ficant loss of microglial cells was noted in all structures of the hippocampal formation except layers II-IV of the entorhinal cortex. The largest decrease in the microglia density was observed in the dentate gyrus (from 226.4 ± 40.7/mm^2 in the control group to 48.9 10.2/mm^2 in AD) and in CA4 sector of the hippocampus (from 140 26.11/mm^2 to 44.4 ± 9.6/mm^2; $p<0.05$). These observations suggest two conclusions: 1. Microglia can secrete an amyloid but it is not its major source. 2. The microglia reaction is not only the response to an amyloid deposition but also to the neuronal degeneration.

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Neuropathology of acquired immune deficiency syndrome in Polish material

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Pathomorphological analysis of the central nervous system in 100 cases of acquired immune deficiency syndrome is presented. The patients were treated in the course of 1987-1995 either in the Institute of Infectious Diseases, School of Medicine, Warsaw or in the AIDS Centre of Wolski Infectious Disease Hospital in Warsaw. The special attention was paid to the incidence and pathomorphological characteristics of AIDS-related and AIDS-associated pathology in the examined cases as well as to their concomitance with various opportunistic infections and neoplastic processes. Neuropathology of Polish material was compared with that described in the comparable foreign reports.

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Neuropathological changes within the CNS in pregnant rabbits with experimentally evoked antiphospholipid syndrome

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A post-mortem neuropathological investigations were carried out on 16 female rabbits of New Zealand race, aged 5-6 months, 3900 - 4600 g of body weight. A material was divided into 3 groups: Group I - 6 pregnant rabbits with experimentally evoked antiphospholipid syndrome; Group II - 5 non-pregnant rabbits with evoked antiphospholipid syndrome; Group III - 5 non-pregnant control animals without antiphospholipid syndrome. Antiphospholipid syndrome was evoked by subcutaneous injections of cardiolipin in increasing dose from 0.3 to 0.4 ml (2250 - 3000 µg) with adjuvant (2% solution of aluminium hydroxide) mixed 1:1. Non-pregnant rabbits were immunized on 0 - 4 - 8 - 12 - 16 - 20th day, whereas pregnant animals on 10 - 14 - 18 - 22 - 26th day. Rabbits were delivered by cesarean section on the 30th day of pregnancy in general anesthesia with intravenous Vetbutal injection. Macroscopic appearance of the CNS was within the norms. Microscopic evaluation revealed three types of changes. There were as follows: 1) perivascular inflammatory infiltrates consisted of mononuclear cells, especially of lymphocytes, located mainly around vessels larger than 30 µm in diameter; 2) intracerebral infiltrates formed with microgliotic and histiocytic cells; 3) round-cell infiltrates within the leptomeninges. In general, above described changes were more frequent in cerebral cortex, but if they had appeared within the white mater they showed tendency to location in the vicinity of ventricular system. Perivascular infiltrates and infiltrates within the leptomeninges appeared more frequently in rabbits of group I compared with group II. All described neuropathological changes were significantly more frequent in group I and II in comparison to control group ($p<0.05$). A necrotic and hemorrhagic changes were not found. The preliminary observations revealed an efficacy of applied model of antiphospholipid syndrome in the development of neuropathological changes both in pregnant and non-pregnant rabbits, in former - the changes in the CNS were more intensive.

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Proposal of classification for CNS involvement by leukemias and lymphomas . Clinical and morphological aspects

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The clinical and post-mortem neuropathological investigations were carried out on 133 randomized patients of both sexes aged 17 to 73 years (mean age 43.6 years) treated and deceased due to acute myeloblastic leukemia (AML) - 49, blastic phase of chronic myelogenous leukemia (BPCML) - 31, acute lymphoblastic leukemia (ALL) - 11 and high grade non-Hodgkin lymphomas (NHL) - 42 cases. Based on accessibility of various central nervous system (CNS) regions for neoplastic cells, the following grading classification has been proposed for CNS involvement by leukemia and lymphoma: grade I - leukostasis within the cerebral and meningeal vessels as a result of white blood cells count evident increase and of local activation of adhesion and aggregation; grade II - meningeal infiltrates with or without concomitant leukostasis; perivascular infiltrates originated from intracerebral vessels filled with leukostasis (grade IIIa) or related to meninges involved by neoplasm (grade IIIb); intracerebral infiltrates invading nervous tissue originated from perivascular infiltrates related to leukostasis (grade IVa) or to meninges (grade IVb).

Very intensive leukostasis may cause hyperleukocytic syndrome leading to vertigo, tinnitus, gait disturbances, somnolence. Meningeal infiltrates are the main source of neurological deficits. Due to their larger frequency in lymphomas, neurological manifestations are more common for lymphoma compared to leukemia. Perivascular infiltrates appeared to be "clinically silent" however, neurological manifestation may be related to hyperleukocytic syndrome (grade IIIa) or to meningeal infiltrates (grade IIIb). Intracerebral infiltrates invading nervous tissue related to meningeal involvement can cause clinical manifestations of focal CNS lesion.

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Neuropathology of central pontine myelinolysis

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Central pontine myelinolysis (CPM) also designated osmotic demyelinating syndrome is a rare, in many instances fatal condition of the central pons with or without associated extrapontine demyelinating lesions. CMP is classically described in alcoholics, other malnourished individuals after correction of severe hyponatremia. We report here clinical and neuropathological data on 15 cases of CPM (14 males and one female age 26 - 62 years, duration of alcohol abuse 8 - 23 years). In some cases, disturbances of sodium (9 cases) and potassium (6 cases) serum levels were observed in a terminal stage of disease. By autopsy, diffuse steatotic changes in the liver were found in all cases. Routine and immunohistochemical characteristics of demyelinating lesions in the pons is described. No extrapontine demyelinative lesions were found in this series of cases.

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Neuropathology and immunohistochemistry of Shy-Drager syndrome

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