Introduction: Diffuse gliomas are the most mundane adult primary encephalon tumors, the majority of which are glioblastomas. Albeit cancer of the encephalon is relatively infrequent, representing only remotely more than 1% of all incipient cancer cases, it is a disproportionately mundane cause of cancer-cognate death and morbidity. The prognosis for patients with glioma is concretely poor due to its infiltrative nature and constrained sensitivity to radiation therapy and chemotherapy.

Historically, gliomas have been relegated and indited histopathologically according to the World Health Organization (WHO) relegation system (grades I–IV, with incrementing degrees of malignancy) and sundry lineages: astrocytoma, oligodendroglioma, and commixed oligoastrocytoma. The WHO relegation provides prognostic information and guidance on treatment, such as radiation therapy and chemotherapy after surgery. However, especially for the diagnosis of grade II and III gliomas, classic histopathologic evaluation is notorious for its interobserver variation. Despite the attested value of the WHO grading system, accumulating data show that molecular analysis of gliomas is far more informative than is classic histopathologic evaluation.

This development was triggered by the apperception of a vigorous and propitious prognostic and predictive effect of the codeletion of chromosomal arms 1p and 19q on overall outcome and sensitivity to chemotherapy, respectively. Next, the major predictive value of methylation of the methylguanine methyltransferase (MGMT) promoter gene for outcome after adjuvant chemotherapy in patients with glioblastoma was reported. The more recent revelation of isocitrate dehydrogenase (IDH) mutations in the immensely colossal majority of grade II and III diffuse gliomas represented another major step forward in the molecular characterization of gliomas. It was shown that IDH-mutated tumors have a much more auspicious prognosis than do tumors of kindred lineage and grade that express IDH gene wild type (IDHwt) and may even sanction identification of chemotherapy-sensitive subgroups of patients. Authors of more recent research have further refined this concept and have suggested that diffuse gliomas can be characterized as one of three different subtypes, depending on the presence of a handful of molecular markers copy number alterations of chromosomal arms 1p and 19q (in coalescence); chromosomal arm 10q and chromosome 7; and mutational status of the IDH, telomerase reverse transcriptase (TERT), tumor protein p53 (TP53), and α thalassemia/establishment incapacitation syndrome X–linked (ATRX) genes. Note that the TERT and ATRX mutations are mutually exclusive and have diagnostic value: TERT mutations are diagnostic of oligodendroglioma (with 1p19q codeletion) or glioblastoma (without 1p19q codeletion), while TP53 mutations in the presence of IDH mutation are indicative of astrocytoma.

This molecular relegation additionally contains more prognostic information than do classic histopathologic evaluation results. According to which low-grade tumors have a better prognosis than do high-grade tumors. Regardless of tumor grade, however, IDHwt tumours, even those that are tenacious to be of low grade at histopathologic evaluation, are, biologically, all of the glioblastoma type (class 3) and have very impecunious prognosis. The WHO tumor grade may retain its prognostic information, albeit perhaps less so in IDH-mutated tumors. Results of studies have shown that, at the molecular level, commixed oligoastrocytomas do not subsist, because the pres-

Image Analysis for the Classification of Brain Tumor Location on MR Images
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ence or absence of 1p19q codeletion, respectively, relegates the tumor either as an oligodendroglioma or an astrocytoma. Some mutations (1p19q, MGMT promoter methylation, IDH) already have had a major effect on diagnostic neurologic oncology. How to integrate the molecular classes into the standard of care and the design of clinical tribulations is a matter of debate, because molecular diagnostics come with a financial burden to the diagnostic workup, and laboratory facilities that are not always routinely available are required.

In parallel, there are several denotements that categorical imaging features of encephalon tumors may contain prognostic information beyond WHO tumor grade. In their hallmark study to presage survival in patients with low- and high-grade gliomas by utilizing dynamic susceptibility contrast magnetic resonance (MR) perfusion imaging, Law et al demonstrated that the prognosis for patients with low-grade but highly perfused tumors was worse than that in patients with high-grade tumors with low perfusion. This finding illustrates how imaging features may accommodate as surrogate markers of tumor deportment and may reflect molecular tumor profiles. Taking this a step further, Diehn et al correlated several structural imaging features such as necrosis, contrast material enhancement, and mass effect to gene expression signatures in glioblastomas, such as epidermal magnification factor receptor overexpression, hypoxia and the extracellular matrix, and proliferation, respectively. In turn, certain imaging features could be identified that were predictive of the outcome.

