For nearly a century, the diagnosis and grading of oligodendrogliomas and oligoastrocytomas has been based on histopathology alone. The first glioma-associated molecular signature was found with complete chromosome 1p and 19q codeletion being particularly common in histologically classic oligodendrogliomas. Subsequently, this codeletion appeared to not only carry diagnostic, but also prognostic and predictive information, the latter aspect only recently resolved after carefully constructed clinical trials with very long follow-up times. More recently described biomarkers, including the non-balanced translocation leading to 1p/19g codeletion, promoter hypermethylation of the MGMT gene, mutations of the IDH1 or IDH2 gene, and mutations of FUBP1 (on 1p) or CIC (on 19q), have greatly enhanced our understanding of oligodendroglioma biology, although their diagnostic, prognostic, and predictive roles are less clear. It has therefore been suggested that complete 1p/19g codeletion be required for the diagnosis of 'canonical oligodendroglioma'. This transition to an integrated morphological and molecular diagnosis may result in the disappearance of oligoastrocytoma as an entity, but brings new challenges as well. For instance it needs to be sorted out how (histopathological) criteria for grading of 'canonical oligodendrogliomas' should be adapted, how pediatric oligodendrogliomas (known to lack codeletions) should be defined, which platforms and cut-off levels should ideally be used for demonstration of particular molecular aberrations, and how the diagnosis of oligodendroglioma should be made in centers/countries where molecular diagnostics is not available. Meanwhile, smart integration of morphological and molecular information will lead to recognition of biologically much more uniform groups within the spectrum of diffuse gliomas and thereby facilitate tailored treatments for individual patients ¹⁾.

1)

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