CEREBROSPINAL FLUID RESEARCH

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ECRG-4 expression in normal and neoplastic choroid plexus

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Background

The choroid plexus is a major site of gene expression of esophageal cancer-related gene (ECRG)-4 during development, suggesting that its gene product may be involved in cerebrospinal fluid (CSF) homeostasis. Yet, ECRG-4 is also a novel candidate tumor suppressor gene whose expression is downregulated and is inversely associated with a worse prognosis in several different cancers. Reduced expression of ECRG-4 has been demonstrated in most tumors, including colorectal carcinoma and malignant glioma, to be mediated by hypermethylation of its promoter.

Materials and methods

In this study, samples of normal human choroid plexus (both fetal and adult) and choroid plexus neoplasms (WHO grade I papilloma, grade II atypical papilloma, and grade III carcinoma) were stained with antibodies that we generated to augurin, the gene product of ECRG-4. DNA was then extracted from the tissue, treated with bisulfite, and subjected to PCR using a 217-base pair region encompassing the ECRG-4 promotor to detect methylation.

Results

Both fetal and adult human choroid plexus cells demonstrated a robust positive immunostaining at the apical surface that is consistent with our prior results in human, rat, and mouse brains. In contrast, there was a near-complete absence of immunostaining in all of the choroid plexus neoplasms examined. The choroid plexus carcinoma demonstrated significant methylation of the ECRG-4 promotor region.

Conclusions

Taken together, these data suggest that ECRG-4 is downregulated in neoplasms of the choroid plexus just as has been observed in other central nervous system (CNS) and non-CNS cancers. This is likely due to hypermethylation of the ECRG-4 promotor, as shown in the choroid plexus carcinoma. Further analysis is underway to determine the (1) physiologic and (2) pathophysiologic consequences of ECRG-4 over- and under- expression in the choroid plexus on CSF formation, function, and composition.

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