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CLINICAL CHARACTERISTICS OF PATIENTS WITH HYPERTENSIVE ENCEPHALOPATHY

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ABSTRACT

Hypertensive encephalopathy (HE) presents with motor, cognitive, psychic and pelvic-reservoir syndromes as well as with dysarthric, extrapyramidal and gate disturbances. The author analyzed the clinical picture of HE and related clinico-diagnostic, pathogenetic, and therapeutic problems. Nine patients, 5 females and 4 males aged between 43 and 72 years (mean age of 52,5 years) with a cardio-cerebral form of hypertension were examined. The diagnosis of HE was proved clinically as well as by electrocardiography, Doppler sonography, x-ray, magnetic resonance tomography, polysomnography, angiography and neuropsychological tests. Symptoms of a coordination and pyramidal-extrapyramidal syndrome and only of a coordination syndrome were established in 3 patients each, while symptoms of pyramidal and frontal corneal syndrome and of pyramidal and coordination syndrome - in 2 patients each. All the patients presented with cognitive and memory disorders. The antiphospholipid antibodies were elevated in 6 patients. Treatment with vasodilatators, calcium antagonists, neuroprotective means, antioxidants, and cholinesterase inhibitors did not cause any subjective and objective improvement of the neurological deficit. The diagnostic criteria of HE clinical signs wee not well defined concerning the degree of their manifestation and specificity and they could indicate the onset of other nervous system diseases, too.

Key words: hypertensive encephalopathy, pathophysiology, clinical syndromes, diagnosis, treatment

The peculiarities of the clinical course of hypertensive encephalopathy (HE) present with motor, cognitive, psychic and pelvic-reservoir syndromes as well as with dysarthric, extrapyramidal and gate disturbances (1,2). They are related with an interruption of the link of prefrontal cortex, basal ganglia and thalamic cortical pathways. Advanced age, hypertension, diabetes mellitus, tobacco smoking, hyperhomocysteiuria, hyperfibrinogenemia and other pathogenetic moments leading to hypoperfusion are considered risk factors for HE. In this aspect, obstructive sleep apnea, congestive heart failure, cardiac arrhythmia and orthostatic hypotension play a crucial role (5). The treatment is symptomatic and consists of drug prevention and continuous control of risk factors. The ischemic cerebral lesions are divided into complete infarctions (lacunar and micro-infarctions) and incomplete ones located in the deep white matter. The lacunar infarctions affect the basal ganglia, internal capsule, thalamus, pons, radial corona and semioval center (7,8). The pathological alterations of the white matter include demyelinization, astrocitary gliosis, dilatation of the perivascular spaces and reduction of oligodendrocytes. The incomplete white infractions correspond to the penumbra of the complete lacunar infarctions and are characterized by a typical hypoperfusion mechanism.

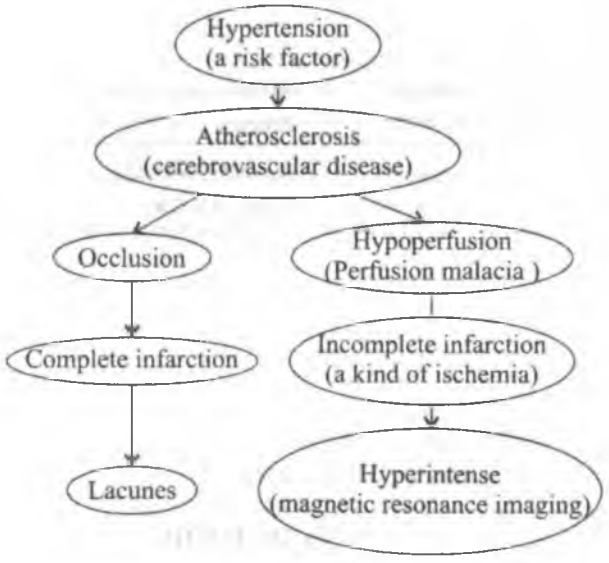

Fig. 1. Pathogenesis of HE

The pathogenetic mechanisms inducing microcirculatory disorders include disturbances of the hemorheologic factors, increased vascular resistance, altered autoregulation, endothelial changes, dysfunction of the blood-brain barrier and dilatation of the perivascular spaces. As a whole, they

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influence upon neuronal metabolism and thus on glia, oligodendrocytes, astrocytes and endothelial cells and result in myelin laceration preceding the vascular ischemia. This selective reduction of the cellular elements appears in case of moderate hypertension and is accompanied by ischemia known as incomplete infarction because of the decreased autoregulatory reserve and stenosis of both arteries and arterioles.

hypertrophy (by means of ECG); tobacco smoking and alcoholism; changes of the immune status; lipid profile, and overweight. The following methods were applied: anamnesis, somatic status, and neurological status; blood pressure control; clinical picture, biochemistry, serology, immunology; Doppler sonography, computer tomography (CT) and MRT of the brain; angiography (in some patients only); neuropsychological examination and polysomno-

