

ON SOME CEREBROSPINAL FLUID CHANGES DURING THE ACUTE STAGE OF CEREBRAL INFARCTION

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Cerebrovascular disease being a socially significant disease imposes a deep obligation upon us to study more profoundly the etiological, epidemiological and social factors as well as to look for new clinical and biochemical criteria for exacter differential diagnosis with this patients' contingent.

In the present paper we report a combined spectrophotometric, cytologic and electrophoretic investigation of the cerebrospinal fluid in patients during the acute stage of cerebral infarction.

Material and methods

Cerebrospinal fluid changes were followed-up with 60 patients hospitalized in the Neurologic Clinic of the Higher Institute of Medicine, Varna. Of them, 30 patients were with a red, and 30 ones — with a white infarction. Diagnosis was confirmed by means of a thorough clinical investigation as well as by paraclinical and CT analyses. It was pathologically-anatomically verified. Cerebrospinal fluid was taken by lumbar puncture in a lying position from the first till the twentieth day after brain accident.

Results and discussion

Cerebrospinal fluid was clear, colourless, transparent and without any fibrin network in the cases with white cerebral infarction. A slightly expressed xanthochromia was established in two patients with extensive brain infarction. Bloody and xanthochromic cerebrospinal fluid was found out in 10 patients (i. e. one third) of the group with red infarction. Xanthochromia was visually estimated in 6 per cent but spectrophotometrically in 46 per cent of white infarction patients. The corresponding percentages were 17.5 and 68, respectively, when verified cases with red infarction were concerned. Xanthochromia was of mixed type: haemorrhagic and congestive. Bilirubin components prevailed with the white cerebral infarction while at the onset oxyhaemoglobin and methemoglobin, and after one week also bilirubin predominated with the red one (1, 3, 4). Cytological examination showed normocytosis in about 75 per cent of white cerebral infarction patients. Pleocytosis was slightly expressed (between 18 and 23.10⁶/l) reaching its maximal level from the second till the seventh day after the onset of accident when the rest patients were concerned. Erythrocytorachia was an important finding in red cerebral infarction. It va-

ried between 1 and 13.0 · 10⁹/l. Cellular reaction was mainly of lymphomonocytes. A considerable polymorphism was presented consisting of mononuclear cells, mainly of lymphocytes, monocytes, reticulocytes and plasmocytes as an expression of leptomenigeal irritation. Macrophagocytosis was weak and due mainly to lipophages in white cerebral infarction patients. However, it was more considerable in red cerebral infarction ones. At the beginning, from the first till the seventh day, erythrophages, and later on, after 10 — 14 days also hemosiderophages could be observed. A slightly expressed hyperproteinorachia (between 0.4 and 1.0 g/l) was found out in 40 per cent of white cerebral infarction patients. However, hyperproteinorachia was more expressed and established in 20 red cerebral infarction patients (2/3 of the cases). It reached its maximal levels between the second and eighth day and then normalized strongly after about 20 days. At protein-cellular dissociation was observed in about 28 per cent of brain infarction patients. No specific phoregrams were estimated by means of the electrophoretic study. The dysproteinorachia observed by us was on the account of the increased gamma globulins. The phoregrams were close to the serum ones in patients with red cerebral infarction. Chlorides and sugars in the cerebrospinal fluid were within normal limits. Thus our results were in concordance with data reported by other investigators (2, 4 — 6, 8 — 11). According to Chankov (7), quantitative analysis of microelectrophoretic fractions demonstrates that gamma globulins are elevated at the average by 36 per cent in the half of the cases with encephalomalacia. Most often, there is an increase of alpha₁ and gamma globulins simultaneously with a decrease of albumins and alpha₂ globulins in cases with cerebral embolism.

We can conclude that cerebrospinal fluid changes during the acute stage of cerebral infarction although not strictly specific help the early differential diagnosis between a red and a white infarction of the brain and thus the correct and timely treatment of this patients' contingent.

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О НЕКОТОРЫХ ЛИКВОРНЫХ ИЗМЕНЕНИЯХ В ОСТРОЙ СТАДИИ ИНФАРКТА МОЗГА**В. Дренски, В. Колчев****РЕЗЮМЕ**

Авторами проведено спектрофотометрическое, цитологическое и электрофоретическое исследование ликвора у 60 больных в стадии инфаркта мозга. При белом мозговом инфаркте визуально установленная ксантохромия наблюдалась у 6% больных, а спектрофотометрично — у 50% больных. При красном инфаркте мозга в одной трети случаев наблюдался кровянистый ликвор с ксантохромией (у 17,5% больных устанавливается визуально и у 68% при спектрофотометрии). Ксантохромия смешанного типа — геморрагическая и застойная. Эритроцитрафия является важной находкой при красном инфаркте. Она варьирует от 1 до 13 $\times 10^9$ /л. При красном инфаркте в первые дни устанавливаются эритрофаги, а через 10—14 дней и гемосидерофаги. На фореграмме устанавливается диспротеинрафия за счет увеличения гамма-глобулинов.