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Postischemic encephalopathy – selected pathogenetic aspects.

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Postresuscitation encephalopathy, resulting from global cerebral ischemia due to cardiac arrest is known, first of all, from the anesthesiological literature. Despite of relatively rich bibliography of the subject, the pathogenetic mechanism(s) of this progressive pathological process remains unclear. To elucidate, at least, some of its aspects, a series of studies was undertaken on the model of experimental cardiac arrest in rats.

The morphological observations revealed, that individually variable structural tissue changes were of selective and progressing nature, and greatly evolving in histological pattern during postischemic observation period, ranging from several minutes to one year. Widespread nonspecific neuronal degeneration and neuronal loss, at first localized in selectively vulnerable brain regions, and than involving other cerebral areas, considered as ischemia-resistant, were the most striking pathological feature. In 20% of cases, generalized brain atrophy, more severely expressed in the cerebral cortex and subcortical white matter, was found. The appearance of the "antibrain" antibodies in the blood sera of experimental animals may be indicative of autoimmunological mechanism of generalized neuronal loss. This is supported by biphasic blood-brain barrier changes, appearing both immediately after ischemia and than during the first postischemic week.

Deep abnormalities in the morphology of brain microvessel system present both in early and late postischemic stages are clearly pointing out to importance of local vascular factors in the development of the brain damage. The same role, although limited to the early postischemic period, may be attributed to disturbed balance between excitatory and inhibitory neurotransmitter systems.

During the whole observation period a tendency for intravascular thrombocyte aggregation was found. In some cases single platelets were observed in extravascular location. This tendency indicative of permanently disturbed endothelium-thrombocyte relations, may point out to microthrombi formation, operating also in late postischemic period.

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Etiology in juvenile infarction – the neuroradiological contribution

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Apart from the ability of imaging procedures to define pathologies mimicking ischemic infarction and to solve topical problems, it is the impact on classification which is of major interest in the young. Obvious reasons for that are the broadness of the etiological spectrum (with the demand of special therapeutic measures in individual cases) and raised life expectancy in this special subgroup of stroke patients.

Personal experience is presented which covers more than 160 patients (<45 y), 95% of them being angiographed either conventionally or by DSA.

In 100 consecutive patients who were followed up, nearly 30% had an embolic infarction as defined by CT/MRI ("territorial" or large ganglionic infarction) despite a source of embolism could not be established after full cardiological and angiographical work up. Detection of the embolus depends strongly on the timing of angiography, being probable only during a few hours after the event. In this group, the recurrence rate was 0. Because embolism was observed as a complication of pneumonia and pulmonary AVM, the search for sources of emboli may include the pulmonary vessels, depending on findings of chest views.

Small, deeply located thalamic infarcts were seen not only together with typical risk factors for small vessel disease (hypertension/diabetes), but were caused also by migraine, embolism and arteritis. Dissection in C1/M1 (3 patients) and circumsript stenosis of M1 represented atypical causes of large ganglionic infarction and were detected angiographically.

In cases of migrainous infarction as identified by CT/MRI (12 patients), hemianopsia and all the motor and sensory deficits, even aphasia could be attributed exclusively to lesions in the supply area of the posterior cerebral artery (cortical, thalamic, capsular). The angiograms, if done early showed either proximal occlusion, or a unique finding, resembling the string of beads pattern with circumsript widenings and constrictions in the proximal course of the vessel.

There were several cases of embolism in whom the source in course of the internal carotid artery could not be detected by ultrasound examination. The etiologies were: dissection, aneurysm (with fibromuscular dysplasia and recurrent tonsillitis, respectively), arteritis (presumed, in C2 and transmitted from the tonsillar niche, respectively).

In four cases, which were not be classified despite numerous diagnostic measures and relapsing course, arteritis could be diagnosed unequivocally based on angiographic findings (widespread constrictions and widenings in middle sized arteries).

Conclusions:

1. Angiography seems to be dispensable only in cases of a. bad condition, b. proven emboligenic heart disease, c. lacunar stroke in the presence of longstanding diabetes and/or hypertension, d. typical migrainous infarction.
2. There are much more embolic infarctions, than well-established sources of emboli.
3. The yield of MR angiography remains unsettled yet, but in some cases lesion characterization is sufficient (in personal experience: 3 D time of flight after administration of Gd-DTPA).

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Accumulation of Alzheimer's β-amyloid protein precursor in rat brain after cardiac arrest-induced complete cerebral ischemia

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Extracellular β-amyloid deposition in the brain is a common feature of the pathological lesions in Alzheimer's disease (AD). We used various antibodies to the ß-amyloid protein precursor (APP) of AD to study changes in the extracellular distribution of APP in experimental ischemic brain injury. Rats underwent 10 min of global cerebral ischemia, with survival time up to 7 days. The APP-immunoreactivity was observed not only intracellularly, within neuronal cells and, less often, glial cells, but also extracellularly, in the perivascular areas. Perivascular APP deposits formed irregular, often asymmetric, well-delineated areas, which were usually located close to the blood vessels, predominantly capillaries. Only rarely did these deposits encircle blood vessels, forming round, perivascular cuffs. Neuropil alterations were more advanced in the rats with concomitant extracellular APP deposits.