

Molecular Genetics of Meningiomas



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Mini Review

Meningiomas arise from arachnoid membrane of the brain and can be developed in various parts of the brain including skull base and rarely in the ventricular system [1]. They show a rising incidence with age. Most of the cases with neurofibromatosis type 2, develop meningiomas [2]. Neurofibromatosis type 2 gene mutations can be seen in about 60 percent of the cases with sporadic meningiomas [3], so neurofibromatosis type 2 gene mutation, is the most common gene alteration in meningiomas [4]. Neurofibromatosis type 2 tumor suppressor gene's location is on the arm of the 22q chromosome [5]. Immunoreactivity reduction or absence of merlin as the neurofibromatosis type 2 gene product, may also be seen in meningiomas [6]. 4.1B/DAL-1 protein is also detected to be involved in the pathogenesis of meningiomas [7]. In about 70 to 80 percent of meningiomas, 4.1B/DAL-1 loss of expression can be seen [8]. In about 70 percent of meningiomas, loss of heterozygosity which involves the 4.1B/DAL-1 region on 18p chromosome, has been detected [9]. Neurofibromatosis type 2 and 4.1B/DAL-1 genes inactivity, can be occurred in anaplastic, atypical and benign meningiomas. In the more malignant tumor forms, some other gene alterations have been detected which are linked to tumors progression. Gene alterations in CDKN2A, P14ARF, CDKN2B genes on 9p and allelic losses on chromosome arms 17q, 10q, 1p, 14q and 9p are related to this category [10-12]. Having knowledge about the molecular genetics of meningiomas, is of importance to understand their pathogenesis and also to find new treatment options for such tumors.

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