Table 1. Diagnostic patterns of HE patients

Patients/ Age (years)	Diagnosis prior to admission	Primary neurological syndrome	Clinical pict-ure, biochem-istry, serology	ACL IgG IgM	MRT-T2 image	Treatment
H.T.K., 60	TCBFD[1] in RMCA[2]	pyramidalcoordination dementia	normal	normal	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
P.M.Z., 49	Vascular dementia	pyramidal- extrapyramidal cerebellar	normal	IgG IgM	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
A.D.T., 49	TCBFD[1] in VBS[3]	coordination pyramidal cognitive	normal	IgG	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
T.H.B., 64	Demyelinizin gprocess	quadripyrami-dal, cerebellar, cognitive	normal	IgG	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
D.G.D., 55	lateral amyotrophic sclerosis	peripheral nerve, pyramidal	normal	normal	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
S.V.D., 48	lateral amyotrophic sclerosis	peripheral nerve, pyramidal	normal	normal	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
T.P.T., 61	TCBFD[1] in RMCA[2]	pyramidal coordination dementia	normal	IgG IgM	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
I.V.D., 45	multiple sclerosis	coordination pyramidal dementia	normal	IgG	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive
K.D.S., 45	multiple sclerosis	pyramidal, cerebellar, cognitive deficit	normal	normal	hyperintense lesions	corticosteroids, antiagr. nootropic, antihypertensive

Abbreviations: TCBFD[1]- transient cerebral blood flow disturbances; RMCA[2] - right median cerebral artery; VBS[3] - vertebro-basilar system

The purpose of the present paper is to analyze the variety of the clinical picture of HE that creates clinico-diagnostic, pathogenetic, and therapeutic problems.

MATERIAL AND METHODS

The study covered 9 patients, 5 females and 4 males aged between 43 and 72 years and at a mean age of 52,5 years, with a cardio-cerebral form of hypertension proved clinically as well as by electrocardiography (ECG), Doppler sonography, x-ray and magnetic resonance tomography (MRT). The following risk factors were looked for: heart diseases such as ischemic heart disease and left ventricular

raphy. A control group of 10 patients with hypertension was examined, too.

The diagnostic patterns of HE patients were summarized on Table 1.

RESULTS AND DISCUSSION

The occurrence of these syndromes on the background of relatively low values of the arterial hypertension represents an essential peculiarity of their clinical course in case of preventive treatment and during the progression of the disease. The role of the neuroimaging methods and especially of MRT for the sensitive detection of isointense,

hyperintense and hypointense manifestations in the white matter of the brain when describing the non-specific symptoms should be emphasized.

The symptoms of a coordination and pyramidal-extrapyramidal syndrome were established in three patients, only of a coordination syndrome in other three patients, while two patients presented with pyramidal and frontal corneal syndrome and two ones - with pyramidal and coordination syndrome. Cognitive and memory disorders were observed in all the patients. Elevated values of antiphospholipid antibodies were established in six patients. The therapy included vasodilatators, calcium antagonists, neuroprotective means, antioxidants, cholinesterase inhibitors, etc. No subjective and objective improvement of the neurological deficit was observed during the clinical follow-up lasting between one and three years. The aberrations of the immunological parameters and MRT findings were not influenced during the dynamic monitoring of the patients.

HE patients with initial cognitive disorders make, usually, the diagnostic process difficult after the appearance of focal neurological symptoms. These cases remain either not diagnosed at all, or diagnosed with a nervous system disease of inflammatory, degenerative or some other nature (5,6,7,8). Their early recognition requires maximal clinical, dynamic radiological, immunological and other examinations. This fact is related with the timely diagnosis and prevention of the patients suffering from hypertension at an early age (4).

According to the clinical picture, HE patients can be divided into two groups. The first group presents with an acute sensory-motor deficiency (a pure motor plegia, pseudobulbar palsies and other lacunar syndromes). The subacute manifestations include cognitive signs, personality and psychic disturbances, gate disorders, motor dysfunctions and pelvic-reservoir syndromes. Besides moderate focal signs prevail such as hand paresis, central facial hemiparesis, reflectory asymmetry, Parkinson syndrome, small-step gate, coordination disorders, pyramidal-extrapyramidal, pyramidal-coordination syndromes, etc. (1,2,3,4). Besides the clinical diagnosis includes a cognitive syndrome, memory deficit, behavioural and psychological symptoms, sometimes a beginning with transcortical sensory aphasia, apraxia and focal lesions detected by MRT, emotional deficit, reduction of attention and association processes along with progression of gate disorders, initiativeness, balance, rotation around the body and mobility.

The presence of certain immunological aberrations such as these of the antiphospholipid antibodies in two thirds of the cases suggests the involvement of an autoimmune mechanism in the pathogenesis of the disease in these patients (5,6).

The diagnostic criteria of HE clinical signs remaining not defined concerning the degree of their manifestation and specificity could indicate the onset of other nervous system diseases mentioned above. One should take into consideration the criteria offered by the National Institute of Neurological Disorders and Stroke (NINDS) including a clear delineation between the cognitive syndromes and relevant radiographic images of cortical and subcortical structures in case of existing risk factors mentioned above (2). However, one does not profoundly look for an autoimmune pathogenetic relation between the established symptoms and syndromes that could contribute to the improved prognosis and more successful treatment of these pathological conditions.

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