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Pattern of neuronal loss in rat hippocampus following experimental cardiac arrest – induced ischemia

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The pattern of neuronal loss in rat hippocampus following 10-minute-long cardiac arrest – induced global ischemia was analyzed using the unbiased, dissector morphometric technique and hierarchical sampling. On the 3 day after ischemia, the pyramidal layer of CA1 demonstrated significant, 27%, neuronal loss ($P<0.05$). At this time, no neuronal loss was observed in other cornu Amonnis sectors of the granular layer of the dentate gyrus. On the 14th postischemic day, further neuronal loss in the CA1 pyramidal layer was noticed. At this time, the sector contained 31% fewer pyramidal neurons, than on the 3rd day ($P<0.05$) and 58% fewer than in the cotrol group ($P<0.01$). On the 14 day neuronal loss in other hippocampal subdivisions also was observed. The pyramidal layer of sector CA3 contained 36% fewer neurons than in the control group ($P<0.01$), whereas the graular layer of dentate gyrus contained 40% fewer ($P<0.05$). The total number of pyramidal neurons in sector CA2 remained unchanged. After the 14 day, no significant alterations in the total number of neurons were observed in any subdivision of the hippocampus until the 12th month of observation. Unbiased morphometry analysis emphasized the exceptional susceptibility of CA1 pyramidal neurons to hypoxia/ischemia but also demonstrated significant neuronal loss in sector CA3 and dentate granular layer previously considered as "relatively resistant". Different timing of neuronal drop out in sector CA1 and in sector CA3 and dentate gyrus may implicate the existence of regional related properties, which condition earlier or later reaction to ischemia. However, as the hippocampus has unique, unidirectional system of intrinsic connection, where the major bulk of dentate granular neurons projection targets the CA3 pyramidal neurons which in turn project mostly to sector CA1, the early neuronal dropout in CA1 may result in retrograde transsynaptic degeneration of neurons in other areas. The lack of neuronal loss in sector CA2 can be explained by the resistance of this sector to ischemia/hypoxia and the fact that this sector is not included in the major chain of intrahippocampal connections and hence is not affected by retrograde changes.

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Fetal globoidal leukodystrophy (Krabbe's disease). Neuropathological picture at 20 gestational weeks Case report

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Globoidal leukodystrophy (Krabbe's disease) is a genetically determined metabolic disease induced by deficiency of lysosomal enzyme – galactocerebroside beta-galactosidase. Morphologically, the disease is manifested by demyelination and dysmyelination, gliosis and the presence of globoid cells accumulating galactocerebroside (cells of the mesenchymal origin) in the damaged white matter. The disease may be detected through prenatal examination involving the analysis of galactocerebrosidase activity in amniocytes.

The neuropathological picture of fetal globoidal leukodystrophy at 20 gestational week (GW) is presented. The diagnosis of globoidal leukodystrophy in the first child was the indication to the prenatal examination. Galactocerebroside beta-galactosidase activity in amniocytes was 0.04 nmol/h/mg protein (control 1.1–3.1 nmol/h/mg protein). The fetal development corresponded with GW defined according to the data of last menstruation. The neuropathological examination revealed the presence of cells with abundant cytoplasm and one or several nuclei in some tracts of the spinal cord (posterior, anterior funiculi and anterior part of lateral funiculi). The electron microscopic observation showed that the structure of theses cells was similar to the globoid cells in the child with Krabbe's disease died at 24 months of age. Globoid cells express the reaction to myelin decomposition. In the case presented, these cells were found in the spinal tracts showing a certain degree of myelination at 20 GW.

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Central nervous system damage suggesting mitochondrial disease with the onset in the pre- and perinatal period. Report of two cases

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Despite of advances of clinical diagnostics mitochondrial diseases are still insufficient known and diagnosed just postmortem. We present neuropathological findings of two such cases.
Case 1. A girl born at term by cesarean section, with birth weight 2800 g. Apgar score 8. From the second week of life apnea, bradycardia, hypotonia, attacks of spasms and failure of thrive were observed. Neurologically there were lack of suckling reflex, brisk reflexes and hypotonia. There was increased level of lactic acid in cerebrospinal fluid – 10.7 mmol/l (standard 1.1–1.5 mmol/l). The brain CT revealed symmetric hypodensity in the periventricular white matter and in the basal ganglia. Girl died at 10 weeks of life. Congenital leukodystrophy was suspected clinically.

Neuropathological investigation showed macroscopically softening of the whole brain mostly in the temporal lobes, periventricular white matter and basal ganglia. In the tegmentum of the brain stem as well as in the gray matter of spinal cord there were bilateral, symmetrical necrotic foci. Microscopically there were spongy necrosis, necrosis with or without macrophagic reaction and proliferation of blood vessels in the cortex, periventricular white matter, basal ganglia and gray matter of spinal cord. Well preserved single neurons in necrotic areas were also found.

Case 2. A girl with birth weight 3400 g born at term by cesarean section because of imminent asphyxia. Apgar score were 1-3-7, respectively. During the first 24 hours of life respiratory disorders and severe acidosis occurred. On consecutive days symptoms of infection, anemia, leukopenia and thrombocytopenia as well as failure of liver and kidneys were observed. Neurological examination revealed increased muscular tension and seizures. Cerebrospinal fluid was normal. USG of the brain revealed