortical involvement compared to isocitrate dehydrogenase-wild-type group (34/46 vs 6/24, P = .0001). Peritumoral edema was less frequent in isocitrate dehydrogenase-mutant tumors (32.6% vs 58.3% for isocitrate dehydrogenase-wild-type tumors, P = .0381). Isocitrate dehydrogenase-wild-type tumors were more likely to have a nondefinable border, while isocitrate dehydrogenase-mutant tumors had well-defined borders (66.7% vs 39.1%, P = .0287). Only 8 (17.4%) of 46 of the isocitrate dehydrogenase-mutant tumors demonstrated marked enhancement, while this was 66.7% in isocitrate-wild-type tumors (P < .0001). Choline-creatinine ratio for isocitrate dehydrogenase-wild-type tumors was significantly higher than that for isocitrate dehydrogenase-mutant tumors. In conclusion, frontal location, well-defined border, cortical involvement, less peritumoral edema, lack of enhancement, and low choline-creatinine ratio were predictive for the definition of isocitrate dehydrogenase-mutant low-grade gliomas. Magnetic resonance imaging can provide an advantage in the detection of isocitrate dehydrogenase status indirectly and indicate the need to explore new design for treatment planning in gliomas. Choline-creatinine ratio in magnetic resonance spectroscopy could be a potential more reasonable reference for the new design of delineation of target volume in low-grade gliomas [7)].

IDH wild-type Grade II diffuse gliomas (DGs) are associated with a lower ADC and poor clinical outcomes. Combining IDH mutational status and ADC may allow more accurate prediction of clinical outcomes for patients with grade II DGs [8)].

1)
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