investigated variants of meningiomas . There was no statistically valid difference, however, in this matter between meningothelial, fibrous and transitionaly types of the tumors.

Tumor cells immunopositive to p53 and bcl2 were found in some examined cases. Cells showing p53 immunoexpression were in atypical meningiomas more frequent than in benign variants of these neoplasms.

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Possible involvement of astroglial expression of cytokines in neuronal degeneration after transient global ischemia in rat brain

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Studies from various laboratories have shown that cytokines may play an important role in ischemic brain injury. Particular interest revolves around the role of the two interleukins: interleukin -1beta (IL-1beta) and interleukin-6. Although these pleiotropic cytokines have been described as having both pro- and anti-inflammatory properties, IL-1beta has been mainly postulated as a mediator in the pathogenesis of ischemic damage, while IL-6 appears mostly involved in the neuroprotective action. The balance between the opposing actions of these two cytokines may be an important element in determining the outcome after ischemic insult.

The aim of the present study was to investigate the effect of transient complete cerebral ischemia induced by cardiac arrest (an experimental model of clinical death), on the pattern of expression of IL-1 beta and IL-6 immunoreactivities (IR) in the hippocampus. The hippocampus and particularly its pyramidal cells of the CA-1 region are well known as vulnerable to ischemia and develop delayed neuronal death over several days after ischemic insult. To identify the cell types expressing the two immunoreactivities we used specific cell markers and combined staining procedures. In the intact brain IL-1beta and IL-6 were mainly localized in neurons particularly in pyramidal and granular cell layers. Ischemic insult resulted in a concomitant induction of IL1-beta and IL-6 immunoreactivities in multiple astroglia, especially in a CA1 region. The number of astroglia expressing both immunoreactivities and the intensity of staining was maximal at 14th day and remained as the same level at 28th day.

Our data suggest that the astroglial IL1-beta and IL-6 may affect the neurodegeneration of CA1 neurons in the ischemic hippocampus. The persisting neurodegeneration suggests however, that the neurodegenerative effects of the prolonged IL1-beta expression (possibly connected, among others with stimulation of arachidonic acid metabolism and/or nitric oxide synthase activity) prevail over the presumed neuroprotective action of IL-6. Although both IL1-beta and IL-6 are known to stimulate the astroglial nerve growth factor (NGF), evidently such NGF, noted also in our experimental conditions, especially in a CA1 zone, seems to be not sufficient or may reach useful levels too late, to protect against neuronal death.

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Spongioblastoma polare. A case report

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A case of polar spongioblastoma occurring in a 25-year-old woman is presented. Immunohistochemical analysis of the tumor cells did not review expression of synaptophysin, neurofilament protein, GFAP and EMA.

Single- stranded conformational polymorphism analysis did not show TP53 mutations in exons 5-8. Differential PCR performed on the tumor tissue confirmed lack of deletion of CDKNaa/p16, and no amplification of MDM2, EGFR, and CDK4 as well.

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Premature aging in man with multiple congenital malformations

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45-year-old mentally disabled man with history of arterial hypertension and legs amputation due to their congenital malformations, was addmitted to the Department of Neurology after sudden loss of consciousness. The patien's status was very severe; he was deeply unconscious with respiratory disturbances and elevated blood pressure to 250/160 mmHg. Neurological examination revealed very narrow, not reactive pupils, initially decerebrate syndrome with limb spasms, later flaccid tetraplegia with decreased deep tendon reflexes. CT-scan showed extensive hemorrhage in brain stem with blood penetration into ventricular system. Twenty hours after admission the patient died.

Microscopial examination revealed numerous gray matter ectopies in brain hemispheres as a result of disturbed neuron migration, lack of anterior part of the corpus callosum and abundant number of pathological blood vessels. We also found changes characteristic for premature aging: granulo-vacuolar degeneration, senile plaques and amyloid fibers in blood vessels wall.

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Amplification of c-erB1 and MDM2 genes in glioblastomas

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Amplification and overexpression of EGFR occur almost exclusively in glioblastomas (38%-58%). In contrast MDM2 gene is amplified in up to 10% of glioblastomas.

In series of 28 glioblastomas we analyzed EGFR and MDM2 gene amplification and the DNA level by differential PCR and gene products were evaluated by immunohistochemistry. Thirteen cases (45%) presented immunopositivity for EGFR. Amplification of EGFR were observed in 9 tumors among which 1 revealed no EGFR immunopositivity. Two tumors with weak EGFR expression showed no gene amplification by differential PCR. The immunohistochemical staining for MDM2 revealed strong immunoreactivity only in 1 case and this tumor also presented MDM2 gene amplification. Our results demonstrate that there is no strict correlation between gene amplification encourtened at the DNA level and at protein level by immunohistochemistry.

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