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Concurrent sequence variation of *TP53* and *TP73* genes in anaplastic astrocytoma

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ABSTRACT. Disruption or loss of tumor suppressor gene *TP53* is implicated in the development or progression of almost all different types of human malignancies. Other members of the p53 family have been identified. One member, p73, not only shares a high degree of similarity with p53 in its primary sequence, but also has similar functions. Like p53, p73 can bind to DNA and activate transcription. Using PCR-SSCP and gene sequencing, we analyzed the *TP53* and *TP73* genes in a case of a grade III anaplastic astrocytoma that progressed to glioblastoma. We found a deletion of AAG at position 595-597 of *TP53* (exon 6), resulting in the deletion of Glu 199 in the protein and a genomic polymorphism of *TP73*, identified as an A-to-G change, at position E8/+15 at intron 8 (IVS8-15A>G). The mutation found at exon 6 of the gene *TP53* could be associated with the rapid tumoral progression found in this case, since the mutated p53 may inactivate the wild-type p53 and the p73α protein, which was conserved here, leading to an increase in cellular instability.

Key words: Astrocytoma; *TP53* gene; *TP73* gene

