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Basic and Translational
 Investigations

Pediatric glioblastomas: A histopathological and molecular genetic study

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Abstract

Glioblastoma multiforme (GBM) occurs rarely in children. Relatively few studies have been performed on molecular properties of pediatric GBMs. To evaluate the genetic alterations in pediatric GBM (age ≤ 18 years) with special reference to p53, p16 and p27 protein expression; epidermal growth factor receptor (EGFR) alterations and phosphate and tensin homolog gene (PTEN) deletion. Thirty cases of childhood GBMs reported between January 2002 to June 2007 were selected and H&E stained slides reviewed. Immunohistochemical staining was performed for EGFR, p53, p16, p27 and MIB 1 labeling index. Fluorescence in situ hybridization (FISH) analysis was performed to evaluate for EGFR amplification and PTEN deletion. Histopathological features and MIB-1 labeling index were similar to adult GBMs. p53 protein expression was observed in 63%. Though EGFR protein over-

expression was noted in 23% cases, corresponding amplification of the EGFR gene was rare (5.5%). Deletion of the PTEN gene was also equally rare (5.5%). One case showed polysomy (chromosomal gains) of chromosomes 7 & 10. Loss of p16 and p27 immunoexpression was observed in 68% and 54 % cases respectively. In pediatric de novo/primary GBMs, deletion of PTEN and EGFR amplification are rare, while p53 alterations are more frequent as compared to primary adult GBMs. Frequency of loss of p16 and p27 immunoexpression is similar to their adult counterparts. This suggests that pediatric malignant gliomas are distinctly different from adult GBMs highlighting the need for identification of molecular targets that may be adopted for future novel therapeutic strategies.

Key Words: epidermal growth factor receptor, glioblastoma multiforme, p16, p27, p53, pediatric, PTEN

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