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Relationship between Ki-67 labeling index and survival in high-grade glioma patients treated after surgery with tamoxifen

L Mastronardi [1], A Guiducci, F Puzzilli, A Ruggeri

Affiliations
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Abstract

Background: The Ki-67 is a nuclear antigen expressed in the G1, S, G2, and M phases of the cell cycle recognizable by the monoclonal antibody MIB-1. The Ki-67 labeling index (LI) is considered as a marker of proliferation (growth fraction rate), even if its use as prognostic indicator of cerebral high-grade gliomas is still debated. The aim of this study is to correlate the Ki-67 LI with survival in patients operated on for a malignant glioma and treated postoperatively with tamoxifen.

Methods: Using the MIB-1 antibody, the Ki-67 antigen staining of surgical specimens was obtained in 26 patients operated on for a malignant cerebral glioma. After operation, 9 patients started to receive 40 mg/day, 8 patients 80 mg/day, and 9 patients 120 mg/day of tamoxifen. In 20 cases one or more cycles of i.v. carboplatin was administered. All patients received radiotherapy (4500-6000 cGy).

Results: The overall mean survival rate was 19.8 months and the median 12 months; the 12-month and 24-month survival rates were 47% and 23%, respectively. The Ki-67 LI ranged from 2.3% to 62% (mean 24.1%, median 20.5%). Excluding 2 patients who died during the postoperative period, we analyzed the survival rates of the remaining 24 patients in relation to the value of Ki-67 LI. In relation to the index, patients have been divided into 3 groups, with different survival rates: L) Ki-67 LI < or =10%, with mean survival rate of 30.7 months, M) from 10.1% to 30%, with mean survival rate of 15.8 months, and H) >30%, with mean survival rate of 20.2 months.

Conclusions: Our preliminary observations seem to confirm the efficacy of TAM in modifying the survival of malignant gliomas and seem to indicate that tumors with a lower LI and those with a LI >30% could be associated, if treated with TAM, with a better survival than gliomas belonging to group M, who could have a higher tumor proliferation rate in comparison to group L and a lower response to protein-kinase-C antagonists than group H.