1. EGFR amplification or 2. Combined whole chromosome 7 gain and whole chromosome 10 loss (+ 7/− 10) or 3. TERT promoter mutation. Later in the 6th update (3), in order to simplify the nomenclature they suggested criteria for the diagnosis of GBM, IDH-wt as follows:

Table 5. Suggested criteria for Glioblastoma, IDH-wildtype.

An IDH-wildtype diffuse astrocytic glioma with:

- Microvascular proliferation, or
- Necrosis, or
- One or more the following molecular features or glioblastoma:
  - *TERT* promoter mutation, or
  - *EGFR* gene amplification, or
  - +7/-10 chromosome copy number changes

It was also found that all IDH mutant diffuse gliomas did not behave similarly. In The cIMPACT update 5, evaluated literature to look for molecular and genetic criteria that could reliably stratify risk among patients with IDH mutant astrocytic gliomas that would behave more aggressively corresponding to WHO grade IV. (4) Among the molecular alterations considered were CDKN2A/B homozygous deletion, CDK4 amplification, RB1 mutation or homozyous deletion, PIK3CA or PIK3R1 mutations, PDGFRA amplification, MYCN amplification and others. Multiple studies have identified homozygous deletion of CDKN2A/B as a marker of poor prognosis in patients with IDH-mutant diffuse astrocytic gliomas and were strongly associated with a shorter survival in grade II/III diffuse astrocytic gliomas. Thus new terminology **Astrocytoma, IDH-mutant, WHO grade IV** has been proposed for a diffusely infiltrative astrocytic glioma with an IDH1 or IDH2 mutation that exhibits microvascular proliferation or necrosis or CDKN2A/B homozygous deletion or any combination of these features.

It is anticipated that many of these cIMPACT-NOW updates will be incorporated in the 5th edition of WHO brain tumor classification expected to be released in 2020-2021. The above updates highlight the need for molecular testing in CNS tumors and that histology and IHC alone will not suffice in the classification and treatment of these tumors. In many cases where single molecular markers may not reveal molecular alterations, broader NGS panels such as the Glioseq panel 5 as in the case above maybe required to look for molecular alterations. GlioSeq amplification based NGS libraries to detect single-nucleotide variant (SNVs) and small insertions/deletions in 30 key brain tumor genes, for copy number changes in 24 genes, and >20 types of gene fusions. In addition, a new version of the test