Multifocal nonenhancing tumor (arrow, a-e) with histologic grade II astrocytoma in a 57-year-old woman. However, molecular profile is consistent with the glioblastoma type (phosphatase and tensin homolog mutation, epidermal magnification factor receptor amplification, IDHwt ). Despite lack of enhancement, relative cerebral blood volume (rCBV) is high, corresponding to more preponderant degree of malignancy. (a, b) Axial three-dimensional T2-weighted fluid-attenuation inversion-recovery 2-mm reconstruction images (repetition time [TR] 6500 msec; echo time [TE], 110 msec; inversion time [TI] 2002 msec; flip angle, 90°; voxel size, 1.0 × 1.0 × 0.6 mm3). (c, d) Axial three-dimensional inversion-instau-ration expeditious spoiled gradient-echo contrast-enhanced T1-weighted 2 mm reconstruction images (TR, 6.1 msec; TE, 2.1 msec; TI, 450 msec; flip angle, 12°; voxel size, 0.9 × 0.9 × 0.8 mm3). (e, f)rCBV maps overlaid on contrast-enhanced T1-weighted images derived from dynamic susceptibility contrast-enhanced imaging (TR, 2000 msec; TE, 45 msec; flip angle 90°; voxel size, 2.1 × 2.1 × 5.0 mm3; 52 dynamics; contrast material bolus of 10 mmol gadolinium contrast agent distributed after 20-second baseline acquisition without contrast material).

During the past few years, encephalon tumor segmentation in Magnetic Resonance Imaging(MRI) has become an emergent research area in the field of medical imaging system. Precise detection of encephalon tumor plays a consequential role in the diagnosis of tumor. Me and my students develop a program which analyses the MR Images of patient and apperceives the tumor by utilizing image processing and detects the location of tumor. Detection of required area is sensitive and critical subject in segmenting medical images. Precision and expeditious computation time is two consequential scales for these segmentation algorithms. These algorithms gave different results depending on data sets and anatomic structures of images. We implement some of these algorithms and amalgamate them. With first test of our implemented algorithm we optically discern that sensitivity and computation time (3 seconds) proficient at Tresholding with minute datasets. These tests withal show us relegation predicated segmentation algorithms (K-NN, SVM, Bayers) segments tumor accurately and engender good results for sizably voluminous data set but undesirable demeanors can occur in case of where a class is under represent- ed in training data. In integration to that, clustering algorithms (K-designates, Fuzzy) performs very simple, expeditious and engender good results for non-noise image but for noise images it leads to solemn inaccuracy in the segmentation. This could be solved by utilizing precise pre-processing algorithms afore segmentation. More tests with this program will per-
petuate with incipient implementations.

Primary encephalon tumors, most commonly gliomas, are histopathologically indited and graded as World Health Organization (WHO) grades I–IV according to incrementing degrees of malignancy. These grades provide prognostic information and guidance on treatment such as radiation therapy and chemotherapy after surgery. Despite the substantiated value of the WHO grading system, results of a multitude of studies and prospective interventional tribulations now betoken that tumors with identical morphologic criteria can have. Molecular markers can sanction subtypes of tumors of the same morphologic type and WHO grade to be distinguished and are, ergo, of great interest in personalization of encephalon tumor treatment. Recent genomic-wide studies have resulted in a far more comprehensive understanding of the genomic alterations in gliomas and provide suggestions for an incipient molecularly predicated relegation. Magnetic resonance (MR) imaging phenotypes can accommodate as noninvasive surrogates for tumor genotypes and can provide paramount information for diagnosis, prognosis, and, ineluctably, personalized treatment. The incipiently emerged field of radiogenomics sanctions categorical MR imaging phenotypes to be linked with gene expression profiles. In this article, the authors review the conventional and advanced imaging features of three tumoral genotypes with prognostic and therapeutic consequences: (a) isocitrate dehydrogenase mutation; (b) the coalesced loss of the short arm of chromosome 1 and the long arm of chromosome 19, or 1p19q codeletion; and (c) methylguanine methyltransferase promoter methylation.