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## **Molecular analysis of anaplastic oligodendroglial tumors in a prospective randomized study: A report from EORTC study 26951.**

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**Background.** Recent studies have shown that the clinical outcome of anaplastic oligodendroglial tumors is variable, but also that the histological diagnosis is subject to interobserver variation. We investigated if the assessment of 1p/19q co-deletion, polysomy of chromosome 7, EGFR gene amplification (EGFR(amp)) and loss of chromosome 10 or 10q offers additional prognostic information to the histological diagnosis and would allow molecular subtyping. **Methods.** For this study the clinical data and tumors samples of the patients included in the multicenter prospective phase III EORTC 26951 trial on the effects of adjuvant procarbazine, CCNU and vincristine chemotherapy in anaplastic oligodendroglial tumors were used. Fluorescent In Situ Hybridization (FISH) was used to assess copy number aberrations of chromosome 1p, 19q, 7, 10 and 10q, and the EGFR gene. Three different analyses were done: on all included patients based on local pathology diagnosis, on the patients with confirmed anaplastic oligodendroglial tumors on central pathology review, and on this latter group but with after exclusion of anaplastic oligoastrocytoma (AOA) with necrosis. As a reference set for glioblastoma, patients from the prospective randomized phase III on glioblastoma (EORTC 26981) were used as a benchmark. **Results.** In 257 of 368 patients central pathology review confirmed the presence of an anaplastic oligodendroglial tumor. Tumors with combined 1p and 19q loss (1p(loss)19q(loss)) were histopathologically diagnosed as anaplastic oligodendroglioma (AOD), were more frequently located in the frontal lobe and had a better outcome. Anaplastic oligodendroglial tumors with EGFR(amp) were more frequently AOA, were more often localized outside the frontal lobe and have a survival similar to glioblastoma. Survival of patients with AOA harboring necrosis was in a similar range as glioblastoma while patients with AOA with only endothelial proliferation had better overall survival. In univariate analysis all molecular factors except loss of 10q were of prognostic significance, however on multivariate analysis a histopathological diagnosis of AOA, necrosis and 1p(loss)19q(loss) remained independent prognostic factors. **Conclusion.** AOA with necrosis are to be considered WHO grade IV tumors (glioblastoma). Of all molecular markers analyzed in this study especially loss of 1p/19q carried prognostic significance, while the others contributed little prognostic value to classical histology.

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